MUSCLE ACTIVITY IN TIGHT HIP AND LOOSE HIP SUBJECTS DURING TWO DIFFERENT HIP EXTENSION TASKS.

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DEDICATION

To Dad

For teaching me that perseverance and hard work are the only worthwhile way to achievement.

To Mother

For helping me see the value of life-long learning.

To Davia and Jamison

For continuing to be the joy of my life and making me so proud despite my many absences.

To Cathy

For your love and support these many years. This is yet another one of many things I would have never achieved without you.

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Dr. Vladimir Janda inspired me to get me started on this project. His insight was that orthopedic problems are multifactoral and at least some of us might as well investigate them that way. His courage to put his ideas out clearly and have them scrutinized has lead to progress in understanding these maladies.

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ABSTRACT

Muscle activity in tight hip and loose hip subjects during two different hip extension tasks.

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Purpose. The purpose of the study was to investigate: 1) whether motor programming has a role in perpetuating muscle imbalance and 2) if subjects with tight hip flexors demonstrate hyperactivity of the erector spinae musculature. **Subjects.** From 254 potential subjects, 16 subjects who met the criteria for "loose hip" and 16 for "tight hip" flexors were selected. Subjects were selected who varied 1.5 sd from the mean on measures standardized for this study using an inclinometer. **Methods.** Subjects performed two movements: 1) voluntary lift; and 2) isometric hold, while in a hip extended or hip flexed prone position. Surface EMG was taken at the right and left erector spinae, right gluteus maximus and right hamstring. Dependent variables taken from the EMG were: 1) duration of muscle onset sequence (DMOS) - the time between the onsets of the first and fourth muscles; 2) latency of the erector spinae muscles (LES latency and RES latency) - the time between muscle onset and movement onset, and 3) early amplitude of the erector spinae (EAC) - average percent MVC during

0.4 sec after muscle onset minus average percent MVC during 0.1 sec prior to muscle onset. **Data Analysis.** The research design was a 2X2X2 repeated measures. Therefore, a three-way multi-variate analysis (MANOVA) was used. **Results.** A three-way interaction was found for DMOS. The difference was between the loose and tight hip subjects for the lifting movement in the flexed hip position ($p<0.0004$). This suggests that the muscle activation of the back and hip extensors is different in these two populations when hip tension is altered, but only for the lift movement. A main effect of movement ($p<0.01$) was found for LES latency. There was no main effect for hip type. For EAC there was a significant movement by position 2-way interaction ($p<0.016$), and no main effect for hip type. **Conclusion.** It does not appear that motor programming has a role in perpetuating this particular muscle imbalance and that subjects with tight hip flexors do not demonstrate hyperactivity of the erector spinae musculature. **Clinical Relevance.** This research seems to support Kendall's suggestion that muscle behaviors respond more to immediate external mechanical changes rather than constant internal mechanical differences.

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Chapter 1

INTRODUCTION

Muscle balance and its antithesis, muscle imbalance, have persisted as a theoretic construct since the late 1930's.[1] Muscle balance is an ideal ratio between strength and length of muscles moving a joint in one direction with opposing muscles moving the joint in another direction. Muscle imbalance is a less than ideal ratio of either strength or length between opposing muscles. Imbalance is seen to develop in predictable patterns and to potentially worsen. Both muscle balance and imbalance are demonstrated by clinical observation of posture and movement. Imbalances have been loosely correlated with various painful or degenerative conditions of the musculo-skeletal system.

A key postulation of muscle imbalance theory is that, left uninterrupted, muscle imbalance tends to worsen. The worsening seems to be related to mechanical demands placed on the imbalanced muscle groups. The muscle imbalance of one group in this study was one in which the anterior hip muscles (and perhaps other anterior soft tissues) were tight and the antagonistic hip extensors were normal strength. When the hip flexors are tight, one source of the mechanical demands affecting the low back are thought to be muscular behaviors related to vestibular and optic reactions

when maintaining an erect trunk during upright standing posture or erect trunk during normal walking. Treatments such as hip flexor stretching have been developed with the intent to interrupt the source of these mechanical demands.These ideas regarding muscle balance/imbalance at the hip have persisted for over a half century with only an occasional attempt at critical investigation or validation.

In the late 1970's and early 1980's, Lewit, Janda and others[2,3] advanced some intriguing new postulations about muscle imbalance. First was the postulation that the motor control system was more widely involved than previously thought in the perpetuation and gradual worsening of muscle imbalance. Earlier it was thought that if neuromotor mechanisms were involved, it was involved through responses to mechanical stimuli. Most notable of these would be vestibular and optical righting reactions and responses related to maintaining an upright posture with eyes balanced with the horizon. The new idea was that over time the vestibular and optical right reactions would cause an alteration of function in the central nervous system area responsible for muscle sequencing and amplitude. Thus, muscle imbalance would worsen not only on the basis of the mechanical demands of optical and vestibular righting reactions, but also because of a secondary neuromotor factor. The muscle sequencing and amplitude centers would control muscles so that the onset of certain muscles involved in a movement would tend to be earlier or later than normal in sequence. Certain muscles

would become hyperactive; that is, onset earlier than normal and have an amplitude that was excessive in relation to the mechanical demand. Other muscles would become inhibited; that is, onset later than normal and have amplitude diminished in relation to normal mechanical demand. These sequencing and amplitude changes would occur whether or not there was a mechanical demand imposed by posture or movement. Simple clinical movement tests would reveal which muscles were hyperactive or inhibited. These tests are movements which were unrelated to those movements which imposed mechanical demands causing the imbalance.[4,3]

These ideas were exciting to physical therapists interested in the treatment of orthopedic conditions. They offered a potential advance in the understanding of the perpetuation and worsening of painful spinal conditions in which muscle imbalance is an underlying cause. They also offered the potential for new ways to evaluate and possibly treat these conditions.

Sequencing and amplitude are muscle behaviors controlled to a large extent in the premotor and supplementary motor areas of the central nervous system.[5] However, these behaviors can be modified by other effects at lower centers of control. Therefore, a review of neurological mechanisms related to muscle function seems to be in order.

Definitions

Internal resistance - The resistance in the opposite direction of a muscle's action that results from the tension in tissues having an opposing action to a

muscle. In this study, the internal resistance resulted from tension in the hip flexor muscles and other anterior hip structures when the hip extensor moved or held the hip toward hip extension.

External resistance - The resistance opposite the direction of a muscle's action resulting from an external source, such as the gravity weight of the extremity, a weight, or any mechanical factor which magnifies these sources. In this study the external resistance was the gravity weight of the right lower extremity. The drop speed of the leg in the hold conditions was a magnifying factor.

Internal/external resistance - A combination of internal and external resistance.

Muscle balance - A condition that exists at a joint when the opposing muscle groups have the optimum ratio of strength and length relative to each other.

Muscle imbalance - A condition existing when opposing muscle groups do not have an optimum ratio of strength and/or length relative to each other.

Motor Pre-Programming - Timing and amplitude commands stored in higher CNS areas for control of well-learned movements. The commands are executed in the pre-movement period and their effects can be seen in the mechanical events during movement.

Hyperactivity - EMG muscle activity that, when compared to normal, is earlier and of excessive amplitude relative to the mechanical demand.

Inhibition - EMG muscle activity that, when compared to normal, is later or of diminished amplitude relative to the mechanical demand.

Assumptions

1. The main reason for hypertrophy and increased strength to develop in a muscle is prolonged application of increased resistance in a direction opposite that muscle's action.

2. It is widely accepted that persons with tight hip flexors have a tendency to stand and walk with an increased anterior pelvic tilt. The mechanical demands of standing and walking in this way may cause the erector spinae muscles to hypertrophy.

3. Tight anterior hip structures can be confirmed by a forwardly rolled pelvis in prone lying and a decreased supine hip flexor length measurement. Persons with these clinical test results usually have hypertrophic and tight erector spinae muscles.

4. When combined with a reliable and valid source of start of movement, surface electromyography gives an accurate representation of the start of contractile activity in a muscle.

5. Surface electromyography gives an accurate and reliable measure of the magnitude of activity in the contractile part of the muscle and correlates well with force output of a muscle, provided the muscle is not extremely slack or extremely lengthened.

6. It was assumed the measurements which classified each subject into a hip type group indicated the type of muscle imbalance each had for several years.

7. When lying in the prone hip extended position, persons with tight anterior hip structures have greater resistance to hip extension than person with loose anterior hip structures.

CHAPTER 2

REVIEW OF LITERATURE

Review of neurological control of muscle behavior.

This study was an attempt to observe changes in muscle behaviors in the pre-movement period. Studies of neurological control mechanisms strongly suggest that muscle behavior in this period is pre-programmed at higher CNS levels. However, there are influences on the behavior from many levels of the sensory and motor systems. Therefore, a review of mechanisms of neurological control over muscles is in order. Emphasis will be on those neurological mechanisms that affect the behavior of muscles in the pre-movement period.

Epicritic sensation and its relation to behavior of individual muscle or muscles in coordinated action.

For the neurological system to regulate, control, or pre-plan, there must be sensory input on which to base these operations. The sensory branch of the neurological system provides pre-movement assessment of the conditions under which movement will occur. Differences in pre-movement sensation, once interpreted, may result in a change in muscle behavior during the pre-movement time. Support surface conditions, the balance and

equilibrium state of the body, muscle length, joint position, and surface contact conditions are the important sensory input. One dependent variable in this study was based on timed onset of muscles during voluntary movements. It was reasonable to suppose that events related to onset of muscle activity at the start of a movement would be influenced by the state-of-the-organism information from the epicritic receptors just prior to the onset of movement.

The epicritic sensory modalities (vision, touch, vibration, joint position or movement, and pain) give us the ability to sense our environment. These sensory modalities can be perceived individually. Often, however, they are perceived in combinations giving such perceptions as texture, stereognosis, and two point discrimination.

Proprioceptors are extremely important to spinal level motor organization. These are the muscle spindles, golgi tendon organs, joint receptors, vestibular receptors and cutaneous receptors. The muscle spindles are receptors located within the contractile part of a muscle amongst and in parallel with the extrafusal (outside-the-muscle-spindle) muscle fibers. A muscle spindle is composed of three different types of intrafusal (inside-the-muscle-spindle) fibers: dynamic nuclear bag fibers, static nuclear bag fibers, and nuclear chain fibers. Two different types of afferent endings (primary afferent Ia ending and secondary II afferent ending) and two different types of gamma efferent endings (dynamic gamma efferent and static gamma

efferent) carry messages to and from the different fibers. There are two morphological parts to the muscle spindle: the nuclear bag and the nuclear chain. The nuclear bag contains one dynamic nuclear bag fiber and one static nuclear bag fiber. The nuclear chain contains only nuclear chain fibers. The dynamic efferent axon enters only the nuclear bag and innervates only the nuclear bag fiber. The static efferent axon enters both the nuclear bag and nuclear chain. It innervates the static nuclear bag fiber and all the nuclear chain fibers.

A Ia primary ending consists of coils around the mid-portion of all intrafusal fibers (dynamic nuclear bag and both types of static). It is, therefore, well-constructed to signal the changing differences between the static and dynamic fibers. The secondary II ending consists of coils around the static fibers only (static nuclear bag fiber and static nuclear chain fibers). It is constructed to signal only changes in static state of stretch.

Before a muscle is stretched, both primary and secondary endings have a discharge rate that signals the state of stretch of the muscle. When a muscle is being stretched, the primary Ia afferent fires at a much higher rate signalling a change in stretch. When the muscle reaches a new length, a steady state static rate is reestablished at a higher rate. When muscle stretch is reduced, during the time when change in length is occurring, the Ia afferent ceases firing while the II afferent keeps firing. A new, slower steady

state is reestablished by both Ia and II afferents when the new length is reached.

The central branch of Ia and II afferents of the lower extremities has at least three targets. One is the somatosensory cortex via and the cerebellum travelling mainly the dorsal column medial lemniscus pathway. This muscle spindle afferent input is very important in higher center movement control and will be considered later. Another target is the inhibitory neuron to the lateral spino-thalamic transmission cell in the nociceptive system. The nociceptive connections of the muscle spindle afferent input was not considered in this study (as far as it is known). Subjects in this study had no painful condition. Therefore, the pain pathways were expected to have no effect on the outcome of this study.

The third target is the anterior horn cell (AHC) pool of the muscle in which the muscle spindles are located. This connection completes the monosynaptic stretch reflex pathway, the shortest and fastest neural pathway. The reflex produces an excitatory influence on the muscle when stretch is applied to the muscle. One might expect that when a muscle contracts, it would stretch its own fibers and produce a reflex excitation. In moving, it might also stretch its antagonist, producing a reflex contraction that would retard movement in the intended direction. In spinal controlled movement, the typical response would match the magnitude of perturbation. If this actually occurred (and it does clinically), it would make for very

inefficient movement. In learned voluntary movements, such as those used in this study, the self-induced stretch reflex is seldom seen in reciprocal inhibition coordinated purposeful movements.

The self-induced stretch reflex does have a regulatory role during co-contraction activities. For example, during precision movement or postural stabilization, a perturbing force disturbs body segments from their intended path of movement or state of postural equilibrium in a direction that would suddenly elongate the muscle. If such a perturbation were to occur, the reflex would cause correction back toward the intended path or posture.

The reason the stretch reflex does not self-induce excitation in the same or antagonistic muscles during learned reciprocal inhibition controlled voluntary movement is that higher command cells inhibit the AHC presynaptically through the Ia inhibitory neuron. The same higher centers control the activity of the gamma motor neurons that innervate intrafusal muscle fibers in the muscle spindles to suppress excitation during initiation. This occurs because there is sensory input from all the sensory modalities which informs the somatosensory area of the amount of stretch before the movement starts. Muscle spindles and anterior horn cells are inhibited slightly to ensure no stretch reflex activity occurs. This higher center inhibitory influence is strong enough to invert the relationship between stretch and excitation. The result is a slower than expected time of muscle recruitment of more elongated muscles.

When a muscle contracts, inhibitory influences are passed through these spinal cord pathways to its antagonist either before or during the contraction. There are three ways this can occur. First, presynaptic inhibition whereby the motor neurons of the muscle to be contracted send collateral branches to the Ia inhibitory interneurons which terminate on the antagonistic AHC.[6] The second is through the Ia afferent neuron of the agonistic muscle spindle which sends a collateral branch to the Ia inhibitory interneuron synapsing with the antagonistic AHC. The third way is through the agonistic efferent motor neuron which sends a collateral branch to the Renshaw interneuron. The Renshaw interneuron is inhibitory to the agonistic AHC (this short recurrent loop will dampen the quick changes in the agonist) which causes a disinhibition of the antagonist's AHC,[7] and to the Ia inhibitory neuron. It would seem that there would always be strong inhibitory influence to any antagonistic muscle. However, both the Ia inhibitory and Renshaw cell have multiple synapses with the higher brain centers through the descending pathways. The higher centers can change the preponderance of inhibitory and excitatory influences on the agonistic and antagonistic alpha motor neurons so that the movement relationship between agonistic and antagonist is under reciprocal inhibition strategy or co-contraction strategy. One performance condition of this study was a voluntary lift into hip extension. It was expected that this movement was coordinated through a reciprocal inhibition strategy. The other performance condition was an

isometric hold. It might seem that co-contraction would be a control strategy with this movement. However, this movement was a hip extension hold in the prone position. In this case, the hip extensor was expected to be the agonist and gravity was the expected antagonist. Gravity, in this case, was expected to perform the same function the antagonist would, if a stable or precise hip extension position were to be held in another body orientation.

The Golgi tendon organ (GTO) is another sensory receptor in muscle that can supply information regarding the external and internal environment. While the muscle spindle is more sensitive to passive stretch, the GTO is usually activated when the muscle actively shortens or tries to shorten. Acting through spinal reflex pathways alone, it is inhibitory to its homonymous muscle and excitatory to the antagonistic muscle in which it is located through interneuron.[8] The central fiber of the GTO takes similar pathways as the muscle spindle. The three targets of the GTO central fiber are: the somatosensory cortex (via the dorsal-column-medial lemniscus pathway), the nociceptive inhibitory neuron, and other spinal interneurons. There are several differences between the muscle spindle and the GTO. One difference is that the GTO reflex pathway goes through interneurons, rather than taking a monosynaptic path as do spindle impulses. Another is that the GTO is inhibitory to the muscle from which it comes and excitatory to its antagonist. Finally, the sensitivity of the GTO cannot be influenced directly through the gamma motor system as can the muscle spindle.

Higher centers, through descending inhibitory and excitatory influences on interneurons and the AHC, modify the spinal reflex behavior that could be attributed to the GTO. The possible influences of these sensors may underlie some of this study's observations.

The third type of epicritic sensory receptor input which is important to this study is the joint receptors. Wyke has extensively studied the effect of joint receptors on muscle activity. He identified four types of receptors in joint tissues. Type I receptors are similar in construction to cutaneous Ruffini corpuscles. They are located in the superficial layers of the joint capsule and attach to Group II myelinated fibers. Type I receptors are more numerous in areas of the joint capsule that undergo greatest changes on stress. They are low-threshold, slowly adapting, and some are active in every angular joint position. There is a constant resting discharge from the receptors in the area of joint capsule stretch when the joint is in a constant position. Increase of stretch due to muscle contraction also increases firing in these receptors. They are constantly active, signaling static joint position when the joint is still, and signaling direction, amplitude, and velocity during dynamic movements.

Type II receptors are elongated, conical-shaped Ruffini corpuscles and are located in the deeper layers of the fibrous joint capsule and over fibro-fatty pads. These receptors are low-threshold, rapidly adapting. They elicit no signal when the joint is motionless; but, at the onset of movement, they

produce a brief high frequency burst. Another brief high frequency burst occurs when the joint motion stops. Therefore, they are classified as dynamic mechanoreceptors signalling changes in acceleration.

Type III Golgi joint receptors are similar to GTOs and seem to operate in the same way; that is, sensitive to tension. They are located exclusively in ligaments and are active only at the extremes of joint motion, or when the ligament is under stress. Their afferent connection is to large myelinated fibers with extremely fast conduction velocity. They adapt very slowly.

Type IV receptors are pain receptors. Since pain was not an issue in the present study they will not be considered here. Wyke monitored the electrical activity from muscles while performing different manipulations of the joint capsule. For most of his experiments, muscles were tenotomized to eliminate muscle spindle effects. He summarized his findings as follows:

> ..."Type I mechanoreceptors contribute reflexively to the maintenance of tone in the muscles of the limbs, spine and jaws at rest and to the coordinated reflex regulation of isotonic and isometric changes in the tone of these muscles when movements are made, or attempted; in addition, these same receptors contribute to conscious perception of static joint position and dynamic joint movement--that is to say kinaesthesis. In contrast Type II and III mechanoreceptors are reflexogenic only, the former provoking transient "booster" changes in the tone of the muscles operating over the joint and during the actual period of acceleration or deceleration of the articulated bones, whilst the latter act as "brakes" to limit excessive joint displacement."[9]

From the foregoing it might appear that type I and II receptors are complementary to the muscle spindle. Type I seems to assist the dynamic

and static spindle receptors in supplying information regarding static position, direction, and velocity of movement. Type IIs seem to assist in another way. When a muscle contracts, the extrafusal fibers shorten, causing a slack in the muscle spindle. The result might be a momentary inhibition of the muscle. However, the type II joint receptor which excites the muscle when the movement begins may offset this effect.

Type II joint receptors also signal velocity changes and excite muscles producing the movement when velocity changes. Since velocity of movement was controlled in the experimental tasks, this type of information supplied by this receptor should have no effect in this study.

Information supplied by Type III receptors could have had a profound effect in this study. These receptors have an inhibitory effect on muscles moving the joint toward the extremes of ligament stretch. Subjects in this study, regardless of group membership, were tested in a prone lying position where the test movement took the hip joint to or very near the end of the joint range of motion. The study was planned so that subjects under different circumstances would start movements closer or further away from the position of maximum joint capsular stretch. A subject who started further away from capsular stretch might have shown a different muscle recruitment than a subject who started the movement closer to the position of maximum capsular stretch. Electromyographic recordings may have reflected this difference.

Joint receptor information follows the same pathways as the other epicritic receptors. The ascending dorsal column medial lemniscus pathway takes information to higher centers. At the spinal level, joint receptors operate through the same reflex loop as do the GTOs and cutaneous receptors. This is the Ib interneuron loop. The Ib interneuron has numerous modifying connections from higher centers.[7] Higher centers often exert a inhibitory influence in the interneuron loop. Therefore, motor responses that one could directly relate to joint receptors and their spinal reflex connections are usually controlled by higher centers. As with other mechanoreceptor information when movement is voluntary instead of reflexive, it exerts and influence rather than induces a recognizable response.

Wyke's experiments were conducted on extremity joints. More recently, Pickar and McLain mapped out mechanoreceptive endings in the lumbar spine. Some of these were in the facet joint capsule and others were in the paraspinal tissues, such as in muscle. All were found to be directionally sensitive and many responded to facet joint movement in a graded fashion. Some of their receptive fields were located quite far from the area of the nerve root.[10]

Cutaneous receptors for skin indentation are the slowly adapting Merkel receptor and Ruffini receptor. These receptors are sensitive for pressure on the skin and stretch of the skin. They both signal when pressure is both placed on the skin and when it is taken off. Both the sensation of

increased skin pressure and decrease in skin pressure may have occurred in this research.

Like the GTO and joint receptor, the spinal reflex loop travelled by the cutaneous receptor input is through the Ib inhibitory interneuron. The Ib inhibitory neuron connects and relays to AHCs of both the muscle overlaid by the skin and its antagonist. This interneuron is synapsed extensively by the higher centers, and higher CNS modulation is expected. The other pathway travelled by messages coming from these receptors is the dorsal column medial lemniscus pathway to the thalamus and ultimately to the somatosensory cortex.

Hagbarth studied the effect of cutaneous receptors on muscle inhibition and excitation. He found that stimulation of the skin over a muscle produced a reflex excitation to a muscle and inhibitory effect on the antagonist. One can assume that this reflex operates like other reflex loops and that, up to a point, the greater the strength of the stimulus the greater the response.[11] It would seem that cutaneous receptors might be the most susceptible to modification of influence due to prior learning. First, they would need to be the most susceptible to accommodation. It would not be a good system that did not allow tactile stimulation to be ignored. The extreme dysfunction that results from inability to accommodate is seen in spinal cord patients. Second, the higher centers must be able to modify the response to tactile stimulation. If increased pressure always resulted in increased activity

in the agonist and inhibition in the antagonist, movement would be difficult. There are countless examples of movement in which increase in agonistic activity is exactly the wrong response to increase in pressure, or vice versa.

Vestibular receptors for the vestibular system are the hair cells of the vestibular epithelium located in the bony labyrinth of the inner ear. Afferent fibers take messages from these receptors and distribute themselves to three brain stem pathways: the medial, lateral, and aminergic. Afferent input from the vestibular receptors is compared with somatic and visual input so that a coordinated motor response can be formed.[12]

Spinal cord influences on individual muscles and on muscles in coordinated action.

One contribution to the neuromotor system by the spinal cord is propriospinal neurons, interneurons, and motor neurons, or AHCs. Interneurons interconnect neurons; e.g., AHC with another AHC, AHC with a descending pathway axon, or interneuron with another interneuron. These neurons stay within the confines of the spinal cord and span only one to two segments of the cord. Propriospinal neurons have similar types of synapses but are more than two segments long. Sometimes they project outside the spinal cord to make a synapse. From their connections and location relative to AHCs, it seems obvious that these neurons modulate and coordinate the activity of AHCs. Interneurons and propriospinal neurons are the hard wiring

for coordination between different neuronal pools. It is assumed that a certain amount of the change in motor behavior observed when one learns a motor skill has to do with reorganizing the pathway taken by impulses through interneuronal and propriospinal pathways.

Muscles can be controlled by spinal cord pathways to produce movement independently (reflexively), or spinal cord pathways can be used to produce movement that is hierarchically controlled. Spinal cord initiated and controlled movements are the simplest type of movements. The simplest movements are spinal reflexes such as the stretch and withdrawal reflexes. The composition of these simple reflexes includes one or two synapses, and may include one interneuron that can modulate the movement. Spinal cord controlled movement, however, can be quite complex. The interneuronal integration for many complex repetitive movements exists in the spinal cord in the form of interneurons and their connections with motor neurons. Such arrangements are called central pattern generators and consist of interneurons that switch on and off patterns of muscle contractions that accomplish the repetitive task. The higher centers initiate the central pattern generator activity and then have that activity regulated at the spinal cord level. This allows the higher centers to be free for decision making, task switching, and other higher processing tasks. Gait is an example of a complex repetitive movement regulated at the spinal cord level. Although the focus of this study was not gait, the central pattern generator

phenomena and its control illustrate what often occurs during voluntary movement. Such observations have prompted researchers to comment that movement is accomplished by higher centers controlling the reflex pathways of lower centers.[13]

Brain stem control of individual muscles and muscles in coordinated action.

The brain stem initiates and controls movement when posture and/or equilibrium is challenged. Movements are initiated in response to perturbations. Examples of movement controlled by brain stem centers are body-on-body adjustments when posture is perturbed, and visual righting reactions when the head is tilted. Brain stem controlled posture and equilibrium responses can be programmed into the motor plan by the higher centers. From the vestibular receptors, afferent fibers take messages from these receptors and distribute themselves to three brain stem pathways: the medial, lateral, and aminergic. Afferent input from the vestibular receptors is compared with somatic and visual input so that a coordinated motor response can be formed.[12]

The medial pathways start in the vestibular nuclei and tectum of the brainstem. Axons from both sources send collaterals to the medial reticular formation as they travel into the spinal cord forming the reticulospinal, vestibulospinal, and tectospinal tracts at the base of the medulla. Axons from these tracts synapse mostly with axons and interneurons located more

medially in the spinal cord. More medially located motor neurons innervate axial muscles that control posture, balance, and equilibrium. The lateral pathway starts in the red nucleus and descends as the rubrospinal tract to synapse with interneurons and motor neurons in the lateral part of the anterior horn. These cells are associated with muscles of the extremities. The pathway also maintains balance and posture, but the muscles coordinated are more in the extremities. The pathway is also involved in the coordination of extremities that are functionally related. The aminergic pathways connect reticular formation neurons with motor neurons throughout the spinal cord. These pathways are said to be responsible for generalized excitation of motor neurons.

Like the receptors of the spinal level, input from vestibular receptors can cause a local reflex motor response. For example, if a person in standing is suddenly and unexpectedly pushed or pulled, there will be a motor response that not only may include a single muscle, but may include several muscles recruited over a wide area of the body. Depending on the magnitude and nature of the disturbing force, the response can be quite complex. It may literally be a total-body response.

Alternatively, the vestibular receptors can be the source of information by which the higher centers select and formulate a response. The response that results from higher CNS control incorporates and modifies the local response into a coordinated action that may be more pre-planned than a

simple reflex response. The receptors inform the total body what is likely to occur if the correct action is not taken to the vestibular input. Just before movement, muscles are contracted in such a way as to counteract the upcoming perturbation.[12] Since the tasks in this study were voluntary, this type of pre-planned postural muscle activity was most likely to be observed.

Subjects in this study performed a movement while lying prone. It would seem that initiating a movement from this position was not a very challenging position for the vestibular motor system to control. However, each subject lifted the right leg from a platform or held it in place while a platform fell from beneath it. This may have caused a pre-planned motor response that occurred before the movement or early after onset. A situation was created in which the subject maintained balance and moved despite the fact that the support surface decreased its area by one fourth, while the load distribution was changed from even to uneven. One muscle that might have responded as part of a vestibular system reflex or pre-programmed response was the left hip flexor. In lifting the right lower extremity into hip extension, the subject contracted the right hip extensor muscle. It would seem that the left hip flexor would be required to stabilize the posture and keep the body from falling in the direction of the lifted or held leg. To test this assumption, a pilot study was conducted employing EMG monitoring of the activity of the left hip flexor while the right hip was extended from a prone position. The study indicated that the left hip flexor did indeed contract just at or slightly

before the right hip was extended. There were two groups in the pilot study, one with tight anterior hip structures (n = 7) and one with excessive anterior hip flexor extensibility (n = 5). It was anticipated that there would be a significant difference in left hip flexor onset between the groups. However, no difference was found. This indicates that the timing of onset of the left hip flexor may not be related to pre-movement differences in sensation through the vestibular system.

The widely-accepted clinical observation of back extensor hypertrophy (excessive strength) when a patient has hip flexor tightness is assumed to be controlled by these mechanisms. In standing, in patients with tight hip flexors the pelvis forwardly rotated in standing. When walking, the patient tends to have the pelvis pulled forward in terminal stance. Both of these give the spine the tendency to fall forward. The vestibular and optic righting reactions are assumed to operate in such a way to return the upper trunk to erect so that the eyes are even with the horizon. Part of the operation to maintain the trunk is assumed to be a fairly constant excessive activity in the lower back erector spinae.

Auditory-motor influences on muscle behavior

The motor behavior of a newborn infant is a mixture of random movement and reflex generated movement. One needs to observe an infant for a short time to see that auditory-motor reflexes are a part of the

repertoire of automatic movement. One need only watch a track meet to observe that voluntary response to a sound stimulus can be highly trained and that certain postures help the sprinter to respond better to the sound stimulus. However, the auditory motor behavior requested of the subjects in this study was a metronome to which a voluntary movement was made. The subject chose the response time. It was more like dancing with timing of the movement to a rhythmic sound stimulus. Anyone who has observed a group of persons dancing will know that keeping time to a rhythmic sound stimulus is probably a normally distributed trait. There is little reason to believe that membership in any hip type group affected the ability to move the leg voluntarily to a metronome beat. Therefore, there was no effort to control for ability to move the limb to a metronome beat. There should have been persons of all abilities distributed among the two groups.

Primary Motor Cortex control of individual muscles and in coordinated action of muscles.

