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A systems-level perspective of the flexion-relaxation phenomenon in the lumbar spine

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by

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ABSTRACT

Standard anatomic classifications such as "trunk", "lower limbs", "upper limbs" can be misleading regarding the functional role and influence that the tissues in these body regions may play in adjacent body regions. In particular, much of the spine biomechanics literature has considered the lumbar spine in isolation, neglecting to account for the influence of the tissues of the lower extremities (muscles, ligaments and fascia) on the performance of the lumbar region of the torso. Some previous literature supports a systems level (i.e., trunk, pelvis and lower extremities) approach for better understanding of trunk stability during flexion-extension motions. The current study presents a new musculoskeletal model of the active spinal stability system that includes the local system (e.g., multifidus muscles) and global system (e.g., lateral erector spinae, rectus abdominis muscles etc.) as proposed by Bergmark (1989), but then adds a super global system that considers the influence of the lower extremity tissues on the responses of the lumbar region. This innovative model was verified throughout *in vivo* experiments involving human subjects that included three different physical exertion tasks that stressed the low back and the lower extremities in different ways to explore these important interactions.

The empirical work in this dissertation focused on gathering data from the local, global and super global biomechanical systems before and after three 10 minute exercise protocols and then during a 40 minute recovery session. Twelve participants performed three separate experiments (three protocols) on different days: Protocol A- alternately perform 25 seconds of full trunk flexion and 5 seconds upright, relaxed posture; Protocol B- alternately perform 25 seconds of isometric exertion in a 45 degree trunk flexion posture and 5 seconds upright, relaxed posture; and Protocol C- consecutively perform 25 seconds of full trunk flexion followed by 5 seconds of upright, relaxed posture followed by 25 seconds of isometric exertion in a 45 degree

trunk flexion posture and 5 seconds upright, relaxed posture. Kinematic and physiological measures were recorded before during after these protocols as well as during the recovery period. In addition, a variable describing the level of fixation of the pelvis was considered to allow for a direct evaluation of the role of the pelvis/lower extremities on the performance of the lumbar region during these exertions: 1) lower extremity restricted stooping posture (pelvis and thigh restriction) and 2) free stooping posture. The data collected in these experimental trials included the peak lumbar flexion angle, the peak hip flexion angle, the peak trunk flexion angle, the EMG-off angle (i.e., flexion-relaxation), and the average normalized integrated electromyography (NIEMG) for the agonist muscles (lumbar extensors (multifidus and iliocostalis)), the antagonist muscles (lumbar flexors (rectus abdominis and external obliques)) and the lower extremity synergistic muscles (gluteus maximus and biceps femoris).

The results of *in vivo* experiments, focused on the role of the pelvis/lower extremities in trunk flexion-extension, showed a 6.4% greater lumbar flexion angle (36° vs. 38.3°), a 10.2% greater (or later) EMG-off angle in multifidus (31.6° vs. 34.8°), and a 8% greater EMG-off angle in the iliocostalis (30.6° vs. 33°) in the restricted stooping posture than in the free stooping posture. Collectively, these results suggest that additional passive moments about the lumbar spine are generated in the restricted stooping posture because of the relative fixation of the pelvis that is seen during the restricted stooping condition. Consistent with these results, 22% greater lower extremity activation (10.5% MVC vs. 8.2% MVC) was observed in the free stooping posture, as compared to the restricted stooping posture. This additional lower extremity muscle activation acts to stabilize the pelvis (the foundation of the spinal column) and generate passive moments in low back through the lumbodorsal fascia. Consequently, the enhanced pelvic stability and passive moments in the low back generated by the lower extremity

active system (i.e. the super global system) led to the an 8% lower low back muscle activation level (15.1% MVC vs. 16. 3% MVC) in the free stooping condition. In addition, under the abnormal low back conditions (after protocols), the agonist muscles showed significant increases in both the free stooping posture and the restricted stooping posture (15% in both) to maintain spinal stability, but the synergist only increased in the free stooping (22%, 11.2% MVC vs. 13.7% MVC) (no difference in the restricted stooping posture). To summarize, these results indicate a significant role of the tissues of the larger super global system as a trunk stabilizer by immobilizing the pelvis during trunk flexion-extension motions and increasing the stiffness of the trunk systems by enhancing tension of the lumbodorsal fascia.

Regarding the effects of the 10 minute protocols on the biomechanical responses, results showed greater full lumbar flexion and deeper biomechanical equilibrium point between passive tissues and external moment (i.e., EMG-off angles) than the baseline (initial measure) after Protocol A: full lumbar flexion increased 7%; EMG-off angle increased 7.2% in multifidus and increased 7.8% in iliocostalis. In Protocol B the trends in the dependent variables were opposite to those seen in Protocols A: full lumbar flexion angle decreased by 4% and the EMG-off angles decreased by 4.9% in the multifidus and by 6.3% in iliocostalis. Protocol C (the mixed protocol) generated similar, but less pronounced results as compared to Protocol A: full lumbar flexion increased by 3.7%; EMG-off angles increased by 3.7% in multifidus and by 5.9% in iliocostalis. The results of Protocol A and B are consistent with the results of previous studies of these responses and demonstrate important biomechanical effects that need to be considered when modeling the lumbar spine in full or near full-flexion postures. Protocol C was a condition that had not been considered in previous studies and these results indicate that the result of a mixed effort protocol may depend on the relative intensity of the passive vs. the

active fatigue. In the current study the passive tissue fatigue appears to have dominated since the results of Protocol C are somewhat similar to those seen in Protocol A. In all three protocols there appears to have been significant compromise of the passive spinal stability system, as the muscle activities in agonist muscles and synergist muscles were significantly increased in all three protocols illustrating an increased need for active control of the lumbar region.

In terms of the recovery process, the *in vivo* experiment, comparing characteristics of the recovery phase in three protocols, showed longer recovery time after the passive tissue elongation protocol (not fully recovered until 40 minutes of rest in all variables) than the muscle fatigue protocol (recovered after 5 minutes of resting in all variables) and the combined protocol (not fully recovered until 40 minutes of resting for the full lumbar flexion angle and the EMG-off angle; fully recovered in agonist muscle activation after 40 minutes of resting; and fully recovered in the synergist muscles after 5 minutes of resting). The results suggest that the slow recovery of the viscoelastic tissues caused by the prolonged stooping of Protocols A and C may lead to periods of spinal instability because of the abnormally lax passive tissues. While not a direct results of this study, these results may indicate an increased risk of injury during this period of passive tissue remodeling. Also, the enhanced activation in the synergist muscles (i.e., super global system) and depression in the antagonist muscles during the recovery session suggest an interaction mechanism between antagonist and synergist which may be planned in skilled motor programs before the initiation of the movement. Meanwhile, contrary to the results of passive tissues elongation protocol, the muscle fatigue protocol showed relatively quick recovery in all responses measures, but higher levels of muscle activity increase immediately after the protocol: Protocol B (agonist: 14.2%; synergist: 12.5%) vs. Protocol A

(agonist: 9.2%; synergist: 4.7%) and Protocol C (agonist: 11.5%; synergist: 5.1%)). In all three protocols, the super global system (i.e., synergist) showed a recovery pattern that was quite similar to the agonist muscle response.

The results of the theoretical modeling and experimental validation components of the current study indicate that a new musculoskeletal model with a more "systems-level" perspective is necessary to fully understand the biomechanical response of the lumbar spine during full flexion and near full flexion exertion. This study has filled a void in the literature in that it addresses 1) the role of the super global system (i.e., lower extremity) in both normal and abnormal condition, 2) the effect of combined effect protocol (both laxity of the passive tissues and fatigue of the active tissues), 3) differences in the biomechanical responses as a function of the type of fatigue developed (passive tissue, active tissue, combined passive and active tissue fatigue), and 4) dynamic and variable responses of the chosen biomechanical measures during recovery. The results of this new systems-level biomechanical model can be used to develop a new EMG-assisted model of spinal loading and spinal stability as well as guidelines for designing safer working environments that can lower the risks of musculoskeletal injury to the low back.

Chapter 1 – INTRODUCTION

1.1. Low Back Disorders

During the last century, a number of scientists and engineers have worked to understand the nature of low back pain (LBP) and provide practical methods for prevention. Despite their devotion the lifetime incidence of LBP is still considerable in modern society (Walker, 2000). The U.S. Department of Labor (2004) reported that LBP is a leading cause of missed work days. However, knowledge about the association between LBP and work activities seen in the industrial setting is still not fully understood.

Risk factors for LBP can be placed in the following categories: (1) psychosocial factors; (2) lifestyle factors; and (3) mechanical factors. First, the psychosocial factors, including job demands and control, social support, individual characteristics, and so on, were shown to have an association with musculoskeletal disease in a review study (Bongers et al., 1993). They suggested that the perceived stress by workers may be a factor in the process of developing musculoskeletal disorders (MSDs) in that the psychosocial factors at work could influence the mechanical load by modifying the lifting posture and strategy (i.e., lifting kinematics and kinetics). Second, Deyo and Bass (1989) suggested associations between LBP and lifestyle factors such as smoking and obesity even after controlling other possible underlying factors such as age, education, exercise level, and employment status. They noted that some of these risk factors (e.g., obesity) may have direct effects on the lifting kinematics and kinetics because the additional body fat adversely impacts the loading of the low back muscles and the spine. In summary, the causation of those two risk factors is still not clear and needs to be clarified.

Mechanical risk factors have been widely investigated and shed light on the association between the exposure variables in the industrial setting and MSDs. The exposure variables in

ergonomic epidemiology can be conceptually divided into the following: (1) posture: the position of the body parts; (2) motion/repetition: the change of body part and its frequency; (3) material handling: the object being handled by workers; (4) demands: work/rest relationships; and (5) external factors: vibration, climate, etc. (Hagberg, 1992). Similar to this conceptual model, the epidemiologic and biomechanical studies have shown an association between LBP and occupational requirements, including heavy lifting, static stooping, awkward trunk postures, whole-body vibration, etc. (Andersson, 1981; Punnett et al., 2005). A prospective study conducted by Adams and Dolan (2005) also suggested that the excessive mechanical stresses on low back during manual lifting or tasks are still an important and controllable cause of LBP by demonstrating how mechanical loading on low back tissues can precipitate spinal injuries.

1.2. Prolonged stooping in industry

Static flexion postures, hyper-flexion of the lumbar spine and repetitive lifting have been given deserved attention to show the underlying injury mechanism in the low back among several factors regarded as mechanical stressors on the low back. These mechanical stressors on low back can be named as the 'stooping' posture, which is frequently and widely accepted term in human motion. In many industrial settings, workers perform a task for prolonged periods, manually lifting or moving products repetitively (e.g., assembly line workers, farmers in crop production and concrete workers in the construction field, etc.). As an example, farmers harvesting ground crops (cucumbers, sweet potatoes, melons, peppers, tomatoes, etc.) are asked to perform static stooped postures, heavy lifting from ground level and long-distance carrying from the field (Meyers et al., 2003; Jin et al., 2009). Research has shown that workers subjected to prolonged stooping and repetitive lifting have an increased risk of low back pain (LBP)

(Goldsheyder et al., 2002; Kelsey et al., 1984; Magora, 1973; Marras et al., 1993; Rosecrance et al., 2006). Studies have further shown that prolonged static stooping in a full flexion posture can accumulate stress on low back passive tissues and precipitate the development of LBP (Solomonow et al., 2003c; Solomonow, 2004; Shin & Mirka, 2007). Also, repetitive lifting in a stooping posture also develops muscular fatigue in low back muscles and increases the bending moment acting on the low back (Dolan & Adams, 1998).

1.3. Flexion-relaxation in the lumbar spine

Under task conditions requiring a static full flexion posture or dynamic lifting over 60° trunk flexion, an important biomechanical characteristic known as flexion-relaxation phenomenon (FRP) occurs (Floyd and Silver, 1951, 1955; Shin et al., 2004). The FRP has been explained as a synergistic load-sharing mechanism between active tissues (i.e., muscles) and passive viscoelastic tissues (e.g., ligaments, tendons, intervertebral discs, etc.) in the low back (Fick, 1911; Schultz et al., 1985). The active muscle force can be estimated by electromyography (EMG), detecting the electrical potential generated by muscles, using electrodes placed on the skin over the target muscle. As the trunk flexion angle increases, the superficial low back muscle activities estimated by electromyography (EMG) increase in order to compensate for increased external moment. However, at some point during the flexion motion passive tissues are placed in tension, and deep low back muscles activated, and begin to offset the external torque. As flexion continues the trade-off accelerates until at peak flexion there is no muscle activity in the superficial low back muscles and the load is carried completely by the passive tissues and deep lumbar erector spinae and quadratus lumborum (Andersson et al., 1996).

This myoelectric silence period often shows interesting alterations depending on the low back condition. The initiation and cessation of the EMG silence could be affected by the coordination of trunk and hip movement (Gupta, 2001), trunk velocity (Sarti et al., 2001), stretched passive tissues in low back (Solomonow et al., 2003a; Shin et al., 2009), low back muscle fatigue (Descarreaux et al., 2008) and gender (Solomonow et al., 2003a). The results suggest that the FRP may be a worthwhile topic for discovering the underlying control mechanism and dysfunction in the low back.

The association between FRP and low back condition has been reported and its significance and reliability for studying abnormal low back condition in both healthy population (i.e., transient low back dysfunctions) and chronic low back patients has been demonstrated (Neblett et al., 2003; Watson et al., 1997). A review study concentrating on clinical research of LBP patients suggested that the FRP is a valuable assessment tool to assist in the diagnosis and treatment of chronic low back patients (Colloca and Hinrichs, 2005). Also, in biomechanics studies of healthy population, Solomonow et al. (2003a) revealed that prolonged stooping modifies FRP in the low back, and Rogers and Granata (2006) showed that the modified FRP could be an indication of spinal instability. Similarly, it was reported that fatigued muscles in the low back create changes in the FRP and increase spinal instability (Descarreaux et al., 2008; Granata and Gottipati, 2008). In summary, the FRP could be employed to reveal the low back condition and understand the fundamental control mechanism of the low back system under normal or abnormal conditions.

1.4. Objective of this study

This study will investigate how the FRP and muscle activation patterns are modified under abnormal low back conditions (e.g., muscle fatigue, passive tissue elongation and combination) in trunk and lower extremity muscles. In addition, a system-level evaluation including both trunk and lower extremity tissues will help to reveal underlying physiology of the load-sharing mechanism between active and passive tissues. A literature review of previous studies on FRP, trunk motion system and functional anatomy will be provided, and a discussion about the strengths and limitations of previous studies will be presented. The experiment in current study will reveal a change in FRP and associated unusual patterns in the trunk system including muscles and passive tissues in upper body and lower extremity. Finally, this study will explain how the FRP changes in abnormal low back conditions, and hence contribute to the knowledge of LBP occurrence.

Chapter 2 – BACKGROUND

2.1. Functional anatomy of the trunk motion system

2.1.1. Connective tissues

Lumbar spine

Various connective tissues in lumbar spine, including bones, ligaments, fascia, cartilage etc., transmit forces and maintain the structural integrity in human trunk motion (Chaffin et al., 1999). The vertebra is an individual bone in the spine, and mainly provides skeletal support of the body. The vertebral column consists of a total of 33 vertebrae, including 7 cervical vertebrae (C1-C7), 12 thoracic vertebrae (T1-T12), 5 lumbar vertebrae (L1-L5), 5 fused sacral vertebrae (S1-S5) and 4 fused coccygeal vertebrae (tailbone) (Dorland, 2007). The lumbar spine will be mainly considered in this study which focuses on LBP. A functional spinal unit consists of two adjacent vertebrae, an intervertebral disc and ligaments, including the superspinal ligament, inter-spinal ligament, posterior longitudinal ligament and anterior longitudinal ligament (White and Panjabi, 1978). A lumbar vertebra consists of a vertebral body and a vertebral arch. The vertebral body is a large and flattened cylindrical-shaped bone which can bear large compression forces. The vertebral arch includes pedicles, laminae, facets and seven processes such as articular, transverse and spinous processes (Figure 2.1). The facet joint provides stability in flexion motion by guiding and limiting vertebrae motion and also bears up to 33% of compression load to the motion segment (Nachemson and Morris, 1964).

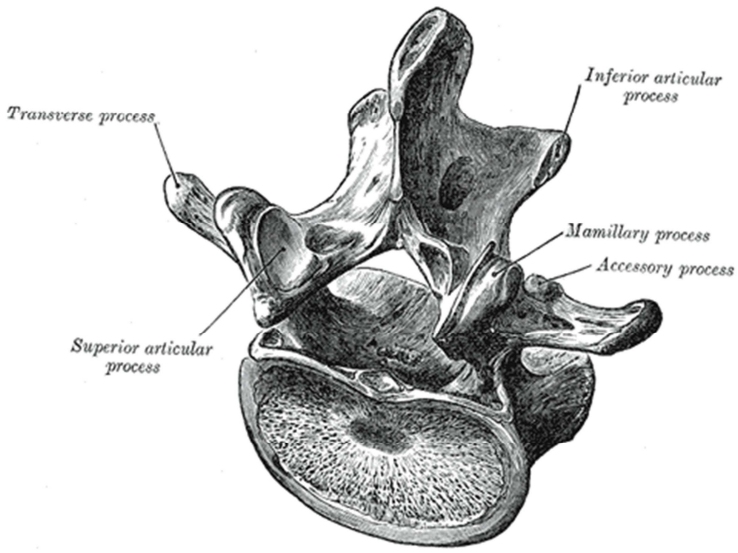

Figure 2.1 Lumbar vertebrae (from 1917 Gray's *Anatomy*)

Between two vertebral bodies, the intervertebral disc lies and this cartilaginous joint allows movement of the spine. Each disc consists of an outer annulus fibrosus which surrounds an inner nucleus pulposus. The nucleus, which acts as a shock absorber, occupies 30-50% of the total cross-sectional area of the disc and primarily consists of 70-90% water (Markolf and Morris, 1974). The annulus fibrosus is composed of concentric layers of bands with more collagen which provides flexibility and resistance to pressure (Figure 2.2). The mechanical tasks of the intervertebral disc can be summarized as follows: (1) firmly attach two adjacent vertebrae; (2) resist shear force when the spine rotates; (3) absorb and distribute compression force on vertebrae; (4) limit the movement of the vertebrae; and (5) space and position the vertebrae (Burkart and Beresford, 1979).

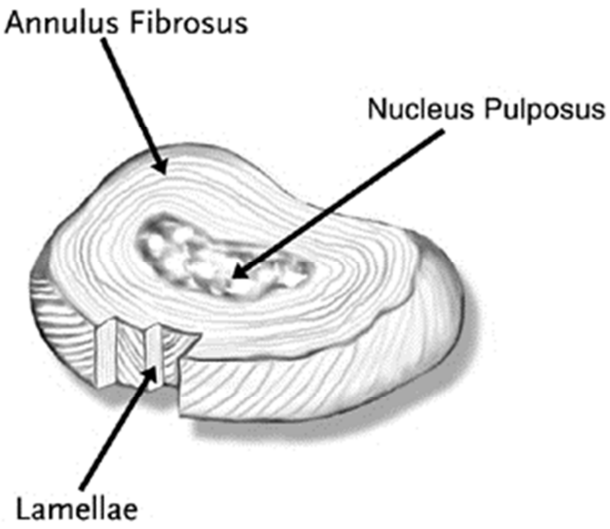

Figure 2.2 Intervertebral disc

Seven primary ligaments exist in lumbar spine (Figure 2.3). The anterior longitudinal ligament is located on the anterior side of the vertebral body, and the posterior longitudinal ligament is located on the posterior side of the vertebral body. Both of them are thin and broad ligaments attached to the anterior surface/posterior surface of the vertebral bodies and intervertebral discs (Maroon and Gianaris, 1990). In addition, they are extended from the surface of the vertebrae bodies to the sacrum (Neumann et al., 1992). The ligamentum flavum connect the laminae of adjacent vertebrae and extends from the second cervical vertebra (C2) to the first sacrum (S1). The capsular ligaments are oriented perpendicularly to the plane of the facet joint and bear both compressive and torsional load (Maroon and Gianaris, 1990). The supraspinous ligament connects the spinous processes from C7 to the sacrum and is thicker and broader in the lumbar region. The interspinal ligament connects adjacent spinous processes and meets the ligamenta flava on its anterior aspect and the supraspinous ligament on its posterior aspect. The intertransverse ligaments are located between the transverse processes. The role of

these spinal ligaments is to connect adjacent vertebrae and hold the lumbosacral spine together for permitting physiologic movement. Hence, the ligaments protect the spinal cord and passively provide spinal stability. Within the near-upright physiological range of trunk motion, the ligaments generate minor resistance to the spinal motion, but as they are passively stretched in trunk flexion postures they stiffened quickly and provide high forces near the full trunk flexion posture.

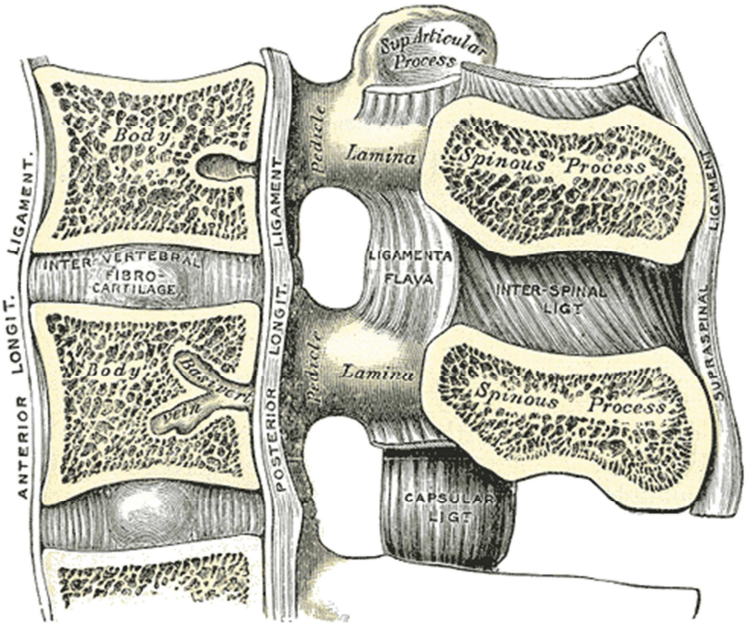

Figure 2.3 Spinal ligaments (from 1917 Gray's *Anatomy*)

The lumbodorsal fascia (or thoracolumbar fascia) is a deep investing membrane with three layers: anterior, middle and posterior. The quadratus lumborum muscle lies between the anterior and middle layer, and the erector spinae (or sacrospinalis) is surrounded between the middle and posterior layers (Figure 2.4). It extends from the iliac crest and sacrum to the thoracic cage, covering the paravertebral musculature (Figure 2.5). The functional role of the fascia in trunk flexion and extension includes the load transfer from the low back to the lower extremities

through the pelvis. Vleeming et al. (1995) showed that the thickest posterior layer of the fascia is stretched and stiffened by contraction of the gluteus maximus, erector spinae or latissimus dorsi. Also, the anterior and middle layers are tensed by the exertion of biceps femoris. The results and anatomical connections suggest that the lower extremity muscles (i.e., hip, pelvic and leg muscles) interact with trunk muscles via lumbodorsal fascia.

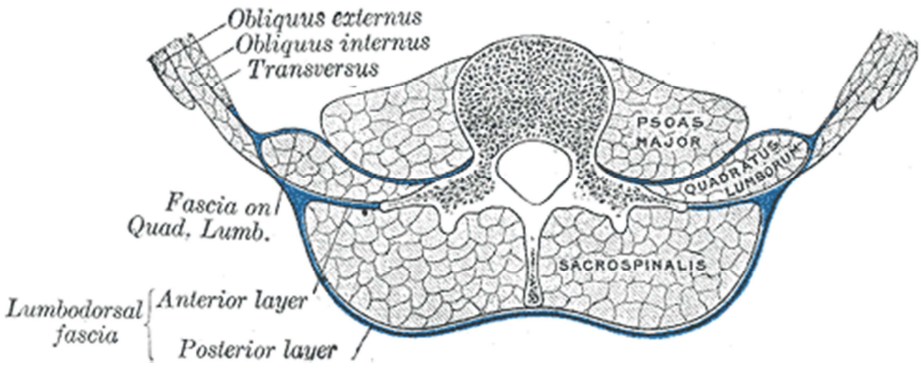

Figure 2.4 The lumbodorsal fascia - a transverse section (from 1917 Gray's *Anatomy*)

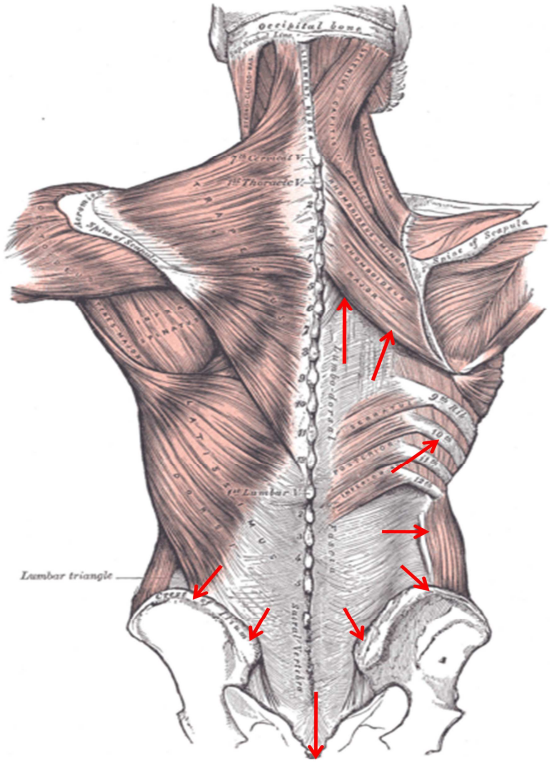

Figure 2.5 The lumbodorsal fascia - a posterior view (from 1917 Gray's *Anatomy*)

Pelvis

The pelvic girdle is a bony structure connecting the spinal column to the femurs. The lumbar spine is directly linked with five fused sacral vertebrae, and the head of each femur is inserted into the acetabulum, which is a concave surface of the two hip bones (Figure 2.6). The three structures, including right and left ilia and sacrum, are bonded together by the sacroiliac (SI) joint between the sacrum and the ilium of the pelvis on the posterior and the pubic symphysis between the two hip bones on the anterior: this structure is called the pelvis.

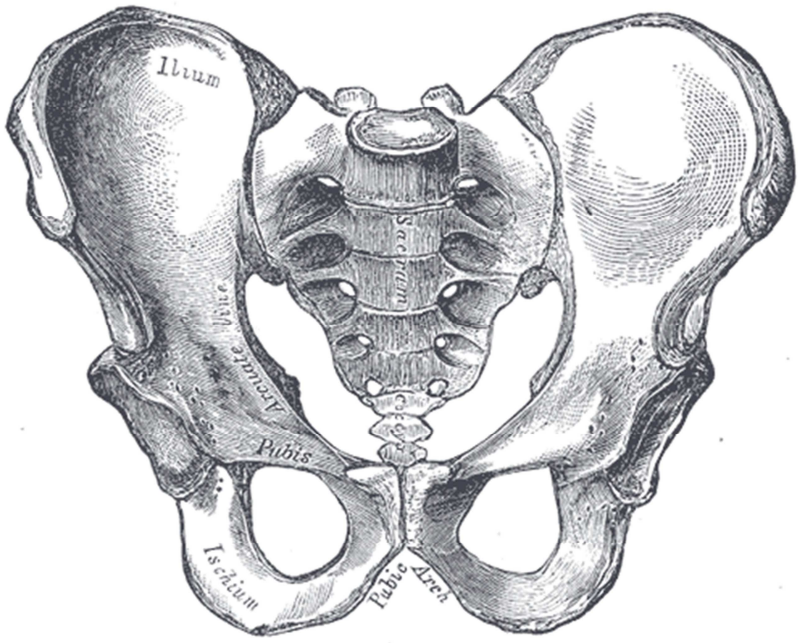

Figure 2.6 Male type pelvis – anterior view (from 1917 Gray's *Anatomy*)

The SI joint connects ilium with sacrum by five strong ligaments (Figure 2.7): (1) anterior SI ligament; (2) interosseous SI ligament; (3) posterior SI ligament; (4) sacrotuberous ligaments; and (5) sacrospinous ligament. The joint is not only very stable but also allows some mobility for walking and supporting the upper body (Pool-Goudzwaard et al., 1998; Schuenke et al., 2006) while absorbing shock like other joints. An additional role of this strong and rigid structure is to provide attachments for the various big muscles in the trunk and lower extremities and withstand the forces (Moore et al., 1992). Hence, the pelvis performs a vital function in the interactions between the upper body and lower body, and controls the length of active and passive tissues in low back throughout forward and backward rotation. For example, less hip flexion (i.e., pelvic rotation) during stooping will increase length of low back tissues (e.g., muscles and lumbodorsal fascia) at the same level of trunk flexion.

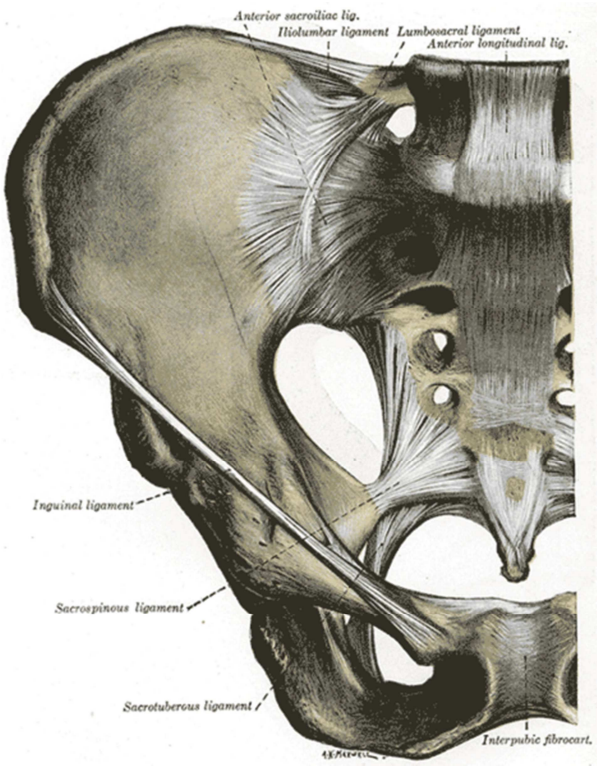

Figure 2.7 Ligaments between sacrum and ilium (from 1917 Gray's *Anatomy*)

2.1.2. Skeletal muscles

Lumbar musculature

The low back musculatures include a large number of muscle groups which control the flexion and extension motion of the torso and stabilize the multi-articular spinal column (Bogduk et al, 1992). The multifidus muscle in the low back arises from the mamillary processes of the lumbar vertebrae or the sacrum, and is inserted into the spinous process of each lumbar vertebra. The multifidus is the most medial muscle in the low back and is known to stabilize and rotate the spinal column. A recent study revealed the unique design of the multifidus muscle for lumbar spine stability with architectural analysis (Ward et al., 2009). This study demonstrated that the muscle has the largest physiological cross-sectional area (PCSA) in low back muscles (i.e., higher muscle force) and relatively short fibers (i.e., smaller shortening distance – not designed for excursion) (Figure 2.8). The unique design of this muscle indicates large force-producing capability over a narrow range of lengths. This study also revealed that the length range of sarcomeres in multifidi are positioned between the ascending portion and peak point of the length-tension curve, so the muscles can produce higher forces even when the low back is fully flexed. In summary, the multifidus muscle is a strong, dynamic stabilizer of the spinal column.

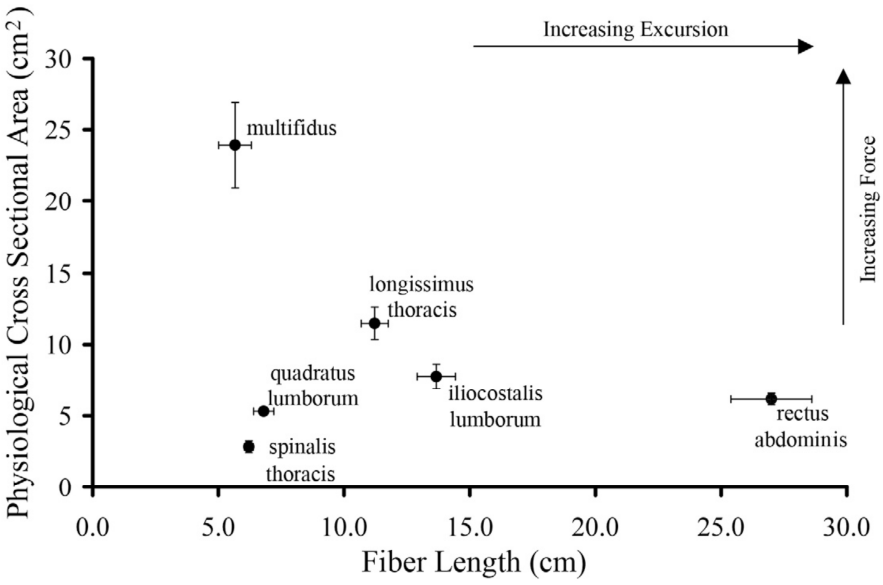

Figure 2.8 Relationship between PCSA and fiber length (adapted from Ward et al., 2009)

The lumbar erector spinae group, located lateral to the multifidus, consists of the spinalis in the medial column, the longissimus in the intermediate column and the iliocostalis in the lateral column. The spinalis arises from the spinous processes of the last two thoracic vertebrae (T11, T12) and the first two lumbar vertebrae (L1, L2), and is inserted into the spinous processes of the upper thoracic vertebrae. The longissimus lumborum is the largest and longest muscle of the erector spinae. The muscle originates from the ventral surface of the posterior superior iliac spine of the ilium and inserts into the transverse processes and the accessory processes of the lumbar vertebrae. In addition, the muscle is attached to the anterior layer of the lumbodorsal fascia (Figure 2.4). The iliocostalis lumborum originates from the ventral edge of the iliac crest and sacrum and inserts into the angles of lower six ribs for generating extensor moment (McGill, 2002).

The latissimus dorsi is the larger, flat, dorso-lateral muscle on the trunk. The muscle is largely attached to spinous processes from T6 to L2, the lateral raphe and the posterior layer of

lumbodorsal fascia and iliac crest. Even though this muscle has attachments on the lumbar vertebrae and pelvis, the main function of this muscle is shoulder adduction rather than generating trunk extensor moment (Macintosh and Bogduk, 1987).

The psoas major muscle is a long, deep muscle originating from the lateral surfaces of the T12 and the lumbar vertebrae and running downwards, laterally and forwards to reach the psoas tendon. The role of this muscle is known as a hip flexor and a spine stabilizer (Bogduk et al., 1992). The muscle generates minor flexion and extension moment in low back because of short moment arm, but it can generate significant compression and shear force on the lumbar spine.

Abdominal musculature

The abdominal muscles are known as the main trunk flexors in the control and movement of the trunk (Cholewicki et al., 1999; Hodges and Gandevia, 2000). In addition, the muscles are also considered trunk stabilizers by co-contracting with the low back muscles and increasing the biomechanical stability (Cholewicki et al., 1998; Gardner-Morse and Strokes, 1998; Granata and Marras, 2000) suggesting a significant role of the abdominal muscles in an underlying trunk control mechanism.

The rectus abdominis muscle consists of two parallel muscles running vertically on the anterior side of abdomen. The muscle is a major trunk flexor and partially produces intra-abdominal pressure by tensing the abdominal wall. Generally, the muscular activity of this muscle is dominated by the gravitational moment requirements. For example, contribution of this muscle in the standing posture is minor during trunk flexion, but during supine posture the muscle mainly generates flexion moment (sit-up motion).

There are three flat muscles in the lateral anterior abdomen, including external oblique, internal oblique and transverse abdominis. The external oblique is the largest and the most superficial muscle among three flat muscles. The muscle originates from the external surface of ribs 5 to 12 and inserts on the anterior iliac crest and abdominal aponeurosis. The actions of this muscle include flexion and rotation of trunk and compression of abdominal cavity raising intra-abdominal pressure.

The internal oblique lies deep to the external oblique and superficial to the transverse abdominis and generally runs perpendicular to line of action of the external oblique. The muscle arises from anterior two thirds of the iliac crest and lumbodorsal fascia of the low back and inserts on costal cartilages of ribs 8 to 12 and abdominal aponeurosis. Its functional roles include flexion and rotation of the trunk, supporting the abdominal wall and raising intra-abdominal pressure.

The transverse abdominis is the innermost of the flat abdominal muscles underlying the internal oblique. The muscle originates from the lumbodorsal fascia of the low back, anterior iliac crest and cartilages of ribs 6 to 12 and inserts on abdominal aponeurosis, xiphoid process and pubic symphysis. The major role of this muscle includes providing compression force on the abdomen, so its activity is most consistently related to changes in intra-abdominal pressure as compared to other abdominal muscles (Cresswell et al., 1992). In addition, Gracovetsky et al. (1977, 1981) proposed that the lateral pulling tension produced by the transverse abdominis and other abdominal muscles could generate an extension moment on the lumbar spine throughout the lumbodorsal fascia, and it was demonstrated by a cadaver study (Fairbank and O'Brien, 1980). However, Macintosh et al. (1987) showed that the effect is minor (less than 5 Nm).

Lower extremity musculature

The big muscles in the anterior and posterior thigh and gluteal region are used for standing, stooping, walking, running etc. In addition, the muscles have been regarded to have an important role in effective load transfer between lower extremity and trunk during lifting and trunk movements (Vleeming et al., 1989). Considering the focus of this study is FRP in the low back and the system level interactions that may influence the FRP, the lower extremity muscles which are suspected to have a direct or indirect influence the response of the tissues of the pelvis and low back will be discussed.

The gluteus maximus is a large, broad muscle in the buttocks. The big muscle is unique in humans in that the other primates have much flatter buttocks. The free trunk and upper extremity motions in bipedal stance could require the counter moment to keep the balance of the human body and develop the muscle. It rises from the posterior crest of the ilium, posterior sacrum, lumbodorsal fascia and sacrotuberous ligament and inserts on the gluteal line of the femur and iliotibial track (lateral thigh). The muscle extends from the femur (i.e., bring the bent thigh) or the trunk from stooping posture by pulling the pelvis backward. Also, it is recruited with trunk muscles in throwing, clubbing, trunk rotation, breaking the upper body motion and stabilizing the pelvis in static flexion of trunk (Marzke., 1988).

The posterior thigh muscles are called the hamstring including biceps femoris, semitendinosus and semimembranosus. The muscles traverse both the hip and the knee joint and are therefore recruited in hip extension and knee flexion. Also, those two-joint muscles are known to have influences on lumbar-pelvis interaction (i.e., lumbopelvic rhythm) and pelvis-femur interaction (i.e., pelvifemoral rhythm) because of the attachment in pelvis (Sihvonen, 1997). The biceps femoris (long head) arises from the posterior surface of ischial tuberosity and

the lower part of the sacrotuberous ligament and insert on the lateral collateral ligament and lateral tibial condyle. The muscle is most lateral one among hamstring muscles, and its function is mainly a hip extensor (i.e., pelvic rotator) and partially a knee flexor. The semitendinous is a medial muscle among the hamstring muscles. It originates from the medial impression on the tuberosity of the ischium and inserts on the upper medial surface of the tibia. The actions of this muscle are to extend the hip by rotating the pelvis and flex the knee. The semimembranosus is the most medial muscle among hamstring muscles. The muscle rises from the ischial tuberosity and inserts on the posterior part of the medial condyle of the tibia. Similar to other hamstring muscles, this muscle is a hip extensor (i.e., pelvic rotator) and a knee flexor. It also helps medial rotation of the knee.

In the anterior compartment of the thigh, the rectus femoris is the only muscle involved in hip flexion and hence is a direct antagonist to the hamstrings. This muscle originates in the anterior inferior iliac spine (AIIS) and ilium above the acetabulum and inserts on the quadriceps tendon to the base of the patella and onto the tibial tuberosity via the patella ligament. The functions of this muscle involve hip flexion and knee extension.

2.2. Trunk flexion and extension

2.2.1. Trunk muscle contractions

Concentric and eccentric exertions

The voluntary muscular contraction can be classified into the concentric contraction and the eccentric contraction according to the changes in muscle length. In terms of the low back musculatures, the flexion motion is the eccentric exertion, and the extension motion is the concentric exertion. In concentric contraction motion, the muscle shortens as it contracts, so

the degree of overlap between myosin and actin increases with the body movement. In eccentric contraction motion, the muscle lengthens as it contracts, so the degree of overlap between myosin and actin decreases with body movement. The eccentric contraction motion is more likely to damage the tissues in that the bonds between myosin and actin are disrupted by mechanical stresses during this lengthening motion (Proske and Morgan, 2001). There are other differences between the two contraction types such as muscular activation level (i.e., electromyographic response) and force generation capacity.

Previous studies revealed that the muscular activation level in eccentric contraction motion is smaller than the concentric contraction motion under the same level of force generation (Tesch et al., 1990, Huang and Thorstensson, 2000). Tesch et al. (1990) also showed that the ratio of EMG/moment is significantly higher in the concentric contraction motion. The result suggests that the eccentric contraction motion necessarily requires an additional source of force generation to provide a similar level of force with the concentric contraction. They hypothesized that the passive viscoelastic tissues can produce additional force to meet the same level of net force with the concentric contraction. Regarding the force generation capacity, the eccentric contraction motion showed greater force generation capacity compared to the concentric contraction in maximum voluntary contraction (Doss and Karpovich, 1965). The greater force generation could be attributable to the elastic force generated by stretched tissues (McCully and Faulkner, 1985).

Coactivation in trunk muscles

A classical definition of coactivation (or co-contraction) is the muscular activity in the antagonist muscles which opposes the intended movement (Williams and Warwick, 1980). An

alternative for co-contraction is the excessive antagonistic activation over the required baseline which is required to generate desired movement (Hughes et al., 2001). Considering the fact that the antagonistic exertion does not contribute to generating any driving force in the movement the coactivation may have a unique role in the system. Prior research studies of spinal stability hypothesized that the additional excessive activation in the antagonist muscle stiffens the spinal column and enhances spinal stability to prevent buckling of the spine under abnormal low back conditions (Bergmark, 1989; Cholewicki and McGill, 1996; Crisco and Panjabi, 1990).

There is clear evidence that the co-contraction contributes to increased biomechanical spinal stability (Cholewicki et al., 1998; Gardner-Morse and Stokes, 1998; Granata and Marras, 2000). The biomechanical models of the spine suggested an increase of trunk stiffness by the recruitment of antagonistic coactivation (Gardner-Morse and Strokes, 1998; Granata and Orishimo, 2001). Recently, Lee et al. (2006) conducted an empirical study to evaluate the influence of coactivation on trunk stiffness with the analysis of impulse response functions (IRFs) of trunk dynamics and demonstrated an increase of trunk stiffness by 37.8% from minimal to maximal coactivation. Based on the studies, the excessive exertion in the antagonist muscle could be attributed to a response of the system to stabilize the movement.

On the other hand, the enhanced activation in antagonist muscles (i.e., abdominal muscles) for stabilizing the spinal column also increases the spinal compression load (Hughes et al., 1995; Marras and Granata, 1996). There may be an increased risk of spinal failure caused by enhanced co-activation in antagonistic muscles at the benefit of the spinal stability. Spinal instability without antagonistic coactivation may also bring about the risk of the spinal column buckling. So, there is a trade-off between coactivation (i.e., increased spinal load) and spinal stability.

Granata and Marras (2000) demonstrated the cost-benefit of increasing spinal stability with co-contraction. They developed a biomechanical model to compute spinal load and stability, and showed a 12% to 18% increase in spinal compression load and a 34% to 64% increase in spinal stability with enhanced antagonistic co-contraction. It can be hypothesized that the antagonistic co-contraction of trunk muscles can possibly be balanced by the optimal control mechanism of the motor control system manipulated by central (cortex) commands and spinal reflexes (Brooks, 1986).

2.2.2. Lumbopelvic rhythm

The trunk flexion and extension motion is a combination of lumbar spinal flexion and hip flexion. This spine-hip interaction is called lumbopelvic rhythm (Cailliet, 1981). This rhythm is accomplished by the predominant lumbar flexion before the initiation of flexion relaxation (FR) in low back muscles and pelvic rotation after the initiation of FR in low back muscles (Paquet et al., 1994; Sihvonen, 1997; Sarti et al., 2001). The opposite occurs during extension motion.

Sihovonen (1997) investigated the muscular activity in the low back and lower extremity and the lumbar angle using two motion sensors placed on the sacrum and the thoraco-lumbar area during trunk flexion and extension. The results showed that the activity of the hamstring muscles lasted longer than that of the low back muscles and ceased its activity at nearly full lumbar flexion (97%). The author proposed that the lumbopelvic rhythm is controlled by the different activation timing of the low back muscles and the hamstring muscles. However, this study did not include the gluteal muscles which are known to directly influence the lumbopelvic rhythm. In the system level perspective of the trunk motion system, the abdominal muscles may also interact with other muscles to accomplish trunk flexion and extension motion. For better

understanding of the lumbopelvic rhythm and the interaction within the trunk motion system, the gluteal and abdominal muscles should be investigated.

Leinonen et al. (2000) investigated the function of the back and hip extensor, including the lumbar paraspinalis, gluteus maximus and biceps femoris, during trunk flexion and extension. The lumbar paraspinalis and biceps femoris simultaneously initiated the flexion motion and were followed by the gluteus maximus activation in both healthy subjects and chronic LBP patients. However, the activation period of the gluteus maximus during flexion and extension was shorter in the low back patient group than the control group. The authors suggested that the back pain patients may have atrophy to recruit hip extensors (i.e., gluteals) and decondition the muscles.

From a biomechanical point of view, the spine-pelvis-leg system is completely integrated to provide a source of driving force of flexion and extension and stability of the spinal column. There are some anatomical evidences for these system-like interactions. First, the trunk and hip extensor muscles are largely attached to the pelvis. So, the relative movement of the pelvis should influence trunk flexion and extension, and hence the hip extensor is attached to the pelvis and femur and controls the pelvic motion also indirectly affecting trunk flexion and extension. Second, the lumbodorsal fascia can be employed for load transfer between low back and lower extremity (Snijders et al., 1993b). The gluteus maximus is coupled with trunk muscles via the lumbodorsal fascia extending from the iliac crest and sacrum to the thoracic cage, and the lamina is tensed by contraction of the trunk and hip extensors (Vleeming et al., 1995). In summary, the anatomically integrated trunk motion system effectively transfers load and the muscles interact with each other.

2.3. Neuromuscular responses of the lumbar spine

The neuromuscular control system receives and integrates the input signals initiated from the various afferent nerves, and recruits the motor unit to control the movement. The sensory receptors in the human body can be distinguished on the basis of the following criteria: (1) location (exteroceptors, proprioceptors and interoceptors); (2) function (mechanoreceptors, thermoreceptors, photoreceptors, chemoreceptors and nociceptors); and (3) morphology (free nerve endings and encapsulated endings). In this chapter, the receptors linked with neuromuscular responses in passive and active tissues will be discussed.

2.3.1. Neural components in passive tissues

The joint receptors function as mechanoreceptors and appear in various locations such as joint capsules, ligaments and loose connective tissues (Enoka, 1994). They are passively excited by mechanical disturbances such as pressure or tension, and activate or inhibit motor neuron known to directly or indirectly control muscle activity. For example, the mechanical energy caused by deformation stimulates the mechanoreceptors in the passive tissues (depolarized to threshold), and, then, the nerve impulses (action potentials) arise and propagate into the spinal cord along a sensory neuron. Within the spinal cord, the sensory neuron activates stimulation (or inhibitory) interneuron making a synapse with a motor neuron. Finally, the stimulation (or inhibitory) neurotransmitter activates (or inhibits) the motor neuron generating more (or fewer) nerve impulse to contract (or relax) the muscle.

The ligaments in the extremity joints and the spine are innervated with four types of mechanoreceptors including Golgi endings, Ruffini endings, free nerve endings and Pacinian corpuscles (Burgess and Clark, 1969; Rothwell, 1987). The Golgi and Ruffini endings are

responsive only to the extreme deformation of the joint capsule or ligaments as a protective mechanism and are known as slowly adapting endings (Clark and Burgess, 1975; Edin, 1990). In addition, the Ruffini endings signal joint position and displacement, angular velocity and intra-articular pressure (Johansson et al., 1991). The Pacinian corpuscles are rapidly adapting afferent nerve endings (Edin, 1990). They have low thresholds to mechanical stress and detect acceleration of the joint (Bell et al., 1994). The characteristics of free nerve endings have not been investigated in detail, but Leunig et al. (2000) suggested a role of a nociceptor (i.e., pain receptor) and proprioceptor (sensing of the relative position).

Those afferent nerve endings were also found in the annulus fibrosus and longitudinal ligaments of the human spine (Roberts et al., 2000). Also, prior studies showed various mechanoreceptors in the human intervertebral discs (Malinsky, 1959; Yoshizawa et al., 1980), the facet joint capsule (Jackson et al., 1966), and the various anterior and posterior ligaments (Rhalmi et al., 1993; Yahia et al., 1988). They may provide proprioception to sense the posture and movement of the trunk and have a distinct role in reflex activation or inhibition as a mechanoreceptor to control the low back muscles.

2.3.2. Neural components in active tissues

There are two types of muscle receptors: muscle spindles and Golgi tendon organs. Muscle spindles are sensory receptors lying in parallel to muscle fibers within the muscle and are able to detect the changes in the length of the muscle. The responses of muscle spindles will help to determine the position and orientation of the joint system and regulate the contraction of the muscle (Enoka, 1994). The Golgi tendon organ is a proprioceptive sensory receptor which is located in the tendon at the end of a muscle. The receptor senses muscle contraction

and prevents overexertion by inhibiting activity of the α-motor neurons (Brooks, 1986). Therefore, it is a sensor controlling muscle tension.

During muscle contractions, two main types of motor neurons in the spinal cord take part in motor control activities, and translate "brain language" into "muscle language". The α-motor neurons are the largest ones, sending out signals to muscle fibers and generating active muscle contractions. The γ-motor neurons are the smallest, regulating the gain of the stretch reflex, providing the baseline level of activation in the α-motor neurons and helping to control muscle length and tone (Brooks, 1986; Enoka, 1994).

2.3.3. Reflex pathways in lumbar spine

The neural components outlined and described in Chapters 2.3.1 and 2.3.2 indicate that the components are present for a ligamentous and muscular reflex response in the lumbar region. A reflex pathway from ligaments to muscles was first observed in the anterior cruciate ligament in 1987 (Solomonow et al., 1987). Subsequently a ligamento-muscular reflex was also found in the lumbar spine (of animal model) (Indahl et al., 1995, 1997; Solomonow et al., 1998; Stubbs et al., 1998), the elbow (Phillips et al., 1997), the shoulder (Guanche et al., 1995; Solomonow et al., 1996) and the ankle joint (Solomonow and Lewis, 2002) by electrical stimulation on the nerve emerging from the ligaments or direct tension on the ligaments using a hook. With specific reference to the lumbar spine there is sufficient literature reporting the reflex pathways between passive tissues and muscles in lumbar spine using the porcine or feline model. Those studies showed activation of the paraspinal muscles (usually multifidus) using the EMG technique where they excited the ligament, the intervertebral disc and the facet joint with electrical, mechanical and chemical stimulation (Indahl et al., 1995, 1997; Kang et al., 2002; Solomonow et al., 1998;

Stubbs et al., 1998). By combining the results of animal model studies with the fact that the mechanoreceptors also exist in the human lumbar spine and are innervated by articular nerves, it would be reasonable to believe that the reflex exists in the human lumbar spine as well.

In addition to this, the muscle spindles detect increases in muscle length. The patellar reflex is a good example of this stretch reflex. When one strikes the patellar tendon, the quadriceps muscles are passively stretched and the muscles spindle receptors trigger an afferent impulse leading to the spinal cord. Then, an efferent impulse conducted from a motor neuron contracts the quadriceps muscles resulting in 'knee-jerk.' So, basically, stretching of the muscle increases spindle output and muscular activity, and compressing the muscle decreases spindle output and muscular activity. Similar kinds of reflex responses may be present in the lumbar region (Kang et al., 2002).

2.4. Spine stability

2.4.1. Concepts of spinal stability

White and Panjabi (1978) proposed the definition of the clinical spinal stability as follows: "The ability of the spine under physiologic loads to limit patterns of displacement so as not to damage or irritate the spinal cord or nerve roots and no development of deformity or pain due to structural changes." The stability concept suggested would be viewed in light of the individual patients' physical capacity or characteristics. In other words, everyone may have different level of tolerance at the same level of perturbation in the spine to maintain spinal stability. The idea can be explained with a simple ball example (Figure 2.9) representing stability of the system. In Figure 2.9 (a), the ball is in equilibrium but is unstable and will easily roll away given a perturbation. This figure can be described as having low level of tolerance to maintain

the stability. On the other hand, the ball in Figure 2.9 (b) is in equilibrium and is stable and will not easily roll away even with a significant perturbation. The system will have high level of tolerance to maintain stability under the same level of perturbation with Figure 2.9 (a). Also, the ball will eventually return to the initial, stable position after perturbation.

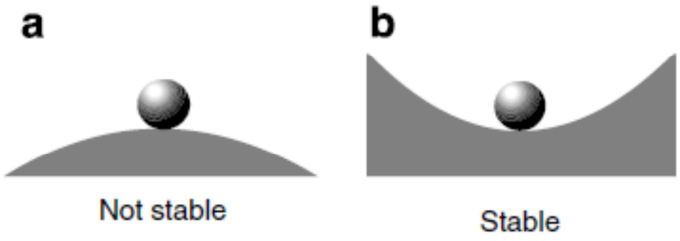

Figure 2.9 Stability of a ball (adapted from Reeves et al., 2007)

Reeves et al. (2007) expanded this analogy and suggested system characteristics in spinal stability including robustness and performance. The robustness of the spinal stability system indicates the ability to keep the spinal column stable under both large and small perturbations by adjusting spinal stiffness. For example, the ball in Figure 2.10 (a) can keep the stable condition under both large and small perturbation without failure of the system (i.e., robust), but the ball in (b) could easily fail with perturbation (i.e., not robust) even though the ball is stable in the static condition. With respect to the spinal column, the steepness of the wall in this example would be linked with the "stiffness of the spine" governed by surrounding musculatures in low back (Figure 2.10 (c) and (d)) (Bergmark, 1989). An increase in spinal stiffness (i.e., a larger "margin of safety") may result in a more stable spinal system like the example in Figure 2.10 (c) (Cholewicki and McGill, 1996). The performance of the spinal stability system includes speed and accuracy in returning to the original undisturbed position. For example, even though both

systems (c) and (d) in Figure 2.10 are successful in maintaining the system stability, system (d) could require a longer time to return the original undisturbed position (i.e., late response). In the spine system, the steeper system (c) represents a faster and more accurate reaction of the spine column, and hence provides a more stable spinal system.

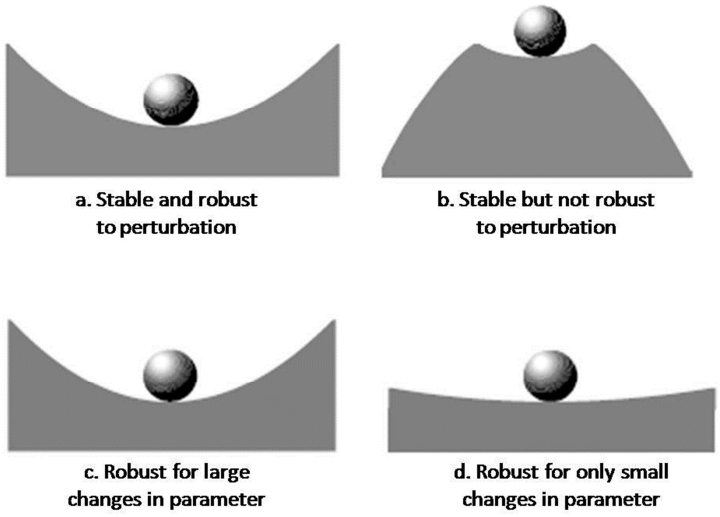

Figure 2.10 Concepts of robustness – The ball example (adapted from Reeves et al., 2007)

2.4.2. Factors influencing spinal stability

The "stiffness of the spine" (i.e., the steepness of the wall in the example) can be influenced by the following three subsystems proposed by Panjabi (1992): (1) the passive musculoskeletal subsystem including facet joints, intervertebral discs, spinal ligaments, joint capsules and passive mechanical tension generated by muscles; (2) the active musculoskeletal subsystem including the muscles stabilizing the spinal column; and (3) the neural and feedback subsystem including the transducers such as mechanoreceptors, the nociceptors, the

proprioceptors, etc., in spinal tissues and the neural control centres. All three subsystems can be faced with abnormal conditions that can cause some trouble in keeping the spinal system stable.

