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A Quantification of Deep Core Trunk Muscles Impact on Lumbar Lordosis and Spine Stability

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A Quantification of Deep Core Trunk Muscles Impact on Lumbar Lordosis and Spine Stability

by

David Aaron Moody

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requirements for the degree of

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in

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Committee in charge:

Professor Oliver M. O'Reilly, Co-Chair
Professor Jeffrey C. Lotz, Co-Chair
Professor Tony M. Keaveny
Professor Steven L. Lehman

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Abstract

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The lumbar multifidus (LM) plays a unique role for adjustment and support of lumbar lordosis. Although exercise therapies have shown that strengthening the multifidus produces reductions in low back pain, the quantification of the LMs role is undetermined. The goal of this *in vitro* study was to test the hypothesis that LM atrophy significantly reduces lumbar lordosis for spines in the upright posture.

The paraspinal muscles of six fresh human thoracolumbar spine (T12-pelvis) were surgically removed, leaving the spinal segments intact. The T12 level was fixed to a torso plate that facilitated anatomically appropriate trunk muscle attachment and dead weight simulation of torso weight (245 N). The pelvis was attached to a custom fixture that allowed pelvic tilt adjustment to the appropriate value for upright stance. In this experiment, three muscle groups were simulated using steel cables: the deep LM, erector spinae (ES), and rectus abdominus (RA). These cables were inserted at anatomically appropriate vertebral sites and were routed directly to digitally controlled torque motors. A total of five loading cases were investigated for three sets of experiments: neutral posture, axial rotation, and flexion/extension stability perturbation.

Results indicated a small change in lumbar lordosis for L4/L5 motion segment for atrophy while in the neutral posture. The data highlights how variations in muscle activation patterns alter lumbar lordosis and tissue stress distribution.

To my parents
who kept asking me,
“Did you finish your chapter?”

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Acknowledgments

This dissertation presents the work that was influenced, directly or indirectly, by a number of people that I would like to specifically express my gratitude to.

I would like to begin by thanking the National Science Foundation (NSF). I was a recipient of an NSF Graduate Research Fellowship. In addition to the NSF fellowship, I was a recipient of the University of California at Berkeley (UCB) Chancellor's fellowship. My work not only benefited from these sources of funding but from the support provided by NSF Grant CMMI 0726675 to my coworkers and advisors. I refer to my coworkers and advisors as my partners in crime and have provided an image of them in Fig. 1. I express my deepest gratitude to my advisors, Professors Oliver M. O'Reilly at UCB and Jeffrey C. Lotz at the University of California at San Francisco (UCSF) for their thorough guidance in this work. I know that the success of this project would not have been possible without their help. I am also grateful to Professors Tony M. Keaveney and Steve L. Lehman for serving on my dissertation committee.

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I'd be greatly remiss if I didn't thank those who have provided me the support and understanding throughout my entire life. I give God all the praise, and I love my parents (Curtis J. Moody Sr. and Elaine S. Moody) and siblings (Curtis J. Moody Jr. and Jonathan D. Moody) for their continual support. My family members were with me in spirit and continued to push me despite all the struggles I faced while working on this project. I don't know if I'll ever be able to thank each of you properly.

Fig. 1 My partners in crime in the Professor Lotz's Research Laboratory in the Department of Orthopaedic Surgery's at UCSF from left to right: Kunwoo Park, Oliver M. O'Reilly, Jeffrey Lotz, Dezba Coughlin, Cory Laws, David Rodriguez, and myself, David A. Moody. Photo by Dr. Daniel Peters.

Chapter 1

Introduction

The Institute Of Medicine (IOM) reported in 2001 that the lifetime prevalence of lower back pain (LBP) approaches 80% among United States (US) workers [52] with the annual prevalence of back pain ranging from 15% to 45%, with point prevalences averaging 30% [5, 6]. Back pain is the most common cause of activity limitation in people under the age of 45 in the US, the second most frequent reason for visits to a physician, the fifth-ranking cause of admission to the hospital, and the third most common cause of surgical procedures [6, 44, 92, 112].

There are many types of LBP with the most painful being chronic low back pain (CLBP). In a US study conducted in 1988, patients suffering from CLBP had a prevalence as high as 90.1% during years 45-64, 80.5% during years 18-44, and 75.9% during years 65-74 [92]. Patients experiencing CLBP often seek treatment. Common treatments of CLBP include acupuncture, and in extreme cases, surgery (spinal fusion, artificial disc replacement (ADR) or total disc replacement (TDR)), and finally, physical therapy. Patients choosing acupuncture often experienced mixed results. Sawazaki *et al.* [103] showed that acupuncture can be an effective treatment for back pain, but the problem remains in reproducibility of results in the research community [77]. Those who suffer extreme cases of CLBP tend to choose surgery due to lack of understanding of the etiology coupled with the few therapeutic inventions with proven clinical benefits [17, 34]. Despite choosing ADR/TDR over spinal fusion for its advantages, increase in mobility/range of motion, there is no evidence that disc replacement reliably, reproducibly, and over longer periods of time fulfills the three primary aims of clinical efficacy: continued motion, and few adjacent segment degenerative problems [59]. Additionally, the efficacy of spinal fusion remains unclear due to the high rates of reoperation and complications [33]. Surgical options should only be considered under special circumstances.

Patients who decided to participate in physical therapy experience better results provided they follow an appropriate regime [17]. Additionally, studies have shown that partic-

ularly among obese, decreased trunk muscle strength are key factors in CLBP and trunk muscle strengthening is helpful in reducing the pain [9]. Furthermore, the only exercise therapy that has been shown to achieve substantial and lasting reductions in pain are special exercises directed at coactivation of the abdominal and multifidus muscles [17, 38, 39, 74, 99, 114]. This research will focus on coactivation of the abdominals and multifidus muscles.

The research efforts on multifidus and coactivation of the abdominals found that multifidus atrophy (50% or more reduction in muscle activity) has been linked to CLBP, likely due to alterations in lumber posture and kinematics. In support of this, human volunteers subjected to prolonged bedrest develop multifidus atrophy that correlates with loss of lumbar lordosis (approximately 1.5 degrees at L4/L5; [11]). Similarly, paraspinal muscle atrophy has been linked to a loss of lordosis in patients with degenerative flat back syndrome [63].

Based on this information, three hypotheses were formed. Hypothesis 1: force comparable to that of moderate to severe atrophy of the deep lumbar multifidus will change lumbar lordosis. Hypothesis 2: Force comparable to that of moderate to severe atrophy of the deep lumbar multifidus produces a greater change in spinal curvature during axial rotation than during neutral posture. Hypothesis 3: Force comparable to that of moderate to severe atrophy of the deep lumbar multifidus produces a significant reduction of spinal stability when the spine is perturbed in flexion or extension from the neutral posture. The goal of the research question was twofold: first, to provide physicians and patients with additional benefits of non-surgical options for CLBP and, second, to provide future researchers with additional information on the contribution between the muscles versus the disc properties.

In order to test the three hypotheses it was necessary to create a physiologic representative mechanical testing apparatus. To support the research, a robust, physiologic representative mechanical quasi-static testing device that simulates flexion, extension, and axial rotation was developed. The testing apparatus presented here has the ability to conduct dynamic testing which is ultimately more desirable than quasi-static testing. This novel testing apparatus and corresponding data are discussed in Chapters 2 -5.

Chapter 2 begins with a brief overview of the anatomy of the human spine and continues with a literature review of various types of spinal testing: pure moment, follower load, and a hybrid loading system. Additional details on muscle anatomy are found in Appendix A. In particular, we conducted thorough investigations of Patwardhan *et al.* [90] work on a spine testing apparatus used for testing the cervical section of the spine, Wilke *et al.* [124] award winning work of an alternate spine tester for the lumbar spine, and finally, Welke *et al.* [120] innovative spine testing device that tests two vertebral bodies versus an entire section. Following the detailed analysis of their work, limitations are provided and discussed in detail to further motivate the purpose for this work. The next subsequent section continues with a brief discussion of spinal stability for this work due to the tremendous variability of definitions depending on the research community i.e., biomechanics community as opposed to the physical therapy community. Finally, the chapter ends with a brief overview of the

multi-body model used versus a finite element or complex intervertebral body models.

Chapter 3 expands on the motivation for the spine testing apparatus provided in Chapter 2 and provides details on the novel design of the spine testing apparatus used in this dissertation. Following the presentation of the spine testing apparatus the rationale for muscle selection and postures are provided. Intimate details of the muscle groups are provided in Appendix A. The chapter ends with the testing methodology for flexion, extension, and axial rotation for the standing posture. More specifically, a testing protocol for simulating the standing posture for three experiments.

Using testing methodologies featured in Chapter 3, the results from the testing are presented in Chapter 4. The three test procedures: neutral posture, right and left axial perturbation, and finally, two stability perturbations: flexion and extension, with varying loading conditions. The dissertation concludes, Chapter 5, with a thorough discussion of the outcomes of this work as well as recommendations for future work.

In closing, the testing of whole thorocolumbar (T12-Sacrum) using a sophisticated pulley system representing the trunk muscles during flexion and extension is a more accurate physiologic simulation of the lower spine than present testing methods, thereby resulting in a testing protocol that can be of great clinical significance. The results provided in this work will further educate physicians, patients, and researchers on the importance the deep cores muscles play in reduction of chronic low back pain.

Chapter 2

Background on Biomechanical Testing of the Spine

This chapter connects all the relevant information on the anatomy of the spine, comments on clinical and structural spinal stability, and concludes with an overview of previous testing measures to motivate this work. A discussion of a multi-body model for the human spine is included.

The data in this chapter will leverage several other works. The main sources for review of spine anatomy are from Bogduk *et al.* [14, 15, 16, 18, 19]. Much of the work presented on stability follows the work of Panjabi *et al.* [83]. Sources for relevant testing methodologies are Patwardhan *et al.* [90], Welke *et al.* [120], and Wilke *et al.* [124]. Each of these researchers present their own unique testing methodologies which have made tremendous contributions to the spine research community. Their contributions to the research community are discussed as well as identifying some of their limitations, following an overview of the two most common testing protocols: pure moment and follower load. Finally, the chapter concludes with a presentation of a multi-body model for the spine based on the work of Christophy *et al.* [26].

2.1 Anatomy and Motion of the Spine

The human spinal column is composed of three main sections: cervical, thoracic, and lumbar as shown in Fig. 2.1. These sections are characterized by their curvatures: cervical lordosis, thoracic kyphosis, and lumbar lordosis. The cervical spine starts at the skull and is composed of seven vertebral bodies, C1-C7. Following the cervical spine is the thoracic spine consisting of twelve vertebral bodies, T1-T12. Finally, the lumbar spine contains five vertebral bodies, L1-L5. It should be noted that there is a section below the five lumbar vertebrae, the sacral spine, S1, and coccyx, which are not directly identified in

Fig. 2.1. The sacral region and coccyx are different from the lumbar, thoracic, and cervical regions because they contain fused vertebral bodies rather than individual vertebral bodies separated by intervertebral discs. These vertebral bodies and intervertebral discs allow the spine to move in three planes: coronal, sagittal, and transverse as seen in Fig. 2.2.

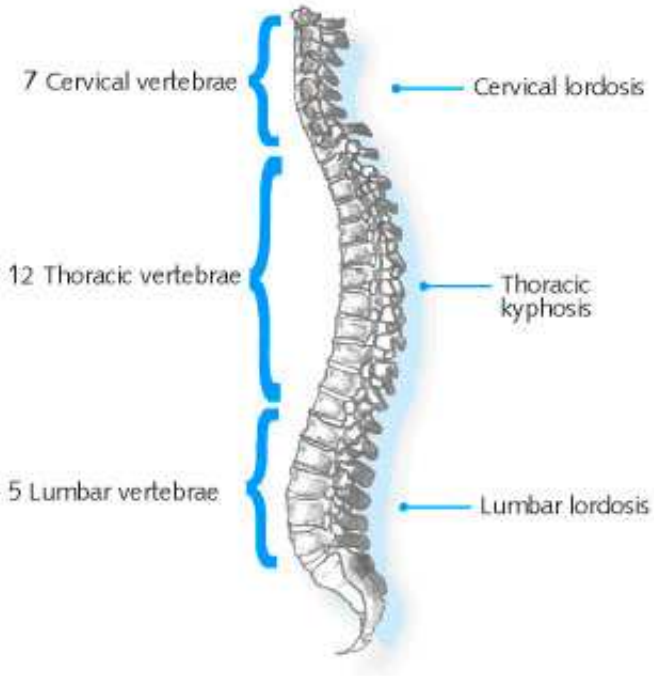

Fig. 2.1 Lateral view of the human spinal column indicating the three main spinal sections and curvatures: cervical lordosis, thoracic kyphosis, and lumbar lordosis. The citation for the source image can be found in Appendix B.

Motion in these planes is achieved by the activation of the 238 muscle fascicles in the trunk as shown in Fig. 2.3.[1] These muscles enable the spine to move in flexion-extension (trunk moving forward/backward) in the sagittal plane, lateral bending (trunk moving side-to-side) in the coronal plane, and axial rotation (twisting of the trunk) in the transverse plane. Thus, replication of *in vivo* motion during *in vitro* experiments are rather challenging. Two of the most common spine testing protocols used to mimic *in vivo* motion are known as pure moment and follower load. These methods do not capture the full essence of spinal motion and loading. Thus, hybrid testing methods have been developed to help close the gap.

[1] Additional details on muscle anatomy can be found in Appendix A.