In 1950, Penfield and Rasmussen confirmed cells existed in areas of primary motor cortex that, when stimulated electrically, caused specific muscles to contract on the contralateral side of the body. This suggested a direct connection between cerebral cortex cells and the anterior horn cells of the spinal cord. Since stimulation of specific areas of the motor cortex produced contraction in specific muscles, a direct connection from primary

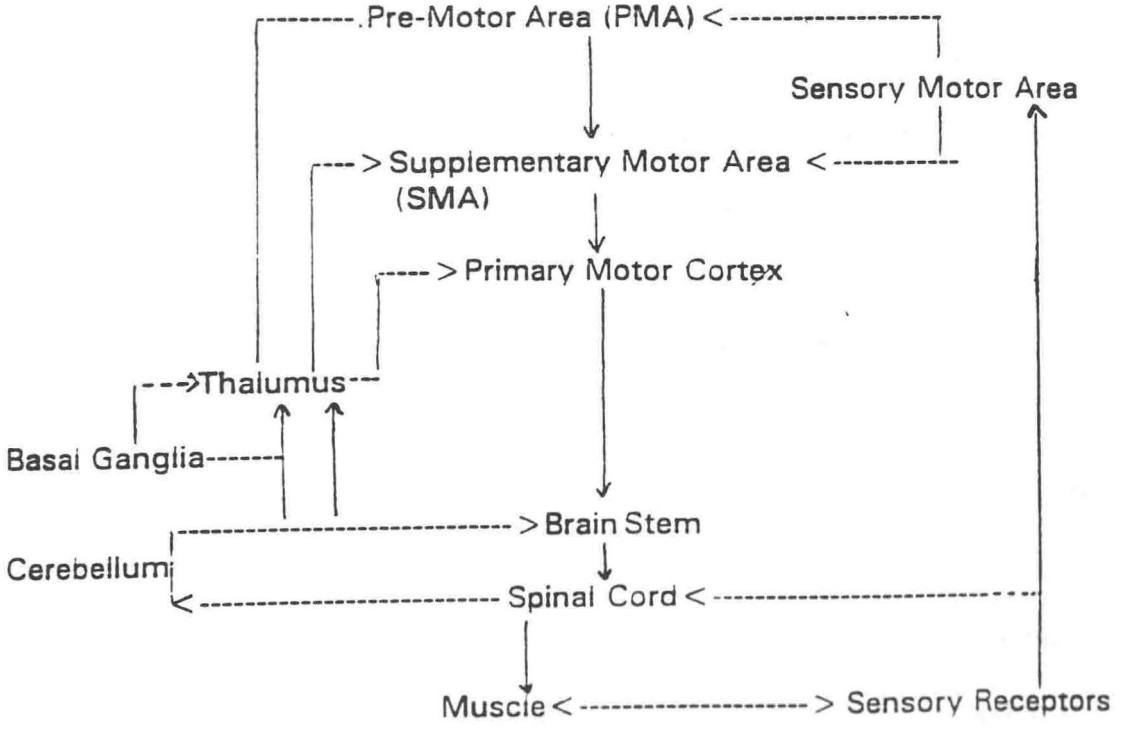
motor cell to motor units in specific muscles through the anterior horn cell was assumed. Their work established that the primary motor cortex was somatotypically organized.[14] Asunuma, et. al., studied how discrete the connection was between primary motor cells and specific muscles. They found that proximal muscles are under very specific command control of specific columns of cells in both the primary motor cortex ; i.e., specific columns of cells cause specific groups of motor units in a muscle to contract. The more distal the muscles, the more diffuse are the locations of control columns.[15] One could say that the greater the involvement in precise movement, the more diffuse the control centers. Electrical stimulation of primary motor cortex cells resulted in contraction of specific muscles with no coordination with other muscles.

Woolsley discovered that electrical stimulation of areas anterior to the primary motor cortex resulted in specific groups of muscles exhibiting somewhat more coordinated movements. Instead of electrical stimulation resulting in isolated uncoordinated movements, stimulation in these areas resulted in movements that resembled functional movements, where the actions of muscles seem to have planned onset and timing. These areas anterior to the primary motor cortex (now known as the premotor area, PMA, and supplementary motor area, SMA) were also found to be somatotypically organized.[16] That electrical stimulation of these areas resulted in specific and reproducible results at individual muscles meant that

muscle activity was to a certain extent a reflection of processes occurring in this area.

In addition to the primary motor cortex, the higher centers include the PMA, SMA, somatosensory area (the source of approximately 25 percent of motor fibers to the main motor tract), the basal ganglia, the cerebellum, and the thalamus. These structures seem to form a loop that starts with the PMA and SMA, to the primary and somatosensory motor axons. Their axons, as well as projecting to the spinal cord, send collaterals to the basal ganglia,

Figure 2.1 Hierarchical Levels of Motor Control



especially the globus pallidus internus and cerebellum. Ascending axons from spinal cord afferents synapse with cerebellar pathways that project to the thalamus. Thalamic axons project back to the somatosensory, pre-motor and supplementary motor areas. Figure 1 shows a schematic illustrating the connections of the motor learning and control loop. It is assumed that, through this loop, motor learning occurs with the development of internal models that assist feed-forward control of movement. It is assumed that the movements used in this study are controlled by this system.

Over the years, the role of the primary motor cortex has become better defined. It is now recognized that it occupies an approximately middle position (see Figure 1 above) in a hierarchical system of muscle function. If movement could be said to have a starting point, it would be in the sensory cortex that informs the highest level motor areas of the need to move and the environmental conditions under which the voluntary movement will take place.[17] The sensory cortex represents the highest level of motor control. The sensory area directs the highest motor centers. PMAs and SMAs produce a motor plan. This plan selects the appropriate primary motor area cells and activates them in such a way as to provide all the force necessary for a movement.[18] The plan is based on sensory information and prior learning. The PMA and SMA encode rises in rate, duration, and sequential onset of all primary area cells they estimate to be necessary to complete the task.

In the not-too-distant past, there was some controversy as to what was encoded in the primary motor cells. Some suggestions at that time were joint position, coordination of reflexes, anticipated sensation, force, change of force, and duration of force. There is a common functional movement error phenomenon that suggested the answer. That error occurs when a person lifts an object that he/she supposes to be heavy but, in fact, is light. The result is an inappropriately strong muscle effort. This suggests that the primary control parameter that the SMA and PMA encode in the command cells is force, change of force, and duration of force. This was confirmed by Evarts who determined that motor cells in the primary motor cortex encode force and change in force in determining muscle function during a voluntary or involuntary movement.[19]

A motor program or plan is a set of pre-structured muscle commands based on a predetermined environmental objective executed to cause movement which achieves that objective in the absence of peripheral feedback. The commands of a motor program can be modified by peripheral feedback.[20] Once the program is formed and selected as part of the motor plan (number of command cells active, frequency, and duration of activity), the program is executed by the command cells. Force and change of force are signalled to the AHC by frequency of impulses generated from the command cells.[19]

Force is magnified by increasing the number of primary motor cortex cells. This expands the pool of active AHCs, and thus the number of active motor units. In addition, there is an increase in firing frequency of active cells. The increases in frequency and number of active motor units are reflected in the EMG record and increases in frequency, amplitude, and density of signal. Measured over time, EMGs reflect PMA and SMA commands. Usually, the image of the motor program is not an exact copy of the program that exists in the PMA and SMA. Sensory and cognitive feedback constantly incoming from various sensory areas cause some modification.

The higher centers initiate all movements having conscious intent and control when a movement is complex and not well-learned. Higher centers are also the seat of motor learning. Once even complex movements are learned, higher centers pass off the execution to lower centers and retain certain aspects of initiation only.

Complex movements are ones in which there are different sequences to the movement, there are many different joints and related muscles involved, and for which postural adjustments must be made. The higher centers typically control movement when complex movements are being learned. Through the sensory cortex (visual, auditory, vestibular, etc.), the SMA and PMA are presented with a goal-oriented problem that needs to be solved by movement. If the problem is not a familiar one, the PMA and SMA

will not have an internal model or motor program to solve the problem. Theoretically, a library of motor programs that have been learned exists in the PMA and SMA. From this, a trial model or program is selected that seems a good solution. The trial program is sent to the primary motor cortex to be executed by the command cells. At the same time, an efferent copy of the program is sent to the basal ganglia, the cerebellum, through the thalamus, and back to the sensory area. The efferent copy arrives back at the sensory cortex first and awaits the arrival back to the sensory cortex of the peripheral proprioceptive sensation associated with the movement. The time difference is a result of the difference in length of the loops travelled by the efferent copy and the movement sensation. The former is short and the latter is a long loop. The returning sensory information is compared with the efferent copy and knowledge of results (success or failure) and knowledge of performance (the correctness of the elements) is obtained. Using the knowledge of results and performance, the original motor plan and program may be modified by the SMA and PMA. On the next trial, a modification of the original trial motor program is executed with a new efferent copy. The voluntary movement is practiced this way until the problem is solved to the desired level of safety and efficiency. At a neural level, the process of developing an internal model or program involves the higher centers gradually developing automatic switches that turn on and off interneuronal activity in the brain stem areas for vestibular control, and in the spinal cord for

sequencing the muscles. Once the internal model is formed and is successfully used to produce the movement, the internal wiring necessary to produce the movement using lower center control is established. Even in very complex movements, the only role of the higher centers is to perceive the need for the movement or the need to stop the movement and execute the commands to do so. Once the movement is started, it is executed without reference to the higher centers unless the higher centers perceive a need to stop or change.[18,21] This explains the almost automatic complex and seemingly effortless movement of performing artists and athletes. It also explains the left foot activity that occurs when a person, familiar with driving a standard transmission car, drives an automatic shift car. If the person is distracted, the left foot will perform the action of manipulating the clutch that is not there.[22]

This study looked at muscle behaviors in the time just before movement. It was presumed that in this pre-movement period the influence of the pre-programming and motor sequencing areas of the cortex is most clearly seen.

Feedback and feed-forward control mechanisms

There appear to be two modes of neuromotor control, feedback control and feed-forward control. These modes are not used exclusively. During feed-forward control, for example, feedback plays a secondary role.

Both control modes are heavily dependent on incoming sensory data, feedback mode needing continuous sensory input and feed-forward requiring post-movement sensory perception.

Feedback control is used when the intent of the movement is to maintain a position, maintain a level of force, or move with precision. In this type of system, a comparator mechanism compares the difference between the reference signal and the feedback signal and adjusts a controller mechanism up or down to eliminate the difference between signals. There is continuous comparison between the reference signal and the feedback signal, and success or failure is a moment-to-moment event. In simpler feedback systems, the reference signal is a stable one and the task is regulator; that is, to maintain a constant relationship with a constant signal. In this study, both tasks might have called for some feedback control. In the first task, the subject lifted the leg from the support surface to the count of a metronome. The reference signal came from the metronome and sensory feedback from this signal may have affected the results of this study. In the second task, the subject held the lower extremity at a constant point while the support surface dropped from beneath it. During this time, the support surface was dropping and thus was giving gradually less support to the limb. There was a changing reference signal and the task was to maintain a constant relationship to a constantly variable signal. In this study, a subject had an initial reference signal just by lying in the test position. He/she had

the sensation of joint position and internal tissue tension that would supply this signal. In both tasks, the subject probably used the joint position sensation and internal tissue tension from the muscles involved as a controller mechanism to match to the reference signal. Monitoring the electrical activity from these muscles should reflect the adjustments the subject made in the controller mechanism both before and during the movement. This activity should reflect what the motor system senses it needs to do to move the leg in time with the metronome or hold the leg in the same position. In other words, it may have been a reflection of the somatosensory area's assessment of the state of the organism. As the leg was held, the position in which the subject held the limb may have been an image of the overall difference the subject sensed between the reference and feedback signals; i.e., the comparator mechanism.

Feed-forward control is used by the neurological system when faster and well-learned voluntary movements are performed, especially when the intent is to efficiently move an extremity or body segment in a specific direction. In these situations, feedback control is too slow and inefficient. Both tasks in this study called for some elements of feedforward control. Both tasks were very simple and subjects were permitted to practice several times until a certain level of performance was met. This practice and other prior experience with similar movements should have caused an element of feedforward control. It may have been that initiation of both tasks were

under feedforward control and the movement under feedback control. Nevertheless, whether there was feedback or feedforward control, the effects of pre-movement sensation may have been seen.

In feed-forward control, success or failure is not immediately known, but usually the result of an after-the-trial comparison among the internal model, the knowledge of results in the form of some external representation of success or failure, and knowledge of performance in the form of the later-arriving sensory feedback. If there is a good match when these three are compared, the internal model will remain the same. If there is a mismatch among the three, the internal model will be modified. The modification will be based largely on the sensory feedback and its comparison to the internal model. One can see the obvious importance of sensory feedback in this development of feed-forward control.

If one has a movement that might be a combination of feedback and feedforward control, when in that movement is the best time to record events related to feedforward control? Before movement, three major aspects of movement control are selected. These are the muscles necessary for the task, their intensity of contraction, and the type of control (feedback or feedforward). Once movement is started, the type of control determines whether there will be any adjustments during the movement. If movement is under feedback control, changes can occur as soon as input returns through the feedback loop. If under purely feedforward control, the motor activity

transpires unchanged from that pre-programmed on the basis of previous experience. It would seem that the best time to gain information regarding the motor programming processes would be just befor or at the onset of movement. Feedback requires some information about which to feedback. On the basis of this and reasons stated later, two major assumptions of this study were that the best time to observe motor programming and control processes is at the onset of movement, and that muscle activity observed at the onset of movement is directly associated with motor planning and programming processes.

Muscle coordination patterns in the pre-movement period

One of the basic tenets of this study was that muscle behavior observed in the pre-movement period can be taken as evidence of the motor pre-planning done by the neuromotor system. A combination of electromyography (EMG), with some related measures of force and/or motion offers a method of studying motor control during movement. The strategy of this type of study is as follows: First, the force and motion characteristics of the onset and actual movement as reflected by the devices measuring these parameters are well known. These measuring devices collect force and motion data simultaneously with EMG data during the pre-movement and continuing throughout onset and actual movement. Logical connections are made between the EMG, force, and motion changes observed during the pre-

movement period and those observed or expected during onset and movement. For example, when a person stands on a force platform, it reflects both the force of the center of gravity and the force of the foot pushing on the plate. Reflective markings record the flexion-extension motion of the knees. EMG recordings are taken during the pre-movement, onset and movement periods, from posterior and anterior ankle muscles. If that person was to perform the rise on toes movement, the expected events are a rise in pressure under the forefoot with a backward movement of the center of gravity. The knees should flex because of forceful activity of the triceps surae muscles at the knee. The EMG should show a great deal of activity from the gastro-soleus muscles with later activity from the anterior muscles because of the backward movement of the trunk (and with it the tibia in relation to the foot). However, if the individual being tested is neurologically well-functioning, events in the pre-movement period modify what is expected and the purpose of each modification can be deduced from knowledge of mechanics. In the pre-movement period, the event is EMG activity in the anterior tibial muscles along with forward body sway. The purpose of this is to reduce the moment arm along the foot before the movement. The large burst of activity following in the gastro-soleus does not cause knee bend because of the previous body lean and activity in the quadriceps muscles. It is only after all these events take place in the pre-movement period that onset of movement occurs. These mechanical events,

along with the pre-movement electrical activity in the muscles, are assumed to be pre-planned in a program for rising on the toes. Use of these methods has returned a wealth of information regarding motor learning, motor planning, motor programming, motor regulation, and motor control. Since, as mentioned previously, the motor program of well-learned movements is passed along unmodified, one can compare the EMG record from a condition with known sensory input with a condition with new sensory input. The difference is evidence of the effect of the new sensory input.[5]

Anticipatory Postural Adjustments

For nearly a century, a large number of studies have examined neural control of movement of the pre-movement period. Much of this research has focused on the relationship between the control of upright posture and movement, particularly the control of posture during the initiation of movement. The events of most interest in this area of investigation are postural adjustments (PAs). These researchers have clearly established the basis for making the previous assumptions. Moreover, they have investigated some of the same observations of muscle behavior (duration of onset sequence, effects on muscle latency, effects on EMG amplitude) as undertaken in this study. The role of pre-movement sensation and prior learning on coordinated muscle activity associated with onset of movement in both normal subjects and subjects with various neurological

pathologies has been studied. The theoretic constructs and logical basis of this study owe much to this body of investigation. Extensive summaries of this area of investigation have recently been done by Oddsson[23] and later by Massion.[24]

The study of postural events associated with voluntary movements in the standing position has been so extensive that investigators have attempted to develop their own nomenclature to describe events. PAs include muscle activity, change in center of gravity or segmental accelerations. Words used to describe these events have been "postural adjustments," "consecutive postural adjustments,"[25] "postural preparations,"[26] "postural bias,"[27] "automatic postural adjustments,"[28] "anticipatory postural adjustments," "postural reflexes," and "automatic postural responses."[29] Although their terms are similar, careful reading of these articles suggests that authors are clearly not discussing the same kind of activity. Frank and Earl present a classification that seems useful in distinguishing the different types of postural motor behavior associated with intentional movement. There appear to be three different types of motor behavior identified with the postural subsystem. Postural preparations (PPs) are more or less static joint positions and muscular activity that occur well before the movement commences. For example, a person may crouch just before jumping. Anticipatory postural adjustments (APAs) are joint position changes, shifts in center of gravity, and muscular events that occur

milliseconds before, simultaneous with, or milliseconds after a movement. An example occurs when a person shifts the center of mass forward just before rising on the toes. APAs seem to anticipate the mechanical events that take place during early movement. Postural responses (PRs) are joint movements and muscular events that occur only if the intended movement is perturbed. For example, a person will rock side to side or throw an arm upward when moving from two-legged to one-legged standing if he/she makes the change too quickly.[30] These events are meant to control the perturbations caused by the subsequent intended movements.

Studies of APAs probably began with observations made by Babinski in 1899. He observed that a patient with cerebellar dysfunction could not perform a backward trunk movement in standing without losing balance. He reasoned that the cerebellar dysfunction patient failed to execute knee flexion that seemed to automatically accompany backward trunk bend in persons with normal cerebellar function. With normal cerebellar function, knee flexion automatically occurs as a PA that is necessary to keep the center of gravity within the base of support.[31] Later, Massion deduced that knee flexion alone was not sufficient to keep the center of gravity within the base of support, but that there needed to be an accompanying ankle dorsiflexion. It was noted that there was close coordination between two joints which are distal to where the intended movements occurred.[32]

Beevor was the first to study the muscular onset phenomena of APAs. He noted that the erector spinae muscles contracted when an arm is moved forward and upward. He stated that the purpose of the contraction was to prevent dysequilibrium of the trunk during the voluntary movement.[33] In the intervening years, the idea developed that there were two systems operating when a normal subject performs a voluntary movement. One system produces the movement and one system maintains equilibrium of the body during the initiation of movement. APAs have become the investigative focus because they offer the opportunity to study the in-situ coordination between two subsystems. APA inquiries use most of the same methodology as this study. For example, the simultaneous collection of kinetic data consisting of center of gravity, center of pressure, and electromyographic and kinematic video analysis of segmental angulation and linear movement.

At first, PAs were thought to be PRs, probably because data at that time were based only on visual or tactile observations. It was assumed that PRs were the result of reflexes in postural muscles that were put in a position of stretch.[34,35] It was thought there was a global postural feedback system that controlled all perturbations during movement.This idea was held until Belen'kii proposed that APAs were pre-programmed; i.e., feed-forward activity instead of feedback activity. The subject's task in this study was to perform unilateral or bilateral shoulder flexion in a standing position. Two types of muscle activity occurring in the pre-movement period were

distinguished: preparatory and compensatory. Preparatory activity took place in the prime mover before movement occurred. Compensatory movement was defined as the electrical activity in trunk and leg muscles whose apparent purpose was to minimize the perturbations of the upcoming movement. Such muscle activity was very task specific; i.e., changed with slightly different tasks. The pattern of muscle activation was stable in both type and sequence.[36] There have been numerous studies subsequent to Belen'kii, et. al., in which the standing position raising one or both upper extremities paradigm was used. In fact, this seems to be the favorite paradigm. Bouisset and Zattara, studying standing subjects, found consistent patterns of accelerations of the center of gravity and accelerations of individual arm and leg segments. They found these force changes could be related directly to consistent patterns of EMG activity of leg and trunk musculature. The consistent pattern of EMG activity included both inhibitory and excitatory muscle activity. Lower extremity and trunk EMG activity always preceded the onset of the agonist's EMG activity (in this case, the anterior deltoid). The trunk and lower extremity activity clearly seemed to have a postural stabilization purpose. Both force changes and EMG were task-specific. For example, there were clear differences between APAs associated with unilateral shoulder flexion and those associated with bilateral shoulder flexion. They found both the kinematic and kinetic aspects of APAs to be highly reproducible.

> "The pattern of local accelerations is consistent and corresponds to a given set of conditions. .....The reproducibility of the EMG pattern in the muscles concerned, and the chronology of their activation, is remarkable, given their multifunctional action and multiple causes of variations in the execution of the movement."[37]

Another study by the same group compared postural adjustments during unilaterally and bilaterally performed shoulder flexion. APAs exhibited as a result of left arm flexion were mirror images of those caused by right arm flexion. APAs produced as a result of bilateral arm flexion were considerably shorter and less complex than unilateral. Weight added to the arm under any condition caused APAs to commence earlier relative to the onset of arm acceleration and be of larger amplitude. This study provided definitive evidence of the pre-programmed control of PAs.[25,38] It also seems to suggest increased resistance caused onset sequence to be prolonged and muscle amplitude to be increased. Caution must be exercised in directly extrapolating the results of this inquiry to this study. In the present study, the subjects were lying prone. In the reported study, subjects were tested in standing. Upright standing is a much more susceptible position for losing balance. Greater perturbation would have more consequences. Therefore, the prolongation of onset sequence with greater resistance may have more to do with preparing to deal with greater perturbation. In prone lying, there may not be the need to onset postural support muscles earlier relative to movement onset. Moreover, not all the the muscles monitored in the present study were postural muscles.

Burbaud, et. al., studied monkeys lifting one upper extremity upward in a standing crouched position. In all subjects, there was clear evidence of APAs even when the head, contralateral arm, trunk, and lower extremities were constrained and externally supported. When the subject reached upward using the flexor muscles, there was simultaneous muscle activity of the extensor muscles of the contralateral upper extremity. Unlike Bouisett and Zattara, however, they found high variability in the latency of activation relative to the onset of movement.[39]

Other methods of studying APAs in standing normal subjects have been pulling and pushing on handles[40,41] and during locomotion.[42] Over the last decade, Oddson has conducted numerous studies of standing subjects performing trunk inclinations of various kinds and with various different movement strategies.[23]

Moore, et. al., studied APAs in normal seated subjects reaching for a near and far target at different speeds. They observed force changes that preceded the onset of EMG activity in the agonist (anterior deltoid). These force changes, as was found in previous studies of standing subjects, seemed to counteract the perturbation of the upcoming movement. However, they found that force changes (in this case, the acceleration of the center of gravity) did not correlate well with muscular activity in the lower extremity or trunk. For example, they found that agonist activity (anterior deltoid) preceded trunk EMG activity in 70% of the trials. Amount of support

offered by the seating surface seemed to determine the amount of paraspinal activity.[43] This seems to suggest that with increasing support, onset sequencing is different from standing in both sequencing and duration of muscle onset. It should again raise questions regarding directly extrapolating the results of standing APA studies to results of studies that use other performance positions.

Since APAs are largely pre-programmed, they tend to occur in the same way under the same circumstances. Therefore, the possible diagnostic value of APAs was investigated. Several investigators interested in problems of motor control compared normals with subjects with various pathological conditions. Horak, et. al., studied APAs in normal subjects vs. hemiplegic patients performing upward reaching under different conditions of velocity of movement, weight moved, and task certainty.[44]

Kaneoke, et. al., studied a specific component of APA, the pre-movement silent period, or PMSP. The PMSP is the latency between a signal to move and the appearance of electrical activity in the EMG record during a muscle contraction. They found that the PMSP of the muscles responsible for PAs was increased in Parkinson's patients compared to a matched group of normal subjects. They also found that once the APAs were executed, other EMG events occurred normally during the remainder of the movement. Qualitative analysis of the EMG records indicated subject uncertainty during the APA period. This led them to hypothesize that programming of APAs is

the basis of bradykinesia seen in Parkinson's patients, rather than programming of the muscles for the intended movement.[26]

Ahern, et. al., compared the patterns of lumbar paravertebral activity between normal and low back pain subjects. They found that there were no differences between subjects at quiet standing. However, when subjects performed dynamic activities from a standing position, clear and consistent differences were found. This group only compared differences in amplitude, not differences in onset of muscles. They were able to classify normals and back pain patients using their results.[45]

Rogers, et. al., compared normal subjects with subjects having Parkinson's disease. They found both delayed onset of APAs and multiple bursts of EMG activity not seen in normal subjects. They concluded that the basal ganglia may serve a linking function between postural and movement systems that helps APAs to be normal.[46]

Recently,considerable interest in the effect of aging on PPs, APAs, and PRs has developed. This is probably due to the observation that the elderly often fall when attempting to perform voluntary movements or seem to use excessive postural preparations that interfere with free movement. Woollacott has investigated young and old to see if observed differences in patterns of muscle onset were due to actual differences in the organization of APAs or to the use of APAs as a mediating factor by the elderly. If these differences were observed, this would indicate an actual altered organization

with age. On the other hand, if organization was the same with latencies merely spread out, this would suggest that APAs were different merely because of slower recruitment of muscle. Woolacott's results indicated that the former hypothesis of actual difference in organization was supported. There were significant differences in organization of muscle onset. Coincidentally, by subjecting the older subjects to detailed neurological evaluation, they were able to show that the difference in organization of muscle onset was greater for those who had more subclinical neurological test results.[47]

A summary of research findings outlining the characteristics of pre-movement and concurrent postural adjustments may help in the interpretation of the present study. They are:

1. Once established (after learning), APAs are pre-programmed.[25]
2. APAs seem to be able to serve three purposes: a) to control movement of the center of mass; b) to generate an actual movement opposite the movement of the center of mass; and c) help position the center of mass over a new base of support.[48]
3. They are highly velocity, task, and instruction specific.[49]
4. They are affected by the aging process.[47]
5. The locus of control seems to be in the motor learning loop which consists of a coordination between the motor cortex, supplemental motor area, the basal ganglia, and the cerebellum.[30]

6. APAs and PRs are related, and when the task can be simplified by combining their effects, this is done.[50]

7. At present, there seems to be a controversy as to which characteristics of APAs are variant and which are invariant.

Some researchers found force and EMG onset highly reliable,[25] while others maintain that APAs, at least in standing, are more loosely tied to the intended movements and more influenced by the current dynamic status of the body.[28] Many authors now question the invariant nature of APAs. Badke found that APAs are influenced by different conditions of postural preparations.[27] Layne and Abraham found APAs to be influenced by the presence of postural responses induced just before the intended movement is initiated. Their study of the influence of PRs suggested that PAs can be influenced, even abolished, by the imposition of conditions requiring postural responses just before a voluntary movement. This prompted them to speculate that the apparent invariant nature of APAs seen in previous studies is due to the simplicity of previous tasks.

> "The imposition of temporal constraints typically found in reaction time paradigms may create a specific and repeatable pattern of (muscle) activation that differs from that found in self-paced or coincidence anticipation paradigms. Likewise, the imposition of a postural perturbation with an accompanying reflex activation should result in a unique activation pattern, apparently even when the reflexive activity and the centrally generated activity would be coincident." [28]

8. There is evidence that certain aspects of APAs are pre-programmed and susceptible to long-term training.

Pedotti showed that trained gymnasts exhibited a coordinated activation of muscles during fast backward bending that was different from untrained subjects. Untrained subjects showed a mass simultaneous activation of muscles, while trained gymnasts showed an activation of distal muscles followed by proximal muscles. This resulted in a better performance in both velocity of movement and control of equilibrium.[51] That trained subjects showed a different muscle activation pattern demonstrates that with different prior experience, muscle activation in the pre-movement period can be changed.

Like the study of APAs, this study examined the pre-movement muscle behavior and mechanical events. All but one (the effect of aging on APAs) of the eight points enumerated above regarding APAs were relevant to this study. It is assumed the muscle behavior and mechanical events studied were pre-programmed.

These studies show that APAs are highly related control movements of the center of mass when in the erect standing and sitting positions. Slight disturbances in posture can cause responses that would obscure observation of the pre-formulated behavior when subjects are tested in standing. For example, in standing there is a constant postural sway that might make muscle onset in the experimental task different based on where the subject is in the postural sway path. To avoid some of these problems, the experimental tasks chosen for this study were less demanding of the

postural control system. Subjects performed in a prone position. It was hoped this would eliminate some of the problems of observing muscle behavior during momentary changes in the position of the center of mass. Therefore, pre-movement muscle behavior related to control of mass in erect posture were not applicable in the interpretation of this study.

It was expected that, as was found in the APA studies, the muscle behavior would be highly velocity, task, and instruction specific. It was assumed that the locus of control was the motor learning loop consisting of a coordination between the motor cortex, supplemental motor area, the basal ganglia, and the cerebellum. It was expected that, like APAs and PRs, the muscle behavior might show evidence of tasks being simplified by combining their effects. Finally, and probably most importantly, it was expected that certain aspects of muscle behavior and mechanical events would be susceptible to long-term muscle tightness. This inquiry was an attempt to see if two groups which may have undergone different training because of different capabilities to move might have shown evidence of this different training.

Electromechanical Delay

There is another large body of research related to the pre-movement muscle behavior. This line of inquiry relates to the behavior of individual

muscles, rather than muscles in coordination. These investigations focused on the phenomenon known as electromechanical delay, EMD.

The interpretation of the behavior of the components of a muscle and eventually the behavior of opposing muscle pairs (and the behavior of related stabilizing muscles) owes a great deal to the observations made by researchers studying EMD in individual muscles. To understand EMD, one must visualize a muscle as composed of two functional components. The contractile component (CC), is the part of the muscle that actively shortens when it recieves sufficient neural stimulation. The CC consists of the actin and myosin fibrils. Since the CC contracts under the influence of efferent motor nerve, waves of depolarization run throughout this area of the muscle which can be picked up by surface electromyography. The passive component (PC), is the fibrous part of the muscle. The PC consists of the fibrous part of the muscle; that is, the fibrous network that supports the actin and myosin and the tendon. The CC pulls on the PC to produce mechanical force.