First, previous literature revealed that prolonged or cyclic stooping brings about laxity in passive tissues of the lumbar spine and contributes to changes in sensitivity of the receptors in the passive tissues (i.e., changes in spinal reflex) (Granata et al., 2005; Rogers and Granata, 2006; Solomonow et al., 2003a, 2003c). For example, Rogers and Granata (2006) investigated paraspinal muscle reflexes, known to be controlled by the receptors in spinal ligaments, after performing a total of 16 minutes prolonged stooping, and showed an increase in lumbar flexion angle and a decrease in spinal reflex. Decreased spinal reflex could be attributable to the laxity in the low back passive tissues (i.e., increased lumbar flexion angle) reducing the sensitivity of the mechanoreceptors and causing neuromuscular reflex errors. In addition, decreased tension in the viscoelastic tissues may reduce mechanical contribution in stability than the normal condition. Those changes will result in spinal instability and elevate the risk of spinal buckling.

Second, the literature is replete with evidence suggesting that muscle fatigue increases variability of muscle force and antagonist coactivation in trunk muscles (Granata et al., 2004; O'Brien and Potvin, 1997; Potvin and O'Brien, 1998). For example, Granata et al. (2004) developed a biomechanical model of spinal stability and investigated the effect of a lifting-induced muscle fatigue, generated by the repeated lifting of a 12.7 kg load from the floor to an upright posture at a rate of 60 lifts/min for minimum of 2 min. The results showed a decrease in spinal stability and a significant increase in antagonist coactivation. Mirka and Marras (1993) developed a stochastic model of trunk muscle coactivation and showed the importance of the biomechanical variability as a low back risk factor. The higher variability in muscle force may have higher potential for stability system failure in that a single big disturbance results in the

system failure (i.e., big disturbance in the ball system). In addition, muscle fatigue is known to impair spine proprioception (Taimela et al., 1999) and the ability to regulate the force (Parnianpour et al., 1988; Sparto et al., 1997). Parnianpour et al. (1988) investigated the effect of muscle fatigue on the maximum torque generation, the range of motion (ROM), the maximum and average velocity during isoinertial movement in the three axes of rotation including sagittal, coronal and transverse plane. During the experiment, the subjects were asked to perform trunk movement, from upright to full flexion and back, as quickly and as accurately as possible while exerting maximum force on the dynamometer. The results revealed significant decreases in the maximum torque, the range of motion (ROM) and the maximum and average velocity in sagittal plane. In addition, the ROM and the maximum and average velocity in coronal and transverse plane were significantly increased under the muscle fatigue condition. The authors suggested significantly less motor control ability under the condition of the low back muscle fatigue. The disturbed neural system causing reflex errors and reduced force generating capacity of muscles could also result in spinal instability and, in turn, elevate the risk of spinal buckling.

2.4.3. Role of the local and global systems in spinal stability

Bergmark (1989) classified the active components (e.g., muscles) of the trunk into a local and a global system for mechanical modeling of the spinal stability system. The local system includes all muscles having their origin and insertion at the vertebrae such as the multifidus and medial erector spinae muscles. Recognizing the functional anatomy of the multifidus and erector spinae discussed in Chapter 2.1.2., the role of the local system is to keep the lumbar lordosis in upright standing and control its curvature during spinal flexion. In addition to this, the local muscle system will mechanically stabilize the lumbar spine by stiffening the spinal

column. The local muscles may also contribute to generating extensor moment in some degree (around 20%) (Bogduk et al., 1992).

The conceptual definition of the global system proposed by Bergmark (1989) is "active components transferring the load between thoracic cage and the pelvis," such as lateral erector spinae, lateral quadratus lumborum, internal and external oblique, transverse abdominis, rectus abdominis, and intra-abdominal pressure. In line with this idea, the main role of the global system is to counteract the external moment generated by trunk weight and hand-held load. The extension force generated by the global system will then be transferred to the spinal column which should be stiffened enough by the local muscle system for dynamic motions.

There is enough evidence that the global system also directly or indirectly contributes to the stability of the trunk system. The intra-abdominal pressure (IAP) has been hypothesized to increase spinal stability (Cresswell et al., 1994; Marras and Mirka, 1996; McGill and Norman, 1987). By developing a physical model, Cholewicki et al. (1999) showed that the IAP produced by coactivation of the abdominal muscles increases the spinal stability. Recently, Hodges et al. (2005) performed an *in-vivo* experiment and provided clear evidence that lumbar spine stiffness (i.e., spinal stability) is concurrently increased with the IAP elevation.

As already discussed in Chapter 2.2.1, there is clear evidence that the agonist-antagonist co-contraction also significantly contributes to increased biomechanical spinal stability (Cholewicki et al., 1998; Gardner-Morse and Stokes, 1998; Granata and Marras, 2000). Based on this, an increase in abdominal muscle activity will result in enhanced IAP and co-contraction and stabilize the spinal column.

2.4.4. Trunk system level stability

Prior research on low back stability have focused on the tissues of the torso and have not considered the potential influence of the structures of the lower extremities; the pelvis being regarded as a rigid, stable body in most previous models (Bergmark, 1989; Cholewicki and McGill, 1996; Cholewicki et al., 1998; Granata and Orishimo, 2001; Granata and Rogers 2007; Rogers and Granata, 2006). For example, in the model developed by the Granata group, the effects of lumbopelvic rhythm were excluded by restricting the pelvis with belts and assumed that the deformation within the pelvis is small. Using the model, they investigated the effects of muscle fatigue and passive tissue elongation in low back on spinal stability (or spinal reflex). Clearly, the models have provided a good theoretical and empirical basis to understand spinal stability, but the models are limited for several reasons described in the next several paragraphs. An investigation of system level (lower extremity included) trunk stability, including all relevant tissues having biomechanical effects on trunk movement and stability, is required to further understand the spinal stabilization system.

Biomechanical linkage – lumbodorsal fascia

As previously mentioned, the lumbodorsal fascia covers the paravertebral musculature and are linked with the gluteus maximus and biceps femoris (caudally) and the transverse and internal oblique (laterally) (see Figures 2.4 and 2.5). Vleeming et al. (1995) showed the functional role of the lumbodorsal fascia in load transfer between spine, pelvis, and lower extremity by dissection in ten embalmed human cadavers and traction to various muscles such as gluteus maximus, external oblique, latissimus dorsi and biceps femoris. The authors proposed that the lumbodorsal fascia plays an important role in stabilization of the trunk motion system including

spine and sacroiliac (SI) joints and trunk rotation. Figure 2.11 represents the superficial layer of the lumbodorsal fascia and its attachment to various muscles and explains its functional roles. The double, red arrow lines (thicker arrow) in the figure show that contraction of the gluteus maximus generates tension in the contralateral side layer via the lumbodorsal fascia which contributes to trunk rotation and stability. In addition to this, the deep layer of the lumbodorsal fascia is also connected with the transverse abdominis and internal oblique and functions similarly (Bogduk and Macintosh, 1984; Bogduk and Twomey, 1987; Vleeming et al., 1995). In summary, a strong fascia could tighten the unity between the trunk (i.e., spinal column) and lower extremity (i.e., pelvis) by bracing the lumbar spine and SI joints, and enhance the trunk-system level stability achieved by both pelvic stabilization and spinal stabilization. The fascia also can transmit the force from the lower extremity to the trunk with this connection (Pool-Goudzwaard et al., 1998).

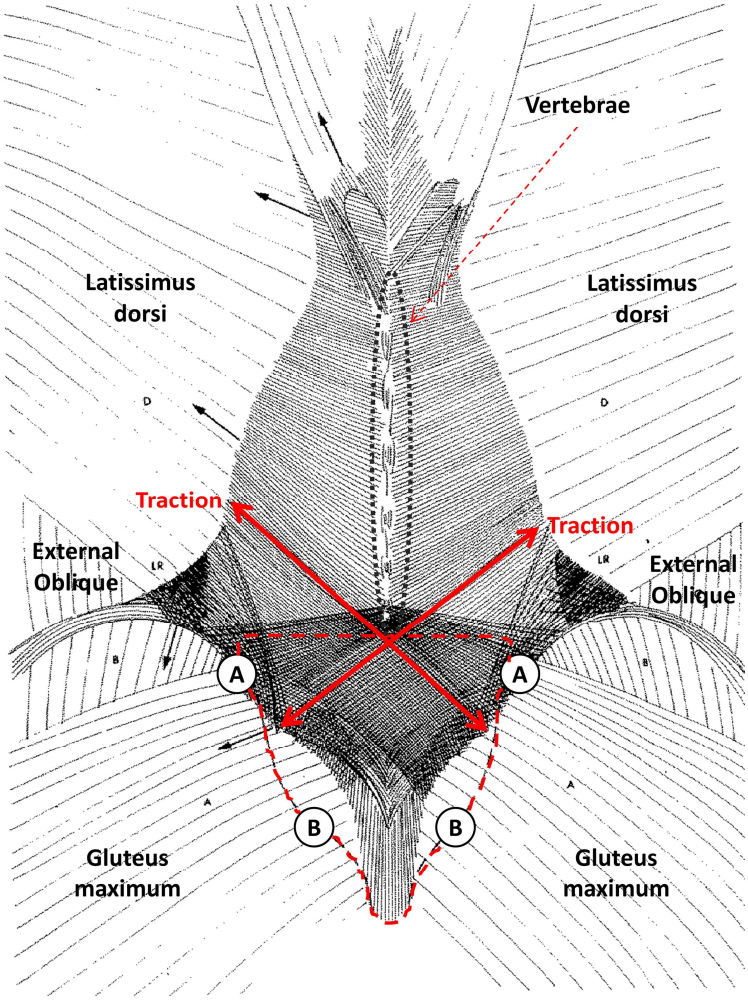

Figure 2.11 The superficial layer of the lumbodorsal fascia (adapted from Vleeming et al., 1995))

Note: Red, thicker dotted line (sacrum); A (posterior superior iliac spine); B (sacral crest).

The ball example presented by Reeves et al. (2007) can be expanded and used to explain this concept (see Figure 2.12). In Figure 2.12, the ball could be considered as spinal column, and the bowl the ball is resting on might be regarded as the pelvis. The red ropes holding the bowl represent pelvic stabilizers such as strong ligaments. The bowl was flat in the previous example in Figure 2.10 only accounting for the low back tissues in spinal stability. This new example takes into account the stability potentially provided by the lower extremities, especially pelvic stabilization for stable foundation and SI joint stability. In this example, the enhanced unity by

the lumbodorsal fascia is relevant to the increased friction between the ball and the bowl and reduces the time required for returning to the stable position. This hypothesis can be explored by observing the activation levels in the lower extremity muscles under the unstable low back conditions.

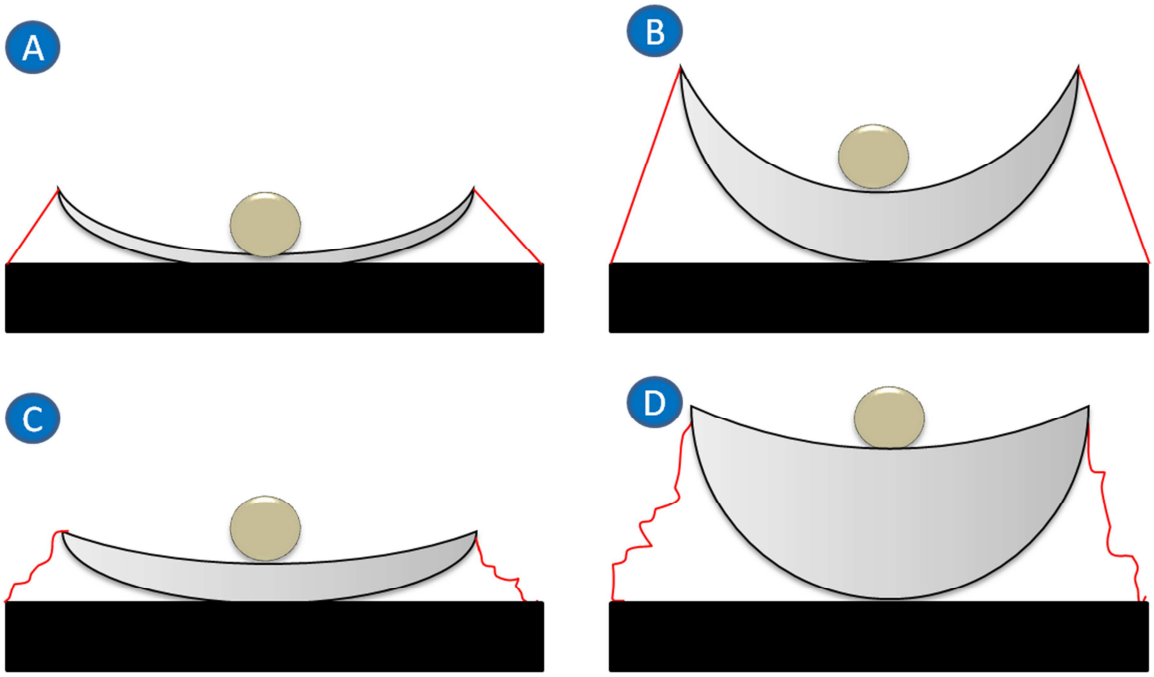

Figure 2.12 Stability of a ball over stable or unstable bowl

Pelvic stabilization

The pelvic-femoral rhythm is a unique feature of the human body, because the bipedal stance requires the counter moment to keep the balance by using buttock muscles (e.g., the gluteus maximus). During trunk flexion and extension, the lower extremity muscles control the pelvic rotation and stabilize the pelvis, which provides a stable foundation for the movements of the spinal column. It is clear that the foundation should be stabilized enough in advance of the

spinal stabilization; otherwise the spinal stability could not be successfully achieved. For an example, the ball upon the unstable bowl (see Figure 2.12) cannot reach the stable position unless the bowl is stabilized. In addition, the unsteady bowl could aggravate the disturbance of the ball.

Similarly, the pelvis should be locked and kept stable during the trunk motion. The pelvic girdle will provide a firm foundation, and stable movement will be achieved via the active tissues around the pelvis, including those in the trunk, hip and thigh (i.e., muscles), along with the passive tissues connecting the two hip bones and the fascia. The main source of instability in the pelvis arises from the SI joint. It is the only flat joint in the human body transferring large amounts of force between the trunk and the lower extremity. It is known that the wedge-shaped flat joint is naturally favourable to compression force and vulnerable to shear force (Snijders et al., 1993a). Snijders et al. (1993a) proposed two types of self-locking or self-bracing mechanism including form and force closure. The form closure can be accomplished by the unique shape and close fit of the two joints such as the wedged character. The force closure is achieved through the compression forces on the SI joint generated by ligaments, muscles and fascia, to avoid shear. The perfect form closure will not allow any mobility, so the optimal combination of form and force closure is required.

Prior studies revealed that the sacrotuberous ligaments can stabilize the SI joint via the activation of the biceps femoris and gluteus maximus muscles and nutation of the sacrum (i.e., forward rotation of sacrum relative to two hip bones) (see Figure 2.13) (Vleeming et al, 1989a and 1989b; Wingerden et al., 1993). In contrast, the sacroiliac ligaments can stabilize the SI joint during counter-nutation of the sacrum and activation of the erector spinae muscles, and the tension of the ligament decreased during activation of the gluteus maximus and traction of

lumbodorsal fascia (see Figure 2.13) (Vleeming et al., 1996). The results suggest that the creeping deformation of sacrotuberous ligaments (i.e., sacroiliac joint instability) can be compensated by the biceps femoris and gluteus maximus muscles and the sacroiliac ligament. Also, the activation of the abdominal muscles and latissimus dorsi muscles could contribute to the traction of the lumbodorsal ligament, and hence increase the sacroiliac joint stability. Recently, Wingerden et al. (2004) measured the SI joint stiffness during relaxed postures and voluntary contractions and verified the idea that the SI joint stability increases with even slight activation of the erector spinae, the gluteus maximus and the biceps femoris muscles.

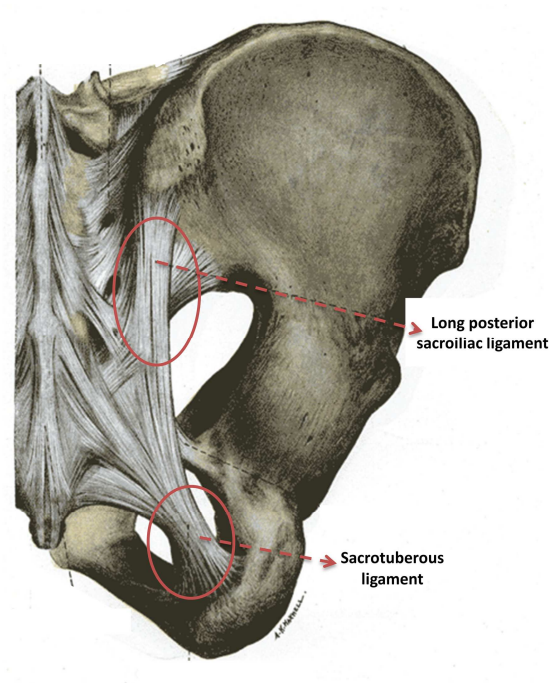

Figure 2.13 Sacroiliac joint and attached ligaments – posterior view (from 1917 Gray's *Anatomy*)

The ball example in Figure 2.12 can explain the role of passive and active mechanisms in pelvic stabilization. The steepness of bottom of the bowls in the figure could represent contribution of the active tissues such as the erector spinae, the gluteus maximus and the biceps femoris muscles on pelvic stabilization. Deconditioned muscles may not successfully stabilize

the pelvis, and hence the condition can be characterized as a steeper bottom of the bowls in Figure 2.12 (B) and (D). Kankaanpää et al. (1998) revealed that the gluteus maximus of low back patients fatigue faster than the normal participants, even when there is no difference in the fatigue of the erector spine muscles. In addition, Leinonen et al. (2000) investigated the function of the back and hip extensor, including the lumbar paraspinalis, gluteus maximus and biceps femoris, during trunk flexion and extension. The lumbar paraspinalis and biceps femoris simultaneously initiated the flexion motion and were followed by the gluteus maximus activation in both healthy subjects and chronic LBP patients. However, the activation period of the gluteus maximus during flexion and extension was shorter in the low back patient group than the control group. The authors point out that the avoidance of the use of this muscle may weaken the hip extensor muscles. Briefly, the deconditioned muscles can increase the pelvic instability and is represented by a steeper bottom of the bowl.

The ropes holding the bowl in Figure 2.12 could be regarded as the passive tissues (e.g., strong ligaments) which provide SI stability. The slack ropes of C and D represent inability of the ligaments (e.g., sacrotuberous ligaments and sacroiliac ligaments) holding the SI joint. The creeping deformation in sacrotuberous ligaments (slack ropes) after prolonged stooping caused by the severe sacral nutation could be a good example of the slack ropes. The loose ropes in C and D will increase the time required for returning to the stable position of the bowl. On the other hand, the tense ropes in A and B will reduce the required time to stabilize the bowl denoting enhanced pelvic stability.

2.5. Flexion-relaxation phenomenon

Fick (1911) first proposed the myoelectric deactivation of erector spinae in full lumbar flexion posture. The hypothesis has since been confirmed by many researchers, and the phenomenon of flexion-relaxation has been tested under various conditions to reveal its underlying mechanisms and significance. The myoelectric deactivation period caused by significantly less muscle activity usually shows interesting alterations depending on the low back condition (e.g., transient muscles fatigue, chronic LBP patients, transient passive tissue stretching) or the task characteristics (e.g., hand-held load, trunk movement speed, flexion-extension posture, body orientation to the gravity vector,). In this chapter, the nature of FR (flexion-relaxation) will be discussed.

2.5.1. Mechanism for muscular deactivation in full flexion: How does it happen?

FRP has been explained as a synergistic load-sharing mechanism between active tissues (i.e., muscles) and passive viscoelastic tissues (e.g., ligaments, tendons, intervertebral discs, etc.) in low back during trunk flexion and extension motion (Fick, 1911; Schultz et al., 1985). One would expect that the low back muscle activity gradually increases as trunk flexion angle increases in order to compensate for increased external moment of the mass of the torso. However, at some point (i.e., ~ 60° trunk flexion) passive tissues are stretched enough to offset the external torque (i.e., generating passive tension), and finally result in no muscle activity in the paraspinal muscles (i.e., EMG-off) (see Figure 2.14). During extension from this position, the low back muscle activity reappears (i.e., EMG-on) and increases up to full extension to generate active extension moment. The point of EMG-off and EMG-on is usually described in the lumbar angle (or lumbar curvature), because the FRP is directly influenced by the lumbar angle.

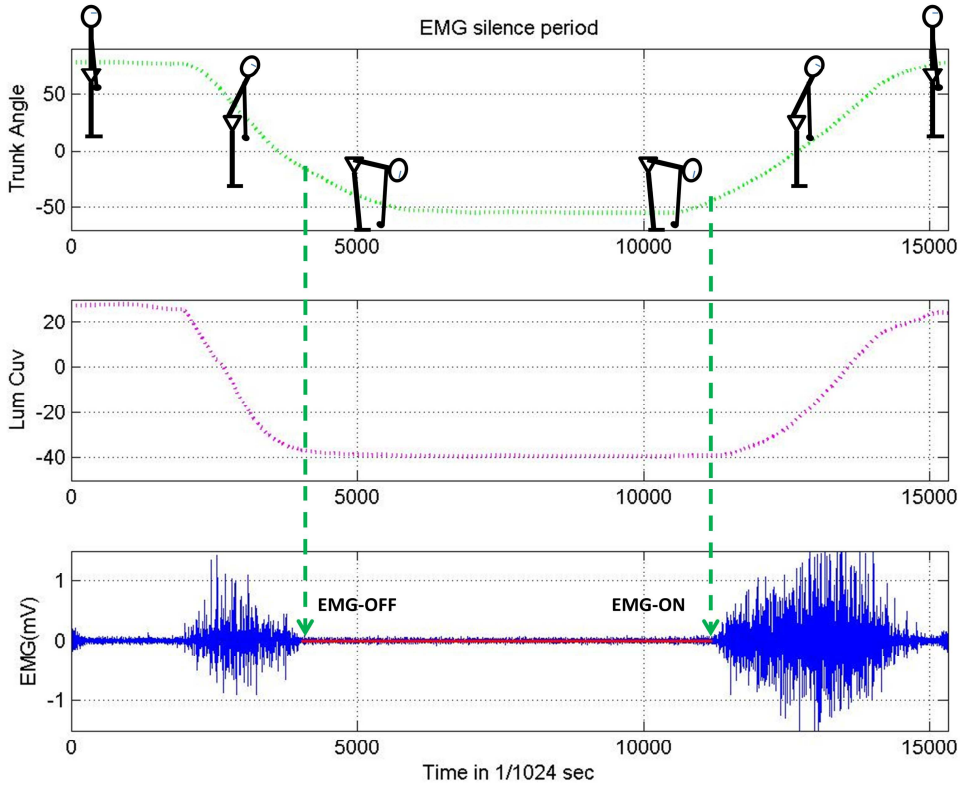

Figure 2.14 Flexion-relaxation phenomenon in a paraspinal muscle (Lum Cuv: Lumbar curvature)

From the viewpoint of the various reflex responses, it might be expected that the mechanoreceptors in stretched passive tissues during FRP will be stimulated at some point and activate the low back muscles like the reflexes in any other joint receptors. It has been suggested that the reflex response is modulated with trunk flexion and extension and controls low back muscle recruitment (Granata and Rogers, 2007; Solomonow et al., 2003c). However, the muscular activation with passive tissue stimulation did not happen in low back; instead of the muscular activation, the muscular deactivation (FRP) has been observed, so stretching of the passive tissues may be related to the muscular deactivation period (Solomonow, 2006). The inhibitory reflex was revealed in the passive tissues of the human subject (Dyhre-Poulsen and Krogsgaard, 2000; Voigt et al., 1998). In a study conducted by Dyhre-Poulsen and Krogsgaard

(2000), electrical stimulation was provided at the sensory nerve fibers inside the anterior cruciate ligament (ACL) while recording muscle activities. The results revealed a short inhibition of the muscle activity in both semitendinosus (flexor) and rectus femoris (extensor). The result suggested that the inhibitory reflexes could be initiated in the ligament-muscular reflex to prevent extreme stress on the joint in addition to the excitatory reflex from the ligaments to muscles. Considering that the passive tissue takes over the external moment at some degree of trunk flexion, the inhibitory reflex in low back may be beneficial from the view point of energy consumption. In addition, Granata and Rogers (2007) showed that the trunk stiffness was enhanced with an increase of lumbar flexion angle, even though there was almost no muscle activity, which they attributed to the tension of the passive tissues. Further studies relating to the reflex mechanism will be discussed in detail below.

Mechanical or neuromuscular control

A series of studies revealed the role and function of lumbar ligament-muscular reflex in flexion-relaxation phenomenon (FRP). First, the effect of gravity on FRP was tested (Olson et al., 2006). In this study, the subject was required to perform flexion-extension from upright standing and from supine positions (i.e., sit-up). In sit-up trials, the low back muscles were active in full flexion of the trunk, but the FR was observed in abdominal muscles and rectus femoris; the muscle activity in rectus femoris was similar with a muscle activity pattern of the low back muscles in flexion-extension from upright standing. The result suggests that the mechanical requirement for dealing with the internal moment which is influenced by the trunk mass and orientation to the gravity vector is a dominant factor in muscular activation during trunk flexion-extension (Solomonow, 2006). Also, Solomonow proposed that the trunk

kinematics and fixed reflex response are not significant contributors in trunk flexion-extension. Recently, Olson et al. (2009) examined the response of viscoelastic tissues and muscles in the low back after passive cyclic flexion-extension in upright standing posture. A dynamometer was used to support the subject's body mass so the subject was not required to actively generate flexion-extension movements. The passive flexion-extension cycle that included a 5 second flexion followed by a 5 second extension were performed over a 10 min period to generate passive tissue elongation. The results showed no muscular activity during trunk flexion and extension revealing no reflexes with passive tissue stretching in low back under the condition requiring no mechanical load on low back. Both study results denote that the mechanical requirement governs the lumbar ligament-muscular reflex and hence have a dominant influence on FR of the low back.

Definition of FR onset and cessation

The 'EMG cessation lumbar angle (EMG-off)' and 'EMG onset lumbar angle (EMG-on)' of FRP are the most common parameters in FRP studies (see Figure 2.14). However, there has been no common quantitative criterion to define the EMG onset and cessation angle of FR. The methods employed in previous studies are summarized in Table 2.1. The common observation in the methods is the use of data-smoothing techniques for having a smoothed curvature of trunk angle during flexion and extension. This process is necessary to smooth the variable EMG signal and establish the muscle activation profile during flexion-extension exertion so that the EMG-off and EMG-on points can be found. Six studies out of nine then employed the visual inspection method to define FRP and/or EMG-off and EMG-on. However, this technique is subjective and time-consuming. The other alternative method used

in a previous study was a reference-based technique (i.e. when EMG values were reduced to 3% or 5% MVC) by measuring the maximum voluntary exertion (MVC) or submaximal contraction. This method is objective and is easily programmed into computer software. However, the most severe problem of this technique is that the reference measurement itself creates variability between trials, so it can increase inter-individual variability (Mathiassen et al., 1995). Also, the MVC is not practical for chronic low back patients. Meanwhile, the submaximal reference technique is known to be more dependent on motor control strategies and hence produce significant variability between trials (Palmerud et al., 1995). In addition, both reference techniques may have higher intra-individual variation in the abnormal low back condition such as passive tissue stretching or muscle fatigue protocols, because the protocol modulates muscle activation patterns and increases the possibility of muscular spasm (unexpected peak of the EMG). On this basis, a new objective method defining the onset and cessation of FR is required.

Table 2.1 Summary of criteria to define onset and cessation of FRP

Authors	Threshold	Signal processing	Method
McGill and Kippers (1994)	3% of MVC	Low pass filtered at 2 Hz	Reference-based
Gupta (2001)	Abrupt changes	N/A	Visual inspection
Sarti et al. (2001)	Abrupt changes	100 ms moving average	Visual inspection
Dickey et al. (2003)	1% MVC	Low pass filter at 6 Hz; Down-sampled to 20.5 Hz	Visual inspection (only for less than threshold)
Solomonow et al. (2003a)	N/A	100 ms moving average	Visual inspection
Olson et al. (2004)	5% of peak EMG during extension	Smoothed at 0.5 Hz	Reference-based
Olson et al. (2006)	N/A	Smoothed at 10 Hz	Visual inspection
Descarreaux et al. (2008)	N/A	10-450Hz bandpass filtered	Visual inspection
Shin et al. (2009)	3% of MVC	Low pass filtered at 3 Hz	Reference-based

2.5.2. Factors influencing FR

The FRP has been investigated in various conditions to reveal its nature and significance in trunk stability and low back pain. First, an increased external load (e.g., hand-held load) retards the initiation angle of FRP during flexion (i.e., greater lumbar flexion) (Kippers and Parker, 1984; Dickey et al., 2003). The increased external load in hand could require the low back muscles to act longer until the tension generated by viscoelastic tissues in low back meets

the moment caused by external load; the later FR occurs at the biomechanical equilibrium point accounting for increased external moment between passive tissues and external moment.

Gupta (2001) conducted similar tests with Kippers and Parker in which the external loads were placed on the subject's hand (anterior load) or subject's back (posterior load: tied around the pelvis). The results showed that both anterior and posterior loads delay the initiation angle of FRP. He explained the results as follows: "Addition of weights, whether anterior or posterior to the hip axis produce increased tensile torque. This requires the balancing act of the erector spinae to continue longer till the extension torque by the posterior vertebral ligaments in increased proportionally enough at greater vertebral flexion." However, it seems unreasonable to believe that the posterior load tied around the pelvis generates the extension torque on the vertebrae. The hand-held load can increase the extension torque on the vertebrae in that the pelvis considered as a rigid body of the trunk movement provides skeletal foundation for the vertebrae. On the other hand, the load tied around the hip may just transmit the additional force to the ground throughout the biomechanical linkage of the lower extremity (see Figure 2.15). In this posture (Figure 2.15 (B)), the load could be employed to keep the body balance during trunk flexion, so the posterior migration of the pelvis controlled by the hip extensors does not happen. During full flexion, the hamstring muscles are fully stretched and provide tension on the pelvis (Olson et al., 2006). Also, the muscles show pretty similar activation patterns and FRP during trunk flexion-extension. It is plausible that the decreased tension generated by the hip extensors on the pelvis during trunk flexion requires increased vertebrae flexion to meet the biomechanical equilibrium between passive and active tissues.

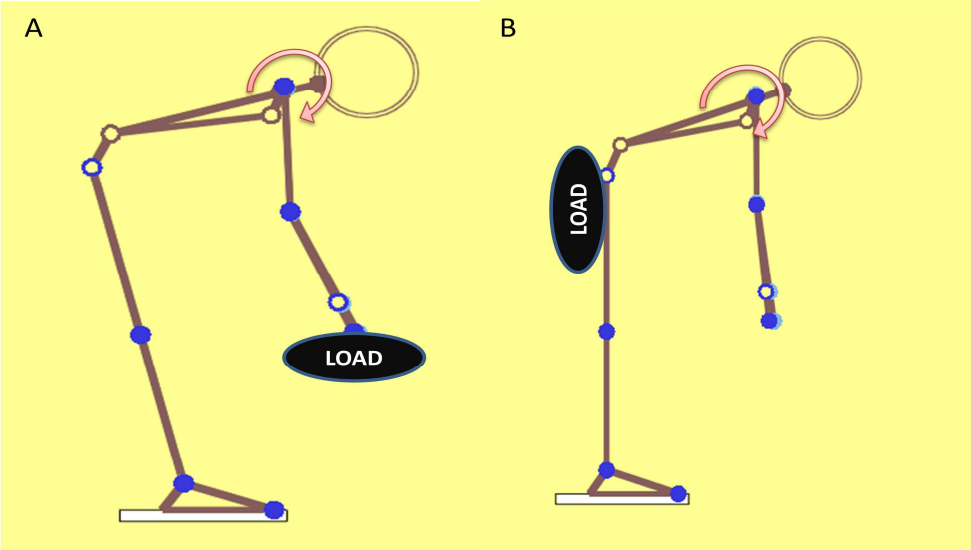

Figure 2.15 Effects of the load on the biomechanical equilibrium

Second, the body posture also influenced the mechanical requirement and hence FRP. Gupta (2001) investigated difference between the free flexion-extension motion (i.e., no restriction) and the buttock restricted condition (held against the wall). During flexion-extension motion, muscle activity in four muscles, including erector spinae, abdominals, hamstrings and the hip extensors, and two motion markers on iliac spine and C7 were sampled. The results showed that the lesser hip flexion in the buttock restricted condition reduced the overall trunk flexion angle at the same level of lumbar flexion and results in the decreased external moment produced by the gravity. Consequently, the FRP is initiated in the lesser trunk flexion angle in buttock restricted trials; the passive tissues may meet the required torque earlier to account for external moment.

Third, the condition of the tissues generating the internal moment in low back also affects the FRP. Laxity of the passive tissues requires deeper lumbar flexion angle in FRP, because of decreased passive moment generation capacity (Dickey et al., 2003; Shin et al., 2009; Solomonow et al., 2003a). Also, the fatigued low back muscles require a lesser lumbar flexion angle to initiate

the FRP, because the low back muscles lose active force generation capacity and cannot keep the lumbar lordosis which influences the passive moment generation (see Chapter 2.7.). The literature review suggests significance of the mechanical requirements in trunk flexion-extension rather than the neuromuscular responses.

In addition, a number of studies have revealed modulation of the FRP depending on a LBP patient's clinical status (Colloca and Hinrichs, 2005; Neblett et al., 2003; Shirado et al., 1995; Watson et al., 1997). The studies showed persistent myoelectric signal in deep trunk flexion and extension of chronic low back patients which discriminates the low back patients from the healthy counterparts. The reason for this different muscle activation pattern is not clear, but the authors usually suggested a protective mechanism of the passive tissues which takes over the external moment instead of the active muscular activation in healthy subjects.

2.6. Effects of prolonged stooping on low back function

The stooping or lumbar flexion posture is commonly adopted both in industrial settings and daily life for ground level tasks. In agriculture and construction industries, the static stooping posture for a prolonged period is known as one of the most challenging tasks for the low back (Goldsheyder et al., 2002; Rosecrance et al., 2006). For example, harvesting ground or bush crops (e.g., cucumbers, potatoes, melons, peppers, grapes, etc.) in the agriculture industry commonly requires prolonged static stooped postures and lateral bending of the torso that could be directly or indirectly linked with development of low back pain (Meyers et al., 2001).

2.6.1. Effects of lumbar passive tissue strain on FRP

Prior studies have acknowledged that prolonged static flexion can modify the nature of FRP. FRP studies have demonstrated its significance and reliability for researching abnormal low back conditions in both chronic low back patients and the healthy population (see Chapter 1.3.) (Shin & Mirka, 2006; Shin et al., 2009; Solomonow et al., 2003a and 2003c; Solomonow, 2004). The prolonged static flexion elicits higher strains on lumbar passive tissues such as interspinous and supraspinous ligaments (Panjabi et al., 1981) and elongates the passive tissues (Rogers and Granata, 2006; Shin & Mirka, 2006; Solomonow et al., 2003a). The deformation in passive tissues is critical in that it has an important role in generating moment, supporting the torso and controlling spinal reflex as joint receptors. There are a multitude of studies revealing the relationship between FRP and passive tissue deformation in low back.

Solomonow et al. (2003a) conducted an *in-vivo* research in which the participants were asked to perform three trunk flexion-extension exertions before and after a 10 min period of the static trunk flexion protocol. The results showed that after prolonged static flexion the erector spinae are active through larger flexion angle (i.e., later deactivation) and initiated earlier in extension motion. In other words, the relaxation period of erector spinae was reduced after the protocol. The authors explained the changes as a compensation for the loss of tension in lumbar ligaments which may result in spinal instability. From the view point of biomechanics, the passive equilibrium point between the external moment (torso) and viscoelastic tissues (ligament) shifts to deeper lumbar flexion posture because of the laxity in the ligaments which have reduced moment-generation capacity compared to before the prolonged static stooping at the same level of lumbar flexion. Deeper lumbar angles could be required to account for the external moment for producing enough tension on the ligaments.

Similar results were reported in later studies in which the subjects were required to have cyclic flexion-extension. Dickey et al. (2003) conducted a study requiring 100 trunk flexion-extension (4.5 sec flexion, 2 sec handing and 4.5 sec extension). The results revealed a shortened silence period with deeper maximum flexion angle after the protocol; the effect of cyclic flexion-extension was similar with the prolonged stooping protocol in this study. On the other hand, Olson et al. (2004) also conducted a study requiring cyclic lumbar flexion-extension (5 sec flexion and 5 sec extension) for 9 min, and showed earlier cessation of EMG during flexion and delayed activation of EMG during extension; the effect of cyclic flexion-extension was similar with the muscle fatigue protocol (more detail in Chapter 2.7). Also, they showed no change in maximum flexion angle. Main difference between two studies was the number of cyclic flexion-extension (100 in Dickey's study and 56 in Olson's study), but it is not clear to explain why they showed opposite results. Further study is needed to clearly understand the effect of cyclic flexion-extension.

2.6.2. Effects of lumbar passive tissue strain on spinal reflexes

Solomonow et al. (2003c) also conducted an *in-vivo* study using a live feline model to reveal the effect of creep of lumbar viscoelastic tissues on spinal reflexes. In this study, a series of three 10 min static flexion protocols using S-shaped hook inserted around the supraspinous ligaments with each session followed by a 10 min resting was performed on the spine of the feline model. The researchers observed an exponential decrease in the reflexive EMG activities of the low back muscles during static loading of the lumbar ligaments. However, the variation of the muscular response was increased because of random or unpredictable muscle spasms (sudden, involuntary contractions of muscles accompanied by pain) (Dorland, 2007). During a

7-hour resting period, an initial hyper-excitable spinal reflex response, which is a rapid, automatic response (e.g., muscle activation) to specific stimuli, was observed in the first 20 to 30 minutes, followed by 1 to 2 hours muscular depression. The reflex disturbance was different according to the magnitude of the load applied to the ligament. The second muscular hyper-excitability was initiated after two or three hours and lasted until the end of the 7-hour rest session. Consequently, the spinal column may lose stability because of the loss of tension in lumbar ligaments and the depression in spinal reflex (i.e., lower EMG response). Those changes after prolonged stretching of viscoelastic tissues in low back could result in different muscle activation patterns in the trunk and influence the spinal stability control and risk of LBP.

Rogers and Granata (2006) quantified the spinal reflexes by using systems identification analyses of the EMG responses in human subjects. They confirmed the passive tissue elongation in the lumbar spine by the increase of lumbar flexion angle first and then showed depression in spinal reflex after 4 cycles of 4 minutes' static lumbar flexion in sitting posture and 1 minute of upright sitting between cycles; note that the reflex gain was measured in upright sitting posture only. The lumbar curvature increased by an average of 17° in male subjects and 22° in female subjects after the protocol. Consequently, the decrease in reflex gain was also higher in female subjects (0.4 %/N (male) vs. 1.7 %/N (female)). Granata and Rogers (2007) attributed this gender difference to the torso mass. They hypothesized that the smaller trunk mass and inertia of the female subjects may bring about greater velocity and acceleration of the torso than the male subject generally having the large mass and inertia for the same force disturbance in both genders, and result in higher reflex gain. Moorhouse and Granata (2007) performed the nonlinear systems-identification analyses to investigate the role of the spinal reflex components of spinal stability during voluntary exertions, and suggested that the

movement velocity of the torso is highly related to the reflex response. In addition, the reflexes did not return to the baseline level during 16 minutes of recovery. However, a previous study showed results where the reflex gain was significantly increased after 15 min of prolonged static full flexion posture (sat on the floor) in female subjects (from 1.333 to 1.724 %/N) (Granata et al., 2005). There was no difference in the reflexes of the male subjects (from 1.070 %/N to 1.038 %/N). Some major differences between the two inconsistent studies should be noted. First, the position of the subject during the reflex trials was different (upright sitting in 2006 vs. upright standing in 2005). Second, the posture during the full flexion protocol was different (sitting on the chair with pelvic restraint in 2006 vs. sitting on the floor without pelvic restraint in 2005).

The main difference between the two studies may be the contribution of the lower extremity. As already discussed in Chapter 2.4.4., the lower extremity muscles may contribute to the spinal stability with significant biomechanical linkage between trunk and lower extremity. The sitting posture generally provides a more stable base than a standing posture. In addition, the lower extremity was not restricted during the full flexion protocol in 2005, so the sacroiliac joint stability could be disturbed. The unstable base during the reflex measurement trials in Granata et al. (2005) could have a more severe mechanical demand in female subjects (i.e., more disturbance) than the male subject because of the smaller trunk mass and inertia, and the female subjects may have tried to compensate by activating more motor units. The external mechanical disturbance is also bigger in Granata et al. (2005) than Roger and Granata (2006) suggesting a more challenging condition for the female; three levels (100, 135 and 170 N) in 2005 and only one level (100 N) in 2006. It is not clear whether the conflicting results reflect simple methodological differences or more fundamental biomechanical response.

A recent study investigated the influence of muscle fatigue or passive tissues elongation on reflex-onset latency (captured by EMG) in the lumbar muscles to the sudden disturbance (Sánchez-Zuriaga et al., 2010). The participants were subjected to two interventions: (1) full flexion in sitting posture for 1 hour to induce laxity in lumbar passive tissues; and (2) the Biering-Sorensen test to induce low back muscle fatigue. They showed delayed reflex response by 36 milliseconds (a 60% increase in onset latency) in soft tissue creep deformation, but there is no difference after the muscle fatigue protocol. The results suggest impaired reflex after prolonged stooping and inability to respond quickly to a sudden loading. At this point, the only thing to be confirmed is that there is a negative modulation in reflex response after passive tissue deformation. Further research is required to reveal the change in spinal reflex after the prolonged static flexion.

Additionally, it should be noted that the experimental conditions in the studies investigating effect of laxity in passive tissues of human lumbar spine was significantly different with Solomonow's work (Solomonow et al., 1998 and 2003c) in that the muscle of the human subjects was also passively stretched during the full flexion protocol. The sustained stretching of the muscles may desensitize the muscle spindles, which is known to activate motoneurons via the stretch reflex to resist muscle stretch. Avela et al. (2004) investigated the effect of repeated and prolonged passive stretching of the triceps surae muscle group on neural and mechanical responses. The result revealed reduced motor unit activation, reduced reflex responses and reduced force-generation capacity. Based on this, the results of Granata's group and Sánchez-Zuriaga et al. (2010) should be considered as a combination of passive and active tissue stretching.

2.6.3. Effects of lumbar passive tissue strain on EMG activities

The muscular activation level has been reported in some studies investigating the effect of prolonged stooping. Shin and Mirka (2007) measured the average normalized EMG (NEMG) in trunk extension phase (isokinetic) during 10 minutes of prolonged stooping protocol and another 10 minutes of recovery. The results revealed an increase of the NEMG in both multifidus and erector spinae of 35% and 40.9%, respectively, after the static stooping protocol. The increased NEMGs of the low back muscles almost recovered to the initial level after a 10-minute recovery session. They pointed out that the trend of extensor muscle activities during the static stooping protocol and the recovery session resemble the magnitude of the passive tissue elongation in the low back (i.e., lumbar flexion angle in full flexion posture).

Shin et al. (2009) investigated the fatigue development in low back muscles after 5 minutes prolonged stooping. They showed a significant increase of NEMG in 15% and 30% isometric contraction and a significant decrease in median power frequency after the protocol. Both studies suggested that the low back system requires more muscle activation to compensate the reduced tension-generation capacity in passive tissues and spinal instability caused by laxity in passive tissues. Also, the authors proposed that the fatigue-like response in lumbar muscles due to the passive stretching of muscles may also reduce the force generation capacity at a given motor unit, and hence increase the EMG activity (i.e., recruit more motor units) for generating the same level of force.

The results concerning the muscular activation level after passive tissue stretching are not directly linked with the reflex response if we accept the principle of reduced reflex response after static flexion. In Granata's studies, the higher reflex suggests greater EMG signals per unit of external perturbation force. The importance of the mechanical requirements in the reflex

response demonstrates a limitation in Granata's studies, namely that the reflex response was only recorded in the upright posture. On the other hand, the muscle activities were recorded during the extension phase of lifting (isokinetic) and 60 degrees of trunk flexion (isometric), denoting different mechanical requirement (i.e., effect of gravity). Further research will be required to reveal the nature of the discrepancy between studies.

2.7. Effects of lumbar muscle fatigue on low back function

The influences of low back muscle fatigue have been largely explored, and reduced force generation capacity (Bonato et al., 2003; Clark et al., 2003), reduced position sensitivity (O'Sullivan et al., 2003), increased spinal load (Dolan and Adams, 1998) and changed lifting patterns (Marras and Granata, 1997) have been suggested. The negative impacts of muscular fatigue may therefore cause spinal instability and modulate the FRP.

2.7.1. Effects of lumbar muscle fatigue on FR

Descarreaux et al. (2008) investigated the influence of low back muscle fatigue on FRP parameters including initiation and cessation angles of FR. The results revealed that the FR was initiated earlier (e.g., less lumbar flexion), and terminated later when muscle fatigue was present (i.e., opposite effect compared to passive tissue stretching). The authors suggested that the presence of low back muscle fatigue limits force generation capacity of the muscles providing sufficient stabilization to the spinal column, so the fresh passive tissues are charged earlier to compensate for the decreased force generation ability of low back muscles. They mentioned that "the low back muscles, in a state of fatigue, are not able to provide sufficient stabilization to the vertebral units, transferring load-sharing to passive structures earlier in trunk flexion."

However, because FR only occurs at the biomechanical equilibrium point between passive tissues and external moment generated by the torso, the hypothesis proposed by Descarreaux et al. (2008) is not true. The occurrence of FR could not be modified by reduce moment generation capacity of the fatigue muscle. The passive tissues are only "passively" activated at a specific length (i.e., at a specific angle) if there is no change in viscoelastic properties of the passive tissues; the FR is controlled by trunk movement (i.e., spine movement). In other words, the FR condition (i.e., transferring load-sharing to passive tissues earlier) can only occur when the passive tissues generate required tension earlier than the normal condition.

It is interesting to note that a literature review showed that the passive elastic tension increased around full stretching length after both isometric fatiguing protocol and eccentric contraction protocol in the triceps surae and right calf muscles (Whitehead et al., 2001; Finlayson et al., 2008). The authors explained it as immediate strain injury contractures, referring to a contraction of the fiber in the absence of an action potential, in damaged muscle fibers after muscle fatigue protocol. It is possible that the strain contracture in low back muscles results in the reduced muscle length and increased passive tension. This hypothesis can explain the earlier biomechanical equilibrium point between passive tissues and external moment generated by the torso. Additional information such as lumbar curvature and trunk angle in full flexion posture before and after the fatigue protocol could be required to confirm the hypothesis.

2.7.2. Effects of lumbar muscle fatigue on reflex response and stability

Reduced neuromuscular control ability of trunk movement after fatigue in low back muscles was suggested by Parnianpour et al. (1988). They investigated the effect of muscle fatigue on the maximum torque generation, the range of motion (ROM), the maximum and

average velocity during isoinertial movement in the three axes of rotation including sagittal, coronal and transverse plane. During the experiment, the subjects were asked to perform trunk movement, from upright to full flexion, as quickly and as accurately as possible while exerting maximum force on the dynamometer. The results revealed significant decreases in the maximum torque, the ROM and the maximum and average velocity in sagittal plane. In addition, the ROM and the maximum and average velocity in coronal and transverse plane were significantly increased under the muscle fatigue condition. The authors suggested significantly less motor control ability under the condition of the low back muscle fatigue. The neuromuscular control is important in that inability in reflex response could result in spinal instability and, consequently, LBP (McGill and Cholewicki, 2001).

Herrmann et al. (2006) investigated the reflex amplitude and delay before and after the muscle fatiguing protocol by using EMG at onset and peak points. They showed a significant increase in reflex amplitude (36%), but the reflex latency was not changed. Results suggest that the decrease in force generation capacity of the fatigued muscles requires greater activation to keep sufficient spinal stability.

The fatigued low back muscles also change the trunk muscle activation pattern and trunk stiffness. Granata et al. (2004) used a biomechanical model to compute the effects of muscle fatigue on spinal stability and revealed a significant increase in abdominal muscles to sustain the spinal stability after the muscles fatiguing protocol. They also showed a linear decrease in trunk stiffness and a linear increase in spinal compression with low back muscle fatigue. Recently, Granata and Gottipati (2008) quantified the trunk stability from the maximum finite-time Lyapunov exponent before and after muscle fatiguing protocol. The stability assessment task was to touch a target located near knee level with hands in time with a metronome sound (30

cycles per min), while recording muscle activity and lumbar motion. The results showed the maximum Lyapunov exponents and mean kinematic rate of expansion were significantly increased after the fatigue protocol suggesting poorer trunk stability. In summary, the muscular fatigue in the low back could bring about reduced neuromuscular control (i.e., decrease reflex) and result in spinal instability.

Chapter 3 – PROBLEM STATEMENT

3.1. Importance of the system-level approach

Standard anatomic classifications can be misleading regarding the functional role of the body segments, which are strongly connected and interact with each other. Chapter 2 provided ample anatomical evidence for the significant biomechanical linkage between trunk and lower extremities and its function were discussed. First, it was shown that the origin and insertion of the lumbar muscles and the lower extremity muscles are directly and indirectly connected with pelvis and ligaments around SI joints, and suggested that the activation of the muscles can influence the trunk and SI joint stability. Second, the lumbodorsal fascia, covering the paravertebral musculature and linked with the gluteus maximus and biceps femoris and the transverse and internal oblique, can transfer load between spine, pelvis, and lower extremity and stabilize the trunk and pelvic systems. Plenty of evidences in previous literature open the possibility to consider a system level (i.e., trunk, pelvis and lower extremities) approach for better understanding of trunk stability. Further, the functional roles of the components of the global and local systems proposed by Bergmark (1989) (Chapter 2.4.3) provide some interactivity, but the role of the lower extremities is required to be investigated as a 'super global system'.

3.2. FRP in abnormal low back condition

Abnormal low back conditions such as muscle fatigue or laxity in passive tissues have been widely investigated to achieve better understanding of LBP. Recent studies start to reveal how the FR is modulated. Given the fact that local system, global system and super global systems are strongly connected, generation of internal torque, especially passive moment generated by ligaments and lumbodorsal fascia around full flexion, for flexion-extension is not

only controlled by the local system but also influenced by global and super global systems as discussed in Chapter 2.4.4. In other words, the FRP can be modified by the contribution of the lower extremity muscles and pelvic angle by indirectly generating passive tension on lumbodorsal fascia. The functional role of global and super global muscles should be revealed for better understanding of the FRP and trunk stability systems.

Figure 3.1 represents a proposed control mechanism for trunk stability during trunk flexion-extension. The passive monitoring system will monitor the current low back condition (e.g., muscle fatigue, viscoelastic tissue deformation) and mechanical requirements, and control trunk stability and flexion-extension. The mechanical requirements are modulated by external torque, including external load (trunk mass, hand-held load, etc.), and internal torque, including trunk kinematics (e.g., passive tissue contribution, force-length relationship, velocity, etc.). As already discussed, mechanical requirements for trunk stability control involves the muscle activation of three active subsystems: global, local and super global. The passive system generating passive moment at some degree of deformation interacts with the active muscle system by a load-sharing mechanism to achieve trunk movement. The body movement may directly modulate the mechanical requirements monitored by the passive monitoring system, and proper adjustment will be initiated again. In this figure, the passive systems are conceptually divided into two main roles in trunk movement as a monitor and a stabilizer; actually they are a single system. The conceptual model suggested in Figure 3.1 should be confirmed in an empirical study.

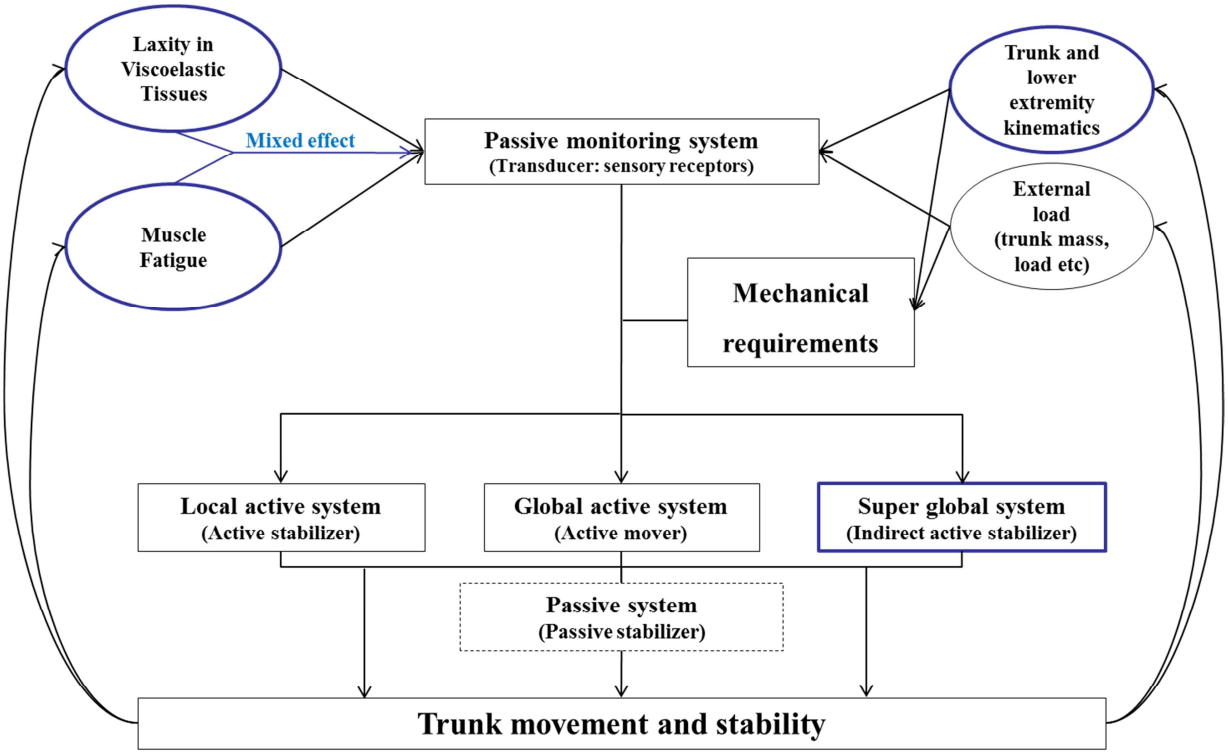

Figure 3.1 The control system of trunk stability and flexion-extension

3.2.1. Effect of prolonged stooping

Previous studies revealed that passive tissue elongation in low back modifies FRP and reduces trunk stability (see Chapter 2.6). The results partially support the conceptual model presented in Figure 3.1, but the studies only include local muscles such as multifidus or medial erector spinae. Consequently, they failed to show the effects of abnormal low back conditions on global and super global muscles. It is possible that the degraded moment generation capacity (i.e., stabilizing ability) caused by laxity in the lumbar passive system and SI joints can modify muscular activation patterns in global and super global systems to achieve better spinal stability. On this basis, the role of the global system and the super global system should be investigated under passive tissue elongation in conjunction with change in FRP revealing modulation of mechanical requirements in a load-sharing mechanism.

3.2.2. Effect of muscle fatigue

It is well known that fatigue in low back muscle modifies muscle activation patterns in trunk muscles including both global and local systems. Recently, researchers showed significant modulation in FRP denoting changes in the load-sharing mechanism and also confirmed a decrease in trunk stability. However, it is still not clear why the FRP is modified in muscle fatigue condition. The hypothesis suggesting increased passive elastic tension in low back muscles seems most plausible at this point (see Chapter 2.7.1). If the hypothesis is true, the lumbar curvature in full flexion posture will be decreased compared with the initial lumbar curvature in full flexion. On the other hand, there is no study investigating the role of the super global system under low back muscle fatigue. A literature review showed a strong biomechanical linkage of lumbodorsal fascia in Chapter 2.4.4. and Figure 2.11 and an increase in tension of

lumbar lamina with super global muscle activation. On this basis, the role of the super global system requires investigation under local muscle fatigue.

3.2.3. Combined effect of muscle fatigue and passive tissue elongation

In an industrial setting, workers usually conduct a task for prolonged period and manually lift or move products periodically (e.g., assembly line workers, farmers in crop production and concrete workers in the construction field, etc.). Those tasks are expected to generate both passive tissue stretching and muscle fatigue simultaneously. Previous studies revealed a single effect of prolonged stooping or manual lifting on low back. However, there is no study considering the combined effect of prolonged stooping and manual lifting. Given that the negative influence of the single abnormal condition, greater instability in the trunk system and greater muscular activation in the global and super global systems are expected. Meanwhile, considering that the two single effects are expected to have opposite effect in FRP and full flexion angle, it is not easy to predict the results. Considering the opposite responses of the two single effects in full flexion angle and EMG-off angle, no significant change from the initial condition is expected under the equal influence of the two effects. However, the muscle fatigue could be a dominant factor modulating the FRP and trunk flexion-extension system than the laxity in passive tissues in that the active system has significant effect in mechanical requirements as compared to the passive system. In other words, the passive system can only be activated by trunk movement initiated by the active system, so inability in the active system could be a dominant factor. Based on this, the combined effect protocol may show a weak effect of the muscle fatigue protocol even the difference is not significantly different.

A conceptual model in Figure 3.2 summarizes the possible mechanism of the low back caused by prolonged stooping and repeated lifting discussed in Chapter 2 and 3.