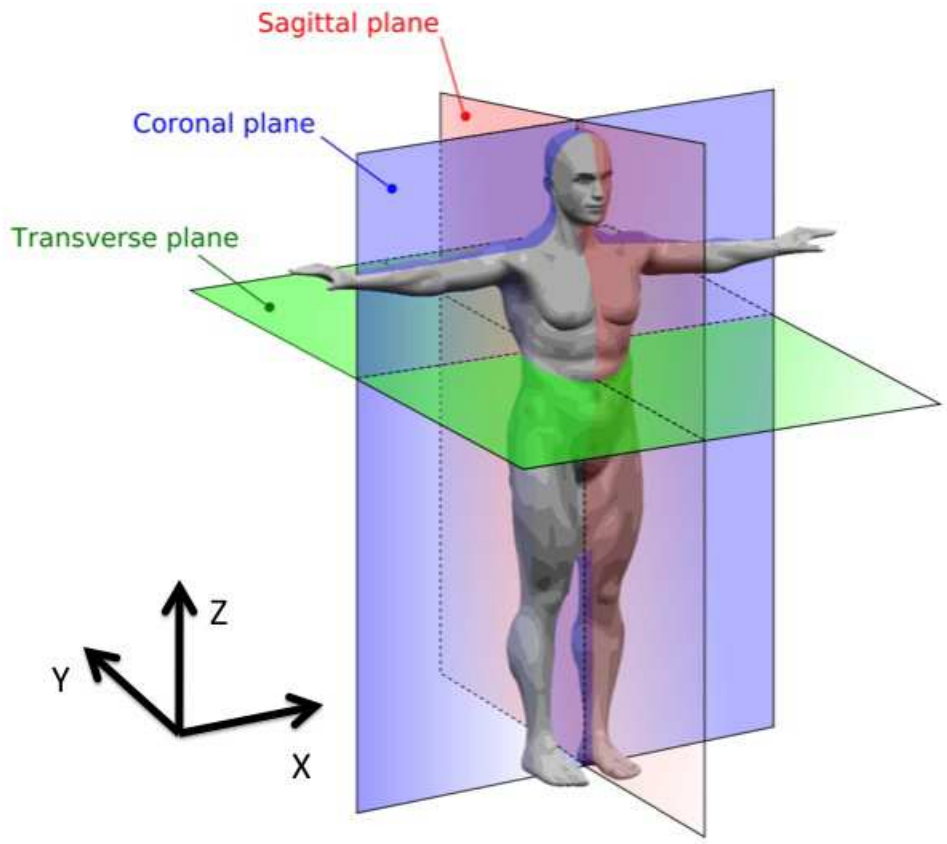

Fig. 2.2 Coronal, sagittal, and transverse planes for the human spine. In terms of X, Y, and Z coordinates, the coronal plane is the equivalent of the X-Z plane, the sagittal plane is the equivalent of the Y-Z plane, and finally, the transverse plane is the equivalent of the X-Y plane. The citation for the source image can be found in Appendix B.

2.2 Clinical and Structural Stability

The human spine supports compressive loads in the range of 1000 N during standing and walking. However, this load increases to several thousand Newtons during various lifting activities [91]. Without the presence of the supporting muscles, it has been estimated that the spine would buckle under a simulated compressive load of 90 N [31]. The trunk muscles serve two main purposes: to actuate the spine, resulting in dynamic motion, and to stabilize the spine during these motions and daily activities, static or dynamic, preventing the spine from buckling. Although the muscles are critical to preventing buckling and maintaining a stable spine, there is great debate in the research community on the precise meaning of the term 'stability' with respect to the human spine.

There are two types of spine stability: structural and clinical. Structural stability gen-

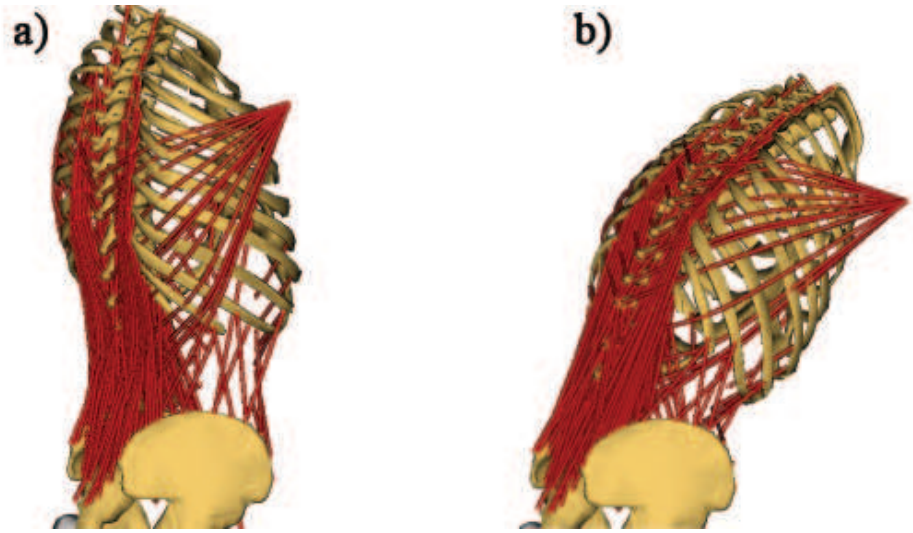

Fig. 2.3 An OpenSim multi-body model of the human spine using all 238 muscle fascicles of the human spine: a) Simulated spine in the standing posture, b) Simulated spine in the standing flexed position in the sagittal plane. The citation for the source image can be found in Appendix B.

erally refers to quasi-static mechanical stability which relates stiffness of the system with its accompanying stability as a function of back pain. In the words of Reeves *et al.* [96], "To discuss stability of a system, whether it is in equilibrium (static) or changing with time (dynamic), we must give a small perturbation and observe the new behavior. If the new behavior is approximately the same as the old, qualitatively speaking, the system is stable." To measure this definition of stability, a number of metrics have been developed and are typically defined in relation to back pain. There are six metrics commonly used to quantify this quasi-static stiffness: neutral zone (see, for example, [80, 82, 84]), range of motion (see, for example, [27]), motion patterns (see, for example, [65, 69, 70, 71]), muscle activation patterns (see, for example, [24, 40, 42, 48, 55, 107]), and finally, reflex response to a sudden permutation (see, for example, [25]). This wide range of metrics results in a broader definition of stability and thus leads to a definition of clinical stability. Panjabi *et al.* [122] defined clinical stability as, "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, in addition, to prevent uncapacitating deformity or pain due to structural changes. Any disruption of the spinal components (ligaments, discs, facets) holding the spine together will decrease the clinical stability of the spine. When the spine loses enough of these components to prevent it from adequately providing the mechanical functions of protection, measures are taken to reestablish the stability." To compare this definition of stability to mechanical stability, clinical stability is the systems"robustness," or rather, its ability to cope with uncertainties in the presence of disturbances or perturbations. The more 'robust' the system, the greater likelihood the system can handle larger perturbations and

changes in trunk stiffness [95].

This work will define spinal stability, rather a spinal stability system similar to Panjabi *et al.* [83], in that the spinal stabilizing system can be thought of as consisting of three distinct yet functionally interdependent subsystems: the passive musculoskeletal subsystem (consisting of the vertebral bodies, articulating facets, intervertebral discs, spinal ligaments and passive mechanical properties), the active musculoskeletal system (comprised of the muscles and tendons surrounding the spinal column), and finally, the neural and feedback system (encompassing various force and motion transducers, located in the ligaments, tendons, muscles, and neural control centers.). Although these systems are separate yet functionally interdependent, it was not possible at the time of this study to fully replicate the neural control system during testing. From this point forward the spinal stability system will consist of the passive (vertebral bodies, articulating facets, intervertebral discs, and spinal ligaments) and active (muscles and tendons surrounding the spinal column) musculoskeletal subsystems.

It is important to note that by focusing on all the trunk muscles that are active during daily activities, the picture of stability becomes almost impossible to analyze. Furthermore, not all active musculoskeletal subsystems directly contribute to stabilizing the spine. A number of assumptions, such as which muscles are active during various postures and activities, must be made in order to simplify the system. Previous studies have shown that stability in the spine increases with application of different muscle groups [127]. The lumbar multifidus muscle group in particular has proven to be essential in daily activities. A degenerated or weakened lumbar multifidus has been linked with chronic back pain in adults [45, 46] and particularly in astronauts when they return from orbit [11]. In addition, MacDonald *et al.* [66])conducted a thorough literature review of the impact of the deep lumbar multifidus and found three significant clinical implications. First, that the deep lumbar multifidus is advantageous to the nervous system due to its ability to control interveretbral shear and torsion without generating torque. This control is helpful because it doesn't require muscle co-contraction of antagonists. Second, the deep lumbar multifidus is more active during postural perturbations than the more superficial multifidus. Thus, for postural perturbations it is best to simulate the deep lumbar multifidus. Finally, it was noted that there is still a great need to investigate the correlation between postural activity of the deep lumbar multifidus regarding low back pain. Therefore, this small yet powerful muscle group, the lumbar multifidus, is not only essential in reducing chronic low back pain but in improving the spine stability, and thereby helping to support a more robust lumbar spine.

Another factor that influences stability of the spine is the intervertebral disc degeneration. Kirkadly-Willis *et al.* [58] defined clinical spinal degeneration as occurring in three phases or stages: temporary dysfunction, unstable, and stabilization, with the idea being that the initial degeneration leads to dysfunction followed by a period of instability before the spine re-stabilizes. To further complicate matters, the duration of these phases or stages varies from individual to individual. Couple this with the fact that disc degeneration is not only common but a natural part of the aging process [12], and the results allude to a rather depressing future. O'Sullivan *et al.* [81] has shown that exercises directed at the abdominal

and lumbar multifidus have proven to be effective in reducing pain and function disability in patients with specialized spine disorders but have implications for the general population suffering from low back pain when 'instability' of the lumbar spine is suspected.

As stated previously, the only exercise therapy that has been shown to achieve substantial and lasting reductions in pain are special exercises directed at coactivation of the abdominal and multifidus muscles [17]. Thus, an *in vitro* experiment that simulates coactivation of the abdominal and multifidus would be of clinical significance to further understanding chronic low back pain.

In summary, the work presented in this research focuses on specific aspects of clinical and structural stability of the human spine. More specifically, the role the deep lumbar multifidus plays in dynamics of the human spine using stability as one measure of biomechanics.

2.3 Biomechanical Testing

Many of the biomechanical tests that researchers use for spinal testing focus on the evaluation of total disc replacements or fusion devices. These tests are paramount to expediting the design-to-clinical trial-to-patient use cycle. There is a wide range of procedures and very little standardization (see, for example, [57, 61, 76, 86, 121]). This spectrum of testing procedures is not surprising given the complexity of the human spine. However, the variability of spine samples makes it difficult to correlate results from the various tests. Consequently, several standardized testing procedures have been championed. The three types of standardized testing are: pure moment, follower load, and a hybrid loading system.

2.3.1 Pure Moment Loading

The origin of the pure moment can be traced back to classical mechanics where a pure moment can be defined as a system of forces applied with a resultant moment but has no resultant force [56]. Thus, many researchers construct custom mechanisms similar to Fig. 2.4 where it can be seen that the sum of the forces are zero but a moment is applied.

In the words of Equizabal *et al.* [37],"Pure moment biomechanical conditions are preferred because they ensure uniform loading along the column of the spine easing the comparison of techniques of previous literatures[1]." Therefore, many researchers use pure moments in model simulations and *in vitro* experiments (see, for example, [8, 10, 13, 29, 35, 41, 73, 85, 86, 105, 123, 128]).

2.3.2 Follower Force Loading

Although the pure moment protocol has proven useful in the biomechanics testing community, pure moments do not paint a complete picture of how the human body applies loads in the spinal region. More specifically, a pure moment is not able to fully simulate the

[1]Equizabal *et al.* observation is based on upon the works of [125, 126].

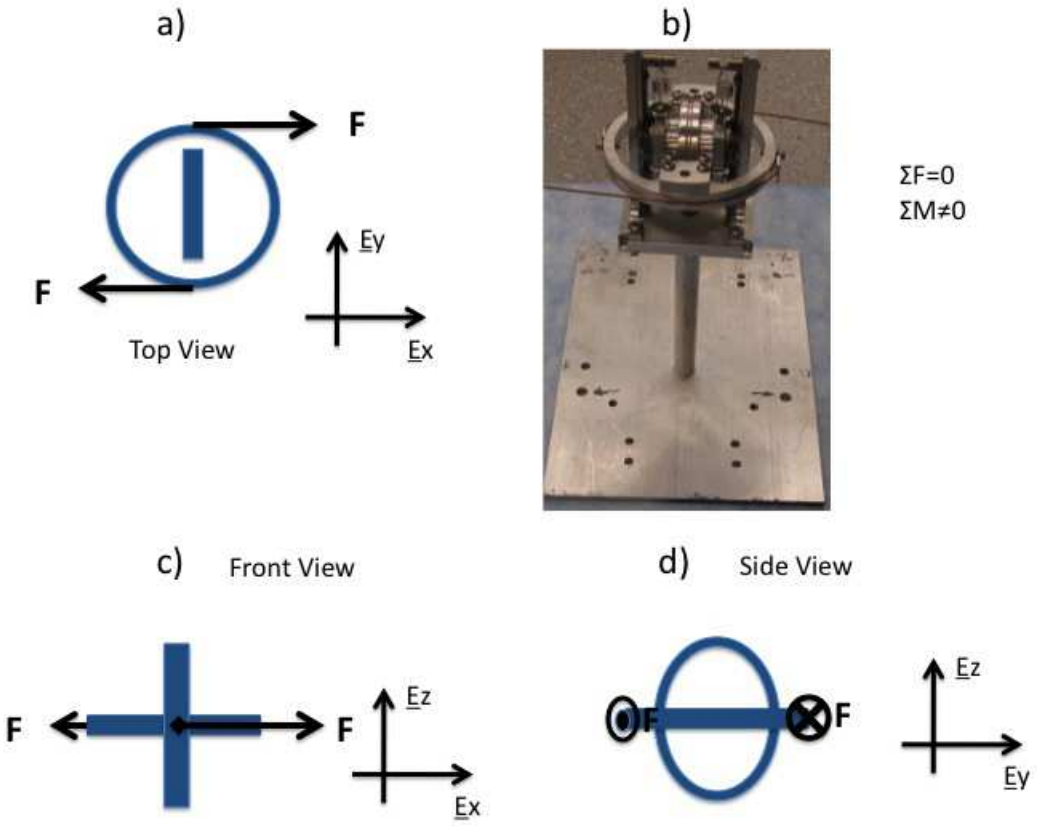

Fig. 2.4 Custom pure moment fixture for flexion, extension and axial rotation. Fixture is attached to top of pole of torso plate used for trunk weight simulation: a) Top view of custom pure moment fixtures, b) Isometric view of custom pure moment fixture attached to torso plate pole used in testing apparatus, c) Front view of custom pure moment fixture, d) Front view of pure moment fixture.

constant compressive preload the spine maintains during physiologic motion. Thus, other testing methodologies have been developed. One such methodology is the follower load. A follower load is a non-conservative load whose line of action follows a structural member [20, 131]. The purpose of the follower load is to simulate a compressive preload and replicate the function of local muscle groups. Nachemson *et al.* [75] was the first to investigate applying compressive loads for daily activities however, Panjabi *et al.* [87] was one of the first to include the effects of preload with the application of physiologic loads. By building upon these earlier works, many researchers [89, 91, 98, 101] were able to demonstrate that the spine could maintain a compressive load of 1200 Newtons in the neutral posture. It has been estimated that there are 1000 N of compressive force on the lumbar spine during standing and walking whereas, the compressive loads increase to thousands of Newtons for

lifting activities [91].