EMD reflects the interdependency of the contractile and fibrous parts of the muscle. Observing EMD requires a method of recording the electrical activity of the muscle, electromyography, and a measure of when movement starts; i.e., an electrogoniometer or accelerometer. Roughly defined, EMD is the time between the start of electrical activity of the muscle (the approximate time the contractile part of the muscle becomes active) and the

start of movement (the approximate time the muscle produces some effective force against the resistance). EMD occurs because the elastic properties of the muscle and tendon require that the contractile part of the muscle must begin its activity and tighten the fibrous part of the muscle before change in tension or movement can occur. Motor control centers must plan EMD into any schemes of movement execution. This allows a small window through which we can examine the motor control system.

Electromechanical delay, as the name suggests, is a time phenomenon. The magnitude of delay can reveal information on how the motor system assesses the pre-movement state of tissues and changes motor recruitment accordingly. The onset of movement or muscle tension is a mechanical event and is usually the constant in measuring EMD. The variable is the onset time of electrical activity.

If one has adequate instrumentation, EMD can be subdivided into two distinct divisions, force time (FT) and contraction time (CT). FT is the time between the start of EMG activity of a muscle until the muscle develops tension. CT is the time between the development of tension in a muscle and the beginning of movement. This allows us to observe that EMD can be defined slightly differently depending on the type of muscle contraction. When the type of contraction is concentric, EMD consists of both FT and CT. This is the time between the onset of electrical activity recorded from the muscle and the beginning of change in state of movement. When the

type of muscle contraction is eccentric, EMD is the time between the onset of muscle activity until there is evidence of a deceleration as measured by the movement measuring device. There is again a chance to observe both FT and CT.

The duration of EMD is very dependent upon the pre-movement state of stiffness in the muscle-tendon. Since tension produces movement, the higher the pre-movement stiffness in a muscle the shorter the EMD. Pre-movement tension can be produced by two different sources. First, a state of partial contraction in the muscle sufficient to develop tension in the tendon can exist. Obtaining EMD data when the contractile component is tonically active is problematic and usually avoided when trying to study the phenomenon. The precise point of EMG increase is difficult to ascertain because the onset cannot be distinguished from the tonic electrical activity without sophisticated EMG filtering methods. The second source of pre-movement tension in the muscle-tendon is the passive elastic force (PEF) produced by passive elongation. The effect of PEF on EMD is much easier to observe when the contractile part of the muscle is electrically silent before movement is attempted. Therefore, EMD is usually measured with the muscle at rest before the onset of movement. This is the method used in this study.

The greater the PEF before movement, the shorter will be EMD. There are several conditions that can change the duration of EMD. First, the length

of the muscle at the time of contraction. Presupposing the rate of recruitment of the CC is the same, the tighter the elastic part of the muscle is pulled (greater PEF), the shorter the EMD, and conversely, the greater the pre-movement slack of the PC, the greater the EMD. The force necessary to cause movement or develop tension against a resistance usually comes from the CC and PC. If more force is generated in the PC before movement (increase in PEF), less force is necessary from the CC to create movement. It takes less time to generate less force from the CC and therefore, EMD is shorter.

In the middle ranges of PC tension, the inverse relationship between PEF and EMD is nearly linear, but becomes less so at either end of the extremes of PC length. The reasons are part physiological and part neurological. When the PC is elongated to extremes, the added passive tension tends to make EMD very short because it requires very little CC activity to boost the force that is already present. However, EMD does not increase linearly near extremes because there is a decrease in the number of available cross-bridges limiting the time tension can be developed by the CC. Moreover, the golgi tendon organ exerts an inhibitory effect at the extremes of PC length.[52]

Extreme slack in the PC also decreases the number of available CC cross-bridges tending to prolong EMD even more than it might. However, the

muscle spindle senses slack in the CC and may boost pre-movement muscle tone tending to shorten the EMD.

The foregoing discussion clearly illustrates the two determinants of EMD: the rate at which the CC develops force during the pre-movement time, and pre-movement PC tension. The rate of CC force development operates rather like an accelerator (rate of increase in velocity) in mechanics. It is the aggregate of motor units increasing their rates of firing (cross-bridges make and break at the myofibril level) and the superimposition of the force generated by one motor unit on the force of others.

The relationship between force and EMG amplitude when the muscle action is isometric is positive and linear or slightly curvilinear for most muscles.[53] For purposes of this study, it was assumed that this positive linear relationship also would hold during the pre-movement period where the relationship is described better as between EMG amplitude and intention to develop force. If this assumption was true, increased intention to develop force would be indicated by increased amplitude of EMG. Similarly, an increase in the perception of demand for force would lead to an increase in amplitude of EMG. If, for example, a person perceives that an object is heavy when, in fact, it is light, the force (and probably the related EMG) is inappropriately excessive for the task.

In the pre-movement period, if the muscle action during a movement is either isotonic or isometric, the muscle activity is usually at some baseline

level of amplitude and increases from this baseline when the CC increases its potential to increase force. The increase in activity from baseline is reflected (depending on the sampling rate) as a more or less sloped increase in the EMG record. A steep rise would tend to be associated with a rapid increase in the CC's attempt by to increase force while a less steep rise would indicate a less rapid increase in force.

The sloped rise in EMG results mostly from summation of action potential coming from several motor units. When a motor unit twitches, the electrical activity associated with that twitch migrates to the skin surface where it is picked up by the recording electrodes. If only one motor unit is active at a slow enough rate, that activity will be recorded as one small discrete sinusoidal wave. If two motor units are active at one time, the waves are often summated into a much larger envelope. The more motor units that are active nearly simultaneous, the greater the EMG amplitude. The twitch rate of each motor unit has some effect, in that the higher the frequency of firing of the motor unit, the more chance it will summate with another. Size of motor units also has an effect on amplitude, in that the larger the motor unit, the greater the amplitude of its action potential. The greater the required force, the greater the number of larger motor fibers that are recruited. All these effects correspond to make increasing ramped appearance of the EMG record when the sinusoidal waves are rectified and enveloped. An increase in amplitude was indicated by an upward slope of

the enveloped EMG record over time, and was taken to indicate an attempt by the CC to develop more force.

Presupposing equal pre-movement PC tension, the greater the amplitude (pre-movement contractile activity), the shorter will be the EMD. As previously observed, in the middle ranges of a scale of increasing pre-movement amplitude (if PEF is held constant), the inverse relationship between pre-movement amplitude and EMD is nearly linear. However, there is a physiologic limit to how fast cross-bridges can be made and broken.

Table 2.1, below, shows the usual effects on EMD by different combinations of PEF and pre-movement amplitude. There are corollaries to this table that are important to this study. First and most clearly, the more elongated a muscle prior to its contraction, the shorter EMD should be, provided pre-movement amplitude (pre-movemennt contractile activity is constant. The second corrallary concerns the relationship between resistance

Table 2.1 The effects on electromechanical delay (EMD-latency) of different combinations of pre-movement passive elastic force (PEF - stretch tension) and pre-movement EMG amplitude/density.

PEF - tendon stretch tension	EMG pre-movement amplitude/density	Electromechanical delay
Greater	Greater	Short
Greater	Lesser	Moderate
Lesser	Greater	Moderate
Lesser	Lesser	Long

and EMD. The greater the resistance to a muscle's action, the shorter the EMD. This usually occurs because with greater resistance, tension caused by CC causes a quicker rise in PEC.

The third corollary is the type of muscle action. Eccentric muscle action usually causes a shorter EMD. This occurs because there is movement in the direction of muscle stretch which causes more PEF. Thus, smaller amounts of CC activity result in more force and shorter EMD.[54]

At the outset, is was stated that the neurological system exerted a major regulatory and control function over the musculoskeletal system. This includes alterations in the relationship between pre-movement amplitude, PEF, and EMD. The muscle spindles, golgi tendon organs, joint receptors, vestibular and cutaneous receptors are assumed to be the main neural receptors, and pathways related to them are assumed to be involved in coordinated responses. Physiological mechanisms such as conduction velocity and physiological rate limits on motor unit frequencies also exert some control. It is assumed that these neural and physiological regulation and control mechanisms influence muscle behavior differently over time based on different input over time. This study was an attempt to test these assumptions.

The concepts related to EMD were used in interpretation of this study. Latency, the time from the start of muscle activity until movement, was a dependent variable. While not divided into its component parts as in EMD

studies, latency was very similar to EMD. The experiment tried to change the length of muscles to see if there were altered combinations of pre-movement amplitude and latency depending on the group to which subjects belonged.

The effect of pre-movement sensory information on muscle coordination.

This study focused on the possibility that differences in sensory information between two groups at the outset of movement affects how muscles are organized in preparation for the movement. Sanes, et. al., showed that patients with large-fiber sensory neuropathy showed gross errors during voluntary movements especially when visual and tactile sensation was denied. They concluded that:

> "The errors in direction arise because the motor systems lack a precise representation of the state of the limb (its position in space and the tension of the different muscles) and its current properties. As a result they cannot select the muscles that are appropriate to move the limb in the desired direction".[55]

Hugon, et. al., have established that not only is pre-movement sensation important to movement control, but also anticipated sensation. They studied normal subjects seated with the elbow flexed. A load was placed on the right forearm of the subject's flexed arm causing the right biceps to contract. A handle was attached to the load that when pulled would unload the forearm. When the subject unloaded her/his forearm by pulling on the handle with the left arm (causing left biceps activity), an anticipatory inhibition of the right biceps muscle preceded the muscle activity

of the left biceps associated with actively unloading the right. In this way, the right forearm stayed in a constant elbow-flexed position, presumably preventing the forearm from flying upward when the right forearm was unloaded. When the right forearm was unloaded by the experimenter, no prior-to-unloading inhibition of right biceps was observed. This indicated that, in the motor program for self-unloading, the subjects included an inhibition of the right biceps that was appropriate in onset and amplitude. For the experimenter-unloading condition, no such inhibition was included in the motor program. The self-unloading condition had an anticipated sensory condition that modified the program. This sensory condition could not be developed in the experimenter-unloaded condition. Presumably, the reason subjects learned a different motor program for the self-unloaded position was that a difference in anticipated sensation existed in the one condition that was not in the other.[56]

More recently, Kaluzny and Wiesendanger investigated whether this effect of pre-movement sensation on posture and movement also was similar when the same paradigm was used in testing the hands. The question was raised because more proximal muscles are assumed to operate more often in the reciprocal inhibition mode, while more distal muscles of the hand operate more often in the co-contraction mode.[57]

Researchers interested in the coordination of movement have been interested in specific areas of the CNS that may be responsible for

coordination of postural motor activity with movement motor activity. The supplementary motor area, SMA, has been the proposed area since coordination of several different types of movement is required. Using the Hugon paradigm of comparison of response to active and passive unloading, Viallet, et. al., compared normal subjects with subjects having known lesions of the SMA. Their study suggested:

> "...that the SMA region contralateral to the postural forearm, together with other premotor and motor areas, may select the circuits responsible for the phasic postural adjustments which are necessary to ensure postural maintenance, whereas the motor cortex contralateral to the voluntary movement controls both the movement and, via collaterals, the pre-selected circuits responsible for the associated postural adjustments."[58]

The importance of the foregoing is it firmly establishes that the control area for coordination between postural (or stabilizing) muscles and moving muscles is in an area contained within the motor learning loop. This means that such coordination will be learned, to a certain extent, from prior experience and pre-programmed, or operating under feedforward rules.

The concept of muscle balance

Definition of muscle balance

There does not seem to be any agreed upon definition of muscle balance. Rather, the definition seems to be that muscle balance exists when muscle imbalance does not. This author could not find a single reference that stated a clear definition of muscle balance. From reviewing the literature, the

following definition was gained: Muscle balance exists at a joint or across a series of joints between opposing muscle groups when there is optimal relative balance of their strength capabilities.

Some clarification is necessary to the understanding of the muscle balance or imbalance definition. It consists of two parts. The first is, "at a joint or series of joints between opposing muscle groups". The second is, "optimal relative balance of strength capabilities". The first part is structural and somewhat easier to encapsulate.

The Opposing Muscle Pair (OMP)

Muscles tend to be arranged in opposing pairs; that is, groups of muscles moving a joint in one direction are opposed by muscles moving that joint in the opposite direction. The resulting functional unit could be called an opposing muscle pair, or OMP. A muscle opposes another muscle if its line of action is on the exact opposite side of a joint from another muscle. For example, the quadricep muscles at the knee opposes the hamstring muscles. The quadriceps extend the knee since their line of action is on the anterior side of the joint, while the hamstrings produce flexion (the opposite action) since their line of pull is on the posterior side of the joint. Sometimes the opposing muscles consist of muscles from the same group. For example, the medial hamstring muscles produce internal tibial rotation at the knee while the lateral hamstrings produce the opposing external rotation. To delimit an

OMP, one needs at least two qualifiers: 1) the axis or potential axis around which the muscles move the segment (this axis defines the plane of motion in which movement will occur); and 2) the lines of action of all the muscles that might move the segment around the axis in either direction in that plane.

Most OMPs consist of several muscles moving the joint in opposite directions. There is only one instance in the human body where a single muscle opposes another single muscle: the flexor pollicis longus vs. extensor pollicis longus at the interphalangeal of the thumb. Nevertheless, when considering movement in a single plane, it is common for therapists to think conceptually about synergistic muscles as if they were one muscle.

It is important to point out that in an opposing muscle pair, all the force that opposes a muscle's efforts to move in a direction does not come from the opposing muscle. Some of the opposing muscle force can come from the tissues of the joint and its structures. This is particularly true when the muscle doing the moving has moved the joint close to the end of its range of motion in a particular direction. For example, when a person is in a hip extended position, further movement into hyperextension will be resisted by the passive tension in the hip flexor muscles and by the anterior joint structures of the hip. Therefore, the term muscle balance can be a somewhat loose description, meaning the balance of a muscle that would tend to move a joint in one direction against an opposing muscle force, plus

any joint structures or other tissue that might become tight if the agonistic muscle moves the joint concentrically.

The terms agonist and antagonist are important to the description of muscle balance. If one selects a certain direction of joint movement in a certain plane, the agonist is the muscle that tends to move the joint in that direction. The antagonist is the muscle that tends to cause movement in the opposite direction. If the considered movement clearly is intended to take place in a specific direction, these labels are easy to assign to muscles. If the intent of the muscle pair's action is to hold a specific position, the labelling of muscles as agonist or antagonist is difficult and arbitrary. Considering the direction of movement as either desirable or undesirable sometimes helps in this case. The agonist tends to hold the position in a desired position, while the antagonist, if active, would take the joint in an undesired direction.

Muscles seem to be arranged in OMPs for functional purposes. These functional purposes provide for fast, but controlled, movement, joint protection, joint stabilization, and movement precision. In serving these functions, muscles of the OMP seem to coordinate their actions using two modes, reciprocal inhibition and co-contraction.

In the reciprocal inhibition mode, one muscle group (the agonist or agonistic muscle) moves the segment in one direction, while the other muscle group (the antagonist) relaxes to allow the movement in the agonistic direction. Relaxation of the antagonist muscle is necessary, because when a

person is awake, muscles are usually in a state of slight contraction, called muscle tone. Muscle tone increases in both paired muscle groups when there is an intention to move the part where the muscles reside. The underlying tone in the antagonistic muscle would retard the efforts of the agonist to move the part quickly. The relaxation of the antagonist provides for the ability to produce fast movement when this is necessary.

When operating in the reciprocal inhibition mode, the relaxation of the antagonist is often more relative than absolute. The degree of relaxation of the antagonist seems to depend on the speed with which the agonist moves the part. If an agonist moves the part slowly, the antagonist contracts very little or not at all. If the agonist moves the part very quickly, the antagonist will contract more vigorously. This contraction of the antagonist is seen as protection for the joint, since if the agonist moves the part without some control, the joint structures might be taken to a position of stretch in which they might be injured.

The second mode of muscle coordination between OMP members is co-contraction. In this mode, both opposing muscle groups contract at the same time. There appear to be two purposes of co-contraction. The first purpose is to provide joint stability. An example of co-contraction for joint stabilization is seen at the knee. When athletes prepare to be very active and subject the knee to quick turning movements, they are often seen crouching with knee flexed and trunk slightly inclined forward. This position promotes

muscle contraction in both the knee extensors and knee flexors simultaneously, apparently providing stability.

The second purpose of co-contraction seems to be movement precision. An example of this type of co-contraction is hand muscles when fingers are being used to perform precise manipulative movements. During a movement toward finger extension, for example, the finger flexors contract to control the degree of movement caused by the finger extensor. This permits the fingers to arrive at very precise positions necessary for highly skilled movement. The same kind of coordinated co-contraction is seen between muscles of the eyes.

The CC is the variable part of the muscle. When relaxed, this part of the muscle is very loose and stretchable. During a muscle contraction, this part of the muscle changes its quality of stiffness depending on requirements for force production. The PC is the invariable part of the muscle. In a healthy muscle, it always has a constantly-stiff quality and a constant length. To develop force, the CC contraction first pulls the PC tight. As the discussion of EMD showed, this can be very important in understanding how a muscle behaves.

The OMP consists of four components that balance each other. An OMP could be described as a first muscle consisting of CC1 and PC1 balanced against an opposing muscle consisting of CC2 and PC2. Figure 2.2A below illustrates a theoretic opposing muscle pair. The designations

CC1 and PC1 are arbitrarily assigned to the contractile and passive components of the agonist (the upper muscle). CC2 and PC2 are arbitrarily assigned to the respective components of the lower muscle.

The Opposing Muscle Pair Complex (OMPC)

The concept of opposing muscle pairs can be expanded to include the muscles that provide stabilization of the bone or bones to which the OMP muscles are attached. With the addition of the stabilizing muscles for either pair in the OMP, an opposing muscle pair complex is formed. Each stabilizing muscle has a CC and a PC. The OMPC, therefore, consists of CC1, PC1, CCS1, and PCS1 balanced against CC2, PC2, CCS2, and PCS2. This idea is illustrated by Figure 2.2B below.

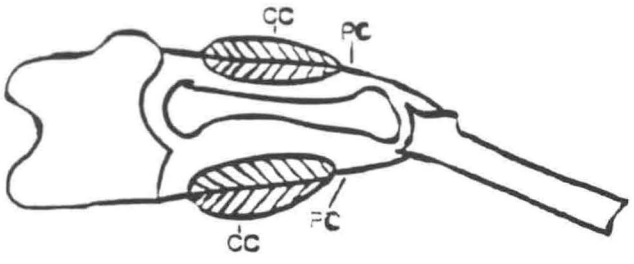
An example is seen at the shoulder where an opposing muscle pair would be the shoulder abductors and adductors. The opposing muscle pair complex, or OPMC, would consist of the adductors with rhomboids as stabilizer opposing the abductors with the trapezius muscle as stabilizer. In this study, the OMP consisted of the hip flexors against the hip extensors. The OMPC consisted of hip flexors with abdominals opposing hip extensors with erector spinae.

The second part of the definition of muscle balance is "optimal balance of relative strength capabilities". The consideration of relative strength capabilities raises the question of the concepts strong and weak

Figure 2.2 Theoretical model of an opposing muscle pair and opposing muscle pair complex.

2.2A. The opposing muscle pair

Agonist

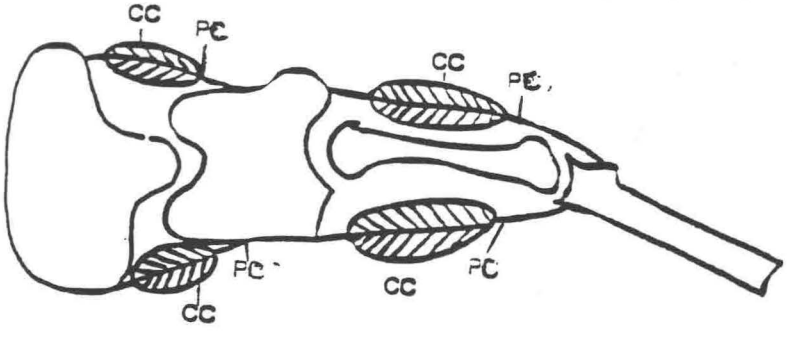


Antagonist

2.2B The opposing muscle pair complex

Stabilizer for the agonist

Agonist



Stabilizer for the antagonist

Antagonist

when used to describe conditions of muscle balance or imbalance. These terms suggest comparisons between a muscle's strength capabilities and a standard that represents normal strength. Sometimes, the standard is relative

to norms developed for individuals of a similar group. Often, the standard is the opposing muscle. The terms strong and weak can also be applied to the passive resistance created in a muscle when stretched. In this study a muscle is strong if its passive tension when stretched a standard quantity is excessive and weak if its passive tension is less than normal when stretched a standard quantity.

Muscle balance and the force generating characteristics of muscles.

Muscle balance or imbalance involves assessing the strength capabilities force of the opposing muscle groups. A review of these capabilities seems basic to the definition of the balance/imbalance concepts.

A muscle's strength is its ability to produce force. For movement to occur, force is usually converted into torque because muscles are attached to bones that act as levers pivoting at joints. A muscle derives its ability to produce force from two different but interdependent sources, active and passive. Active muscle force is produced in the muscle's CC. Here, myofibrils interact to shorten and stiffen the muscle and pull both ends of the contractile portion toward the middle. The effect transforms the CC, gradually or suddenly according to need, from a very yielding, easily stretchable tissue to a very stiff, difficult-to-elongate tissue. This stiffening of the contractile part of the muscle can result in graded amounts of force as needed to move, hold or decelerate opposing forces. However, the CC of the

muscle would not actually produce much force were it not for the passive part of the muscle, the passive component, PC.

The PC consists of two parts, the parallel elastic component, PEC, and the series elastic component, SEC. (The reason for the addition of the term elastic will be apparent in the succeeding discussion.) The PEC consists of most of the fibrous tissue existing within the contractile part of the muscle; i.e., the fibrous wrappings of the muscle fibers, bundles of fibers, fasciculi, and enveloping fascia. It is the fibrous skeleton of the contractile part of the muscle. The SEC consists of the tendons which most muscles have on either end of the CC. The PEC blends into and is continuous with the SEC. The PC is the bridge between the CC and the bones to which the muscle attaches. When a muscle contracts, the CC pulls on the PC which transfers that pull to the bones. Therefore, the CC is dependent on the PC to transfer its force of contraction to the bone.

The PC also is capable of producing force somewhat independently of the contractile part. Passive elongation of the PEC and SEC causes a reactive force to develop throughout the PC similar to pulling an elastic cord taut. Elongation of the passive component is done by taking the joint in the opposite direction of the action of the muscle. This may be done by some external source, such as a therapist, gravity, a machine, or by an internal source such as the active contraction of the opposing muscle. The SEC or tendon has very little elasticity. Most tendons are capable of only five

percent strain until failure.[59] The PEC is capable of much greater elongation before failure so that most of the elasticity of the PC is due to the PEC.

In a muscle contraction, a muscle has two interdependent sources of force. For the muscle to produce force that is externally effective, the CC first must pull the PC tight. This eliminates its elasticity and thereby develops a reactive force in the PC. Both components are contributing some force during the contractions.

In a less than maximal contraction, the motor control centers partial the amount of force contributed by either component in some way. In most movements, the motor centers combine the proper amount of stiffness in the contractile element so that the passive component can produce the force necessary to perform the function. Only in a maximal eccentric contraction (the muscle decelerating the greatest amount of opposing force of which it is capable) can one be reasonably sure it is using both contractile and passive components to their maximum abilities.

The active force generating capabilities of a muscle are measured more indirectly than the casual observer would think. It is usually measured by its ability to produce contact force on a measuring device or visible evidence of overcoming the resistance torque developed by an object of a known weight. The word strength, as it is typically used to refer to muscle, is a relative term. In general, a muscle's strength is judged by its ability to meet certain defined expectations. This might be illustrated by a seated

person to which a weight of 30 pounds is applied at the tibia via a weight boot to the foot. The quadriceps muscles are said to be strong, if, through the tibia, they generate enough reactive force against the weight or resistance arm to lift that weight so the knee is extended. This is true only because the expectation of lifting the 30-pound weight meets the standard definition of strong. Lifting the tibia without the weighted boot does not meet the criteria for strong. Here, as in most instances, the actual force applied by the muscle at its attachment to the tibia is far greater than the measured reactive force. However, seeing the weight move (or be held or decelerated) is far simpler than calculating the actual muscle force. Only in very rare circumstances is the actual direct measurement of human muscle force possible.[60,61] For example, by trigometric calculations, holding a 30-pound weight with the knee at 30 degrees flexion requires approximately 380 pounds of quadriceps force.[62]

When strength of the contractile portion is measured and strength of the passive component is measured at the same time, strength of the PC is implied because no tears develop in the tendon or PEC when the muscle contracts. There are times, however, when it is important to measure the active force capabilities and the passive force capabilities separately. There are separate standards by which to judge the CC's ability to generate active contractile force and PC's ability to generate passive reactive force.

In addition to the methods mentioned above, active and passive force capabilities of muscles are often assessed functionally. This is done done by therapists who have extensive experience in observing a wide variety of persons performing functional movements and postures. If muscles involved in a functional movement or posture have the expected active strength, the functional movement will be performed or posture held without deviation from the expected. Strength deficits are indicated by certain characteristic deviations from the expected normal movement or posture.

The passive force of the muscle also is measured indirectly, and most times not as precisely. Actually what is measured is its passive stiffness which is a combination of elongation and force. A muscle and tendon must be elongated to produce force. There is usually more interest in length testing in the amount of movement toward elongation than the actual force. The reason is that it clinically it is much easier to measure the elongation than to measure the reactive force in the muscle caused by the elongation.

The passive strength of a muscle-tendon complex is measured by the amount of elongation of which it is capable, and the reactive force it produces when at full elongation. This is done by subjecting the muscle to a length test during which the joint or joints the muscle crosses are fixed in some standard position or posture. One joint from these is selected as a target joint to measure. This target joint is moved in a direction of muscle stretch until a posture is maintained by passive reactive muscle force. Any

other joints that the muscle might move are stabilized. The target joint posture is measured or inspected to see if it meets the expectation of a normal posture. Again, the muscle is judged on pre-set expectations of normal. If the muscle allows the expected posture (normal angulated position) at the target joint, the muscle is judged normal. If less than the expected angulation is achieved, the passive component judged to be tight or have excessive strength. If greater than the expected angulaition is permitted, the PC is judged as having excessive length, or is weak. The force is expected to be constant, that is the force necessary to hold the target joint at the point of maximum elongation.

The interdependence of the muscle's active and passive abilities to produce force is important to the eventual understanding of muscle imbalance. As with most organs, muscles follow a conservation of energy principle. The production of active muscle force requires the expenditure of short-term energy stores at higher physiological cost, while the development of passive force does not. Therefore, muscles are equipped with specialized sense organs (muscle spindles and GTOs). One of the functions of these organs is to assess the force produced by the contractile element (muscle spindle) and the passive element (GTO). Since most muscle contractions are sub-maximal, and sub-maximal contractions are always a combination of force by both components, the input from spindles and tendon organs helps the motor control system regulate the contractile element. This regulation

and control is necessary so that the use of active force is minimized and the use of passive force is maximized. From an energy expenditure standpoint, it is assumed that this is the most efficient way a muscle can produce force. Further, it is assumed that the neurological system always seeks to regulate and control the way muscles produce force so that this efficiency is maintained.[21] That the control and regulation mechanisms seek to operate muscles according to this efficiency principle should be evident from the foregoing discussion of electromechanical delay.

The last part of the definition of muscle balance is that relative balance in strength capabilities is maintained. Troup and Chapman studied the balance of muscle forces at the spine. Using isometric and isokinetic contractions, they found the ratio of trunk flexors to extensors was 3:4.[63] This was confirmed by Reid and Costigan.[64] It has been suggested that the ideal ratio of hamstrings to quadriceps strength is 2:3. However, this ratio was found when isometric or slow speed isokinetic contractions were the method of testing.[65] Other authors found an ideal ratio was 1:1 when high speed isokinetic contractions were used as a test.[66]

The results presented above illustrate some of the problems of defining muscle balance other than the non-equity of muscle pairs. First, rather than equity between pairs, muscle balance seems to be some optimal ratio between strength capabilities of two or more opposing muscles. It is

conventional that the stronger of the two muscle groups is used as the reference muscle.

Second, the ratio found is highly dependent on the instrument and methods used to test the muscles. Strength measurements on which a ratio is developed are usually between the contact force a subject can produce against the arm of a certain testing machine while in a certain testing position. The ratio may change with a different machine, a different method or position, and even when the same machine is used. However, the Reid and Costigan study illustrates that in recent studies, the calculation of force capabilities of opposing muscles are being done on more sophisticated bases. Through MRI, they determined the effort arm and cross section of each muscle. This was combined with a machine-obtained force measurement giving a more accurate estimate of the force comparison. Since these measurements are based more on the torque production and cross section muscles rather than contact force alone, they more accurately represent a true comparison.

Finally, a muscle balance ratio may or may not be clinically relevant. Sometimes the ratio is a simple comparison between the force generating capabilities of opposing muscle without a suggestion that this is the optimal ratio. There is no suggestion that if a subject had a different ratio, he/she also would have some clinical condition related to the strength difference. This would be an example of a clinically neutral ratio. At other times, the

ratio has been developed by testing a group of healthy subjects and another group with a clinical condition related to the muscles or joints of the OMP. This ratio is clinically relevant. There is a clear suggestion that the ratio demonstrated by the healthy subjects is the state of muscle balance, while the ratio shown by the subjects with the clinical condition represents the state of imbalance. McNeil, et. al., compared the trunk strength of healthy subjects to patients with low back pain. Subjects with low back pain were found to have a greater flexor to extensor difference than normal subjects.[67]

Definition of muscle imbalance

Muscle imbalance is a term often used in physical therapy to describe abnormal relationships between muscles in the OMP or OMPC. One authority on muscle testing defines muscle imbalance as:

> "Inequality in strength of opposing muscles; a state of muscle imbalance exists when a muscle is weak and its antagonist is strong; leads to faults in alignment and inefficient movement."[68]

This definition, taken in a proper context, is as good as any that have been developed. Dissecting it can lead to a comprehensive definition of muscle imbalance.