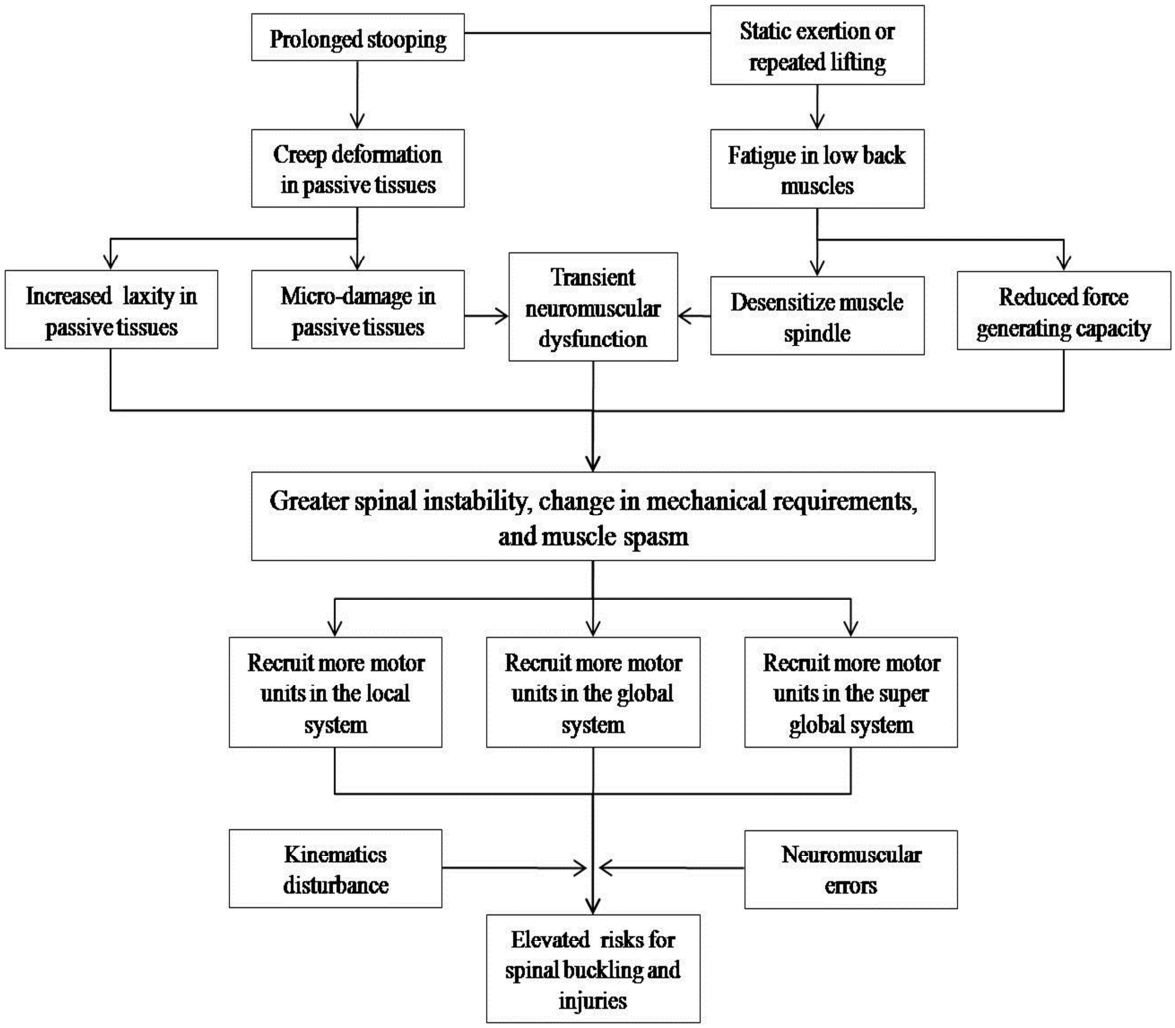

Figure 3.2 A conceptual model for combined effect of prolonged stooping and manual lifting

Chapter 4 – PRELIMINARY STUDY

Jin, S., and Mirka, G.A. (In press). "The Effect of a Lower Extremity Kinematic Constraint on Lifting Biomechanics", *Applied Ergonomics*. doi: 10.1016/j.apergo.2011.02.003

4.1. Relevance

A strong biomechanical linkage between the torso and the lower extremities and the functional role of lower extremities in trunk flexion-extension are central to the system-level modelling perspective pursued in this dissertation. To some extent this interaction has been explored to reveal the hip-spine coordination (the lumbopelvic rhythm) (see Chapter 2.2.2 and 2.4.4). Also of importance is that during flexion and extension movement, the posterior movement of the unconstrained pelvis is usually adopted to keep the balance of the body, and the gluteus and hamstring muscles actively control the movements. Although the posterior movement of the pelvis is useful to maintain balance of the body it also increases the moment arm between L5/S1 and the load, requiring more lumbar muscle activity (Gupta, 2001; Nelson et al., 1995). Based on this, it is possible that a change in the lower extremity kinematics can modify the trunk muscle activity and passive tissue moment. The focus of this pilot work study was to explore the effects of a kinematic constraint (constraining motion of the thigh) on this interaction.

4.2. Objectives

In a previous study (Shu et al., 2007), the role of kinematic constraint of the lower extremity was investigated, focusing on the effect of a shin-level kinematic constraint on low back biomechanics during lifting. This study evaluated the differences in activation levels of trunk extensor muscles while kneeling on a knee support (i.e., loss of the degree of freedom of

the ankle joint). In this study the participants were asked to maintain a designated trunk flexion angle and then receive and hold a weight that was released into their hands by the experimenter. The kinematic constraint eliminated the motion of the ankle joint but allowed participation of the knee joint in supporting this load. Their results showed that the loss of the degree of freedom at the ankle joint had little effect on the activation level of latissimus dorsi and multifidus muscles during this task. While this previous study provided some information regarding the effect of a kinematic constraint, it was somewhat limited in that it only considered the constraint on the ankle joint – a joint with relatively limited direct impact on low back function. It was felt that limiting the participation of the knee joints through a kinematic constraint may be much more impactful on the function of the low back. The goal of current study was to investigate the effect of a thigh-level kinematic constraint by leaning against the barrier on trunk muscle activation (i.e., active tissue activation), trunk kinematics and moment generated by passive tissues in low back.

4.3. Methods

4.3.1. Overview of the study design

The lower extremity kinematic constraint employed in this study led to the loss of two degrees of freedom in the kinematic chain (ankle and knee joints). There were two phases in this study: a static phase that involved static weight-holding tasks and a dynamic phase that involved free dynamic lifting tasks. The static trials were designed to understand how the muscles of the lumbar region function under leaning and no leaning conditions. The dynamic trials were designed to quantify the trunk kinematics and ground reaction forces during the leaning and no leaning conditions.

Thirteen male participants were recruited from the university undergraduate and graduate student population of Iowa State University. They did not report any chronic problems or current pain in the low back or lower extremities. Each participant provided written informed consent prior to participation. The average and standard deviation of age, stature and whole body mass of participants were 28.1 yr (4.0), 172.5 cm (2.7), and 71.5 kg (7.2), respectively.

4.3.2. Experimental apparatus

The experimental setup was designed to simulate a 82 cm height barrier which served as the lower extremity kinematic constraint during leaning conditions. The load was a 60 cm (L) × 60 cm (W) × 35 cm (H) box with a mass of 9 kg.

During the static phase, surface electromyography was used to capture the activities of the ten sampled muscles (Model DE-2.1, Bagnoli™, Delsys, Boston, MA) (data collected at 1024 Hz), and a magnetic-based motion analysis system was used to capture the instantaneous lumbar curvature (The MotionMonitor™, Innovative Sports Training, Chicago, IL) (data collected at 102.4Hz).

During the dynamic phase, the lumbar motion monitor (LMM) (Chattanooga Group Inc., Chattanooga, TN) was used to capture the three-dimensional trunk kinematics (data collected at 60 Hz). A Bertec force platform (Bertec, Columbus, OH) was used to capture ground reaction forces and moments (data collected at 60 Hz).

4.3.3. Experimental design

A 2 × 3 repeated measure design was employed that had two levels of posture (POSTURE: leaning, no leaning) and three levels of load height (HEIGHT; 85 cm, 70 cm and

55 cm from the ground level) which refer to the height of the hands as the participant grasped the box. There was one replication of each of the six conditions in each phase resulting in twelve trials in both the static and dynamic phases of the experiment. All trials within each phase were completely randomized.

In the static phase there were six dependent measures and during the dynamic phase there were two dependent measures. The average (across muscle pairs), normalized EMG included five bilateral muscles: erector spinae (ES), latissimus dorsi (LD), rectus abdominis (RA), external oblique (EO) and gastrocnemius (GAS). The extensor moment generated by the passive tissues low back was estimated using the technique of Dolan et al. (1994) (described in more detail in following Chapter). In the dynamic phase of the experiment, the peak sagittal plane angular acceleration was found from the LMM data and the peak anterior-posterior ground reaction force was found using the force platform. Both were captured during the concentric lifting motion.

4.3.4. Experimental procedures

Upon arrival the experimental procedure was described to the participant and informed consent was obtained. The participants then participated in a five minute warm-up session to prepare the muscles of the low back and lower extremity. The ten surface electrodes were secured on the skin over the selected muscles. The sampling locations for these muscles are as follows: (1) erector spinae: 3.5 cm from the vertebral midline at L2 level, (2) latissimus dorsi: most lateral portion of the muscle at the level of T9, (3) rectus abdominis: 5 cm above the umbilicus and 3 cm lateral to the midline, (4) external oblique: 10 cm from the midline of the abdomen and 4 cm above the ilium at an angle of 45° and (5) gastrocnemius: 2 cm medial from

the midline of calf (location of largest muscle mass). The participant completed a series of isometric maximum voluntary contraction (MVC) exertions. For the erector spinae, rectus abdominis and external oblique muscles, a lumbar dynamometer was used to provide a static resistance at the 40 degree trunk flexion angle (Marras and Mirka, 1989). For the gastrocnemius muscles, participants were asked to rise on the balls of their feet against manual resistance on their shoulders provided by the experimenter. For latissimus dorsi, participants asked to bend their elbow to 90 degrees, abduct their shoulder to 90 degrees and maximally adduct against manual resistance provided by the experimenter. Two magnetic sensors were then secured on the skin on the midline of the spine - one at the L1 level and the other at the S1 level. The participants were then asked to stand in an upright posture and then to bend forward to a full trunk flexion posture to establish their full sagittal plane range of motion. As they performed this activity data from the magnetic motion sensors on L1 and S1 were captured. This was used to calibrate (express as % of range of motion) the lumbar motion data collected during the experimental trials.

Before beginning the experimental trials, verbal instructions were provided describing the leaning and no leaning postures. Participants were told that during the leaning condition they were to lean against the railing with both thighs and that they should not touch the railing during the no leaning condition (see Figure 4.1). The participants were asked to step on to the force platform and find a comfortable width of their feet. This location of their feet was marked and they were told to keep their feet in this position throughout the experimental trials. The trials in the static phase required that the participant flex the torso and grasp the load (9 kg box) and lift it ~5 cm from its resting height and hold that posture for 5s while EMG and magnetic motion

sensor data were collected. Between trials, participants were given a rest period of 20 seconds. After completion of all trials, the electrodes and the magnetic sensors were removed.

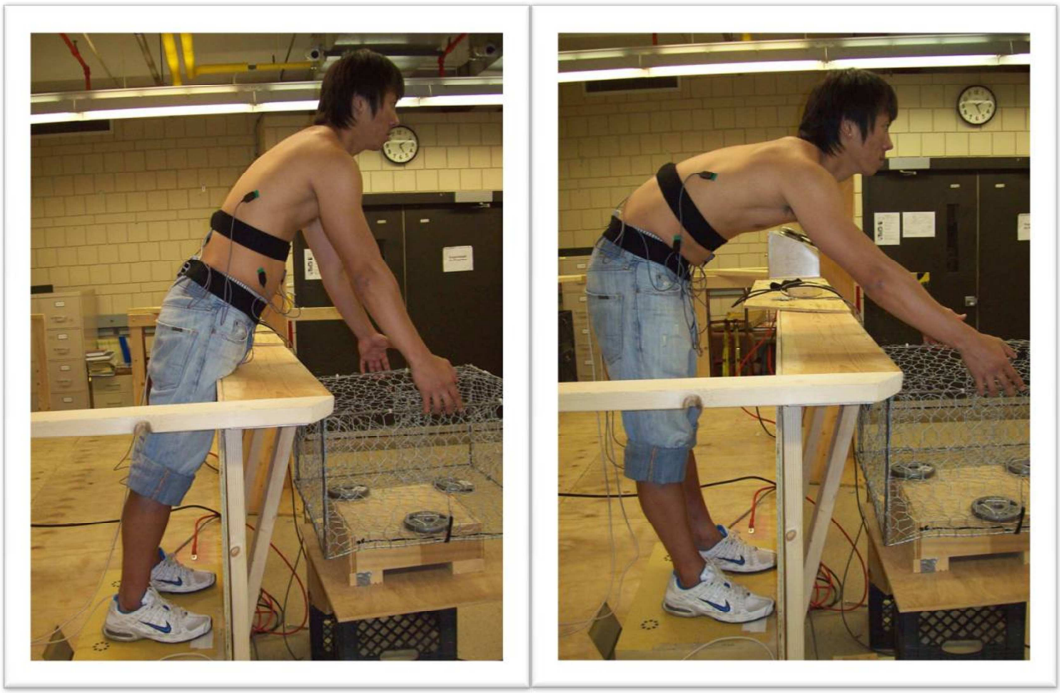

Figure 4.1 Experimental task: comparison of leaning and no leaning condition.

The second phase began by securing the LMM to the back of the participant and they returned to their position on the force platform. During the lifting trials the participants began in an upright position, bent over to grasp the top of the load and come to an upright position, lifting the pot into the boat. During the trials, both LMM and force platform data were collected. Two trigger signals, one at the point when the participant first touched the crab pot and the other at the end of lifting motion (full upright posture), were also recorded. A rest period of 20 seconds was provided between trials.

4.3.5. Data processing

The unprocessed EMG data collected during static phase of the experiment were filtered (high-pass 10 Hz, low-pass 500 Hz and notch filtered at 60 Hz and 102.4 Hz and their aliases). For the MVC exertions, the filtered signals were full wave rectified and averaged into 1/8 second windows. The maximum 1/8 second window was indentified for each muscle group and was used as the denominator in order to normalize the EMG data during lifting tasks. For the EMG data collected during experimental trials, the filtered signals were full wave rectified and then averaged over the static weight holding time period. These values were used as the numerator in the normalization process. Finally, the normalized EMG of the right and left muscles of each bilateral pair were averaged.

The sagittal plane angles measured by magnetic sensors placed on L1 and S1 were used to calculate passive moment on low back during static trials. Lumber curvature (LC) was calculated for both the including upright standing and full flexion postures, and the static experimental trials using Equation 1. These values were then used to measure percentage of range of flexion using Equation 2. Finally, this percentage flexion value was used to calculate the passive tissue moment employing by Equation 3 (Dolan et al., 1994).

$$\text{Lumbar curvature (LC in deg)} = \text{Sagittal Angle}_{(L1)} - \text{Sagittal Angle}_{(S1)} \quad (1)$$

$$\text{Percentage Flexion (PF in \%)} = \frac{[LC - LC_{standing}]}{[LC_{fullflexion} - LC_{standing}]} \times 100 \quad (2)$$

$$\text{Passive tissue moment (in Nm)} = 7.97 \times 10^{-5} \times PF^3 + 12.9 \quad (3)$$

All statistical analyses in this study were conducted using SAS®. Prior to model analysis, diagnostic tests were performed on the data, including, test for homoscedasticity (Bartlett's Test and Levene's Test) and normality (Anderson-Darling Normality Test) (Montgomery, 2001).

Dependent variables that violated one or more assumption were transformed so that the ANOVA assumptions were fully satisfied (Montgomery, 2001).

Due to the multivariate nature of the data collected in this study, both MANOVA and univariate ANOVA techniques were used. Multivariate analyses of variance (MANOVAs) were conducted on all response measures to control the experiment-wise error rate. Only those independent variables found to be significant in the MANOVA were pursued further in the univariate ANOVA. Post hoc tests employing Bonferroni's method were then performed on these significant main effects. A p-value less than 0.05 were regarded as the standard level of significance of an effect in current study.

4.4. Results

The results of MANOVA for average NEMG showed significant effects of POSTURE and HEIGHT, but there was no significant interaction effect between POSTURE and HEIGHT (see Table 4.1). Accordingly, the interaction effect was not considered in subsequent data analysis. Univariate ANOVAs were conducted on each of the five muscles and revealed a significant effect of POSTURE on all five selected muscle activities. The results showed that a leaning posture requires significantly lower muscle activation as compared to no leaning posture in the trunk extensors, trunk flexors and the gastrocnemius (see Figure 4.2). The effect of HEIGHT was to have ~20% reduction in gastrocnemius activity at the higher load position.

Table 4.1 MANOVA and ANOVA results for average, normalized EMG

Independent Variables	MANOVA (Wilks' lamdba)	ANOVA results				
		Dependent Variables				
		ES	LD	RA	EO	GAS
Posture	**p < 0.0001**	**P < 0.0001**	**P < 0.0001**	**P < 0.0001**	**P < 0.0001**	**P < 0.0001**
Height	**p < 0.0001**	p = 0.2753	p = 0.5628	**p = 0.0003**	p = 0.6261	**p = 0.0002**
Posture × Height	p = 0.0673	N/A	N/A	N/A	N/A	N/A

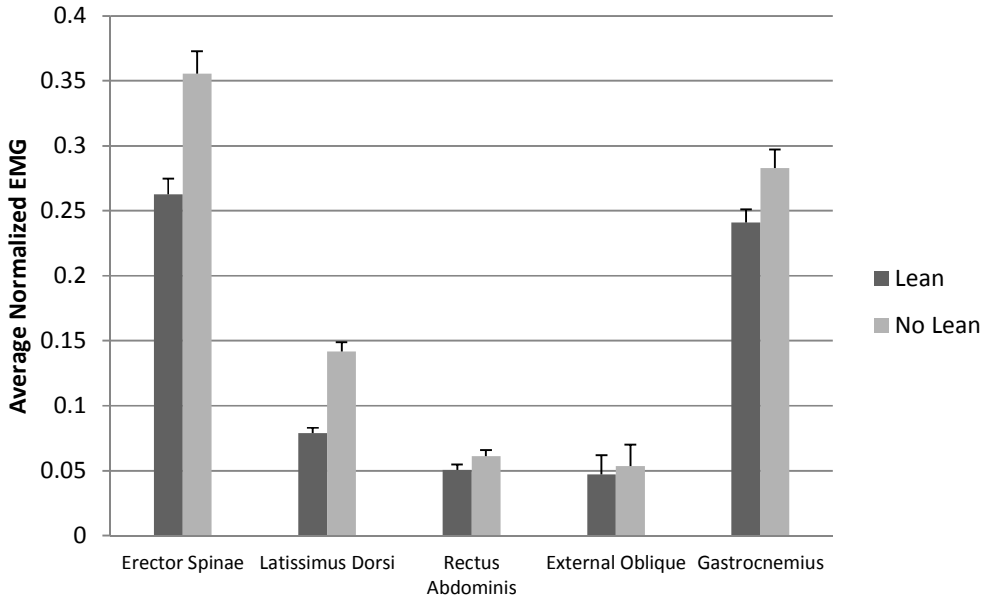

Figure 4.2 Effect of the POSTURE on NEMG (Error bars show standard error.)

The results of the analysis of the passive tissue moment showed a significant effect of POSTURE ($p < 0.0001$), HEIGHT ($p < 0.0001$) and their interaction ($p = 0.0255$) (see Figure 4.3) (simple effects analysis confirmed that both main effects were significant). Percentage of flexion of lumbar spine measured by two motion sensors, one at the L1 level and the other at

the S1 level, also showed that the leaning, 55 cm condition stands comparison with the no leaning, 85 cm condition and the no leaning, 70 cm condition (see Table 4.2).

Table 4.2 Percentage of the flexion range of motion of the lumbar spine (standard errors in parentheses.)

HEIGHT	POSTURE	
	Leaning	No leaning
55 cm	60.55% (3.62)	76.67% (3.73)
70 cm	41.07% (3.17)	68.94% (3.76)
85 cm	25.10% (2.58)	53.81% (2.60)

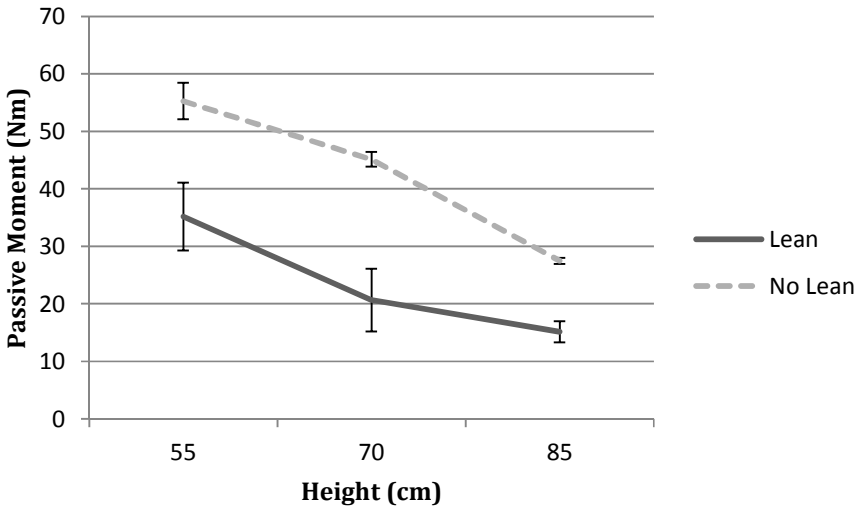

Figure 4.3 Interaction of POSTURE and HEIGHT on passive tissue moment. (Error bars show standard error.)

Regarding lifting kinematics, the result of ANOVA for angular acceleration in sagittal plane during a concentric lifting motion showed significant effects of POSTURE ($p < 0.0001$), HEIGHT ($p < 0.0001$) and its interaction ($p < 0.0001$) (see Figure 4.4). Simple effects, however,

revealed that there is no difference between leaning and no leaning conditions at the height of 55 cm ($p < 0.4319$) but confirmed HEIGHT as a significant main effect ($p<0.0001$).

In regards to the ground reaction force, the results of ANOVA for peak ground reaction force in A-P axis showed significant effects of POSUTRE ($p < 0.0001$), HEIGHT ($p = 0.0004$) and its interaction ($p = 0.0002$) (see Figure 4.5). Simple effects analysis revealed that HEIGHT was not significant in the no lean condition, but confirmed POSTURE as a significant main effect.

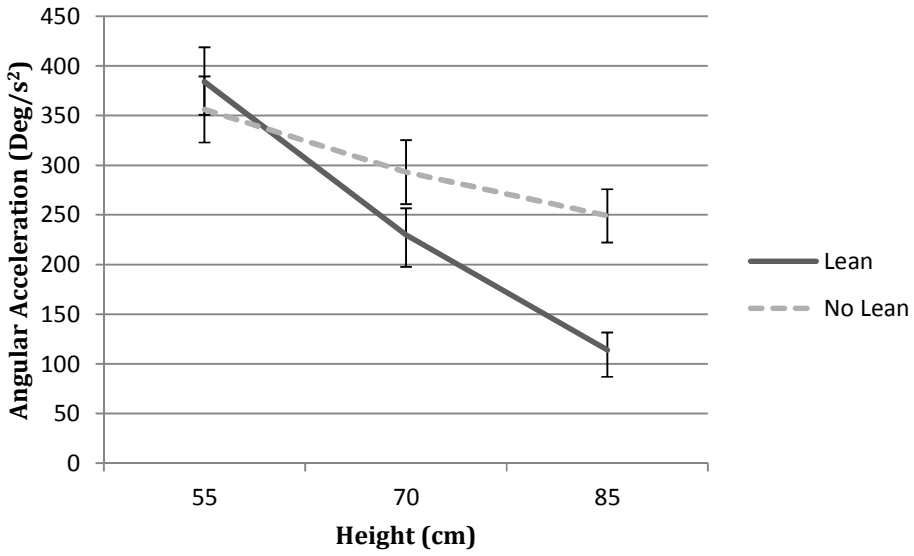

Figure 4.4 Interaction of POSTURE and HEIGHT on peak sagittal plane angular acceleration. (Error bars show standard error.)

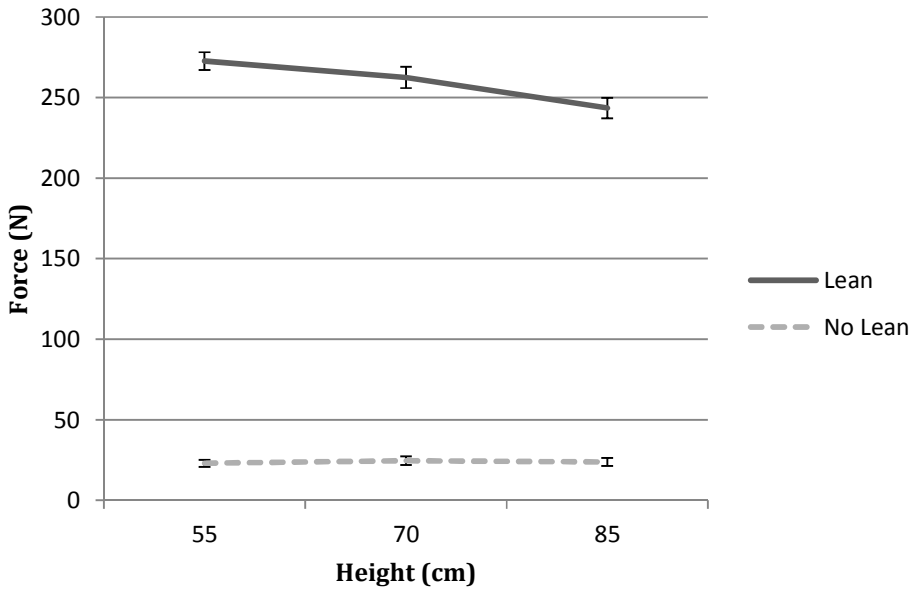

Figure 4.5 Interaction of POSTURE and HEIGHT on peak anterior ground reaction force. (Error bars show standard error.)

4.5. Discussion

Understanding the impact of a leaning posture on low back biomechanics and injury risk can provide valuable insight into possible ergonomic interventions for lifting in many scenarios. Quantifying the trunk kinematics through motion analysis, spine loading through electromyography, and slip risk through ground reaction force assessment can provide the type of quantitative data that will indicate whether this would be effective intervention in this particular work environment.

Normalized EMG results showed that the no leaning condition requires significantly greater trunk muscle activities (both agonist and antagonist) than did the leaning condition. The first thing that one notes in evaluating the postures assumed during these static contractions is that the leaning posture allows the pelvis to move anteriorly (see Figure 4.1), thereby moving the fulcrum of the biomechanical system closer to the load and reducing the moment generated by

the external load. This observation is consistent with the results of Kingma and van Dieën (2004) which showed a reduction in the distance between L5/S1 and external loads by supporting the upper body with the free hand during lifting. The second aspect of the results that was not as clear a priori, was that the leaning posture reduced antagonist muscle activity as well. This can be explained by noting that the leaning posture reduced the linear distance between the center of mass of the torso and lowest point of joint freedom and thereby increased the stability of the system (especially lower extremity) over that which would be seen in the free standing (i.e., no leaning) case and a reduced need for significant antagonist muscle activity. Third, while not part of the active trunk extension mechanism, the moment generated by the passive tissues of the low back were greater under the no leaning condition. The participants were able to keep a more upright trunk posture during the leaning condition, instead of the hyper trunk flexion observed during the no leaning condition for reaching an object (Figure 4.1). Consequently, the lumbar flexion angle during the leaning condition was significantly smaller than that observed in the no leaning condition resulting in a lower passive tissue moment. With regard to low back loading, it is clear that the leaning posture is superior. Finally, the leaning condition had slower trunk acceleration, so the leaning condition may have smaller spinal forces and moments as compared to the no leaning condition.

Less clear are the impacts of the leaning posture on lower extremity biomechanics and the resulting slip potential from this technique. The EMG results of gastrocnemius showed a significant (~15%) reduction in the necessary plantar flexion moment during the leaning condition as compared to the no leaning condition, indicating a positive effect of leaning. However, the nature of the leaning posture generated significantly higher anteriorly-directed ground reactions forces than the no leaning condition. The nature of the leaning posture

required that the participants push against the barrier with the thighs. With this pushing force comes an equal and opposite ground reaction force that is monotonically related to slip potential. In the current study this anteriorly-directed ground reaction force was shown to vary significantly as a function of load height during the leaning condition with the lower load heights generating the greater anterior shear force. While our laboratory simulation of the process of lifting the load from three different heights had high fidelity in some characteristics, the realistic working conditions (wet surface, oil and other particles) will reduce this coefficient of friction and may alter the strategies employed by workers performing this constrained lifting task.

The results showed that the lower extremity kinematic constraint during trunk flexion and extension provides some biomechanical benefits over the free lifting strategy by (1) reducing moment arm between pelvis and the external load, (2) increasing trunk-system stability and (3) reducing trunk acceleration. Meanwhile, this posture generated greater ground forces in posterior direction (anteriorly directed ground reaction force), so there is potential, under certain environmental conditions, for the leaning posture to increase slip potential.

This pilot study showed importance of the lower extremity kinematics on lifting biomechanics and suggested the needs to further investigate its biomechanical roles in various lifting conditions.

Chapter 5 – PILOT WORK

5.1. Overview of the chapter

The purpose of this study was to achieve a better understanding of the muscle activation patterns of the trunk and low extremity muscles (system-level perspective) during trunk flexion and extension under conditions involving prolonged stooping and muscle fatigue. This chapter presented preliminary experiment methods and results with three subjects which were performed to 1) demonstrate the ability to reproduce results of the previous literature, 2) test new hypotheses suggested in this dissertation, and 3) refine the experimental design and procedures of this study.

Subjects visited the lab three times for three separate experiments (three protocols) on different days with an interval of at least one week. Protocol A was designed to generate laxity in viscoelastic tissues through prolonged stooping; Protocol B was designed to cause muscle fatigue in low back muscles; and Protocol C was designed to have a combined effect of both passive tissue stretching and active tissue fatigue (see Figure 5.1). Each protocol lasted 10 min. After the 10 minute protocol, the subject had a 40 min recovery period. A variety of physiological/biomechanical data were recorded before, during and immediately following each protocol and during the recovery sessions to reveal modulation in biomechanical responses. In particular, this chapter explored the alteration in full flexion angle, EMG-off angle and intensity of muscle activation (NEMG) while performing the three protocols. The goal was to verify that the proposed method including the data collection and the data analysis procedures could generate reliable results in this *in-vivo* study.

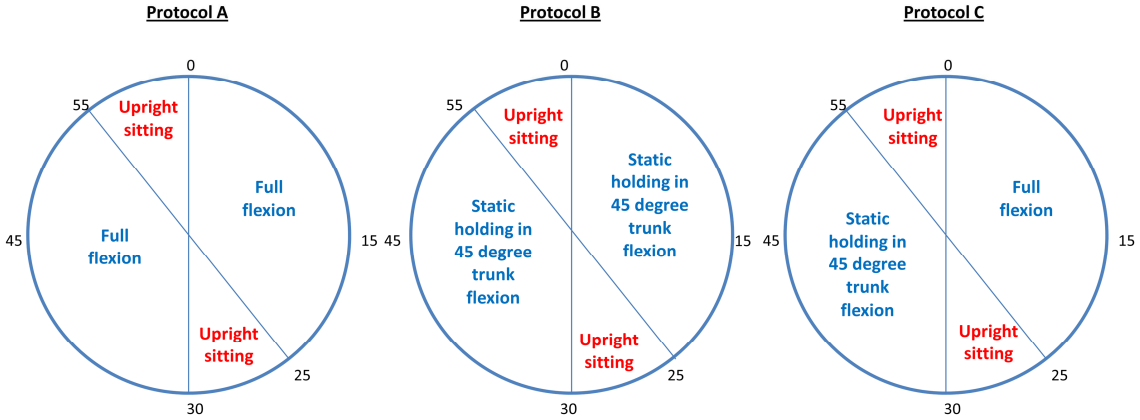

Figure 5.1 Diagram of experimental protocols for a minute (replicated for 10 min)

5.2. Methods

5.2.1. Participants

Three male participants were recruited from the university undergraduate and graduate student population of Iowa State University. They did not report any chronic problems or current pain in the low back or lower extremities. Each participant provided written informed consent prior to participation. The average and standard deviation of age, stature and whole body mass of participants were 28.0 yr (5.6), 176.3 cm (2.1), and 71.0 kg (3.6), respectively.

5.2.2. Experimental equipment

A lumbar dynamometer (Marras and Mirka, 1989) was used to provide the static resistance (both trunk flexion and extension) during maximum voluntary contractions (MVC) and submaximal contractions and a waist harness prevented falling over during full flexion exertions (using waist strap) (see Figure 5.2). During the experiment, surface electromyography was used

to capture the activities of the fourteen sampled muscles including right and left multifidus, iliocostalis, rectus abdominis, external oblique, gluteus maximus, biceps femoris and rectus femoris (Model DE-2.1, Bagnoli™, Delsys, Boston, MA) (data collected at 1024 Hz) (see Figure 5.2). Also, a magnetic-based motion analysis system was used to capture the instantaneous lumbar flexion angle, thoracic flexion angle, trunk flexion angle and hip flexion angle (The MotionMonitor™, Innovative Sports Training, Chicago, IL) (data collected at 102.4Hz) (see Figure 5.3). An electrical metronome was used to help participants control the speed of the trunk flexion and extension motions (Weird Metronome 1.4, Jone/Stone Production).

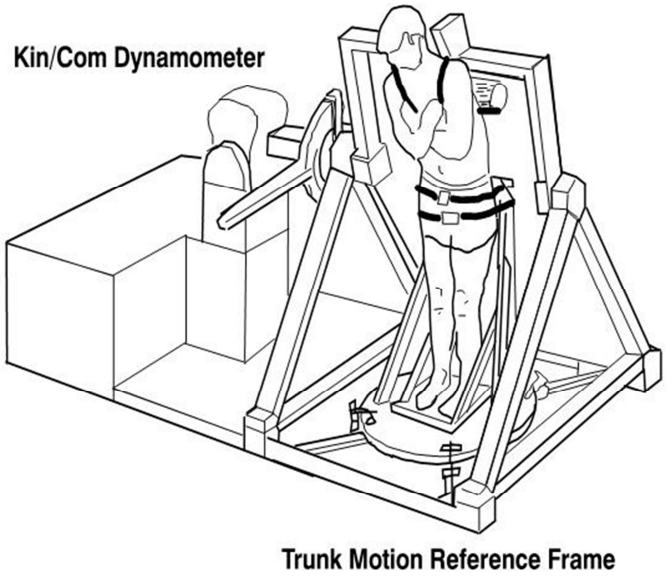

Figure 5.2 Drawing of the lumbar dynamometer

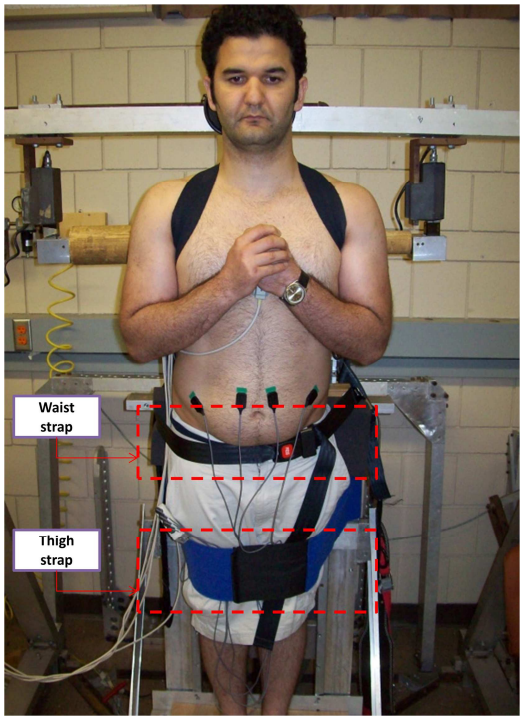

Figure 5.3 Lower extremity constraint in the lumbar dynamometer

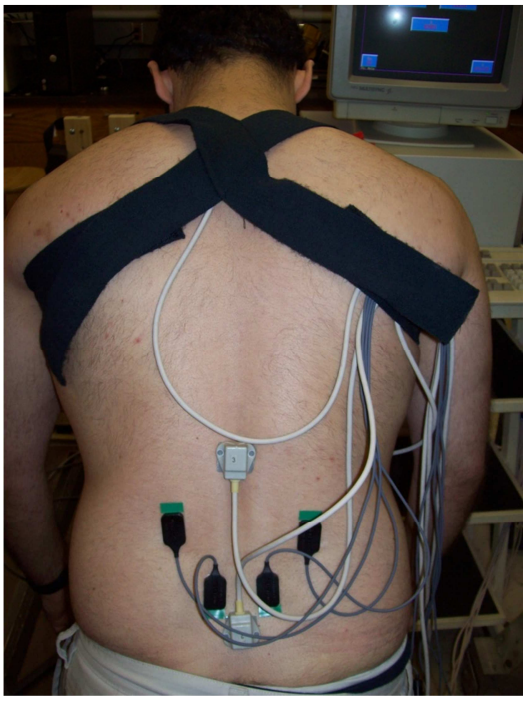

Figure 5.4 Electrodes and motion sensors on low back

5.2.3. Design of experiment

There were three within-subject independent variables in this study: 3 levels of PROTOCOL (A, B and C described graphically in Figure 5.1), eight levels of TIME (0 (initial), 1 (after protocol), 2 (5 min resting), 3 (10 min resting), 4 (15 min resting), 5 (20 min resting), 6 (30 min resting) and 7 (40 min resting), Figure 5.5) and two levels of POSTURE (free stooping and restricted stooping, Figure 5.3).

Dependent variables were the peak lumbar flexion angle, peak thoracic flexion angle, peak hip flexion angle, peak trunk flexion angle, average normalized EMG (NEMG) for agonist (average of bilateral multifidus and iliocostalis), antagonist (average of bilateral rectus abdominis and external oblique), synergist (average of bilateral gluteus maximus and biceps femoris) and average of bilateral rectus femoris during isometric exertions and isokinetic trunk flexion-extension, EMG-off angle (as defined in Chapter 2.5.1) during isokinetic trunk flexion-extension, and median power frequency (more detail in Chapter 5.2.4. and Figure 5.8) (see Table 5.1). The peak lumbar flexion angle was used to document the degree of passive tissue elongation, and the downward shift of the median frequency of the EMG data showed the level of muscle fatigue. The NEMG in isometric and isokinetic trials revealed the muscle activation pattern among agonist, antagonist and synergist muscles. All dependent measures were recorded before and after the protocol and during the recovery session. In addition, the maximum static flexion angle measured during the 10 min protocol (except muscle fatigue protocol) was used to show the trend of viscoelastic tissue deformation in each protocol.

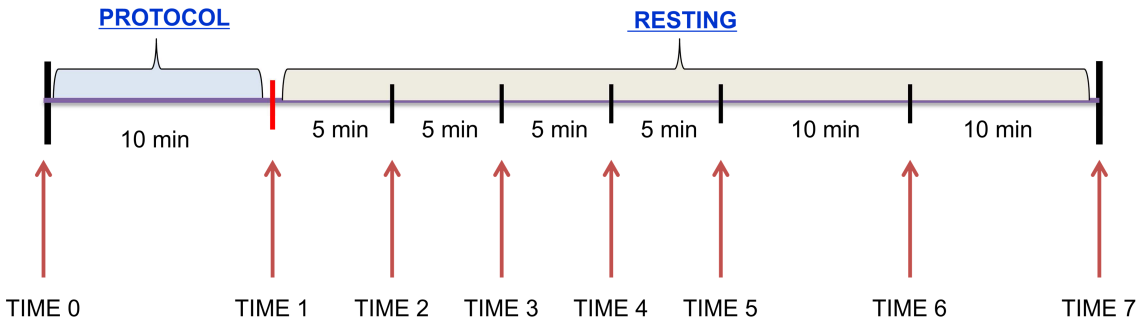

Figure 5.5 Schedule of experimental trial events

Table 5.1 A summary of timing of collection of dependent variables

	Before protocol			During protocol (10 minutes)	After protocol			Recovery session (40 min)		
	Isometric extension (35% MVC) (3 rep.)	Free flexion and extension (2 rep.)	Restricted flexion and extension (2 rep.)		Isometric extension (35% MVC) (3 rep.)	Free flexion and extension (2 rep.)	Restricted flexion and extension (2 rep.)	Isometric extension (35% MVC) (3 rep.)	Free flexion and extension (2 rep.)	Restricted flexion and extension (2 rep.)
Peak lumbar flexion angle		*	*	*		*	*		*	*
NEMG (Isometric)	*				*			*		
EMG-off angle		*	*			*	*		*	*
NEMG (Isokinetic)		*	*			*	*		*	*
Median frequency of EMG	*				*			*		

5.2.4. Experimental procedures

Subjects visited the lab three times for three separate experiments (three protocols) on different days with an interval of at least one week. Upon arrival, the experiment was described and the subjects were asked to sign an informed consent form. Participants' anthropometric data were then recorded (first visit only). A brief (5 minute) warm up routine was provided to let subjects stretch and warm up the muscles of the low back and lower extremities.

The subjects were then fitted with a set of sensors designed to capture muscle activation levels (electromyography: EMG) and 3-dimensional trunk positions (magnetic motion sensors). EMG sensors were bilaterally placed over the following muscles: (1) multifidus; (2) iliocostalis; (3) rectus abdominis; (4) external oblique; (5) gluteus maximus; (6) biceps femoris; and (7) rectus femoris. Also, motion sensors were place over the first sacral vertebrae (S1), the twelfth thoracic vertebrae (T12), the seventh cervical vertebrae (C7) and xiphoid process (bone below sternum).

Subjects then stepped into the lumbar dynamometer apparatus and perform a series of maximum voluntary contractions (MVC, two trunk extensions and two trunk flexions) in a 20 degree trunk flexion angle against the static resistance provided by the reference frame for measuring maximum capacity of the multifidus, iliocostalis, rectus abdominis and external oblique. Also, they were asked to perform a series of maximum isometric leg flexion and extension trials and knee flexion and extension trials for each leg for measuring maximum capacity of the gluteus maximus, biceps femoris and rectus femoris. During these trials, an experimenter manually provided static resistance on the ankle. A one minute rest period was provided between exertions. These data are collected for normalizing EMG activity of each muscle for each subject with respect to their maximum EMG activity.

Before starting the recording session, the participants were asked to stand upright and reach full flexion posture for calibration of the motion sensor data. These data were used to define full range of trunk flexion. The participants were then asked to perform two trunk extension exertions wherein they generated a trunk extension torque equal to 35% of their capacity at 20 degree trunk flexion from the standing posture for 6 seconds (Recording 1). A three minute break was provided before next recording. The participants were then asked to do a slow, controlled flexion and extension trunk motions consisting of two free stooping trials (no restriction) and two trials where the lower extremities were constrained (secure the thighs and pelvis with straps) (Figure 5.3) (Recording 2). These trials consisted of a 5 second flexion motion (to full flexion), 4 seconds of holding at full flexion and then 5 seconds to extend back to upright posture in time with a metronome sound (one beat per second). The full flexion posture was defined by a trigger signal manually controlled by one investigator during the trials. The point was used to find the FR angle.

The subjects then conducted one of the three experimental protocols that was assigned for that day: (1) alternately perform 25 sec full flexion in the seated posture (see Figure 5.6) and 5 sec upright sitting (see Figure 5.7) for 10 min (Protocol A); (2) alternately perform 25 sec static holding (see Figure 5.8) at 45 degree trunk flexion (no external load, just holding weight of torso) and 5 sec upright sitting for 10 minutes under the seated posture (Protocol B); and (3) consecutively perform 25 sec full flexion, 5 sec upright sitting, 25 sec static holding at 45 degree trunk flexion and 5 sec upright sitting for 10 minutes under the seated posture (Protocol C) (see Figure 5.1). Protocol A is designed to cause passive tissue elongation only, and Protocol B is aimed to generate low back muscle fatigue only. Protocol C is designed to bring force the combined effect of both passive tissue elongation and lumbar muscle fatigue. During the static

full flexion, there was restriction on the subject's pelvis. When the 10 minute protocol was completed, the Recording 2 session (described above) was conducted. Once completed, Recording 1 session (described above) was then performed. The 40 minute recovery process then began.

During this recovery period, the Recording 1 and Recording 2 sessions were conducted every five minutes until the 20 minute mark and then were performed at 30 minutes and finally at 40 minutes. After the final recording session, electrodes and magnetic sensors were removed and the subject was free to leave.

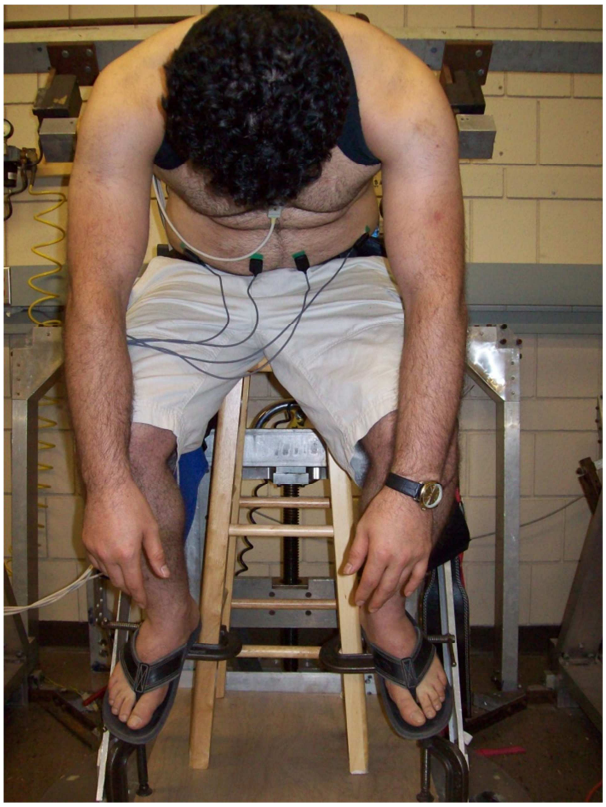

Figure 5.6 Full flexion in the seated posture

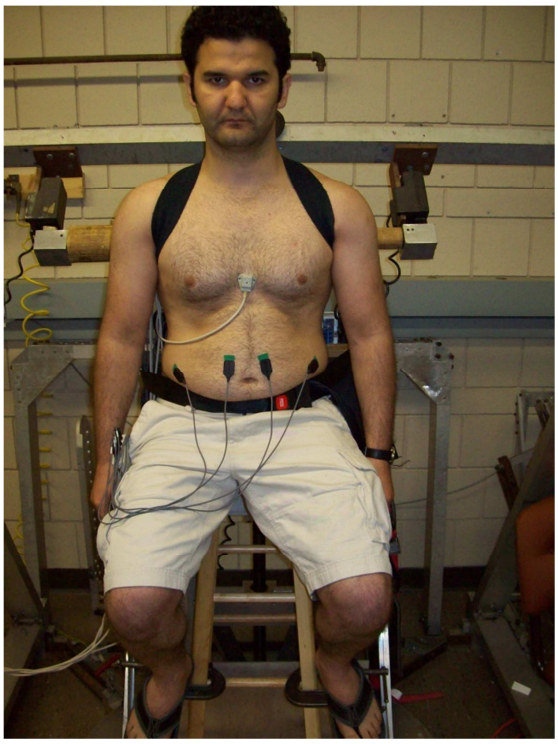

Figure 5.7 Upright sitting

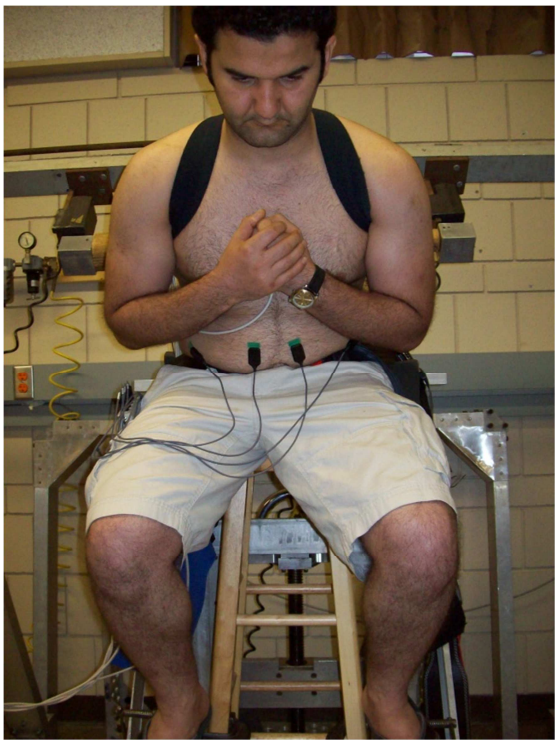

Figure 5.8 Static holding at 45 degree trunk flexion

5.2.5. Data analysis

Kinematic data

The sagittal plane angles (i.e., pitch angle) measured by four magnetic sensors placed on S1 (represent pelvic rotation), T12 (represent lumbar rotation), C7 (represent overall spine rotation) and xiphoid process (represent trunk rotation) were used to calculate the thoracic flexion angle, the lumbar flexion angle, the trunk flexion angle and the hip flexion (pelvic rotation) angle. The thoracic flexion angle was captured by the difference of the pitch angles between the sensor on xiphoid process and the S1 sensor, representing the gross movement of the vertebrae and the pelvic movements (Equation 1) (see Figure 5.9) (Rogers and Granata, 2006). The lumbar flexion angle (i.e., lumbar curvature) was captured by the difference of the pitch angles between the T12 sensor and the S1 sensor, representing total movement of the five lumbar spine segments (Equation 2) (see Figure 5.9). The lumbar flexion angle in full flexion posture was used to confirm the passive tissue elongation in low back. The percentage of range of flexion was calculated using the lumbar flexion angle during flexion-extension. Two calibration data included sagittal angles of the sensors in upright standing measured before each protocol and sagittal angles of the sensors in full flexion measured after the muscle fatigue protocol (Equation 3) (Dolan et al., 1994). The full flexion data after muscle fatigue protocol were employed to provide fair condition to calculate the percentage of flexion in all three protocols because less flexion was expected in this condition.

$$\text{Thoracic flexion angle (TF in deg)} = \text{Sagittal Angle}_{(\text{Xiphoid})} - \text{Sagittal Angle}_{(\text{S1})} \quad (1)$$

$$\text{Lumbar flexion angle (LF in deg)} = \text{Sagittal Angle}_{(\text{T12})} - \text{Sagittal Angle}_{(\text{S1})} \quad (2)$$

$$\text{Percentage Flexion (in \%)} = \frac{[\text{LF} - \text{LF}_{\text{standing}}]}{[\text{LF}_{\text{fullflexion}} - \text{LF}_{\text{standing}}]} \times 100 \quad (3)$$

The trunk flexion angle was captured by the pitch angle (i.e., sagittal angle) of the sensor on C7 which is normalized to the pitch angle when standing upright (see Figure 5.9). The upright standing posture captured before experimental trials was used to establish the participant's upright standing posture. The hip flexion angle (i.e., pelvic rotation angle) was captured by the sagittal rotation angle (i.e., pitch angle) of the S1 sensor which is normalized to the pitch angle when standing upright (see Figure 5.9). The sacroiliac (SI) joint is known to have a very stable connection between sacrum and ilium (Pool-Goudzwaard et al., 1998; Schuenke et al., 2006). Also, laxity in a ligament can be restored by other ligaments around the SI joints (see Chapter 2.4.4 and Figure 2.13). So, the deformation in SI joints after the protocol may be minimal, and the sensor on S1 can provide the pelvic rotation angle.

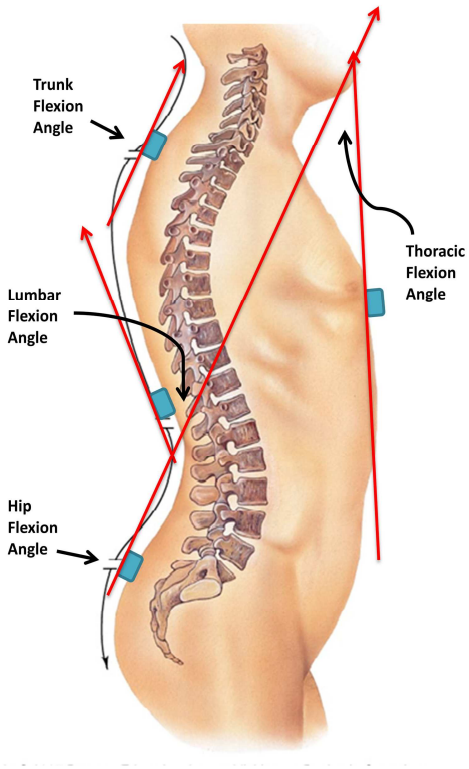

Figure 5.9 Definition of the kinematic variables - lumbar flexion angle, thoracic flexion angle, trunk flexion angle and hip flexion angle

Initiation angle of FR

The FR initiation angle were calculated for right and left multifidus and iliocostalis (see Figure 5.10). First, the unprocessed EMG data collected during Recording 2 session were filtered (high-pass 10 Hz, low-pass 512 Hz and notch filtered at 60 Hz and 102.4 Hz and their aliases). Second, the filtered signals were full-wave rectified and smoothed by averaging to the 1/4 second of moving window (256 data points) for removing the bumpy nature of the EMG signal. For example, the new first data were the average from 1st to 256th data, and the second data were the average from 2nd to 257th data. Finally, a method finding the FR period were applied on the data.

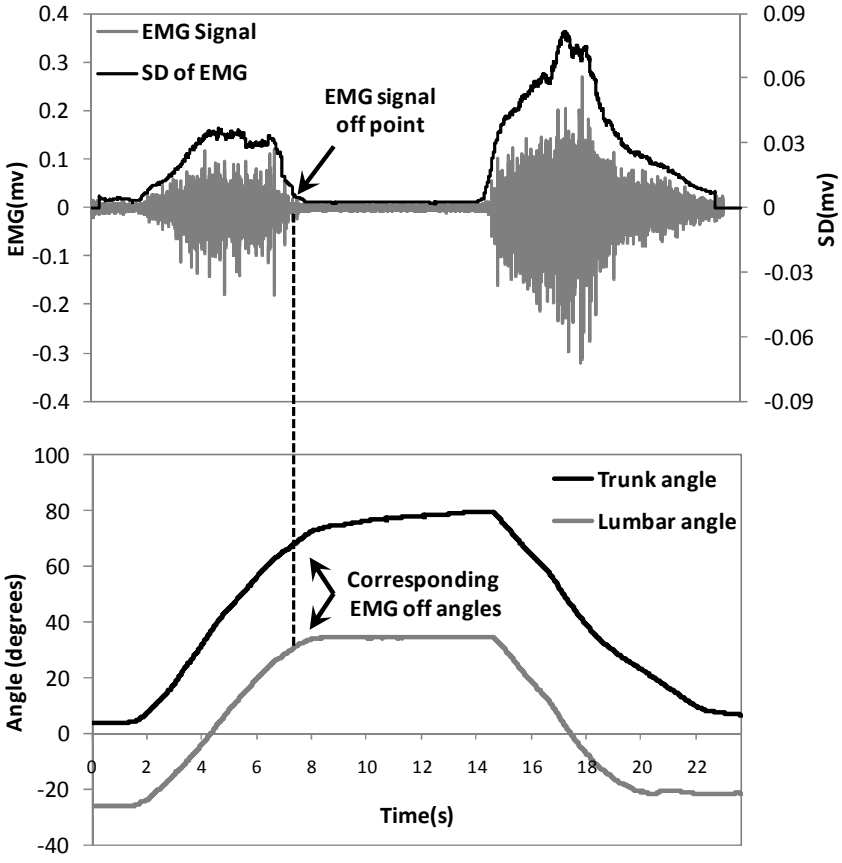

Figure 5.10 The EMG-off angle during trunk flexion-extension

To find the FR angles, standard deviation (SD) profiles of the EMG data for each muscle were developed by calculating the standard deviation in a moving data window of 512 data points (see Figure 5.10). This moving SD value was then compared with the SD calculated in the steady state, full-flexion posture (FSD). A total of 1024 EMG data (1 sec window) were selected from the initial point of the trigger marked in full flexion to 1024th data to calculate the FSD. A computer algorithm then identified the EMG-off point (during flexion) for each muscle by comparing the moving window SD value with the FSD and identifying the first time that this value is less than 2 times of the FSD value. The lumbar flexion angle and thoracic flexion angle values corresponding with this instant in time were found (Figure 5.10). If there exist a sudden peak in EMG signal near the trigger mark, known as muscle spasm (Solomonow et al., 2003a; Shin et al, 2009), the trigger mark was moved manually where the steady signal is observed. The MATLAB (Version 7.7.0) was used to calculate the FR initiation angle and provide the summarized result after this processing.