Despite the fact that previous researchers have been able to demonstrate that the spine can experience these large compressive loads during *in vitro* experiments, the method in which these compressive loads are applied (see for example Fig. 2.5) are unphysiologic. Thus, the follower load is useful in proving the concept of the spine being able to maintain a large compressive load without buckling but loses value when searching for a more robust physiologic testing solution. A more appropriate testing protocol would be a combination of the previous two methods with an alternate application of the follower load.

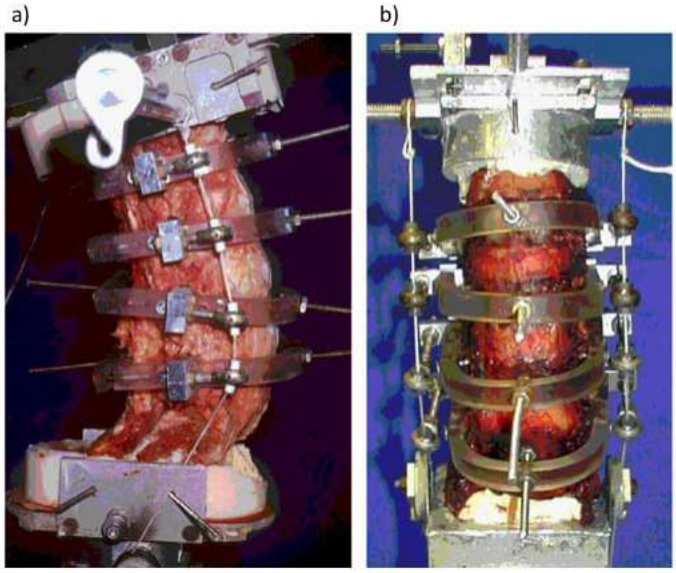

Fig. 2.5 Custom lumbar follower load: a) Lateral view of the *in vitro* follower load technique, b) Anterior-posterior view of the *in vitro* follower load technique. The citation for the source image can be found in Appendix B.

Patwardhan *et al.* [90] was the first to develop a more physiologic representative testing apparatus using a follower load on a whole cervical spine as shown in Fig. 2.6. The benefit of this tester was the ability for the cervical spine to maintain a lordotic posture while withstanding larger compressive *in vitro* loading without any damage or instability. The ability to simulate these large compressive loads while maintaining lordosis and avoiding stability is critical to simulating *in vivo* motion. For example, without the presence of the follower load or simulated muscles, the lumbar spine would buckle under a compressive load 90 N, a load which is typically less than the upper torso [31] during *in vitro* loading. This work led researchers to believe that more advanced *in vitro* testing was possible on

entire regions of the spine, cervical or lumbar, without the spine becoming unstable and maintaining a physiologic posture. This inspired researchers to develop several hybrid loading apparatuses that conceptually mimicked a follower load protocol but replaced the follower load with simulated muscles.

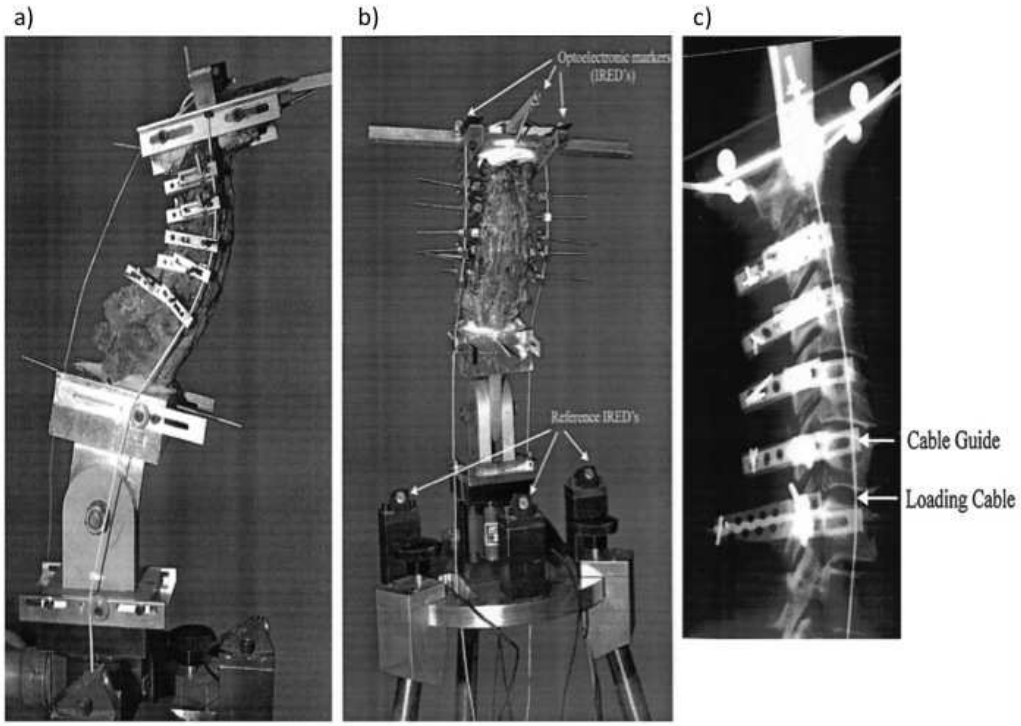

Fig. 2.6 A human cervical spine (C2-T2) subjected to a compressive follower load: a) Lateral view of novel fixture, b) Front view of novel fixture, c) Lateral x-ray of follower load highlighting cable guiding mechanism. The citation for the source image can be found in Appendix B.

2.3.3 Hybrid Loading Systems

Even though pure moment and follower load testing methodologies have proven useful, they are unable to fully represent physiologic loads and motion. In particular, they are limited in what information they provide in regards to muscle activation and spine response. As Wilke *et al.* [127] discovered, stability in the spine is increased with multiple muscle groups simulated during *in vitro* experiments. Knowing this, Wilke *et al.* [124] developed one of the most robust lumbar spine testing apparatuses as seen in Fig. 2.7. This spine tester was the first to include multiple muscle groups for the lumbar spine, Erector Spine (ES),

Rectus Abdominis (RA), a supporting abdominal force, vertical preload and finally, a follower load. This tester was able to simulate lumbar flexion angles between -15°(extension) and +20° (flexion) in 5° increments. Furthermore, this tester could simulate hip flexion angles between 0° and 30° in 10° increments for a variety of loading conditions. This award winning research demonstrated the possibility for *in vitro* loading to closely approximate *in vivo* loading conditions for axial preload and use of local (follower load) and 'global' muscles (RA, ES, abdominal force).

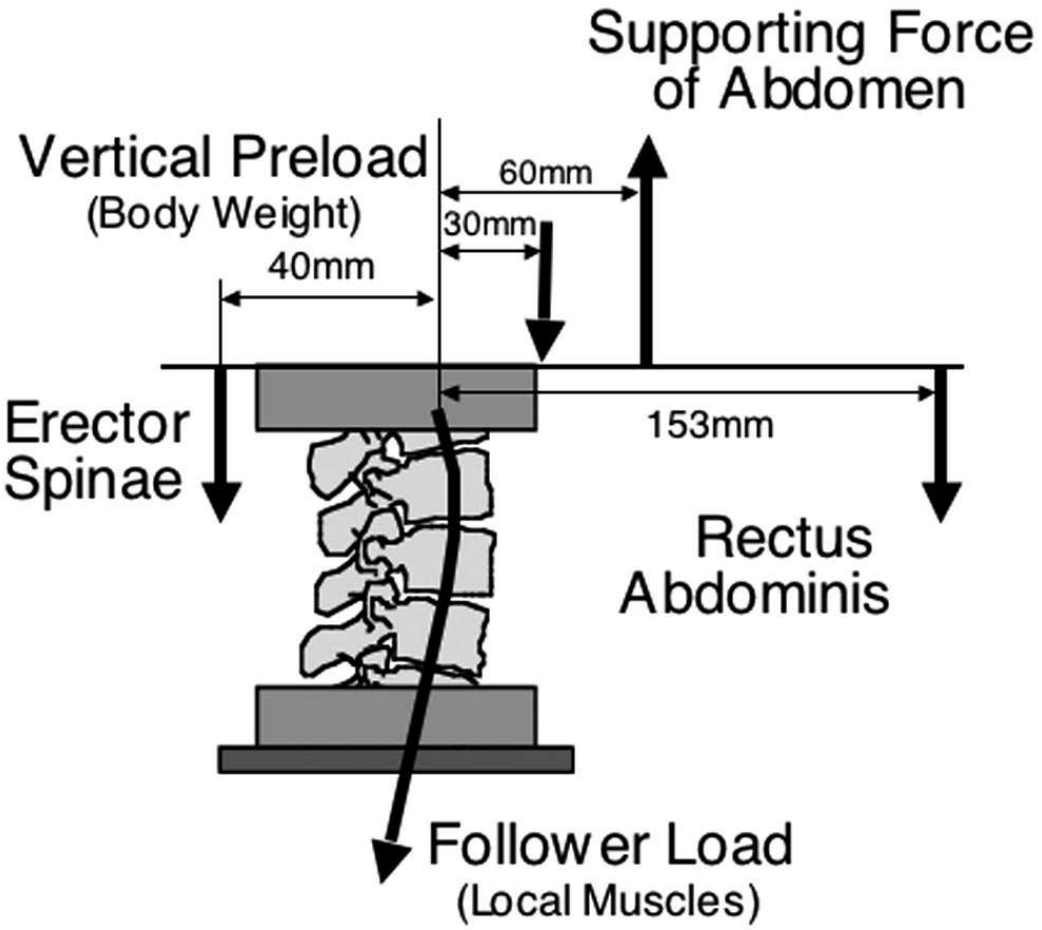

Fig. 2.7 Schematic diagram of the Wilke Spine Tester for loads applied to the L1-L5 spine specimens. The citation for the source image can be found in Appendix B.

2.4 Improvements to Existing Testing Devices

In light of these advantages, it seems difficult to improve upon Wilke *et al.* spine test. However, there is great debate in the research community about the role of the supporting

abdominal force during daily activities [22, 49, 110]. There are those who believe that an increase in intra-abdominal pressure will help stabilize the spine [7]. However, [94] has determined that lumbar support belts, which are used to increase intra-abdominal pressure with the hope of lowering low back pain, did not reduce the incidence of low back injury in baggage handlers. Thus, this lead to the belief that the supporting abdominal force is unnecessary. Second, the use of a follower load implies that all local muscles apply a constant load during routine activities and are less important than the larger, global muscles. This simply isn't the case as a growing number of researchers are finding how important these small muscles groups are to the reduction of chronic low back pain [17].

In an attempt to address these shortcomings, Welke *et al.* [120] designed a similar tester that didn't include a supporting abdominal force and simulated all the muscle groups associated with the lumbar spine as shown in Fig. 2.8. In spite of including all these muscle groups, the tester had its limitations as well. First, it only used two lumbar elements which limited the overall implications for the entire lumbar region. Second, it can be argued that the robot-assisted approach was unphysiologic. Finally, the simulated muscle groups failed to simulate physiologic loads since they all applied the same load.

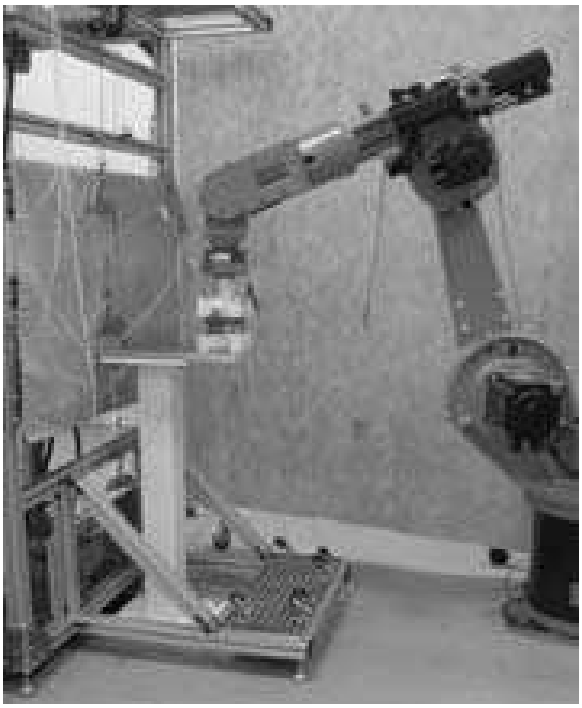

Fig. 2.8 Robot-assisted spine tester for simulation of five muscle groups on a calf lumbar spine segment (L4/L5). The citation for the source image can be found in Appendix B.

Despite the fact all muscles are created equal, they should not be valued as such. Therefore, discrimination is not only useful but necessary during *in vitro* experiments. Welke *et*

al. and Wilke *et al.* each found ways to discriminate that were useful for their hypotheses. In this dissertation, Wilke *et al.*'s work is expanded upon by removing the supporting abdominal force, simulating a local muscle group, the deep lumbar mulitifidus, with physiologic loads in place of the follower load. It it our belief that this modified approach will produce greater results and more physiologic *in vitro* experiments.

In summary, the pure moment is useful because it applies uniform loading along the column, the follower load helped to demonstrate the spine's ability to handle large compressive loads, and the various hybrid testing apparatuses each extended upon the previous two testing methodologies to created a more advanced experiment. Despite their many advances over their predecessors, an upgraded approach is necessary. In the next chapter a detailed novel spine testing apparatus will be presented as well as the testing methodology used for the *in vitro* experiments. However, it is useful to talk about spine stability briefly here before moving forward.