The first part of the definition is: "inequality in strength of opposing muscles". From studies that will be presented later, it is obvious that the term inequality should be used in a relative sense. That is, it should not be used in an absolute sense to suggest that if equality of muscle strength is

established, normal balance will seldom be restored. The normal state of balance is inequity. The inequality that makes for imbalance is greater or different than the imbalance demonstrated by healthy subjects for the same age, occupation, body size, etc. Muscle balance is the optimum relative imbalance and muscle imbalance is a less than optimal relative imbalance.

The next part of the definition is: "exists when one muscle is weak and its antagonist is strong". There are four basic parts to an OMP: the agonist's CC and PC and the antagonist's CC and PC. Each of the four parts can be nomal, weak, or have excessive strength (normal, excessive length, and tight, in case of PC). If all four parts are normal, then muscle balance exists. If any one or more of the four parts are not normal, then imbalance exists.

The final part of the definition is: "leads to faults in alignment and inefficient movement". This implies the muscle imbalances of interest to physical therapists would be those that have some association, however loose, with the development of some recognizable clinical condition. The association may take the form of cause, effect, or exacerbation. Some associations between a muscle imbalance and a clinical condition have been studied extensively and the association seems clear. For example, the shoulder muscle endurance of three groups have been compared: non-swimmers, competitive swimmers with painful shoulders, and swimmers (who swim the same stroke) without painful shoulders. The ratio of external

rotation to internal rotation endurance in the non-swimmers was found to be 67.8%. For swimmers with pain the ratio was found to be 42.0%. For swimmers with no pain the ratio was found to be 67.8%.[69,70] Moreover, it was found that when swimmers with shoulder pain performed external rotation endurance exercises to the point that external to internal endurance ratio was greater than 50%, their shoulder pain subsided.[70] Using electromyographic recordings, Wise, et. al., found that the ratio of amplitude between vastus medialis and vastus lateralis was less for subjects with knee pain than subjects without knee pain. It was found that with training, subjects could increase the ratio of activation of vastus medialis relative to lateralis. When this occurred, patello-femoral pain subsided.[71]

Most associations between muscle imbalances and clinical conditions are conjectures based on clinical experience. Imbalance between gastro-soleus plantar flexors and ankle dorsiflexors has been associated with the development of anterior compartment of the leg. It is felt in those persons with plantar flexor tightness, the dorsiflexor muscles are required to work harder than those without plantar flexor tightness.[66] However, the same validation through statistical testing of those with and without the clinical condition, or those before and after treatment that alters the imbalance, has not been done.

The association that this experiment tried to study is an example. It has been suggested that persons with tight hip flexor muscles (actually tight

tissues in general that would limit hip extension) are more susceptible to mechanical low back pain. The usual reason given is that in both standing posture and in walking, the pelvis is pulled into an anteriorly tilted position, since in both activities, the tight tissues reach the end of their length. With the pelvis anteriorly rotated, the trunk should be pulled into forward inclination both by the tight hip flexor muscle and by the fact that the pelvis, on which it sits, is inclined forward. The trunk most often does not incline forward substantially. It is surmised that the trunk stays erect because the thoracic and back extensor muscles contract vigorously to maintain the upright position. Over time, it is reasoned this should cause hypertrophy of the back extensors, fatigue of those same muscles and painful compression of the lumbar facet joints. This muscle imbalance consists of at least two abnormalities in the OMP, tightness of the hip flexors and excessive strength of the back extensors. The condition, often called mechanical low back pain, fits the part of the description stated above, leads to faults in alignment and inefficient movement.

Recognition and treatment of muscle imbalance

The logic of recognition and treatment of muscle imbalance is also based on descriptions as stated above. A part of the definition suggests the first way OMP or OMPC imbalance is discovered clinically; that is, "...leads to faulty alignment." A joint or series of joints takes a posture dependent on

the balance of forces affecting these segments. Physical therapists are trained and subsequently develop great skill in observing and interpreting general and local posture so that imbalance of forces at joints are recognized.

Another part of the definition ("...leads to...inefficient movement") suggests another way imbalance is discovered clinically. Normal movement is usually smooth and segments take predictable paths at predictable velocities. Therapists spend years perfecting observational skills to discern normal from abnormal movement patterns during functional movements. By watching a patient's movement pattern, both when the patient is aware and unaware, a therapist is often able to recognize abnormal movement that indicates OMP or OMPC asymmetry. For example, physical therapists easily recognize OMPC imbalance in the scapular stabilizing muscles, such as serratus anterior weakness, when a patient performs upward reaching. Sometimes, instrumented motion analysis is also helpful in discerning more subtle muscle imbalance problems.

"Inequality in strength of opposing muscles" is also part of the definition of muscle imbalance. CC assymetries may be recognized by means of clinical manual muscle tests. Manual muscle tests are usually adequate to discern only gross CC imbalances. Instrumented testing that graphically displays the comparative muscle abilities are often needed to uncover subtle imbalances.

PC imbalance is often recognized by tests of muscle length. This technique has already been mentioned in the section above entitled, force generating capabilities of muscle. A muscle is judged as having normal length, diminished length, or excessive length. These parallel with the terms normal, excessive strength, and weak. Two subclassifications of the tight or excessive strength category are often used. The first is tight, meaning the muscle restricts joint movement but not severely. This restriction may or may not interfere with function. The second is contracture, which indicates the muscle restricts joint movement to a severe degree. Less often, two subcategories of exessive length may be used. These are: loose, indicating a mildly excessive length that may or may not interfere with joint function, and elongated or ruptured, indicating an excessive length so great that a major deficit in functional movement results.

One problem with both recognition and treatment is the issue of gross vs. subtle imbalance. Gross imbalances are easy to recognize. No instrumentation is required. More of these imbalances are clinically important; that is, they result in marked lack of efficiency and, if treatment is indicated, need more drastic means. Subtle imbalances are harder to recognize, especially against a background of individual variations and asymmetries of body segments. Because it is well known that the body has several compensatory mechanisms that may ameliorate the effects of subtle

imbalances, at least in the short run, it is assumed that less of the subtle imbalances are clinically significant.

Treatment is based on the same logic. Strengthening exercises are prescribed for the weaker of the two muscles if contractile weakness is found. If tightness is found, stretching exercises are prescribed. The treatment for excessive length is less certain than for contractile weakness. Sometimes, orthotics are used and as a result, the excessively long tissues remodel to shorter length. Most times, the orthotic keeps the joint from entering a range that cannot be controlled. Orthotics are used also to gradually stretch tight tissue. Once strength has been increased and length has been gained, training exercises are often used to develop efficient movement. If excessive length persists, patients learn movements or postures which will help to avoid persistance or further development of excessive length. Training exercises also are prescribed so patients learn movement or postures that avoid the persistance or development of excessive strength in one of the muscle pairs.[72,73]

Classification of muscle imbalance

As a prelude to classification of muscle imbalance, it seems important to review the theoretical states in which either muscle component can be. The contractile component can be weak, normal, or strong relative to some standard measure. In common language, the terms normal and strong are

usually synonomous. In the classification of muscle imbalance strong can mean excessive strength, i.e., so much strength that the opposing muscle is overpowered during function.

The passive component is also capable of being weak, normal, or strong, although these terms are not commonly used. Instead, the terms excessive length, normal tautness, and tightness (or contracture) are used. Excessive length is equal to weak, and tightness (or contracture) is synonymous with excessive PC strength.

It seems necessary for some method of classification of muscle imbalances. It should be clear that, according to the classification above, there is only one state of muscle balance at an OMP. That condition exists when the agonist has a CC with normal strength and a PC with normal length balanced against an antagonist with the same qualities. An abbreviation for this arrangement would be agonist(CC normal/PC normal) = antagonist(CC normal/PC normal).

It has already been stated that the ideal ratio of hamstring to quadriceps strength is 2:3. A somewhat loose correlation with susceptibility to injury or decrease in success of rehabilitation has been suggested when there is a large variation in this ratio. If quadriceps are designated the agonist, this imbalance could be classified in several categories depending on the main source of the imbalance. If the quadriceps are excessively strong (such as would be seen in extensor spasticity where a 2:4 ratio might exist),

an agonist(CC excessive strength/PC normal) =/= antagonist(CC normal/PC normal) imbalance could exist. If the quadriceps are weak, an agonist(CC weak/PC normal) =/= antagonist(CC normal/PC normal) imbalance could exist. If the hamstring PC is tight, an agonist CC weak/PC normal) =/= antagonist (CC normal/PC excessive) imbalance could exist. All of these might result in altered postural alignment, inefficient movement, and possibly the development of a clinical condition.

Perhaps no aspect of muscle imbalance makes the concept so overwhelming as the consideration of the number of combinations that are theoretically possible. There are two contractile elements on the agonist side and two on the antagonist. There are two passive elements on each side, one for each muscle. Each contractile element can be too strong, normal, or weak. Each passive element can be excessively tight, normal, or loose. This gives four elements on each side of the equation. Each of the four elements can have one of three outcomes. This gives 3! X 3! X 3! X 3! or 1296 possible combinations of muscle imbalance. (actually, 1295, when the since condition in which all four components are normal is excluded).

Muscle imbalance in the OMP or OMPC

Gross muscle imbalance

In physical therapy, variations in magnitude of imbalance between OMPs are observed. The spectrum of inequity can range from gross to subtle. Gross imbalances are observed in catastrophic states following major

neurological or mechanical injury. Muscles are severely imbalanced when there is spasticity following closed head injury or stroke. Severe muscle imbalance in the upper extremity is illustrated by the well-known "dropped hand," "claw hand," and "papal paw" configurations following peripheral nerve injury to the radial, ulnar, and median nerve, respectively. A few clinical examples of gross muscle imbalance are described and classified in table 2.2, below.

Table 2.2 Clinical examples of gross muscle imbalance.

Agonist		Antagonist		Clinical Condition
CC	PC	CC	PC	
Normal	Normal	Weak (absent)	Normal	Wrist just after radial nerve injury above the elbow.
Strong	Tight	Weak (absent)	Weak (stretched)	Wrist after radial nerve injury with no recovery and untreated.
Excessive	Normal	Weak (inhibited)	Normal	Spastic muscle following CVA or CHI

With gross CC imbalance, at rest the joint segment may take a readily observable posture deviated toward the stronger muscle. This occurs because the resting tonic activity in the stronger muscle tenses the PC of the

stronger muscle pulling the segment toward the strong muscle, whereas there is no tonic activity in the opposing muscle to balance the tonic activity of the strong muscle. Gross contractile imbalance is often easier to observe when movement occurs. The path taken by the moving segment will deviate dramatically in the direction of the action of the stronger muscle. This occurs for a similar reason as resting deviated posture; i.e., the contractile portion of the stronger muscle induces more tension in the PC of the weaker muscle than its CC can control. The result of the grossly distorted posture and movement can be severe alteration from normal joint mechanics. This often, but not always, results in disorders of joint tissue.

Gross imbalances are easy to see clinically so they seem to need no enhancement by instrumentation to recognize. In a few instances, these clinical conditions are studied using EMG, force and motion analysis technology. One aspect that has been discovered is that gross muscle imbalance can metamorphose from one classification to another. The metamorphosis seems related to the neurological regulation and control mechanisms.

> "...many secondary effect of CNS lesions also contribute to the postural behavior seen in patients. These secondary problems are not a direct result of the CNS lesion, but rather develop as a result of the original problem".[74]

An example is seen in children with cerebral palsy. Patients with bilateral spasticity have an imbalance at the ankle that could be described

(with the triceps surae as agonist) as an agonist(CC excessive/PC normal) =/= antagonist(CC normal/PC normal) imbalance. These children often walk with a equinus gait. Over time the achilles tendon may hypertrophy and stiffen so that an agonist(CC excessive/PC excessive) =/= antagonist(CC normal/PC normal) imbalance develops.

Sometimes this condition is treated by lengthening the achilles tendon. If the tendon is lengthened too much, the child may develop an agonist(CC excessive/PC weak) =/= antagonist(CC normal/PC normal) imbalance. This often results in a gait that is described as a crouch gait. It is surmised the child may adopt this walking pattern in search of stability against destabilizing forward acceleration of the tibia at initial contact. In time, the child may loose the ability to plantar flex the ankle. This would be described as an agonist(CC excessive/PC weak) =/= antagonist(CC normal/PC excessive) imbalance.[75]

Subtle muscle imbalance (Postural Dysfunction)

The habitual posture and movement approach

In gross muscle imbalance there is usually a major deficit in the neurological system or musculoskeletal system. The idea that there is a more subtle form of muscle imbalance that can exist in persons with apparently normal neurological and musculoskeletal systems has a long history. The idea that the basis of these subtle muscle imbalances are under conscious

control and the result of habitual postures and movements has an equally long history. These ideas have probably existed as long as parents have told children to "stand up straight". In this country Kendall and Kendall were probably the first to extensively describe these imbalances and correlate certain postures and movements with imbalances.[1] Of muscle imbalance, Kendall states:

> "Muscle imbalance distorts alignment and sets the stage for undue stresses and strains in joints, muscles and ligaments."[68]

He went even further to make correlations of imbalances with clinical conditions. Their observations can be collectively described by the title, habitual posture and movement approach. The main ideas seem to be:

1. The origins of the imbalances are mechanical.
2. The muscle imbalances can metamorphose.
3. If the imbalance or related clinical condition exacerbates, the main reason is the continued use of the habitual posture or movement.
4. Each imbalance has a recognized but somewhat loose connection with a clinical condition.
5. The clinical relevance of the imbalances are variable. In some subjects the apparent imbalance is marked but the person has no related clinical symptoms. In others, the same imbalance seems slight, but the clinical symptoms are marked.

6. If the condition exacerbates or metamorphosizes, the change seems related to the chronic use of certain habitual abnormal postures or movements.

Postulations have been made regarding the imbalance metamorphosis process in response to the mechanical forces of the abnormal posture or habitual movement. The first postulation is called "stretch weakness".

Stretch weakness was proposed by Kendall.[1] It comes from a clinical observation that if a person adopts a posture that habitually places a muscle in a stretched position, that this muscle will show weakness on a clinical test of muscle strength. On the other hand, observation of functional movement does not reveal a suggestion of weakness. The suggestion is that since the muscle is habitually working in a more stretched position, the PC supplies more than the normal portion of the muscle force. The muscle spindle and GTOs sense this, and through their connections cause a decrease in the participation from the CC. Therefore, the muscle weakens. Neumann studied the stretch weakness phenomenon. He suggested that stretch weakness might be an error in measurement. He reasoned that the observed difference could result from the fact that the comparison of strength was made between the affected side and the normal side. The clinical test used is performed at a precise joint angle on both sides. This may place the PC at a more slack position on the affected side. Therefore, the same CC activity is less effective at holding the limb in the test position.

He found that if the affected joint was placed in a position where the PC was slightly more elongated, the test results were the same.[76] In a subsequent study, he found no significant differences in EMG between sides during isometric contractions at comparable points in the ROM.[77]

Another theoretical concept related to a muscle's combination of CC and PC to produce force is the concept of tight weakness. Again, this is a concept developed purely on the basis of clinical observation. In this concept, a habitual posture or movement is adopted which demands less excursion from a PC. The theoretical result is that a muscle becomes less capable of generating normal force when tested in an elongated position compared to the same muscle on the opposite side of the body when tested in the same elongated position.[78] The explanation may be similar to stretch weakness. In stretch weakness, the standard test position puts the affected muscle closer to an actively insufficient position (PC more slack) so that CC force is less effective. In the tight weakess phenomenon, the affected muscle may be closer to a passively insufficient position and also less able to obtain the same participation by the CC.

The observations by Neumann, et. al., and Gossman, et. al., are important to this study. Although not conclusive or understood, there is evidence that with different habitual uses, muscles may change the participation by the CC and PC in producing force. These differences may underlie certain clinical observations and may be important in explaining

muscle imbalance. More importantly, the idea that muscles change their behavior over time when subjected to subtle differences in loading was one of the main ideas tested in this study.

PC tightness and excessive length occur by serial length remodelling. The main constituent of PC is collagen. Collagen remodels slowly according to the stresses applied to it. This is an active process modulated through fibrocytes, the basic cell of collagen. When increasing tension is applied repeatedly to collagenous tissue, fibrocytes become active in producing more collagen and proteoglycan binding substances. This results in gradual lengthening of the tissue in the direction of stress. Relatively light forces applied daily and sustained for as little as 20 minutes per day can produce this elongation.[79]

When the PC is deprived of tensile stress, the fibrocytes become active in removing collagen.The tissue remodels so that it is shorter. This may occur when the collagenous tissue is habitually held in a slackened position. In addition, the tissue may become crimped or pleated. Adhesions may occur between the crimps or pleats of the tissue, further shortening the tissue. There will be a gradual decrease in the tissue's ability to elongate.

Early study of muscle imbalance

Opposing muscle interaction in an OMP has a long history of study. Sherrington was first to discover the neural integrative principle of reciprocal

inhibition.[80] He found that when a muscle contracted, the tonic activity in the opposing muscle would be seen to cease or decrease. If the tonic activity were to persist or increase in the opposing muscle, then the movement in the desired direction would be retarded or stopped. Such an imbalance could be described as agonist(CC normal/PC normal) =/= antagonist(CC excessive strength/PC normal).

For many years reciprocal inhibition was considered to be the only mode of interaction between opposing muscle pairs. Most early studies investigated muscle interaction in the OMP exclusively using the reciprocal inhibition coordination model. Later, with better technology, the co-contraction mode of opposing muscle interaction, co-contraction, was revealed.

It is simpler to study the interaction of muscles operating in a reciprocal inhibition mode because at least one of the muscles is physiologically active and the other is not. This simplifies the assignment of forces to either active or passive sources. The purpose of the co-contraction mode of opposing muscle interaction is for the joint to hold a precise position, move in a precise movement, hold a stable position, or make a movement that is dynamically stable. If one of the paired muscles exerts a stronger pull than it should, the joint might be stabilized in a abnormal position or the movement may not occur with the required precision or stability.

The latter two points emphasize the problem created when one extrapolates behavior in a grossly imbalanced OMP (one or more of the four components severely dysfunctional) to a situation where the OMP is completely intact with subtle imbalances between the components. The problem is that the CC works on the PC to make force, and in doing so, can alter its participation depending on the state of the PC. When there are subtle changes that require adjustment in the muscle, the corrections might be performed in alternative ways. In one situation the adjustment might be done by increasing the participation of the CC, but in the next situation the adjustment might be done by adopting a position that pre-stretches the PC, increasing the participation by the PC and requiring no increase in CC participation. Therefore, to understand the role of muscle imbalance and its possible contribution to the development of symptomology in patients with intact OMPs one needs better information than clinical end results.

The Kendalls validated the faulty habitual posture and movement approach mainly by long-standing clinical observations. To be fair, those persons who have made conjectures based on clinical observations were using the best tools available at the time. Their observations were remarkable and many of their findings based on the logic of mechanics and physiology have been confirmed by newer techniques. Still, there is a need to discover what underlies the development of muscle imbalance. Armed with this information, questions such as why imbalance develops in two

individuals differently under similar mechanical circumstances and what measures may be truly effective at preventing or treating muscle imbalance might be answered.

Despite other possible interpretations for muscle imbalance development and metamorphosis, treatment of muscle imbalance causing postural dysfunction, weak tightness, and stretch weakness has remained the same for many years. This treatment consists of stretching structures affected by weak tightness, strengthening structures affected by stretch weakness and patient education to avoid the faulty habitual postures and movements.[81,82,72]

Proponents of the faulty posture and movement approach have identified two types of abnormal standing postures: kyphosis-lordosis and the sway back posture. These postures describe abnormalities in the sagittal plane. Figure 2.3, below, illustrates these two postures with an illustration of ideal posture. Kendall has observed specific muscle weakness and tightness patterns in the abnormal postures. Her descriptions are as follows:

> "**Kyphosis-Lordosis Posture**
>
> **Short and strong**: Neck extensors, and hip flexors. The low back is strong and may or may not develop shortness.
>
> **Elongated and weak**: Neck flexors, upper back Erector spinae, External oblique. Hamstrings are slightly elongated but may or may not be weak.
>
> **Sway-back Posture**
>
> **Short and strong**: Hamstrings, upper fibers of Internal Oblique, Strong but not short: Low back muscles.
>
> **Elongated and weak**: One joint hip flexors, External oblique, upper back extensors, neck flexors."[68]

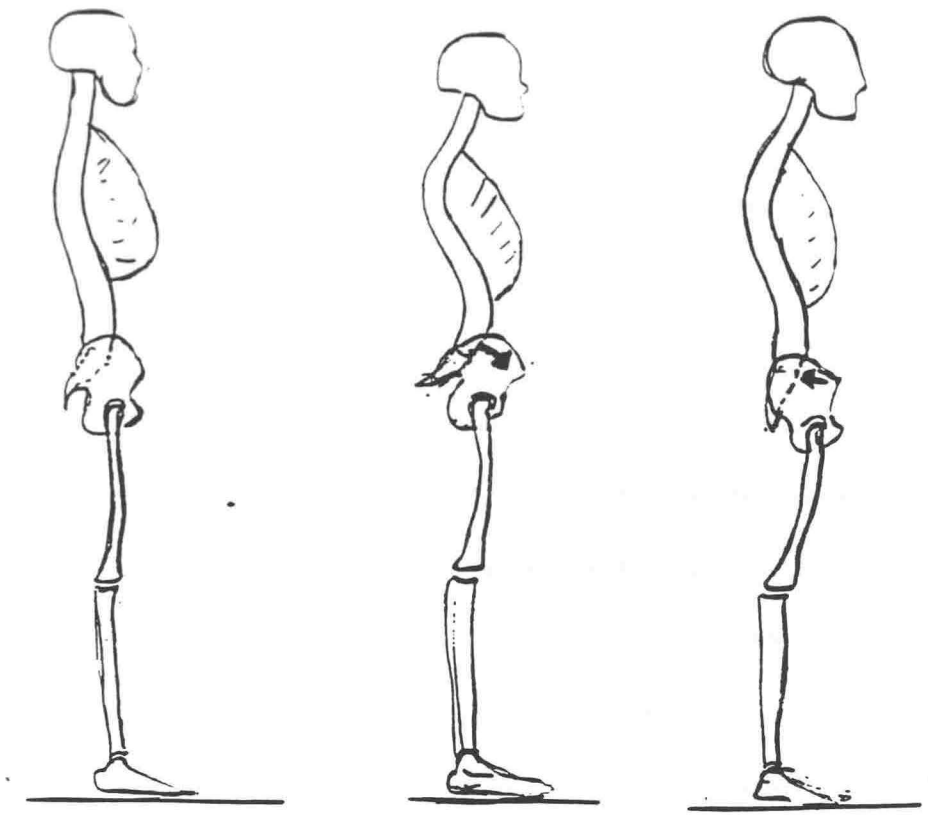
The two abnormal postures were important to this study. The tight hip subjects are assumed to have as one of their characteristics a tendency to use the kyphotic-lordotic posture. The loose hip subjects are assumed to have a tendency to use the sway-back posture.

Proponents of the habitual posture and movement approach would say that the main stimulus for the development of these postures is body mechanics. In standing, the line of gravity in the two postures would be located at different distances from weight-bearing joints. For example, the line of gravity would fall more posterior to the hip joint in the sway back posture, causing a greater tendency for the hip to extend. In the kyphotic posture, the line of gravity falls more toward the anterior hip joint causing a tendency toward hip flexion. The greater lumbar lordosis may be related to the tendency toward hip flexion. If hip flexion does occur, the pelvis tilts forward and the spine tends to fall forward. To remain erect, the person must contract the erector spinae. This may lead to excessive strength in the erector spinae.

Persons with the kyphotic-lordotic posture have tight hip flexors. This presents a second mechanical problem for them during gait. Hip extension of the trailing leg is required to obtain normal step length. If hip flexors are tight, normal step length is difficult. The person with tight hip flexors often will substitute for the lack of hip extension of the trail leg by tilting the pelvis

forward in the terminal stance sub-phase of gait. The tilt of the pelvis allows greater step length, but also tends to throw the trunk forward. These

Figure 2.3 Ideal, Kyphotic-Lordotic and Sway-Back Standing Postures



Ideal | Kyphotic-Lordotic (Crossed pelvic syndrome) | Sway-Back

persons often use the erector spinae muscles to keep the trunk from pitching forward. This is a second use of the erector spinae muscles and would tend to make them stronger.

The person with tight hip flexors has two mechanical reasons for developing excessively strong low back erector spinae. One is related to the

abnormal posture and the other to a faulty movement during gait. However, excessively strong and tight low back extensors may or may not occur. Kendall states in her description of the kyphotic-lordotic muscle imbalance, The low back is strong and may or may not develop shortness.

Functional Movement Pathology

There is another theoretical approach related to the habitual movement and posture approach. This is functional movement pathology, or FMP. FMP theory does not deny that posture and faulty movement habits may underlie the development of muscle imbalances and resultant insidious non-traumatic musculoskeletal pain. However, FMP suggests that factors beside mechanics may underlie muscle imbalance development or progression. Proponents propose that alterations in motor control mechanisms, related to mechanical demands of posture and movement, are an additional factor. In other words, neural mechanisms associated with motor control of the OMP gradually become altered to favor perpetuation of the muscle imbalance.

FMP is a combination of mechanical, physiological, neurological, and psycho-social approaches. It was first advanced by Lewit. A main tenet of FMP is that alterations in central neuromotor control of OMPs may cause chronic painful orthopedic. These alterations are caused by abnormal mechanical events of movement and posture.[3] The usual scenario is that some mechanical events cause one of the opposing muscles to work harder

than it normally would. This harder work causes the motor control system to pre-program neural behavior associated with meeting greater resistance. Physiological changes occur in the muscle that cause a hypertrophy of the CC and PC. This causes one or muscles in an OMPC to become strong and tight in relation to its opposing muscles. The psycho-social element has its influences. Many of the precipitating mechanical conditions are part of habitual postures and movements in cultural behavior. For example, in the North American culture there are a great number of sedentary behaviors. Sitting seems to precipitate mechanical events favoring certain muscle imbalances. Pain comes from joint pathology, sudden unanticipated stretch to the tight muscle or fatigue of the tight muscle that occur when the imbalanced OMPC is used in posture and movement.

FMP theories also propose how weakness or excessive length might imbalance the OMP equation. Weakness of the CC might occur if habitual posture or movement is performed in such a way that there is less than normal mechanical demand on a CC1. If all the other three OMP components (PC1, CC2 and PC2) remain the same, the result might be imbalance. It is also possible for mechanical forces of habitual faulty postures or movements to cause gradual excessive elongation of the one of the OMP muscle's PC. Joint pathology and muscle symptoms may also be the result of this phenomenon. In many cases, FMP theory is an elaboration and exemplification of habitual posture and movement approach observations.

However, instead of relying only on clinical observations for validation, FMP uses the tools of EMG, kinetics, and kinematics. In this way, the participation of all components of the OMP can be surmised and conjectures related to neural control can be made.

The suppositions of FMP proponents are perhaps best illustrated by their analysis of a pathological functional movement known as early upward translation of the shoulder girdle during gleno-humeral abduction. The shoulder abduction, considered here, is called "pure frontal plane abduction" (PFPAb). It has a specific description. The humerus moves from the side of the body in an upward arc. The subject attempts to move in the pure frontal plane (parallel to the frontal plane of the body) toward overhead.

Initiating and sustaining full PFPAb takes fine-tuned coordination between muscles that move the gleno-humeral joint, joints of the shoulder girdle, and the spine. An important part is the muscle activity in the spinal and shoulder girdle muscles (for trunk stabilization and shoulder girdle stabilization) which precede and accompany the onset of the prime movers. Joints move precisely at given times and maintain an exact movement ratio in coordination with other joints. For this to occur without conscious thought a motor program exists which coordinates the onsets of the muscles (deltoid, rotator cuff, sections of the trapezius, and levator scapulae) that move several joints. The program also ensures muscles that would interfere with the movement (rhomboids, levator scapula, and latissimus) are relaxed.

The muscles involved form an opposing muscle pair complex. The deltoid is the agonist (thus deltoid is CC1 and PC1) that abducts the shoulder. It also tends to cause upward translation of the humerus in the glenoid. Its antagonist is the supraspinatus muscle that, by tightening on the top of the humerus, causes a downward translation of the humerus on the glenoid. Thus, the supraspinatus is CC2 and PC2. The stabilizing muscle for the agonist (AgStab CC1 and PC2) is the upper and lower sections of the trapezius. Were it not for gravity, the role of antagonistic stabilizer (AntagStab CC2 and PCS2) might be played by the rhomboid muscles, but such stabilization is not necessary because the trunk is erect position.

In the normal and efficient performance of PFPAb, sections of the trapezius muscle and deltoid work in concert. Trapezius provides dynamic stabilization of the scapula. The increasing resistance torque of the abducting humerus causes an increasing downward rotating force on the scapula. At the onset of PFPAb the trapezius's initial task seems to be to stabilize the shoulder girdle against the downwardly rotating force. Normally, the magnitude of muscle contraction seems to be just adequate to match the resistance torque but not to exceed it. Evidence of this matching force is that no gross movement of the scapula is usually seen until the humerus moves approximately 30 degrees into abduction. Early upward movement of the scapula during PFPAb, therefore, could be defined as scapular movement occuring before the humerus reaches 30 degrees abduction.

A frequent clinical observation during PFPAb is early upward translation of the shoulder girdle. The abnormal movement is easily discerned. The initial movement period as the humerus normally moves to 30 degrees abduction is relatively long. The the shoulder girdle normally moves very little. Any upward shoulder girdle movement in the before-30-degree-abduction point is abnormal.

If there is a painful condition in the subacromial space of the gleno-humeral complex, early upward scapular elevation usually occurs. Under these conditions it seems to have a useful purpose; i.e., to prevent compression of painful subacromial tissues. However, the abnormal movement may persist after the painful shoulder condition resolves. It is also sometimes seen when no painful condition has ever developed in the shoulder.

Hypertrophy of the upper section of the trapezius is a frequent observation associated with early shoulder girdle elevation during PFPAb. This hypertrophic muscle seems to cause a shoulder posture in which the scapula is in slight upward rotation and elevation. Even in relaxed erect sitting or standing when the weight of the upper extremity is pulling downward on the shoulder girdle, the shoulder posture persists. Remarkably, the muscle seems to be disobeying the law of gravity. The posture suggests that the upper trapezius is either contracting or its PC is tight and is unable to elongate to full normal resting length.