Median frequency and NEMG during isometric exertions

The level of muscle fatigue was calculated by median power frequency of EMG using the data from 35% isometric extension exertions at 20° trunk flexion. First, the raw data were transferred into the frequency domain (by fast Fourier transformation: FFT) and filtered at 60 Hz and 102.4 Hz and their aliases using the notch filter. Second, the cumulative sum of the frequency domain was calculated, and the half of the area was determined. Finally, the frequency of the half area was found. The outcome was used to confirm that the Protocol B and C (muscle fatigue only and both passive tissue elongation and muscle fatigue) develop muscle fatigue after completing the protocols. Also, it was used to confirm a previous study

result revealing trunk muscle fatigue after static stooping for 6 min (Shin et al., 2009). The muscle fatigue level was compared within the protocol and between the protocols, and the result was used to discuss the changes in FRP and muscle activation patterns.

The data of 35% isometric extension exertions at 20° trunk flexion and MVC were also used to reveal the modulation in muscle activation pattern after the protocol and during the resting session. The raw data were filtered (high-pass 10 Hz, low-pass 512 Hz and notch filtered at 60 Hz and 102.4 Hz and their aliases) and full-wave rectified. The rectified data were averaged into 1/8 second windows for both 35% isometric exertion and MVC. For the MVC data, the maximum 1/8 second window was identified for each muscle group and was used as the denominator in order to normalize the EMG data. For the EMG data of the 35% isometric exertion, the filtered data were averaged over the static exertion time period (3 seconds) and used as the numerator in the normalization process.

NEMG during slow isokinetic trunk flexion-extension exertions

The data from slow dynamic (i.e., isokinetic) flexion-extension trials (Recording 2) were used to reveal the modulation in muscle activation patterns. The unprocessed EMG data were processed with the same method described in Chapter 5.6.2. The processed data were then divided into 40 sections (5 % interval for both flexion and extension) according to the percentage of lumbar flexion (see Equations 2 and 3). Then the data were averaged over each section. These values were used as the numerator in the normalization process. The MVC results described in Chapter 5.6.3 were used as the denominator in order to normalize the EMG data. A total of 40 normalized EMG (NEMG) data showing muscle activity during flexion-extension were generated for each trial and muscle. Finally, the NEMG of each muscle in each

section was averaged as agonist including right and left multifidus and iliocostalis, antagonist including right and left rectus abdominis and external oblique and synergist including right and left gluteus maximus and biceps femoris. In a preliminary test, a total of five ranges during trunk flexion-extension were investigated as follows for selecting a sensitive range to reveal difference between postures: (1) from 95% flexion to 5% extension for selecting a sensitive range of motion; (2) from 80% flexion to 20% extension; (3) from 80% flexion to full flexion (100%); (4) from full flexion to 20% extension; and (5) from 30% flexion to 70% extension. In line with the expectation, 'from 80% flexion to 20% extension' was most sensitive to reveal the hypotheses. Previous studies also supported this result in that the lumbopelvic rhythm is accomplished by the predominant lumbar flexion before the initiation of flexion relaxation (FR) in low back muscles and pelvic rotation after the initiation of FR in low back muscles (Paquet et al., 1994; Sihvonen, 1997; Sarti et al., 2001). The opposite occurs during extension motion. So, it could be reasonable to believe that the most significant action and role of the pelvis in lumbopelvic rhythm occurs within 80% flexion and 20% extension phase. Consequently, 'from 80% flexion to 20% extension' was used to test the hypotheses in this pilot work.

5.3. Experimental hypotheses

Each of the experimental tasks was designed specifically to explore one or more hypotheses related to the effects of protocol types (PROTOCOL), flexion-extension posture (POSTURE) and protocol and resting effects (TIME) on peak lumbar flexion angle, peak thoracic flexion angle, peak trunk flexion angle, peak hip flexion angle, EMG-off angle, NEMG during isometric exertion and isokinetic trunk flexion-extension. The primary focus of current study was to test the system-level responses in three different protocols and reveal the effects of

the muscles fatigue protocol (B) and the combined protocol (C) on the peak lumbar flexion angle and EMG-off angle. In addition, the recovery phase of each protocol was investigated to reveal the characteristics of the phase such as required time to return the initial condition.

Hypothesis 1

Aim: Investigate the main effect of the Protocol A (TIME: 0, 1) on the peak (or full) lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point).

Hypothesis: It was hypothesized that after passive tissue elongation protocol the peak lumbar flexion angle and the occurrence points of FR will be deeper (increase) than the initial condition.

H_0: Dependent variables (the peak lumbar flexion angle and the occurrence points of FR) will be equal for initial condition and after protocol condition.

Contrast: TIME (0, Protocol A) vs. TIME (1, Protocol A)

Table 5.2 Summary of Hypothesis 1

Hypothesis	Independent Variable	Dependent Variables	Contrast
H1	TIME	1. Peak lumbar flexion angle 2. Lumbar angle at EMG-off for multifidus 3. Lumbar angle at EMG-off for iliocostalis	DATA USED: Protocol A TIME (0) vs. TIME (1)

Hypothesis 2

Aim: Investigate the main effect of the Protocol B (TIME: 0, 1) on the peak (or full) lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point).

Hypothesis: It was hypothesized that after muscle fatigue protocol the peak lumbar flexion angle and the occurrence points of FR will be smaller (decrease) than the initial condition.

H_0: Dependent variables (the peak lumbar flexion angle and the occurrence points of FR) will be equal for initial condition and after protocol condition.

Contrast: TIME (0, Protocol B) vs. TIME (1, Protocol B)

Table 5.3 Summary of Hypothesis 2

Hypothesis	Independent Variable	Dependent Variables	Contrast
H2	TIME	1. Peak lumbar flexion angle 2. Lumbar angle at EMG-off for multifidus 3. Lumbar angle at EMG-off for iliocostalis	DATA USED: Protocol B TIME (0) vs. TIME (1)

Hypothesis 3

Aim: Investigate the main effect of the Protocol C (TIME: 0, 1) on the peak (or full) lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point).

Hypothesis: It was hypothesized that after the protocol the peak lumbar flexion angle and the occurrence points of FR will not change as compared to the initial condition.

H_0: Dependent variables (the peak lumbar flexion angle and the occurrence points of FR) will not be equal for initial condition and after protocol condition.

Contrast: TIME (0, Protocol C) vs. TIME (1, Protocol C)

Table 5.4 Summary of Hypothesis 3

Hypothesis	Independent Variable	Dependent Variables	Contrast
H3	TIME	1. Peak lumbar flexion angle 2. Lumbar angle at EMG-off for multifidus 3. Lumbar angle at EMG-off for iliocostalis	DATA USED: Protocol C TIME (0) vs. TIME (1)

Hypothesis 4

Aim: Investigate the effect of PROTOCOL and TIME on muscle activities of agonist, antagonist and synergist in 35% isometric exertions at 20° trunk flexion.

Hypothesis: (**A**) It was hypothesized that muscle activities of agonist (NEMG) will increase after all three protocols. The NEMG of agonist will increase more rapidly to maintain higher levels of stability in Protocol C than in Protocol A and B; (**B**) The NEMG of antagonist will show no significant change after protocols; and (**C**) The NEMG of synergist will increase after all three protocols. The NEMG of agonist will increase more rapidly to maintain higher levels of stability in Protocol C than in Protocol A and B.

H_0: Dependent variables will be equal between TIME × PROTOCOL conditions.

Contrast: Combination of PROTOCOL (Protocol A, B and C) × TIME (0, 1).

Table 5.5 Summary of Hypothesis 4

Hypothesis	Independent Variable	Dependent Variables	Contrast
H4	TIME × PROTOCOL	1. NEMG for agonist in 35% MVC 2. NEMG for antagonist in 35% MVC 3. NEMG for synergist in 35% MVC	3 × 2 interaction

Hypothesis 5

Aim: Investigate the main effect of POSTURE on the peak lumbar, thoracic, trunk and hip flexion angle and the occurrence points of FR (lumbar and thoracic flexion angles at the EMG-off point).

Hypothesis: It was hypothesized that the peak trunk and hip flexion angle in the free stooping will be significantly higher (more flexion) than the restricted stooping. However, the lumbar flexion angle and the occurrence points of FR in the restricted stooping will be significantly higher than the free stooping.

H_0: Dependent variables (peak lumbar, trunk and hip flexion angle and the occurrence points of FR) will be equal for the free stooping and the restricted stooping.

Contrast: POSTURE (free stooping) vs. POSTURE (restricted stooping)

Table 5.6 Summary of Hypothesis 5

Hypothesis	Independent Variable	Dependent Variables	Contrast
H5	POSTURE	1. Peak trunk flexion angle 2. Peak hip flexion angle 3. Peak lumbar flexion angle 4. Lumbar angle at EMG-off for multifidus 5. Lumbar angle at EMG-off for iliocostalis	DATA USED: TIME 0 POSTURE (free stooping) vs. POSTURE (restricted stooping)

Hypothesis 6

Aim: Investigate the main effect of POSTURE on the muscle activities between free and restricted postures in agonist, antagonist and synergist.

Hypothesis: It was hypothesized that the muscular activation (NEMG) of agonist during the restricted stooping posture will be higher than the free stooping posture, but the NEMG of synergist during the free stooping posture will be higher than the restricted stooping posture. No difference is expected in antagonist.

H_0: NEMGs for agonist and synergist in two stooping postures will be the same, but the NEMG for antagonist will have difference between the postures.

Contrast: POSTURE (free stooping) vs. POSTURE (restricted stooping)

Table 5.7 Summary of Hypothesis 6

Hypothesis	Independent Variable	Dependent Variables	Contrast
H6	POSTURE	1. NEMG for agonist in isokinetic trials 2. NEMG for antagonist in isokinetic trials 3. NEMG for synergist in isokinetic trials	DATA USED: TIME 0 POSTURE (free stooping) vs. POSTURE (restricted stooping)

Hypothesis 7

Aim: Investigate the interactive effect of POSTURE and TIME (0, 1) on muscle activities of agonist, antagonist and synergist during isokinetic trunk flexion-extension.

Hypothesis: (**A**) It was hypothesized that NEMG of agonist will increase after the protocols, but there will be no significant interaction between POSTURE and TIME; (**B**) the NEMG of synergist will have a significant interaction between POSTURE and TIME (no increase in restricted stooping and significant increase in free stooping); and (**C**) the NEMG of antagonist will have a significant interaction between POSTURE and TIME (no increase in free stooping and significant increase in restricted stooping).

H_0: NEMGs for agonist and synergist in two stooping postures will be the same, but the NEMG for antagonist will have difference between the postures.

Contrast: Combination of POSTURE (free and restricted) × TIME (0, 1)

Table 5.8 Summary of Hypothesis 7

Hypothesis	Independent Variable	Dependent Variables	Contrast
H7	POSTURE × TIME	1. NEMG for agonist in isokinetic trials 2. NEMG for antagonist in isokinetic trials 3. NEMG for synergist in isokinetic trials	DATA USED: TIME 0 and 1 across all protocols 2 × 2 interaction

5.4. Verification of the proposed methods

5.4.1. Hypothesis 1

The Hypothesis 1 aimed to test the effect of the passive tissue elongation in low back (Protocol A) (TIME: 0, 1) on the peak (or full) lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point). The result showed increases in the peak lumbar flexion angle and the occurrence points of FR after the Protocol A, bringing laxity in viscoelastic tissues in low back (Figure 5.11, 5.12 and 5.13). The peak lumbar flexion angle before the protocol (TIME 0) was observed as 38.3° and it showed consistent increase to 40.1° after the protocol (TIME 1), which was 4.7% increase. This amount of angle change was smaller than Shin and Mirka's (2007) observation (9.3% for a 10 minute full flexion) and McGill and Brown's (1992) observation (6.1% for a 20 minute full flexion) but greater than Solomonow et al. (2003a) observation (1.6% for a 10 minute full flexion). The EMG-off angle was also increased about 1.9° in multifidus (5.5%) and 1.3° in iliocostalis (3.8%). This amount of change was smaller than Solomonow et al. (2003a) observation (7.4%) and greater than Dickey et al. (2003) observation (2%). The smaller changes than some studies could result from the inclusion of female subjects in these studies and differences in measurement methods. Solomonow et al. (2003a) and Rogers and Granata (2006) reported significantly greater lumbar flexion in female subjects after the passive tissue elongation protocol as compared to the male counterparts. So, the inclusion of female subjects may increase the magnitude of significance. The measurement method was also somewhat variable between studies. For example, Dickey et al. (2003) placed the sensors on the sacrum and T6, and Solomonow et al. (2003a) put the circular markers on greater trochanter, iliac crest and rib cage for capturing the lumbar flexion. Consequently, the flexion angle measured in those studies were significantly higher than the current study,

suggesting greater magnitude of change: initial, peak lumbar flexion angle was 38.3° in current study, but they reported 60° (Dickey et al.) and 58.6° (Solomonow et al.). In addition, the experimental protocol employed in current study included 5 sec resting in every 25 sec. This periodical resting during 10 minute protocol could result in smaller change in peak lumbar flexion angle. In spite of this limitation, the result of this pilot work suggested that the experimental protocol and setup are capable of generating passive tissue elongation of low back and detecting creep responses such as EMG-off points.

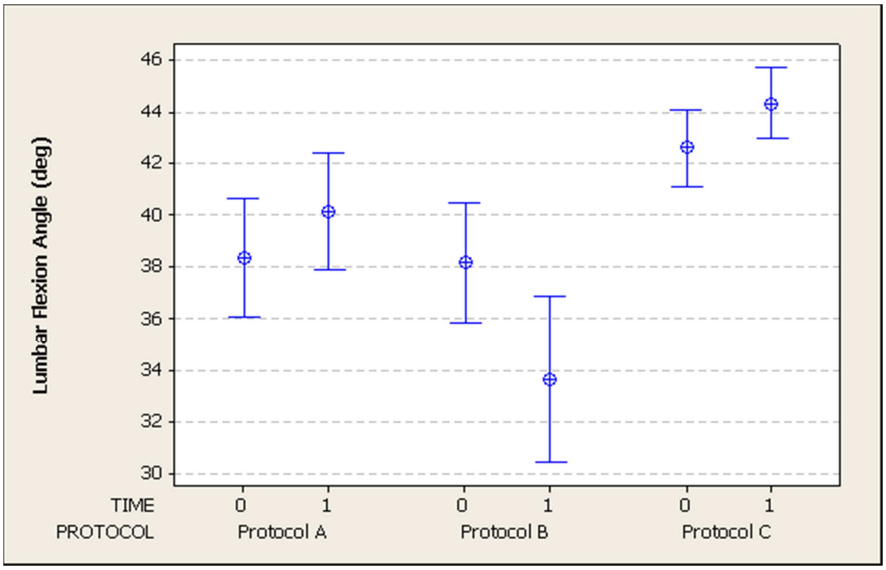

Figure 5.11 Lumbar flexion angle before and after three protocols (Error bars show 95% confidence interval.)

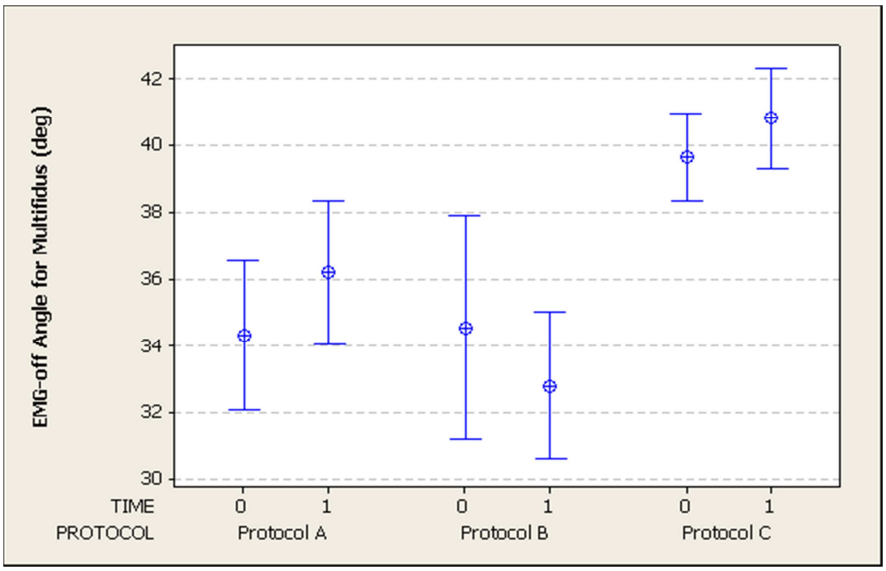

Figure 5.12 EMG-off point for multifidus before and after three protocols (Error bars show 95% confidence interval.)

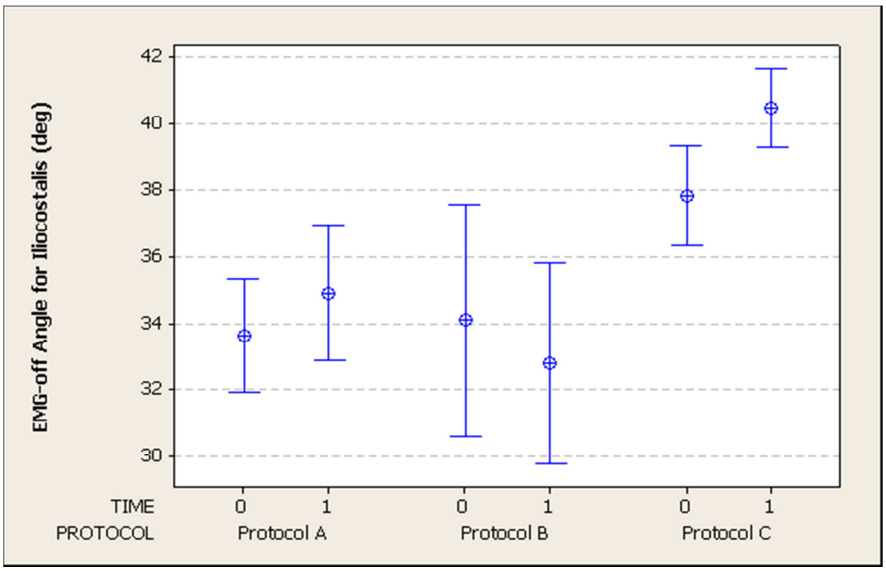

Figure 5.13 EMG-off point for iliocostalis before and after three protocols (Error bars show 95% confidence interval.)

5.4.2. Hypothesis 2

The goal of Hypothesis 2 was to investigate the effect of the muscle fatigue (Protocol B) (TIME: 0, 1) on the peak (or full) lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point). In line with the hypothesis, the peak lumbar flexion angle and the occurrence points of FR was decreased after 10 minutes protocol which is designed to generate low back muscle fatigue (Protocol B) (see Figure 5.11, 5.12 and 5.13). The low back muscle fatigue was confirmed by the median frequency shift from 110 Hz to 92 Hz in multifidus denoting presence of low back muscle fatigue, but there was almost no change in iliocostalis (from 88.7 to 87.5) (Figure 5.14 and 5.15). Presence of muscle fatigue in low back reduced the peak lumbar flexion angle about 4.5 degree suggesting less peak lumbar flexion. It could result from the decreased ability of the fatigued local muscles around the lumbar spine to keep the lumbar lordosis. There is no study investigating the changes in lumbar flexion angle in full flexion, but this result can be supported by Takihara et al. (2009) observing significantly decreased lumbar curvature (i.e., hypolordotic) after low back muscle fatigue in the upright standing, 30 degree trunk flexion and 40 degree trunk flexion postures.

The lumbar flexion angles at EMG-off point were also reduced about 4.6% (1.6°) in multifidus and 3.3% (1.2°) in iliocostalis supporting the result of Descarreaux et al. (2008) (Figure 5.12 and 5.13). This amount of angle change was smaller than Descarreaux et al. (2008) employed the overall trunk flexion angle to calculate the EMG-off points (5.7% decrease at L5 level (4.3°)), but it could result from methodological difference. They used the overall trunk flexion angle instead of the lumbar flexion angle, by placing light-emitting diodes on acromion, iliac crest and greater trochanter. As a result, the full flexion angle measured by total trunk flexion was around 90° which is over double what the current study measured in the peak

lumbar flexion angle (around 39°). The overall trunk flexion angle measured in this study using the sensors on C7 showed 10.5% decrease (from 77.7° to 69.5°) which greater than Descarreaux et al. (2008). However, the lumbar flexion angle was chosen because the lumbar flexion angle provides most stable results in that the most significant changes after protocols occurred in lumbar region.

In summary, the results of pilot work suggested that the experimental setup and protocol designed for testing Hypothesis 1 are capable of generating low back muscle fatigue and detecting alteration in low back curvature and EMG-off points.

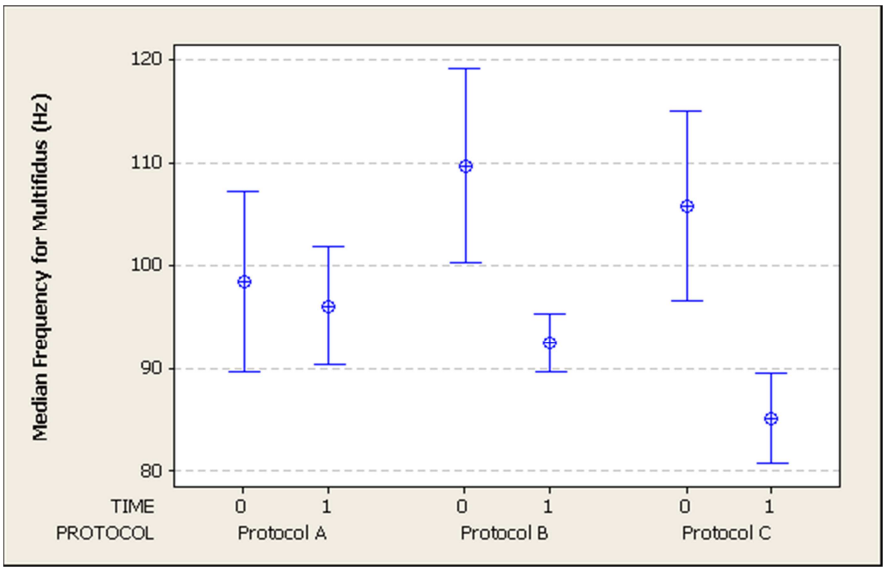

Figure 5.14 Median frequency of multifidus before and after three protocols (Error bars show 95% confidence interval.)

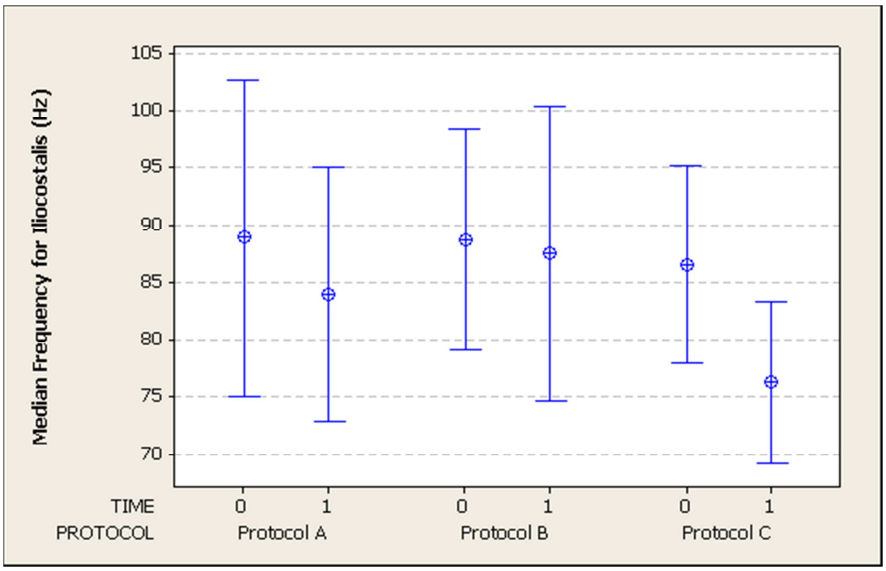

Figure 5.15 Median frequency of iliocostalis before and after three protocols (Error bars show 95% confidence interval.)

5.4.3. Hypothesis 3

The goal of Hypothesis 3 was to test the combined effect of passive tissue stretching and muscle fatigue in low back (Protocol C) (TIME: 0, 1) on the peak lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point). Considering the independent effect of low back muscles fatigue and elongation of the passive tissues in low back on lumbar flexion angle (less flexion in 'Fatigue' and more flexion in 'Passive' in Figure 5.11), no changes in lumbar flexion angle and EMG-off points were expected in the combined effect protocol in that individual effects of muscle fatigue and passive tissue stretching could counteract each other. However, the results showed increase in peak lumbar flexion angle after the protocol like the Protocol A (viscoelastic tissue elongation) but the magnitude of the change was smaller (2° vs. 1.7°) (Figure 5.11). This result suggested that the elongation or deformation

of passive tissues in low back have a dominant influence on the low back condition than the active low back muscle fatigue. Also, the smaller amount of change could be attributable to the counteraction of the low back muscle fatigue which is confirmed by shift in median power frequency in this protocol (from 105 Hz to 85 Hz in multifidus; from 86 Hz to 76 Hz in iliocostalis) (Figure 5.14 and 5.15). In line with the result of peak lumbar flexion angle, the EMG-off points were also increased (more flexion) after the protocol (Figure 5.12 and 5.13). In summary, even the results did not support our hypothesis it is still in line with our model representing the independent effect of passive tissue stretching and muscle fatigue on low back. Based on this, the experimental setup and the protocol used for testing Hypothesis 2 are competent in answering the questions of the effect of the combined protocol.

5.4.4. Hypothesis 4

The Hypothesis 4 aimed to investigate the interactive effect of PROTOCOL and TIME on muscle activities of agonist, antagonist and synergist during isometric exertions (35% MVC) at 20° trunk flexion. In line with the hypothesis, results showed increase in the normalized EMGs for agonist and synergist and almost no change in the NEMG for antagonist after all three protocols (TIME: 0 vs. 1) (see Figure 5.16, 5.17 and 5.18). Regarding agonist and synergist muscles, the magnitude of increase was higher in the passive tissue elongation protocol (Protocol A) (agonist: 2.5%; synergist: 2.1%) than the other two protocols right after the protocols (TIME: 1). In the muscle fatigue protocol (Protocol B), NEMGs were increased 1.4% in agonist and 1.9% in synergist right after the protocol. In the combined effect protocol (Protocol C), NEMGs were increased 1.8% in agonist and 1% in synergist right after the protocol. The highest increasing magnitude in the combined effect protocol in comparison of

TIME 0 with 1 was expected due to inability in both active and passive tissues, but the passive tissue elongation protocol showed the highest increase. The results may also suggest a more dominant effect of the laxity in lumbar passive tissues than the active muscle fatigue in line with the results of Hypothesis 2. In accordance with the hypothesis, the NEMG of antagonist was very weak (between 3 and 5% of the max) and the effect of the protocol was negligible (less than 1% changes).

The muscle activities during 35% isometric exertions at 20° trunk flexion throughout the experiment showed an effect of each protocol on the NEMG. Greater muscle activity after the protocols indicated that the muscles had to activate more to meet the 35% of their extension moment which is set before experiment. The increase of NEMG in agonist after passive tissue elongation protocol could be explained as the reduced ability of the low back passive tissues to keep the spinal stability because of the laxity in the ligaments. Previous studies observed similar results in low back muscles after 10 minutes prolonged stooping (Shin and Mirka, 2007) and 100 repeated spinal flexion (Dickey et al., 2003), but they had different experimental setting such as the weight holding task. Both of them observed deeper lumbar flexion after the passive tissue elongation protocol, and attributed the increased muscle activity to creep of passive tissues. The increase of NEMG after the muscle fatigue protocol may be explained by the decrease in force generation capacity of the fatigued muscles. The fatigued muscle can result in the significant increase in reflex amplitude (Herrmann et al., 2006) and hence increase the EMG activity. Similarly, it is reasonable to believe that the combined protocol could have the effects of both fatigued muscle and laxity in passive tissues on low back muscle activity during 35% MVC exertions.

The muscle activities in synergist also showed an effect of all three protocols on NEMG. Previous literature showed a direct and indirect connection between torso and lower extremity and suggested a possibility to consider a system level investigation (Snijders et al., 1993b; Vleeming et al., 1995). So, the results could be attributable to the synergistic activation of the 'super global muscles' including gluteus maximus and biceps femoris for assisting extension moment generation and spinal stability.

In summary, in spite of the weak effect in NEMG, the results suggested the procedures and setup for this validation work were capable of showing the effect of protocols on the muscle activities measured in 35% isometric exertion throughout the experiment.

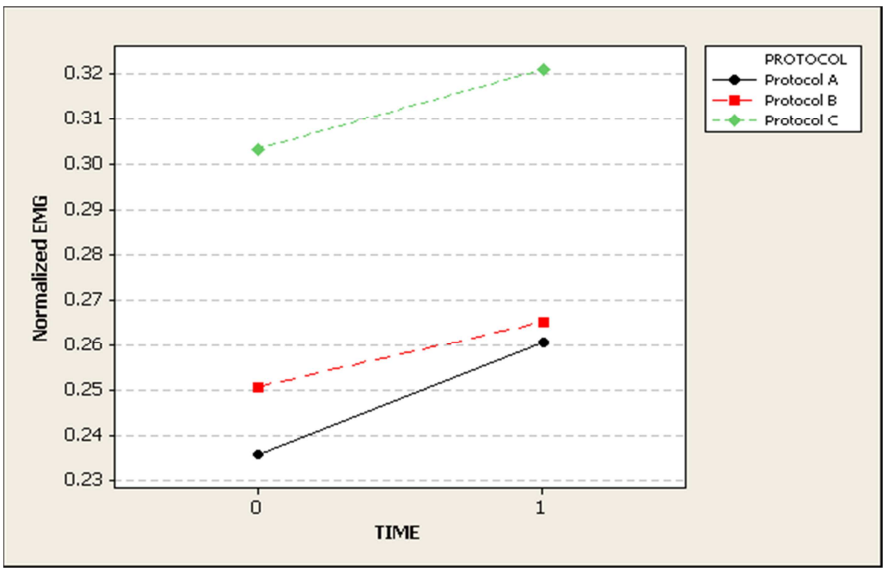

Figure 5.16 Interaction between PROTOCOL and TIME in NEMGs of agonist

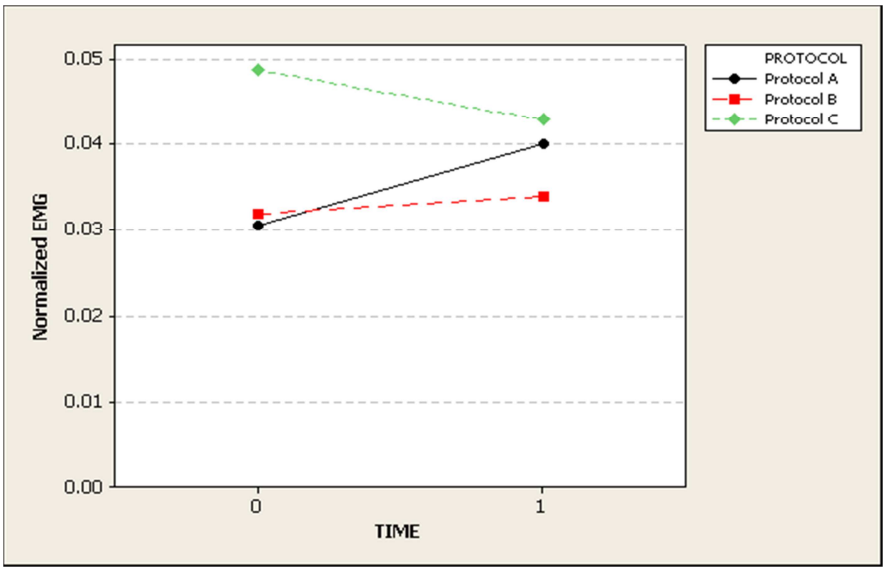

Figure 5.17 Interaction between PROTOCOL and TIME in NEMGs of antagonist

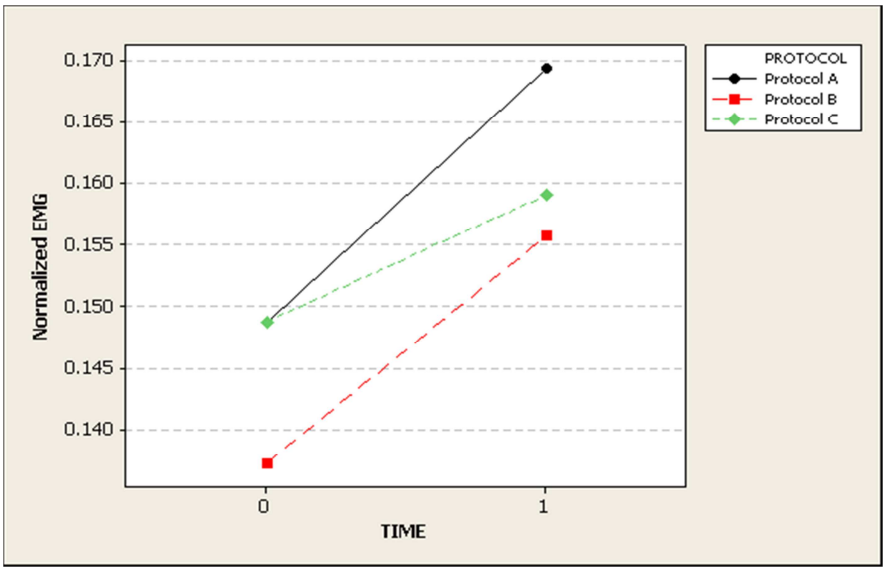

Figure 5.18 Interaction between PROTOCOL and TIME in NEMGs of synergist

5.4.5. Hypothesis 5

The goal of Hypothesis 5 was to investigate the main effect of POSTURE on the peak (or full) lumbar, thoracic, trunk and hip flexion angle and the occurrence points of FR (lumbar and thoracic flexion angles at the EMG-off point). The result showed significantly higher peak trunk and hip flexion angle in free stooping posture (Figure 5.19), but the difference between two postures was smaller in trunk flexion angle (33° in hip flexion angle and 26° in trunk flexion angle). It suggested that there was a significant difference between two postures in the flexion magnitude (i.e., curvature) of the spinal columns at full stooping.

The main source of smaller change in trunk flexion was explained by additional flexion of spinal columns during sagittal trunk movement in restricted stooping posture. The restricted posture was 2.4° deeper in the peak lumbar flexion angle and 6.3° deeper in the peak thoracic flexion angle than the free stooping posture (Figure 5.20) even the hip and trunk flexion angles were significantly smaller in the posture. In line with these results, the EMG-off points in multifidus and iliocostalis were also delayed in the restricted posture (3.6° (10.4%) increase in multifidus, and 3° (8.7%) increase in iliocostalis) (Figure 5.22). It could be attributable to the longer moment arm in the restricted posture, denoting bigger external moment generated by the mass of torso at full flexion posture, than the free stooping. The distance between the centre of mass (COM) of the torso and pelvis (e.g., a rigid body) increases until the trunk reaches horizontal line (i.e., max in 90°), and decreases again after passing the horizontal line. The pitch angle of the peak trunk flexion in sagittal plane in the restricted posture was 88° denoting the maximum point of the external moment (Figure 5.21 and 5.23). In free stooping posture, the trunk pitch angle was further increased (more deeper flexion) to 114° (24° more flexion from the horizontal line), so the magnitude of external moment was smaller than the restricted

posture. As already discussed in Chapter 1.5.1, the full flexion angle and the FRP significantly depend on the external torque in that the passive tissues have to offset the external moment at some point. Previous studies also showed deeper peak flexion and FRP angle of spinal column when increasing the external load (using hand-held load) (Kippers and Parker, 1984; Gupta, 2001; Dickey et al., 2003). Based on this, the results may suggest the role of hip flexion (i.e., lower extremity) in flexion-relaxation phenomenon which controls the EMG-off points and show that the measurement system is capable to exactly capture the lumbar, thoracic, trunk and hip flexion angle.

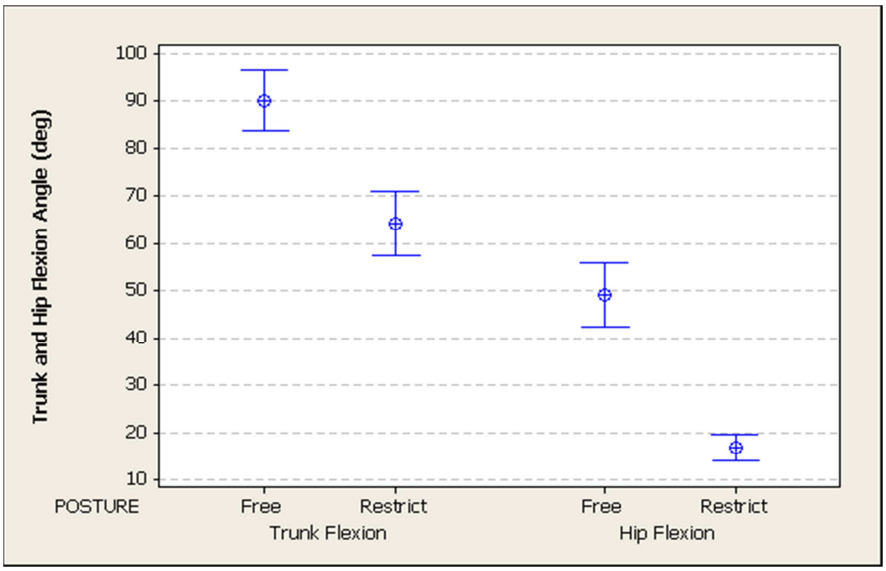

Figure 5.19 Effect of posture on trunk and hip flexion angles (Error bars show 95% confidence interval.)

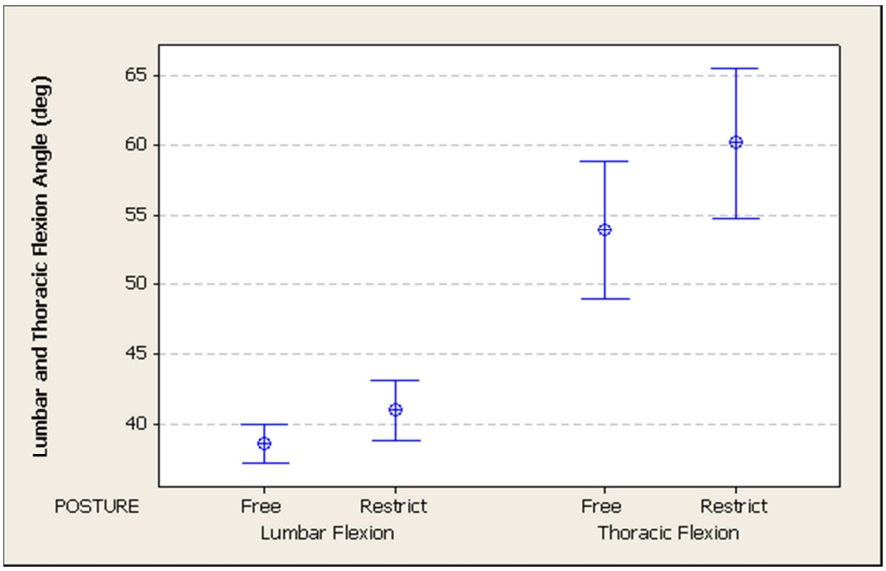

Figure 5.20 Effect of posture on lumbar and thoracic flexion angles (Error bars show 95% confidence interval.)

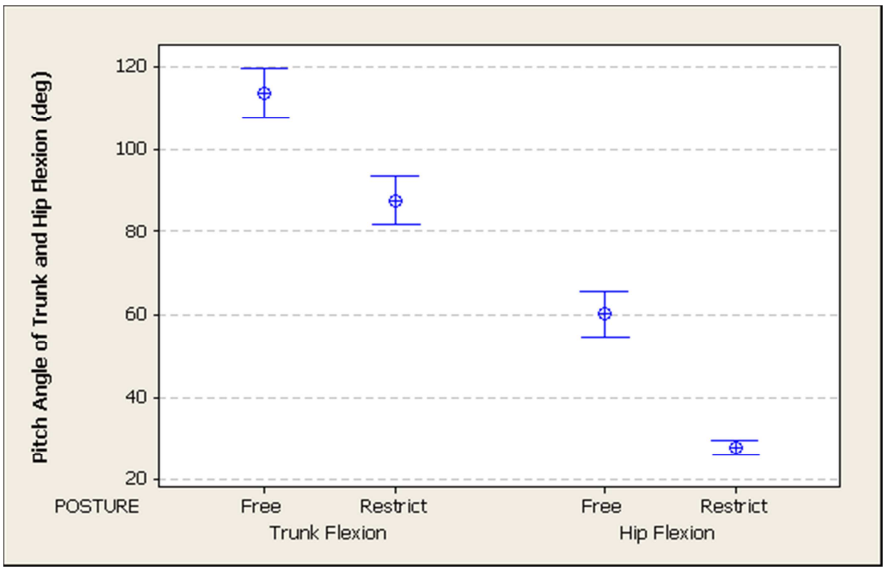

Figure 5.21 Effect of posture on the pitch angles of trunk and hip flexion(Error bars show 95% confidence interval.)

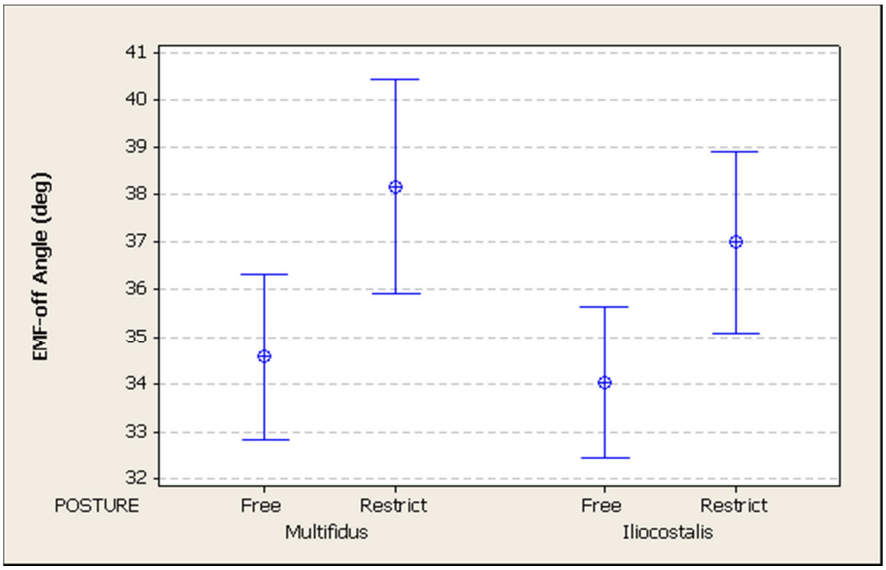

Figure 5.22 Effect of posture on EMG-off angles (Error bars show 95% confidence interval.)

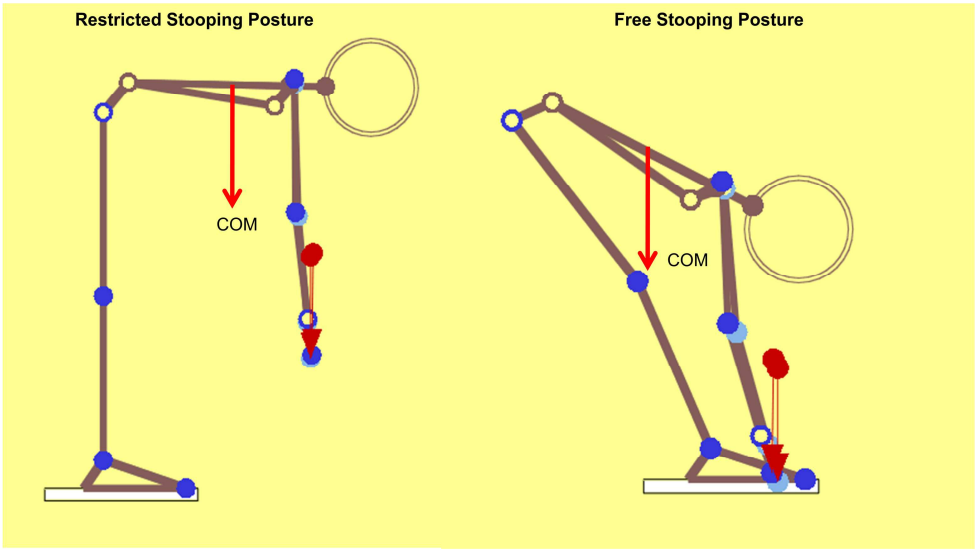

Figure 5.23 Comparison of the posture between free and restricted stooping (COM: Center of mass)

5.4.6. Hypothesis 6

The Hypothesis 6 aimed to test the main effect of POSTURE on the muscle activities during isokinetic trials in agonist, antagonist and synergist. The result may reveal a system-level response in trunk flexion-extension by showing different recruitment patterns in two postures. In line with our expectation, the restricted posture showed higher NEMG in agonist than the free posture, and the free posture showed higher NEMG in synergist than the restricted posture at the range of motion of 'from 80% flexion to 20% extension' (see Figure 5.24). It is plausible that in the restricted posture the synergist such as gluteus maximus and biceps femoris could not be activated to assist the trunk flexion-extension, so the agonist may be asked to generate the extension moment without help of the synergist by having greater muscular activation than the free stooping. In contrary, the free stooping had less activation level of the agonist in low back by recruiting the synergist in lower extremity to assist the trunk flexion-extension. The benefit of the synergist recruitment during the trunk flexion-extension can be summarized into three aspects: (1) providing stable basis (i.e., pelvic girdle) of the flexion-extension motion of the spinal column; (2) generating extension moment during trunk motion throughout the large attachment of synergist on pelvis; and (3) enhancing spinal stability throughout the lumbodorsal fascia. The results suggested that the trunk flexion-extension is accomplished by a system-level response including the activation of both trunk and lower extremity rather than the trunk muscle activity alone.

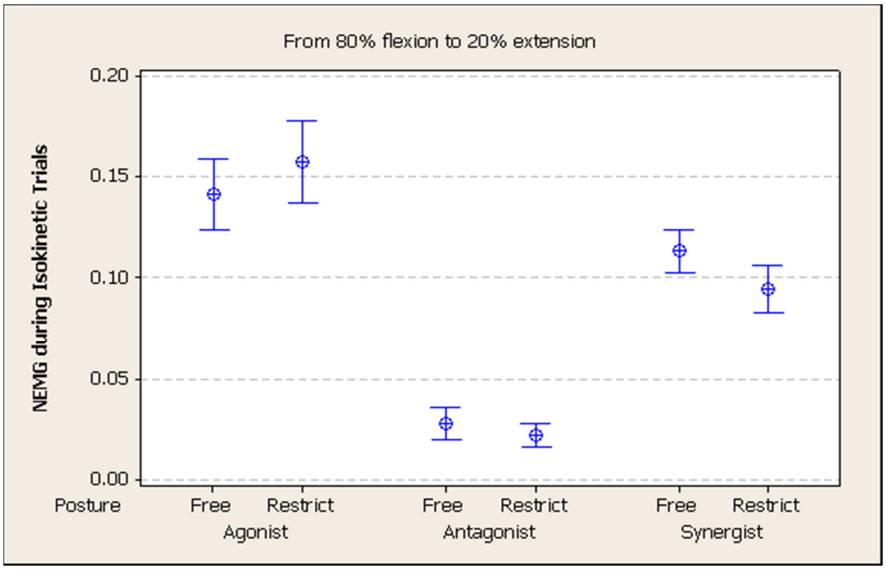

Figure 5.24 Effect of the posture on NEMGs from 80% flexion to 20% extension (Error bars show 95% confidence interval.)

5.4.7. Hypothesis 7

The Hypothesis 7 aimed to investigate the interactive effects of POSTURE and TIME (0, 1) on muscle activities of agonist, antagonist and synergist during isokinetic trunk flexion-extension. In accordance with the hypothesis, the synergist showed interactive effects between POSTURE and TIME during trunk flexion-extension, but the agonist did not have an interaction effect showing similar level of effect of the POSTURE in both TIME 0 and 1 (see Figure 5.25, 5.26 and 5.27). However, the muscle activation level of the antagonist was negligible (less than 3% of MVC), so it may be reasonable to regard that there is no significant effect.

Regarding the increased activation in synergist it is plausible that the free stooping posture utilized the synergist including gluteus maximus and biceps femoris to enhance the stability of trunk flexion-extension system after the protocol, bringing about spinal instability caused by the

inability of passive tissues or active tissues in low back. A ball over the bowl example in Figure 2.12 may explain the result in detail. The new concept in Figure 2.12 took into account the stability potentially provided by the lower extremities, especially pelvic stabilization for stable foundation. The low back condition after the protocol (TIME 1) could be regarded as an unstable ball (e.g., greater rolling) on the bowl. Consequently, the unstable ball on the bowl may increase the level of instability of the bowl which should be stabilized prior to stabilization of the ball, so the system may require more stable basis (i.e., bowl) to prevent system failure. Finally, it could result in greater activities of the active stabilizer such as lower extremity muscles.

On the other hand, the restricted stooping posture simply recruits more low back muscles (i.e, agonist) at the same level of increase with the free stooping than utilizing the synergist under the unstable condition (TIME 1). The restricted stooping posture may not need any additional action to enhance the stability of the basis, because a stable basis was already provided. This condition could be regarded as the old concept (firm basis) in Figure 2.10 suggested by Reeves et al. (2007). It is plausible that the ball example system with a firm basis (i.e., restricted posture) just need to stabilize the ball (i.e., spinal column), so activation in the agonist of low back could be the only requirement to maintain the spinal stability.

In summary, the results may suggest the role of lower extremity as super global muscles in trunk flexion-extension. Also, the results of Hypothesis 5, 6 and 7 suggested that the experimental procedures and setup were capable of revealing the role of local, global and super global muscle group for testing a system-level response during trunk flexion-extension.

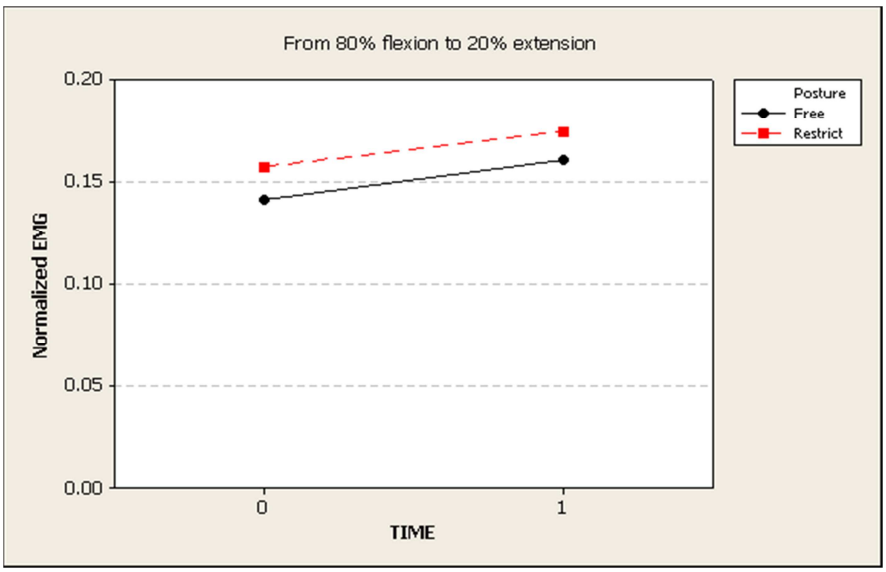

Figure 5.25 Interaction between POSTURE and TIME in NEMGs of agonist

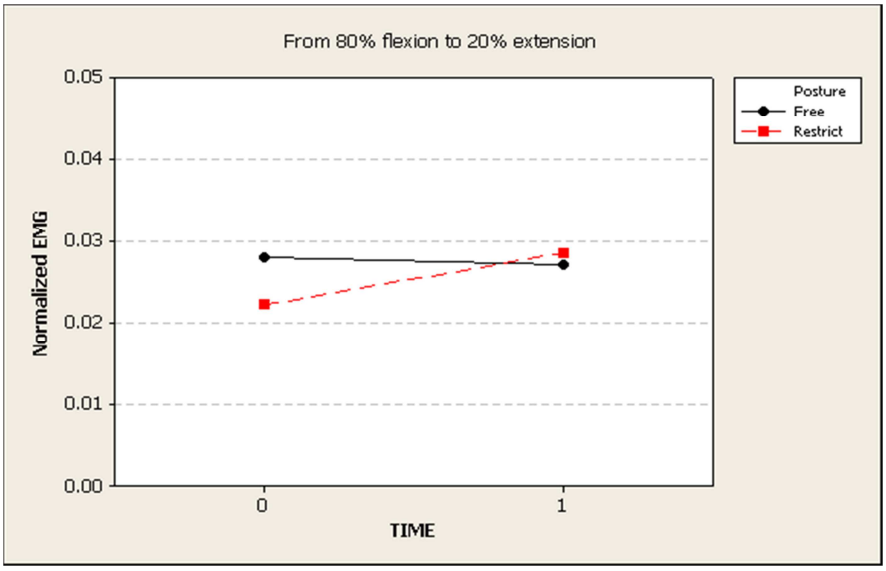

Figure 5.26 Interaction between POSTURE and TIME in NEMGs of antagonist

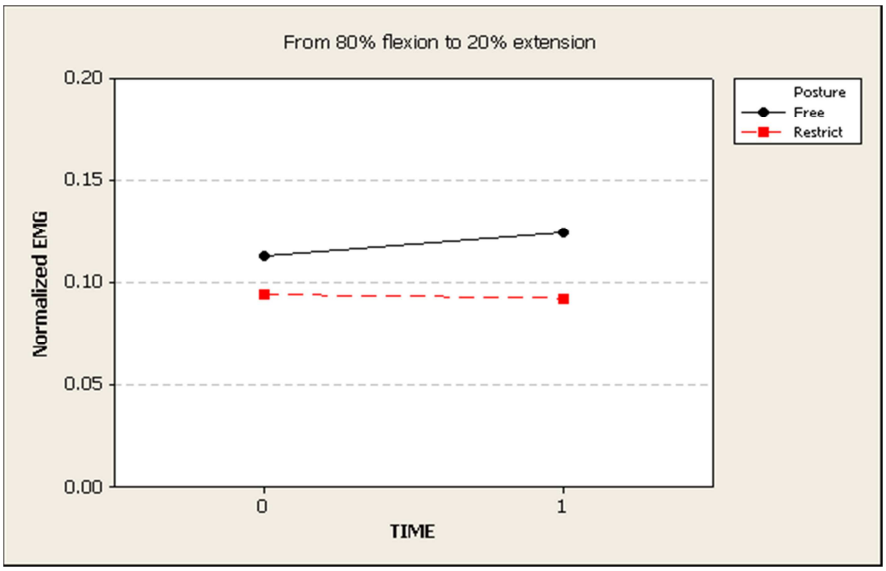

Figure 5.27 Interaction between POSTURE and TIME in NEMGs of synergist

Chapter 6 – METHODS

The iterative process used in the pilot work phase of this project resulted in experimental methods that proved to be very effective for exploring the hypotheses listed. Therefore, the methods described in the subsequent sections are largely the methods described in Chapter 5 but are reiterated here for clarity.

6.1. Participants

Twelve male participants were recruited from the undergraduate and graduate student population of the Iowa State University. The average and standard deviation of age, stature and whole body mass of participants were 28.3 yr (4.7), 175.9 cm (2.7), and 73.5 kg (6.6), respectively. This exploratory study included male participants only, because the location of the motion tracking sensors (well below the waistline) and the electromyography on buttocks makes the recruitment of female subjects problematic.

Eligible participants did not have any chronic problems or current pain in the low back or lower extremities. Each participant provided written informed consent prior to participation, using a form approved by the institutional review board (IRB) at Iowa State University.

6.2. Experimental equipment

A lumbar dynamometer (Marras and Mirka, 1989) was used to provide the static resistance (both trunk flexion and extension) during maximum voluntary contraction (MVC) and submaximal contraction sessions (35% MVC) and prevent falling over during full flexion (using waist strap) (see Figure 5.3). During the experiment, surface electromyography was used to capture the activities of the fourteen sampled muscles including right and left multifidus,

iliocostalis, rectus abdominis, external oblique, gluteus maximus, biceps femoris and rectus femoris (Model DE-2.1, Bagnoli™, Delsys, Boston, MA) (data collected at 1024 Hz). Also, a magnetic-based motion analysis system was used to capture the instantaneous lumbar flexion angle, thoracic flexion angle, trunk flexion angle and hip flexion angle (The MotionMonitor™, Innovative Sports Training, Chicago, IL) (data collected at 102.4Hz). An electrical metronome was used to assist constant trunk flexion and extension (Weird Metronome 1.4, Jone/Stone Production).

6.3. Experimental design

There were three within-subject independent variables in this study: 3 levels of PROTOCOL (A, B and C: see Figure 5.1), eight levels of TIME (0 (initial), 1 (after protocol), 2 (5 min resting), 3 (10 min resting), 4 (15 min resting), 5 (20 min resting), 6 (30 min resting) and 7 (40 min resting)) and two levels of POSTURE (free stooping and restricted stooping).