2.5 Models of the Spine

In addition to considering stability and developing a novel spine testing apparatus focusing on the lumbar multifidus, a spine model was necessary for the experiment. Combing through the literature, many researchers have developed complex models of the human lumbar spine (see, for example, [16, 23, 36, 51, 62, 68, 106, 109, 130]). These models can be separated into two distinct groups: multi-body models of the spine and finite element models of the spine.

The investigation begins with finite element models. One major drawback of finite element models is the large computation associated with these models often requires a supercomputer to process them. The researcher is challenged with extracting relevant clinical data from these models [117]. The significant financial resources required coupled with the high level of expertise required to generate a detailed finite element model steered this work towards other models and systems. More specifically, nonfinite element mulitbody models.

In regards to multibody models of the spine, it is imperative that the muscles and tendons represented in the model, which are responsible for production of force and movement, properly reflect the mechanical characteristics. Traditionally, there are three models or categories which are used to describe the mechanical behavior of muscles: cross-bride models, one-dimensional Hill-type three-component models, and three-dimensional continuum models [108]. Use of these models also require accurate prediction of the three muscle contractions: isometric, concentric, or eccentric. These models have often been implemented in finite element studies (see, for example, [54, 60, 64, 115, 129]). The challenge with finite element studies is not limited to proper representation, but also extracting clinically relevant information from these finite element models.

In this work the model of choice was a multi-body model of the human spine based on an OpenSim platform [32] that uses the Hill-type constitutive model for the mechanical

behavior of the muscles and tendons.[1] A complete model of the human spine with all 238 muscle fascicles in the trunk was developed by Christophy *et al.* [26], using OpenSim as shown in Fig. 2.3. The advantage of using the OpenSim platform are the open source nature of the platform which allows greater access for the larger researcher community, its internal inverse dynamics, and the ability to replicate all 238 muscle fascicles in the trunk. Intimate details on this full model are provided in Appendix A. Although this full model is of tremendous value to the research community, the numerous muscle groups were simplified and limited to muscles that were activated during the standing posture. Using these simplifications and assumptions, a simplified model of the human spine was developed and used for this experiment and can be found in Fig. 2.9.

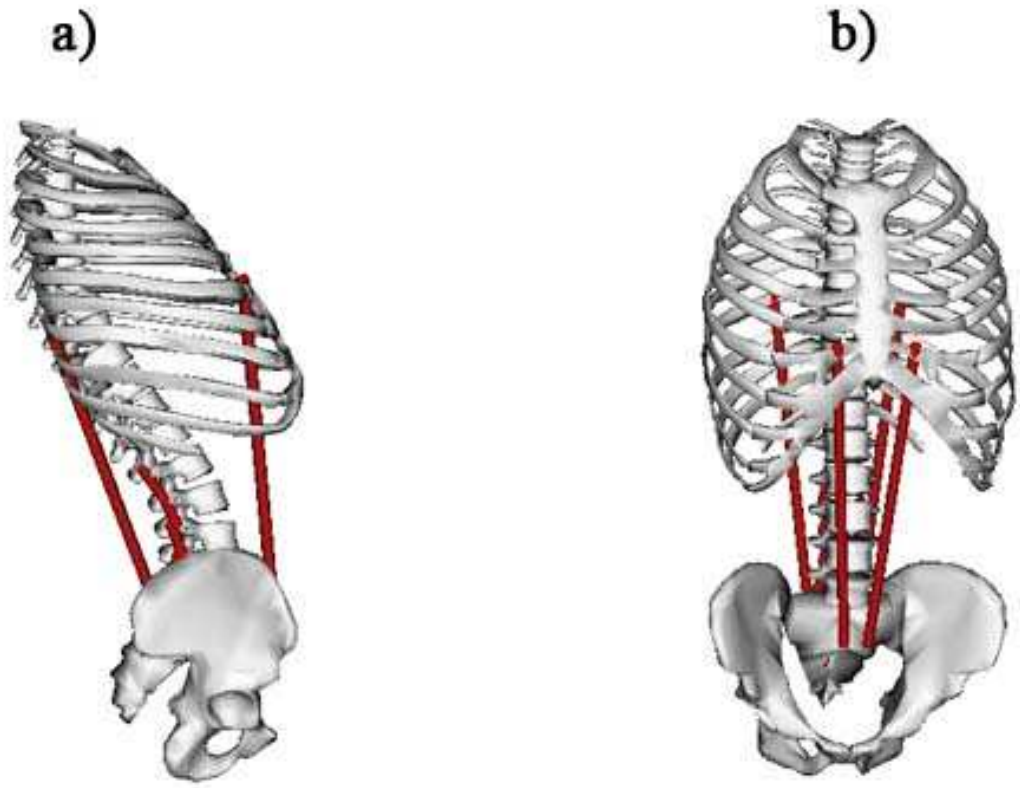

Fig. 2.9 An OpenSim multi-body model of the human spine with three muscle groups (Rectus Abdominis, Erector Spinae, and Lumbar Multifidus) activated during the standing posture: a) Lateral view of simulated spine in the standing posture, b) Front view of simulated spine in the standing position.

[1] Additional details on Hill-type constitutive models can be found in Appendix A.

Chapter 3

Experimental Methods

The human spine is an amazing structure that provides the body with the necessary support and functionality to execute daily activities while protecting the spinal cord. It is arguably one of the most sophisticated parts of the human body consisting of 24 vertebral bodies each separated by an intervertebral disc that allows for motion when the brain's neural system activates a portion of the 238 muscle fascicles needed to execute a desired motion. Thus, replicating all the intricacies of this wonderfully designed portion of the body is extremely challenging. In this chapter, a novel spine testing apparatus is presented that seeks to replicate some of the functionality of the human spine by building upon earlier researcher contributions (which were discussed in Chapter 2) with the hope of developing greater insight into back pain. This unique testing apparatus is a part of a series of steps to better replicate spinal motion so researchers can improve their understanding of spinal function and the role of various muscles during daily activities.

3.1 Testing Apparatus

The testing apparatus described here is similar to that of Wilke *et al.* [124] with two major modifications. First, the follower load has been removed and replaced with the actuation of the deep lumbar multifidus to provide a more physiologic boundary condition. Second, the supporting abdominal force has been removed in response to comments in the research community [3, 21, 53]. The attachment points of the Erector Spinae (ES) and Rectus Abdominus (RA) are consistent with those found in Wilke *et al.*. The forces generated for a neutral standing posture for these two groups was also based on Wilke *et al.*. In order to mimic the multifidus, inserting points were used from Bogduk *et al.* [18] stating that the multifidus attaches to the mammillary processes of the lumbar spine, which is consistent with multifidus modeling of Macintosh *et al.* [67].

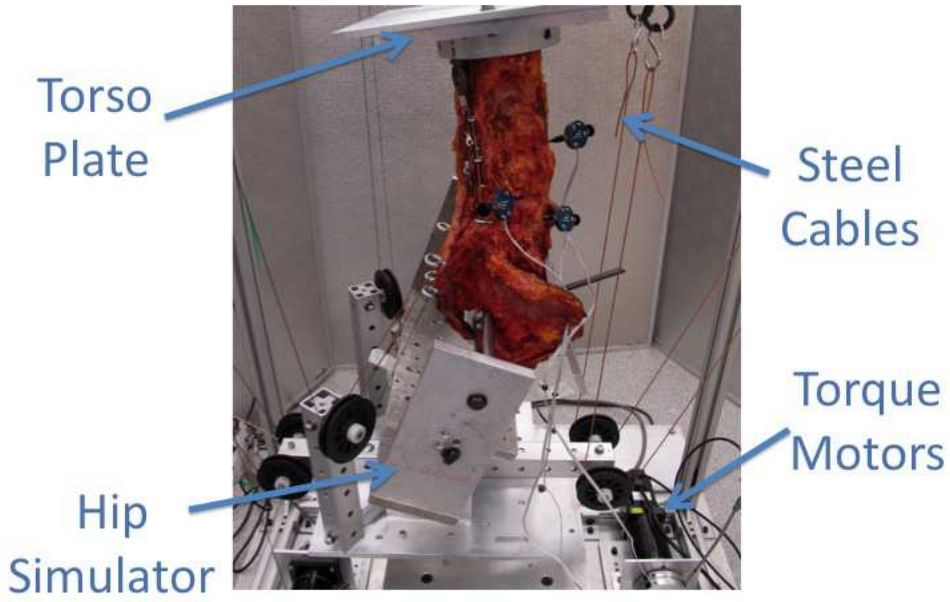

Fig. 3.1 Sagittal view of the lumbar and pelvis with the testing apparatus showing pelvic attachment frame, muscle cables, torque motors, torso plate, and OptoTrak target for measuring vertebral position.

3.1.1 Determination of the Muscle Forces

The determination of the muscle forces for the groups represented was determined by combing the literature and using a variety of best practices. For example, electromyographic data was used to determine potential forces for trunk muscles. Other data was also used for two reasons: the large variability in outcomes are as high as 160% [50] and second, the fact that electromyographic data is only able to be captured at the surface and unable to predict the behavior of deeper muscles, like the lumbar multifidus. Therefore, the data used in this study reflects the physiologic range of force for the multifidus which was found to be between 30 and 90 N [72, 88, 93, 100]. However, El-Rich *et al.* [36] conducted a muscle activation study for standing postures using a combined model and *in vivo* studies and determined a different range for muscle force in the multifidus. Their work suggested an average load of 22.5 N for the multifidus activity in L3-L5 and an average load of 32 N for L4-L5. Based upon their work and the previously mentioned researchers, we selected a constant load of 30 N to simulate the forces generated by the multifidus for this study. Coupling this information with a representative wire loading pattern shown in Fig. 3.2, yielded a improved simulation of the lumbar multifidus during *in vitro* experiments compared to simply lumping the muscle groups together as part of a follower load.

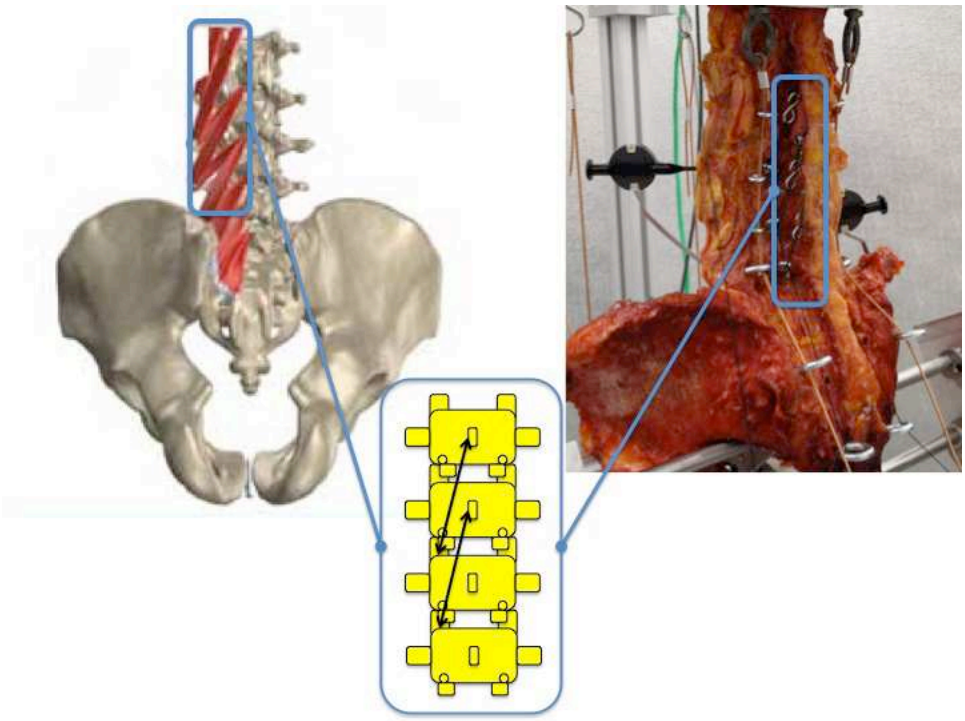

Fig. 3.2 Lumbar Multifidus (LM) simulation for *in vitro* experiments.

3.1.2 Kinematics of the Testing Device

It should be noted that in the testing apparatus shown in Fig. 3.1, the T12 level is fixed to a torso plate that facilitates anatomically appropriate trunk muscle attachment and dead weight simulation of the torso weight. The pelvis is attached to a custom fixture that permits pelvic tilt adjustment to the appropriate value for upright stance [2]. In this test apparatus, we simulated the deep lumbar multifidus (LM), erector spinae (ES), and rectus abdominus (RA) muscle groups using steel cables. The cables were inserted at anatomically appropriate vertebral sites and were routed directly to digitally controlled torque motors. With the help of these guides and motors, physiologic boundary conditions were imposed. However, in order to simulate axial rotation, another device was added to the apparatus shown in Fig. 3.3 which shows a sliding ring cable driven mechanism typically used in pure moment testing. A sliding ring cable driven mechanism was used instead of a traditional fixed ring system because of its increased efficiency of the resultant pure moment [37].

3.1.3 The Possibility of Dynamic Testing

In addition to the various quasi-static tests, a seamlessly transition from quasi-static to dynamic testing was required. To accomplish this task a National Instrument's (NI) real-

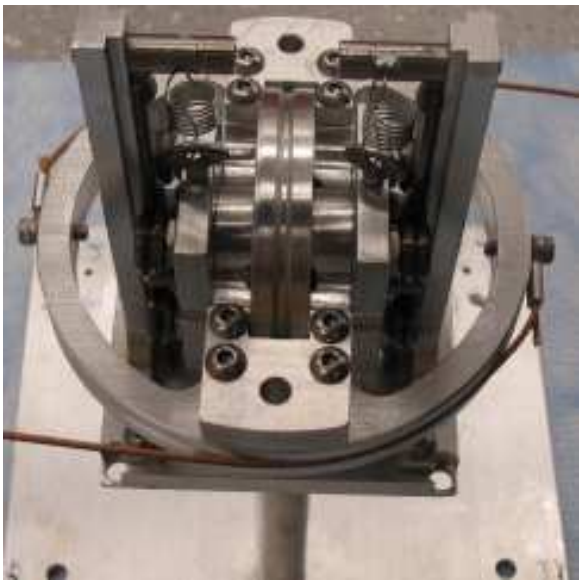

Fig. 3.3 Isometric view of a sliding ring cable driven mechanism used for pure moment testing in axial rotation (with current setup is simulating right axial rotation).

time embedded controller, CompactRIO cRIO-9004 (National Instruments Corporation, Austin, TX). Pairing NI's hardware with their real-time software, LabVIEW Real-Time (National Instruments Corporation, Austin, TX), allowed for PID control loops greater than 1kHz to be used. An added bonus of this controller is that the chassis is modular and thus additional off-the-shelf actuators and sensors can be added by the addition of NI modules. This embedded controller has been programmed to accurately execute the specific tasks (force and positions control loops) and provide safety buffers to the end user. Changing the performance requirements of this controller are realized through manipulation of the sampling rate for smoother operation and close approximation to dynamic motion. The transition from quasi-static to dynamic testing can be realized by making small changes in the software for the controllers instead of dramatic changes to the testing apparatus.