Early upward scapular translation, according to FMP theorists, may develop from OMP imbalance due to habitual posture. Many persons with early upward translation also exhibit slight protraction of the shoulder girdle. Slight protraction causes the scapula to rest in a more superior position on the top of the upper thorax. The normal resting position is on the top posterior surface of the thorax. The top surface is less congruent with the scapula's anterior surface. As a result, the scapula tilts slightly forward. In this position the downward pulling force of the upper extremity moves the arm and shoulder girdle forward of the plane of the body. The weight of the arm is no longer supported by the scapular contact with the posterior-superior thorax. The trapezius muscle must now support part of the weight of the upper extremity. This requires a constant tonic contraction of the upper trapezius. Over time, the trapezius gains excessive strength relative to its OMP counterparts, the rhomboids. When required to produce stabilization during early PFPAb, the trapezius overpowers its OMP partner.

Janda has studied subjects performing pure frontal plane shoulder abduction from a seated position. Two groups were contrasted, one whose head-neck-shoulder posture was judged normal and one group whose head-neck-shoulder posture was judged abnormal. Electromyography (EMG) recordings were taken from the right and left upper trapezius and from the right and left lower trapezius. Their onsets were normalized to the onset of the right middle deltoid. Differences between groups in onset sequence of

these muscles were observed. Subjects from the normal group showed onset of the contralateral upper trapezius was just prior to the onset of the deltoid. The ipsilateral upper trapezius onset was simultaneous or after the deltoid. Subjects from the group with abnormal head-neck posture showed that the ipsilateral upper trapezius was usually the first muscle to onset and seemed to have excessive amplitude. Moreover, the entire onset sequence of stabilizing muscles and prime movers was longer in the subjects with abnormal posture compared to a shorter onset sequence in persons with normal posture.[4,83]The important observation here is that there is a difference between normal persons and persons with shoulder muscle imbalance in timing of onset and inappropriate amplitude for the trapezius muscle.

FMP proponents would describe the upper trapezius as being "hyperactive". Labelling a muscle as having hyperactivity seems unfortunate since this term implies different things to different fields of scientific inquiry and clinical practice. FMP theorists seem to use this term to denote a muscle whose onset is earlier than normal and whose early activity is more forceful than seems normal for the usual mechanical demand of the movement.

FMP theory also proposes certain secondary effects of muscle hyperactivity. This is also illustrated by the early upward scapular translation. First, hyperactivity affects the muscle's antagonists. In this case, downward scapular translators and rotators (rhomboid major and minor) are theorized to be "inhibited" causing them to be weak. In time, the early initiation of

shoulder abduction with early upward translation becomes pre-programmed into the motor control system where the motor program for abducting the shoulder is stored. When the dysfunction reaches the stage where the abnormal muscular activity is pre-programmed, shoulder abduction cannot take place without upward translation even after any need for this motor behavior is eliminated.

The posture and habitual movement approach is criticized for considering only one explanation. Postulations of FMP are similarly criticized. There are alternative explanations for the upward posture of the shoulder girdle and early upward translation during PFPab. Serratus anterior tightness is one. Another is anterior gleno-humeral joint tightness. DiVeta, et. al., tested the notion that this agonist-hyperactive/tight-antagonist-weak relationship existed at the shoulder girdle.They questioned whether upward elevation suggesting hyperactive upper trapezius always accompanied weakness of rhomboids.They first measured the postural position of the scapula by the usual clinically accepted bony landmarks. Recognized clinical tests of muscle elongation, that FMP advocates would use to designate muscles as hyperactive or tight, were applied to shoulder girdle protracting muscles. Recognized strength tests were applied to antagonistic scapular retracting muscles. The authors found no correlation between measures of tightness of protracting muscles, strength measurements of scapular retracting muscles, or measurement of scapular postural position.[84]

This study shows that speculation regarding muscle imbalance phenomena need further evaluation. One could say that FMP explanations might have more validity because they are based on measureable representations of movement onset and EMG rather than based on conjectures from clinical observations. These tools might lead to a fuller explanation of muscle imbalance phenomenon.

The addition of EMG, force, and movement indicators does more than help discern the response to mechanical events occurring during posture and movement. It also allows evaluation of the idea that, with the development of muscle imbalance, there is an associated change in the organization of the neural mechanisms controlling the OMP. The muscle tendon unit is elastic. Therefore, the CC must develop sufficient stiffness before it can use the PC to develop force. EMG-detected waves of depolarization must begin in the CC several milliseconds before force or movement begins. For this reason, the onset of the CC must be pre-programmed by the neural control system. The state of pre-movement OMP sensation is assumed be a determinant of this pre-programmed EMG activity. Previous learning also is a determinant. This makes recording of mechanical and muscle EMG in the movement onset period important where both pre-movement sensory assessment and prior learning may be represented by muscle timing and amplitude behaviors. Time to peak activity, time between onset of CC and onset of force or movement,

and sequential order of muscle onsets have been used to study behavior in the pre-movment period.

The postulations by FMP proponents regarding the development of hyperactivity or inhibition in muscles of the imbalanced OMPC are particularly intriguing. It is suggested that with developing muscle imbalance, disordered sequencing, hyperactivity, and inhibition become well established. These behaviors are presumed to be established by motor programming. The behaviors become so well established that they are evident during simple uniplanar movements using only the resistance of an extremity. It is implied that the simple resistance of these uniplanar movements may, besides other mechanical demands, further encourage the development of the muscle imbalance. These ideas are especially enticing because, if true, recording of muscle behaviors during these simple movements could possibly be used to study and understand phenomena related to muscle imbalance development. Simple tests might be developed for diagnosis and validation of treatment methods.

FMP theorists have studied the behavior of OMPs affected by muscle imbalance almost exclusively using a reciprocal inhibition rather than a co-contraction model. Simple uni-planar, unidirectional movements are used. Under these conditions, mechanical events are predictable. The conditions of uniplanar movement allow easier deduction of the muscle behavior of OMP components. In these movements the agonist of the OMP works

concentrically. Muscles are usually grouped into OMPCs for study. Agonist, antagonists and their stabilizers are usually included for observation. Muscles should be coordinated so their actions make movement efficient. Monitoring muscles offering stabilization along with those acting as prime movers allows assessment of another FMP concept. This is known as generalization. Generalization is the idea that the imbalanced OMP seems to have effects wider than the imbalanced pair itself or the joint it moves. Observing the behavior of the related stabilizing muscles offers an opportunity to model how the process of generalization might occur. Muscle action of related stabilizing muscles are usually isometric in simple movements involving the OMP. This simplifies analysis of the relationship between stabilizing muscles and the OMP.

Perhaps no other researcher has devoted more attention to the behavior of the imbalanced OMP and related stabilizing muscles than Janda. He has devoted his attention to the order of sequential muscle onset. He felt that sequential order might yield the greatest amount of information regarding motor planning and control. Emphasis on sequential order also may have been a technical decision. Janda's tools for observing events were raw EMG and accelerometer. Using raw EMG and an accelerometer marking movement onset (but no indication of force onset), he found sequential order of muscle onset to be a reliable discriminator between normal subjects and subjects with muscle imbalance.[85]

From early in his studies, Janda felt that development of muscle imbalance had a motor control factor in addition to mechanical factors. The results of his first study indicate why he would feel this way. He studied five hundred patients who had developed chronic spinal pain early in life. These subjects had been unusually resistant to all the forms of treatment. Using recognized methods of neurological examination, he concluded that 80 percent of this group had slight but distinct signs of minimal brain dysfunction. One characteristic of these patients he found striking.

> "In relation to the general character of movement pattern, patients with minimal brain dysfunction seem to show a greater tendency towards an overflow of muscle activity. This means that they have a decreased ability to perform fine movements and to adjust themselves. Their performance is rather poor with lack of normal variability. Often it is even difficult to estimate which muscle plays the role of the prime mover."[85]

He then had each subject perform three simple uniplanar movements while monitoring the onset of EMG activity in selected muscles. He related muscle onset to movement onset. The movements he used were prone hip extension, side-lying hip abduction, and seated shoulder abduction. He noted consistent onset patterns of muscles that were different from normal subjects. He then tested subjects In prone lying. Muscle latencies were recorded from the thoracic erector spinae, the lumbar erector spinae, the ipsilateral gluteus maximus, and the ipsilateral hamstring. He observed that in normal subjects, gluteus maximus activity was accompanied by contralateral lumbar erector spinae. Ipsilateral erector spinae onset was next

with thoracic extensors being last to onset. In some patients, gluteus maximus was often delayed and the ipsilateral rather than the contralateral erector spinae was seen to onset next. In some patients, the initial muscles to onset were the thoracic extensors. Those who showed first onset of thoracic muscles were usually those with the most severe pain complaints. Order seemed to make a difference.

Subsequently, Janda compared muscle behaviors of chronic non-neurogenic back pain patients with normal subjects. Prone hip extension was the task. The back pain subjects' onset sequences were similar to the subjects with minimal brain dysfunction. Normal subjects seemed to show a different sequential order of onset.[2]

Eventually, Janda studied sequential muscle onset order during several tasks. He observed patients performing hip abduction from a side-lying position. Those with spinal pain seemed to perform a rostral movement an ipsilateral pelvic tilt before movement at the hip. During side-lying hip abduction, the quadratus lumborum should stabilize the pelvis against the downward pulling hip abductors. Early ipsilateral pelvic tilt before hip movement was interpreted as early abnormal activity of the quadratus lumborum. An experiment using EMG was designed to test this notion. EMG recording were taken from the gluteus medius and tensor fascia lata. An electrogoniometer signaled onset of hip abduction. Movement of the ipsilateral pelvis marked the onset of quadratus lumborum activity. Chronic

back pain and normal subjects were contrasted. Normals started the movement by contracting the gluteus medius group first, while chronic back pain patients contracted tensor fascia lata first. Observation from this experiment confirmed findings from the study based on kinematic analysis alone. Normal subjects contracted the abductor muscle and the pelvis did not tilt downward. This indicated the quadratus was probably performing its normal function. Chronic pain subjects often started the movement with an ipsilateral upward pelvic tilt. This indicated an imbalance in the quadratus lumborum vs. hip abductors OMP. On this basis he suggested that quadratus lumborum was hyperactive while the hip abductor was inhibited in the chronic pain subjects.[86]

Janda has used other tasks to study the difference in sequential muscle onset between spinal pain patients and normals. Active neck flexion, prone hip extension, and supine curl-ups have been used. Observations were made by observing the onset of movement using an inclinometer and the sequential onset of muscles using EMG.[87]

There seem to be two themes from Janda's work: First is the hypothesis that there is sequential order of muscle onset associated with normal subjects and other sequential orders associated with subjects having muscle imbalance. Furthermore, there is evidence in the abnormal sequential order shown by subjects with imbalance of hyperactivity and inhibition of specific OMPC muscles. The hyperactive muscle is usually on one side of the

joint and the inhibited muscle on the other. Janda's definition of hyperactivity and inhibition is vague. However, from his writings and presentations it seems fair to define hyperactivity as muscle activity which, compared to normal, occurs earlier in the sequence and is of excessive amplitude. A muscle can be defined as hyperactive if it consistently demonstrates this behavior under the same standard conditions. Inhibition, it is surmised would be the opposite; that is, a muscle which, compared to normal, shows activity starting later in the sequence and which has diminshed amplitude. It is suggested that normal and abnormal activity can be distinguished both during clincal observation tests and using EMG. There seems to be no control for speed but during the clinical tests it is recommended that patients move slowly.

Janda has written and presented extensively about the behavior of muscles about the hip and pelvic region. In this area there are two OMPs operating in the sagittal plane that have been given greatest attention. They are the abdominal muscles vs. the erector spinae muscles and the hip flexor muscles vs. the hip extensor muscles. Janda and others have studied these OMPs using a hip extension movement. The manner in which these OMPs have been considered implies that they can also be considered as an OMPC with the hip extensors as the agonist with erector spinae as the stabilizer for the agonist. Hip flexors can be considered the antagonist with abdominal muscles as the stabilizer for the antagonist. Janda states that an imbalance

can develop in which the hip flexors have excessive tightness in relation to the hip extensors. Eventually, the hip extensor muscles become inhibited while the stabilizing erector spinae become hyperactive and tight. Abdominal muscles also become inhibited. Janda calls this imbalance the "crossed pelvic syndrome".[87] It is important to note that the crossed pelvic syndrome described by Janda is very similar to the description given by Kendall to the hip-pelvic muscle characteristics of the hyperlordotic-hyperkyphotic posture.

Janda suggests that the hyperactivity in the erector spinae develops in a similar way as hyperactivity of upper trapezius causing early upward scapular translation in PFPAb. The hyperactive muscle may work against an abnormal source of resistance causing worsening of the condition. In upward scapular translation the source of resistance is external, that is, a redistribution of the shoulder girdle weight.

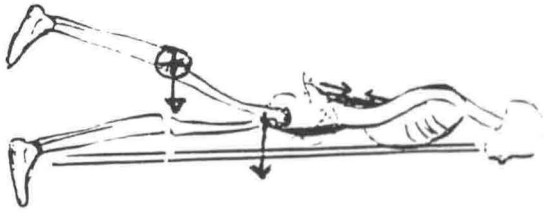
With tight hip flexors there seem to be two sources of resistance that would cause the erector spinae to be hypertrophic and tight. Persons with tight hip flexors often exhibit a posture with a forwardly rotated pelvis. Since the lumbar spine sits on the pelvis a forward rotated pelvis tend to impart a forward inclination to the spine. To keep the trunk erect that person would need to use the back extensor muscles. Since the resistance producing this postural phenomena is the trunk's line of gravity it is considered to result from an external source of resistance. This phenomena is illustrated in figure 2.4A, below.

Figure 2.4 The three possible sources of resistance to erector spinae in person with kyphotic-lordotic (cross-pelvic syndrome) posture.

2.4 A Erect standing

2.4 B. Terminal stance

2.4 A Internal resistance



Another possible source of resistance could make the erector spinae work harder in the person with crossed pelvic syndrome. When a person with tight hip flexor attempts to step forward with one lower extremity the opposite extremity must more toward hip extension. Hip extension of the trail leg is one determinant of step length. If hip extension is insufficient, a person may gain step length by allowing the pelvis to tilt forward achieving a normal step length. This would cause the spine to incline forward requiring the back extensors to work to maintain an erect trunk. This source of resistance for this phenomena is also an external resistance. The resistance of the trunk during terminal stance is illustrated in Figure 2.4B, above.

With the cross pelvic syndrome or hyperlordosis/hyperkyphosis there may be another source of resistance. This source is considered internal. It is the passive force of the tight opposing hip flexor and perhaps other anterior hip structures. If tight, they resist the hip extensors' attempts to extend the hip even when a person is not standing or walking. Figure 2.4C, above, illustrates this internal source of resistance.

Both Janda and Kendall seem to agree that these sources of resistance exist and the first two may cause and perpetuate the muscle imbalance associated with tight hip flexors. Both agree that the internal source is useful in distinguishing tight patients from normal persons. What is different is the way Janda surmises that the internal resistance can be used to distinguish tight from normal persons. This is evident by a clinical test

recommended in confirming the cross-pelvic syndrome (hyperlordotic-hyperkyphotic posture). Like Kendall, he recommends observation of standing posture, terminal stance of gait, and available hip extension during the hip flexor length test to confirm that the cause of the posture is tight anterior hip structures. However, in addition, Janda recommends a prone hip extension test as a confirmatory tests. During the prone hip extension test muscles are observed and palpated for their onset time in relation to each other. It is suggested that during the test a sequence of onset that is normal and other sequences that are abnormal can be determined by visual inspection and/or palpation. The prone test seems to have been developed from EMG studies by Janda mentioned previously.

The normal sequence of activation proposed by Janda for hip extension is gluteus maximus first, contralateral erector spinae, ipsilateral erector spinae and finally hamstring. He proposes that with tight hip flexors, "postural" muscles become hyperactive and "phasic" muscles become inhibited. In this classification, postural muscles are erector spinae and hamstrings. The phasic muscle is gluteus maximus. Thus in the most common pathological condition erector spinae and hamstring tend to be the first muscles or be contracted, while gluteus maximus tends to be last.[4]

Janda's work has been widely quoted in published texts and articles. He has made numerous conference presentations on this topic. Clinical inspection and palpation tests have been based on these observations.[88]

Validation of the use of muscle onset sequence to distinguish normal subjects have eluded other researchers. Pierce and Lee had 20 normal subjects perform 534 trials of an active prone hip extension movement. The movement was performed in a hip-flexed position. Speed was controlled at 30 degrees per second. EMG recordings were taken from the erector spinae, gluteus maximus, and biceps femoris. No consistent pattern of sequential muscle activation was found. However, a high degree of variability of firing order was found. This seemed to give evidence contrary to Janda's claim of a consistent pattern of firing order for normal subjects. Sequential order was so inconsistent it led the investigators to speculate that high variability might distinguish normal subjects from subjects with pathology.[89] They speculated that high variability was the rule rather than the exception for normals. Janda implied a similar notion in discussion of his results of minimal brain dysfunction subjects. He stated that a distinguishing characteristic between normal subjects and minimal brain dysfunction subjects was poor performance with lack of variability. (see quote p. 109 above).

There were two important differences between Janda's studies and the Pierce and Lee study. First is a difference in required speed of movement. Janda makes no mention of how speed of movement was controlled. One can assume from the clinical test recommended, that speed of movement was self-selected but slow. Pierce and Lee gave visual cues that helped subjects to move the limb at the same speed for each trial. The

requirement of moving at a certain speed may introduce a level of skill not required of subjects moving at a speed a self-selected speed. These findings seem to agree with Lee, et. al., in which the latency of onset was more variable when a target speed condition was imposed.[90] Another difference between Janda's studies using the prone hip extension paradigm and the Pierce and Lee study is the test position. In the Janda studies, subjects were tested in the prone hip extended position. In the Pierce and Lee studies, the test position was prone with 30 degrees hip flexion.

It seems clear that Janda believes that the internal resistance of the hip flexors causes a distinct alteration in muscle behavior that can distinguish normal subjects from subjects with tight hip flexors. His comparison of subjects in the prone hip extended position leads to this conclusion. The prone hip extended position would seem to create more tension in the hip flexors for a person with tight hip flexors than a person with normal length. The external resistance in the prone flexed position would be the same for both hip-tight and normal persons.

The Pierce and Lee study seems to give evidence contrary to Janda's postulation but only for the normal group. However, the lift movement in this study was done from a hip flexed position. In the hip flexed position there may be no internal resistance from the hip flexors. If subjects had been tested in a hip extended position perhaps muscle onset sequence would have

been more consistent. Nevertheless, their evidence argues against consistent behavior during a prone hip extension movement.

The idea of different muscle behaviors as shown by EMG based on a difference in muscle balance is intriguing. It would seem that changing persons from a hip extended position to a hip flexed position would change the balance between hip flexors and hip extensors. A pilot study was conducted to see if changing position of subjects could would cause a change in EMG. Five subjects with tight anterior hip flexors were compared with seven subjects with loose anterior hip flexors. Subjects were classified using the same inclinometer measurements as used in this study. However, inclusion criteria were not as stringent. This study showed a main effect for position and two way hip type by movement interaction for erector spinae muscles (See Appendix, tables A1 and A2). The study seemed to show that manipulation of position (changing muscle balance) might cause a systematic change in erector spinae EMG. However, the power for this interaction was not sufficient to truly assure significance. Hip tight and hip loose subjects did not seem distinguished by this study. Therefore, it was decided to conduct a study with more stringent subject classification and more subjects with the idea that in this subsequent study change in position might distinguish not only movement but also persons of different hip types. This would provide support for Janda's idea that internal resistance during a prone hip extension

test might cause changes in EMG for tight hip subjects that distinguish this group from persons with other types of muscle balance at the hip.

CHAPTER 3

PURPOSE

The first purpose of this study was to see if there is any evidence that muscle behavior is influenced by distinct types of long-standing muscle imbalance. If Janda's suppositions are true, persons with one type of muscle balance would exhibit muscle behaviors that are different from persons with another type of muscle balance under the same movement conditions.

Muscle behavior was observed in the pre-movement period. This is assumed to be the time when muscle behavior is most influenced most by neurological pre-programming processes. Therefore, the first question was, do subjects with tight anterior hip structures develop a motor program for prone hip extension that is different from subjects with loose anterior hip structures.

The second purpose was to see that, if there were differences between hip tight and hip loose subjects, was there evidence the differences were due to hyperactivity in the tight group as Janda suggests. If so, is this hyperactivity related to changes in internal resistance? Change in position should change internal resistance but unequally for the tight and loose groups. A different result occuring in response to change of position would support the hypothesis that internal resistance operating during prone hip

extension can distinguish groups especially if the muscles of the tight group appear to have a hyperactive response.

The final purpose was to compare conditions of internal and external resistance. An important question in understanding muscle imbalance seems to be whether the motor control system responds the same or differently to internal and external resistance. Comparisons between conditions in the experiment could be classified as variations of only external resistance, as variations of only internal resistance, or as a varying because of a combination of internal and external resistance. By studying the pattern of results of these comparisons insight could be gained as to the relative influence of these types of resistance.

Significance of the Research

The effect of gross muscle imbalance on posture and movement is clear to see. Persons with this type of imbalance have severe neurological or mechanical injuries. The four elements of an OMP with their related stabilizing muscles become severely imbalanced in predictable ways. It is equally easy to see how use of grossly imbalanced OMPs in functional movements might worsen imbalance and cause pain. The effect of subtle OMP imbalances in persons with no apparent neurologic and gross mechanical injury is unclear. It seems that there may be ways in which the motor control system may alter the participation of the contractile

component and passive component to accommodate imbalances. Despite this, the idea that subtle OMP muscle imbalances can be created, perpetuated, worsen, and eventually cause musculoskeletal pain in apparently normal persons is strongly supported in texts and literature related to therapeutic exercise. Despite this strong support, the phenomena are not well understood. Proponents of functional movement pathology approach have speculated that motor programming may play an influential role in the development and perpetuation of muscle imbalances causing painful conditions.

This study specifically tested the ideas related to the cross pelvic syndrome as proposed by Janda. The idea that abnormal erector spinae activity associated with limited hip extension occurring during standing and walking become so programmed that the abnormal behavior is evident during a more simple movement is intriguing. If this does occur the study of the phenomenon could lead to a better understanding of muscle imbalance. Although these ideas seem logical, research support does not seem particularly strong. There have been studies that provide contradictory evidence. It was hoped that this study could test these ideas more fully and more directly than previous studies.

The search for more objective methods to evaluate muscle imbalance or validate treatment techniques is ongoing. Clinical tests frequently show low inter-rater reliability and often have questionable validity. Using

sufficiently reliable EMG methods, studies of these ideas advanced by Janda might lead to the development of EMG techniques that might be helpful in identifying problems related to muscle imbalance. The effect of current treatment techniques such as stretching and strengthening exercises might be more objectively tested.

CHAPTER 4

METHODOLOGY

Design

The design was a 2 X 2 X 2 repeated measures design. The three independent variable were hip type, position and movement. There were five dependent variable measures of muscle behavior.

Subjects were assigned to levels of the first factor, hip type on the basis of measures of anterior hip tightness. There was a deliberate attempt to make groups as different as possible on classification measures and yet have a large enough sample size for statistical power. A sample of 32 subjects was chosen from a larger group of 254 potential subjects from a population of random healthy subjects. Sixteen of these subjects were assigned to the hip loose group on the basis of high hip flexor length scores and low pelvic inclination change test scores. Sixteen subjects were assigned to the hip tight group on the basis of low hip flexor length scores with confirmatory high pelvic inclination change scores. The mininimum deviation of hip tight subjects from hip loose subjects was 3 standard deviations for all classification measures.

Each subject was subjected to the same four tests. Each test was a combination of the factors position and movement. The two positions were

flexed and extended hip. The two movements were lift and hold of the right lower extremity. The tests were: lifting the leg in extended in hip position, lifting the leg in flexed hip position, holding the leg in extended hip position, and holding the leg in flexed hip position. Combining the hip type factor with the test created eight experimental conditions of the study: 1) tight group lifting in extended (TLiE); 2) loose group lifting in extended (LoLiE); 3) tight group lifting in flexed (TLiF); 4) loose group lifting in flexed (LoLiF); 5) tight group holding in extended (THE); 6) loose group holding in extended (LoHE); 7) tight group holding in flexed (THF); and 8) loose group holding in flexed, LoHF.

Independent variables

Independent Variable # 1. Hip type: Hip tight vs. Hip loose

Before hip tight and hip loose subjects could be tested for distinguishing muscle behaviors, they need to be identified. The clinical tests traditionally used to distinguish these groups are clinically useful but clinician-dependent and have poor inter-tester reliability. There are no established norms for test of anterior hip tightnesss. Therefore, it was necessary to select tests that might distinguish hip tight from hip loose subjects. It was then necessary to apply these tests to a large group of volunteer subjects to develop norms and variability on which to base subject selection criteria. The tests chosen were the hip flexor length test (HFL test)

and a test of change in pelvic inclination from standing to prone. The hip flexor length test has a long history of clinical use but norms and standard deviations have not been developed. The second test, pelvic inclination change, was chosen for specific purpose of identifying those subjects whose anterior hip structures were tight enough to force a pelvic change in inclination. It also had no norms and standard deviations developed. Therefore, preliminary to the first part of the study was to develop norms and standard deviations on both tests. Norms were developed on 40 volunteer subjects. For the entire subject selection process 254 persons eventually were assessed using the two tests.

The HFL test (often called the Thomas test) is a commonly-used test of hip flexor muscle extensibility. Hip extension is measured in supine by dropping the lower extremity from a flexed position toward hip extension while holding the pelvis stable. Clinically, this test is usually measured by sighting the angle formed by the femur and the horizontal surface of the table.[91] The test can be measured with equal or greater objectivity using a goniometer with its axis at the hip with one arm on the lateral thigh and one arm on the lateral pelvis. The estimated error of goniometry in measuring the lower extremity is 4 - 5 degrees.[92,93] The estimated error of measuring the distance of the femur from the table is not known. There are no norms known for this test. The reference for normal is considered slight (approximately 15 degrees) hip hyperextension. If a person can achieve this

position without stretch discomfort in the HFL position a normal result is assigned. Tight and loose results are much less than or much greater than slight hyperextension.

Physical therapists usually measure pelvic inclination in the saggital plane by inspection and palpation of the anterior superior iliac spine (ASIS) and posterior superior iliac spine (PSIS). Most imagine a line connecting these points and reference this line to horizontal. Normal is considered anywhere from 0 to 15 degrees forward tilt.

It was determined that the usual clinical measurement of these two tests lacked precision necessary for classifying subjects. Therefore methods were devised to measure these tests using a the EDI-320 inclinometer (Cybex, Inc. Ronkonkoma, NY). This device has proven to be highly reliable and valid for measuring joint movement particularly of the hip-pelvis-spine area.[94,95] The inclinometer technique used in this study was derived from that used by Adams, et. al.,[96] and later Dolan, et. al.,[97] to measure lumbar curvature. Dolan comments:

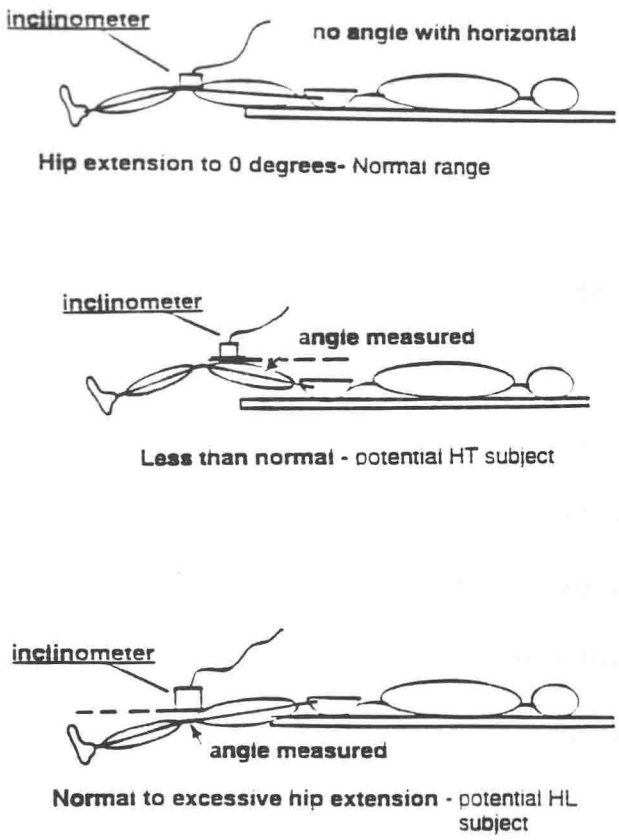
> "The lumbar curvature measured by this technique is not directly comparable with that normally obtained in x-ray studies, since the inclinometer measurement depends on the relative orientation of the dorsal surfaces of the spinous processes, whereas the lumbar curvature obtained from x-ray studies depends on the relative orientation of the vertebral bodies. However, when compared with x-ray studies the inclinometers were shown to measure the same angular movements of the vertebrae without systematic error."[97]

In the first subject classification measurement, the EDI-320 was modified to measure the supine hip flexor length test.The EDI-320 has a bar that connects both feet. When this bar is connected to both feet and the bar is applied to a horizontally level surface, the angle measured with reference to horizontal is 0. If one end of the bar is fixed to the surface when the opposite end is lifted, the device measures the angle of inclination with reference to horizontal. The EDI-320 was modified by attaching a bubble level device to its handle. For HFL tests, the bubble device was calibrated so that bubble registered level when the bar was horizontal with earth. The inclinometer could then be used in a somewhat inverse way. The inclinometer could be placed on a sloped surface. In this position the bubble of the level device would be offset toward the high side. The end of the bar closest to the bubble would be fixed to the sloped surface. The device could then be tilted until the bubble level indicated the inclinometer bar was level. The tilt of the inclinometer necessary to level the device was thereby measured.

Using this method, the angulation with horizontal of the anterior thigh was used to measure the supine HFL test. This was possible because the anterior thigh is a nearly horizontal surface which can take any angulation in space during the HFL test.