Protocol A was designed to develop laxity in viscoelastic tissues of the low back through prolonged stooping in the seated posture; Protocol B was designed to cause muscle fatigue in low back muscles through static holding at 45 degree trunk flexion in the seated posture; and Protocol C was designed to have a combined effect of both passive tissue stretching and active tissue fatigue (see Figure 5.6, 5.7 and 5.8). Each protocol lasted 10 min. After 10 minutes protocol the subject had a 40 min recovery period. The lower extremities of the participants were constrained with straps on the thighs and pelvis in the restricted stooping posture, but there was no restriction during the free stooping posture (Figure 5.3).

Dependent variables were the peak lumbar flexion angle, peak thoracic flexion angle, peak hip flexion angle, peak trunk flexion angle, average normalized EMG (NEMG) for agonist

(multifidus and iliocostalis), antagonist (rectus abdominis and external oblique), synergist (gluteus maximus and biceps femoris) and rectus femoris during isometric and isokinetic exertions, EMG-off angle (as defined in Chapter 2.5.1) during isokinetic trunk flexion-extension, and median power frequency (more detail in Chapter 5.2.4, Figure 5.9 and Table 5.1). The peak lumbar flexion angle was used to document the degree of passive tissue elongation after the 10 min protocol, and the median power frequency showed the level of muscle fatigue. The NEMG in isometric and isokinetic trials revealed the change in muscle activation pattern after the protocols. All dependent measures were recorded before and after the protocol and during the recovery session.

6.4. Experimental procedures

Subjects visited three times for three separate experiments (three protocols) on different days with an interval of at least one week. Each subject was assigned to one of the following experiment sequences: (1) Protocol A – Protocol B – Protocol C; (2) Protocol B – Protocol C – Protocol A; and (3) Protocol C – Protocol A – Protocol B. Upon arrival, the experiment was described and the subjects were asked to sign an informed consent form. Participants' anthropometric data were then recorded (first visit only). A brief (5 minute) warm up routine was provided to let subjects stretch and warm up the muscles of the low back and lower extremities.

The subjects were then fitted with a set of sensors designed to capture muscle activation levels (electromyography: EMG) and 3-dimensional trunk positions (magnetic motion sensors). EMG sensors were bilaterally placed over the following muscles: (1) multifidus: 1 cm from the vertebral midline at L5 level; (2) iliocostalis: 5 cm from the vertebral midline at L2 level; (3)

rectus abdominis: 5 cm above the umbilicus and 3 cm lateral to the midline; (4) external oblique: 10 cm from the midline of the abdomen and 4 cm above the ilium at an angle of 45°; (5) gluteus maximus: over the center of the muscle approximately 5–6 cm below the cranial origin of the muscle; (6) biceps femoris: center of the muscle at the back and mediolateral side of the thigh; and (7) rectus femoris: 10 cm above the knee joint cleft at the anterior side. Also, motion sensors were placed over the first sacral vertebrae (S1), the twelfth thoracic vertebrae (T12), the seventh cervical vertebrae (C7) and xiphoid process (bone below sternum).

Subjects then stepped onto a lumbar dynamometer and performed a series of maximum voluntary contractions (two trunk extensions and two trunk flexions) in a 20 degree trunk flexion angle against the static resistance provided by the reference frame. Also, they were asked to perform a series of maximum isometric leg flexion trials for each leg. During the trials, an experimenter manually provided static resistance on the ankle. A one minute rest period was provided between exertions. These data were collected for normalizing EMG activity of each muscle for each subject with respect to their maximum EMG activity.

Before starting the recording session, the participants were asked to stand upright and reach full flexion posture for calibration of the motion sensor data, used to calculate percentage of trunk flexion. The participants were then asked to perform two trunk extension exertions wherein they generated a trunk extension torque equal to 35% of their capacity at 20 degree trunk flexion from the standing posture for 6 seconds (Recording 1). A three minute break was provided before next recording. The participants were then asked to do slow, controlled flexion and extension trunk motions consisting of two free stooping trials (no restriction) and two trials where the lower extremities were constrained (secure the thighs and pelvis with straps to the lumbar dynamometer) (Recording 2). These trials consisted of a 5 second flexion motion (to full

flexion), 4 seconds of holding at full flexion and then 5 seconds to extend back to upright posture in time with a metronome sound (one beat per second). The subjects were asked to exhale in full flexion posture for reaching full flexion. The full flexion posture was defined by a trigger signal manually controlled by one investigator during the trials. The point was used to find the FR angle.

The subjects then conducted one of three experimental protocols that is assigned for that day: (1) alternately perform 25 sec full flexion in the seated posture (see Figure 5.6) and 5 sec upright sitting (see Figure 5.7) for 10 min (Protocol A); (2) alternately perform 25 sec static holding (see Figure 5.8) at 45 degree trunk flexion (no external load, just holding weight of torso) and 5 sec upright sitting for 10 minutes under the seated posture (Protocol B); and (3) consecutively perform 25 sec full flexion, 5 sec upright sitting, 25 sec static holding at 45 degree trunk flexion and 5 sec upright sitting for 10 minutes under the seated posture (Protocol C) (see Figure 5.1). Protocol A was designed to cause passive tissue elongation only, and Protocol B was aimed to generate low back muscle fatigue only. Protocol C was designed to bring about the combined effect of both passive tissue elongation and lumbar muscle fatigue. During the static full flexion, there was restriction on the subject's pelvis using strap, but there was no restriction in the legs. When the 10 minute protocol was completed, the Recording 2 session (described above) was conducted. For minimizing possible recovery effect during the recording session, half of the subjects conducted the free stooping trials first, and another half of the subjects performed the restricted stooping trials first. Once completed, Recording 1 session (described above) was performed. The 40 minute recovery process then began.

During this recovery period, the Recording 1 and Recording 2 sessions were conducted every five minutes until the 20 minute mark and then were performed at 30 minutes and finally

at 40 minutes. After the final recording session, electrodes and magnetic sensors were removed and the subject was free to leave.

6.5. Data analysis

6.5.1. Brief description of the data analysis

All data analysis procedures were the same as those presented in Chapter 5.2.4. The kinematic data were captured by the four magnetic sensors and used to calculate the thoracic flexion angle, lumbar flexion angle, trunk flexion angle and hip flexion (pelvic rotation) angle. Also, the lumbar flexion angle was employed to divide trunk flexion and extension motion into the 40 sections with 5% interval (20 sections for flexion and 20 sections for extension).

The initiation angle of flexion-relaxation used the data recorded from the isokinetic trunk flexion-extension and defined the EMG-off point using a computer algorithm. The characteristic of standard deviation profiles of the EMG data was used to define the EMG-off point, and the point was matched with corresponding lumbar flexion angle.

The isometric exertions in 35% MVC at 20° trunk flexion were used to capture the muscle activities during the static exertions and the level of muscle fatigue. The muscle activities revealed the effect of the protocol by showing alteration in its activation level under the same level of extension moment (35% of their capacity). The median frequency was calculated to represent the level of muscle fatigue which can be detected by the shift in median frequency to low level of frequency (hertz).

The isokinetic trunk flexion-extension was used to capture muscular recruitment pattern in trunk and lower extremity under different postures and time. Each flexion-extension motion was divided into 5% interval (40 sections), and the range of motion (from 80% flexion to 20%

extension) was used to show the effect of POSTURE and TIME on the muscular recruitment pattern.

Data preparation process was conducted after finishing all data analysis process. For H1, H2 and H3, the two replications at each of TIME 0 and TIME 1 were averaged respectively, and they were paired within each subject and posture (free or restricted) for a pair-wise comparison. For H4, the processed data were modified into the magnitude of the increase or decrease from TIME 0 to TIME 1 (normalized to TIME 0). Hence, the two observations in TIME 0 within a protocol of a subject were averaged and used to calculate the changes in each data.

6.5.2. Graphical representation of the resting data

To reveal the characteristics of the recovery phase (from TIME 2 to TIME 7), the full lumbar flexion angle, EMG-off points and muscle activities during isometric and isokinetic trials were plotted after data standardization process within each subject and each protocol as follows:

$$Z_{ij} = \frac{X_{ij} - \bar{X}_j}{s_j}$$

where $i = 1, \ldots, 16;\ j = 1, \ldots, 9$

There were total 16 observations (*i*) (2 replication × 8 levels of TIME) within a column, and each column (*j*) represented 9 dependent variables such as peak lumbar flexion angle, EMG-off points in multifidus and iliocostalis, and agonist, antagonist and synergist muscle activities in isometric and isokinetic trials. The raw data were standardized to remove natural variation caused by different baseline (TIME 0) in each subject and each protocol that also cause larger variation in the following trials (from TIME 1 to TIME 7). The standardized data has mean vector (*j*) all zeros, and variances all equal to 1. After the standardization procedure, the average

of all twelve subjects in each TIME was plotted for visual analysis. In this figure, '+1 or -1' in Y-axis represented 1 standard deviation from mean (0).

6.5.3. Statistical analysis

All statistical analyses in this study were conducted using SAS® and Minitab®. Prior to model analysis, diagnostic tests were performed on the data, including, test for homoscedasticity (Bartlett's Test and Levene's Test) and normality (Anderson-Darling Normality Test) (Montgomery, 2001). Dependent variables that violated one or more assumption were transformed so that the ANOVA assumptions were fully satisfied (Montgomery, 2001).

The univariate ANOVA was conducted for selecting significant main effects and interaction effects, and the Post hoc tests employing Bonferroni's method were then performed on these significant effects. The paired *t*-tests were also performed for testing difference between two groups. A *p*-value less than 0.05 was regarded as the standard level of significance of an effect in current study. Table 6.1 provides summary of the statistical analysis required to test the hypotheses.

Table 6.1 Summary of statistical analysis

Hypothesis	Independent variable	Dependent variable	Contrast	Experimental design
H1	TIME (0, 1)	1. Peak lumbar flexion angle 2. Lumbar angle at EMG-off for multifidus 3. Lumbar angle at EMG-off for iliocostalis	TIME 0 vs. TIME 1 (Protocol A)	Paired *t*-test ($\mu_d = \mu_0$ vs. $\mu_d \neq \mu_0$) where μ_d is the population mean of the differences
H2	TIME (0, 1)	1. Peak lumbar flexion angle 2. Lumbar angle at EMG-off for multifidus 3. Lumbar angle at EMG-off for iliocostalis	TIME 0 vs. TIME 1 (Protocol B)	Paired *t*-test ($\mu_d = \mu_0$ vs. $\mu_d \neq \mu_0$) where μ_d is the population mean of the differences
H3	TIME (0, 1)	1. Peak lumbar flexion angle 2. Lumbar angle at EMG-off for multifidus 3. Lumbar angle at EMG-off for iliocostalis	TIME 0 vs. TIME 1 (Protocol C)	Paired *t*-test ($\mu_d = \mu_0$ vs. $\mu_d \neq \mu_0$) where μ_d is the population mean of the differences
H4	TIME (1) × PROTOCOL (A, B, C)	1. NEMG for agonist in 35% MVC 2. NEMG for antagonist in 35% MVC 3. NEMG for synergist in 35% MVC	Protocol A at TIME 1 vs. Protocol B at TIME 1 vs. Protocol C at TIME 1	1-way ANOVA (GLM)
H5	POSTURE	1. Peak trunk flexion angle 2. Peak hip flexion angle 3. Peak lumbar flexion angle 4. Peak thoracic flexion angle 5. Lumbar angle at EMG-off for multifidus 6. Lumbar angle at EMG-off for iliocostalis	POSTURE (free stooping) at TIME 0 vs. POSTURE (restricted stooping) at TIME 0	1-way ANOVA (GLM)
H6	POSTURE	1. NEMG for agonist in isokinetic trials 2. NEMG for antagonist in isokinetic trials 3. NEMG for synergist in isokinetic trials	POSTURE (free stooping) at TIME 0 vs. POSTURE (restricted stooping) at TIME 0	1-way ANOVA (GLM)
H7	POSTURE × TIME	1. NEMG for agonist in isokinetic trials 2. NEMG for antagonist in isokinetic trials 3. NEMG for synergist in isokinetic trials	2 × 2 interaction	2-way ANOVA (GLM)

Chapter 7 – RESULTS

7.1. Effects of protocols on FRP

Recall that there were three protocols that were tested to reveal their effects on lumbar flexion angle and flexion-relaxation phenomenon (EMG-off and -on angles). First, the Hypothesis 1 aimed to test the effect of the passive tissue elongation in low back (Protocol A) (TIME: 0, 1) on the peak (or full) lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point). Second, the goal of Hypothesis 2 was to investigate the effect of the muscle fatigue (Protocol B) (TIME: 0, 1) on the peak (or full) lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point). Third, Hypothesis 3 was designed to test the combined effect of passive tissue stretching and muscle fatigue in low back (Protocol C) (TIME: 0, 1) on the peak lumbar flexion angle and the occurrence points of FR (lumbar flexion angles at the EMG-off point). In all three hypotheses, the lumbar angle at the EMG-on point was also tested for a comparison with prior studies.

Paired *t*-tests were employed to test the effect of each protocol. Prior to conducting statistical analyses, the two replications at each of TIME 0 and TIME 1 were averaged respectively, and they were paired within each subject and posture (free or restricted) for a pair-wise comparison. Also, the assumptions of paired *t*-test (normality, equal variance and independence of observations) were evaluated using graphical approaches advocated by Montgomery (2001).

7.1.1. Laxity in low back viscoelastic tissues – Protocol A

To reveal changes in the effect of Protocol A on lumbar spine, paired *t*-tests were conducted. Paired *t*-tests on the peak lumbar flexion angle and the lumbar flexion angle at

EMG-off and -on points of multifidus and iliocostalis showed statistically significant difference between TIME 0 (initial condition) and TIME 1 (after Protocol A) (Table 7.1). The results are in accordance with Hypothesis 1 and previous studies, suggesting increases (i.e., deeper) in peak lumbar flexion angle and the lumbar angles at EMG-off and EMG-on points after the passive tissue elongation protocol. The 10-minute protocol (alternately perform 25 sec full flexion in the seated posture and 5 sec upright sitting for 10 min) caused 2.57° deeper full lumbar flexion angle (from 36.61° to 39.18°), 2.31° deeper EMG-off angle in multifidus (from 31.88° to 34.19°), 2.43° deeper EMG-off angle in iliocostalis (from 31.11° to 33.54°), 2.38° deeper EMG-on angle in multifidus (from 37.04° to 39.42°) and 2.41° deeper EMG-on angle in iliocostalis (from 37.00° to 39.41°) (Table 7.1 and Figure 7.1, 7.2, 7.3, 7.4, 7.5). The results suggested elongation of viscoelastic tissues and modification in the load-sharing mechanism between active and passive tissues in low back.

Table 7.1 Results of paired *t*-tests for Protocol A (H1)

	Mean Difference	*t*-value	*p*-value
Lumbar Flexion	2.57°	4.69	< 0.001
EMG-off (Mul)	2.31°	4.63	< 0.001
EMG-off (Ilio)	2.43°	4.65	< 0.001
EMG-on (Mul)	2.38°	4.17	< 0.001
EMG-on (Ilio)	2.41°	4.38	< 0.001

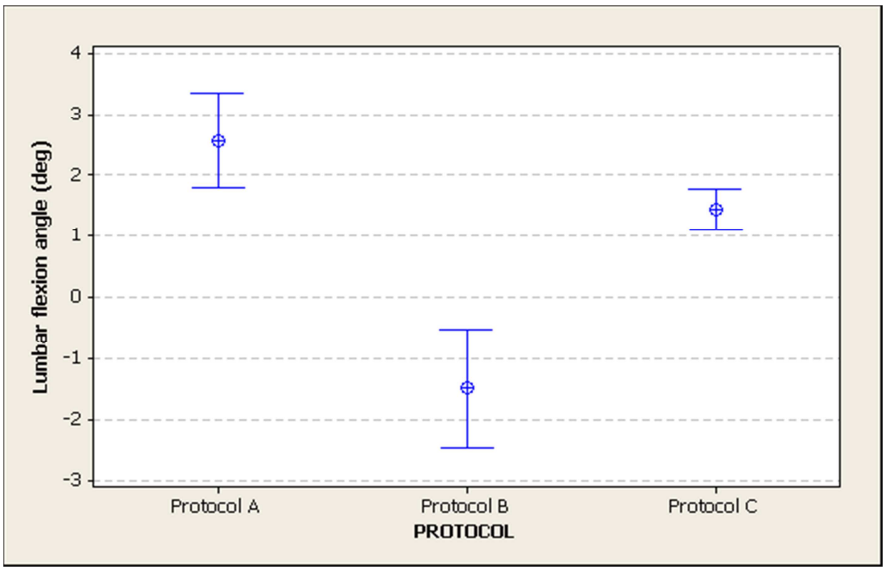

Figure 7.1 Amount of changes in the peak lumbar flexion angle after each protocol (Error bars show 95% confidence interval)

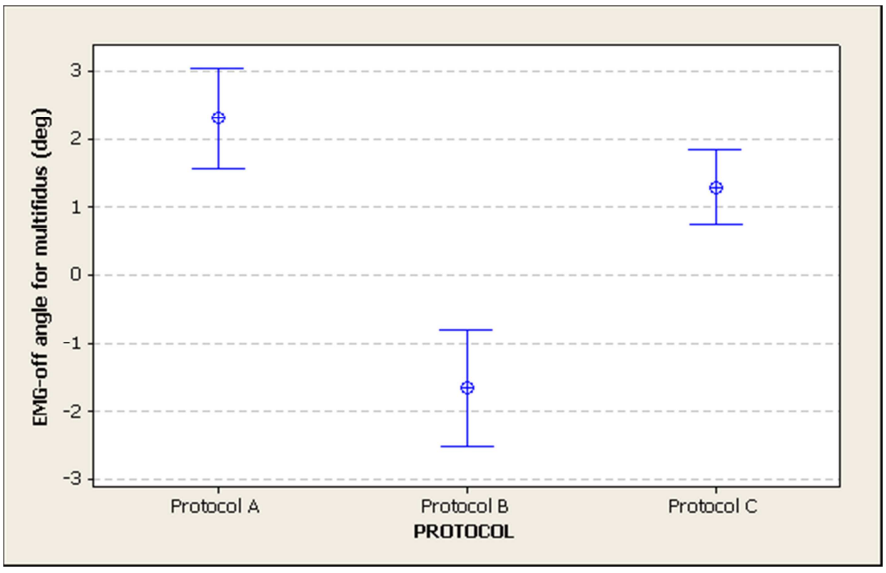

Figure 7.2 Amount of changes in the lumbar flexion angle at the EMG-off point for multifidus (Error bars show 95% confidence interval)

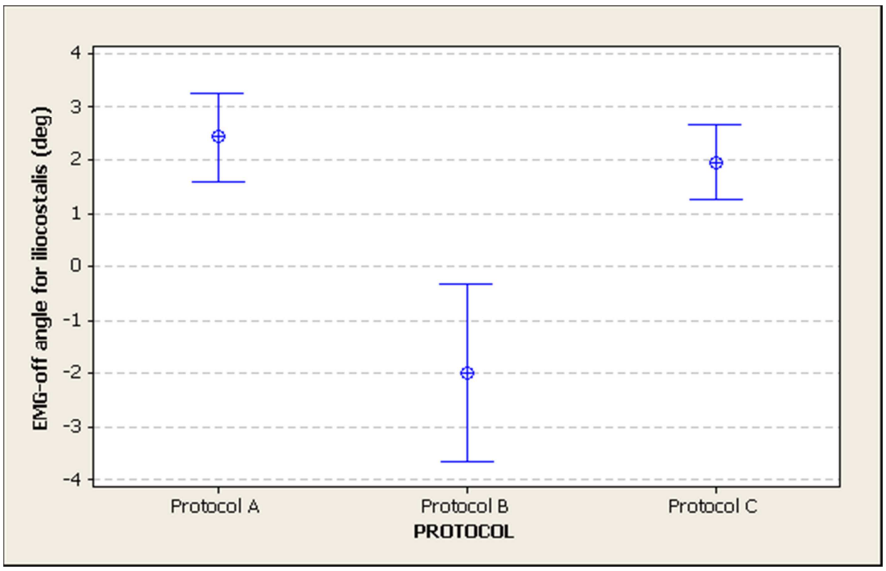

Figure 7.3 Amount of changes in the lumbar flexion angle at the EMG-off point for iliocostalis (Error bars show 95% confidence interval)

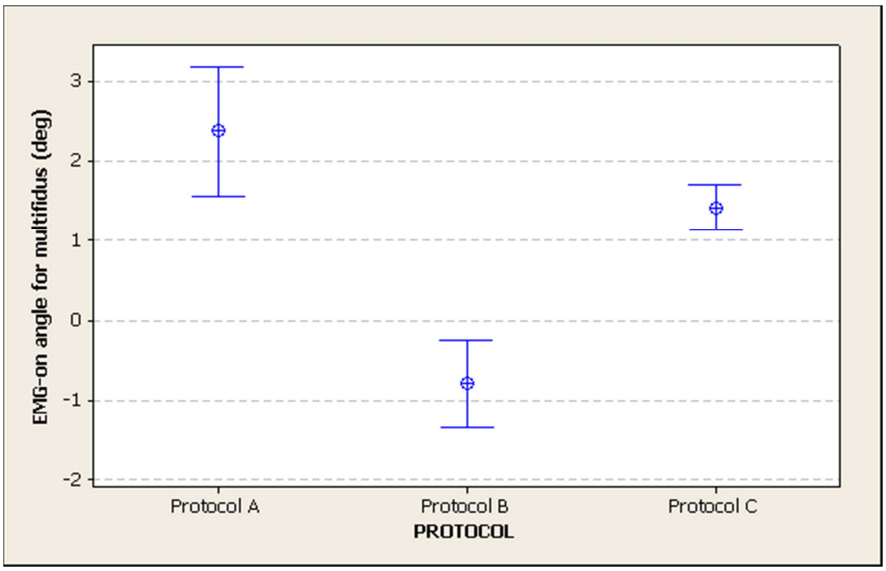

Figure 7.4 Amount of changes in the lumbar flexion angle at the EMG-on point for multifidus (Error bars show 95% confidence interval)

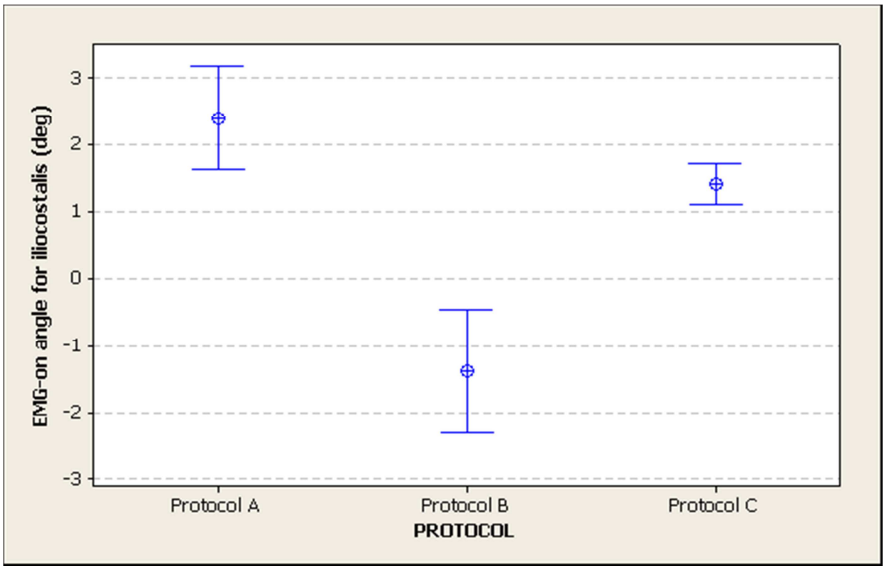

Figure 7.5 Amount of changes in the lumbar flexion angle at the EMG-on point for iliocostalis (Error bars show 95% confidence interval)

7.1.2. Low back muscle fatigue – Protocol B

Paired *t*-tests were used to reveal the effect of Protocol B on the peak lumbar flexion angle and the lumbar flexion angle at EMG-off and -on points of multifidus and iliocostalis. The results showed statistically significant difference between TIME 0 (initial condition) and TIME 1 (after Protocol B) in all dependent variables (Table 7.2 and Figure 7.1, 7.2, 7.3, 7.4, 7.5). In line with the Hypothesis 2, the peak lumbar flexion angle and the occurrence points of FR were decreased (i.e., less flexion) after 10 minutes protocol which is designed to generate low back muscle fatigue (Protocol B). The low back muscle fatigue was confirmed by the median frequency shift from 96.3 Hz to 88.9 Hz in multifidus denoting presence of low back muscle fatigue (p-value < 0.001), but there was almost no change in iliocostalis (from 78.2 to 77.6) (p-

value = 0.462) (Figure 7.6 and 7.7). The results showed that the presence of muscle fatigue in low back reduced the peak lumbar flexion angle about 1.5 degree suggesting less peak lumbar flexion. Also, the 10-minute protocol (alternately perform 25 sec static holding at 45 degree trunk flexion and 5 sec upright sitting for 10 minutes under the seated posture) caused 1.65° less EMG-off angle in multifidus, 2.01° less EMG-off angle in iliocostalis, 0.79° less EMG-on angle in multifidus and 1.38° less EMG-on angle in iliocostalis (Table 7.2).

Table 7.2 Results of paired *t*-tests for Protocol B (H2)

	Mean Difference	*t*-value	*p*-value
Lumbar Flexion	-1.50°	-2.28	0.032
EMG-off (Mul)	-1.65°	-3.25	0.004
EMG-off (Ilio)	-1.58°	-2.53	0.021
EMG-on (Mul)	-0.79°	-2.36	0.028
EMG-on (Ilio)	-1.38°	-2.29	0.032

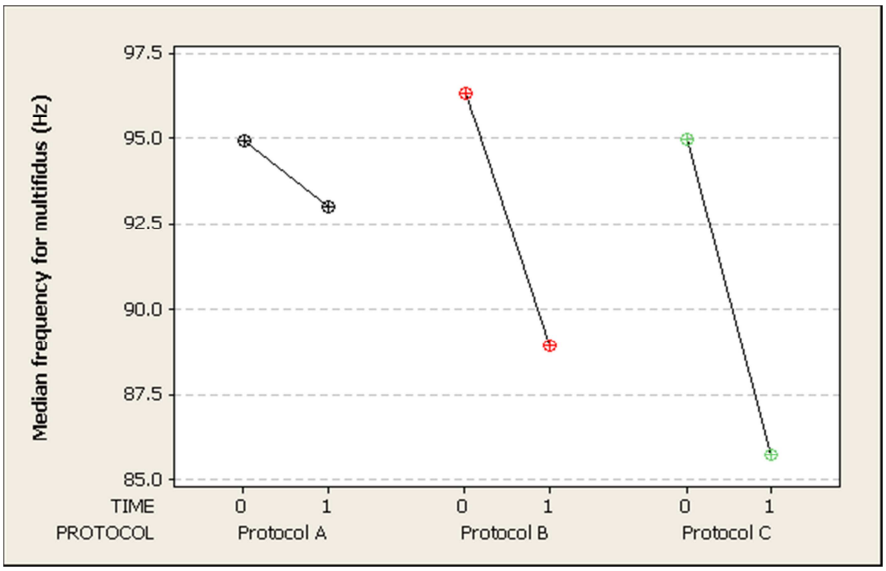

Figure 7.6 Median power frequency of multifidus before and after three protocols

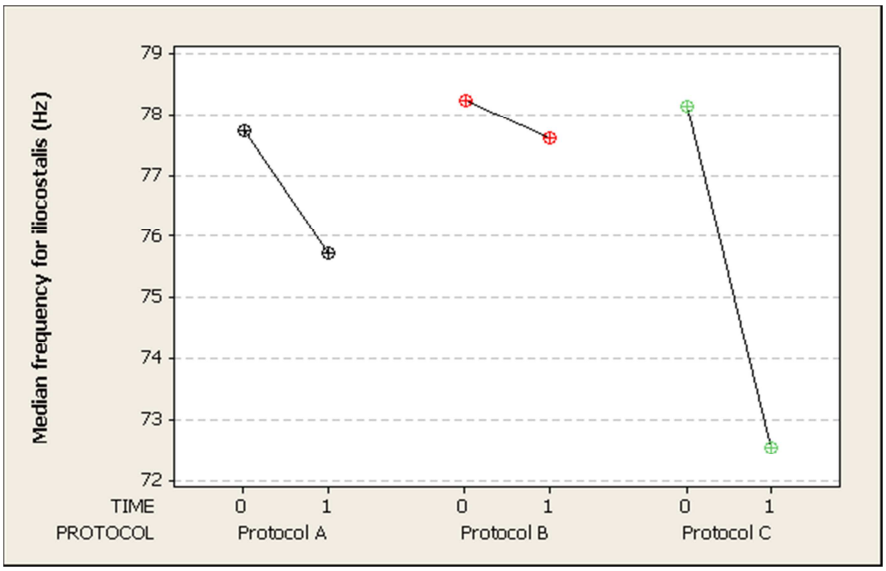

Figure 7.7 Median power frequency of iliocostalis before and after three protocols

7.1.3. Combination of laxity in viscoelastic tissues and muscle fatigue – Protocol C

To investigate the effect of Protocol C (consecutively perform 25 sec full flexion, 5 sec upright sitting, 25 sec static holding at 45 degree trunk flexion and 5 sec upright sitting for 10 minutes under the seated posture) paired *t*-tests were conducted. The results showed statistically significant difference in the peak lumbar flexion angle and the occurrence points of FR (Table 7.3 and Figure 7.1, 7.2, 7.3, 7.4, 7.5). The peak lumbar flexion angle and the EMG-off and -on points were increased (i.e., deeper) after the combined effect protocol (Protocol C) like the Protocol A (passive tissue elongation protocol), but the magnitude of increases was smaller in both full lumbar flexion angle (Protocol A: 2.57° vs. Protocol C: 1.44°) and EMG-off and -on points (EMG-off (mul): 2.31° vs. 1.29°; EMG-off (ilio): 2.43° vs. 1.96°; EMG-on (mul): 2.38° vs. 1.41°; EMG-on (ilio): 2.41° vs. 1.42°). The smaller amount of change could be attributable to the counteraction of the low back muscle fatigue in both muscles which is confirmed by statistically significant shift in median power frequency after the protocol (multifidus and iliocostalis: p-value < 0.001) (from 95 Hz to 86 Hz in multifidus; from 78 Hz to 72 Hz in iliocostalis) (Figure 7.6 and 7.7). The results suggest higher levels of muscle fatigue in Protocol C than the muscle fatigue protocol (Protocol B), even though the static holding time was half as long (Protocol B: 500 sec vs. Protocol C: 250 sec). It is possible that total 5 minutes of the passive tissue elongation period performed in the Protocol C also contributed to the development of the low back muscle fatigue in addition to total 5 minutes static holding period, and consequently boosts the level of muscle fatigue in Protocol C. Shin et al. (2009) showed development of fatigue like responses in low back after the 6 minutes prolonged passive stretching. Also, Avela et al. (2004) revealed reduced motor unit activation, reduced reflex

responses and reduced force-generation capacity after prolonged passive stretching of the triceps surae muscle group indicating a muscle fatigue response..

Table 7.3 Results of paired *t*-tests for Protocol C (H3)

	Mean Difference	*t*-value	*p*-value
Lumbar Flexion	1.44°	6.12	< 0.001
EMG-off (Mul)	1.29°	3.88	0.001
EMG-off (Ilio)	1.96°	4.78	< 0.001
EMG-on (Mul)	1.41°	7.16	< 0.001
EMG-on (Ilio)	1.42°	6.59	< 0.001

7.2. Effects of protocols on muscle activities in static exertions

The effect of PROTOCOL and TIME on muscle activities of agonist, antagonist and synergist in 35% isometric exertions was investigated to reveal changes in muscle recruitment pattern after each protocol and difference among protocols (Hypothesis 4). The assumptions of ANOVA (normality, homoscedasticity and independence of observations) were evaluated using graphical approaches advocated by Montgomery (2001) (APPENDIX A).

The effect of each protocol was tested using one-way ANOVA. In line with Hypothesis 4, the results revealed statistically significant increases in muscle activation level of agonist and synergist after all three 10-min protocols (Table 7.4 and Figure 7.8, 7.9, 7.10). No significant

change in antagonist after the protocols was expected, but the muscle fatigue protocol (Protocol B) showed a significant increase in antagonist after 10-min static trunk holding protocol.

Considering the amount changes in muscle activation level after each protocol, the muscle fatigue protocol (Protocol B) showed larger increase in all muscle groups (agonist, antagonist and synergist) as compared to the passive tissue elongation protocol (Protocol A) and the combined effect protocol (Protocol C). The Protocol B showed a 4.2 % increase from TIME 0 (from 0.295 to 0.337) in agonist, a 0.8 % increase from TIME 0 (from 0.077 to 0.085) in antagonist and a 3.7 % increase from TIME 0 (from 0.295 to 0.332) in synergist. The larger increases after the Protocol B suggested an interactive effect between PROTOCOL and TIME.

Table 7.4 Results of one-way ANOVAs for isometric exertions of each protocol (H4)

		Mean Difference	*F*-value	*p*-value
Protocol A	Agonist	0.027	8.74	0.006
	Antagonist	- 0.005	0.41	0.715
	Synergist	0.014	6.20	0.018
Protocol B	Agonist	0.042	22.46	< 0.001
	Antagonist	0.008	5.71	0.022
	Synergist	0.037	26.06	< 0.001
Protocol C	Agonist	0.034	17.72	< 0.001
	Antagonist	0.002	0.01	0.942
	Synergist	0.015	9.03	0.005

To investigate the interaction effect between PROTOCOL and TIME one-way ANOVAs were used to test difference among three protocols at TIME 1 instead of the two-way ANOVA (PROTOCOL (3) × TIME (2)); remember that the raw data were normalized to TIME 0 (all three protocols have zero at TIME 0). The result showed significant difference in antagonist and synergist (p-value=0.017 and p-value=0.006, respectively), but there is no significant difference among three protocols in agonist (p-value=0.196) (Figure 7.8, 7.9 and 7.10). Regarding the antagonist, the following post-hoc test using the Bonferroni method revealed significantly higher antagonist muscle activities in the Protocol B and C than Protocol A. However, considering the negative antagonist muscle activity in Protocol A (i.e., random error) and no significant difference between Protocol B and C in antagonist (Table 7.4) it is reasonable to believe that there are no significant difference among protocols. In regards of the synergist, the post-hoc test showed a significantly larger increase in synergist muscle activity in Protocol B as compared to the Protocol A and C. The amount of increase in Protocol B was more than twice of the Protocol A and C (Protocol B: 0.037 vs. Protocol A: 0.014 and Protocol C: 0.015). The results suggested different muscle activation patterns after three different 10-min protocols.

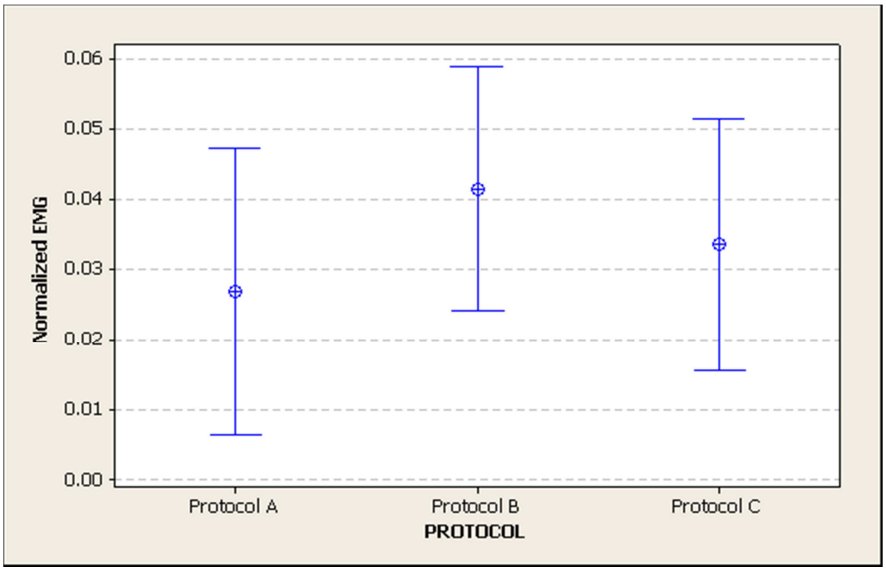

Figure 7.8 Amount of increase in NEMG of agonist after 10-min protocols (Error bars show 95% confidence interval)

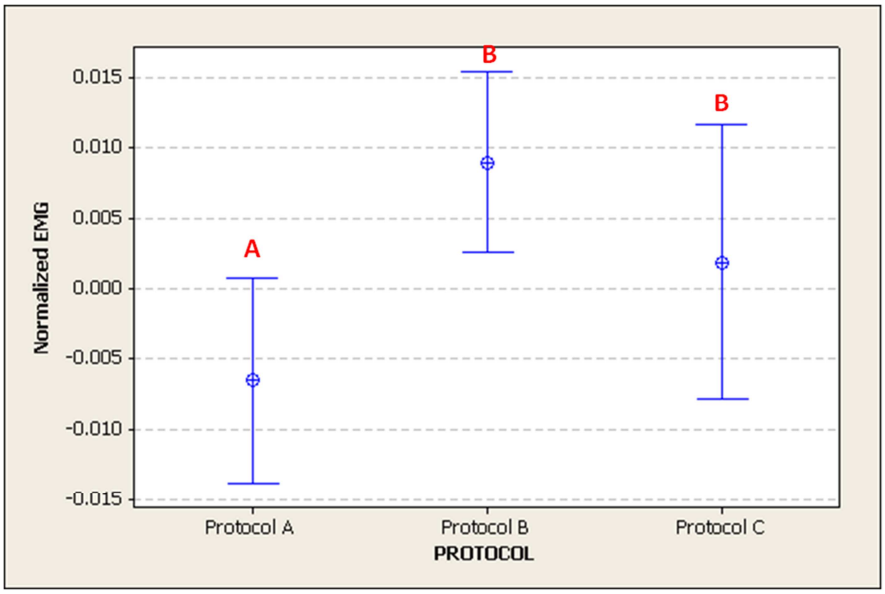

Figure 7.9 Amount of change in NEMG of antagonist after 10-min protocols (Error bars show 95% confidence interval. Different letters indicate levels that are significantly different.)

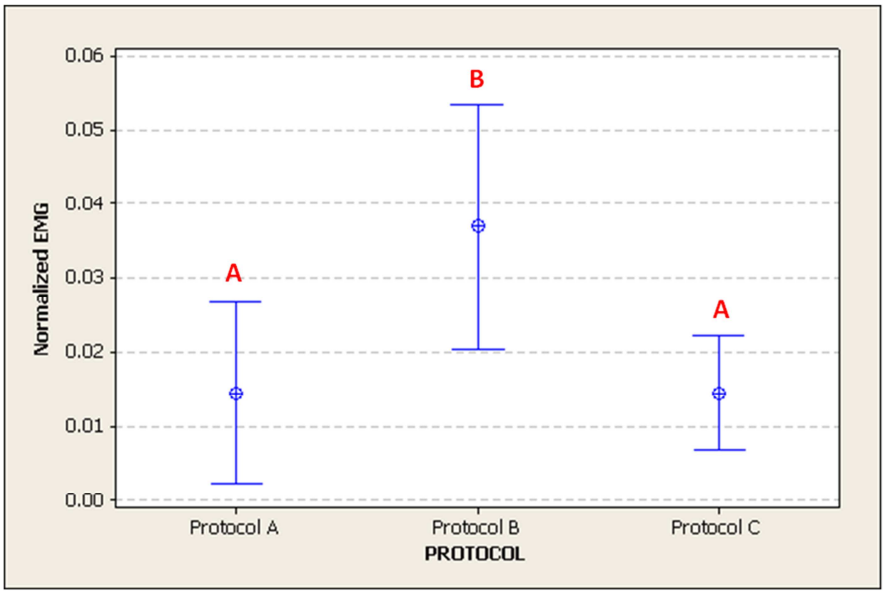

Figure 7.10 Amount of increase in NEMG of synergist after 10-min protocols (Error bars show 95% confidence interval. Different letters indicate levels that are significantly different.)

7.3. Role of lower extremity during trunk flexion-extension

7.3.1. Effect of lower extremity kinematics on trunk kinematics and FRP

To better understand the role of lower extremity on flexion-relaxation phenomenon during trunk flexion-extension, trunk kinematics (the peak lumbar, thoracic, trunk and hip flexion angle) and the occurrence points of FR (lumbar and thoracic flexion angles at the EMG-off point) were investigated in two different conditions including the lower extremity constraint stooping and the free stooping (Hypothesis 5). For a comparison between two conditions, one-way ANOVAs were applied for data at TIME 0. Prior to conducting statistical analyses, the assumptions of ANOVA (normality, homoscedasticity and independence of observations) were evaluated using graphical approaches advocated by Montgomery (2001).

The one-way ANOVAs revealed significantly higher peak trunk and hip flexion angle in free stooping posture than the restricted posture (Table 7.5 and Figure 7.11). The results are

reasonable in that the pelvic movement, significantly related with hip angle, was restricted under the lower extremity constraint condition. However, the difference between two postures was smaller in trunk flexion angle than hip flexion angle (29.9° in hip flexion angle and 25.2° in trunk flexion angle). It may suggest that there was a significant difference between two postures in the flexion magnitude of the spinal columns (i.e., spinal curvature) at full stooping.

The one-way ANOVAs for lumbar and thoracic flexion angles showed significantly greater lumbar and thoracic flexion angles in restricted stooping than the free stooping (Table 7.5 and Figure 7.12). So, the main source of smaller change in trunk flexion angle was explained by additional sagittal flexion of spinal columns in restricted stooping posture. The restricted posture was 2.3° deeper in the peak lumbar flexion angle and 2.7° deeper in the peak thoracic flexion angle than the free stooping posture (Table 7.5) even the hip and trunk flexion angles were significantly smaller in the posture. The difference between peak trunk flexion angle and hip flexion angle (4.7°) was bigger than the gap between the restricted stooping and the free stooping in peak thoracic flexion angles (2.7°). This two degree discrepancy could be caused by location of sensors. Recall that the trunk flexion angle and the hip flexion angle were captured by the pitch angle of the sensor on C7 and S1, respectively, and the thoracic flexion was measured by difference between S1 sensor and xiphoid process sensor. The sensor on C7 may have bigger pitch angle than the sensor on xiphoid process, so it is possible that the difference between peak trunk flexion angle and hip flexion angle is larger than the difference between the restricted stooping and the free stooping in thoracic flexion angles.

In line with these results, the EMG-off points in multifidus and iliocostalis were also delayed in the restricted posture (3.2° deeper in multifidus, and 2.46° deeper in iliocostalis)

(Table 7.5 and Figure 7.13). The result may suggest that the lower extremity constraint, influencing the pelvic rhythm, modifies the trunk flexion-extension kinematics and FRP.

Table 7.5 Results of one-way ANOVAs for two different stooping postures - Kinematics (H5)

	Mean Difference (= free – restricted)	*F*-value	*p*-value
Lumbar flexion angle	-2.29°	8.38	0.004
Thoracic flexion angle	-2.71°	7.93	0.006
Trunk flexion angle	25.20°	188.98	< 0.001
Hip flexion angle	29.96°	271.12	< 0.001
EMG-off angle (multifidus)	-3.22°	14.11	< 0.001
EMG-off angle (iliocostalis)	-2.46°	14.42	< 0.001

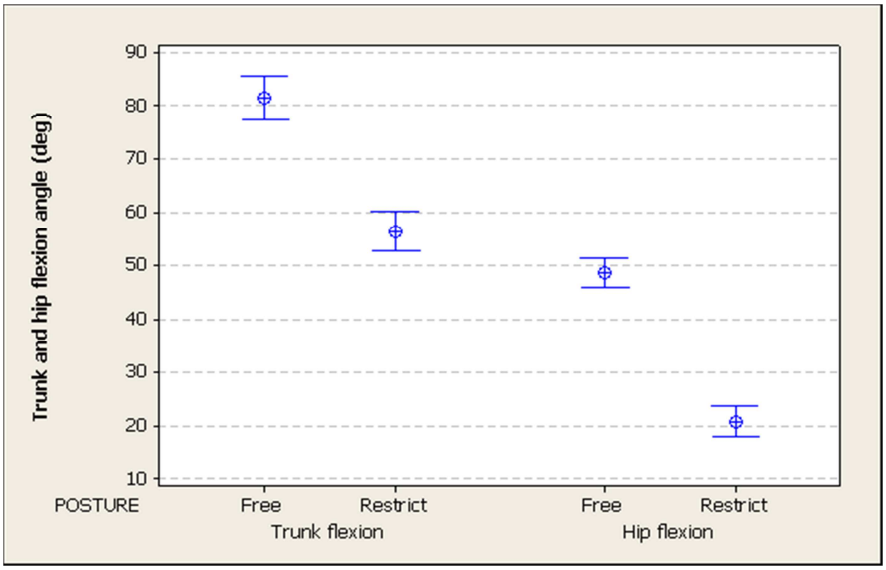

Figure 7.11 Comparison between two stooping postures in trunk and hip flexion angles (Error bars show 95% confidence interval)

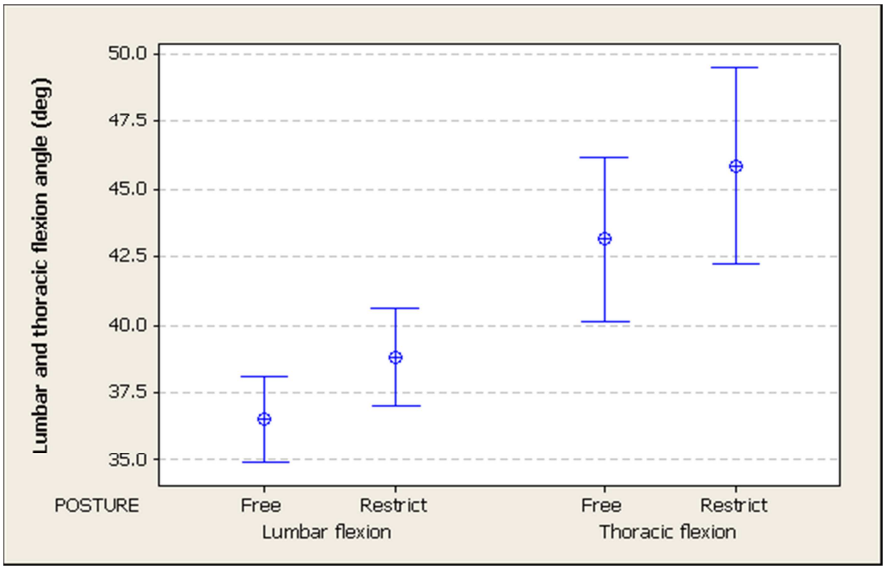

Figure 7.12 Comparison between two stooping postures in lumbar and thoracic flexion angles (Error bars show 95% confidence interval)

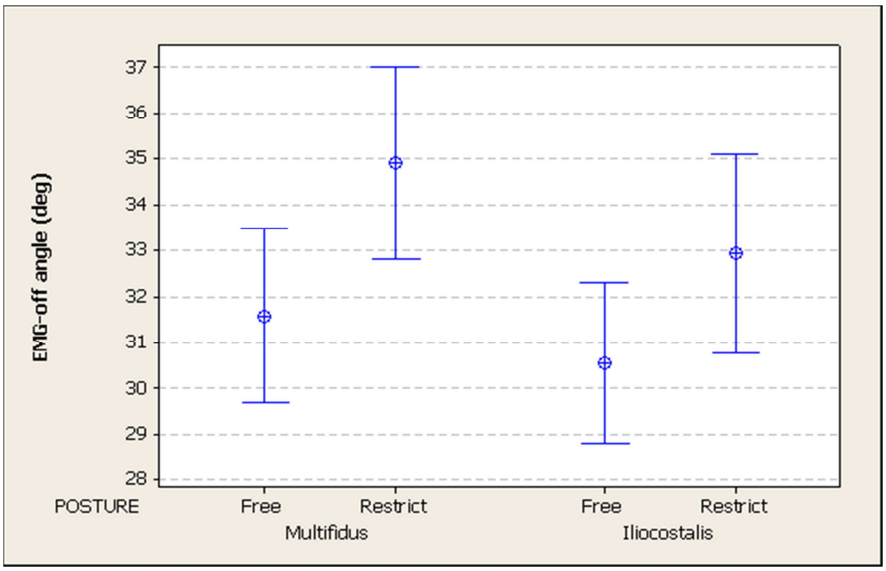

Figure 7.13 Comparison between two stooping postures in EMG-off angles (Error bars show 95% confidence interval)

7.3.2. Effect of lower extremity kinematics on muscle recruitment strategy

To better understand the role of lower extremity in trunk flexion-extension (isokinetic trials), the muscle recruitment pattern in trunk system (i.e., local and global system) and lower extremity system (i.e., super global system) were investigated in two different conditions, including the lower extremity constraint stooping and the free stooping (Hypothesis 6), under normal and abnormal low back conditions (Hypothesis 7). For a comparison between two stooping conditions in H6, one-way ANOVAs were applied for data at TIME 0. Also, two-way ANOVAs were used to test the interactive effect between POSTURE (free and restricted) and TIME (TIME 0 and 1) (H7). Prior to conducting statistical analyses, the assumptions of ANOVA (normality, homoscedasticity and independence of observations) were evaluated using graphical approaches advocated by Montgomery (2001).

Comparison between two stooping postures at TIME 0 - normal condition

In line with the hypothesis, the one-way ANOVA revealed significantly higher NEMG of agonist in the restricted stooping than the free stooping, and the NEMG of synergist showed significantly higher NEMG in free stooping as compared to the restricted stooping at the range of motion of 'from 80% flexion to 20% extension' (Table 7.6 and Figure 7.14). However, there was no difference in the muscle activity of antagonist between two stooping techniques.

Table 7.6 Results of one-way ANOVAs for two different stooping postures – Muscle activity (H6)

Section	Muscle groups	Mean Difference (free – restricted)	*F*-value	*p*-value
80% flexion ~ 20% extension	Agonist	-0.012	5.03	0.027
	Antagonist	0.012	1.96	0.164
	Synergist	0.023	16.33	< 0.001

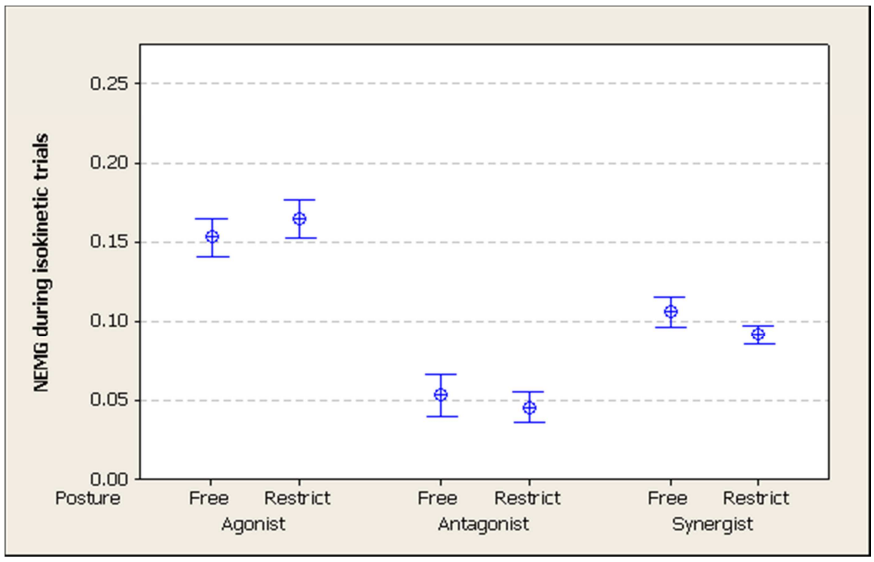

Figure 7.14 Comparison between two stooping postures in NEMG from 80% flexion to 20% extension (Error bars show 95% confidence interval)

Effects of POSTURE and TIME on muscle activation strategy

The main effects and interactive effects of POSTURE and TIME on three muscle groups were investigated to reveal the effects of lower extremity kinematics (e.g., free and restricted) on muscle recruitment strategy under the normal and abnormal low back conditions

(e.g., TIME 0 and 1). In accordance with Hypothesis 7, both agonist and synergist showed statistically significant difference in the main effect of TIME (0 vs. 1) denoting the effect of 10-min protocols on muscle activation level (Table 7.7). The agonist increased by 2.5%, and the synergist increased by 1.5% after the protocols (Figure 7.17). Also, the significant main effect of POSTURE supported the result of Hypothesis 6 suggesting bigger agonist muscle activity in the restricted stooping technique (1.6%) and greater recruitment of synergist muscles in the free stooping technique (2.8%) (Table 7.7 and Figure 7.18).

Regarding the interaction effects, the synergist showed interactive effects between POSTURE and TIME during trunk flexion-extension, but the agonist did not have an interaction effect showing similar level of effect of the POSTURE in both TIME 0 and 1 (Table 7.7 and Figure 7.19, 7.20, 7.21). The synergist showed almost no increase (0.5%) in the restricted stooping technique, but it revealed a significant increase (2.5%) in the free stooping technique after the protocols (Figure 7.21). The following analysis of the simple effects revealed that TIME was not significant in the restricted stooping condition (p-value = 0.148), but the main effect of TIME was significant in the free stooping condition (p-value < 0.001). Also, the simple effects confirmed the significant main effect of POSUTURE in both TIME 0 and 1 (p-value < 0.001).

Contrary to the Hypothesis 7, there was no significant interaction in the antagonist (Table 7.7). However, the main effects of POSTURE and TIME showed significant difference in antagonist. The result showed greater recruitment of antagonist muscles in the free stooping condition (1.3%) as compared to the restricted stooping condition (Figure 7.18). Meanwhile, the NEMG difference between TIME 0 and TIME 1 was negligible (0.14%), so it may be reasonable to regard that there is no physically significant effect (Figure 7.17).