3.2 Testing Methodology

In order for specimens to be included in the study, intervertebral discs could not be herniated and had to have a disc height no less than 50% of sex and level-specific values published in the literature [4, 111]. Previous studies (UCSF internal data) discovered that the aforementioned criteria result in test populations with mild to moderate levels of disc degeneration: Thompson Grade 2 to Grade 4 [113]. Furthermore, specimens were x-rayed to ensure that they were not osteopenic. Using these criteria, 6 fresh human thoracolumbar spines (T12-pelvic) were procured through the UCSF Willed Body Program. Paraspinal

muscles were surgically removed, leaving the spinal segments intact. The T12 was potted into a top potting cup using a urethane plastic, Smooth-Cast®300 (Smooth-On Inc., Easton, Pa) that connected to the torso plate simulator with the pelvis being potted into the hip simulator (see Fig. 3.1). Spines were preconditioned through three cycles using neutral posture, flexion permutation, extension perturbation, and finally, right and left lateral bending perturbation. Each perturbation had a maximum 10° perturbation from the natural posture and provided adequate time for settling between cycles [86]. Cycling through these physiologic ranges helps to reduce the impact of the testing apparatus [30]. Note, these precondition cycles did not simulate full range of motion, rather we limited the range to reflect the potential range of motion during the experiments.

The lumbar vertebral body and torso data was collected using an OptoTrak Analog Data Acquisition Unit associated software (Northern Digital Inc., Waterloo, ON). The positions and orientations of the vertebral bodies (L2, L3, L4, L5) and torso plate were simultaneously collected using a commercial infrared light-emitting diode (IRED) optoelectronic stereophotogrammetry system (OptoTrak 3020 Position Sensor, Northern Digital Inc., Waterloo ON). Furthermore, a total of 15 IREDs were rigidly attached to the vertebra of interest (three IREDs on the torso, and three on each of the vertebral bodies L2, L3, L4, and L5). Prior to testing, all position sensors were digitized to a global coordinate system defined by three points on the frame identifying the x-y and y-x plane. Hirasaki *et al.* [47] found the accuracy of the OptoTrak markers to be 0.1° in rotation, while Schmidt *et al.* [104] determined the translations accuracy to be 0.1 mm with a resolution of 0.01 mm. Each sensor yielded three translations (x,y,z) and three rotations (Rx - flexion/extension, Ry - lateral bending, and Rz - axial rotation) alternatively, in Euler angles: axial rotation (ψ), lateral bending (θ), and flexion/extension (ϕ). Here, the X-Y plane is the transverse plane, X-Z plane is the corneal plane, and finally, the Y-Z plane is the sagittal plane as shown in Fig. 2.2. In terms of an Euler basis, letting X, Y, Z be denoted by $\{\mathbf{p}_1, \mathbf{p}_2, \mathbf{p}_3\}$ and a 3-2-1 set of Euler angles, results in the following expressions for the Euler basis vectors:

$$\begin{bmatrix} \mathbf{g}_1 \\ \mathbf{g}_2 \\ \mathbf{g}_3 \end{bmatrix} = \begin{bmatrix} 0 & 0 & 1 \\ -sin(\psi) & cos(\psi) & 0 \\ cos(\theta)cos(\psi) & cos(\theta)sin(\psi) & -sin(\theta) \end{bmatrix} \begin{bmatrix} \mathbf{p}_1 \\ \mathbf{p}_2 \\ \mathbf{p}_3 \end{bmatrix} \tag{3.1}$$

As discussed in detail by Crawford *et al.* [28] and O'Reilly *et al.* [78], the 3-2-1 is the optimal choice of Euler angles for this particular set of experiments.

3.3 Neutral Posture

Initially, each spine was placed in a neutral posture with a minimal trunk load (22.24 N from the torso and pure moment fixture) such that the torso plate angles (ψ, θ, and ϕ) were as close to zero as possible following three cycles of preconditioning. Following preconditioning and manual neutral posture placement of the torso, measurements were taken for each of the IREDs for a period of three seconds. After a waiting time of 30 seconds,

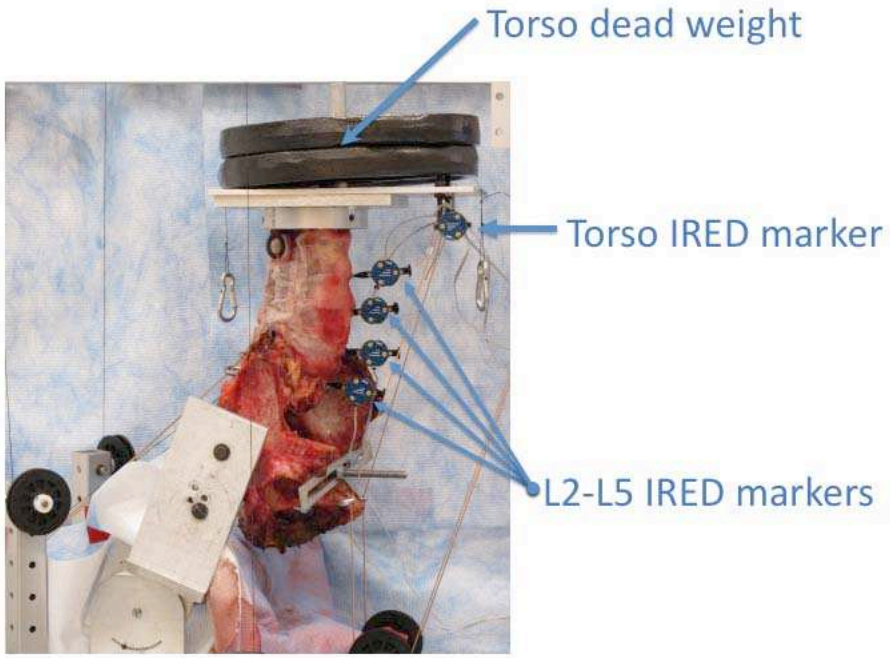

Fig. 3.4 Sagittal view of the lumbar and pelvis with the testing apparatus showing pelvic attachment frame, muscle cables and torque motors, torso plate with dead weight, and five IRED (torso, L2, L3, L4 and L5) OptoTrak targets for measuring torso and vertebral position.

the measurements of each of the IREDs were taken two more times with an additional 30 second grace period between recordings. Once the data had been collected for these cases, known as passive, each spine was again placed in the neutral posture representing a standing or upright posture and were preloaded with a compressive load of 245 N to simulate upper trunk weight as shown in Fig. 3.4[1]. Next, the loads in the cables simulating were ramped up slowly to match the *in vitro* experiments shown in Table 1 where TW denotes torso weight, AR denotes axial rotation perturbation, S denotes stability perturbation (flexion or extension), L LM denotes left lumbar multifidus, R LM denotes right lumbar mulitifidus, ES denotes erector spinae, and RA denotes rectus abdominus. Activity in this table represent the percent activity of the lumbar multifidus. Thus, active is equivalent to 100% activation of the lumbar multifidus, 75% active represents a 25% reduction in lumbar multifidus force from the active case, 50% active represents a 50% reduction in lumbar multifidus force from the active case, and finally, 25% active represents a 75% reduction in lumbar multifidus force from the active case. As with the passive case, each *in vitro* case

[1]It should be noted that a small portion of the right upper pelvic crest bone was removed to ensure that IRED markers could be observed by the OptoTrak camera.

Table 1 Neutral posture *in vitro* experiments.

In vitro Experiment	TW	AR	S	L LM	R LM	ES	RA
Passive	0 N	0 Nm	0°	**0 N**	**0 N**	0 N	0 N
Active	245 N	0 Nm	0°	**30.0 N**	**30.0 N**	195 N	50 N
75 % Active	245 N	0 Nm	0°	**22.5 N**	**22.5 N**	195 N	50 N
50 % Active	245 N	0 Nm	0°	**15.0 N**	**15.0 N**	195 N	50 N
25 % Active	245 N	0 Nm	0°	**7.50 N**	**7.50 N**	195 N	50 N

was tested a total of three times on each spine with a settling period of 30 seconds between each case.

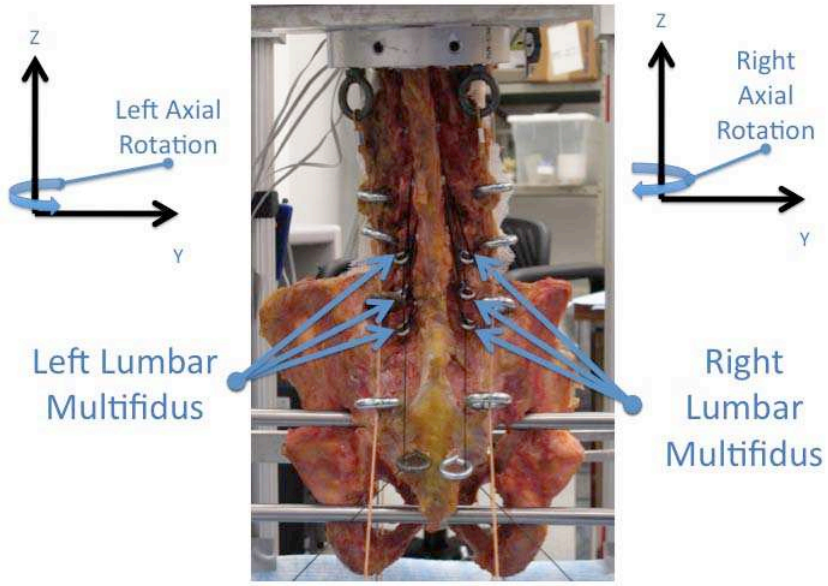

Fig. 3.5 Rear view of lumbar spine identifying right and left lumbar multifidus as well as right and left axial rotation perturbations.

3.4 A Perturbation to the Axial Rotation

Using the sliding ring cable driven mechanism shown in Fig. 3.3, a total of twenty loading cases were investigated to quantify the impact of the multifidus for a right and left axial perturbation. The twenty load cases are displayed in Tables 2, 3, 4, and 5. The experi-

Table 2 Right axial rotation perturbation I *in vitro* experiments.

In vitro Experiment	TW	AR	S	**L LM**	**R LM**	ES	RA
Passive	0 N	4.52 Nm	0°	**0 N**	**0 N**	0 N	0 N
Active	245 N	4.52 Nm	0°	**30 N**	**30 N**	195 N	50 N
75 % Active	245 N	4.52 Nm	0°	**22.5 N**	**30 N**	195 N	50 N
50 % Active	245 N	4.52 Nm	0°	**15 N**	**30 N**	195 N	50 N
25 % Active	245 N	4.52 Nm	0°	**7.5 N**	**30 N**	195 N	50 N

Table 3 Right axial rotation perturbation II *in vitro* experiments.

In vitro Experiment	TW	AR	S	**L LM**	**R LM**	ES	RA
Passive	0 N	9.04 Nm	0°	**0 N**	**0 N**	0 N	0 N
Active	245 N	9.04 Nm	0°	**30 N**	**30 N**	195 N	50 N
75 % Active	245 N	9.04 Nm	0°	**22.5 N**	**30 N**	195 N	50 N
50 % Active	245 N	9.04 Nm	0°	**15 N**	**30 N**	195 N	50 N
25 % Active	245 N	9.04 Nm	0°	**7.5 N**	**30 N**	195 N	50 N

Table 4 Left axial rotation perturbation I *in vitro* experiments.

In vitro Experiment	TW	AR	S	L LM	R LM	ES	RA
Passive	0 N	4.52 Nm	0°	**0 N**	**0 N**	0 N	0 N
Active	245 N	4.52 Nm	0°	**30.0 N**	**30.0 N**	195 N	50 N
75 % Active	245 N	4.52 Nm	0°	**30.0 N**	**22.5 N**	195 N	50 N
50 % Active	245 N	4.52 Nm	0°	**30.0 N**	**15.0 N**	195 N	50 N
25 % Active	245 N	4.52 Nm	0°	**30.0 N**	**7.50 N**	195 N	50 N

Table 5 Left axial rotation perturbation II *in vitro* experiments.

In vitro Experiment	TW	AR	S	L LM	R LM	ES	RA
Passive	0 N	9.04 Nm	0°	**0 N**	**0 N**	0 N	0 N
Active	245 N	9.04 Nm	0°	**30.0 N**	**30.0 N**	195 N	50 N
75 % Active	245 N	9.04 Nm	0°	**30.0 N**	**22.5 N**	195 N	50 N
50 % Active	245 N	9.04 Nm	0°	**30.0 N**	**15.0 N**	195 N	50 N
25 % Active	245 N	9.04 Nm	0°	**30.0 N**	**7.50 N**	195 N	50 N

mental procedure for the axial perturbations were identical to that of the natural posture i.e., starting with the passive case and migrating to the active cases with the only difference being the application of the axial rotation and varying loads of the right and left lumbar multifidus. Again, as far as procedure, they were identical with those two changes from the neutral case. It should also be noted that whatever direction (right or left axial rotation) the perturbation was applied, the lumbar multifidus (right or left lumbar multifidus) did not change. Fig. 3.2 shows the right and left lumbar multifidus as well as the right and left axial perturbations. The spine in this figure has slight lean to the left in the lateral rotation, θ, which was to be expected since there were no lateral muscles simulated to balance the spine for lateral postures.