Figure 4.1 illustrates how the HFL tests were measured. The subject lay supine. The hip measured was always the right and the stabilized leg was always the left. The experimenter first helped the subject achieve the starting position. By palpation and

Figure 4.1 Method of measuring hip flexor length test (HLF- Thomas test.



sighting the subject's pelvis was positioned so that an imaginary line drawn through the ASIS and PSIS was perpendicular to the table surface. The subject self-stabilized in this position by holding the left

lower extremity in hip and knee flexion with both hands. It was important to stabilize the pelvis because tilt of the pelvis might have caused an error in measurement.

While the subject held this standard position, the experimenter passively extended the subject's right hip with knee straight. Passive movement was very important to maintain because any contraction of hip flexors might cause forward pelvic tilt and an error of measurement. Passive hip extension was stopped when the subject's pelvis started to roll forward. This indicating the end of length of anterior hip structures. Using pillows, the thigh was stabilized in this position. After stabilizing the thigh, the inclinometer device with the bar attached was applied to the thigh. A bubble level device showed no offset, offset toward the knee, or offset toward the hip. If the bubble showed no offset the inclinometer bar was level with horizontal and the hip hyperextension achieved was 0. If the level bubble was offset toward the hip it indicated the knee was lower than the hip, meaning the hip was in hyperextension. The experimenter pressed the start record button to start the measurement. The end nearest the knee was lifted until the level showed the bubble to be in the middle. The stop-and-record-measurement button was pressed. The angle displayed was the degrees of hip hyperextension.

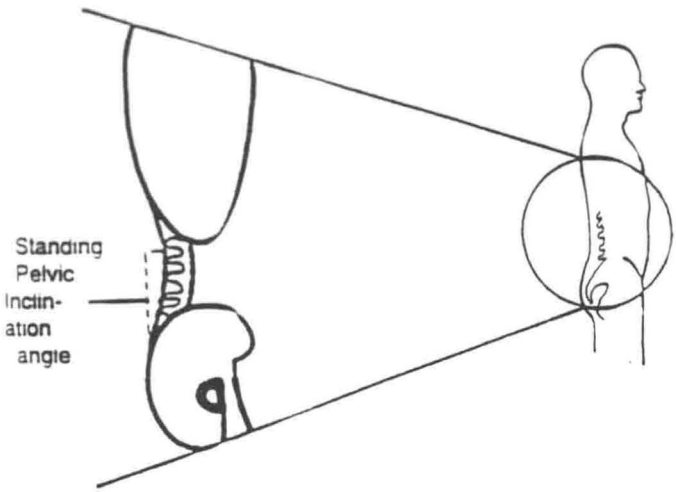
If the bubble was offset toward the knee it indicated the knee was higher than the hip and the hip was still in a position of flexion. In this case, measurement was taken by lifting the end of the bar nearest the hip after pressing the start-measurement button. The result was a minus score indicating the degrees less than 0 the hip could be extended.

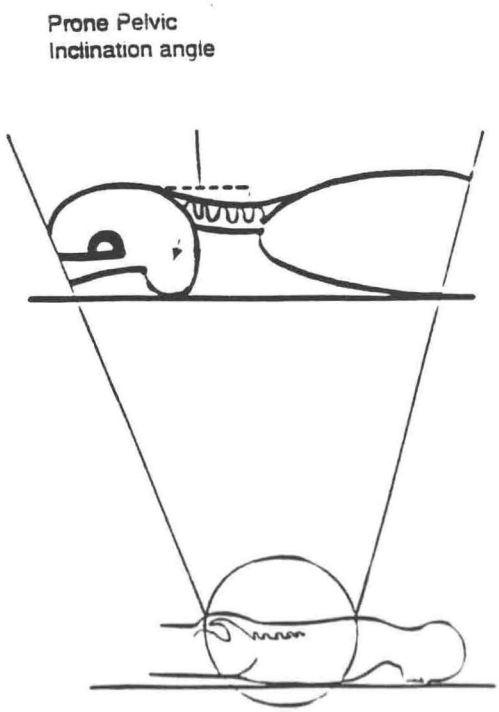
The second test to distinguish tight hip from loose hip subjects was the difference between a measure of the pelvic inclination with the subject standing and a measure of the pelvic inclination with the subject in prone lying. Figure 4.2, below, illustrates the angles measured to obtain this value.

The subject then lay prone with feet just off the table. The same procedures were used to measure the angulation of the pelvis. The subject was asked to stop breathing in the exhaled position. The inclinometer placement and procedures were exactly the same, except the stop-and-record button was pressed when the bubble level indicated the inclinometer is being held parallel with horizontal. The standing score was then subtracted from the prone lying inclination score to obtain the change in pelvic inclination test score.

Prior to this experiment a pilot study of 40 volunteer subjects was conducted. Subjects were tested using the HFL test and pelvic inclination change test. Both tests were applied to each subject on successive days. The objective was to establish the norms and reliability for the tests.

Figure 4.2 Angles from which pelvic inclination difference measurement were taken.





Another objective was to find subjects for the main study who might have met the criteria for inclusion in either the hip tight group or hip loose group. (Only one subject actually qualified).

The results are shown in table 4.1. A significant negative correlation, (-.818), was found between change in pelvic inclination from standing to prone lying and hip flexor length test result. This indicates an inverse relationship between the HFL test result and the pelvic inclination change value. The lower the HFL test score, the higher the change in pelvic inclination score.

The correlation might have been higher except for three possible problems. The reason the pelvic inclination change test may work is that persons with tight hip flexors tend to stand in a compensatory position. If they stand in an uncompensated position for any length of time results in discomfort from their lumbar facet joints. In the compensated standing the pelvic is inclination less. When lying prone the pelvis of the tight hip subject is forced to roll forward by the tight structures increasing the pelvic tilt. Problems arise in that some tight hip subjects do not develop pain sensitive facet joint and tend to stand in the uncompensated position. There is no change in pelvic inclination for these subjects when going from standing to prone. Another problem subject is the obese subject. Even if this subject has tight hip flexors the abdomen acts as a pillow and, by flexing the hips,

releases tension on the hip flexors. These subjects also might not change from standing to prone.

Table 4.1 Hip flexor length and change in forward pelvic inclination from standing and prone lying in 40 subjects.

INCLINOMETER STUDY #2															
S #	AGE	WGT	Date 1	Stand Inclin.	Lying Incln.	HFLT	Inclin. Change	Date 2	Stand Inclin.	Lying Incln.	HFLT	Inclin. Change	Ave Chg.	Ave. HF
1	48	162	12/8/94	13	15	18	2	12/9/94	12	16	12	4	3	15
2	22	117	12/8/94	16	19	16	3	12/9/94	21	24	15	3	3	15.5
3	31	181	12/8/94	16	21	21	5	12/16/94	16	17	16	1	3	18.5
4	27	127	12/8/94	11	13	19	2	12/14/94	16	17	15	1	1.5	17
5	43	105	12/9/94	13	14	23	1	12/23/94	18	17	16	-1	0	19.5
6	29	141	12/9/94	14	17	24	3	12/15/94	23	25	1	2	2.5	12.5
7	45	210	12/9/94	31	36	1	5	12/16/94	27	33	-1	6	5.5	0
8	29	138	12/9/94	18	23	10	5	12/10/94	22	24	15	2	3.5	12.5
9	30	155	12/9/94	13	20	7	7	12/14/94	15	23	10	8	7.5	3.5
10	40	129	12/12/94	15	21	8	6	12/15/94	14	21	12	7	6.5	10
11	35	123	12/14/94	24	25	18	1	12/16/94	18	25	13	7	4	15.5
12	37	193	12/14/94	16	25	7	9	12/19/94	12	27	1	15	12	4
13	24	151	12/15/94	10	27	2	17	12/16/94	10	27	-2	17	17	0
14	26	158	12/16/94	19	21	10	2	12/17/94	21	21	12	0	1	11
15	25	108	12/16/94	20	26	10	6	12/7/94	23	26	23	3	4.5	16.5
16	39	177	12/16/94	16	25	9	9	12/21/94	14	20	12	6	7.5	10.5
17	32	145	12/16/94	19	21	18	2	12/17/94	17	21	12	4	3	15
18	29	237	12/16/94	9	22	8	13	12/21/94	12	19	5	7	10	6.5
19	23	164	12/16/94	12	15	14	3	12/21/94	21	24	13	3	3	13.5
20	30	118	12/16/94	10	19	5	9	12/23/94	12	19	7	7	8	6
21	40	152	12/16/94	13	15	17	2	12/20/94	12	12	21	0	1	19
22	36	131	12/19/94	5	20	-1	15	12/20/94	7	19	3	12	13.5	1
23	29	136	12/1/94	10	18	9	8	12/20/94	10	17	16	7	7.5	12.5
24	40	115	12/20/94	13	11	16	-2	12/22/94	14	13	15	-1	-1.5	15.5
25	45	136	12/21/94	14	12	10	-2	12/27/94	19	14	16	-5	-3.5	13
26	32	119	12/22/94	11	17	10	6	12/23/94	12	18	2	6	6	6
27	27	140	12/23/94	15	22	3	7	12/23/94	10	21	4	11	9	3.5
28	24	108	12/23/94	15	13	27	-2	12/27/94	11	10	20	-1	-1.5	23.5
29	45	128	12/23/94	10	20	3	10	12/27/94	12	26	1	14	12	2
30	25	122	12/26/94	19	22	11	3	12/29/94	8	14	8	6	4.5	9.5
31	41	153	12/27/94	13	17	8	4	12/27/94	15	20	6	5	4.5	7
32	39	138	12/27/94	18	21	13	3	12/27/94	16	19	18	3	3	15.5
33	26	165	12/27/94	18	19	17	1	12/28/94	12	14	16	2	1.5	16.5
34	25	109	12/27/94	16	23	8	7	12/28/94	14	22	6	8	7.5	7
35	28	221	12/27/94	19	23	19	4	12/28/94	17	19	18	2	3	18.5
36	42	146	12/27/94	22	32	-6	10	12/28/94	22	29	-4	7	8.5	-5
37	23	154	12/27/94	18	35	-12	17	12/28/94	18	33	-3	15	16	-10
38	22	121	12/28/94	19	24	9	5	12/29/94	21	25	10	4	4.5	9.5
39	22	105	12/28/94	5	7	15	2	12/29/94	10	17	12	7	4.5	13.5
40	23	119	12/28/94	20	23	11	3	12/29/94	18	26	9	8	5.5	10
	31.95	143.7	Mean	15.20	20.48	10.88	5.28	Mean	15.55	20.85	9.90	5.30	5.29	10.39
	7.85	31.56	Stdev	4.98	6.00	7.98	4.66	Stdev	4.72	5.38	7.32	4.83	4.53	7.10
							Test / Retest=		0.651	0.779	0.720	0.822		
								Correlation: HFL test with Change in inclination =						-0.818

A third problem affecting the correlation between the HFL test and inclination change test relates to the loose hip subjects. In normal and hip loose subjects, there is no posterior structure causing posterior roll to the same extent as tight anterior structures cause anterior roll in tight-hip subjects.

The normal shape of the anterior pelvis is such that the symphysis pubis is more prominent anteriorly than the ASISs. This causes the pelvis to have a natural tendency to roll forward simply because of gravity. Therefore, the loose hip subject's HFL test usually correlates with a slight (often anterior) change in pelvic inclination from standing to prone. The negative correlation would probably be higher if the loose hip subject's pelvis was forced toward posterior rotation as much as the tight hip subject's pelvis is pulled into forward rotation.

Intra-rater reliability for the HFL test was .720, and for inclination change was .822 (see table4.1). These correlations together with the -.818 correlation between HFL test and change in pelvic inclination test gave the confidence to use these two measures as criteria for classifying subjects into tight hip and loose hip groups. Means and standard deviations from 40-subject pilot study were used as criteria for accepting subjects and assigning to either group.

Originally, it was decided to find subjects two standard deviations (SD) below the mean on HFL test and two SD above the mean on the pelvic

inclination change scores for the tight group. The reverse would be used as criteria for the hip loose group. The pilot study showed relatively high variability of scores and it was felt that these criteria would ensure that the groups were truly different. However, using the 2 SD criteria proved too prohibitive for finding sufficient subjects. Therefore, the criteria were reduced to + or - 1.5 SD on each measure.

Each subject was classified using two criteria, HFL test result and pelvic inclination change test. In the 40-subject norm study, the mean hip flexion length test angle was 10.39 (+/- 7.10) degrees hyperextension. Therefore, a candidate was considered for the tight (T) group if her/his HFL test was less than -0.26; i.e., (10.39 - (1.5 * 7.10)). A candidate was considered for the loose (Lo) group if the HFL test was greater than 21.04 degrees; i.e., (10.39 + (1.5 * 7.10)).

The mean change of pelvic inclination from standing to prone lying was 5.29 (+/- 4.53) degrees. The upper critical value for pelvic inclination change at +1.5 sd was 12.09 degrees, i.e., (5.29 + (1.5*4.53)) . Any potential subject whose measurement was above the 12.09 degree level was considered for the T group. The lower critical value on the pelvic inclination change test was -1.51 degrees, i.e., (5.29 - (1.5*4.53)). However, this value was not used to classify subjects into the hip loose group. It was mentioned earlier that hip loose subjects have no force to roll the pelvis posteriorly and that gravity may roll the pelvis forward for a few

degrees. There the pelvic inclination change test criteria for the Lo group was no greater than 2 degrees.

To be classified in a group, a subject had to meet both group criteria. A summary of the classification criteria is contained in table 4.2, below.

Table 4.2 Summary of subject classification criteria.

	Hip Tight		Hip Loose
1.	Pelvic inclination change score > 12.09 degrees	1.	Pelvic inclination change . no > 2 degrees
2.	HFL test measurement < -0.26 degrees	2.	HFL test measurement > 21.04 degrees

Independent Variable # 2. Test Positions: Extended vs. Flexed

The two positions were prone with hips extended (E) and prone with hips flexed (F). In both positions the subjects lower extremities were horizontal. The flexed position was achieved by lowering the trunk. The horizontal position for the lower extremities was important. This equalized the resistance torque for each trial. If the flexed position had been achieved by dropping the legs and keeping the trunk horizontal there would have been a difference in resistance torque between conditions.

There was expected to be an interaction between position and hip type that varied internal resistance. In the extended position the tight group was expected to have greater tension in the anterior hip structures (hip

flexors) than the loose group thus greater internal resistance. In the flexed position the two groups were expected to have about the same internal resistance.

For the minor hypothesis position was classified as an internal source of resistance. As with hip type, the source of difference in resistance is tension of the anterior hip structures.

Independent Variable #3. Test Movement: Lift vs. Hold

For half the trials, subjects performed a concentric lift at a controlled speed. These were called the lift (Li) trials. A metal rod was positioned above the posterior thigh of the subject so that the subject was required to complete an arc of 30 degrees to touch the rod with the leg. The metronome was set and counted at a steady rate of 90 beats per minute. The subject was required complete the movement within six counts of the metronome, i.e., within 5 counts. Therefore the approximate movement speed was 9 degrees per second. The subject was permitted to observe the movement by use of a large mirror positioned so that the subject could see the distance between thigh and rod. Subjects were permitted to practice until movement was smooth and matched to the metronome.

During the other half of the trials subjects performed a eccentric/isometric hip extension movement. The rod was brought down to rest on the back of the thigh in the starting position. At 3, 5, or 7 seconds

following an audible tone at 3, 5, or 7 the platform holding the right leg dropped. The platform dropped from beneath the leg at approximately 5 degrees per second. The subject's task was to keep the posterior thigh touching the rod. These trials were called the hold (H) trials. The subject did not know when, after the tone, the platform would drop. The mirror was turned so the subject could not see the rod, the experimenter or the movements.

Support dropped from beneath the leg while the subject was relaxed. This caused the subject to respond with a brief eccentric contraction followed by an isometric contraction.

This variable was included for two reasons. First was to observe whether the effect of internal resistance was the same for the two types of movement. Functional movements involving the hip-pelvis-lumbar muscles involve both concentric and eccentric types of contraction. All previous studies had used only a voluntary lift paradigm.[98,99,89] Pilot work showed there may be muscle behaviors that are different for the types of contraction.

Pilot work showed that the drop of the support platform made resistance come on faster, increasing the required speed of contraction. This lead to the classification of movement as a variation in external resistance. The gravity weight of the limb was the same for both movements. However,

the acceleration of that same weight was an external source of variation in this resistance.

The Independent Variables Combined (Experimental Conditions)

Figure 4.3 below illustrates the four conditions under which the subjects were tested. Each condition is a different combination of test position, (E or F hip) and test movement (Li or H leg). The left column depicts the Loose (Lo) group undergoing four test conditions: 1) loose group lifting in extended position (LoLiE); 2) loose group lifting in the flexed position (LoLiF); 3) loose group holding in the extended position (LoHE); and 4) loose group holding in the flexed position (LoHF). The right four diagrams illustrates the hip tight (T) group undergoing the same four conditions: 1) tight group lifting in extension (TLiE); 2) tight group lifting in flexion (TLiF); 3) tight group holding in extension (THE); and 4) tight group holding in flexed position (THF).

The Dependent Variables

Dependent variables were five measures of muscle behaviors taken for each experimental condition. These were: 1) duration of muscle onset sequence (DMOS); 2) left erector spinae latency (LES latency); 3) right erector spinae latency (RES latency); 4) left erector spinae early amplitude

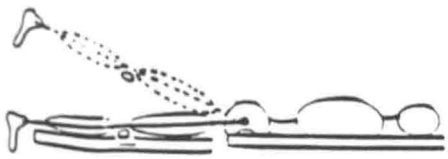
Figure 4.3 Experimental Conditions

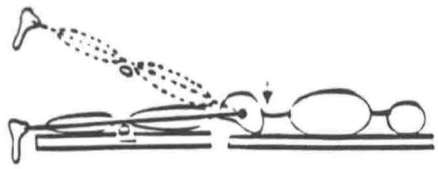
Figure 4.3A Lift

Loose Hip (Lo)

Tight Hip (T)

Hip extended test position

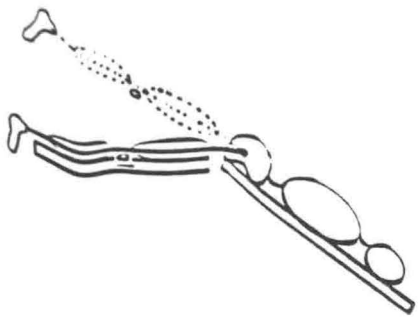


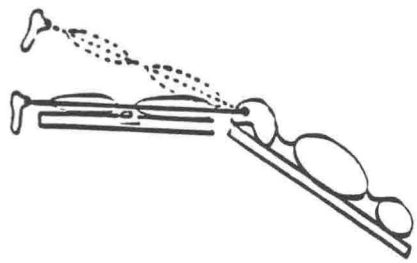


LoLiE

TLiE

Hip flexed test position





LoLiF

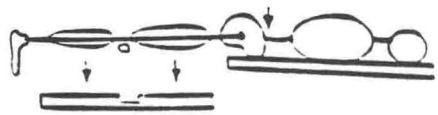
TLiF

4.3B.Hold

Loose Hip (Lo)

Tight Hip (T)

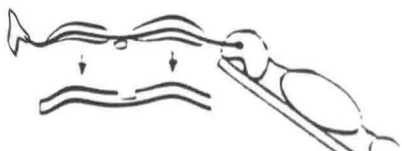
Hip extended test position

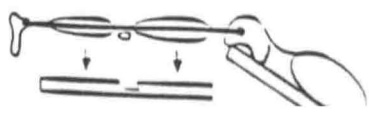


Hip flexed test position

LoHE

THE





change at or just before muscle onset (LES EAC); and 5) right erector spinae early amplitude change at or just before muscle onset (RES EAC).

Dependent variables were meant to examine what muscle behaviors might show about how they prepare to meet greater resistance. Three possible types of preparatory behavior were chosen. First, several synergistic muscles might time their contractions to be more concurrent. The dependent variable associated with this behavior was called duration of muscle onset sequence (DMOS). It was surmised that with greater resistance, DMOS might be shorter indicating muscles were contracting more concurrently.

Second, in preparation for greater resistance, a muscle might begin its contraction earlier in relation to the movement. This might make its peak force more coincident with start of movement. The dependent variable associated with this behavior was latency.

Third, a muscle preparing to meet greater resistance might increase the number of active motor units before and during the start of movement. The dependent variable associated with this behavior was early amplitude change, i.e., amplitude change just prior to and at movement onset (EAC).

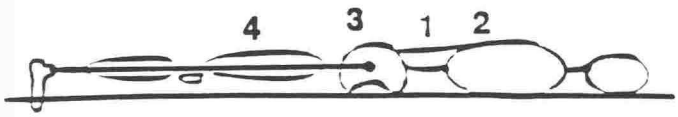
These dependent variable behaviors were derived from EMG data. The data was taken from the immediate pre-movement period and at movement onset. DMOS was chosen because it had been used in a previous study that distinguished groups with different muscle balance at the hip. Latency and

early amplitude change were chosen because it was surmised that they would be most related to Janda's postulations of hyperactivity and inhibition.

Dependent variable #1: Duration of Muscle Onset Sequence (DMOS)

Four muscles of the OMPC related to hip extension were selected for recording. Two are prime movers and two are stabilizers. Muscles were chosen because each is likely to be a participant in the motor program for prone hip extension. These are: 1) left erector spinae (LES); 2) right erector spinae (RES); 3) right gluteus maximus (RGM), and 4) right hamstrings (RHM).[85,89,98] Their expected role in the OMPC for hip extension is listed in figure 4.4 below.

Figure 4.4 Expected role of muscles in OMPC for lifting or holding the right hip in hip extension.



1. **LES** - Stabilizer of pelvis
2. **RES** - Stabilizer of pelvis
3. **RGM** - Prime mover
4. **RHM** - Prime mover

The onset times of these four muscles relative to the onset of movement were ranked. The first muscle to onset were ranked OM1, the second OM2, the third OM3 and the fourth, OM4. The onset time OM1 was subtracted from the onset time of OM4 to obtain DMOS. DMOS (referred to as OM4-OM1) for the same muscles was a dependent variable in a recent study by Bullock-Saxton. In that study, groups with different muscle balance at the hip were distinguished using this variable. The difference between the two groups seemed to indicate a general difference in pre-programmed muscle behavior.[98]

Figure 4.5 Sample DMOS for the tight group.

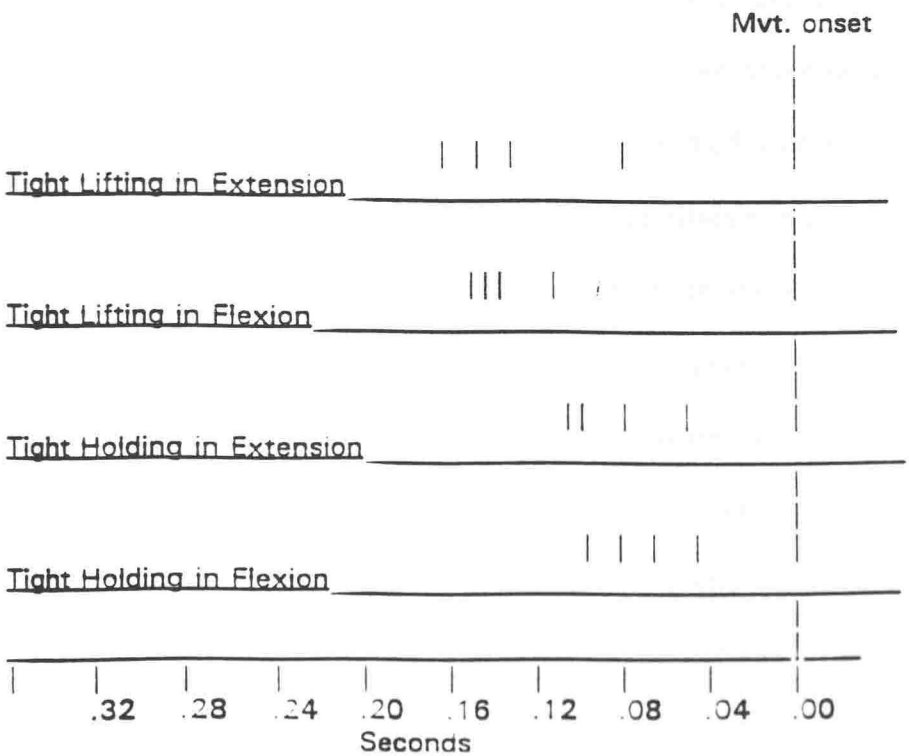


Figure 4.5, above, illustrates the DMOS variable. This figure was derived from the DMOS values for the tight subjects in this study. The short vertical markers show the onset time of the different muscles. The horizontal lines represent the pre-movement time. The common vertical line indicates the onset of movement. The top two conditions show a difference in DMOS. In the tight lifting in flexion condition the onset markers are closer together. Muscle onsets were more synchronous, i.e., there was a shorter DMOS. In the tight lifting in extension condition the muscle onset markers are more spread out, i.e., there was a longer DMOS.

Dependent Variable #2. Latency of Erector Spinae Muscles

Latency was defined as the time between the onset of a muscle and the onset of movement. The latency of erector spinae muscles were chosen for study to test the hypothesis regarding the effect of muscle imbalance on the stabilizing muscles. A strongly-supported hypothesis is that tightness in the hip flexors will ultimately lead to strengthening in the lumbar extensor muscles. Treatments for muscle imbalance at the hip-pelvis-lumbar spine complex have been devised on this basis. Janda states that with anterior hip tightness, postural muscles become hyperactive. The erector spinae muscles are included in the list of postural muscles that become tight. Although Janda does not specify the characteristics of hyperactivity, it seems logical

to observe latency to see if this change in timing is part of change in muscle behaviors.

EMD studies have shown that muscles that are working harder (as in overcoming greater resistance) often show a shorter latency. Since it was assumed that tight hip subjects have greater resistance to hip extension (the test movement in this study), it also was assumed that the stabilizing muscles for hip extension might show a shorter latency.

Dependent Variable #3: Early amplitude change in the Erector Spinae

One aim of this study was to test a hypothesis regarding exacerbation of muscle imbalance in the neurologically intact individuals. The hypothesis that a sign of exacerbation of the muscle imbalance that includes anterior hip joint tightness is that back extensor muscles hypertrophy and strengthen. Strengthening of back extensors might be a response to having to provide greater stabilization so that hip extensor muscles can more effectively move against the increased resistance offered by the tight hip flexors. If back extensors strengthen in persons with tight hip flexors and not in those without there might be amplitude differences when EMG records from these two groups are compared. Furthermore, if these differences are observed during pre-movement and movement initiation, it would be evidence that the amplitude differences are pre-programmed.

Janda's states that with anterior hip tightness, back extensor muscles become hyperactive. His definition of hyperactivity seems clearly to rest on two observations. First, is the observation that during clinical inspection tests back extensors of tight hip subjects are often more hypertrophied. Second, is that EMG recording and clinical observation/palpation recommended by Janda demonstrate earlier activity that seems of greater amplitude than normal.

Moritani and Devries have observed that with training approximately 5% of the increase in strength capabilities of muscles can be ascribed to neural changes. These neural changes are reflected by a greater intergrated EMG amplitude during a maximum voluntary contraction.[100]

It is generally accepted that when speed and type of contraction are the same, greater resistance will result in a greater amplitude of the intergrated EMG record.[101] It is surmised that a person with tight hip flexors in the prone hip-extended position (Janda's test position for EMG studies and recommended clinical tests) will have greater resistance to hip extension than a person without tight hip extensors. Back extensors would be expected to be required to supply greater stabilization so that the pelvis is stabilized. Therefore, it is reasonable to expect that back extensors might show greater amplitude in persons with tight anterior hip structures during prone hip extension. It is possible that Janda's observation of hyperactivity in hip tight subjects could have been a result from a combination increased

neural effects due to training and the more immediate effects of increased resistance caused by the position. To explore this idea, manipulation of position was included in this study.

There are reasons to expect that persons with tight hip flexors might not show a greater EMG amplitude when performing same type and speed prone hip extension. Trained muscles hypertrophy partly because of increase in diameter of muscle fibers. Increased diameter fibers have greater force capabilities. These muscle fibers may be able to complete the same task with fewer active motor units. This might cause lower amplitude of the integrated EMG.

To try to observe if there are pre-programmed amplitude differences between subjects with tight hip flexors and subjects with loose hip flexors the construct early amplitude change, EAC, was formed. This construct was defined as the change in percent maximum amplitude during the period spanning .1 second before muscle onset until .4 seconds after muscle onset.

Relationship between EAC and Latency

EMD studies show that muscle behaviors almost always involve interplay between the length (pre-tensioning) of a muscle and contractile force supplied by the CC. Therefore, it would seem that latency and amplitude would usually be highly related. All other things equal, when there is greater CC amplitude, latency decreases. On the other hand, when the

pre-movement tension of the tendon is increased, a muscle may develop the same force with a decrease in latency and no increase in contractile activity. Therefore, testing the hypotheses regarding hypertrophy or hyperactivity of erector spinae with hip flexor tightness would seem to require trying to observe the simultaneous changes in amplitude and latency. If only one of these dependent variables was used, a finding of no change would always lead one to question if the other one had changed.

Subjects

A pilot study contrasted 5 tight-hip and 7 loose-hip subjects using four of the same dependent variables as in this study. One of the purposes of this study was to obtain power estimates from the results that would indicate the number of subjects needed for the study. The pilot study showed a main effect for hip type. However, the power for this study was only .62. Power estimates computed from this pilot study indicated that 30 subjects were needed. The number of subjects (30) was based on a power formula applied to effect sizes shown during pilot studies.[102] To obtain 30 subjects with scores on the criteria measurements at least + or - 1.5 sd, it was calculated that measurement of at least 215 potential subjects was required. This was established by using the Z distribution table, approximately 14% of subjects were outside + / - 1.5 sd therefore, 30.1 is 14% of 215.

Potential subjects were recruited from faculty, staff, and students at The University of Texas Medical Branch at Galveston, from local fitness centers, local colleges in the Galveston, Texas area. Eventually, 254 subjects were tested to obtain the subjects needed. Thirty-six potential subjects met the criteria for either the T or Lo group. Three potential subjects were eliminated to make the groups more equal in age, height, weight, and gender. Data was taken on 33 subjects. Data were reported from 32 subjects, as one subject's data was lost due to an experimenter computer error.