Table 7.7 Results of two-way ANOVA between POSTURE and TIME in all three protocols (H7)

	Dependent variables		
Independent variables	Agonist	Antagonist	Synergist
Posture	F (1, 272) = 11.77, 0.001	F (1, 271) = 7.57, 0.006	F (1, 271) = 57.08, < 0.001
Time	F (1, 272) = 25.89, < 0.001	F (1, 271) = 4.54, 0.034	F (1, 271) = 17.15, < 0.001
Posture × Time	F (1, 272) = 0.44, 0.506	F (1, 271) = 0.71, 0.401	F (1, 271) = 4.36, 0.038

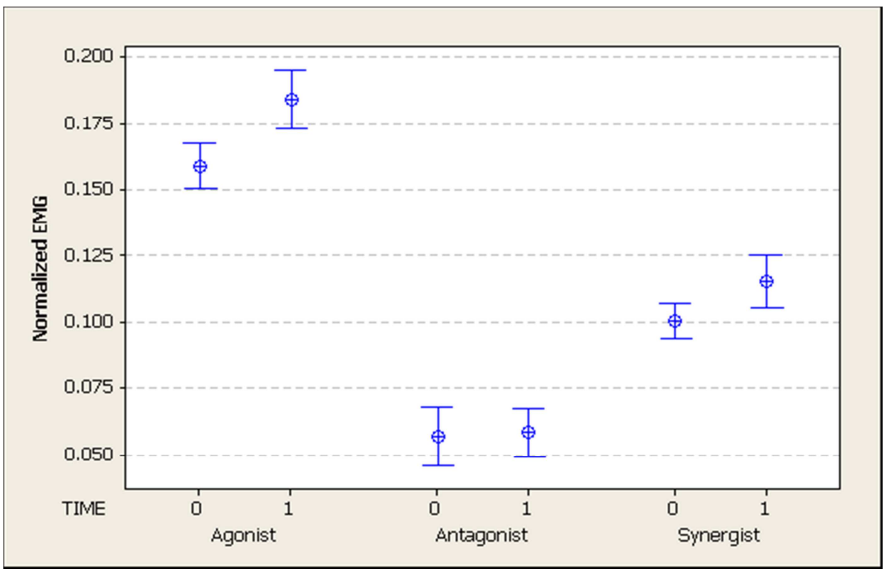

Figure 7.15 Main effect of TIME in three muscle groups (Error bars show 95% confidence interval)

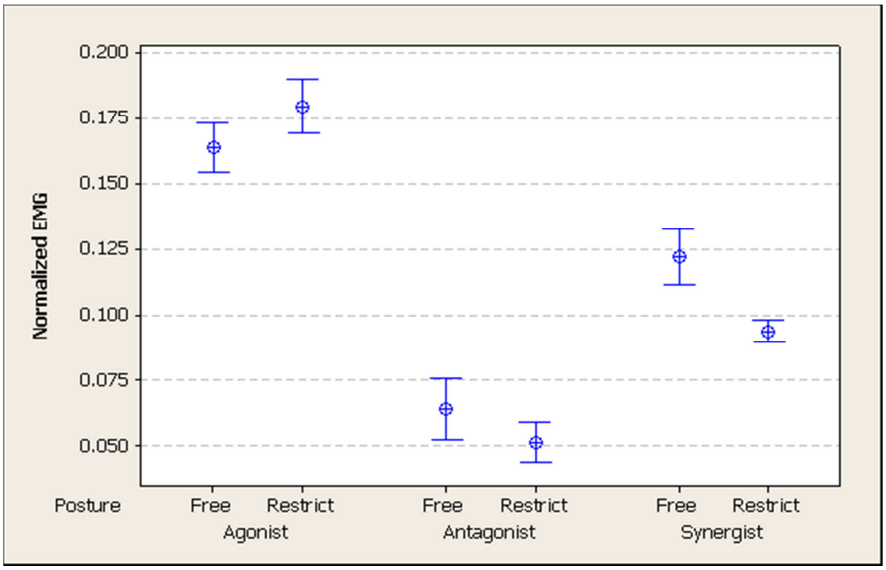

Figure 7.16 Main effect of POSTURE in three muscle groups (Error bars show 95% confidence interval)

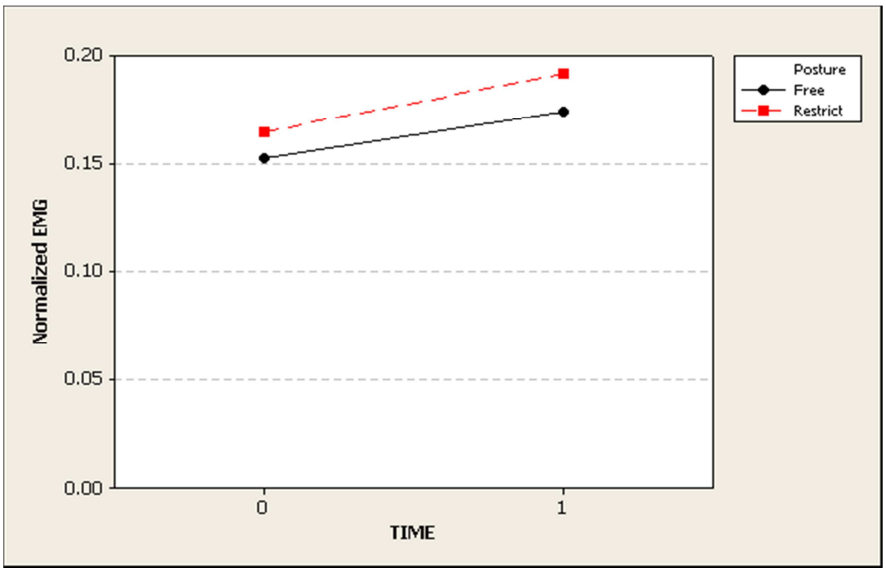

Figure 7.17 Interaction plot between POSTURE and TIME in all three protocols - Agonist

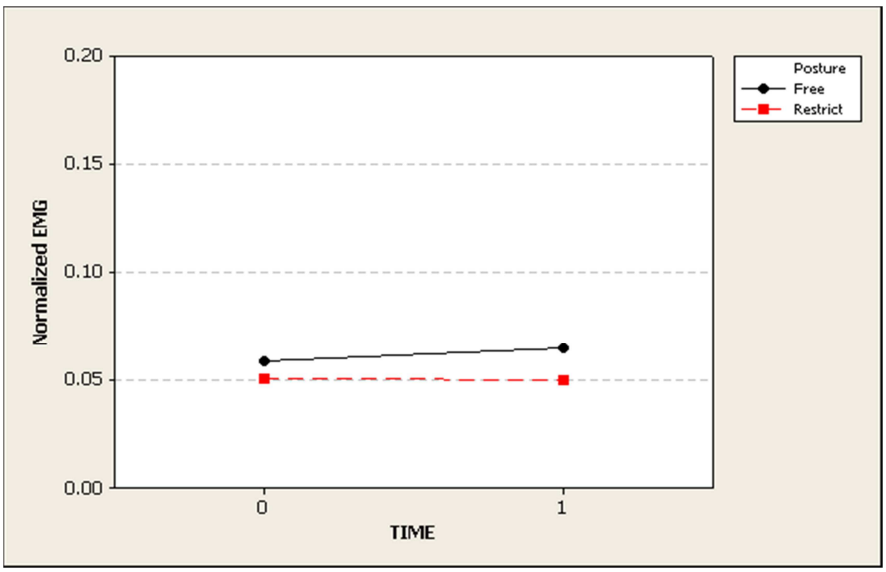

Figure 7.18 Interaction plot between POSTURE and TIME in all three protocols - Antagonist

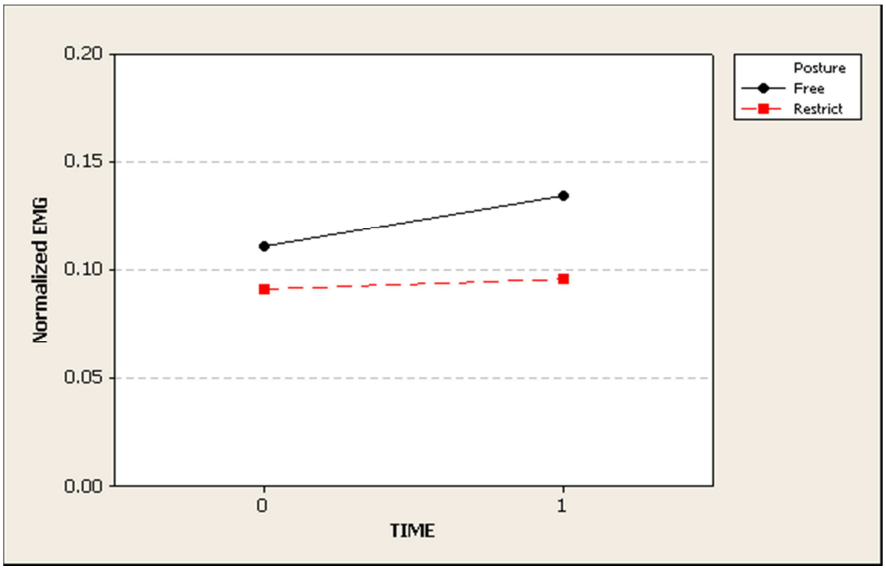

Figure 7.19 Interaction plot between POSTURE and TIME in all three protocols - Synergist

Additional analyses were conducted to reveal the different characteristics of the interactive effects between POSTURE and TIME in each protocol. First, the two-way ANOVAs for the Protocol A showed significant interactive effects on agonist and synergist (Table 7.8). The analysis of simple effects in agonist confirmed a significant effect of TIME in both free and restricted stooping conditions (p-value < 0.001 in both), suggesting the effect of 10-min passive tissue elongation protocol on agonist in both stooping conditions (3.1% increase in free and 1.9% increase in restricted). Also, simple effects sliced by TIME showed a significant effect of POSTURE in TIME 0 (p-value < 0.001), but POSTURE was not significant in TIME 1 (p-value = 0.634) (Figure 7.22). Regarding the synergist, simple effects revealed that there is no difference between TIME 0 and TIME 1 in the restricted stooping condition (p-value = 0.622; 0.3% increase) but showed a significant increase in the free stooping (p-value < 0.001; 2.7% increase) (Figure 7.24). Also, simple effects confirmed significant difference of POSTURE in both TIME 0 and 1 (p-value = 0.0463; p-value < 0.001). The result of two-way ANOVA on antagonist showed no interaction between TIME and POSTURE, but a significant difference was observed between free and restricted stooping conditions (Table 7.8 and Figure 7.23). However, the result was considered as biomechanically insignificant because of minor increase (1.2 %).

Table 7.8 Results of two-way ANOVA between posture and time for Protocol A

	Dependent variables		
Independent variables	Agonist	Antagonist	Synergist
Posture	F (1, 81) = 12.99, 0.001	F (1, 80) = 9.72, 0.003	F (1, 81) = 28.91, < 0.001
Time	F (1, 81) = 64.13, < 0.001	F (1, 80) = 1.84, 0.179	F (1, 81) = 10.34, 0.002
Posture × Time	F (1, 81) = 8.57, 0.004	F (1, 80) = 0.04, 0.837	F (1, 81) = 6.32, 0.014

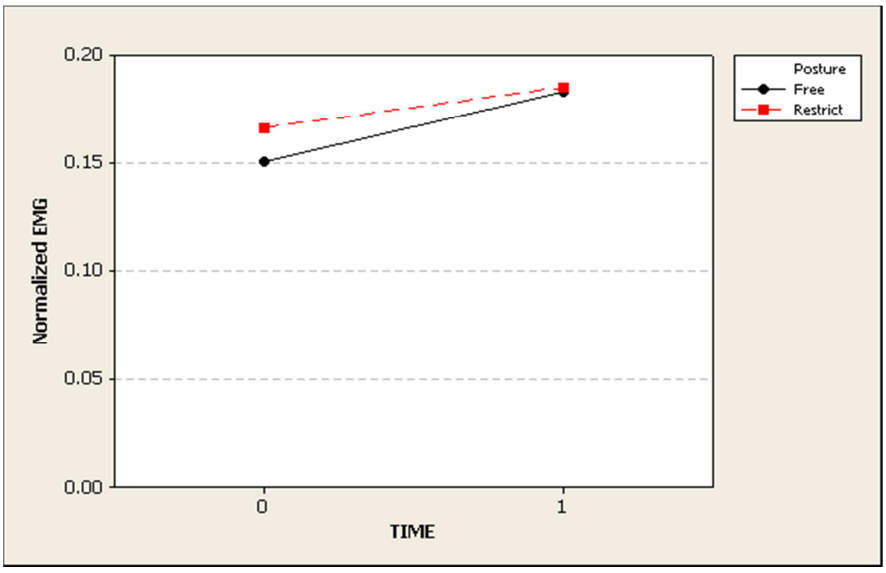

Figure 7.20 Interaction plot between POSTURE and TIME in the Protocol A - Agonist

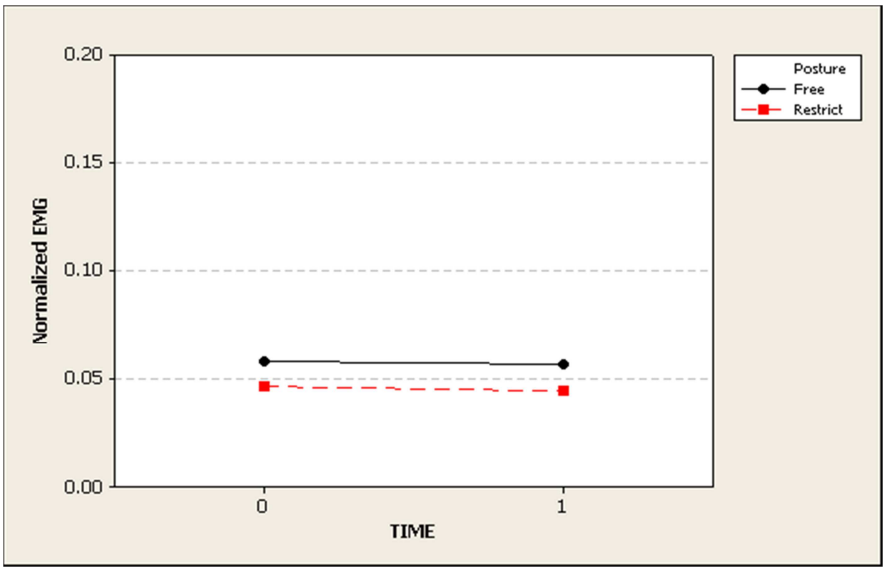

Figure 7.21 Interaction plot between POSTURE and TIME in the Protocol A - Antagonist

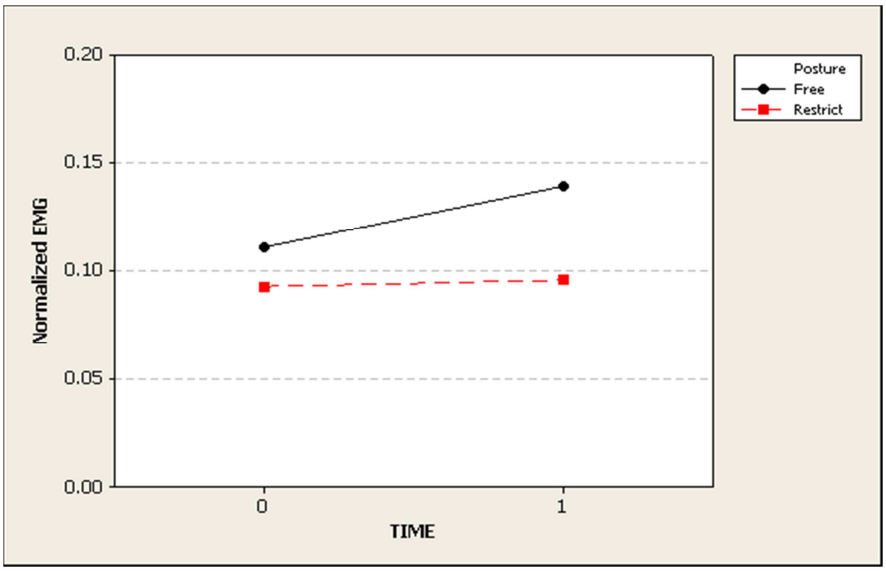

Figure 7.22 Interaction plot between POSTURE and TIME in the Protocol A - Synergist

Second, the two-way ANOVAs showed that the main effects and its interactions for Protocol B are broadly significant, except an interactive effect in synergist (Table 7.9). As all main effects and its interaction were statistically significant in the agonist, simple effects were investigated. Simple effects in agonist, sliced by the POSTURE, showed no significant difference between TIME 0 and TIME 1 in the free stooping condition (p-value = 0.378; 0.7% increase in TIME 1) but revealed a significant increase in the restricted stooping condition (p-value < 0.001; 3.8% increase in TIME 1) (Figure 7.25). The results suggested no effect of 10-min Protocol B in the free stooping but a significant effect of the protocol in the restricted stooping. Regarding the antagonist, the results also showed statistical significance in all main effects and its interaction (Table 7.9). Simple effects, sliced by the POSTURE, showed a significant difference between TIME 0 and TIME 1 in the free stooping condition (p-value < 0.001; 1.8% increase in TIME 1), but there was no difference in the restricted stooping condition (p-value = 0.863; 0.3% increase in TIME 1) (Figure 7.26). It may denote no effect of 10-min Protocol A in the restricted stooping but a significant effect of the protocol in the free stooping (opposite result with the agonist). Considering the main effects on synergist, the free stooping showed significantly higher NEMG than the restricted stooping (1.8%), and the TIME 1 showed a significant increase in NEMG from TIME 0 (1.2%) (Table 7.9). Even the antagonist showed significant main effects and its interaction, the synergist did not have a significant interaction because of a minor increase in NEMG of the free stooping from TIME 0 to TIME 1 (1.6%) (Table 7.10 and Figure 7.27).

Table 7.9 Results of two-way ANOVA between posture and time for Protocol B

	Dependent variables		
Independent variables	Agonist	Antagonist	Synergist
Posture	F (1, 80) = 20.87, < 0.001	F (1, 79) = 11.93, 0.001	F (1, 79) = 23.37, < 0.001
Time	F (1, 80) = 13.10, 0.001	F (1, 79) = 12.18, 0.001	F (1, 79) = 10.56, 0.002
Posture × Time	F (1, 80) = 5.63, 0.020	F (1, 79) = 10.52, 0.002	F (1, 79) = 0.81, 0.370

Table 7.10 Difference between TIME 0 and TIME 1 in each POSTURE (Bolded numbers show significant interaction)

PROTOCOL	POSTURE	Agonist	Antagonist	Synergist
Protocol A	Free (TIME 0 - TIME 1)	**0.031**	-0.001	**0.027**
	Restricted (TIME 0 - TIME 1)	**0.019**	-0.002	**0.003**
Protocol B	Free (TIME 0 - TIME 1)	**0.007**	**0.018**	0.016
	Restricted (TIME 0 - TIME 1)	**0.038**	**0.003**	0.007
Protocol C	Free (TIME 0 - TIME 1)	0.025	-0.016	0.017
	Restricted (TIME 0 - TIME 1)	0.031	0.000	0.003

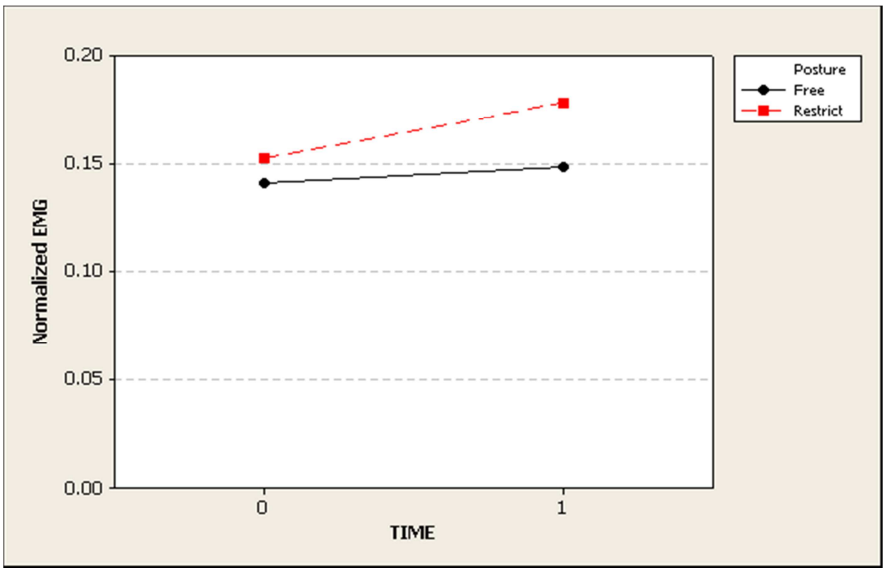

Figure 7.23 Interaction plot between POSTURE and TIME in the Protocol B - Agonist

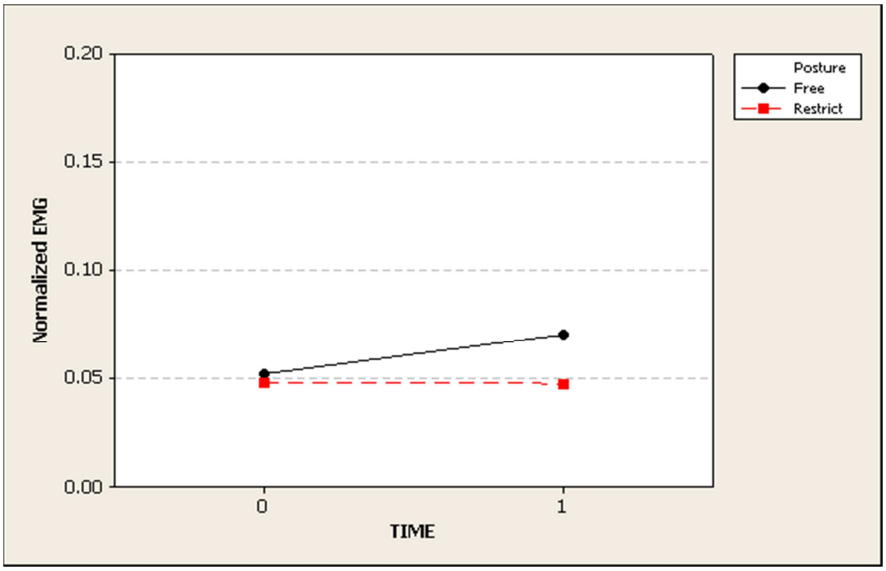

Figure 7.24 Interaction plot between POSTURE and TIME in the Protocol B - Antagonist

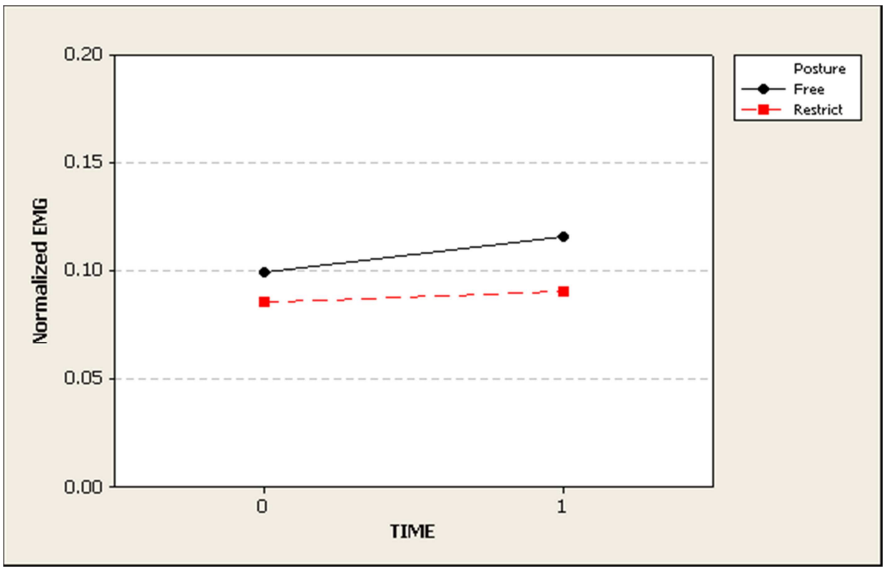

Figure 7.25 Interaction plot between POSTURE and TIME in the Protocol B - Synergist

Third, the two-way ANOVAs for Protocol C showed statistically significant main effects of POSTURE and TIME in agonist and synergist, but there was no significant interaction in any muscle groups (Table 7.11). That is because the agonist showed significant increase of NEMG from TIME 0 to TIME 1 in both free stooping (2.5%) and restricted stooping (3.1%), and the synergist did have no significant increase in NEMG (1.7% in free) and larger standard deviation (2.5 times bigger than Protocol B) (Figure 7.28 and 7.30). In regards of the antagonist, the result revealed no significant main effect and its interaction.

Table 7.11 Results of two-way ANOVA between posture and time for Protocol C

	Dependent variables		
Independent variables	Agonist	Antagonist	Synergist
Posture	F (1, 81) = 7.52, 0.007	F (1, 81) = 0.22, 0.637	F (1, 81) = 108.93, < 0.001
Time	F (1, 81) = 60.36, < 0.001	F (1, 81) = 0.00, 0.992	F (1, 81) = 7.57, 0.007
Posture × Time	F (1, 81) = 0.51, 0.479	F (1, 81) = 2.30, 0.133	F (1, 81) = 2.38, 0.127

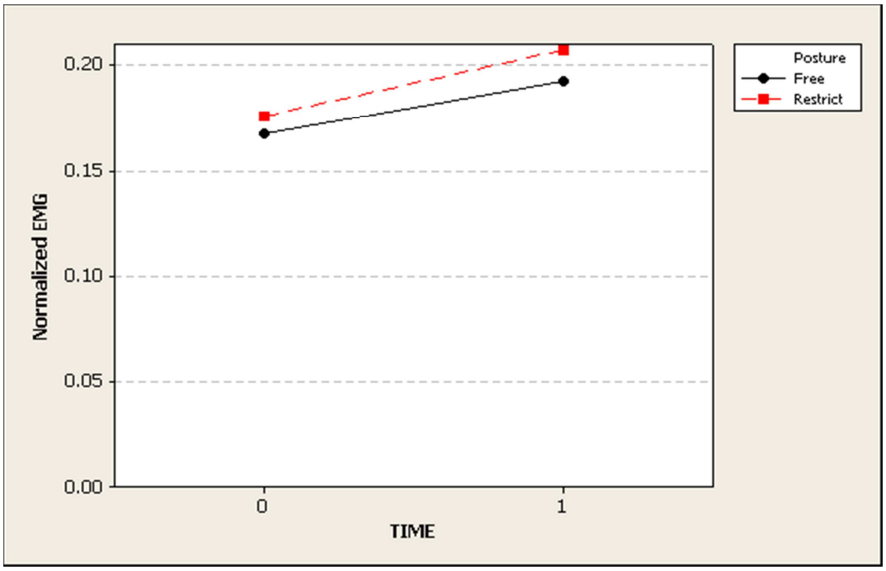

Figure 7.26 Interaction plot between POSTURE and TIME in the Protocol C - Agonist

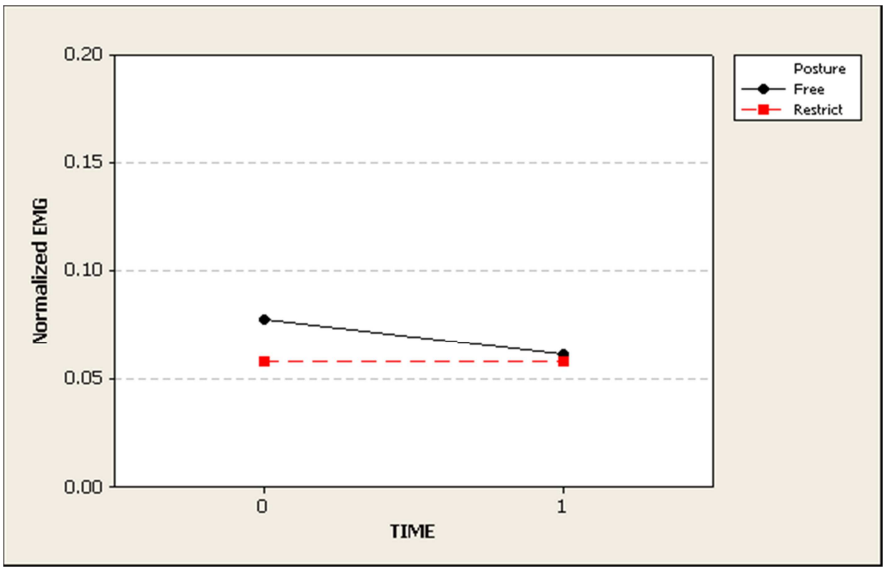

Figure 7.27 Interaction plot between POSTURE and TIME in the Protocol C - Antagonist

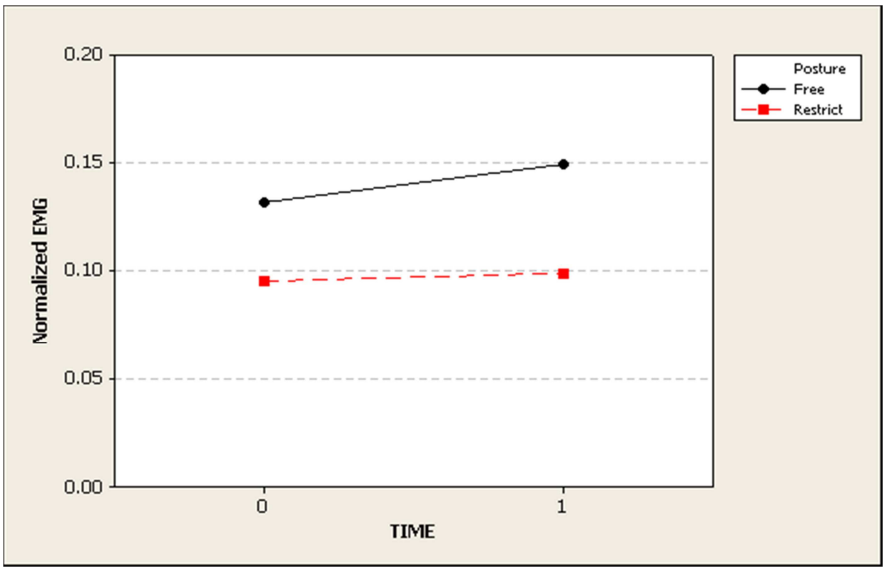

Figure 7.28 Interaction plot between POSTURE and TIME in the Protocol C - Synergist

7.4. Characteristics of the recovery phase

The characteristics of the recovery phase after the 10 minutes protocol were investigated throughout the statistical analyses and graphical analysis approach where each data point in the figure represents an average of standardized value of 12 subjects. Note that the data only included the free isokinetic trials to avoid the compounding effect of the stooping posture. In addition to this, the followings should be noted in this chapter: TIME 0 (baseline); TIME 1 (after 10 minutes protocol); TIME 2 (after 5 minutes resting); TIME 3 (after 10 minutes resting); TIME 4 (after 15 minutes resting); TIME 5 (after 20 minutes resting); TIME 6 (after 30 minutes resting); and TIME 7 (after 40 minutes resting).

7.4.1. Passive tissues elongation in low back

In line with the previous study, the peak lumbar flexion angle in Figure 7.29 showed considerable recovery during very earlier 5 minutes resting (TIME 2) (Shin and Mirka, 2007), but no remarkable recovery was observed after the 5 minutes resting (from TIME 3). The post-hoc tests showed no difference among TIME 3, 4, 5, 6 and 7 (p-value > 0.90), and they were significantly greater than the baseline (TIME 0) (p-value < 0.001). The results suggested a rapid, remarkable recovery in elongated tissues at very earlier phase of the resting session and no full recovery during 40 minutes resting period. Regarding the EMG-off points in multifidus and iliocostalis, both of them showed almost no recovery during 40 minutes resting session, even thought the full lumbar flexion angle was quickly recovered at TIME 2. The post-hoc tests also confirmed no difference among TIME 1, 2, 3, 4, 5, 6 and 7 (p-value > 0.40) but significant difference between TIME 0 and any others (p-value < 0.0001). The results denoted no clear recovery to the baseline during 40 minutes resting session.

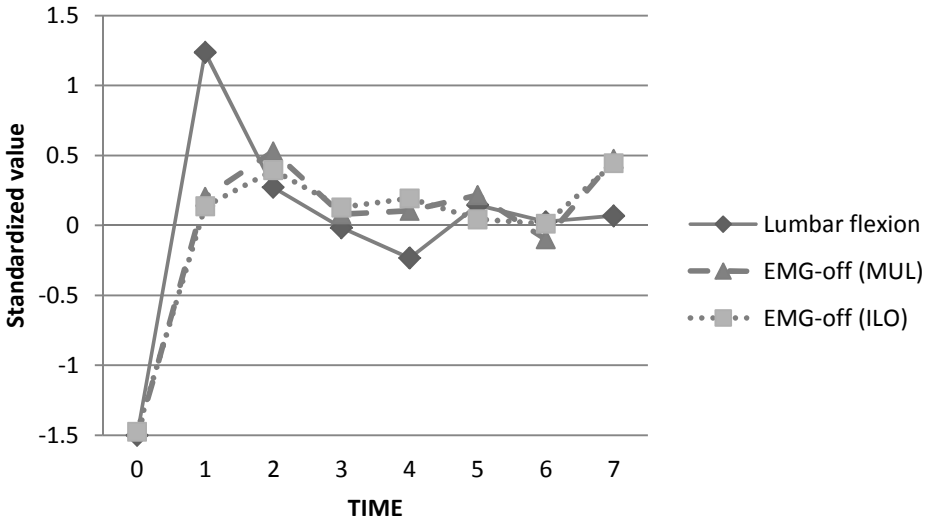

Figure 7.29 Recovery phase of lumbar flexion and EMG-off points after passive tissue elongation protocol

The muscle activities in agonist and synergist during isokinetic trunk flexion-extension showed a similar recovery phase with the peak lumbar flexion angle (Figure 7.30). Especially, both agonist and synergist revealed a considerable reduction (~ 8% in agonist and ~ 14% in synergist) in muscle activation level after 5 minutes resting (p-value < 0.05), but there were no remarkable changes in the following 35 minutes resting period (p-value > 0.60). Considering the isometric trials in 20 degree trunk flexion, the trend of recovery was generally similar with the isokinetic trials, but it was somewhat variable than the results captured at the isokinetic trunk flexion-extension (Figure 7.31).

In regards of the antagonist, no significant trend was expected during the recovery phase in that the antagonist showed no difference between TIME 0 and TIME 1 in both isometric and isokinetic trials (H4 and H7). However, the antagonist showed significant depression in its activation level (~ 33%) after five minutes resting (TIME 2), and it was lasted until at the end of

resting session (40 minutes) (TIME 1 vs. TIME 2, 3, 4, 5, 6 and 7: *p*-value < 0.001) (Figure 7.32). The recovery phase of the antagonist in both 35% isometric exertion and free isokinetic trunk flexion-extension was pretty similar, but the 35% isometric exertion was somewhat variable than the isokinetic trials in line with the results observed in the agonist and synergist.

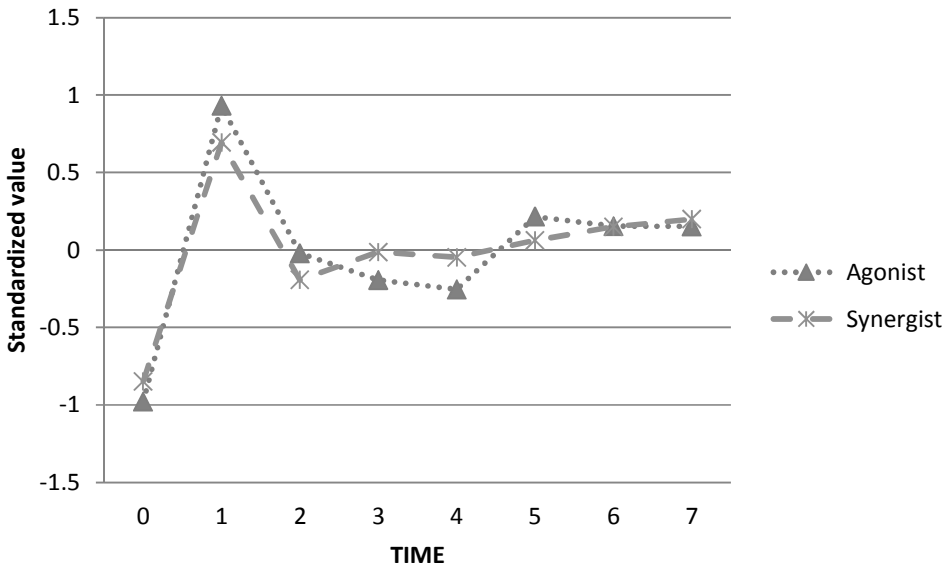

Figure 7.30 Recovery phase of agonist and synergist in isokinetic trials after passive tissue elongation protocol

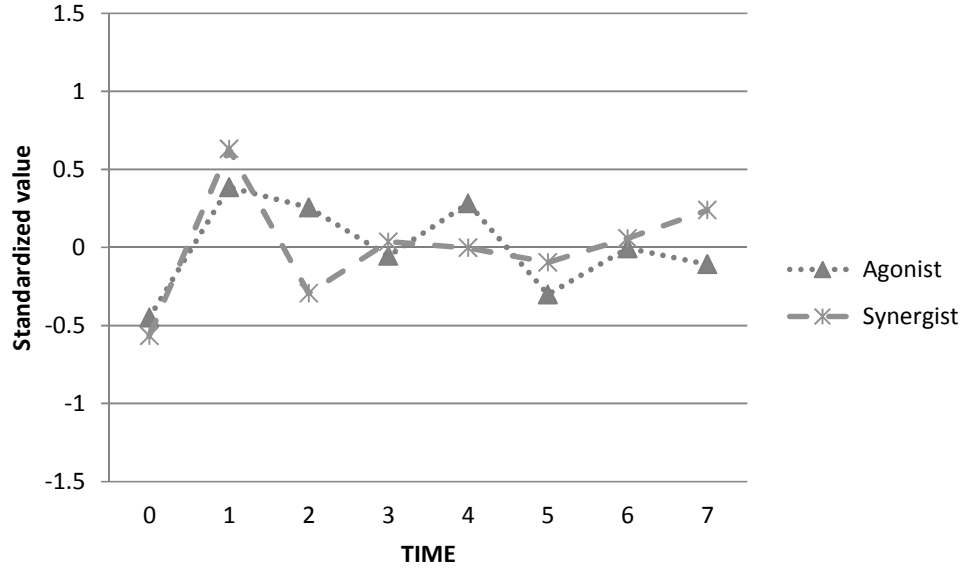

Figure 7.31 Recovery phase of agonist and synergist in isometric trials after passive tissue elongation protocol

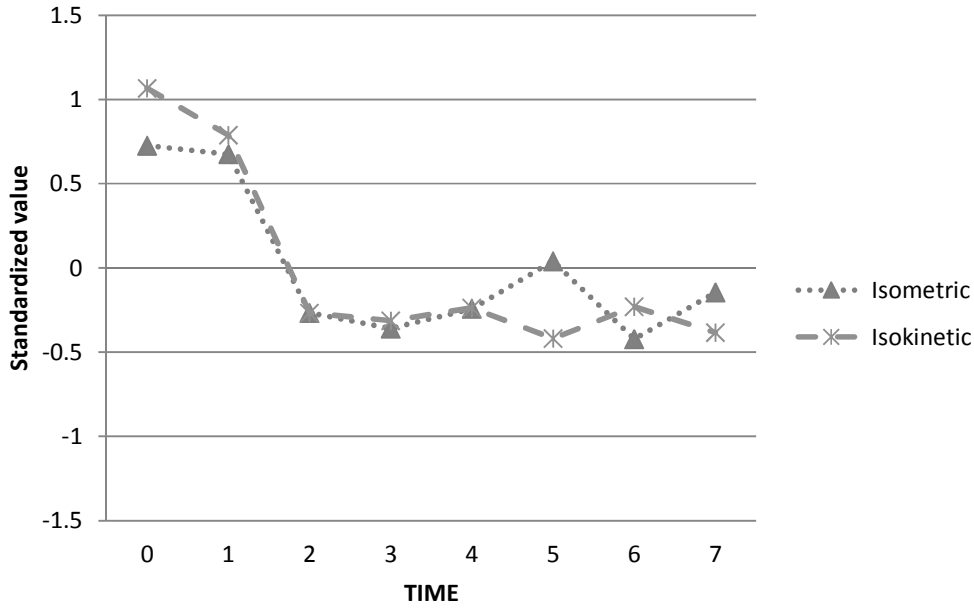

Figure 7.32 Recovery phase of antagonist in isometric and isokinetic trials after passive tissue elongation protocol

7.4.2. Muscle fatigue in low back

In line with the expectation, the EMG-off angles followed the trend of peak lumbar flexion angle, and they were decreased (less flexion) after the 10 minutes protocol at TIME 1 (Figure 7.33). However, there were no significant difference among eight levels of TIME in the EMG-off angles and lumbar flexion (p-value > 0.05).

Considering the recovery phase of the muscle activation in the isokinetic trials, the agonist did show no remarkable changes in all eight levels of TIME (p-value > 0.05) (Figure 7.34). However, the agonist activity in the 35% isometric exertion at 20 degree trunk flexion showed a significant effect of the muscle fatigue at TIME 1, 2 and 3 (until 15 minutes resting) (p-value < 0.01), and it was almost fully recovered at the end of resting session (40 minutes resting) (no difference between TIME 0 and TIME 7; t-value = 0.128).

In regards of the antagonist and synergist, both of them showed a significant effect of the muscle fatigue protocol at TIME 1 in the isokinetic trials (H7), but fully recovered right after the 5 minutes resting session (TIME 0 vs. TIME 2: p-value > 0.15) (Figure 7.34). Similarly, in the 35% isometric exertions, the antagonist was recovered after the 5 minutes resting (TIME 2), but the synergist showed the persistence of the protocol effect until TIME 2 (10 minutes resting) (Figure 7.35). The synergist was not fully recovered until at the end of the 40 minutes resting session, even though its activity from TIME 3 to TIME 7 did not show a statistically significant difference with the baseline (TIME 0) (p-value > 0.20) .

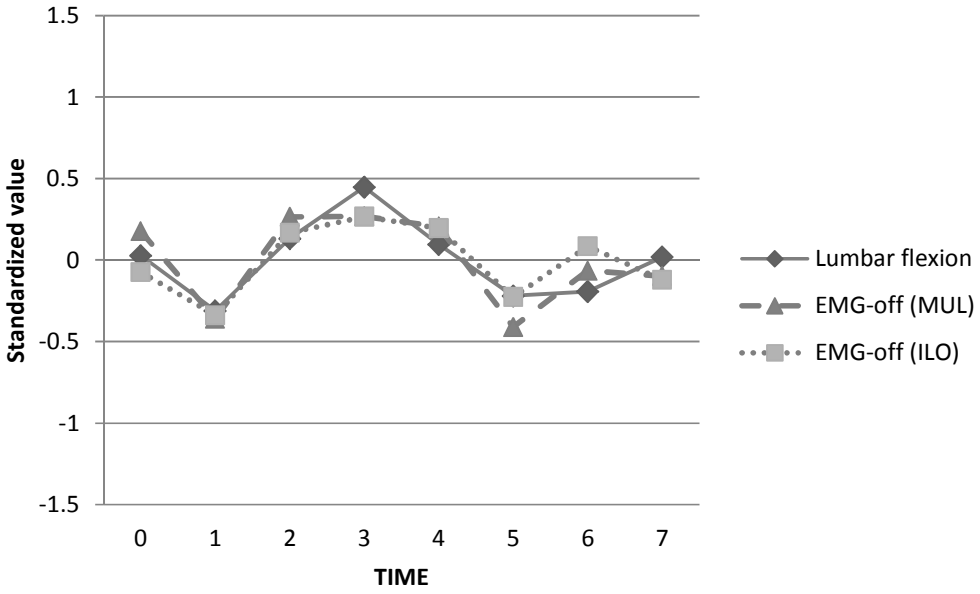

Figure 7.33 Recovery phase of lumbar flexion and EMG-off points after muscle fatigue protocol

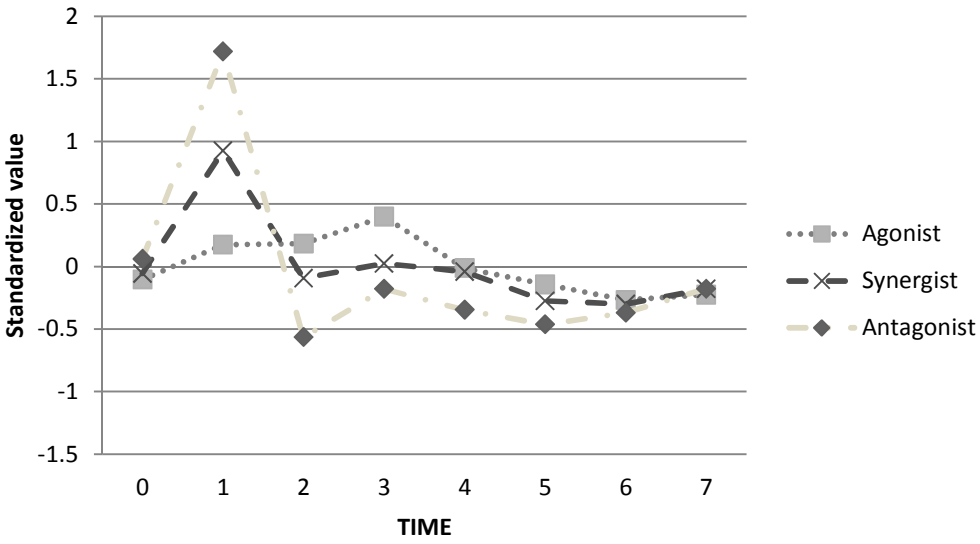

Figure 7.34 Recovery phase of agonist, antagonist and synergist in isokinetic trials after muscle fatigue protocol

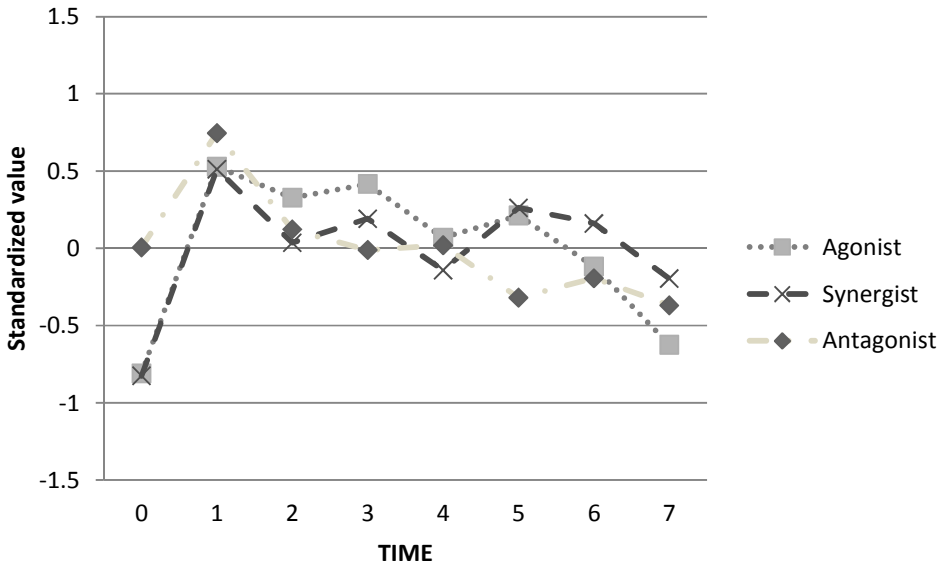

Figure 7.35 Recovery phase of agonist, antagonist and synergist in isometric trials after muscle fatigue protocol

7.4.3. Combined effect of muscle fatigue and passive tissue elongation in low back

The trend of recovery in the full lumbar flexion and EMG-off points after the combined effect protocol was similar with the passive tissue elongation protocol (Figure 7.36). The full lumbar flexion angle and EMG-off angles were not fully recovered until the end of resting session (TIME 0 vs. TIME 7: p-value < 0.05), but the combined protocol showed more clear recovery phase than the passive tissue elongation protocol; they showed a decreasing trend from TIME 2 (no significant).

In regards of the agonist during isokinetic trials, the effect of protocol was maintained from TIME 1 to TIME 3 (until 15 minutes resting) (TIME 0 vs. TIME 1, 2, 3: p-value < 0.01), and it was fully recovered after 40 minutes resting (TIME 0 vs. TIME 7: t-value = 0.038) (Figure

7.37). Meanwhile, the synergist showed a significant increase after the protocol at TIME 1 (H7) (p-value < 0.0001), but it was fully recovered after the 5 minutes resting (TIME 0 vs. TIME 2: t-value = 0.162). Considering the isometric trials in 20 degree trunk flexion, the trend of recovery was generally similar with the isokinetic trials, but it was somewhat variable than the results captured at the isokinetic trunk flexion-extension (Figure 7.38). The antagonist showed a similar recovery pattern with the result observed in the passive tissue elongation protocol, but there is no significant difference between conditions (Figure 7.39).

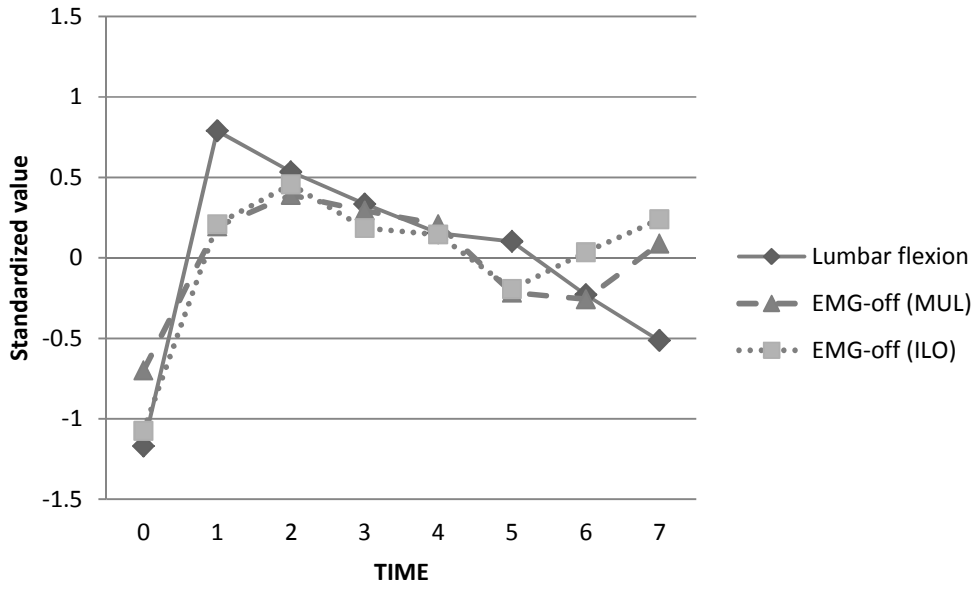

Figure 7.36 Recovery phase of lumbar flexion and EMG-off points after combined effect protocol

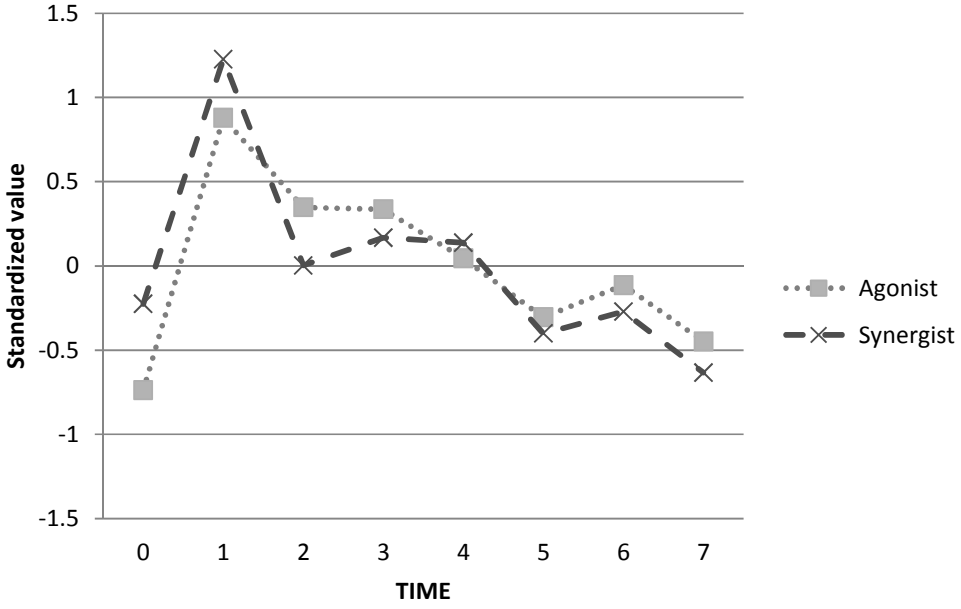

Figure 7.37 Recovery phase of agonist and synergist in isokinetic trials after combined effect protocol

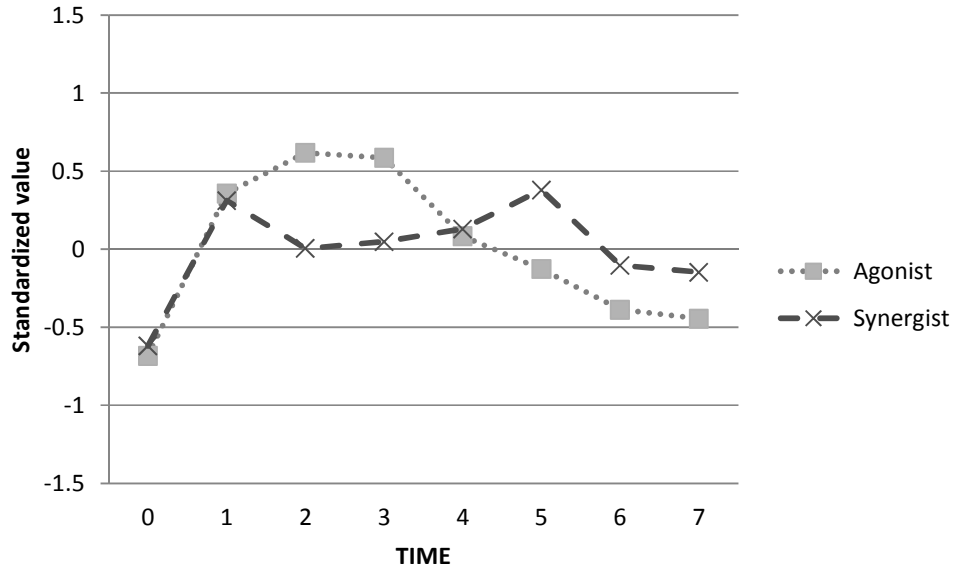

Figure 7.38 Recovery phase of agonist and synergist in isometric trials after combined effect protocol

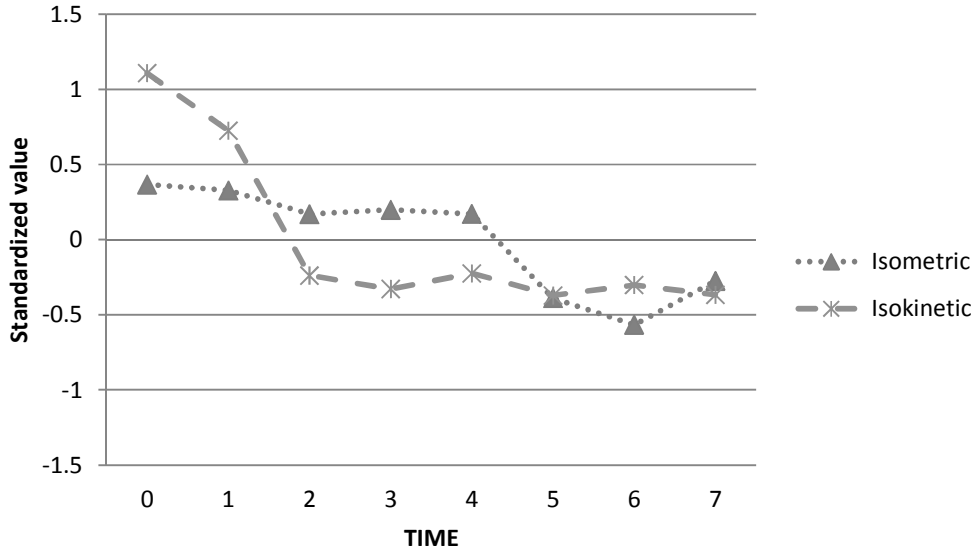

Figure 7.39 Recovery phase of antagonist in isometric and isokinetic trials after combined effect protocol

Chapter 8 – DISCUSSION

8.1. Comparison of three different abnormal low back conditions in FRP

One of the main goals of this study was to investigate the differences among flexion-relaxation responses in three different protocols, designed to generate laxity in low back viscoelastic tissues, to generate low back muscle fatigue, and to generate both laxity and fatigue in low back tissues. The effect of laxity in low back viscoelastic tissues on FRP are well understood from previous studies, but our knowledge about the influence of low back muscle fatigue on FRP is still far from complete. Also, there are no studies considering the combined effect of prolonged stooping and muscle fatigue in low back.

8.1.1. Alteration of full flexion angle and FR responses

Prior studies usually employed both EMG-off and -on points as dependent variables. However, considering that the trunk extension phase requires activation of the low back muscles to initiate trunk movement the EMG-off and -on points may show different characteristics in FRP. During the eccentric phase, the EMG-off points were significantly earlier than the full lumbar flexion angle in both free stooping (EMG-off in multifidus: 31.56°; EMG-off in iliocostalis: 30.53°; peak lumbar flexion: 36.50°) and the restricted stooping (EMG-off in multifidus: 34.91°; EMG-off in iliocostalis: 32.94°; peak lumbar flexion: 38.79°), showing that the external moment generated by the torso was carried completely by the passive mechanism from 5° (in lumbar flexion) prior to reaching the full flexion. On the other hand, during the concentric phase, there were no differences between the EMG-on points and the full lumbar flexion angle in either free stooping (EMG-on in multifidus: 36.66°; EMG-on in iliocostalis: 36.64°; peak lumbar flexion: 36.50°) or the restricted stooping (EMG-on in multifidus: 38.71°;

EMG-on in iliocostalis: 38.72°; peak lumbar flexion: 38.79°), showing that the low back muscles were simultaneously activated with the initiation of the trunk extension motion. Hence, the results lead to the conclusion that there is no turn-off of the active mechanism during the concentric motion, even when the weaker load-sharing between passive and active systems is still working.

Regarding the active system (muscle activity), previous studies revealed that the muscular activation level in eccentric contraction motion is smaller than the concentric contraction motion under the same level of force generation (Tesch et al., 1990, Huang and Thorstensson, 2000). Tesch et al. (1990) showed that the ratio of EMG/moment is significantly smaller in the eccentric contraction motion than the concentric contraction motion, suggesting that the eccentric contraction motion necessarily requires an additional source of force generation to provide a similar level of force with concentric contraction. In this study, they hypothesized that the passive viscoelastic tissues can produce additional force to meet the same level of net force with the concentric contraction. Regarding the force generation capacity, the eccentric contraction motion showed greater force-generation capacity compared to the concentric contraction in maximum voluntary contraction (Doss and Karpovich, 1965). The greater force generation could be attributable to the elastic force generated by stretched tissues (McCully and Faulkner, 1985). All together, the eccentric contraction motion can more effectively make use of the passive mechanism when compared to the concentric contraction motion.

Prolonged stooping – Protocol A

The effect of the passive tissue elongation protocol (Protocol A) on FRP revealed a deeper peak flexion angle (2.57°) and EMG-off and -on angles (2.4° in average), supporting the

findings of previous studies (Rogers and Granata, 2006; Shin & Mirka, 2006; Solomonow et al., 2003a; Shin et al., 2009) (Hypothesis 1). The prolonged static flexion may elicit a higher strain on lumbar passive tissues such as interspinous and supraspinous ligaments (Panjabi et al., 1981) and elongate the passive tissues (Figure 8.1). Consequently, the laxity in passive tissues could shift the passive equilibrium point between the external moment (torso) and viscoelastic tissues (ligament) to deeper lumbar flexion posture because of the laxity in the ligaments (which have reduced moment-generation capacity compared to before the prolonged static stooping at the same level of lumbar flexion). On this basis, deeper lumbar angles could be required to account for the external moment for producing enough tension on the ligaments as a simple compensation for the loss of tension in lumbar ligaments during FRP. Moreover, , it is important to note that the laxity in the viscoelastic tissues denotes spinal instability in that the loosened ligaments around the spinal column could lose the ability to hold or stabilize the structure (Solomonow et al., 2003c).