3.5 Perturbations to Flexional Motions

To investigate the sagittal plane stability of the lumbar spine, each spine was manually flexed to a +5° ϕ perturbation from the neutral posture as shown in Fig. 3.6. This was accomplished by using a hand load cell with the experimenter pulling the torso pole until the spine had reached +5° from the previous neutral posture determined from the first

experiment. Data was recorded in the same fashion as the previous experiments with the only difference being that the experimenter used the hand load cell to ensure the spine held steady at the $+5^\circ$ ϕ perturbation for the duration of the experiment (see Fig. 3.6).

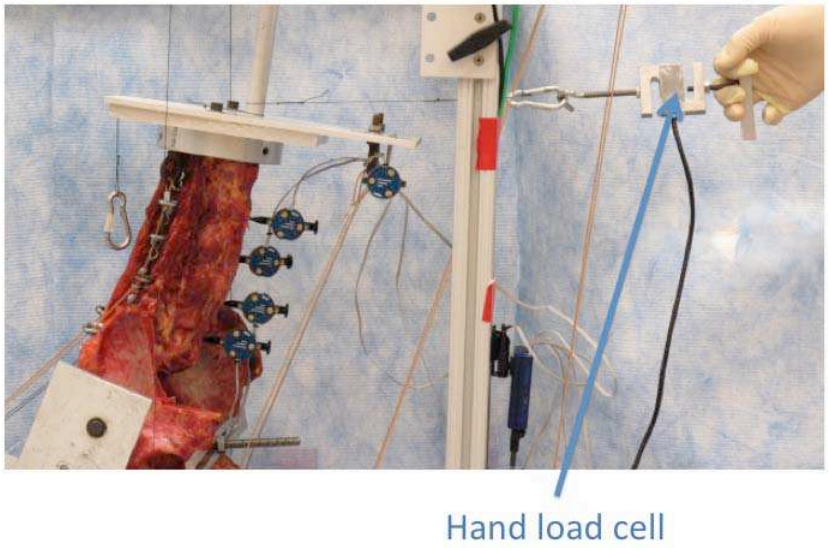

Fig. 3.6 Sagittal view of the lumbar spine and pelvis with the testing apparatus when the spine is flexed $+5^\circ$ (i.e., the torso is flexed $\phi = +5^\circ$) from the neutral posture using a hand load cell.

3.6 Perturbations to Extensional Motions

The final investigation of sagittal plane stability of the lumbar spine focused an extension perturbation, -5° ϕ perturbation from the neutral posture as shown in Fig. 3.7. This was accomplished by using a hand load cell with the experimenter pulling the torso pole until the spine had reached -5° from the previous neutral posture determined from the first experiment. As with each of the previous cases, data was recorded in the same fashion as the previous experiments with the only difference being that the experimenter used the hand load cell to ensure the spine held steady at the -5° ϕ perturbation for the duration of the experiment.

Table 6 Flexion perturbation stability *in vitro* experiments.

In vitro Experiment	TW	AR	S	**L LM**	**R LM**	ES	RA
Passive	0 N	0 Nm	+5°	**0 N**	**0 N**	0 N	0 N
Active	245 N	0 Nm	+5°	**30.0 N**	**30.0 N**	195 N	50 N
75 % Active	245 N	0 Nm	+5°	**22.5 N**	**22.5 N**	195 N	50 N
50 % Active	245 N	0 Nm	+5°	**15.0 N**	**15.0 N**	195 N	50 N
25 % Active	245 N	0 Nm	+5°	**7.50 N**	**7.50 N**	195 N	50 N

Table 7 Extension perturbation stability *in vitro* experiments.

In vitro Experiment	TW	AR	S	**L LM**	**R LM**	ES	RA
Passive	0 N	0 Nm	−5°	**0 N**	**0 N**	0 N	0 N
Active	245 N	0 Nm	−5°	**30.0 N**	**30.0 N**	195 N	50 N
75 % Active	245 N	0 Nm	−5°	**22.5 N**	**22.5 N**	195 N	50 N
50 % Active	245 N	0 Nm	−5°	**15.0 N**	**15.0 N**	195 N	50 N
25 % Active	245 N	0 Nm	−5°	**7.50 N**	**7.50 N**	195 N	50 N

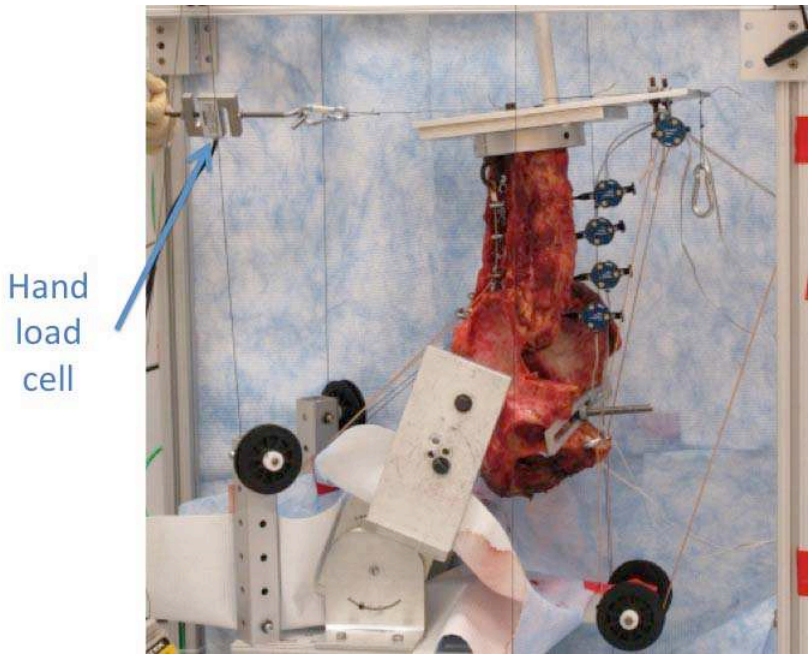

Fig. 3.7 Sagittal view of the lumbar spine and pelvis with the testing apparatus when the spine is extended $-5°$ (i.e., the torso is extended $\phi = -5°$) from the neutral posture using a hand load cell.

Chapter 4

Hypothesis and Experimental Results

4.1 Overview

Prior to discussing our experimental results of this work, it is helpful to summarize what methods were used in generating them. As stated in Chapter 3, 6 fresh human thoracolumbar spines (T12-pelvic) were procured through the UCSF Willed Body Program. The paraspinal muscles were surgically removed, leaving the spinal segments intact before potting them into the spinal testing apparatus shown in Fig. 3.4. Each spine was then subjected to a series of *in vitro* experiments: neutral posture, right and left axial perturbation, and finally, two stability perturbations: flexion and extension, with varying loading conditions. During these experiments, lumbar vertebral body positions and orientations were measured to quantify the postural changes as a function of deep lumbar multifidus (LM) atrophy.

For the given posture, this data was used to help determine the stability of the lumbar region of the spine by perturbing the spine from a position (neutral posture) while simulating atrophy of the deep LM. The data was then analyzed and organized with the help of three hypotheses presented in the following sections: Section 4.2, Section 4.3, and Section 4.4.

The first section, Section 4.2, focuses on the neutral posture and presents an additional subsection on muscle compensation. Section 4.3 is devoted to an investigation of the impact of right and left axial rotation for the two pure moment loading cases. The discussion in Section 4.4 focuses on the stability of the lumbar spine by studying the impact of a flexed and extended perturbations of the neutral spine. Each section has at least two subsections, the first states the hypothesis for the section and the second, summarizes the results presented in their respective section. The chapter concludes with a brief summary of all results presented before moving on to the next chapter.

4.2 Multifidus - Neutral Posture

4.2.1 Hypothesis 1

Force comparable to that of moderate to severe atrophy of the deep lumbar multifidus will change lumbar lordosis.

To test this hypothesis, 6 fresh human thoracolumbar spines (T12-pelvic) were tested using the methods outlined in Section 3.3. Typically, muscle atrophy is defined by the reduction in cross sectional area of the muscle of interest, however, in this work, moderate to severe atrophy was defined by a 50% or more reduction in muscle force. The relative angles[1] for the position of the adjacent vertebral bodies, L2 relative to L3, L3 relative to L4, and L4 relative to L5 are provided in Table 8. Furthermore, the five load cases are as follows:

Passive - a completely unloaded spine, i.e., no upper trunk weight.

Active - a loaded spine with a torso weight of 245 N and all muscles (Erector Spinae (ES), Rectus Abdominius (RA), and LM) fully activated.

75% Active - a loaded spine with a torso weight of 245 N, full activation of ES and RA but only a 75% activation of the LM.

50% Active - a loaded spine with a torso weight of 245 N, full activation of ES and RA but only a 50% activation of the LM.

25% Active - a loaded spine with a torso weight of 245 N, full activation of ES and RA but only a 25% activation of the LM.

Relative angles were calculated by first determining ϕ for each vertebral body in terms of the absolute coordinate system then subtracting the relative angels between adjacent vertebral bodies. In this table and subsequent tables, the active case is the basis for how data is compared, i.e., relative angles for each posture (L2 relative to L3, L3 relative to L4, and L4 relative to L5) are zero for the active posture and measured relative the active loading case. Finally, it was noted that one of the data sets values were drastically different than the previous sets. However, the data set was still included as part of the analysis due to the small sample size. However, if this data set was removed the relative change in angle between L4/L5 would change from 1.1° - 1.3° to 0.1° - 0.3°.

4.2.2 Neutral Posture with Muscle Compensation

During the experiments it was noted that despite the decrease in LM force, there was little to no change in posture of the torso or adjacent vertebral bodies as shown in Table 8.

[1] It should be noted that a positive angle direction is into flexion.

Table 8 Neutral posture *in vitro* experiments results. In this, and subsequent tables, ES denotes the Erector Spinae, RA denotes the Rectus Abdominius, and LM denotes the lumbar multifidus.

	Passive	**Active**	75% Active	50% Active	25% Active
ES Force (N)	0.0	**200**	200	200	200
RA Force (N)	0.0	**50**	50	50	50
LM Force (N)	0.0	**30**	22.5	15.0	7.5
L2-rel-L3 (deg)	0.3	**0.0**	0.0	0.0	0.0
L3-rel-L4 (deg)	1.9	**0.0**	0.0	0.0	0.0
L4-rel-L5 (deg)	4.4	**0.0**	1.1	1.1	1.3

Table 9 Neutral posture *in vitro* experiments results with muscle compensation.

	Passive	**Active**	75% Active	50% Active	25% Active
ES Force (N)	0.0	**200**	230	260	290
RA Force (N)	0.0	**50**	50	50	50
LM Force (N)	0.0	**30**	22.5	15.0	7.5
L2-rel-L3 (deg)	-4.8	**0.0**	0.0	0.0	0.0
L3-rel-L4 (deg)	2.4	**0.0**	0.0	0.1	0.1
L4-rel-L5 (deg)	2.3	**0.0**	0.0	0.0	0.1

This result was contrary to a previous internal study. Therefore, an additional testing protocol was added: muscle compensation of the ES based upon the aforementioned internal study. In this approach for every 25% decrease in LM force the ES force was increased by 15%. The resultant data using using this approach is presented in Table 9. However, it should be noted that only two of the six spines were subjected to this type of testing and thus the sample isn't large enough to generate firm conclusions. Nonetheless, it was interesting to note that the changes in angels shown in Table 9 were similar to those found in Table 8 even when a 45% increase in muscle force of the ES was present.

4.2.3 Summary of Results for Neutral Posture

The results of both neutral posture and neutral posture with muscle compensation were contrary to what was expected. To verify that was not simply an artifact of the testing facility, a simple stiffness calculation was performed. Using a modified relationship of the

stiffness matrix shown in Eq. (4.1):[2]

$$\mathsf{F} = \mathsf{K}\mathsf{d}. \tag{4.1}$$

In this equation, the generalized force vector F, generalized displacement vector d, and stiffness matrix K are

$$\mathsf{F} = \begin{bmatrix} \mathbf{F}\cdot\mathbf{p}_1 \\ \mathbf{F}\cdot\mathbf{p}_2 \\ \mathbf{F}\cdot\mathbf{p}_3 \\ \mathbf{M}\cdot\mathbf{g}_1 \\ \mathbf{M}\cdot\mathbf{g}_2 \\ \mathbf{M}\cdot\mathbf{g}_3 \end{bmatrix}, \qquad \mathsf{d} = \begin{bmatrix} \mathbf{y}\cdot\mathbf{p}_1 \\ \mathbf{y}\cdot\mathbf{p}_2 \\ \mathbf{y}\cdot\mathbf{p}_3 \\ \psi \\ \theta \\ \phi \end{bmatrix}, \qquad \mathsf{K} = \begin{bmatrix} k_{11} & k_{12} & \cdots & k_{16} \\ k_{21} & k_{22} & \cdots & k_{26} \\ \vdots & \vdots & \ddots & \vdots \\ k_{61} & k_{62} & \cdots & k_{66} \end{bmatrix}. \tag{4.2}$$

For the present purposes, the stiffness matrix of the intervertebral disk (taken from the literature [78]) was specified as

$$\mathsf{K} = \begin{bmatrix} 80307 & 155477 & 29398 & -65.43 & -1102 & 1892.3 \\ 34377 & 382690 & 91836 & -113.8 & -1779 & 1585.5 \\ 8882.4 & -17381 & 3227.2 & -400 & -537.3 & 1630.6 \\ 599.5 & 1439.9 & 399.83 & -11.52 & -11.97 & 13.468 \\ 7544.9 & 16460 & 2832 & -50.85 & 174.02 & -276.9 \\ -105 & 7014.3 & 1468.5 & 7.9949 & -154.8 & 307.05 \end{bmatrix}. \tag{4.3}$$

Here, the displacement was measured in meters and rotations measured in radians.

To proceed, we used the measured values of **F** and **M** along with the stiffness matrix stiffness matrix (4.3), to conclude that a maximum change of 0.03° and minimum change of −0.02° in the relative angles was produced in the neutral posture. Thus, all cases (experimental and modeled) produced similar results in that there was negligible change in lumbar lordosis even when corresponding muscles forces increased due to simulated multifidus atrophy.

4.3 Multifidus - Axial Rotation Perturbation

4.3.1 Hypothesis 2

Force comparable to that of moderate to severe atrophy of the deep lumbar multifidus produces a greater change in spinal curvature during axial rotation than during neutral posture.

[2] It should be noted that this equation does not include the generated residual force vector F_o found in O'Reilly *et al.* [78] for simplification purposes.