Sixteen tight-hip subjects were found. That is, each subject in this group had a pelvic inclination change from standing to prone lying of greater than 12.09 degrees and a hip flexor length measurement less than 0.26 degrees. These subjects were compared with 16 loose hip subjects whose pelvic inclination changed no greater than 2 degrees from standing to prone lying and whose hip flexor length test was greater than 21.04 degrees. Duration of onset sequence, latency of erector spinae muscle, and percent rise in EMG amplitude for the first second after onset of the erector spinae muscles were taken. Sixteen subjects from each group were compared for the DMOS and latency variables. Fourteen subjects from each group were compared on the EAC variables. A computer failure caused the loss of EAC data for two subjects from each group.

Table 4.3 below shows the demographics of each group. There were 14 females and 2 males in the loose group. Thirteen females and 3 males comprised the tight hip group. It was important to have the groups be equal or nearly equal in gender constituency, since it has been found that males usually have a shorter electromechanical delay than females. The age of the groups was nearly the same. Height and weight were similar.

Table 4.3 Demographics of the Tight and Loose Subjects

Loose hip g	Gender	Age	Height	Weight	ncr chang	IHFLT	
1	m	40	70	165	2	22	
2	f	25	62	112	3	28	
3	f	41	61	105	-3	27	
4	f	25	66	118	-2	32	
5	f	23	65	119	2	22	
6	f	25	70	132	2	22	
7	f	17	62	128	2	22	
8	f	24	62	114	3	27	
9	f	27	65	133	2	23	
10	f	51	67	147	3	23	
11	f	28	66	127	2	22	
12	f	31	63	125	1	23	
13	f	19	62	134	2	25	
14	m	17	72	162	3	23	
15	f	22	64	144	0	32	
16	m	18	68	157	2	24	
		27.1	65.3	132.6	1.5	24.8	Mean
F = 13		9.2	3.3	17.5	1.7	3.3	Stdev
M = 3		51	72	165	3	32	Max
		17	61	105	-3	22	Min
Tight hip group							
1	f	24	65	144	14	-1	
2	f	41	64	136	17	-1	
3	f	42	63	133	15	-2	
4	f	38	64	175	21	-5	
5	f	25	63	120	19	-4	
6	f	44	64	128	19	-3	
7	f	22	62	121	13	-1	
8	f	34	62	126	14	-1	
9	m	19	69	147	13	-1	
10	m	18	67	158	13	-1	
11	f	39	64	136	15	3	
12	f	37	61	120	17	-2	
13	f	31	66	132	13	-1	
14	f	17	62	127	15	-1	
15	f	55	68	144	14	-4	
16	m	19	71	187	14	-1	
		31.6	64.7	139.6	15.4	2.0	Mean
F = 13		11.0	2.7	18.8	2.4	1.3	Stdev
M = 3		55	71	187	21	-1	Max
		17	61	120	13	-5	Min

None of the subjects had a recent history of low back pain for which treatment had been sought. None had any hip problems. In addition, no

subject had a history of cruciate ligament injury to the knee or severe ankle injury. None had any brain injury, balance or equilibrium problems, nor any other neurological disease. No subject had a leg length discrepancy greater than 1 cm. No subject participated under duress, and each gave informed consent. Subjects had the opportunity to discontinue participation at any time without penalty. The research procedures were approved by the institutional review board of both the Texas Woman's University and The University of Texas Medical Branch at Galveston.

Test procedures

Each subject was asked to lie prone on a specially built table that supported the trunk. Two platforms, independent of the trunk-support table, provided support on the anterior surfaces of the thigh and leg with a cut-out area for the patella. The subject was positioned with the hip joint directly above the junction between the trunk support table and the leg support platforms. The trunk-supporting table had a cranking mechanism that could change the angle of the trunk from horizontal to a head-down angulation, changing the angle of hip flexion while keeping the legs horizontal. For-half the trials, the table was positioned so that the subject was in a prone position with hips at 0 degrees extension. For the other half of the trials, the cranking mechanism was used to position the subject in 30 degrees of hip flexion by inclining the trunk.

Each lower extremity support was attached to and supported by an AMTI Model 1630 strain gauge force plates (Advanced Mechanical Technology, Inc., Newton, MA 02158). The support platforms were offset from and attached to each force platform so that force applied produced a moment on the x axis. This was done in order to magnify the torque readings around this axis. Each force plate was oriented so that the platform fixed to its top caused the greatest signal on the channel displaying the magnitude of the x axis moment. In fact, the platforms were levers that magnified the moment. Pilot work found that with the force of one lower extremity, the signal from this channel was inadequate even at the highest gain to clearly show the onset of movement, but quite adequate using the offset platform. The offset platforms were necessary because force plates are usually used to measure changes in direction and magnitude of the full body weight of human subjects in the standing position, rather than partial body weight of an extremity. It was felt that a weakness of previous studies might be an inadequate signal of onset of movement from a potentiometer[89] or the inclinometer.[98] A potentiometer or inclinometer might not be sensitive enough to show the preparation for onset of movement. This study used both a force plate and motion analysis as movement onset markers. The signals from force plate and motion analysis gave both movement and preparation-for-movement data. Gait initiation studies show there are significant force changes before movement occurs. This movement might

not be captured by a potentiometer or inclinometer attached to the outer surface of an extremity.

When a prone subject lifts one extremity, there is stabilization by the opposite extremity. To capture this data a force plate was located in the platform supporting the left lower extremity. EMG recordings were also taken from the left erector spinae muscle. It was hoped that this data would provide a more complete description of the pre-programmed muscle activity required for the test movements. This data were not used in the analysis of this study.

The force plate supporting the left lower extremity was always stationary. The right force plate was supported by an air cylinder which could be vented so that the attached platform dropped approximately six inches. For the lift trials the air cylinder was not vented so that the right leg support platform also remained stationary and horizontal. In the hold trials, the air cylinder was vented, dropping the right support platform at an approximate rate of 5 degrees per second.

As a control for speed of movement during the lift trials, a horizontal steel rod was suspended above the posterior thigh. During the lift trials, the height of the rod was fixed so that the subjects were required to lift the thigh to an angle of approximately 30 degrees to touch the rod. A mirror was positioned so that the subject was able to see the thigh move in relation to the rod. A metronome was set to click at a rate of 90 beats per minute. The

subject was instructed to start the movement at any first click and, with an evenly paced movement, complete the movement (touch the rod with the back of the right thigh) on the sixth click. This spread the movement approximately across 3.3 seconds making the approximate velocity 9 degrees per second. Several practices were given until the subject performed this adequately. The motion analysis data provided a check to see that movements were done as required.

For observing the actual onset of movement, reflective markers were placed at three points: the subject's right malleolus, the subject's greater trochanter, and to a point on the lateral surface of the force plate. During each trial, the movement of these reflective markers was recorded by motion analysis system and analyzed by way of a 2 dimensional computerized video digitizing system (Expertvision, Motion Analysis, Santa Rosa, CA 95403). The system produced graphic representation of the change of angle between the three points, with the greater trochanter point being the axis. Data were recorded at a rate of 60 frames per second. During the lift trials this measured the increasing angulation caused by the movement of the leg from the plate. For the hold trials, the angulation caused by movement of the plate away from the leg was recorded.

The skin over the selected muscles was prepared for EMG electrode placement in the standard way by shaving, lightly abrading, and cleansing to reduce sources of impedance to less than 3000 ohms. Pre-amplifier EMG

recording electrodes (IODE Motion Control, Inc. Salt Lake City, UT 84119) were placed over the following muscles according to the technique advocated by DeLuca and Basmajian:[53] left erector spinae (LES), right erector spinae (RES), right gluteus maximus (RGM), and right hamstring RHM). Orientation of recording electrodes was parallel with the muscles' fibers. The sampling rate was 600 samples per second so that it could be time-matched with the motion analysis data. The data was collected using a bandpass filter with the low pass at 10 Hz and the high pass at 2000 Hz.

Each channel was tested to see that an adequate signal was emanating from each muscle. The subject then performed a maximum isometric contraction with each muscle. This reference data were used to normalize EMG relative to a maximum for the EAC calculations.

The subject performed as many practice voluntary lifts as necessary to obtain a smooth 9-degree-per-second movement of the right lower extremity into hip extension with knee straight. A mirror was positioned so that the subject could see his/her right leg and the rod that was the target for the movement. The subject used the same practice lifts as in the test lifts. First, an audible tone was presented. This tone started the simultaneous collection of video, force plate, and EMG data. After the tone, the subject started the movement with the right leg at a self-selected metronome click and finished the movement on the sixth click (3.3 seconds) when the metal rod was touched. Trials that did not meet this criteria were rejected.

Next, the subject practiced for the hold conditions. Before movement occurred in the test trials, the metal rod was positioned so that it was in contact with the subject's posterior right thigh with the right platform horizontal. The contact force between thigh and leg was at a point where the subject was first aware of distinct contact with the rod. An audible tone signalled the beginning of the trial. At either 3, 5, or 7 seconds after the tone, a button was pressed, venting the air cylinder. After these practice sessions, the subject knew that when this button was pressed, a loud hissing sound would be heard. After a brief delay(approximately .15 seconds after the start of the venting sound), the platform started its drop from beneath the right leg. The subject's task was to hold the right leg against the metal rod while the platform dropped from beneath it.

After the subject received instructions and practiced, he/she was tested. Conditions were randomly presented by coin flip. Before any trials commenced, there were two coin flips. The first flip established whether a lift or hold condition would be presented. The second flip established whether a flexed or extended condition would be presented. After the first condition was presented, the two coin flips were repeated. After the second two coin flips, only one coin flip was necessary to establish whether the third condition would be flexed or extended condition. There were seven trials for each condition. All trials for a condition were recorded before moving on to the next condition.

Each trial consisted of a 10-second sampling period. During each period, raw EMG signals were captured and pre-amplified by the electrodes, passed through an amplifier, and band-passed (10-2000 Hz) the signals. The signals were recorded at a rate of 600 Hz by an analog to digital data translation system (Data Translation, Inc., Marlboro, MA 01752). Data from the force plates were captured simultaneously by the same system, also sampled at 600 hz. The data translation system passed the data through the Motion Analysis MP280 integrator (Motion Analysis, Inc., Santa Rosa, CA 95403) to a Video Analog Collection software program (Video Analog Collection, Motion Analysis, Inc., Santa Rosa, CA 95403) operating on a Sun computer platform (Sun 3COM Vector Interface Drive, Sun Microsystems, Inc., Mountain View CA 94043). The VAC program stored the analog data in files on the Sun computer. These were transferred to data disks for later analysis.

The motion analysis system used in this study was able to collect analog and video data simultaneously. This video recorder was linked to the system through the MP280 integrator. The same signal that started the analog collection also started the video recorder, and continued the video collection for the same time as the analog data were taken. Following the analog collection, the VAC software package was used to digitize and store the video files on the Sun computer (Sun Microsystems, Inc., Mountainview, CA 94043). Each video file was linked automatically by code to the analog

file for the same subject. Copies of the analog files were off-loaded onto data disks for later analysis.

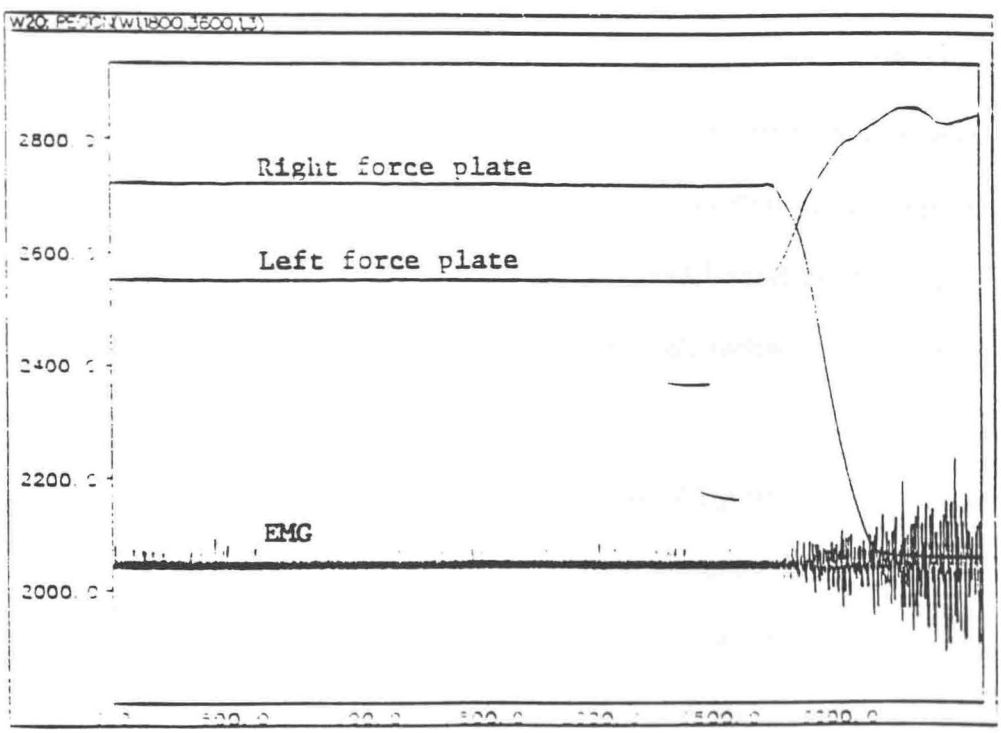
This motion analysis data collection system permits the recording of up to 32 channels of data within one file. In this study, seven of those channels were used. Two of the channels were dedicated to the force plates. Four channels were dedicated to EMG.

Force plate and EMG data were copied onto 3-1/4 floppy disks in a 7-column ASCII format. These ASCII files were analyzed by a program written for this purpose using the DADISP Data Analysis and Display Software package (DSP Development Corporation, Cambridge, MA 02139) running on a 486 PC (Gateway 2000, N. Sioux, SD 57049).

In the first step of this analysis, the 7-column ASCII file was imported into the worksheet environment of the DADisp program. The program then plotted the first column of the entire 6000 sample (600 samples per second for 10 seconds) which were the data for the left force plate. The data were plotted with time on the x axis and magnitude in millivolts on the y axis. Using the crosshairs function, the estimated time of onset of change on the left force plate was determined. This time was input into the program which caused a 1800-sample region to be selected with 600 samples (1 second) preceding and 1200 samples (2 seconds) following the estimated time of onset. The same 1800-sample region was then selected from each of the other six columns.

In the second step of the program, the onset time of movement on the two force plates was determined. The baseline activity on these channels was very clear, that is, without noise, making even small deflections from baseline easily seen. The program first set the baseline to 0. The levelcross function of the program then picked the first sample (samples were six consecutive data points in duration) that was 2 mv. above the baseline. Figure 4.6 below shows the clarity of the force plate signal and ease with which the change in amplitude could be selected visually.

Figure 4.6 Example of force plate data from one trial



The third step began by rectifying all the 1800-sample selections from the EMG channels and normalizing each record so that the baseline was 0. In most studies with EMG onset as a dependent variable, onset of muscle activity is determined by using a 5-percent-above-baseline criteria.[103] The 5% added to the baseline is calculated relative to a peak amplitude EMG reading that has been established by recording from a muscle during a maximum isometric contraction just preceding data collection. This peak amplitude reading is used to calculate the five percent criteria regardless of the performance of the muscle during any individual trials. For chosing the muscle onset, however, the maximum was defined as the peak amplitude EMG reading attained from an individual muscle during each individual trial. For example, if, during a trial, the maximum reading for a muscle was 150 mv, the onset time of muscle activity was the time that six consecutive samples averaged 7.5 mv. The muscle onset was based upon the peak amplitude of the muscle during the individual trial, rather than a maximum established independent of the trial.

To obtain the muscle onset value, a sub-program (the "davepk" program) was written which first calculated the average of the first 60 samples. It then returned to the beginning of the trial and averaged the amplitude values of the first six samples (.01 seconds of data).

If the average of those six samples equaled or exceeded the average of the first 60 samples plus 5% times the peak amplitude EMG for the trial,

the reading stopped, and the number of the beginning sample of that six-sample group was returned as the onset. If the average of that group was less than baseline plus 5% of the maximum, the next six samples were selected and averaged. Reading and averaging of six-sample groups continued sequentially until the first sample was found that met the criteria. If we use the previous example, where the maximum value for a particular muscle for a particular trial was 150 mv, for that trial the program stopped reading at the first six-sample group that averaged 7.5 mv. across the six samples. The onset of the muscle was the time of the first of the six samples.

To ensure the reliability and validity of selecting muscle onset, the results of determining onset manually was compared to onset selected by the sub-program. Two persons (the primary investigator and a person having nothing to do with the study) used the visual crosshairs function on the DaDisp display program to pick muscle onset. Using the crosshairs function, each was asked to estimate and record the muscle onset time for 20 EMG records selected at random. The same trials were then subjected to the davepk sub-program (the previously described sub-program that automatically selected the first five-percent-above-baseline sample). The results of the inter-rater correlation between the two, when using visually-selected onset times, is shown below in table 4.4. The table shows that the correlation between experimenter 1 and 2 using the visual method was .941.

The correlation between experimenter 1 and 2 using the davepk macro was 1.00. The estimated time selected visually by each experimenter was correlated with the time as selected by davepk. The correlation for experimenter 1 with davepk was .968, while the correlation for experimenter 2 with davepk was .955. It is interesting to note that in almost every instance, the davepk macro selected the onset times later than when either experimenter used the visual method.

Table 4.4 **A. Muscle onset times as estimated by two independent estimators (experimenter 1 and experimenter 2) using visual selection methods. Correlation between exp. 1 and exp. 2 is shown. B. Muscle onset times as estimated by exp. #1 and exp. #2 using a sub-program that automatically selects the first six samples that average 5% above baseline. Correlations are shown between the experimenters using visual and sub-program methods.**

A Muscle onset selected by experimenter # 1 using visual selection

	Trial 5	Trial 9	Trial 11	Trial 13
Muscle 1	414	734	760	2718
Muscle 2	346	674	692	2651
Muscle 3	365	711	903	2681
Muscle 4	515	835	896	2959
Muscle 5	433	787	926	2756

Muscle onset selected by experimenter # 2 using visual selection

	Trial 5	Trial 9	Trial 11	Trial 13
Muscle 1	500	723	745	1687
Muscle 2	380	658	700	1506
Muscle 3	369	655	1046	2688
Muscle 4	632	824	941	2620
Muscle 5	380	779	896	2620

CORRELATION #1 with #2 = 0.9413706

B Muscle onset selected by #1 and #2 using Davepk

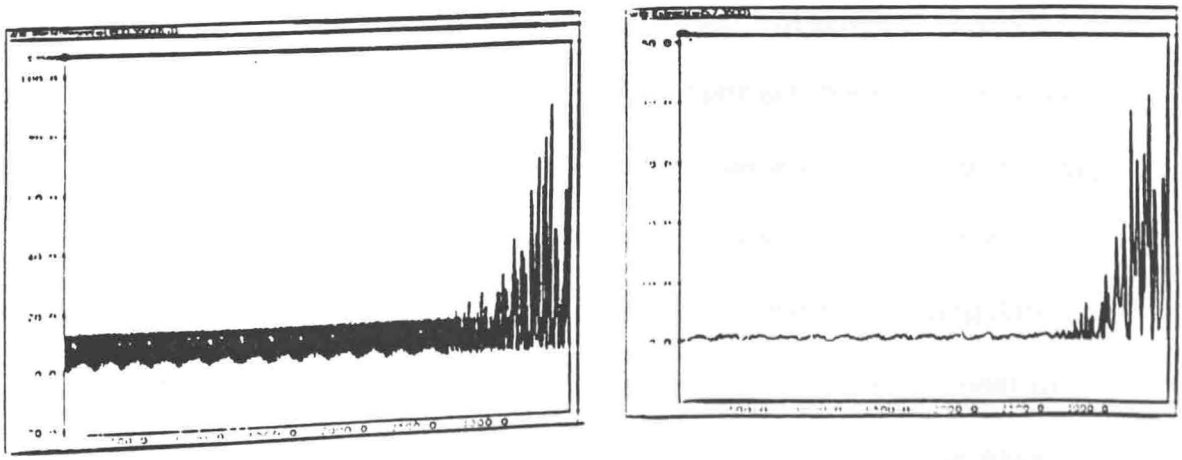
	Trial 5	Trial 9	Trial 11	Trial 13
Muscle 1	525	769	809	1687
Muscle 2	408	687	582	2673
Muscle 3	383	736	961	2726
Muscle 4	524	873	961	2880
Muscle 5	443	801	783	2763

Correlation #1 using visual with #1 using Davepk = 0.967901

Correlation #2 using visual with #2 using Davepk = 0.955093

Pilot studies revealed that an intermittent 60-cycle interference occasionally affected the EMG records. The source of the interference was could not be found. Therefore, it seemed necessary to filter the EMG signal during affected trials. The filter was a low pass filter having a stop edge of 35 hz and a band edge of 61. It was very effective at eliminating the interference as illustrated in figures 4.7a and 4.7b, below. Unfortunately, it also resulted in an attenuation of the signal by approximately 30%. Since time of onset is the dependent variable it seemed that using the filter on some trials and not on others might contaminate the results. Therefore, the filter was applied to all EMG records whether there was interference or not. All records were equally attenuated.

Figure 4.7 Comparison of EMG signal before (6a) and after (6b) filter applied.



The third step began by rectifying all the 1800-sample selections from the EMG channels and normalizing each record so that the baseline was 0. In most studies with EMG onset as a dependent variable, onset of muscle activity is determined by using a 5-percent-above-baseline criteria.[103] The 5% added to the baseline is calculated relative to a peak amplitude EMG reading that has been established by recording from a muscle during a maximum isometric contraction just preceding data collection. This peak amplitude reading is used to calculate the five percent criteria regardless of the performance of the muscle during any individual trials. For this study, however, the maximum was defined as the peak amplitude EMG reading attained from an individual muscle during each trial.

The DaDisp program procedures resulted in six onset-time values for each trial. These were the onset time of: 1) movement on the left force plate (the stabilizing limb); 2) movement on the right force plate (unweighting of the right limb from the force plate); 3) onset of the left erector spinae muscle activity; 4) onset of the right erector spinae muscle activity; 5) onset of the right gluteus maximus muscle activity; and, 6) onset of the right hamstring muscle activity. Each onset value from channels 2 through 6 was subtracted from the time of first increase in hip angulation from the video file. Most of the values were negative, indicating onset of muscle activity or force before hip movement onset. Therefore, for ease of computation, the value of 1 was added to each.

These values were cast into table form using a Microsoft Excel for Windows spreadsheet program (Microsoft Corporation, Redmond, WA 98073) running on a Gateway 2000 4DX2-66V (Gateway 2000, N. Sioux, SD 57049) 486 computer. Each row of the spreadsheet file represented one trial. In each row, the onset time of the first muscle to onset, OM1, was subtracted from the onset time of the fourth muscle, OM4. The mean OM4-OM1 was then calculated from the seven trials for each condition.

The latency value for LES and RES muscles were obtained during the OM4-OM1 calculations. Taking the absolute of the result of subtracting the LES or RES onset value from the movement onset gave a positive number. The mean and standard deviations were calculated for the seven trials for each condition.

The early amplitude change (EAC) of the LES and RES was also calculated for each trial. To do this, the time segment for collection of the EAC was isolated. First, the muscle onset was obtained using the davepk sub-program (the first six samples that averaged greater than 5% above baseline). The sub-program provided the onset information in sample number (the first sample of the six-sample segment). Next, .1 seconds were subtracted from the onset value by subtracting 60 samples. Next, .4 seconds were added to the onset - .1 value by adding 300 samples. This provided a .5-second window which included the .1 second before muscle onset until .4 seconds after onset. For example, if the LES muscle onset was

at 5 seconds during a trial, the onset time would be at sample 3000. Sixty samples would be subtracted from 3000 to yield a value of 2940, or 4.9 seconds. Three hundred samples would be added to 2940 to end the segment at sample 3240, or 5.4 seconds. EAC data were collected from this segment of the EMG record.

To obtain early amplitude change, the command line "mean (1,100)" was applied to the segment isolated for analysis by the DaDisp window. This yielded the mean amplitude value of the first 100 samples of the 300-sample segment. Next, the command "mean (200,100)" was applied obtaining the mean of the last 100 samples. The first mean value was subtracted from the last to obtain the difference in mean amplitude. Finally, the difference was divided by the maximum voluntary contraction (MVC) obtained during trial preliminary to testing to obtain a percent increase. Taking a percent increase was necessary to normalize the data.

Data Analysis

In keeping with the research design, a three-way factor analysis of variance statistical technique was used (SPSS, Inc., Chicago, IL 60611). The SPSS MANOVA program[104] was run on a VAX mainframe computer located at Texas Woman's University, Houston Center. For the major hypothesis a two way hip type by position interaction was expected for all variables.

Therefore, the first order interactions for each MANOVA were of greatest interest.

It was expected that the two-way interaction would show significance mean difference when loose and tight were compared for the extended but no difference for the flexed position. This pattern was expected to be shown for both the hold and lift movements. It was planned to perform these post-hoc comparisons using t tests assuming equal variances. Level of significance was divided by the number of tests according the Bonnferoni adjustment. Since four tests were involved in analyzing the two-way interaction, the significance level was set at $p<.0125$.

It may seem that a five-way MANOVA could be used. However, there were some compelling reasons for using five separate MANOVA analysis. The first was the relationship of erector spinae latency and erector spinae EAC. If these two dependent variables operated as expected, they would always have an inverse relationship; that is, an increase in one would be accompanied by a decrease in the other. Effects would cancel each other in a MANOVA. The DMOS variable may have had a inverse relationship to either latency or EAC as well. Second is the probable strong correlation between the dependent variables. MANOVA is a good statistical technique when independent variables are correlated but not when dependent variables are highly correlated.[105]

For the minor hypothesis the pattern of significant differences for all possible comparisons of the eight conditions was observed. There were 28 different comparisons. Therefore, the significance level used was $t < .0018$. This level was arrived at by dividing .05 by 28. Interpretation of the pattern of significant differences was done visually rather than statistically. Conditions were identified by type (external only, combination of external and internal, or internal only). The test was qualitative, that is, how close the pattern of significant differences came to a hypothesized pattern. This was done by counting the number of agreements with a pattern where contrasts of a certain type were significant and other types of contrasts were not.

Using chart inspection, first, the number of comparisons that were significant in a group was compared with the number that were expected to be significant. Next, a comparison of those that were significant was made to see if they agreed with those that were expected to be significant. To match the hypothesized table both the number of expected significant comparisons had to be matched and those found significant were required to have a weighted difference of 2 or greater.

Hypotheses

There are two hypotheses formulated for this study, a major hypothesis and a minor hypothesis. The major hypothesis develops expectations about differences in muscle behaviors of two different types of

muscle imbalance. For this hypothesis the hip type and position factors were most important. Position was manipulated to see if muscle behaviors of the two hip types were different for the two positions. For this hypothesis subjects were not compared on the movement factor.

The manner in which this hypothesis is formulated implies a causative element for the differences in muscle behaviors. That element is a difference in internal resistance between compared conditions, i.e., in the relative tightness of the anterior hip structures (see definitions of internal resistance, in the INTRODUCTION section). Conditions with greater internal resistance are expected to be significantly different from conditions with less internal resistance. There is a relationship between the hypothesis and suppositions made by proponents of the FMP approach. This will be discussed later.

The minor hypothesis deals with comparing the effects on muscle behavior of the two different types of resistance in this experiment. Conditions in this experiment can be typified on the basis of the type of resistance to hip extension. In this experiment resistance could come from purely external sources, from purely internal sources, and from two combinations of internal and external sources. Do internal and external resistance affect muscle behavior similarly? Is there a difference when the two types of resistance are combined and compared with either pure type? The relationship of this hypothesis and suppositions made by proponents of the FMP approach will be discussed later.

Weighting experimental conditions in preparation for formulation of both hypotheses

A basic step in development of both hypotheses was to generate a system of weighting conditions on the basis of hypothesized resistance. The weighting system was important for development of both the major and the minor hypothesis. Table 4.5 shows the weightings for each condition.

Each factor had two levels. One level was associated with more internal or external resistance, the other associated with less. For weighting the levels of the first two factors, hip type and position, it seemed reasonable

Table 4.5 Total weighting for each experimental condition

Lift	Hold
TLiE = 2+1+2+1= 6	THE = 2+2+2+1= 7
TLiF = 2+1+1 = 4	THF = 2+2+1 = 5
LoLiE = 1+1+2 = 4	LoHE = 1+2+2 = 5
LoLiF = 1+1+1 = 3	LoHF = 1+2+1 = 4

to make an important assumption.The assumption was that internal resistance of the stretched antagonist muscle (the hip flexors and anterior hip structures) would operate on the muscle behavior of the agonistic hip extensor muscles and their stabilizing muscles (the hip extensor and stabilizing muscles) like any other form of externally applied resistance.

Two of the three independent variables in this study were expected to cause alterations in muscle behavior on the basis of length of the anterior hip structures. These two factors were hip type and position. Each level of these two factors could be assigned a weight of either 1 or 2. The weight of 1 indicated an expectation the element on a factor would cause less stretch of the anterior hip, thus, less internal resistance. The weight of 2 indicated an expectation the element would cause the hip extensors to work against more stretch force of the anterior hip structures and, thus, against more internal resistance.

For the hip type factor, the hip loose subjects were expected to have less internal resistance to hip extension. The subjects were assigned to this group on the basis of their high scores on the HFL test. This indicated they would have less resistance to passive hip extension. Therefore, hip loose subjects were given a weight of 1 on this factor. Hip tight subjects were expected to have greater resistance to hip extension because their HFL test results were low indicating greater resistance to hip extension. They were given a 2 on the hip type factor.

For the position variable, weights were assigned according to magnitude of hip extension of the position. The greater the magnitude of hip extension, the greater the magnitude of anterior hip tissue stretch. Greater hip extension was expected to cause greater resistance to further hip extension. Since the hip flexed position had the lesser magnitude of hip

extension, this level was assigned a weight of 1 on the position factor. Extended positions were expected to create more stretch on the anterior hip structures and, therefore, more internal resistance to movement toward hip extension. Extended positions were assigned a weight of 2.