Prior studies also showed negative modulations after prolonged stooping such as depression and delay in spinal reflex, suggesting impaired reflex after prolonged stooping and inability to respond quickly to a sudden external loading (Moorhouse and Granata 2007; Sánchez-Zuriaga et al., 2010). When all factors are considered, the shift in FRP after the passive tissue elongation caused by prolonged stooping is a significant signal of temporary spinal instability.

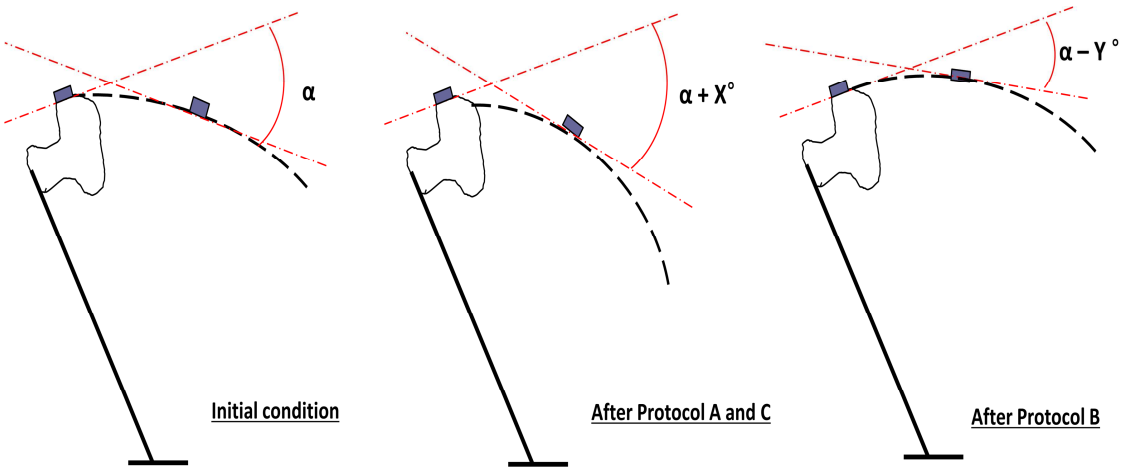

Figure 8.1 Conceptual model representing the effect of three protocols

Low back muscle fatigue – Protocol B

The negative impacts of muscular fatigue in the low back have been largely explored, and the spinal instability and modulation of the FRP suggested (Granata et al., 2004; Descarreaux et al., 2008; Granata and Gottipati, 2008). In line with the findings of Descarreaux et al. (2008), the EMG-off and -on lumbar flexion angles were decreased (i.e., less flexion) after a 10-min protocol designed to cause low back muscle fatigue (i.e., the opposite effect when compared to the passive tissue stretching protocol) (Hypothesis 2). In addition, the peak lumbar flexion angle was investigated for better understanding of the modulation in the FRP after the muscle fatigue condition; the full lumbar flexion angle is highly correlated with the measures of FRP such as EMG-off and EMG-on angles and shows consistency in the property of the passive lumbar tissues. Descarreaux et al. (2008) did not investigate the peak lumbar flexion angle (Figure 8.1). As discussed earlier, the current study contradicts the suggestion of Descarreaux and colleagues that "the low back muscles, in a state of fatigue, are not able to provide sufficient stabilization to the vertebral units, transferring load-sharing to passive structures earlier in trunk flexion"(2008).

The authors suggested that the presence of low back muscle fatigue limits the force generation capacity of the muscles providing stabilization to the spinal column, so the fresh passive tissues are charged earlier to compensate for the decreased force-generation ability of the low back muscles. Given the fact that the FRP only occurs at the biomechanical equilibrium point between passive moment and external moment generated by the torso, the hypothesis proposed by Descarreaux et al. (2008) is difficult to reconcile. In other words, the occurrence of FR cannot be modified by the reduced force-generation capacity of the fatigued muscles. The passive tissues are only "passively" activated at a specific length (i.e., at a specific angle) if there is no change in the viscoelastic properties of the passive tissues; the earlier FR (i.e., transferring load-sharing to passive tissues earlier) can only occur when the passive tissues generate the required tension earlier than in the normal condition.

To support the new hypothesis, a smaller full-flexion angle was expected to explain the earlier transference to passive tissues, based on previous studies showing increased passive elastic tension after both isometric fatiguing protocol and eccentric contraction protocol in the triceps surae and right calf muscles (Whitehead et al., 2001; Finlayson et al., 2008). The increased tension could be explained as immediate strain injury contractures, referring to a contraction of the fiber in the absence of an action potential, in damaged muscle fibers after the muscle fatigue protocol. It is possible that the strain contracture in low back muscles results in the reduced muscle length and increased passive tension. Reduced low back muscle length was observed earlier by Parnianpour et al. (1988) showing a significantly smaller peak lumbar flexion angle after the muscle fatigue protocol. In line with this, the result of the current empirical study supported this new hypothesis by revealing a reduced peak lumbar flexion angle after the muscle fatigue protocol, suggesting that the earlier FRP (i.e., EMG-off and -on points) are highly related

with the strain injury contracture of the fatigued muscles which reduces the peak lumbar flexion angle. In summary, empirical evidence and previous study results showed how the inability of the local muscle system modulates the flexion-relaxation phenomenon from a biomechanical perspective, and the earlier occurrence of FRP could be a good signal of an abnormal low back condition caused by inability in the local system, such as the strain injury contractures.

Combination of laxity and fatigue in low back tissues – Protocol C

Hypothesis 3 aimed to reveal the combined effect of laxity in passive tissues and fatigue in low back muscles. No previous study tested the combined condition, even though the individual effects of muscle fatigue and passive tissue elongation on FRP have been studied.

Contrary to the hypothesis, the results showed an increase in peak lumbar flexion angle and EMG-off and -on points after the protocol (like Protocol A, viscoelastic tissue elongation), but the magnitude of the change was smaller than in Protocol A (Figure 8.1). No significant change in the peak lumbar flexion angle and the occurrence points of FR was expected because of the fair counteraction (i.e., equal contribution) between individual effects of muscle fatigue and passive tissue stretching. However, the result suggested that the elongation or deformation of passive tissues in low back have a more dominant influence on the FRP than the active low back muscle fatigue. It is not clear that Protocol C's smaller change in EMG-off and -on points (0.86° less than Protocol A) denotes the contribution of low back muscle fatigue which is confirmed by a shift in median power frequency in this protocol, because it was confounded with the factor that the full, passive stooping posture in Protocol C was exactly half of the amount of time in Protocol A. It is plausible that the reduced amount of passive stooping time during Protocol C results in less change in both peak lumbar flexion angle and EMG-off and -

on points; the amount of change of EMG-off and -on points in Protocol C was 64% of that in Protocol A on average.

Considering the fact that the EMG-off point is the biomechanical equilibrium point between passive tissues and external moment generated by the torso, the results revealing a dominant contribution of passive tissue elongation in Protocol C may suggest a significant role of the passive structures in trunk flexion-extension, especially around the full flexion. Actually, the result is not surprising regarding the basic concept of FRP – that is, the transfer of the role of extension moment generator from the active mechanism (muscle tissue) to the passive mechanism (ligaments, discs and fascia) over a 60° trunk flexion. Because of the significant role of passive tissues around full flexion, the FRP in Protocol C could follow the result of Protocol A under the condition of both laxity in passive tissues and muscle fatigue in low back confirmed by deeper peak lumbar flexion and shift in median power frequency of multifidus and iliocostalis.

As already demonstrated and discussed in previous sections, the passive tissue elongation resulted in deeper FRP points and the muscle fatigue resulted in earlier FRP points. Even the direction of modulation in FRP points from the normal condition was different in two abnormal low back conditions; both the passive tissue elongation and muscle fatigue in the low back suggested spinal instability for different reasons. Considering that Protocol C showed both laxity in passive tissues and muscle fatigue and revealed similar modulation in FRP with Protocol A, spinal instability could be expected after the Protocol C like the Protocol A and B.

8.1.2. Muscle recruitment pattern in isometric exertions

The agonist muscle activity

Regarding the effect of the protocols on muscle activity in low back, the NEMG in agonist muscles (i.e., multifidus and iliocostalis) during the isometric exertion was significantly increased after all three 10-min protocols. First, a significant increase after Protocol A may suggest that the low back system requires more muscle activation to compensate for the reduced tension-generation capacity in passive tissues and the resulting reduced spinal stability caused by laxity in passive tissues (Shin and Mirka, 2006; Shin et al., 2009). To keep the spinal stability, the low back muscles may be required to increase their force generation capacity at the given motor unit, and hence increase the EMG activity. In addition to this, Shin et al. (2009) proposed that the fatigue-like response in lumbar muscles is due to the passive stretching of muscles. However, there is no sign of muscle fatigue (no shift in median frequency) after the passive tissue elongation protocol of this study. Further research is required to confirm the fatigue-like response of the stretched low back muscles.

Second, a significant increase in agonist activity after the low back muscle fatigue (Protocol B) could be attributable to the reduced force-generation capacity of the preselected motor unit, and hence recruitment of additional motor units (Bonato et al., 2003; Clark et al., 2003). To keep the same level of extension moment during a 35% isometric exertion, the fatigued muscle in low back could be required to recruit more motor units, resulting in an increase in EMG amplitude.

Third, Protocol C also showed a significant increase in agonist muscle activity after a 10-minute protocol. It could be attributable to the decreased force-generation ability in the active mechanism (muscles) caused by muscle fatigue and the reduced tension-generation capacity in

passive mechanisms (ligaments, discs and fascia) caused by passive tissue elongation. A greater increase in agonist muscle activity after Protocol C compared with Protocol A and B was expected, but there was no significant difference among protocols. Even though there was no statistical difference among protocols, Protocol B showed a greater increase than Protocols A and C (4.2% in Protocol B, 2.7% in Protocol A and 3.4% in Protocol C). Considering the trunk posture during the 35% isometric exertion trial was almost upright (20 degree trunk flexion from the standing posture), the role of the passive mechanism in low back may be minimal, but the active mechanism in low back may be significantly engaged. Consequently, the temporary dysfunction in low back muscles caused after the 10-minute muscle fatigue protocol could cause a greater increase in NEMG than the 10-minute passive tissue elongation protocol, even though it is not significantly different. No reduced ability in the active mechanism (no muscle fatigue) in Protocol A could result in the lowest increase (64% of the Protocol B).

The antagonist muscle activity

The antagonist showed no increase in Protocols A and C after the 10-min protocol, but Protocol B revealed a significant increase in antagonist activity after the 10-min muscle fatigue protocol. The result could be related to a greater increase in agonist activity after Protocol B. It is well known that the antagonist muscles can be considered trunk stabilizers by co-contracting with the agonist and increasing the biomechanical stability (Cholewicki et al., 1998; Gardner-Morse and Strokes, 1998; Granata and Marras, 2000), suggesting a significant role of the antagonist in an underlying trunk control mechanism. Even though the activation of antagonist generates flexion moment during the 35% isometric extension exertion, a significant co-contraction in antagonist muscles may be required after Protocol B to enhance trunk stability. A

larger moment arm of the antagonist could significantly increase the compression force on L5/S1 and also require greater agonist muscle activation to generate the same level of force. Meanwhile, there was no difference among protocols in the increase rate after 10-min protocols.

The synergist muscle activity

The subjects tended to increase the activation level of the synergist muscles, including gluteus maximus and biceps femoris, after all three protocols to perform the 35% isometric trunk extension exertion. It may suggest that the trunk system is not a simple unit as the previous biomechanical models have described, in which the role of lower extremity was not considered for simplicity and ease of the model. Plenty of evidence showed both a direct and indirect connection between the torso and lower extremities and suggested a possibility to consider a system-level investigation (Snijders et al., 1993b; Vleeming et al., 1995). For example, the lower extremity muscles can directly and indirectly increase the tension of the lumbodorsal fascia (or thoracolumbar fascia) that is a deep investing membrane with three layers. The gluteus maximus is coupled with trunk muscles via the lumbodorsal fascia extending from the iliac crest and sacrum to the thoracic cage (i.e., generating direct tension), and the lamina is tensed by contraction of the trunk and hip extensors (i.e., generating indirect tension) (Vleeming et al., 1995). The anatomically integrated trunk motion system may effectively transfer load, and the muscles may interact with each other to enhance the spinal stability and increase the extension moment. Based on these biomechanical observations, the results could be attributable to the synergistic activation of the 'super global muscles,' including gluteus maximus and biceps femoris, for assisting extension moment generation and spinal stability.

In line with the result of the agonist, the synergist showed a significant increase in Protocol B as compared to Protocols A and C. The synergist revealed 2.5 times greater increase in NEMG after Protocol B than Protocols A and C. The result supports our discussion in the previous section that the temporary dysfunction of the active mechanism in low back could result in greater muscle activity than the disability in the passive mechanism because of the significant engagement with the active mechanism during almost-upright 35% static exertions.

8.2. Evaluation of trunk flexion-extension in a system-level perspective

8.2.1. The role of lower extremity in trunk flexion-extension: normal condition

In line with Hypothesis 5, the result showed significantly greater trunk flexion and hip flexion in the free stooping technique and significantly larger lumbar flexion and thoracic flexion in the restricted stooping technique. The major source of greater trunk flexion in the free stooping technique could be the larger pelvic rotation (hip flexion) in the posture, showing a significant contribution of pelvic rotation in trunk flexion-extension. Meanwhile, it is interesting to observe that the difference between the two stooping techniques was greater in hip flexion than trunk flexion (about 4.7°), suggesting less flexion in lumbar and thoracic vertebra in free stooping (Figure 7.11). In line with that suggestion, the result of lumbar flexion angle and thoracic flexion angle revealed less flexion in lumbar and thoracic vertebrae (i.e., less curvature) in the free stooping posture as compared to the restricted posture. Considering that the main difference between the two postures was existence of the lumbopelvic rhythm (lumbar-pelvis interaction) and the pelvifemoral rhythm (pelvis-femur interaction), the lower extremity kinematics could be a significant factor influencing the lumbar curvature. The lower extremity

kinematics may have an effect on the low back biomechanics through the following two mechanisms.

First, the lower extremity kinematics may exert influence on the passive moment generated by the lumbodorsal fascia. Deprivation of the lumbopelvic rhythm in restricted stooping could reduce the passive tension of the lumbodorsal fascia, mainly generated by the super global system (gluteus maximus and hamstring muscles) and consequently require deeper lumbar flexion to increase the passive moment and meet the biomechanical equilibrium point between the external moment and the passive system of the low back (Figure 8.2). Vleeming et al. (1995) revealed that the thickest posterior layer of the fascia is stretched and stiffened by contraction of the gluteus maximus and the anterior and middle layers are tensed by the exertion of biceps femoris. The current study designed and showed a condition of the lumbopelvic rhythm deprivation (H5), and significantly less recruitment of the super global system (e.g., synergist muscles) was shown in the restricted isometric trunk flexion-extension (H6). Again, decreased activation of the super global system may reduce the tension of the lumbodorsal fascia which contributes the passive moment around the full flexion. In these bases, more flexion of the lumbar vertebrae may be required in the restricted stooping condition to compensate for the less passive tension of the lumbodorsal fascia; greater lumbar flexion could result in an increase of the passive moment generated by the viscoelastic tissues in the low back, such as supraspinous and interspinal ligaments.

The result of the muscle recruitment pattern in the agonist (e.g., multifidus and iliocostalis) also revealed the role of the lumbodorsal fascia (H6). During the isometric trunk flexion-extension, the agonist showed a greater muscle activation level in the restricted stooping as compared to the free stooping. The activation level could be attributable to the smaller

extension moment generation capacity of the passive mechanism around the full flexion (80% flexion ~ 20% extension) in the restricted stooping, specifically less tension in the lumbodorsal fascia. The EMG-off angles in both multifidus and iliocostalis revealed a significantly late initiation of FRP in restricted stooping to compensate for the reduced passive tension by prolonging the active mechanism. Based on this, significantly less agonist activity in the free stooping could be attributable to the earlier transition to the passive mechanism in the free stooping posture as compared to the restricted stooping posture; note that the silence period (FRP) was not included in the data analysis.

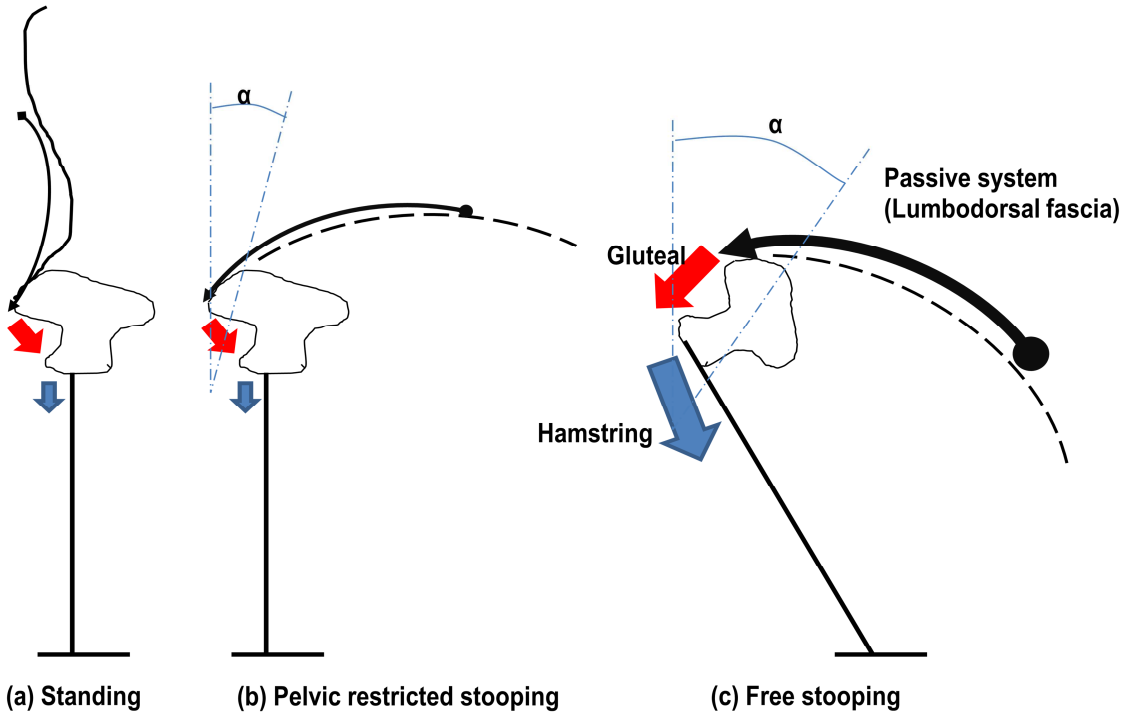

Figure 8.2 Conceptual model representing the role of the super global system

Second, the lower extremity kinematics may also influence the passive moment generated by the low back muscles. The lower extremity muscles, largely attached to the pelvis (e.g., gluteus maximus, biceps femoris, semitendinosus and semimembranosus), have indirect connections to the low back muscles, rising from the iliac crest and inserted into the vertebrae (e.g., multifidus, longissimus lumborum, iliocostalis lumborum), throughout the pelvis, so the activation of the lower extremity muscles can pull the pelvis backward and simultaneously increase the passive tension of the low back muscles around full trunk flexion. Olson et al. (2006) showed that the hamstring muscles are fully stretched around the full flexion and suggested passive pulling tension generated by the muscles on the pelvis. They also showed that the biceps femoris and semimembranosis have similar activation patterns with the low back muscle (i.e., FRP-like responses in the lower extremity muscles). Considering the length-tension relationship of muscle (Figure 8.3), denoting a decrease in active force and an increase in passive force when fully stretched, the backward migration of the buttock and increased passive pulling force on the pelvis could increase the passive moments generated by the low back muscle on the lumbar vertebrae.

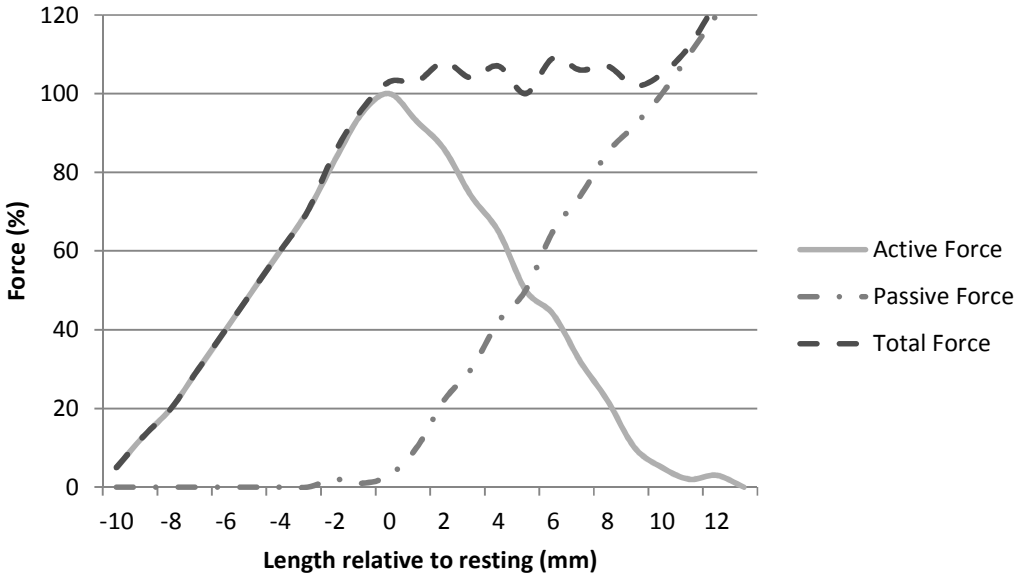

Figure 8.3 Muscle length-tension relationship

Prior studies revealed later initiation of FRP during flexion (i.e., greater lumbar flexion) with an increased external load (e.g., hand-held load) (Kippers and Parker, 1984; Dickey et al., 2003). It is clear that the later FR occurs at the biomechanical equilibrium point accounting for increased external moment between passive tissues and external moment. These studies were different from the current study because there is no difference in the external moment between the two stooping postures. Gupta (2001) conducted a study that is relevant to the current study in which the external loads were placed on the subject's hand (anterior load) or subject's back (posterior load: tied around the pelvis). The results showed that both anterior and posterior loads delay the initiation angle of FRP. He explained the results as follows: "Addition of weights, whether anterior or posterior to the hip axis produce increased tensile torque. This requires the balancing act of the erector spinae to continue longer till the extension torque by the posterior vertebral ligaments increased proportionally enough at greater vertebral flexion" (Gupta, 2001).

However, it seems unreasonable to believe that the posterior load tied around the pelvis generates the extension torque on the vertebrae. The hand-held load can increase the extension torque on the vertebrae when the pelvis is considered a rigid body of the trunk movement and provides the skeletal foundation for the vertebrae. On the other hand, the load tied around the hip may just transmit the additional force to the ground throughout the biomechanical linkage of the lower extremity (Figure 8.4). In this posture (Figure 8.4 (B)), the load could be employed to keep the body balance during trunk flexion, so the posterior migration of the pelvis controlled by the hip extensors does not happen. It means that there is less pulling force generated by the lower extremity muscles on the pelvis and the lumbodorsal fascia which provide passive moment of low back. It is essential to realize that the backward migration of the buttock and activation of the lower extremity muscles provide the counter moment to keep the balance of the human body and hence the mechanism increases the passive moment in trunk flexion-extension. All together, the decreased tension generation caused by deprivation of the lumbopelvic rhythm and pelvifemoral rhythm (e.g., no backward migration of buttock) requires increased vertebrae flexion to meet the biomechanical equilibrium between passive and active tissues.

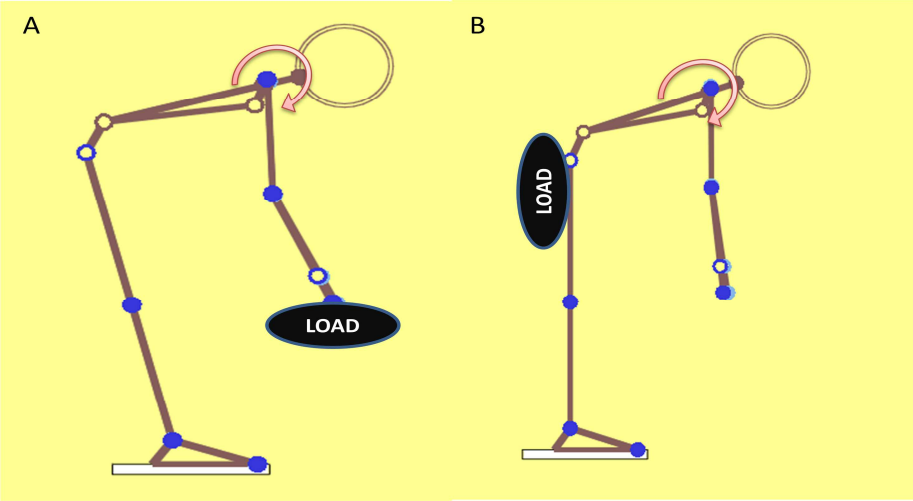

Figure 8.4 The external load locations during trunk flexion-extension in Gupta (2001)

8.2.2. The role of the lower extremity in trunk flexion-extension: abnormal condition

For a better understanding of the role of lower extremity in trunk flexion-extension around full flexion angle (80% flexion ~ 20% extension), the muscle activation pattern of local, global and super global systems were investigated under the abnormal low back conditions including laxity in low back viscoelastic tissues, low back muscle fatigue and both laxity and fatigue in low back tissues. Remember that the full flexion angle of Protocol B (muscle fatigue protocol) at TIME 1 was regarded as the full flexion angle to calculate the percentage of flexion employed to match each trial for a fair comparison.

Protocol A – viscoelastic tissue elongation

In line with the results in Hypothesis 4 (35% isometric trials at 20° trunk flexion), the agonist and synergist muscle activities increased significantly after the viscoelastic tissue elongation protocols. Regarding the interaction effect, the agonist showed a significant increase in both free and restricted stooping techniques after the protocol (effect of TIME), but the

significant difference between two postures observed in TIME 0 was not observed in TIME 1 (effect of POSTURE). This effect means that the incremental rate of agonist muscle activity in the free stooping was significantly larger than the restricted stooping after Protocol A. Recalling the ball example, it could be attributable to the difference in the foundation of the two postures such as the pre-stabilized base (i.e., pelvis) in the restricted stooping posture and the mobile base in the free stooping posture. It is already well described and discussed in a literature review (see Chapter 2.4.4.) that the foundation (pelvis) should be stabilized enough in advance of achieving the spinal stabilization; otherwise spinal stability could not be successfully achieved. For an example, the ball (e.g., spinal column) upon the unstable bowl (i.e., pelvis) (Figure 8.5) cannot reach the stable position unless the bowl is stabilized. In addition, the unsteady bowl could aggravate the disturbance of the ball. The unstable spinal column (i.e., disturbed ball) in free stooping after the protocol (Granata et al., 2005; Rogers and Granata, 2006; Shin and Mirka, 2007; Solomonow et al., 2003a, 2003c) may require a greater increase in the agonist to stabilize the low back system to compensate for the less stable foundation in the free stooping technique than the restricted stooping technique.

The ball example showed that the trunk stability and the pelvic stability should be achieved simultaneously for guaranteeing spinal stability. The synergist's result (super global system) supports the hypothesis. The synergist showed a significant increase in free stooping and no difference in the restricted stooping after the protocol. The free stooping showed significantly greater synergist activation in both TIME 0 and TIME 1 than the restricted stooping, but the difference between the two postures were 2.3 times greater in TIME 1 (4.2%) than TIME 0 (1.8%). The results suggested the increased role of the synergist as a pelvic stabilizer under the condition of spinal instability (i.e., after low back passive tissue elongation

protocol) for providing a stable foundation and a passive torque. The evidence showing the significant role of the super global system as a preliminary stabilizer of the spinal system (e.g., stabilize pelvis) was also revealed in a previous study. Kankaanpää et al. (1998) showed that the gluteus maximus of low back patients fatigue faster than the normal participants, even when there is no difference in the fatigue of the erector spine muscles. The result may suggest the increased role of the super global system in low back patients to provide a stable foundation when the ball (spinal column) is unstable because of inability in the low back system. Altogether, it is important to recognize the role of the super global system to achieve pelvic stability and hence spinal stability.

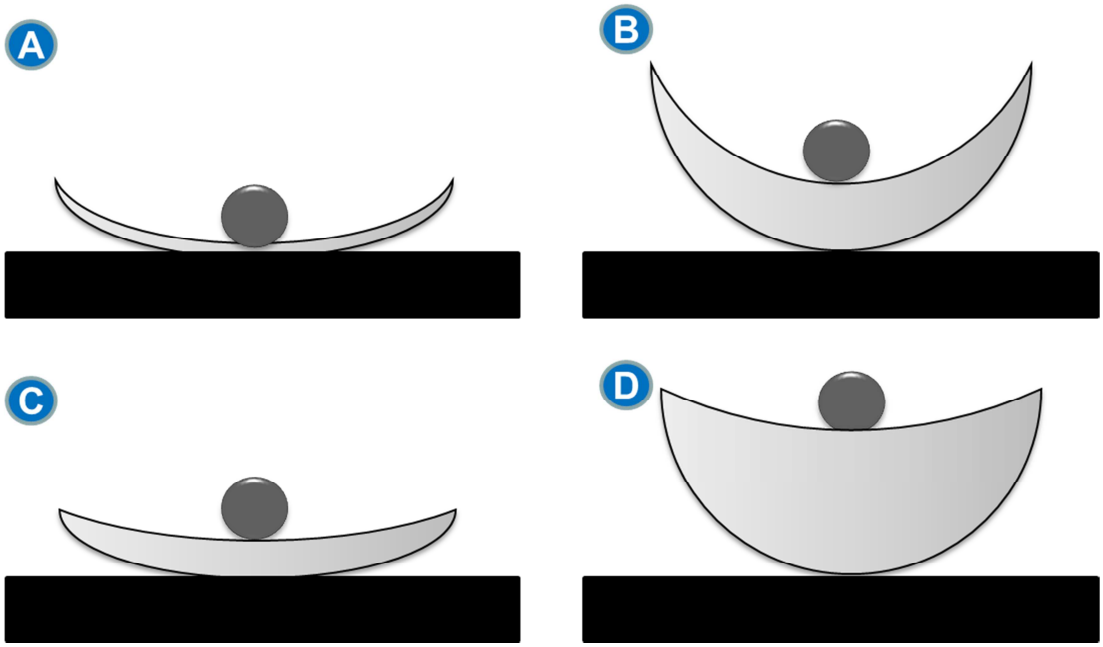

Figure 8.5 Stability of a ball over stable or unstable bowl

Protocol B – low back muscle fatigue

The results of isokinetic trials also showed significant increases in agonist, antagonist and synergist activity after the muscle fatigue protocol, supporting the results in the 35% isometric trials at 20° trunk flexion. However, the agonist activity in the free stooping technique showed no difference between TIME 0 and TIME 1. This lack of agonist activity may be caused by the smaller lumbar flexion angle (i.e., decreased lumbar flexion) and earlier EMG-off angle after the muscle fatigue protocol discussed in Hypothesis 2. Note that the full lumbar flexion angle in the free stooping after the protocol (Protocol B at TIME 1 in free stooping) was the most upright posture throughout all other conditions, and the EMG-off point after the protocol in the free stooping was also observed earlier than any others. In the current study, the full lumbar flexion angle in the free stooping after the protocol was regarded as the upright standing posture to calculate the percentage of flexion for a fair comparison between each condition, which is a combination of PROTOCOL (3) and TIME (2). So, all conditions are reasonably compared at the same level of lumbar flexion, and only the free stooping condition of Protocol B at TIME 1 partially included flexion-relaxation (i.e., silence period) in the range of motion (80% flexion ~ 20% extension) analysed in the isokinetic trials. Consequently, the earlier transition from the active mechanism to the passive mechanism in the free stooping technique at TIME 1 resulted in a weak increase in the agonist. The observation may suggest a cooperative mechanism between active and passive systems in low back to keep the stiffness of the spine and hence spinal stability. However, it is clear that the stiffness of the viscoelastic tissues in low back is less than the normal condition (TIME 0) around full lumbar flexion on the analogy of the earlier EMG-off point after the muscle fatigue protocol (TIME 1) (i.e., earlier transition to passive mechanism). In other words, the viscoelastic properties of the passive tissues in low back are

intact in the muscle fatigue protocol, so it could be inferred that the tissues generate less passive moment than the normal condition (TIME 0) because of less lumbar flexion at the EMG-off point after the protocol (TIME 1); remember that the passive tissues are only passively stretched and generate passive moments.

The analogy led me to question how the earlier biomechanical equilibrium point (i.e., earlier EMG-off point) could be satisfied where less passive moment of the low back viscoelastic tissues is expected. Considering the fact that more passive moment is necessary to meet the earlier equilibrium point, the passive tension or passive elastic stiffness of the muscle can be a candidate to fill the void of the analogy. A literature review showed that the passive elastic tension increased around full stretching length after both isometric fatiguing protocol and eccentric contraction protocol in the triceps surae and right calf muscles (Whitehead et al., 2001; Finlayson et al., 2008). The authors explained it as immediate strain injury contractures, referring to a contraction of the fiber in the absence of an action potential, in damaged muscle fibers after the muscle fatigue protocol. It is possible that the strain contracture in low back muscles results in the reduced muscle length and increased passive tension, supported by significantly less peak lumbar flexion angle after the muscle fatigue protocol (H2) (Parnianpour et al., 1988). All in all, the increased passive elastic tension after muscle fatigue protocol around the full flexion may provide additional passive moment instead of the decreased active moment-generating potential of the low back muscles, and consequently result in an earlier equilibrium point between the external moment and the passive moment generated by the passive tissues and muscles in the low back. It could be regarded as a compensatory mechanism between the active system and the passive system in the low back to assist each other under the abnormal condition that is also observed in Protocol A.

As already discussed, the alteration of the load-sharing mechanism between active and passive tissues in the low back after the muscle fatigue protocol may be a signal of trunk instability that is caused by inability in low back muscles. Regarding the role of the antagonist, prior studies pointed out a unique role of the antagonistic exertion under the trunk instability condition. They proposed and showed that the muscles do not contribute to generating any driving force in the movement, but the additional excessive activation stiffens the spinal column and enhances spinal stability to prevent buckling of the spine under the muscle fatigue in low back (Bergmark, 1989; Cholewicki and McGill, 1996; Crisco and Panjabi, 1990). In addition, the biomechanical models of the spine suggested an increase of trunk stiffness by the recruitment of antagonistic coactivation (Gardner-Morse and Strokes, 1998; Granata and Orishimo, 2001). Regarding the muscle fatigue condition, Granata et al. (2004) used a biomechanical model to compute the effects of muscle fatigue on spinal stability and revealed a significant increase in abdominal muscles to sustain the spinal stability after the muscle fatiguing protocol. The result of this study also revealed a significant increase in the antagonistic coactivation after the muscle fatigue protocol in free stooping to enhance trunk stiffness. The earlier transition into the passive mechanism after the protocol may reduce the spinal stability and hence the antagonist activity was increased to enhance the stability around the full flexion. It is possible that the increased passive elastic tension of the fatigued low back muscles can account for the external moment in a similar fashion to the tensed rope, but cannot provide enough stability in the spinal column because of totally different origination and insertion of the muscles than the ligaments.

The synergist also showed a significant increase in the free stooping posture after the protocol, and there is no difference in the restricted stooping. As already discussed in Protocol A, the results may suggest the increased role of the synergist as a pelvic stabilizer under the

condition of the spinal instability (i.e., after the muscle fatigue protocol) for providing a stable foundation and a passive torque. The trunk system may be required to enhance the pelvic stability and increase moment generation potential under the spinal instability by recruitment of the super global system. Based on these, the global and super global systems including the antagonist and synergist contribute to the trunk system stability under abnormal conditions such as low back muscle fatigue.

Protocol C – combined effect of viscoelastic tissue elongation and muscle fatigue

Noting that the trunk kinematics variables in Protocol C (such as EMG-off angle and full lumbar flexion angle) were similar with to those seen in Protocol A, comparable results in the muscle activation profiles during the isokinetic trials were expected. However, the results of Protocol C were somewhat different from those of Protocol A. For example, a significant interaction was observed in the agonist of Protocol A because of the greater increment rate in free stooping than restricted stooping after the protocol, but Protocol C showed nearly equivalent increment rates in both free and restricted stooping techniques after the protocol (i.e., no interaction). That interaction is caused by a greater increase (3.1%) in the restricted stooping at Protocol C (1.9% increase in Protocol A). When only viscoelastic tissues in the low back are relaxed (Protocol A), the restricted stooping may take advantage of the stable foundation (pelvis) discussed in the 'ball on the unstable bowl' example of Figure 8.5 and hence, show a smaller increment rate in agonist activity of the restricted stooping than the free stooping after the protocol. However, the difference in agonist activity between two postures (e.g., greater activity in restricted posture) was maintained after Protocol C. It seems that the restricted stooping after Protocol C could be asked to increase low back muscle activation levels to account for the

inability in both stabilizing mechanisms, such as decreased force generation potential in the active mechanism (muscles) caused by muscle fatigue and the reduced tension-generation capacity in passive mechanisms (ligaments, discs and fascia) caused by passive tissue elongation. In other words, more severe conditions after Protocol C (i.e., inability in both stabilizing mechanisms) may partially eliminate the advantage of the stable pelvis observed in the passive tissue elongation condition. It could be also explained by the 'ball on the unstable bowl' example. Under the inability in both stabilizing mechanisms caused by Protocol C, the ball (spinal column) on the bowl may be faced with more unstable conditions as compared to the other abnormal low back condition caused by Protocols A and B, so additional potential to stabilize the ball may be required. The enhanced agonist activity in the restricted posture after Protocol C could be an additional potential to stabilize the ball. The lack of assistance from the super global system (i.e., synergist) in the restricted stooping may cause the agonist to stabilize the unstable spinal column (ball). This condition can be described as 'the ball on the stable bowl' example represented in Figure 2.10.

The synergist in the free stooping also increased its activation level after Protocol C to maintain the pelvic stability and increase the passive tension generated throughout the lumbodorsal fascia and affected on the trunk stability. Considering a significant increase in the activation level of the super global system during the free stooping, it may be reasonable to believe that the super global system interacts with the global and local systems in the low back to keep the trunk stability and generate the extension torque under abnormal low back conditions. Altogether, the results supported the analogy of 'the ball on the stable bowl' describing the importance of considering the super global system as a part of the trunk flexion-extension system.

8.3. Recovery after the protocols

8.3.1. Passive tissues elongation protocol

Some of previous studies investigated biomechanical responses during the recovery phase after creep of lumbar viscoelastic tissues and fatigue of the lumbar muscles, but there have been no studies investigating the recovery phase of FRP variables such as EMG-off points and viscoelastic creep (monitoring passive system) and trunk and lower extremity muscle activities (monitoring active system) of the human subjects. In previous studies, they usually employed the spine of feline to assess the multifidus EMG and viscoelastic creep after static or cyclic lumbar flexion (Solomonow et al., 2003b and 2003c), except Shin and Mirka (2007) investigating full lumbar flexion angle and low back muscle activity of human subjects during 10 minutes of resting session. Scientific knowledge about the recovery phase after viscoelastic creep and validation of the hypotheses suggested in the feline model studies are still lacking.

The result of full lumbar flexion angle denoting recovery of the viscoelastic creep and passive muscle elongation supported Shin and Mirka (2007) showing that the creep developed during 10 minutes passive trunk flexion protocol were not fully recovered by the 10 minutes standing recovery session. Both current study and Shin and Mirka (2007) showed a remarkable recovery of the viscoelastic creep in very early resting period (within 5 minutes), but the elongated passive tissues were not even fully recovered until the end of resting session (40 minutes). Regarding the recovery of FRP, it is interesting to note that the EMG-off points did not follow the recovery trend of the peak lumbar flexion angle. Generally speaking, the FRP are highly affected by the full lumbar flexion angle in that modification of the full lumbar flexion angle is directly related with the passive tension capacity of the passive tissues. This inconsistency with our expectation could be explained by the non-linear stress-strain relationship

of the lumbar viscoelastic tissues (Dumas et al., 1987; Nachemson and Evans, 1968). Shin (2006) also suggested that the ratio of the strain increase at the given level of viscoelastic creep is greater than the ratio the stress increases because of the non-linear stress-strain relationship of the tissues. In other words, the effect of modification in the strain (i.e., deformation of the passive tissue) does less affect on the changes in the stress (i.e., applied load on passive tissues); the stress is not sensitively modified as compared to the strain. It is possible that the observed recovery in full lumbar flexion angle (hence, recovery in viscoelastic tissues) was not enough to change the stress level (i.e., passive force generation capacity of the tissues). Consequently, the load-sharing mechanism between active and passive tissues could be as it stands, and still requires later EMG-off point to meet the biomechanical equilibrium point between external torque and the passive tissues until the end of resting session (40 minutes).

The observation in lumbar flexion angle and EMG-off points could be linked with the recovery studies using feline model investigating the recovery characteristics of relaxed viscoelastic tissues for 7 hours and suggesting a long process outlasting more than 60 times of the developed period (Solomonow et al., 2003b and 2003c). Solomonow et al. (2003b) employed 20 minutes of static or cyclic elongation of the supraspinous ligaments using a hook and observed that the creep developed in the ligaments does not fully recover over the 7 hours of following resting session. In addition, their model predicted that the developed creep in viscoelastic tissues and hyperexcitabiliy in lumbar muscles could recover only after 24-48 hours of rest. The follow-up study of Solomonow et al. (2003c) also showed that the cumulative creep in viscoelastic tissues of the feline, developed by three 10 minutes sessions of static flexion with each session followed by a 10 minutes rest, was not fully recovered during a 7 hours rest period. In a series of studies, they suggested that the abnormal condition in low back viscoelastic tissues

is not an isolated mechanical phenomenon, rather a transient neuromuscular disorder, because of reflexive spasm and hyperexcitability in multifidus caused by micro-damage in viscoelastic tissues. This study also showed an initial hyperexcitability of the agonist muscles after creep development in viscoelastic tissues and following quick recovery in very early period of resting session (within 5 minutes) observed in Solomonow et al. (2003b), but failed to observed delayed hyperexcitability in agonist because the resting session was only lasted for 40 minutes in this study; they usually observed delayed hyperexcitability in multifidus after 1 hour of rest. According to the Solomonow group, the increase in agonist muscle activity (hyperexcitability) right after the protocol could be attributable to the hyper-excitable spinal reflex response, which is a rapid, automatic response (e.g., muscle activation) to specific stimuli, caused by the micro-damage in viscoelastic tissues. It is possible in that the passive tissues in low back are known to have a reflex pathway from ligament, the intervertebral disc or the facet joint to paraspinal muscles throughout the electrical, mechanical and chemical stimulation (Indahl et al., 1995, 1997; Kang et al., 2002; Solomonow et al., 1998; Stubbs et al., 1998). In addition to this, the structural compliance of the viscoelastic tissues during the recovery phase, confirmed by the minimal recovery in the full lumbar flexion angle, can detrimentally influence on the force transmission from the active tissues to the spinal column and result in an increase in the muscle activity to further generate stabilizing potential. In summary, it is reasonable to believe that the low back viscoelastic tissues require at least more than 4 times of resting period of the creep development time to fully recover its normal condition in length and spinal reflex.

The synergist also followed the recovery trend of agonist. Considering the fact that there is no reflex pathway between low back passive tissues and the lower extremity muscles, and the synergist was intact after the 10 minutes passive tissues elongation protocol, it could be

explained as a synergistic activation of the muscles to enhance spinal stability and extension torque; recall that the pelvis was restricted in the seated posture during the protocol to limit the creep development within the lumbar region. Contrary to the enhanced recruitment of the synergist, it is interesting to observe a significant depression (~ 33%) of the antagonist after 5 minutes resting. A significant co-contraction was expected in that the additional excessive activation of the antagonist (i.e., co-contraction), including rectus abdominis and external oblique, is known to stiffen the spinal column and enhance spinal stability to prevent buckling of the spine under abnormal low back conditions (Cholewicki et al., 1998; Gardner-Morse and Stokes, 1998; Granata and Marras, 2000). However, instead of increasing antagonistic co-contraction, the synergist significantly enhanced its activation level right after the 10 minutes protocol (TIME 1). Furthermore, the antagonist showed a remarkable depression from 5 minutes resting to the end of observation (40 minutes), but the synergist maintained its enhanced activation level at that time. It is possible that the synergist complements the role of antagonist to enhance and keep spinal stability when the developed creep in the viscoelastic tissues causes transient neuromuscular dysfunction in the agonist of the low back. Based on these, the co-work mechanism between antagonist and synergist could be highly related with the activation mechanism of the antagonist that could be planned in skilled motor programs before the initiation of the movement (Brooks, 1986).

Previous studies revealed that both agonist-antagonist co-contraction or inhibition are mainly controlled by the cerebellum (Deluca and Mambrito, 1987; Frysinger et al., 1984; Tilney and Pike, 1925) in which excitation of the Renshaw cells (and depression of I*a* inhibitory neurons) can facilitate antagonistic co-contraction, usually employed for weak and finely tuned movements, and excitation of I*a* inhibitory neurons (and inhibition of the Renshaw cells) can

induce the reciprocal inhibition, usually corresponding with the crude and strong movements (Hultborn et al., 1971). Considering that the participants were asked to have a constant trunk flexion-extension speed controlled by a metronome sound (one beat per second), a finely tuned trunk movement requiring antagonistic co-contraction may be performed in the normal condition (TIME 0). However, it seems like that the reciprocal inhibition, describing the relaxation of the antagonist muscles to accommodate contraction of agonistic muscle exertions, are active during the resting period (Sherrington, 1909). It is not clear why the pre-planned motor program (i.e., strategy of the movements) controlling agonist-antagonist co-activation or inhibition would be modified, but some benefits of the reciprocal inhibition with the synergistic activation to enhance the spinal stability could be expected. For example, the enhanced activation in antagonist muscles (i.e., abdominal muscles) for stabilizing the spinal column also increases the spinal compression load (Hughes et al., 1995; Marras and Granata, 1996). There may be an increased risk of spinal failure caused by enhanced co-activation in antagonistic muscles at the benefit of the spinal stability. So, a reciprocal inhibition with the synergistic activation could reduce the risk of spinal failure and enhance the spinal stability simultaneously, even thought the stabilizing role of the synergist could be weaker than the antagonist. It is plausible that the higher spinal instability right after the protocol (TIME 1) may necessarily require both antagonistic co-contraction and synergistic activation at the cost of increased spinal compression load, but the partially recovered spinal system after 5 minutes resting could require a synergistic activation only to keep the minimal level of spinal stability and control the risk spinal failure. It can be hypothesized that the antagonistic co-contraction or inhibition of trunk muscles can possibly be balanced by the optimal control mechanism of the motor control system manipulated by central (cortex) commands (Brooks, 1986).

The isometric exertion also showed similar recovery trend with the isokinetic exertions, but the recovery phase was far more variable than the isokinetic exertions. For example, the standardized value of the isokinetic exertions was plotted between ± 1 standard deviation (SD) of the mean (0) because of smaller SD within each TIME, but the standardized value of isometric exertion was located within ± 0.5 SD of the mean (0) because of bigger SD within each TIME. The bigger variance in the 35% isometric exertions could be attributable to the more upright trunk posture (20 degree flexion from upright standing) and bigger exertion level than the isokinetic exertions, requiring control of the upper body mass only. In the upright posture, the relaxed passive tissues may suffer with more severe spinal instability because of reduced friction generated by the passive tissues that oppose the movement but hold the spinal column. Also, the greater exertion level under the abnormal low back condition could increase the variability in the recruitment of the required muscles. Mirka et al. (2000) revealed that an increase in the exertion level of the erector spinae results in increases in both average and variance of the muscle activity. Reducing variability is important in that a task having higher variability in the stresses on low back can have likelihood to bring unexpected higher force and moment on low back (Mirka and Baker, 1996). All together, the 40 minute recovery phase in muscle activities, full lumbar flexion angle and EMG-off angles revealed the need of a long recovery process after the passive tissue elongation protocol and showed some evidences of abnormal low back conditions causing spinal instability.

8.3.2. Muscle fatigue protocol

The Hypothesis 2 revealed a decrease (i.e., less flexion) in full lumbar flexion angle and EMG-off points after the 10 minutes low back muscle fatigue protocol, but the analyses of

recovery phase, including free stooping only, did not show any significant difference between eight levels of TIME. Each data point in the Figure 7.33 showed small standardized value denoting significant variance within each level of TIME and did not clearly show a significant trend (i.e., close to the mean (0)). The result could be attributable to the weak statistical power caused by small number of observations (24 observations within each level of TIME). However, considering the fact that the other two protocols, including passive tissue elongation, showed significant effect of the protocols, no significance after the muscle fatigue protocol may be regarded as a weak effect on the lumbar flexion angle and EMG-off points as compared to the laxity in viscoelastic tissues. The hypothesis could be supported by the result in H3 showing that the elongation or deformation of passive tissues in low back has a more dominant influence on the FRP than the active low back muscle fatigue. Those results indicate that the low back muscle fatigue have a weak effect in modifying the FRP than the inability in the passive tissues.

In regards of the muscle activities, the agonist activity in the isokinetic trials did show no difference in all eight levels of TIME. As already discussed in H7, the weak agonist activity could be caused by the smaller lumbar flexion angle (i.e., decreased lumbar flexion) and earlier EMG-off angle after the muscle fatigue protocol. Consequently, the earlier transition from the active mechanism to the passive mechanism in the free stooping technique at TIME 1 resulted in a weak increase in the agonist. However, the 35% isometric exertions at 20 degree trunk flexion showed a significant increase in the EMG amplitude that is known to be caused by the reduced motor unit action potential since it cannot turn fast twitch fibers on (Allen et al., 2008). To keep the same level of extension moment during the 35% isometric exertion, the fatigued muscle in low back could be required to recruit more motor units, resulting in an increase in the EMG amplitude until 30 minutes rest. The EMG amplitude of the agonist was gradually

recovered during 40 minutes of rest and reached full recovery after the 40 minutes rest, but the median power frequency was fully recovered right after the 5 minutes rest. This discrepancy may be related with the underlying physiology of detecting EMG amplitude and power frequency. An increase in the EMG amplitude suggests fatigue-induced increases of additional motor unit and firing rate (deVries 1968; Edwards and Lippold, 1956; Moritani et al., 1986), but a decrease in EMG median power frequency reflects reduced muscle fiber conduction velocity (Eberstein and Beattie, 1985; Sadoyama et al., 1983). A recent study measured trunk muscle fatigue using the median power frequency also revealed that 90% of subjects are fully recovered in 5 minutes rest, supporting our observation (Shin and Kim, 2007).

Regarding the synergist and antagonist in the isokinetic trials, both of them showed significant increases right after the muscle fatigue protocol to keep the spinal stability. It is interesting to note that the motor control system enhances the recruitment of the antagonistic co-contraction and the synergistic activation of the lower extremity simultaneously right after the protocol. Granata et al. (2004) suggested that the transient inability in the low back muscles may necessarily require increasing the stiffness of the trunk system because of the reduced spinal stability. In the follow-up study, they showed that the antagonistic co-contraction increases the bending stiffness of the torso and hence reduces the spinal instability (Lee et al., 2006). In addition, the current study showed the functional role of super global system (i.e., lower extremity muscles) in trunk flexion and extension as an effective synergist to keep the spinal stability under the normal and abnormal low back condition. So, it is reasonable to believe that both antagonistic co-contraction and the synergistic activation are employed to enhance the spinal stability right after the muscle fatigue protocol. However, it also should be noted that the antagonistic co-contraction and the synergistic activation returned to the baseline levels after the

5 minutes rest. The results may show a quick, partial recovery in the trunk muscles and less spinal instability after 5 minutes rest in free isokinetic trunk flexion-extension. Comparing the results with the passive tissue elongation protocol, relatively much longer recovery process can be expected in the viscoelastic tissue elongation condition, and relatively quick recovery with rapid severe spinal instability can be supposed in the low back muscle fatigue condition.

8.3.3. Combined effect protocol

As the full lumbar flexion angle and EMG-off angles after the combined effect protocol (Protocol C), causing laxity in passive tissues and fatigue in low back muscles, followed the results of the passive tissue elongation protocol (Protocol A), the recovery phase of the combined effect protocol was also similar to the passive tissue elongation protocol. However, the Protocol C showed more clear recovery phase than the Protocol A, even though the full lumbar flexion and EMG-off angles in Protocol C also did not fully recover after the 40 minutes resting session. Regarding the agonist and synergist muscle activities in isokinetic trials during the recovery session, both of them were fully recovered after 40 minutes rest. The agonist showed a gradual recovery during the 40 minutes rest and finally reached to the baseline activation level at TIME 7 (40 minutes rest). The synergist was fully recovered to its baseline activation level right after 5 minutes rest. In the recovery session of the Protocol A, both muscle groups showed a remarkable recovery in very earlier resting period (5 minutes rest), but were not fully recovered during the 40 minutes recovery session. Meanwhile, the recovery phase of the isometric exertion in Protocol C was also very variable like the Protocol A, but the statistical analyses showed no difference between the baseline activity and TIME 4, 5, 6 and 7 (from 15 minutes rest to 40 minutes rest), suggesting full recovery in both muscle groups. Even though

the isokinetic trials in Protocol C showed a clear difference in the recovery phase with the Protocol A, no clear difference was observed between two protocols in the isometric exertions because of the greater variance.

Comparing the recovery phase between Protocol A and C in the isokinetic trials, the main difference was magnitude of the recovery to the baseline. Those results may be highly related with the design of each protocol in which the developed period of viscoelastic creep was exactly half in the combined effect protocol. In a comparison of the protocol effect (H3), the amount of change of EMG-off points in Protocol C was 64% of that in Protocol A on average. It is plausible that the reduced amount of passive stooping time during Protocol C results in less change in both peak lumbar flexion angle and EMG-off points at TIME 1, and, consequently, the recovery phase in the Protocol C also does not require a long time like the Protocol A. In addition, it seems like that the muscle fatigue developed in the Protocol C can be quickly recovered as shown in the recovery phase of the muscle fatigue protocol (Protocol B). If there is an interactive effect between passive tissues elongation and muscles fatigue the recovery phase could also require longer time than the independent effect of the Protocol A and B. However, as we already observed in H3, H4 and H7, there was no significant boosting effect in Protocol C on both muscle activities and FRP. Considering that the passive tissue elongation requires longer recovery than the muscle fatigue and the combined effect, it is clear that the inability in viscoelastic tissues caused by the prolonged stooping may have bigger potential to lead to low back injury as compared to the muscle fatigue or combined effect of muscle fatigue and passive tissue elongation.

In regards of the antagonist, the Protocol C showed similar recovery trend with Protocol A, but there was no statistically significant difference between TIMEs. It may also be related

with the less creep development in viscoelastic tissues. Again, the synergist was fully recovered within 5 minutes rest, suggesting no need of an enhanced synergistic activation to keep spinal stability after 5 minutes rest. The increase in agonist activation level may be enough to maintain the spinal stability under the condition of viscoelastic tissues inability to transmit the active force. The results also support a greater potential risk of low back dysfunction after the prolonged stooping (i.e., laxity in viscoelastic tissues) than the low back muscle fatigue condition or its combination with the repetitive stooping.

8.4. Limitations of this study

There are several limitations to this study that should be mentioned. First, the participants in current study were physically fit, male college students. Solomonow et al. (2003a) and Rogers and Granata (2006) reported significantly greater lumbar flexion in female subjects after the passive tissue elongation protocol as compared to male counterparts. Consequently, the decrease in reflex gain was also higher in female subjects (0.4 %/N (male) vs. 1.7 %/N (female)) (Rogers and Granata, 2006). Even though the source of gender difference is not clear at this point the results of previous studies and the findings in the current study suggests that females may experience more severe trunk instability under the viscoelastic tissue elongation in low back. Future research including female participants could provide valuable insight into the gender difference and may reveal characteristics of susceptible populations for development of LBP caused by inability in low back passive tissues.

Second, this study assumed a linear relationship between muscle force and EMG without considering the force-length and force-velocity relationships and only the biomechanical responses were investigated. It is well known that the EMG signal is directly related with the

motor unit density relative to muscle length, and also an increase in muscle contraction velocity yields increased EMG activity without a concomitant increase in force output. EMG-assisted models of the low back may provide better understanding of the load-sharing mechanism between active and passive tissues in low back biomechanics.

Third, although the role of super global system as an important synergist was shown throughout a new musculoskeletal model in this empirical study, the effect of synergistic activation on the spinal compression force and spinal stability was not directly calculated. Granata et al. (2004) developed a biomechanical model for calculating the spinal stability and compression force, but the model considered global and local systems only, defined by Bergmark (1989); note that their model did not account for the passive tissues in low back. Shu (2007) developed a musculoskeletal model considering both active and passive system in low back, but he also did not consider the super global system as a part of the passive system. A new EMG-assisted biomechanical model of spinal stability, accounting for the super global system, may provide a better understanding in the local system, global system and super global system in trunk flexion-extension (i.e., the role of antagonistic coactivation and synergistic activation) under both normal and abnormal low back condition.