Table 10 Right axial rotation, 4.52 Nm, *in vitro* experiments results.

	Passive	**Active**	75% Active	50% Active	25% Active
L LM Force (N)	0.0	**30**	22.5	15.0	7.5
L2-rel-L3 (deg)	6.9	**0.0**	-0.1	-0.2	-0.3
L3-rel-L4 (deg)	-7.6	**0.0**	-0.1	-0.1	-0.1
L4-rel-L5 (deg)	1.1	**0.0**	-0.2	-0.3	-0.4

Table 11 Right axial rotation, 9.04 Nm, *in vitro* experiments results.

	Passive	**Active**	75% Active	50% Active	25% Active
L LM Force (N)	0.0	**30**	22.5	15.0	7.5
L2-rel-L3 (deg)	3.2	**0.0**	-0.1	-0.2	-0.2
L3-rel-L4 (deg)	-4.1	**0.0**	0.0	0.0	0.0
L4-rel-L5 (deg)	-0.8	**0.0**	-0.1	-0.1	-0.2

As with the previous hypothesis, the same six spines were tested using the methods outlined in Section 3.4. The relative angles of the vertebral bodies during axial rotation, ψ, were calculated by determining ψ for each vertebral body from the absolute coordinate system then subtracting the difference between adjacent bodies. Once this relative angle was calculated they were then modified to make the active case the zero basis. Thus, all relative angles shown in Tables 10, 11, 12, and 13, are relative angles changes with respect to the active case. Although the ES and RA forces are not shown, they held constant at 200 N and 50 N respectively. The only exception was the passive cases when they were 0 N. Finally, the congruent deep lumbar multifidus were held constant during the right or left axial rotations, for example: right axial rotation, right lumbar multifidus held at 30 N for all cases except for passive when it was 0 N.

4.3.2 Summary of Results for Axial Rotation

In both right and left axial rotation the largest change in axial rotation (ψ) occurred at L2 relative to L3. Furthermore, each had a negative change in L3 relative to L4 for the passive cases. In general, there were no significant changes in spinal curvature during axial rotation as moderate to severe atrophy was simulated.

Table 12 Left axial rotation, 4.52 Nm, *in vitro* experiments results.

	Passive	**Active**	75% Active	50% Active	25% Active
R LM Force (N)	0.0	**30**	22.5	15.0	7.5
L2-rel-L3 (deg)	0.9	**0.0**	0.1	0.1	0.1
L3-rel-L4 (deg)	-1.8	**0.0**	0.1	0.1	0.1
L4-rel-L5 (deg)	1.1	**0.0**	0.1	0.1	0.1

Table 13 Left axial rotation, 9.04 Nm, *in vitro* experiments results.

	Passive	**Active**	75% Active	50% Active	25% Active
R LM Force (N)	0.0	**30**	22.5	15.0	7.5
L2-rel-L3 (deg)	4.3	**0.0**	0.1	0.2	0.2
L3-rel-L4 (deg)	-2.3	**0.0**	0.1	0.1	0.1
L4-rel-L5 (deg)	1.8	**0.0**	0.1	0.1	0.1

4.4 Multifidus - Flexion/Extension Perturbation

4.4.1 Hypothesis 3

Force comparable to that of moderate to severe atrophy of the deep lumbar multifidus produces a significant reduction of spinal stability when the spine is perturbed in flexion or extension from the neutral posture.

To test the stability of the spine in the sagittal plane, six spines were tested using the methods outlined in Section 3.5. The relative angles were measured using the same process described in Section 4.2. The results for a flexion perturbation are provided in Table 14 and results for the extension perturbation are provided in Table 15. It should be noted that there was signal interference during data collection on one spine during extension and two during flexion.

4.4.2 Summary of Results for Neutral Posture

Flexion perturbation results were very similar to natural posture with muscle compensation with the exception being a change in L2 relative to L3 in the passive case. Nonetheless, for both flexion and extension there was little to no change in relative vertebral body position. Thus, despite the perturbations from the neutral posture and simulation of muscle atrophy, the spine appears to be very stable.

Table 14 Flexion perturbation *in vitro* experiments results.

	Passive	**Active**	75% Active	50% Active	25% Active
ES Force (N)	0.0	**200**	200	200	200
RA Force (N)	0.0	**50**	50	50	50
LM Force (N)	0.0	**30**	22.5	15.0	7.5
L2-rel-L3 (deg)	-0.1	**0.0**	0.0	0.0	0.0
L3-rel-L4 (deg)	2.2	**0.0**	0.0	0.0	0.0
L4-rel-L5 (deg)	2.4	**0.0**	-0.1	-0.1	-0.1

Table 15 Extension perturbation *in vitro* experiments results.

	Passive	**Active**	75% Active	50% Active	25% Active
ES Force (N)	0.0	**200**	230	260	290
RA Force (N)	0.0	**50**	50	50	50
LM Force (N)	0.0	**30**	22.5	15.0	7.5
L2-rel-L3 (deg)	-0.2	**0.0**	0.0	0.0	0.0
L3-rel-L4 (deg)	1.6	**0.0**	0.0	0.0	0.0
L4-rel-L5 (deg)	3.8	**0.0**	0.0	0.0	0.0

4.5 Summary of Results

All three sets of *in vitro* experiments, neutral posture, right and left axial perturbation, and finally, two stability perturbations: flexion and extension, with varying loading conditions, yielded similar results, little to no change in adjacent vertebral body position as function of multifidus atrophy. The only significant change in spinal curvature was found in Table 8 where there was a 1.1° to 1.3° change in L4 relative to L5 as multifidus atrophy was simulated.

Chapter 5

Conclusions

The goal of this work was to provide the researcher or clinician greater insight into the role of deep muscles in chronic low back pain. More specifically, to quantify the impact of the deep lumbar multifidus on lumbar lordosis and spinal stability. To accomplish this goal, a unique spine testing facility was designed and constructed. Second, a series of *in vitro* experiments (neutral posture, neutral posture with muscle compensation, axial rotation, and flexion/extension perturbations) were conducted to answer the three hypotheses: *one* - force comparable to that of moderate to severe atrophy of the deep lumbar multifidus will change lumbar lordosis, *two* - force comparable to that of moderate to severe atrophy of the deep lumbar multifidus produces a greater change in spinal curvature during axial rotation than during neutral posture, *three* - force comparable to that of moderate to severe atrophy of the deep lumbar multifidus produces a significant reduction of spinal stability when the spine is perturbed in flexion or extension from the neutral posture. In this final chapter, the results of the various *in vitro* experiments are analyzed in Section 5.1, recommendations on how to improve the experiments are presented in Section 5.2, and finally, this work concludes in Section 5.3 with several suggestions for future work to better understand chronic low back pain disorders.

5.1 Role of Muscles in Chronic Lower Back Pain

Three hypotheses were tested to quantify the impact of the deep lumbar multifidus. Of the three hypotheses, only hypothesis one was close to being validated. In that hypothesis, moderate to severe atrophy resulted in a change in spinal curvature. There was approximately a 1.1° to 1.3° change in relative flexion angle between L4/L5 during neutral posture *in vitro* experiments. This result was similar to other studies that have shown that multifidus atrophy correlates with approximately a 1.5° change in the flexion angle at L4/L5 [11]. However, that was not the case in this study for all *in vitro* experiments. All the other

experimental results were on the order of error of the measurement equipment (i.e., $\pm 0.1^\circ$) and thus the corresponding hypotheses could not be verified. Nevertheless, using the data from these experiments, three conclusions can be made:

1. The spine under loading conditions of erector spine, rectus abdominus, deep lumbar multifidus, and body weight is stable under torso perturbations ($\pm 5^\circ$ ϕ) in flexion or extension from the neutral posture.

2. Postural changes found in patients with chronic low back pain probably occur when compressive loading has been reduced or disc properties have changed beyond a threshold of the deep lumbar multifidus compensation.

3. It is unclear whether changes in lumbar spine posture are due to singe spinal level degeneration or to a more global degeneration.

The first conclusion followed the general trend of the data. The spine did not have significant changes in lordosis or changes in axial rotation regardless of the loading condition with the exception being the neutral posture and change in angle at L4/L5. This was particularly evident for flexion and extension stability perturbations where there was no little to no change in the relative angles between vertebrae. However, in the case of flexion and extension perturbations ($\pm 5^\circ$ ϕ), this could have been a byproduct of the torso perturbation being too small to generate a measurable change in adjacent lumbar vertebral segments. Torso perturbations of $\pm 5^\circ$ ϕ are similar to sudden movements of the arms, head, and initiation of locomotion to reach items in the sagittal plane. It should be noted that the changes in spinal kinematics in these mechanical experiments are very similar to those observed in the mathematical model under comparable loading and muscle activations and *in vivo* images of spines in the case of $\pm 5^\circ$ ϕ flexion or extension.

It should also be noted that because the spine is stable in the sagittal plane using the loading conditions mentioned above does not require constant activation of the deep lumbar multifidus. Thus reducing the amount of activity required to maintain this posture reduces the likelihood of fatigue. As it stands, if all the muscle groups were constantly active during the experiments, then the deep lumbar multifidus would have a 33% activation of its maximum contractile force, a level of activation that would probably lead to fatigue. This percentage is well above those for the other muscle groups used in this experiments.

Though the first conclusion (concerning the stability of the spine in the sagittal plane) originated entirely from the data collected during this work, the second conclusion arose from a combination of this study's results and results currently in the literature. Specifically, Belavy *et al.* [11] found that volunteers subjected to prolonged bed rest develop multifidus atrophy and this atrophy was correlated with a loss of lordosis (1.5° change in lordosis at L4/L5). Many believe that such alterations in lumbar posture (change in lordosis at L4/L5) and kinematics are lead to pain. Furthermore, paraspinal muscle atrophy has been linked to loss of lordosis in patients with flat back syndrome [63]. However, these correlations were for individuals exposed to prolonged bed rest or astronauts returning from space that exceed typical diurnal variations. Coupling this research with the data from our study

suggests that posture changes most likely occur when the intervertebral discs are subject to a lower loading condition (i.e., reduced compressive spinal loading due to bed rest or absence of gravity in space). It is well known that changes in spine compression alters disc properties. More specifically, disc height, and occur during sleep, extended bed rest, or loss of gravity all of which are changes in spinal compression. The data suggests that once individuals are subjected to normal loading conditions after prolonged exposure to adverse loading conditions, they will experience small changes in posture regardless of their multifidus atrophy level which is consistent with clinical diurnal variation studies (see, for example, [1]). Thus, if these individuals have already experienced significant changes in spinal curvature, then they are on the cusp of low back pain and it will only take a small deviation from their current position to induce back pain.

The third conclusion was drawn from an inability to vary the load of the deep lumbar multifidus from segment to segment. Thus, a constant load was applied to each set of adjacent vertebral bodies even though such loading is not physiological. Ideally a distribution of the forces exerted by the multifidus on individual vertebra should have been imposed because level specific simulation of the deep lumbar multifidus would allow for greater range of motion of the vertebral bodies and would more accurately mimic *in vivo*. Such a distribution would have resulted in a variation in the amounts of relative rotation experienced by the individual vertebra. For instance, the rotation of L2 relative to L3 would be distinct from the rotation of L3 relative to L4. A consequence of the constant loading in our experiments resulted in these relative rotations being similar.

In summary, a weakened multifidus is a contributing factor to the change in posture of the lumbar lordosis. However, the thresholds in muscle forces needed to produce the change in posture are still not completely known. Furthermore, some enhancements to the testing facility are required before concrete answers to these questions can be found.

5.2 Recommendations

While the spinal testing apparatus used to conduct the experiment in this study was novel, it is not without fault. Four items that could be included to improve this novel testing apparatus are

1. Use of artificial muscle in place of cables.

2. Incorporation of a level specific multifidus activation.

3. Provision of a simpler method to manipulate the torso weight.

4. Addition of muscles required for lateral motion in order that other coupled motions can be examined.

A general limitation of muscle simulation was the choice of cables used in this experiment. The cables used in this experiment were combinations of steel cables and fishing wire which have varying compliance. Although these cables were sufficient for simulating

and maintaining a constant load through the duration of the experiment, they were not capable of replicating the natural compliance of human muscle. It would be beneficial to use a more compliant material like artificial muscle to better replicate muscle activation.

One of the limitations of this testing apparatus was the inability to vary the loads of the level specific multifidus. Thus, all the levels of the multifidus simulated in this study had the same load even though physiologically the load varies as function of vertebral body level. By improving the implementation of the multifidus, the experimental results could be compared to some finite element studies that already feature this (see, for example, [102]). Furthermore, it would help researchers and clinicians to determine if bucking/loss of stability of the spine is a local or global phenomena.

Although the dead weights were used to simulate torso weight, the addition and subtraction of these weights was tedious and time consuming. Cadaver experiments should not exceed 12 hours in length (see, for example, [30, 86]) and the approach used here was on the order of ten hours. Simulation of various torso weights to simulate bedrest, among other activities, would add a tremendous amount of time unless this process is improved.

Finally, one adjustment that could be included is the addition of muscles responsible for lateral movement. One muscle group in particular that is of great interest to clinicians are the transverse abdominals. The existing testing facility is equipped to add up to four additional muscle groups however, the challenge would be determining where to attach and how to route these muscle groups to provide a physiologic simulation without making the testing apparatus too complex and increasing setup time. Nonetheless, the addition of these groups would allow for more complex postures that represent a larger pool of daily activities while helping gain insight as to what activities individuals with specific conditions should avoid.

5.3 Future Work

One of the goals of this experiment was to provide clinicians greater insight into the role of the deep lumbar multifidus. More specifically, to aid physical therapist by quantifying the impact of the deep lumbar multifidus impact on lumbar lordosis since the only exercise therapies shown to achieve lasting and substantial reductions in pain are those that are directed at coactivation of the abdominal and multifidus muscles (see, for example, [17, 38, 39, 74, 99, 114]). Although this experiment was able to simulate coactivation of the abdominal and multifidus, it was unable to simulate a variety of things that physical therapist also do with patients in vivo. For example, physical therapy provides additional benefits to the patient other than muscle strengthening, it provides a relearning or retraining on how to execute specific tasks and which muscles to use, it is patient specific and allows for customized range of motion tasks, and accounts for injury to specific regions of the trunk. These are things that were not able to be simulated in vitro but could be if all the information is known.

Back pain and particularly chronic low back pain disorders are multi-faceted. The results of this study underscores that statement. Although the deep lumbar multifidus atrophy

has been linked to chronic low back pain, it is just one of many contributing factors. It is imperative to better understand the underlying pain mechanisms when individuals experience chronic low back pain since 85% of chronic low back pain disorders have no known diagnosis [79]. One area of interest that would be extremely valuable to clinicians would be a greater understanding of the neural system. The testing apparatus used in this work has the capability to simulate muscle contractions as effectively as the brain's neural system. However, how the brain executes the dynamic movement of the human body is not fully understood. With the advancements in digital computing capacity and high performance sensors that can map the brain's activity, there will be tremendous opportunities in the future to better understand how the neutral system operates and executes these dynamic human body movements. Coupling a neurological model with a program like OpenSim that can be validated by studies like the one presented in this work, would likely provide clinicians some of the insight they seek to understand the underlying pain mechanism present in individuals suffering from chronic low back pain disorder.

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Appendix A

Details of Muscle Anatomy and Function

Muscles in the human body are one of the key components involved in locomotion. Although this work focused on some of the muscles activated in association with trunk movement, intimate details on muscle anatomy and function were not discussed in detail. In this Appendix, a general overview of muscle anatomy and function are provided as well as a generic model that is typically used to describe their behavior. Finally, this Appendix concludes with additional details about the deep lumbar mutlifidus which is this work is based upon.

A.1 Muscle Anatomy

Motion of the human body is achieved through complex, controlled actuation (involuntary or voluntary) of the muscles in the human body. Gray *et al.* ([43]) described voluntary muscles by their cross-stripped appearance of fibers characteristic displayed under the microscope coupled with it being capable of being put into action and controlled by the will. Thus, involuntary muscles were described by their lack of cross-stripped fibers and for the most part are not controlled by the will. An example of the cellular structure can be seen in Fig. A.1. Although involuntary muscles are extremely important in the human body, this work focuses on skeletal voluntary muscles. Skeletal muscles are voluntary and for the most part are controlled by the will of the subject.

Skeletal muscles are a collection of fascicles, which are bundles of individual cells. Each fascicle is surrounded by a connective tissue layer known as perimysium. The collection of fascicles wrapped in perimysium are enveloped by epimysium. All these components connect to tendons, which connect the arrangement of connective tissues or, in this case, skeletal muscle, to the bone as seen in Fig. A.2. Within the fascicles there exists an additional tissue layer, the endomysium, which acts an electrical insulator and separates the muscle cells from each other. These muscle cells are composed of a number of myofibrils

Fig. A.1 The three types of muscle cells: skeletal, smooth, and cardiac. Skeletal muscles cells are considered to be voluntary whereas smooth and cardiac muscle cells are involuntary. The citation for the source image can be found in Appendix B.

as shown in Fig. A.3 where the sacroplamic reticulum acts as a sleeve around each myofibril. Myofibrils are made up of individual contractile proteins known as myofilaments. There are two types of myofilaments: thin and thick. Thin myofilaments are mainly composed of the protein actin whereas the thick myofilament are mostly made up of the protein myosin. The thick and think filaments in each myofibril are arranged in a repeating pattern along the length of the myofibril, commonly known as sarcromeres. Furthermore, there is a space between these overlapping thick and thin filaments which is bridged by projections known as cross bridges. These cross bridges make contact with the thin filaments during muscle contraction and exert force on them. It should be noted that muscle contraction does not necessarily mean shortening; rather it refers only to the turning on of force-generated sites, the cross bridges, in a muscle fiber [116].

A.2 Muscle Function

Force is generated in muscle when thin and thick filaments in each sarcomere moves past the other via movements of the cross bridges which is known as the sliding-filament mechanism for muscle contraction. The muscle fibers ability to generate this force is a function of the two proteins, myosin and actin, with energy provided by Adenosine-5'-triphosphate (ATP). Although these components are always present in the muscle, the muscle is not a continual state of contraction. The reason for this is due to two proteins, troponin and tropomyosin, which prevent the cross bridges from interacting. The cross bridges can interact once calcium binds to troponin which drags tropomyosin away allowing for contraction provided there is a sufficient calcium ion concentration in the muscle fiber [116].

The careful release of calcium originates from the sacroplasmic reticulum and is a function of action potential similar to nerve cells. Action potential in the muscle fiber endures for 1 to 2 milliseconds and is followed by a latency period (a few milliseconds) before activity is observed. The mechanical response of a single muscle fiber to a single action potential is known as twitch [116]. There are two types of twitch: fast and slow. Fast twitch

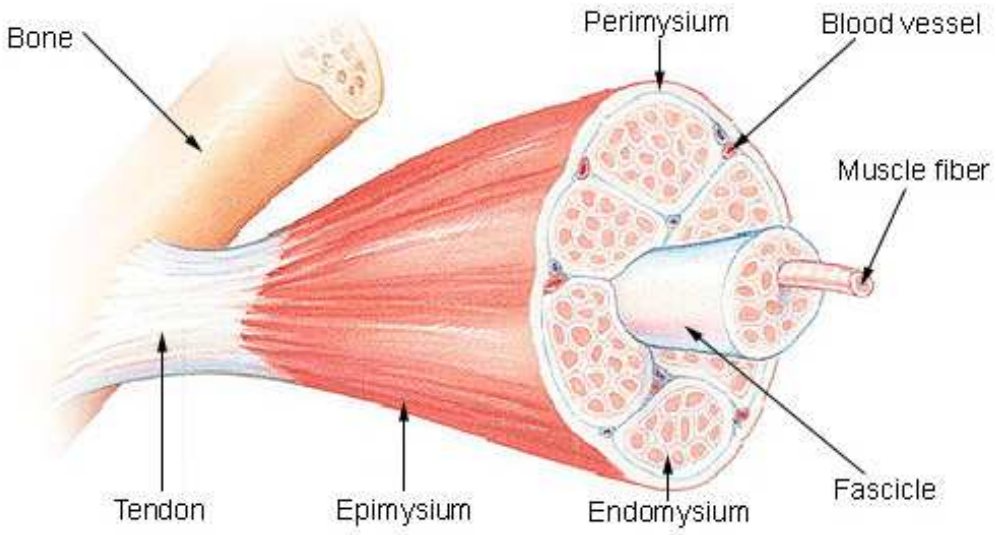

Fig. A.2 Basic components of skeletal muscle: epimysium, perimysium, blood vessels, fascicles, endomysium, and muscle fibers. The citation for the source image can be found in Appendix B.

is typically on the order of 10 milliseconds and slow twitch is about 100 milliseconds or more. The resultant force or tension produced in each muscle depends the type of twitch, the length of the muscle, muscle fatigue, and number of fibers contracting at any time.

A.3 Hill-Type Muscle Models

Hill-Type muscle models are used to predict the maximum force of a muscle. OpenSim uses a modified version of a Hill-Type muscle model to predict the loads the simulated muscles generate through inverse dynamics. A simple generic Hill-Type muscle model is shown in Fig. A.4.

Where:

CE - contractile element

PE - passive viscoelastic element

α - pennation angle

$\mathbf{l}_o^m$ - optimal muscle fiber length

$\mathbf{l}_{TS}$ - tendon slack length

$\mathbf{l}_{MT}$ - musculotendon unit length

$\mathbf{F}_o^M$ - maximum isometric force

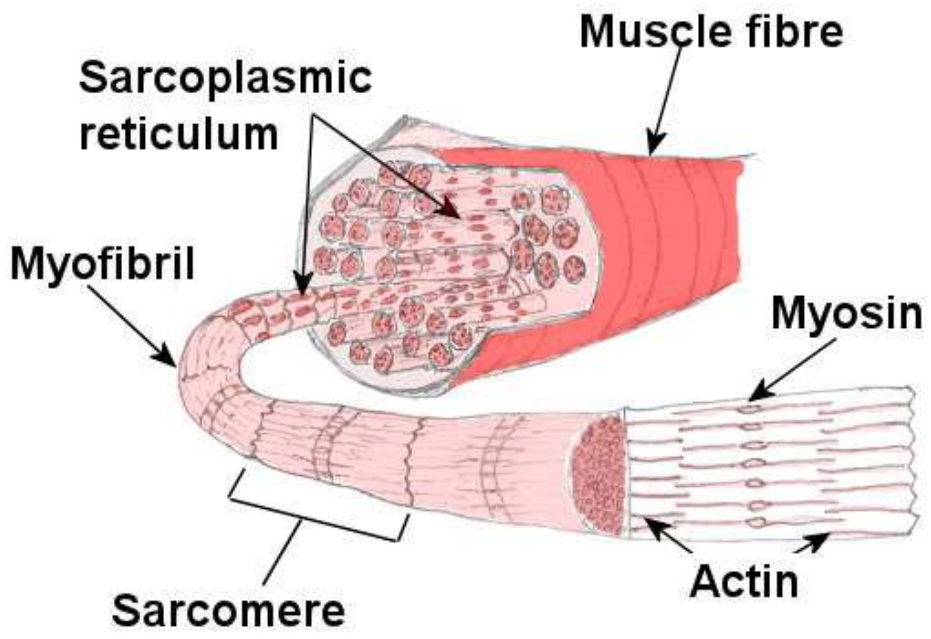

Fig. A.3 Basic components of muscle cells: sacroplasmic reticulum, myofibrils, and myofilaments. The citation for the source image can be found in Appendix B.

$\mathbf{F}_{MT}$ - muscle force

Not shown are two important input parameters: muscle excitation, u, and muscle activation, a. It should be noted that this model can be extended to include parameters like: motoneuron output, force-length relation, force velocity relation, sacroplasmic reticulum response, fitness variable (for fatigue), and several others resulting in a more accurate muscle model. The more variables the model can accommodate, the closer these models will get to generating physiologic simulations.

A.4 Deep Lumbar Multifidus

This section is devoted to providing information about the deep lumbar multifidus. More specifically, it contains a reviews evidence showing that the mutlifidus is a stabilizer, rather than initiator of motions, and concludes with evidence of shallow versus deep lumbar multifidus fiber types.

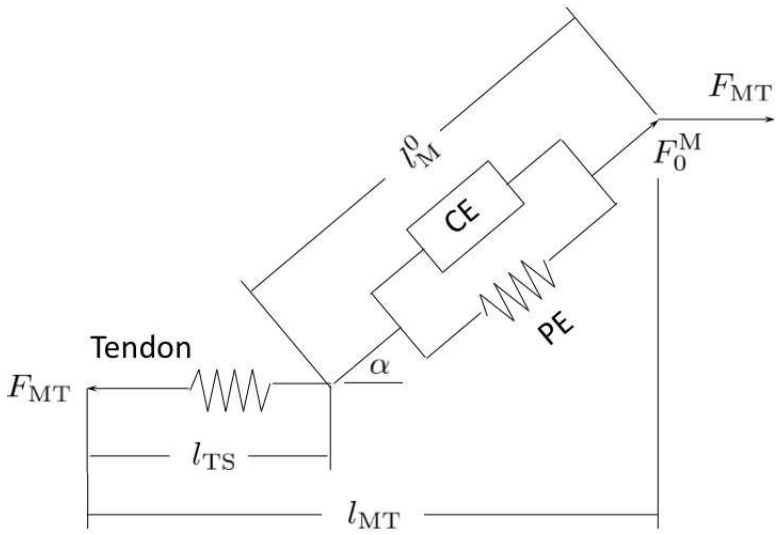

Fig. A.4 Generic Hill-Type Muscle Model used in OpenSim. Model defines boundaries on maximal muscle force.

The deep lumbar multifidus is a small yet powerful muscle that helps stabilize the human spine. Ward *et al.* [119] presented significant evidence demonstrating that the deep lumbar multifidus has a unique architecture among posterior spinal muscles. Based on its very short fiber lengths, very large physiological cross-sectional area, and specialized sarcomere length operating range functions, the multifidus is specialized as a stabilizing muscle for the human spine capable of producing large forces particularly during forward leaning posture.

Comparing passive properties of posterior spinal muscles, Ward et al. [118] measured stress-strain relationships of single-fiber and fiber- bundle material properties from biopsies from multifidus, longissimus, and iliocostalis muscles. Although all three muscles had similar material properties at the single-fiber level, multifidus fiber bundles had significantly greater stiffness (45%) than longissimus and iliocostalis muscles. This finding is important in this work because it demonstrates the high potential for passive stiffness of the multifidus, based on the structure of its extracellular matrix.

Another other misconception about the lumbar multifidus was the difference between the shallow and deep muscle. It was believed that the shallow and deep lumbar multifidus possess different fiber types, Type I for shallow lumbar multifidus and Type II for deep lumbar multifidus. However, Regev *et al.* [97] were able to determine that there was no difference in fiber type. They used a minimally invasive surgical approach to obtain biopsies from the multifidus, longissimus, iliocostalis and psoas muscles at specific predefined depths. They determined fiber types by measuring myosin heavy chain isoform distributions using SDS-PAGE electrophoresis. The fiber type distribution was similar among the

posterior paraspinal muscles and was composed of relatively high percentage of type I (63%), compared to type IIA (19%) and type IIX (18%) fibers. In contrast, the psoas muscle was found to contain a lower percentage of type I fibers (42%) and a higher percentage of type IIA (33%) and IIX fibers (26%; $P<0.05$). No significant difference was found for fiber type distribution among three different depths of the multifidus and psoas muscles.

Appendix B

Sources for Images

B.1 Links to images

Several of the images used in this dissertation were collected from various papers and websites on the internet. The location for all of the original images cited in this work are provided in Table 16.

Table 16 Source links for images used in this work.

Figure Label	Corresponding links
Fig. 2.1	http://t.co/BB4xIoj5
Fig. 2.2	http://bit.ly/qe9OHn
Fig. 2.3	Christophy *et al.* [26]
Fig. 2.5	Renner *et al.* [98]
Fig. 2.6	Patwardhan *et al.* [90]
Fig. 2.7	Wilke *et al.* [124]
Fig. 2.8	Welke *et al.* [120]
Fig. A.1	http://t.co/LpYGSMtF
Fig. A.2	http://t.co/cwDuSibf
Fig. A.3	http://t.co/WwljgAEt