Finally, only sagittally symmetric trunk flexion-extension was investigated in this study. Certainly, the orientation of low back ligaments and muscles are fundamentally changed during asymmetric trunk flexion-extension, thereby altering their force generation potential, especially in the passive elastic tension. Ning et al. (2010) showed the influence of asymmetry in low back FRP where the contralateral muscles showed earlier EMG-off in the asymmetry posture than the sagittal symmetry posture under the pelvic restricted stooping posture. The authors suggested greater passive tension in the contralateral side generated by the lateral flexion of the vertebrae

as a cause of earlier FR. Similarly, it is possible to observe different results in a comparison of the free and restricted stooping conditions under the asymmetric posture. For example, the hamstring muscles on the contralateral side may not be fully stretched around full asymmetric trunk flexion postures and also have different activation patterns during trunk flexion-extension in asymmetric postures. Consequently, the alteration in lumbopelvic rhythm may result in no difference between the free and restricted stooping conditions. Also, considering that the passive moment generated by viscoelastic tissues and passive stiffness of the muscles could be initiated by different mechanisms in asymmetric posture, investigations of the asymmetric postures in low back FRP may help to reveal the underlying physiology of the load-sharing mechanism between the passive tissue and active tissue.

Chapter 9 – CONCLUSION

The results, comparing the role of the pelvis/lower extremities in trunk flexion-extension, showed greater lumbar flexion angle and EMG-off angle in the restricted stooping posture than the free stooping posture. These results suggest that additional passive moments about the lumbar spine are generated in the restricted stooping posture because of the relative fixation of the pelvis that results in the reduced capacity of the passive mechanism (working through the lumbodorsal fascia mechanism and lumbopelvic rhythm) under the pelvic restricted condition. In line with the results, the lower extremity muscles revealed greater activation level in the free stooping than in the restricted stooping posture that acts to stabilize the pelvic and generate the passive moments in low back through the lumbodorsal fascia. Consequently, the enhanced pelvic stability and passive moments in the low back resulted in a significantly lower low back muscle activation level in the free stooping. In addition, under the abnormal low back condition, the results also showed a significant role of the super global system (i.e., lower extremity muscles) as a trunk system stabilizer by revealing a significant increase in the synergist muscle activation only in the free stooping. Collectively, these results indicate a significant role of the tissues of the larger super global system as a trunk stabilizer by immobilizing the pelvis during trunk flexion-extension motions and increasing the stiffness of the trunk systems by enhancing tension of the lumbodorsal fascia. The new musculoskeletal model of the active spinal stability system has proven the necessity of considering the super global system as an active stabilizer with the local system and global system, suggested by the Bergmark (1989), and can have implications for the loading of the spine during trunk flexion-extension, especially near full flexion.

The *in vivo* experiment, focused on the effects of three protocols in the biomechanical responses, showed greater full lumbar flexion and later EMG-off angles in both passive tissue

elongation protocol and combined effect protocol, but less pronounced results were observed in the combined effect protocol. The mixed effect of low back muscle fatigue and laxity in the passive tissues had not been investigated in previous studies, and these results may indicate relatively dominant contribution of passive tissue elongation in the mixed protocol. In the low back muscle fatigue protocol, the opposite trends in the lumbar flexion and EMG-off angles were observed. The results are consistent with the results of a previous study and explain the earlier biomechanical equilibrium point between active and passive tissues as an increase in passive elastic tension of the muscles caused by the strain injury contractures after the muscle fatigue. In all three protocols there appears to have been significant compromise of the passive spinal stability system, as the muscle activities in agonist muscles and synergist muscles were significantly increased in all three protocols illustrating an increased need for active control of the lumbar region. These results are important because workers performing a task for prolonged period in the stooped posture and then periodically manually lifting or moving products (e.g., farmers in crop production) would experience both passive tissue stretching and muscle fatigue simultaneously. The results of this study would imply a potential risk of low back injury in those workers, because of the compromise of both the passive and active stabilizing systems.

The results of the recovery phase in passive tissue elongation protocol showed longer recovery time (not fully recovered with 40 minutes of rest), and this was much longer than the muscle fatigue and the combined effect protocols. These results suggest that the inability of viscoelastic tissues to recover quickly may lead to greater low back injury risk in that the stress or fatigue on low back passive tissues may not recover before the next work activity begins. This creates an accumulation of passive tissue elongation over time. Also, the enhanced activation in

the synergist (i.e., super global system) and depression in the antagonist during the recovery session suggest an interactive mechanism between antagonist and synergist which may be planned in skilled motor programs before the initiation of the movement. Contrary to the passive tissues elongation protocol, the muscle fatigue protocol showed relatively quick recovery in all responses, but higher levels of muscle activity immediately after the 10 min protocol. In all three protocols, the super global system (i.e., synergist) showed quiet similar recovery pattern with the agonist. It also indicates the significant role of super global system in trunk flexion-extension as a trunk system stabilizer. The results during the recovery session can have implications to develop guidelines for designing safer working environments (e.g., work-rest cycles), especially where the stooped posture are required, that can lower the risks of musculoskeletal injury to the low back.

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APPENDIX A: INFORMED CONSENT DOCUMENT

Title of Study: A systems-level perspective of the flexion-relaxation phenomenon in the lumbar spine

Investigators: **Sangeun Jin**

Dr. Gary A. Mirka

This is a research study conducted as work for PhD dissertation. Please take your time deciding if you would like to participate. Please feel free to ask questions at any time.

INTRODUCTION

The purpose of this study is to achieve a better understanding of the muscle activity of trunk and leg muscles during full range trunk bending exertions.

DESCRIPTION OF PROCEDURES

If you agree to participate in this study, you will be asked to visit our lab three times with an interval of at least one week. Your participation will last no more than 90 minutes for each visit. During the study you may expect the following study procedures to be followed: Upon arrival the experiment will again be described and you will be asked to sign an informed consent form. Some simple measurements will be gathered (height, weight, etc.) (first visit only). A brief (5 minute) warm up routine will be provided to let you stretch and warm up the muscles of the low back and leg.

You will then be fitted with a set of sensors designed to capture muscle activity and three-dimensional trunk motions. In total 18 sensors (14 electrodes and 4 motion sensors) will be secured to your low back and legs. You will then step onto a type of exercise equipment, and perform two maximum trunk extension exertions (lifting motions) and two trunk bending exertion (sit-up motion) in a 20 degree trunk bending angle against the resistance provided by the exercise equipment. You will then be asked to perform a series of stationary leg flexion exertions where an experimenter will manually hold your ankle in position and you will pull against their hands. A one minute rest period will be provided between trials.

Before starting the recording session, you will be asked to stand upright and bend forward to reach a peak trunk bending posture. You will then be asked to push against an immovable bar located on your back using your trunk muscles (35% of your maximum capacity) while in 20 degree trunk flexion from standing posture (TEST A). Next, you will be asked to move slowly to a full trunk bending posture (reaching towards the ground) under conditions where there are straps around your waist and thighs and under conditions with no constraints (TEST B).

Next, one of three experimental protocols will be conducted for 10 minutes among the following: (1) alternately perform 25 sec full flexion in the seated posture and 5 sec upright sitting for 10 min; (2) alternately perform 25 sec static holding at 45 degree trunk flexion (no external load, just holding weight of torso) and 5 sec upright sitting for 10 minutes under the seated posture; and (3) consecutively perform 25 sec full flexion, 5 sec upright sitting, 25 sec static holding at 45 degree

trunk flexion and 5 sec upright sitting for 10 minutes under the seated posture. As soon as the 10 minute protocol is completed you will be asked to perform TEST B and TEST A protocols.

You will then be allowed to move around the room and periodically will be asked to perform TEST B and TEST A protocols at time 5 minutes, 10 minutes, 15 minutes, 20 minutes, 30 minutes and 40 minutes. After the final recording session, electrodes and magnetic sensors will be removed and you will be free to leave.

RISKS

While participating in this study you may be exposed to certain risks of injury. There is a risk for lower back injuries as well as some muscle or joint discomfort while performing the maximum exertions and the full trunk flexion motions. If at any point you do feel pain, please stop the task and alert the experimenter. If you have any chronic problems or recent injury or pain in your low back, hips, knees, or ankles, you should not participate in this experiment. If you do not have any trouble with muscles and joints (back, knee, wrist, neck, shoulder, etc.), please mark your initials here: _____. Finally, you may experience some low back muscle soreness for a couple days after the experiment similar to that felt after a strong workout.

BENEFITS

If you decide to participate in this study there is no direct benefit to you as a participant. You may derive some indirect benefits including an understanding of ergonomics research methods. It is hoped that the information gained in this study will benefit society by knowing more about the effect of prolonged stooping and muscle fatigue on low back.

COSTS AND COMPENSATION

For participating in this research study you will receive an Ergonomics Laboratory t-shirt.

PARTICIPANT RIGHTS

Your participation in this study is completely voluntary and you may refuse to participate or leave the study at any time. If you decide to not participate in the study or leave the study early, you will still receive the t-shirt.

RESEARCH INJURY

Emergency treatment is available at a medical facility at the location of the research activity. Compensation for any injuries will be paid if it is determined under the Iowa Tort Claims Act, Chapter 669 Iowa Code. Claims for compensation should be submitted on approved forms to the State Appeals Board and are available from the Iowa State University Office of Risk Management and Insurance.

CONFIDENTIALITY

Records identifying participants will be kept confidential to the extent permitted by applicable laws and regulations and will not be made publicly available. However, federal government regulatory agencies, auditing departments of Iowa State University, and the Institutional Review Board (a committee that reviews and approves human subject research

studies) may inspect and/or copy your records for quality assurance and data analysis. These records may contain private information. To ensure confidentiality to the extent permitted by law, the following measures will be taken. The biomechanical analysis is numerical and does not contain video that could identify the participant. Your data will be kept confidential by using alphanumeric identifiers that are unrelated to your name. Your name and information/data will be kept in separate locations. Your informed consent document will be kept in a locked file cabinet. The research team will keep private all research records that identify you to the extent allowed by law. When the results of the study are reported, the combined information that has been gathered will be presented. If the results are published, your identity will remain confidential.

QUESTIONS OR PROBLEMS

You are encouraged to ask questions at any time during this study.

- For further information about the study contact Sangeun Jin (515) 520-2191 or Dr. Gary Mirka (515) 294-8661.
- If you have any questions about the rights of research subjects or research-related injury, please contact the IRB Administrator, (515) 294-4566, IRB@iastate.edu, or Director, (515) 294-3115, Office of Research Assurances, Iowa State University, Ames, Iowa 50011.

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PARTICIPANT SIGNATURE

Your signature indicates that you voluntarily agree to participate in this study, that the study has been explained to you, that you have been given the time to read the document and that your questions have been satisfactorily answered. You will receive a copy of the written informed consent prior to your participation in the study.

Participant's Name (printed) _____

_____ _____

(Participant's Signature) (Date)

INVESTIGATOR STATEMENT

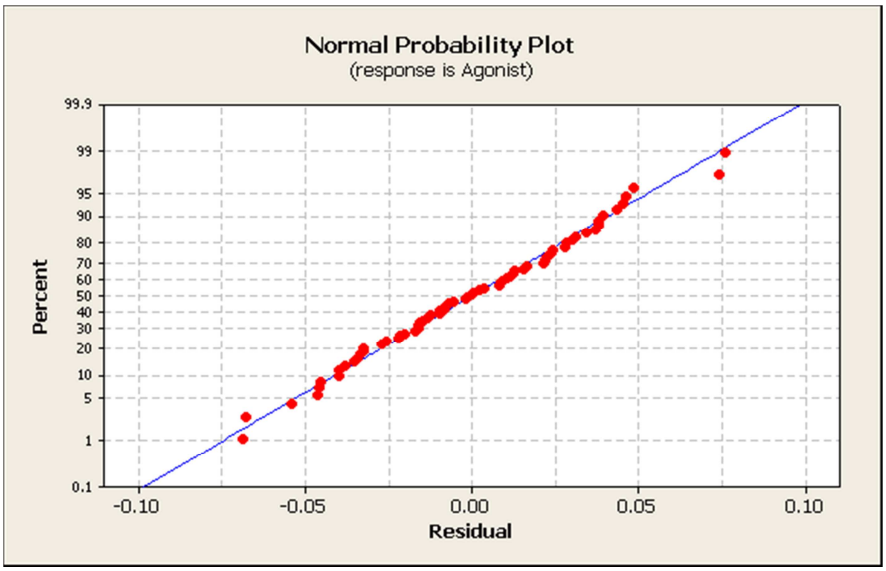
I certify that the participant has been given adequate time to read and learn about the study and all of their questions have been answered. It is my opinion that the participant understands the purpose, risks, benefits and the procedures that will be followed in this study and has voluntarily agreed to participate.

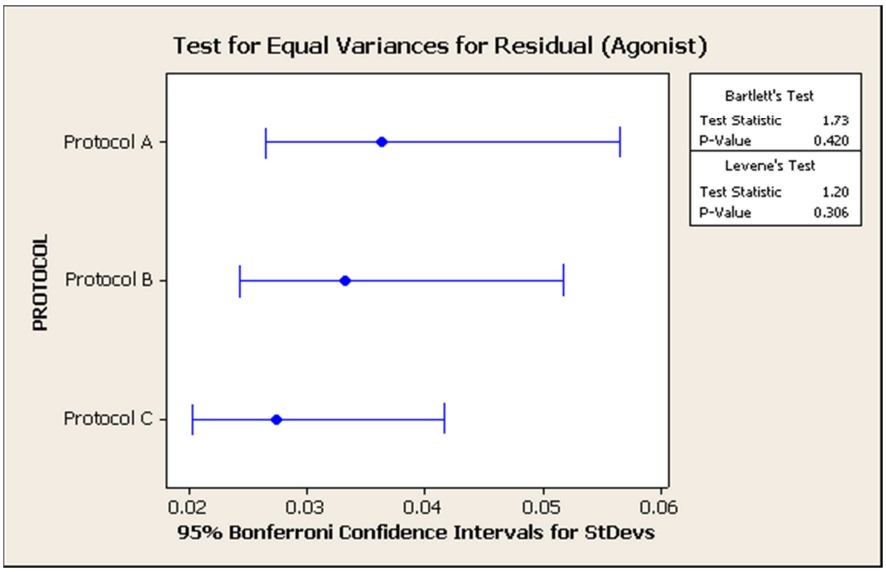
_____ _____

(Signature of Person Obtaining Informed Consent) (Date)

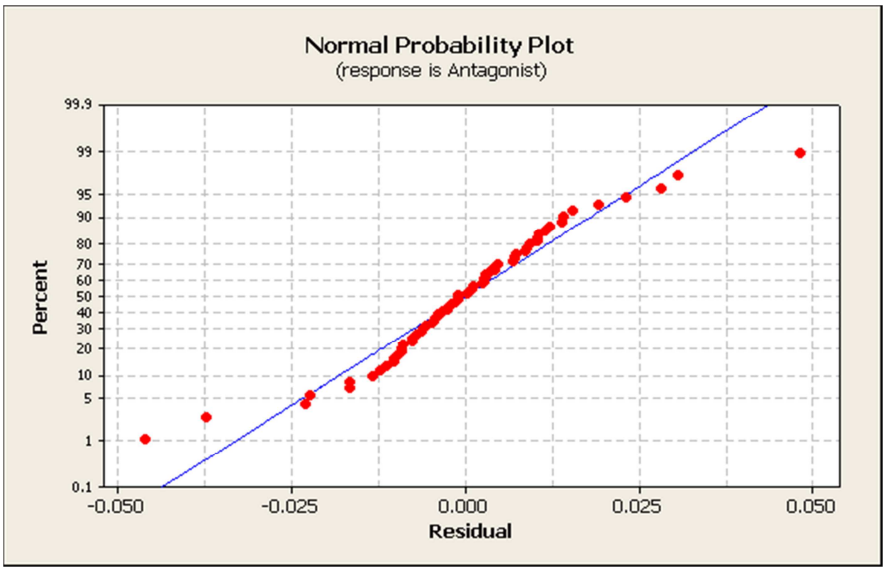
APPENDIX B: STATISTICAL MODEL ADEQUECY CHECKING

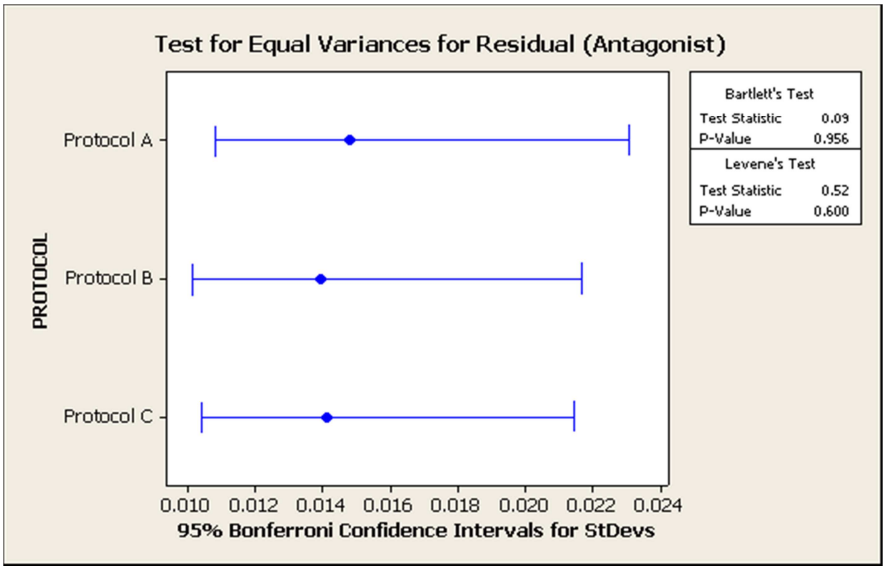
Model for Hypothesis 4 – One-way ANOVA for agonist



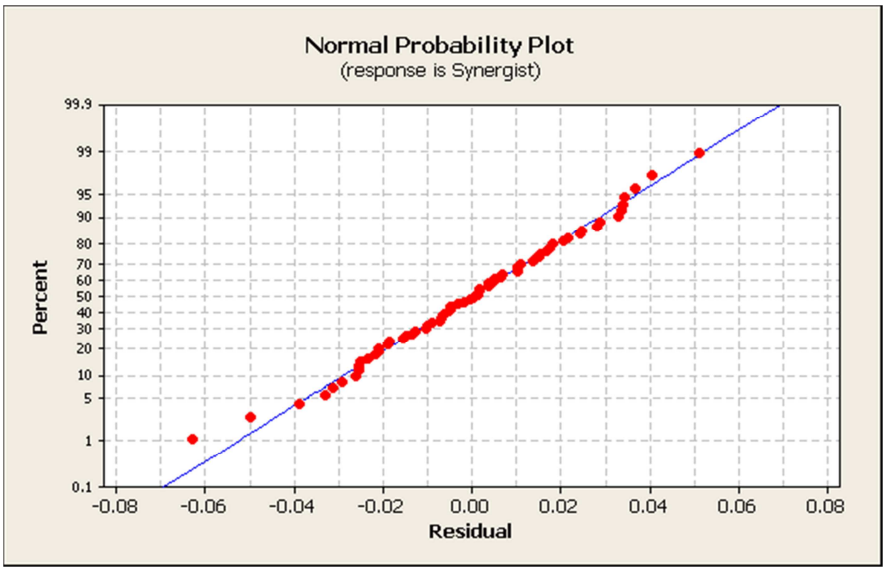


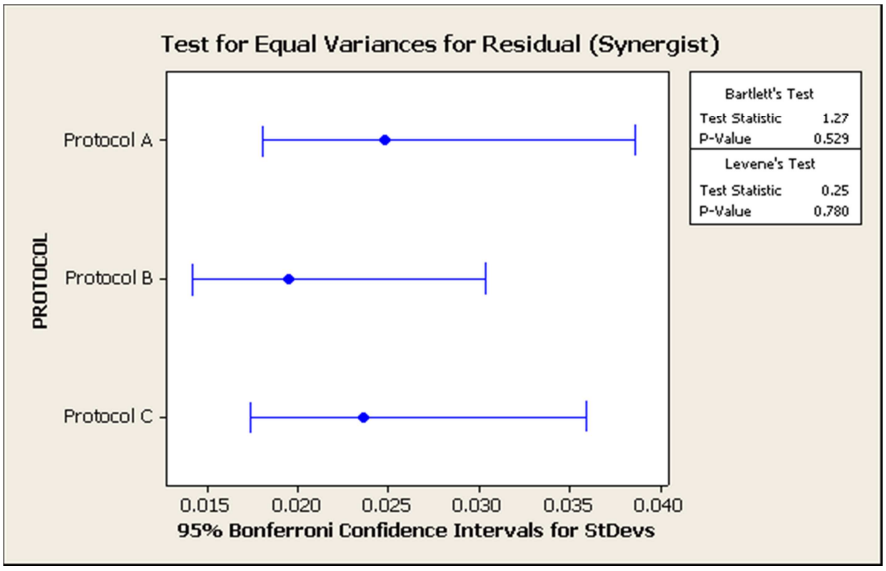
Model for Hypothesis 4 – One-way ANOVA for antagonist



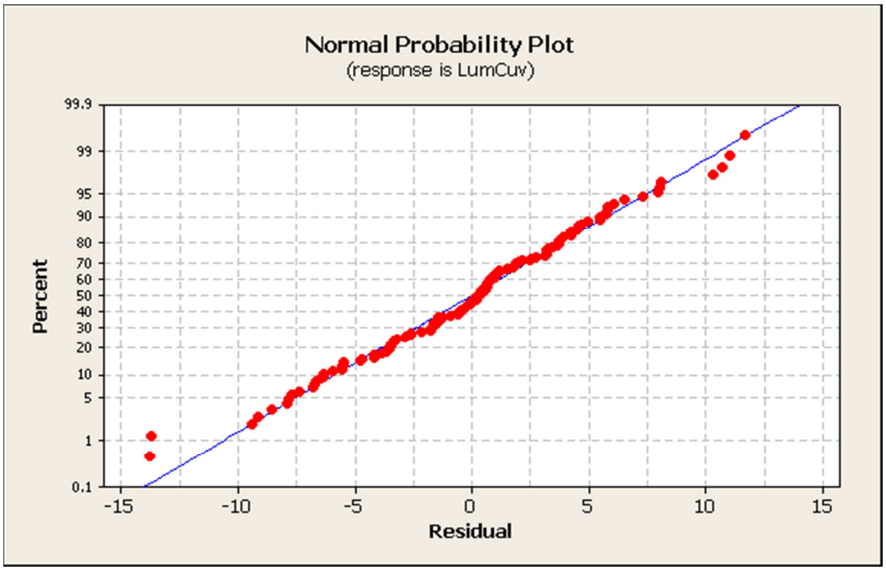


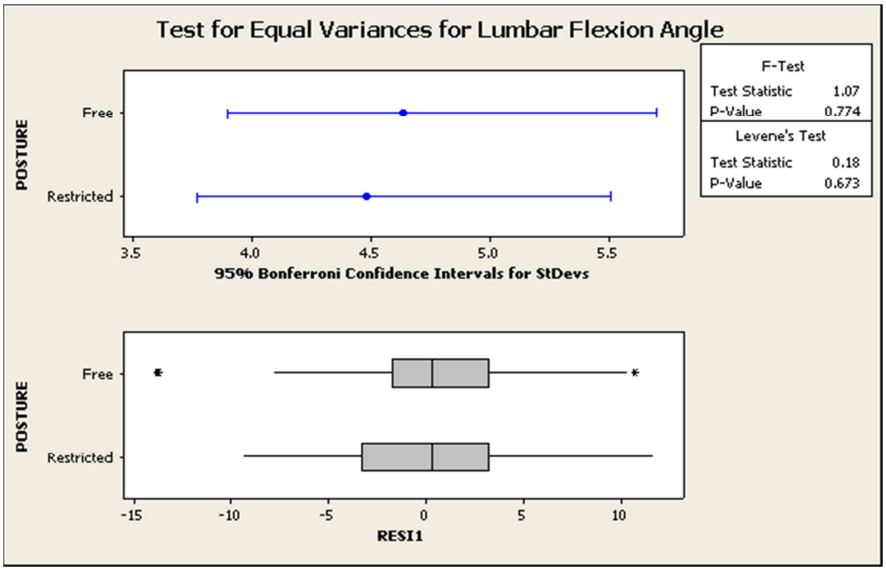
Model for Hypothesis 4 – One-way ANOVA for synergist



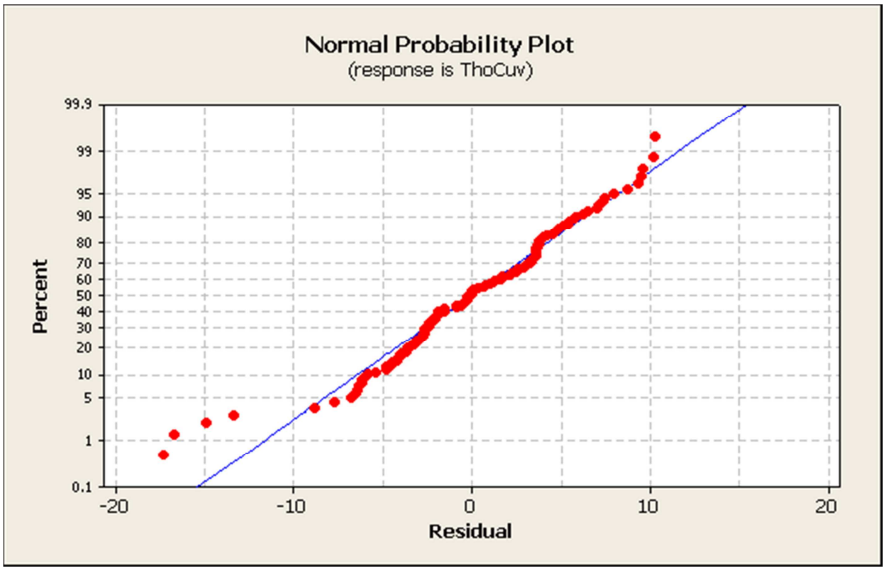


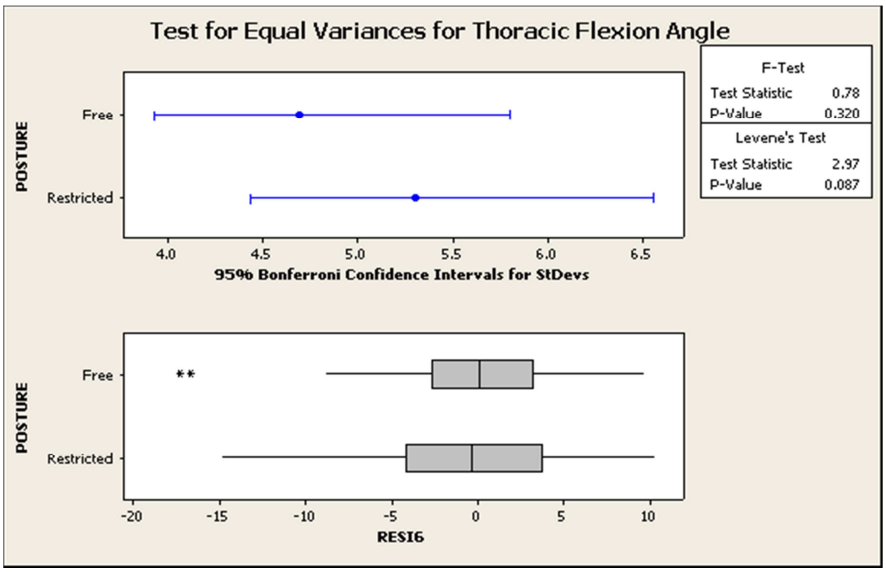
Model for Hypothesis 5 – One-way ANOVA for lumbar flexion angle



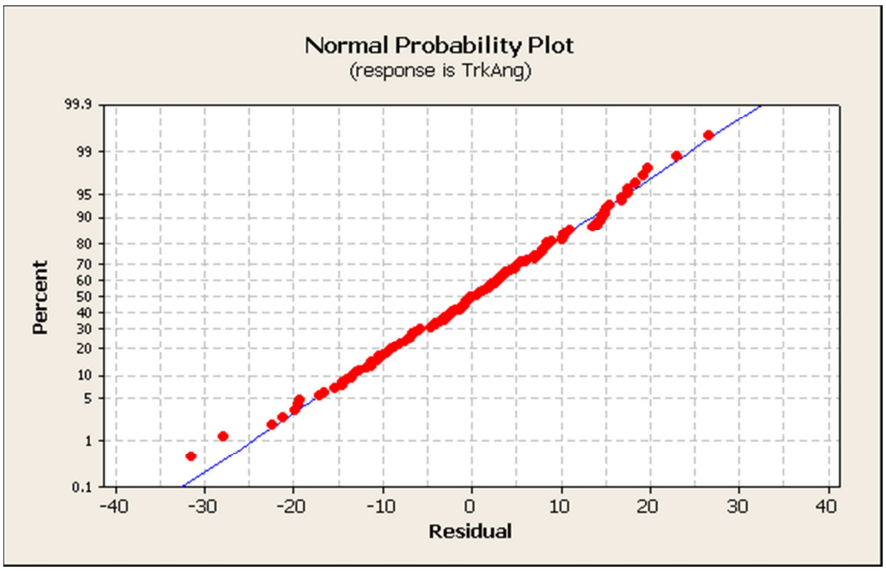


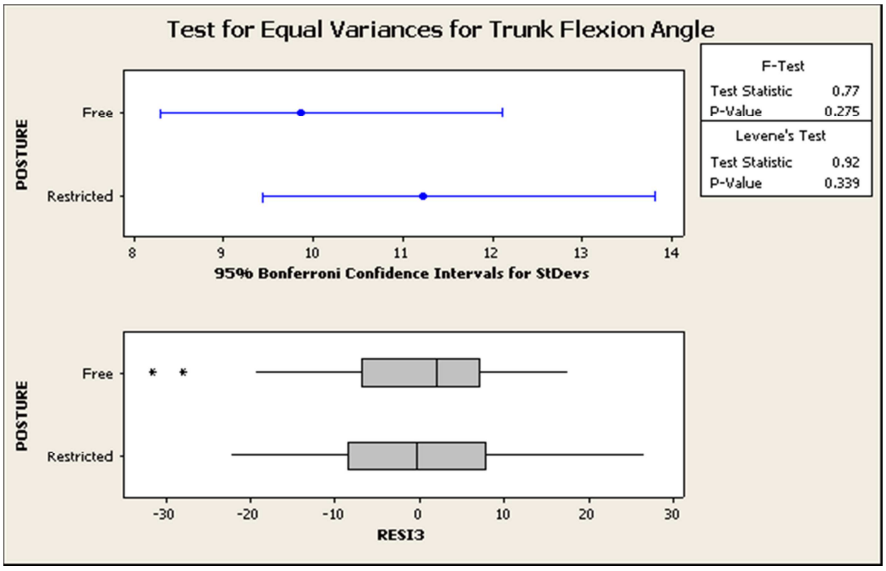
Model for Hypothesis 5 – One-way ANOVA for thoracic flexion angle



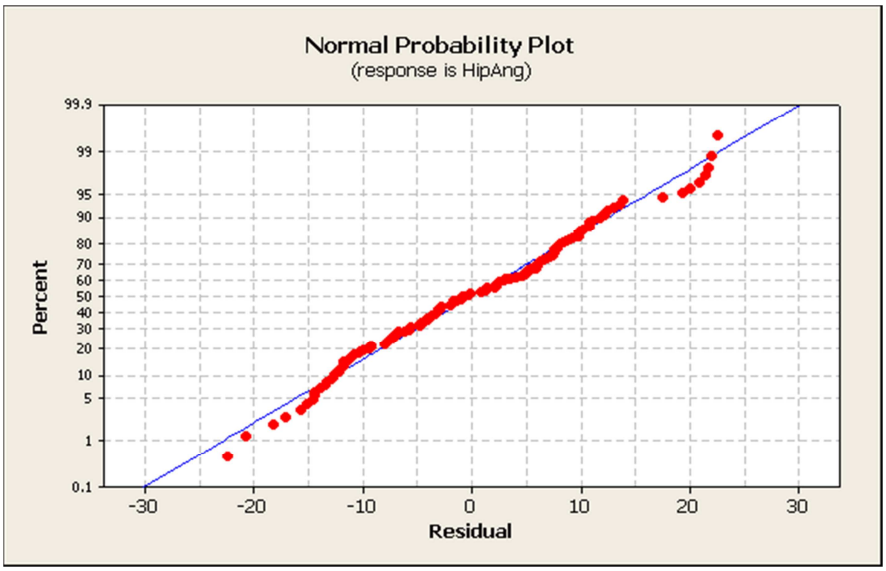


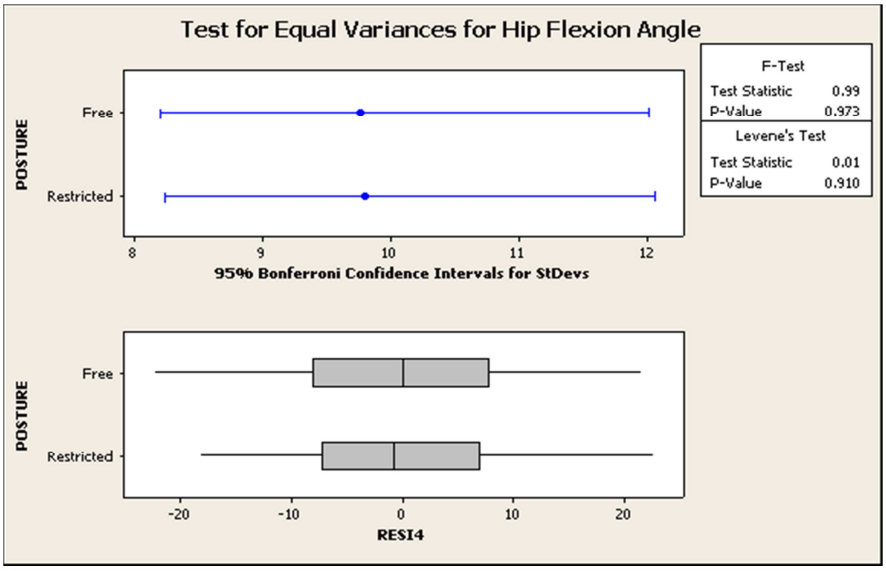
Model for Hypothesis 5 – One-way ANOVA for trunk flexion angle



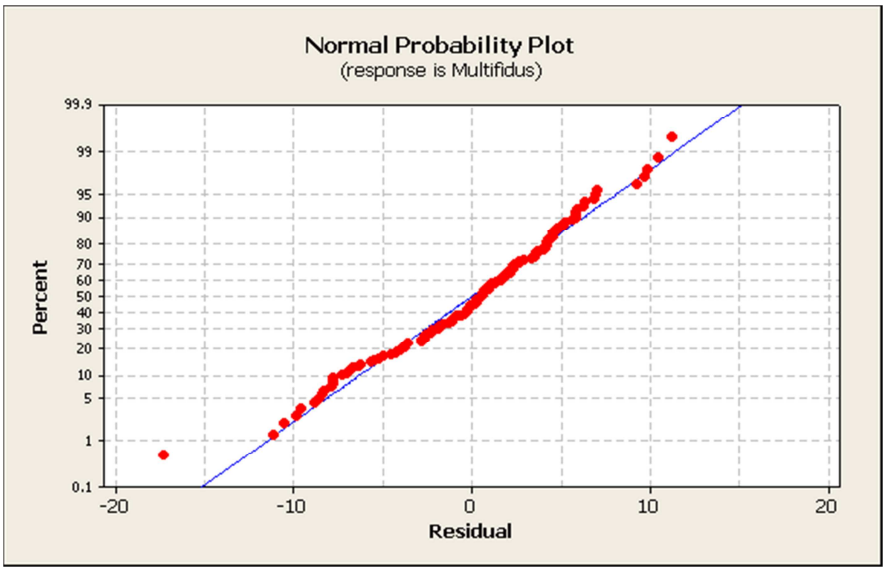


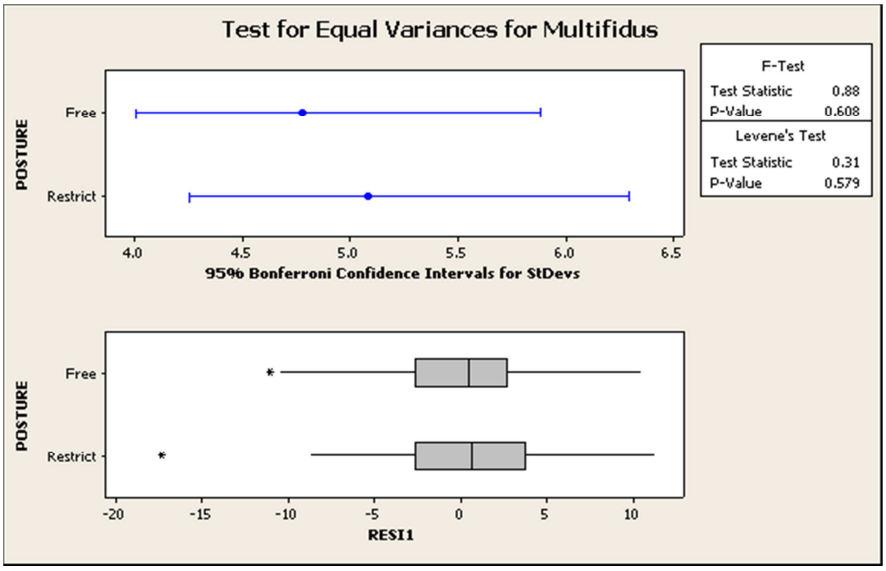
Model for Hypothesis 5 – One-way ANOVA for hip flexion angle



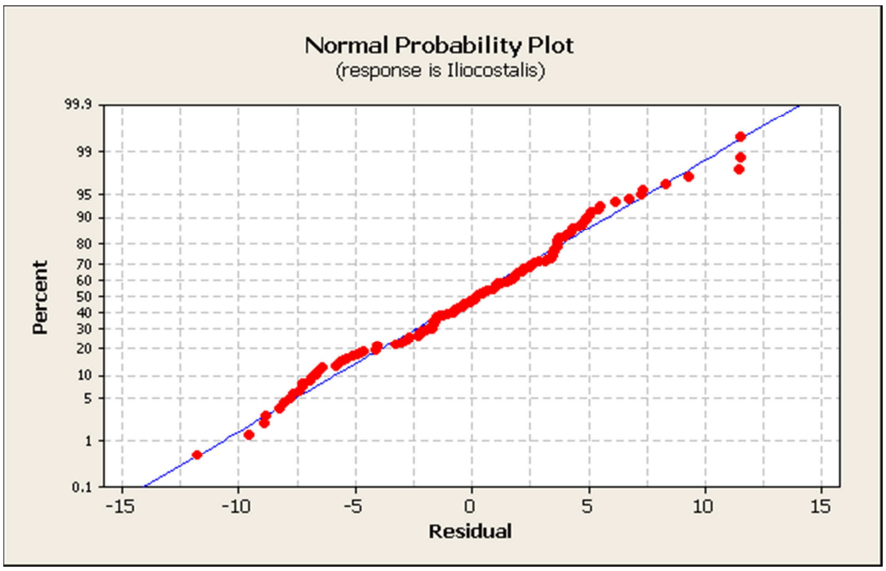


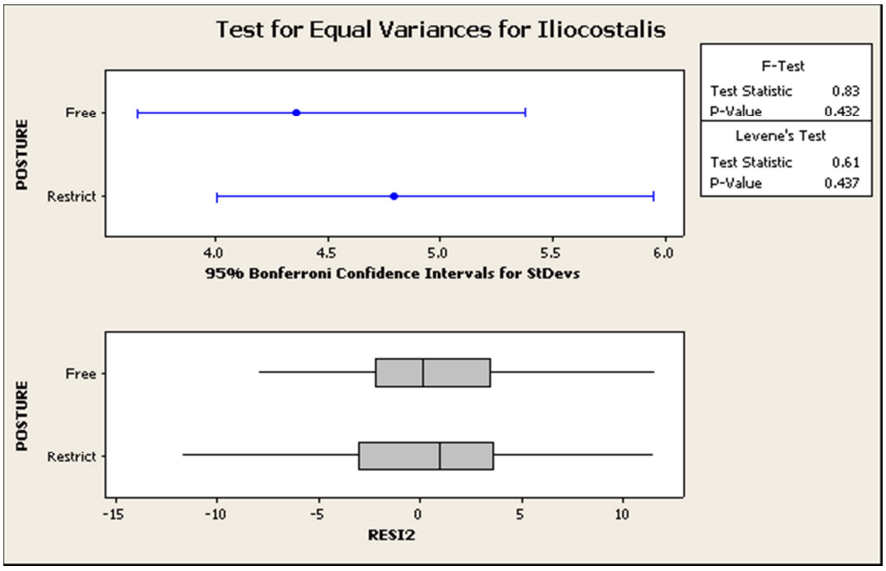
Model for Hypothesis 5 – One-way ANOVA for EMG-off angle of multifidus



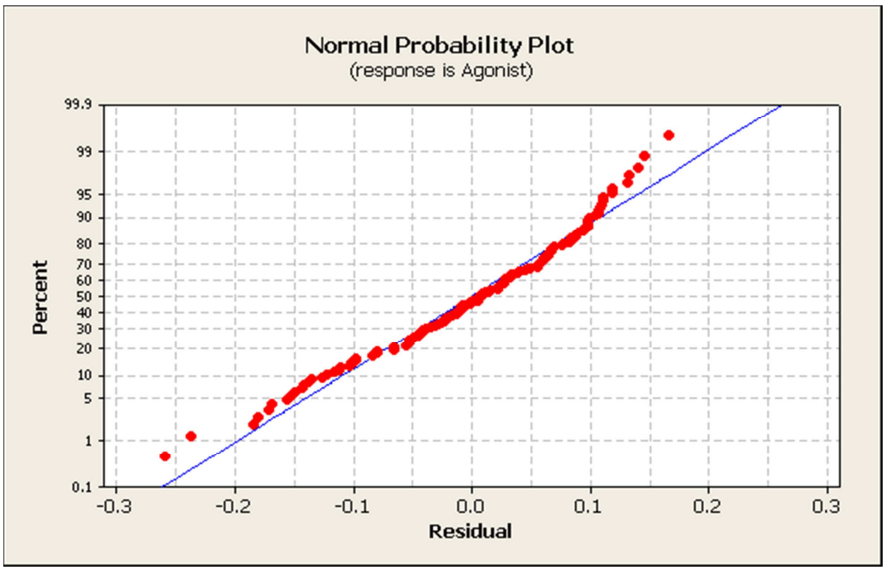


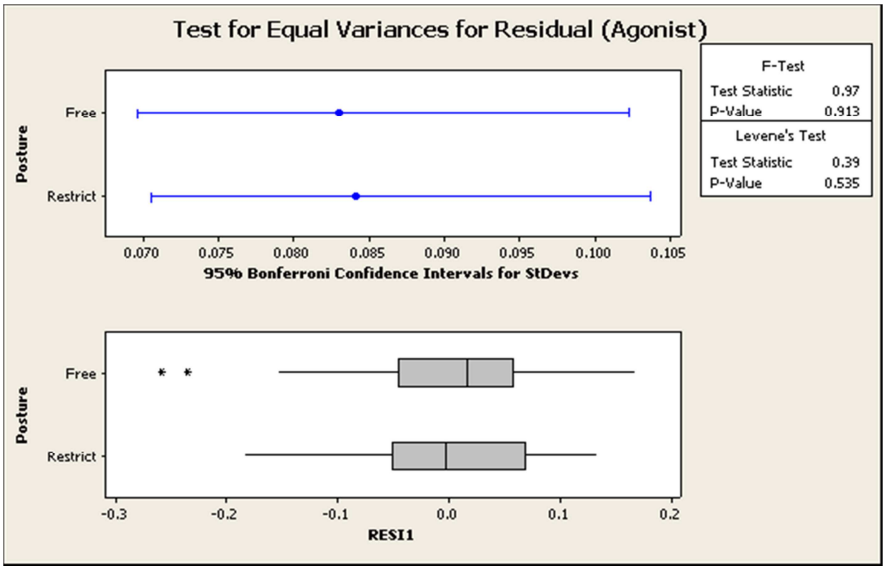
Model for Hypothesis 5 – One-way ANOVA for EMG-off angle of iliocostalis





Model for Hypothesis 6 – One-way ANOVA for agonist (transformed)





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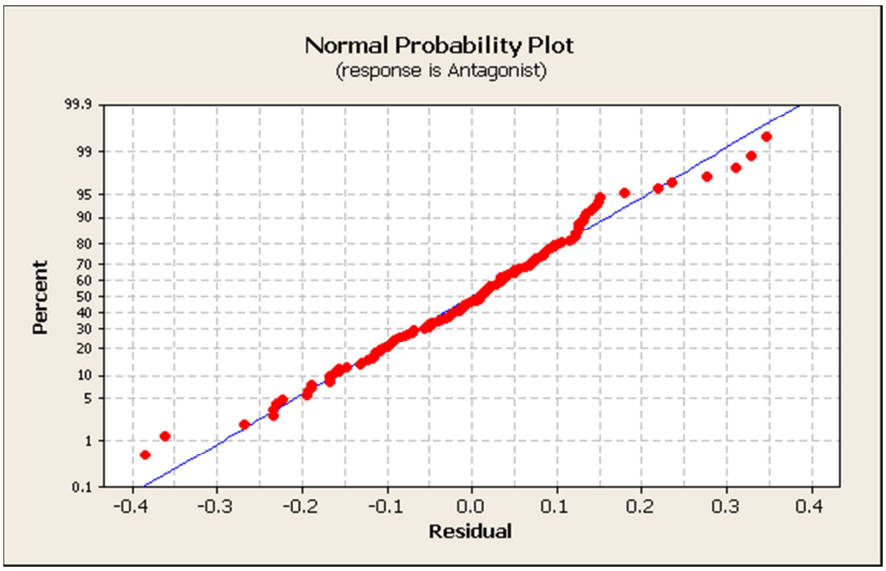

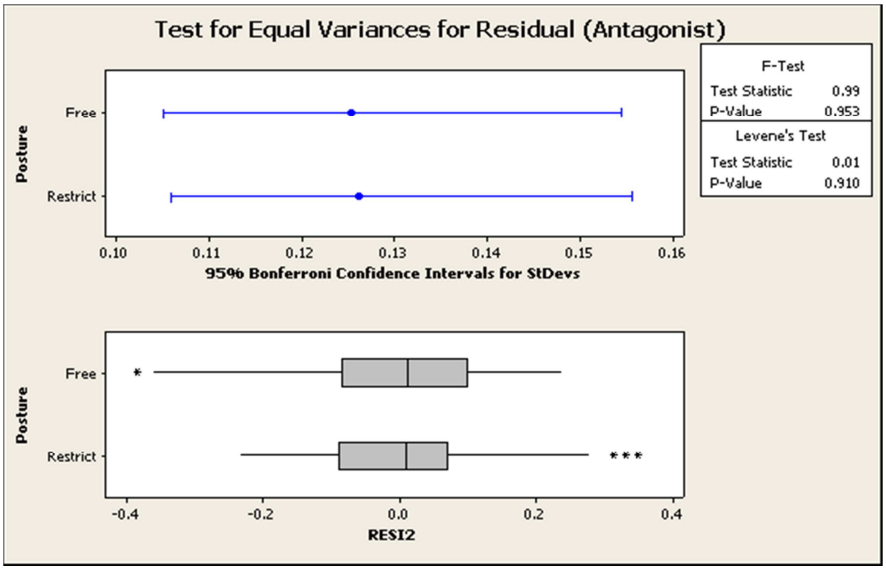

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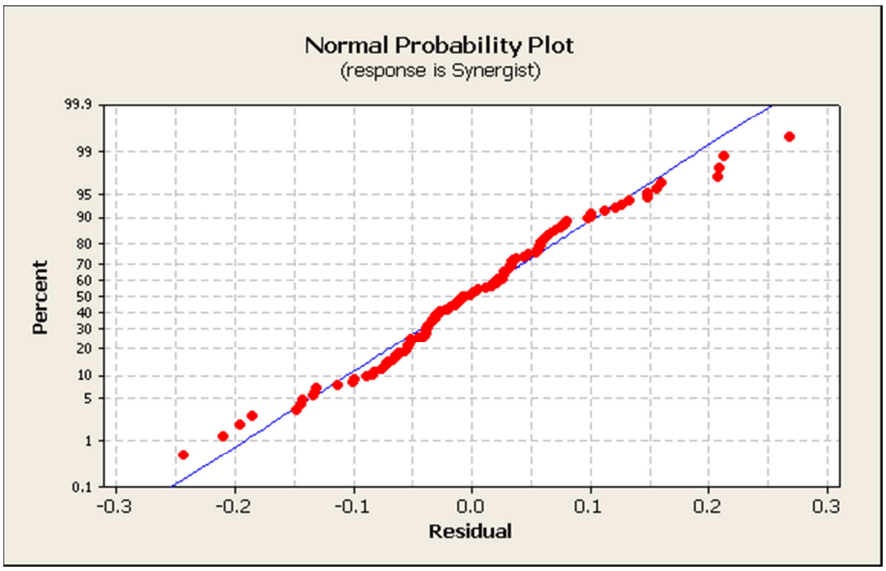
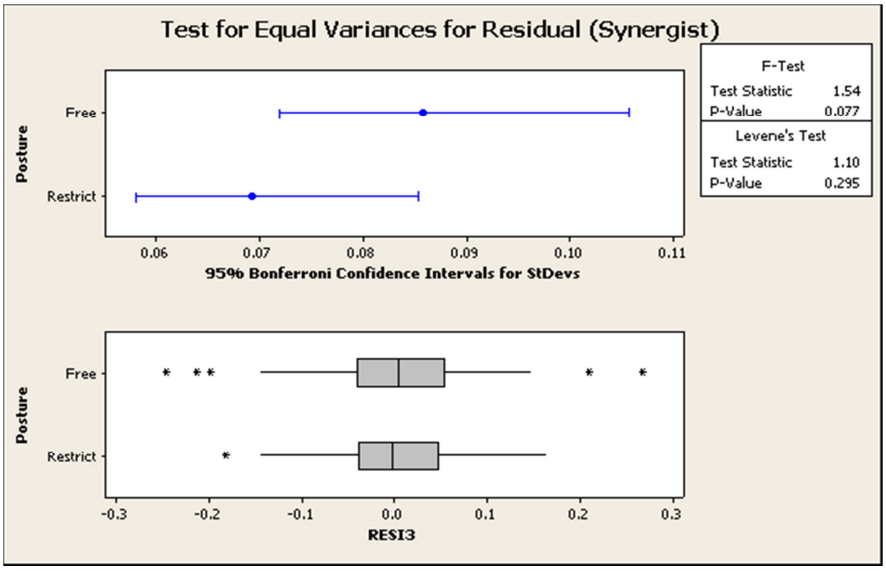
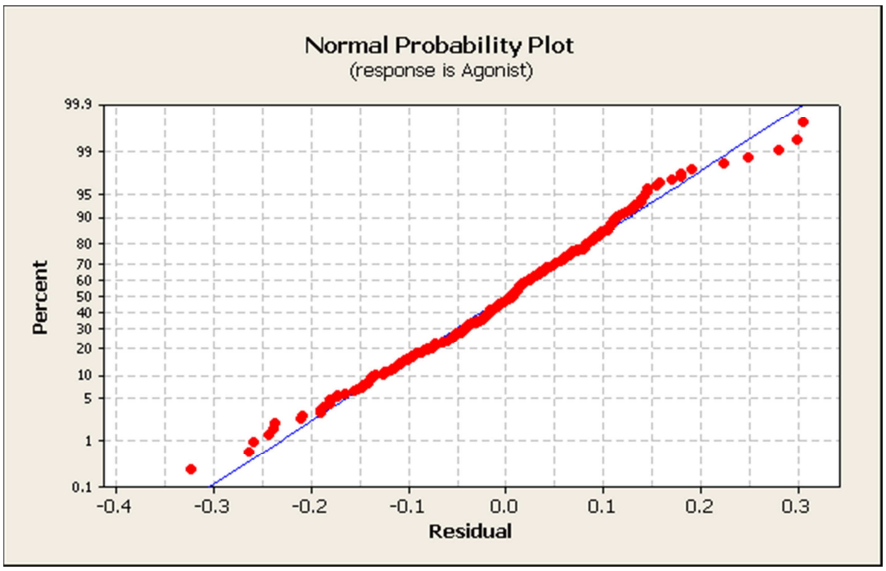
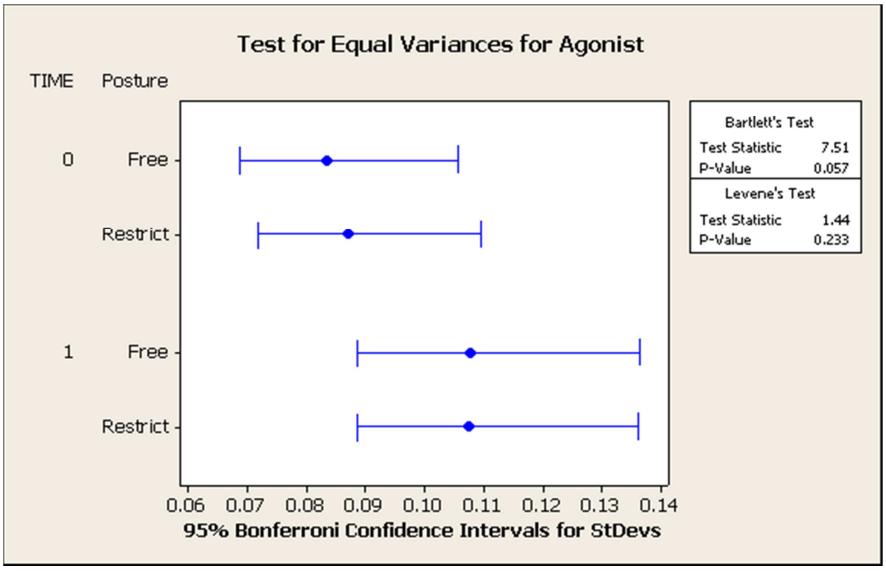


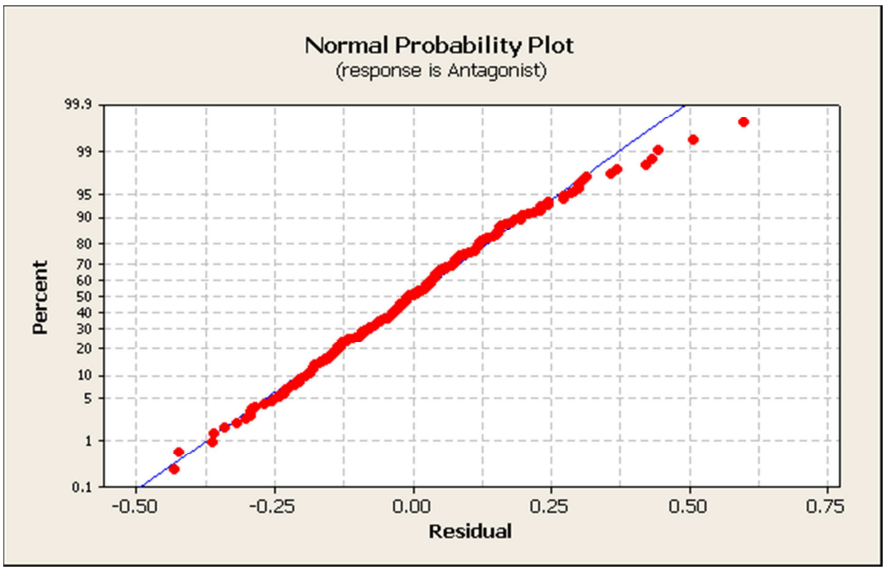


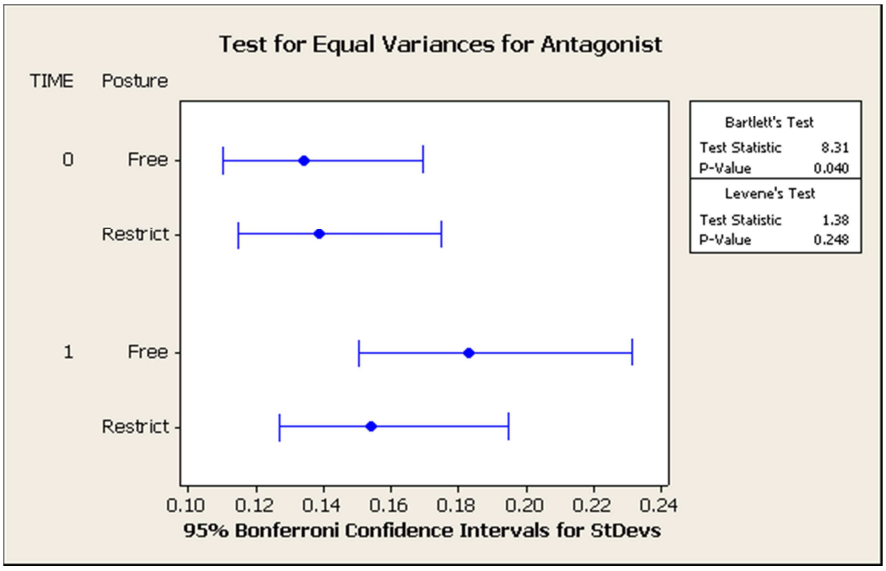
Model for Hypothesis 7 – Two-way ANOVA for agonist (transformed)





Model for Hypothesis 7 – Two-way ANOVA for antagonist (transformed)





Model for Hypothesis 7 – Two-way ANOVA for synergist (transformed)