As part of the hypothesis it was expected that there would be a special relationship between the tight hip type and the extended position that would not be present in other combinations of conditions. Therefore, any condition in which there was a combination of tight hip type and extended position was given an extra weighting factor of 1. In table 4. 5 the top conditions in each column (TLiE and THE) are such conditions. Therefore, each is given an extra weight of 1 that the other conditions are not given.

Assignment of weights on the third factor, movement, was not according to internal resistance, i.e., more or less hip flexor stretch. Resistance for this factor was considered external. Pilot studies clearly showed that the required speed of contraction of the hold movement was faster than for the lift condition. The lift allowed the subjects to choose their own movement onset, while in the hold condition, they needed to react more quickly to the platform falling from beneath the leg at an unknown time. Therefore, to be successful at the hold movement, subjects needed to recruit their muscles faster. Weights were assigned to levels of this on the requirement of greater or lesser speed of contraction. The greater the required speed of contraction, the more resistance; it requires more force to

move the same weight faster. Therefore, the lift level of the movement factor was given a weight of 1, because of the slower required contraction speed. The hold element was given a weight of 2 based on a greater required contraction speed.

Each condition was given a total weighting that was a sum of the weightings given for its level on each factor. For example the TLiF condition was given a 4, (a 2 for hip type, a 1 for the lift movement and a 1 for the flexed position). This was done for each condition of the experiment. (See table 4.5 above). Each condition had a hypothesized total weighting based on magnitude of internal or external resistance.

The next step in hypothesizing was to develop expectations for differences between means when comparisons were made between conditions. These were developed by comparing the weighting differences for the two conditions in a comparison. Table 4, below, shows the expectations for all possible comparisons of the eight experimental conditions. The first two columns describe the comparison with the weighting for each condition; The third shows the weighting difference between conditions; and the fourth column whether a comparison is expected to be significantly different. This chart illustrates a basic rule that was used in formulating both the major and minor hypothesis. Comparisons with a weighting difference of 2 or greater are expected to show a

significant difference between means. Comparison with weighting differences less than 2 are not.

Table 4.6. Expected results for the 28 possible comparisons among the eight experimental conditions. Comparisons are based on their hypothesized difference in magnitude of internal and/or external resistance weighting.

First	vs	Second	difference	significant?
1. THE (7)	vs	LOLIF(3)	4	y
2. THE (7)	vs.	TLIF (4)	3	y
3. THE (7)	vs	LoLiE (4)	3	y
4. THE (7)	vs	LoHF (4)	3	y
5. TLIE(6)	vs	LoLiF(3)	3	y
6. THE (7)	vs.	THF (5)	2	y
7. THE (7)	vs.	LoHE(5)	2	y
8. TLIE(6)	vs	TLIF (4)	2	y
9. TLIE(6)	vs	LOLIE(4)	2	y
10. TLIE(6)	vs	LOHF (4)	2	y
11.THF(5)	vs	LOLIF(3)	2	y
12.LOHE(5)	vs	LOLIF(3)	2	y
13. THE (7)	vs.	TLIE (6)	1	n
14. TLIE(6)	vs	THF (5)	1	n
15. TLIE(6)	vs	LOHE (5)	1	n
16. THF(5)	vs	TLIF (4)	1	n
17. THF(5)	vs	LoLiE(4)	1	n
18. THF(5)	vs	LoHF (4)	1	n
19.LOHE(5)	vs	TLIF(4)	1	n
20.LOHE(5)	vs	LOLIE(4)	1	n
21.LOHE(5)	vs	LOHF (4)	1	n
22. TLiF(4)	vs	LOLIF(3)	1	n
23.LoLIE(4)	vs	LOLIF(3)	1	n
24.LOHF(4)	vs	LOLIF(3)	1	n
25. THF(5)	vs	LOHE (5)	0	n
26. TLIF(4)	vs	LOLIE(4)	0	n
27. TLIF(4)	vs	LOHF (4)	0	n
28.LOLIE(4)	vs	LOHF(4)	0	n

The Major Hypothesis

It is generally accepted that persons with tightness in the anterior hip develop stronger and tighter erector spinae muscles than normal persons. It

is suggested that the strengthening of these muscles probably is caused by increased resistance to the back extensor muscle due to the forwardly displaced line of gravity of the trunk during stance and the forward acceleration of the trunk during gait. Although accepted, these observations of trunk position and behavior in relation to the behavior of the back extensor muscles are difficult to observe. Janda has suggested that the unique behavior of muscles causing hip extension (including the erector spinae muscles) in persons with tight hip flexors can be observed using a simple prone hip extension test. The mechanical demands are different than those imposed in standing and walking. Nevertheless, tight hip subjects can be distinguished from subjects with no hip tightness.

In the prone hip extension test, there is no difference in mechanical demand between the tight hip group and subjects with no hip tightness that can be observed externally. It seems intuitive that the difference in muscle behavior might be due to the difference in tightness of anterior hip structures between groups. To test this idea, experimental conditions were developed which supposedly varied in magnitude of the anterior hip structure tension. If muscle behaviors vary in some relationship to changes in anterior hip tightness, the hypothesis that alteration of internal resistance is a possible cause of differences between subjects with tight hips and those without tight hips is will be supported.

The major experimental question concerns whether the difference in muscle behaviors between tight hip subjects and subjects with no hip tightness can be discerned using a prone hip extension test. If so, what might be the cause of that difference? Is the cause internal resistance from the tight anterior hip structures?

By examining tables 4.5, 4.6 and 4.7 one can see the development of the major hypothesis. Specific contrasts from table 4.6 above were selected. These were the difference between tight and loose subjects in the extended position (TLiE vs. LoLiE and THE vs. LoHE) vs. the difference between tight and loose subjects in the flexed position (TLiF vs. LoLiF and THF vs. LoHF). In chart 4.6 these comparisons are numbers 9, 22, 7, and 18. These comparisons have been selected out and presented separately in table 4.7.

Table 4.7 Specific comparisons selected to test the major hypothesis.

Comp. number	Comparison	Weighting difference	Expected Significance
9.	TLiE(2+1+2+1=6) vs.LoLiE(1+1+2=4)	2	Y
22.	TLiF(2+1+1=4) vs. LoLiF(1+1+1=3)	1	N
7.	THE(2+2+2+1=7) vs.LoHE(1+2+2=5)	2	Y
18.	THF(2+2+1=5) vs. LoHF (1+2+1=4)	1	N

It was hypothesized that tight and loose subjects could be distinguished on the basis of the behavior of their muscles during a prone hip

extension tasks. In addition, internal resistance is the cause of these muscle behavior differences. In the extended position, internal resistance was expected to be greater for both tight and loose groups compared to the flexed position. However, the tight group's internal resistance in the extended position was expected to be even greater than that for the loose group. In the flexed position, the disparity between tight and loose group was expected to be less. It was expected that a significant difference between loose and tight would be found for the extended position but not for the flexed position. For the lift conditions, the TLiE condition had a weighting of 6, while the LoLiE condition had a weighting of 4. Since this comparison has a difference of 2 it was expected to be significantly different. The TLiF condition, on the other hand had a total weighting of 4, while the LoLiF condition had a total weighting of 3. This comparison had a weighting difference of only 1 and was not expected to be significantly different.

The same pattern was expected for the hold conditions. The THE condition had a total weighting of 7, while the LoHE had a weighting of 5. With a weighting difference of 2, this comparison was expected to be significantly different. The THF condition had a total weighting of 5, while the LoHF condition had a total weighting of 4. With a weighting difference on only 1, this comparison was expected to show means that were not significantly different. It was hypothesized that a two-way interaction would

be found for all five dependent variables (DMOS, LES Latency, RES latency, LES EAC, and RES EAC).

It is important to note that for the major hypothesis comparisons in which there was a difference in movement were not contrasted. The movement factor was one in which the external resistance changed between levels. The hip type and position factors were ones in which the internal resistance changed with external resistance held constant. Since the major hypothesis concerns the whether loose and tight subjects can be distinguished using manipulation of internal resistance (change in position), movement was not important.

Hypothesis Statements - Major Hypothesis

Research Hypothesis - It is hypothesized that muscle behaviors of tight hip subjects are different from loose hip subjects when performing simple hip extension movements. Differences are related to the effect on muscle behavior of internal resistance from tight anterior hip structures. These effects are seen in the behavior of muscles opposing the hip flexors in general and specifically in the behaviors of the erector spinae muscles.

Statistical Hypothesis - There is a two-way hip type by position interaction. Follow-up tests will show a difference between tight and loose groups in extended position but not in the flexed position. The

same tight-loose significant difference in extension but not in flexion will be shown for both lift and hold. All five dependent variables will show the same statistical findings.

The Minor Hypothesis

Those persons with tight anterior hip structures are assumed to develop the unique way of hip and back muscle behavior due to long term exposure to different external resistance. That external resistance is the position and acceleration of the center of gravity of the trunk. Janda states that persons with tight hip flexors can be distinguished on the basis of their EMG muscle using a prone hip extension movement. In this test, it would appear that the only difference between subjects with tight anterior hips performing prone hip extension and subjects without tightness performing the same movement is the tightness of the anterior hip structures in the tight subjects. This is an internal source of resistance. Therefore, the assumption is made that the response of hip and back muscles to external resistance is the same as their response to internal resistance. The minor hypothesis attempts to test this assumption.

Although it is assumed muscle response to internal and external resistance is similar, there are reasons to believe otherwise. The discussion of epicritic sensation and its effect on movement (see Review of Literature section) presented several different excitatory and inhibitory influences on

the motor neuron pool that might make muscle response to internal and external sources of resistance different. One difference is that the sensation associated with internal resistance may be different from that associated with external resistance. For example, when an external source of resistance is applied, there is usually an increase in contact force somewhere on the skin surface. This contact may operate as an input into the sensory system that alters the muscle behavior. With internal sources of resistance, such as used in this experiment, there is often no contact on the skin surface. Another difference could be input to the vestibular system of the position of center of gravity of body segments relative to each other. Of particular relevance in this study is the possibility that receptors may be able to sense how quickly the center of gravity of a segment may be moving. The muscle response to this type of external input may be different than the muscle response to gradual tightening of opposing tissues.

Another issue in the discussion of the comparison between response to internal versus external resistance is the possible effects of internal resistance on external resistance. In most functional movements and exercises, the hip extensors are at a mid-lengthened range when used to work against external resistance. In this middle range, the contractile part can be more efficient, since the tendinous part of the muscle is at an optimal length and there is also an optimal number of cross bridges available within the myofibrils.[62] In addition, there is no hindrance in this mid-length from the

passive tension in the opposing antagonist. The range of motion one must achieve to get resistance from a stretched antagonist (the range used in this study) requires the agonistic muscle to be taken to its actively insufficient range (slack), or slack length. Here, the sensation affecting a muscle's performance may be different than when the muscle is meeting resistance in mid-length. The response to resistance may be different when overcoming the external resistance when at the same time there is internal resistance compared to overcoming resistance when there is no accompanying internal resistance. This experiment presents the opportunity to compare different combinations of internal and external resistance to test the assumption of equal response to all types of resistance.

By examining tables 4.5 and 4.6, one can also see how the minor hypothesis was developed. Table 4.6 shows that comparisons 1 through 12 have a weighting difference of 2 or greater. It was expected that means for these 12 comparisons would be significantly different. Comparisons 13 through 28 had weighting differences less that 2. These comparisons were expected to be not significant.

The next step is to identify those comparisons in which the resistance is purely external, comparisons where there is a mixture of internal and external resistance and comparisons in which the resistance is purely internal. This is done in table 4.8. In the first column the comparison is identified. The weighting on the basis of resistance is given in parentheses

Table 4.8 Hypothesized pattern of 28 comparisons of the eight experimental conditions. Significance = $p < .0018$ (.05/28)

Comparison	Weight difference	Expected significance	
External Resistance		**Expected = 6/16;**	
External Only	Difference	Expected Significance	
1. THE (7) v. TLiE (6)	1	N	
2. THF (5) v. TLiF (4)	1	N	
3. LoHE (5) v. LoLiE (4)	1	N	
4. LoHF (4) v. LoLiF (3)	1	N	Expected = 0/4
Combined Resistance			
External/Internal X 2			
5. THE (7) v. LoLiF (3)	4	Y	
6. TLiE (6) v. LoHF (4)	2	Y	
7. THF (5) v. LoLiE (4)	1	N	
8. LoHE (5) v. TLiF (4)	1	N	Expected = 2/4
External/Internal X 1			
9. THE(7) v. LoLiE (4)	3	Y	
10. THE (7) v. TLiF (4)	3	Y	
11. THF (5) v. LoLiF(3)	2	Y	
12. LoHE(5) v. LoLiF(3)	2	Y	
13. TLiE (6) v. LoHE (5)	1	N	
14. TLiE (6) v. THF (5)	1	N	
15. TLiF(4) v. LoHF (4)	0	N	
16. LoLiE (4) v. LoHF(4)	0	N	Expected = 4/8
Internal Resistance		**Expected = 6/12**	
Internal X 2			
17. THE (7) v. LoHF(4)	3	Y	
18. TLiE (6) v. LoLiF(3)	3	Y	
19. THF(5) v. LoHE (5)	0	N	
20. TLiF (4) v. LoLiE(4)	0	N	Expected = 2/4
Internal X 1			
21. THE (7) v. THF (5)	2	Y	
22. THE(7) v. LoHE(5)	2	Y	
23. TLiE(6) v. LoLiE (4)	2	Y	
24. TLiE (6) v. TLiF (4)	2	Y	
25. THF(5) v. LoHF(4)	1	N	
26. LoHE(5) v. LoHF(4)	1	N	
27. TLiF (4) v. LoLiF(3)	1	N	
28. LoLiE(4) v. LoLiF(3)	1	N	Expected = 4/8

for each of the conditions in the comparison. In the second column is the difference in weighting for the comparison. In the third column is the expectation for that comparison being significant. This is indicated by a "Y" or "N". A "Y" is given if the comparison has conditions that differ in weight by 2 or more. A "N" is given if the comparison has conditions that have a weighted difference of less than 2. Comparisons are grouped first as either external or internal. External comparisons were those which had a difference in movement regardless of the other factors. Internal comparison were those that were the same on the movement factor. External comparisons were further subdivided into three categories, external only, external/internal X 2, and external/internal X 1. External only comparisons were those that were different on the movement factor only, i.e., there was no difference between conditions on either hip type or position. External/internal X 2 were comparisons in which all factors differed, i.e., they differed on the movement factor and the two internal resistance factors. External/internal X 1 were comparisons that differed in movement and one of the internal resistance factors (either hip type or position).

Internal comparisons were subdivided into two groups. Internal X 2 comparisons were those in which the movement was the same but there was a difference on both internal resistance factors. Internal X 1 comparisons were those in which there was a difference on one internal resistance factor only.

The hypothesis was constructed in this way to compare the different types of resistance. Expectations for being significant was based on the magnitude of hypothesized difference in resistance. If there was a difference of 2 or greater the comparison was expected to be significant. If the comparison had a weighting difference of less than two it was not expected to be different.

The expectation was that the pattern of significant and non-significant comparisons would match the pattern described in table 4.8. This would support the hypothesis that there is no difference in muscle response to distinctive types of resistance supporting Janda's claim that muscle response to internal resistance can accurately reflect the effects of external resistance.

A full compliance with the pattern shown in table 4.8 was expected despite the stringent significance level, $p < .0018$. Janda and Lewit propose that order of muscle onset and relative amplitude are visible to the naked eye during clinical tests.[106,3,88] Therefore, an expectation that this table would be matched exactly using EMG measures, a supposedly much more sensitive measure of muscle performance.

Hypothesizing in this way gave the opportunity not only to test whether internal/external resistance is effective in influencing muscle behavior but also to observe how extensive is the effect. Full agreement with table 4.8 indicates absolutely no difference between types of

resistance. Less agreement indicates there might be a difference in response to different types of resistance.

Hypothesis Statements - Minor Hypothesis

Research Hypothesis - Muscle behaviors are the same regardless of the source of resistance. Behaviors will be the same whether the source is external, internal or any combination thereof. The implication is that the Janda hip extension test which uses internal resistance is valid for showing the long-term effects of external resistance.

Statistical Hypothesis - There are eight experimental conditions. Each can be weighted on the basis of internal and/or external resistance. There are twenty-eight possible comparisons between the eight conditions of the experiment. When all twenty-eight comparisons are done, the results show a specific pattern of significant and non-significant comparisons in which comparisons in which the conditions have a weighting difference of 2 or greater are significant but comparisons in which the conditions have a weighting difference less than 2 are not significant regardless of the source of resistance.

Directional Hypotheses

It is important to note that t tests done as follow ups for the major hypothesis are one-tailed tests. Therefore, there are directional hypotheses

for hypothesizing regarding the behavior of the erector spinae muscles. FMP proponents propose that a hyperactivity of the erector spinae develops when the anterior hip structures are tight. Therefore, the erector spinae should behave in a manner indicating hyperactivity. Directional hypotheses were derived for each dependent variable (type of muscle behavior) based on what should occur if erector spinae are hyperactive.

A pilot study compared two groups with different hip flexor length measurements as this study does. However, the requirements for inclusion into a hip type grouping was not as stringent as that used in this study. To be included in the hip tight group, subjects had to have an HFL test that was at least 1 standard deviation below the mean. To be included in the hip loose group, subjects had to have an HFL test that was at least 1 standard deviation above the mean. Appendix, table A.1 shows the results of this study. The results of this pilot study were used in the formation of directional hypothesis. The results of a pilot study, a study by Ross, et. al.,[107] and electro-mechanical delay studies[54,78] were also used.

DMOS Directional Hypothesis

It was hypothesized that a inverse relationship would be found between internal resistance and DMOS. Anticipatory postural adjustment studies showed duration of onset sequence should be greater with greater resistance. The typical APA study uses a standing arm lift paradigm. These

studies show that when subjects lift a weight rather than just the arm, a longer onset sequence duration of the functionally related muscles occurs.[108,25,109,110] However, caution must be used in applying this interpretation to this study, since in APA studies subjects are performing in a standing position. In standing, the increase in DMOS might be due to time needed for muscles performing posture stabilization to deal with greater perturbation of the upright stance when resistance increases. The present study used a prone lying paradigm in which this apparent reason for prolongation of onset sequence might not have been necessary.

Bullock-Saxton has demonstrated a greater DMOS in subjects with ankle dysfunction, one of the same voluntary lift conditions as used in this study. The same four muscles were sampled by EMG. This affected-greater-than-unaffected difference was seen both when the subjects' unaffected lower extremity was compared with the affected extremity, and when subjects with ankle dysfunction were compared with subjects without ankle dysfunction.[98] This might indicate that persons with ankle dysfunction are overcoming greater resistance when they extended their hips, because their muscles may have been weaker due to disuse. However, strength measurements were not used to classify subjects. Therefore, these results are not very helpful in hypothesizing the results of this study. It is not clear whether the ankle dysfunction group would behave most like the hip tight group or hip loose group.

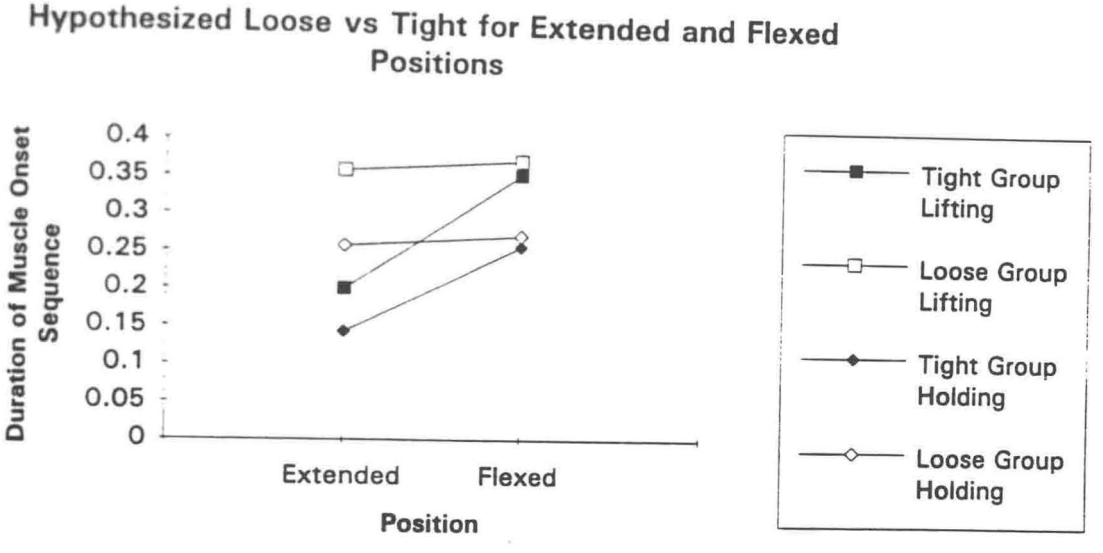
The pilot study seems more helpful in formulating the directional hypotheses. The procedures in this study were identical to the present study, except for the subject classification. The subjects in this pilot study did not have HFL test and pelvic inclination change tests results that were as divergent. There were no significant differences for the DMOS variable. However, a trend was shown (eleven times out of eleven) in which the condition with the greater hypothesized internal resistance had the lesser DMOS. In addition, the greatest mean difference was between the THE and the LoLiF conditions. The condition with the greatest combined resistance should be THE and the least LoLiF. THE had the shortest DMOS and the LoLiF had the greatest. This supports a directional hypothesis for an inverse relationship between magnitude of internal resistance and DMOS.

Ross, et. al., had subjects perform back extension against four levels of resistance. They found a decrease in DMOS (which they called agonist-antagonist latency) with each increased level of external resistance. These differences were statistically significant.[107] This also supports a directional hypothesis of decreased DMOS with increased resistance.

Figure 4.8, below, illustrates the major hypothesis for the DMOS variable. It shows that the directional hypotheses for DMOS is that there is an inverse relationship between internal resistance and DMOS. A two-way interaction shows that the tight group has a shorter DMOS in the extended position but no difference in the flexed position. The graph shows that the

pattern of differences for lift and hold was expected to be the same indicating a two-way interaction. In addition for the both lift and hold, the flexed conditions show that a greater DMOS was expected for these conditions. This reflects the expectation that the greater resistance is

Figure 4.8 Hypothesized Differences for DMOS



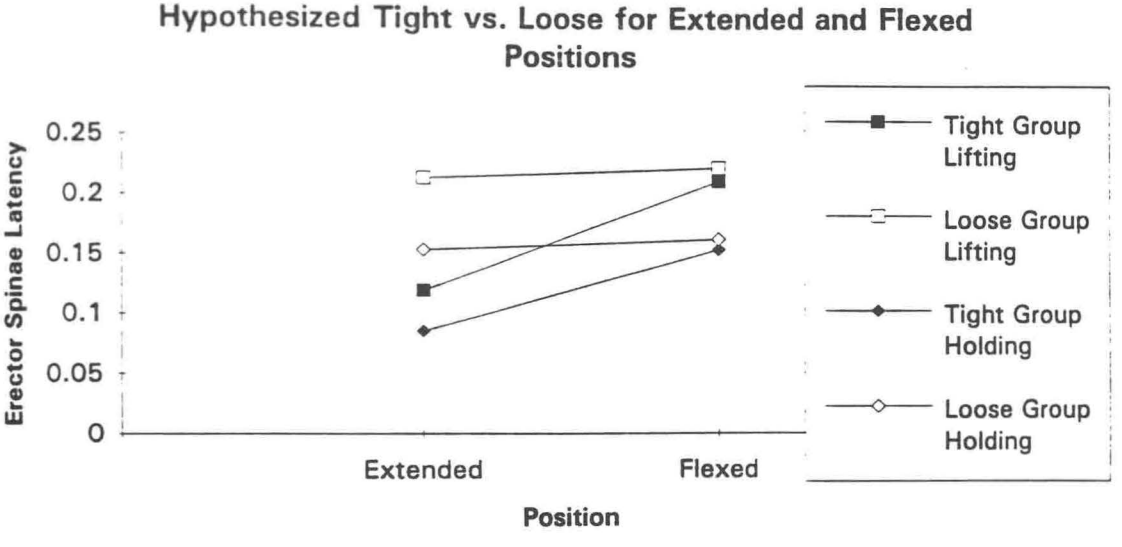
hypothesized for the extended position where the anterior hip structures are expected to be tighter, thus giving more resistance in the flexed positions.

Latency Directional Hypothesis

Figure 4.9 below illustrates the expectations for erector spinae latency. An inverse relationship between internal resistance and latency of erector spinae muscles is hypothesized. It is a widely accepted clinical

observation that patients with apparently tight hip flexors develop tight and hypertrophic erector spinae muscles. This is thought to be due to the mechanical demands of maintaining an erect trunk during standing and gait. Janda suggests that, in addition to the mechanical demand, the erector spinae take on a hyperexcitability. He suggests that muscles show this hyperexcitability regardless of mechanical demand. A specific definition of hyperexcitability is not given but by deduction it seems to mean excessively

Figure 4.9 Hypothesized differences for erector spinae latency.



early and increased amplitude of contractile activity in the erector spinae. The deducted definition includes early activity. In this study increased resistance is hypothesized to be the trigger for this behavior. Since the tight group conditions have greater resistance, the expectation is for early activity

in this conditions. An inverse relationship between resistance and latency is hypothesized. With increased resistance, latency is expected to be shorter.

This notion is supported by electro-mechanical delay studies. If muscle length is the same, when muscles meet greater resistance, EMD (latency) is shorter.[54,108] However, EMD studies used external source resistance. No studies exist on the effects of internal resistance on EMD.

A pilot study (n = 12) showed a significant two-way movement by hip type interaction. The mean latency for loose lift (the condition with the least hypothesized resistance) was greater than the mean for tight lift. This supports an inverse relationship between latency and internal resistance. However, the mean for tight hold (the condition with the greater hypothesized internal resistance) was greater than the mean for loose hold. The trends showed that, 17 times out of 22 comparisons, the condition with the greater hypothesized resistance showed the lesser latency. (See appendix A.1.)

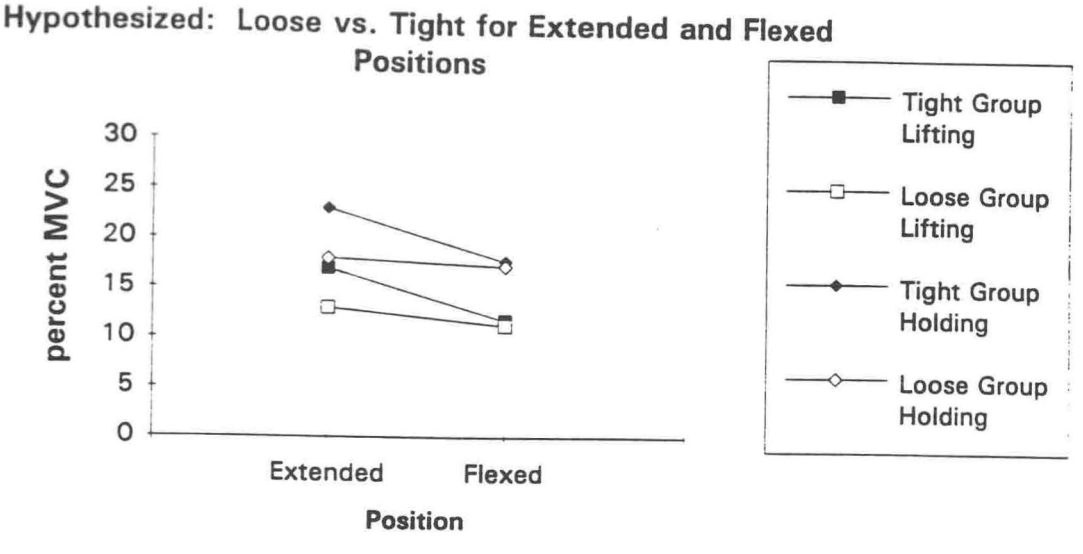
The hypotheses for latency is a two-way hip type by position interaction exists with the tight hip group in the extended position showing a shorter latency in the extended position but no difference in flexed positions.

EAC Directional Hypothesis

Figure 4.10, below, illustrates the expected two-way interaction for EAC for the major hypothesis. A direct relationship between internal resistance and early amplitude change in the pre-movement period is

expected. A widely-accepted clinical observation is hypertrophy of erector spinae muscles with hip flexor tightness. This suggests that amplitude of erector spinae might be greater in persons with hip tightness due to increased resistance. The source of resistance is assumed to be the position and acceleration of the trunk weight during stance and gait. Janda's work suggests the development of hyperactivity in the erector spinae muscles so that early and inappropriately increased amplitude activity occurs in these

Figure 4.10 Hypothesized differences for early amplitude change



muscles regardless of mechanical demand. Therefore, it is logical that the directional hypothesis for EAC is that conditions with greater resistance produce greater early amplitude change.

The pilot study (n = 12) led to more doubt than confirmation of this hypothesis.(See Appendix tables A.1 and A2) Surprisingly, it showed that in only six out of eleven comparisons, the condition with the greater resistance had the greater pre-movement amplitude change . Ross, et. al., found an increase in EMG amplitude of erector spinae with increase in external resistance to back extension. However, the resistance was increased by 20% for each level. Mean amplitude was not always significantly different at adjacent levels of resistance. For example, the mean for 30% was not significantly different from the 50% mean. Nevertheless, there was a tendency for increase in amplitude with each increase in resistance. Therefore, a direct relationship between LES and RES EAC and resistance is hypothesized. In the two-way interaction supporting the major hypothesis, for both lift and hold, the tight group is expected to show greater EAC in the extended position but no difference in the flexed position.

CHAPTER 5

RESULTS

Table 5.1 below shows the mean DMOS for each of the experimental conditions for this variable.

Table 5.1 Mean and standard deviation DMOS in seconds for each experimental condition.

Condition	Mean sec.	Stand.Dev.
Loose lifting in extension (LoLiE)	.301	.151
Tight lifting in extension (TLiE)	.222	.092
Loose lifting in flexion (LoLiF)	.373	.182
Tight lifting in flexion (TLiF)	.187	.074
Loose holding in extension (LoHE)	.170	.078
Tight holding in extension (THE)	.126	.054
Loose holding in flexion (LoHF)	.146	.072
Tight holding in flexion (THF)	.143	.066

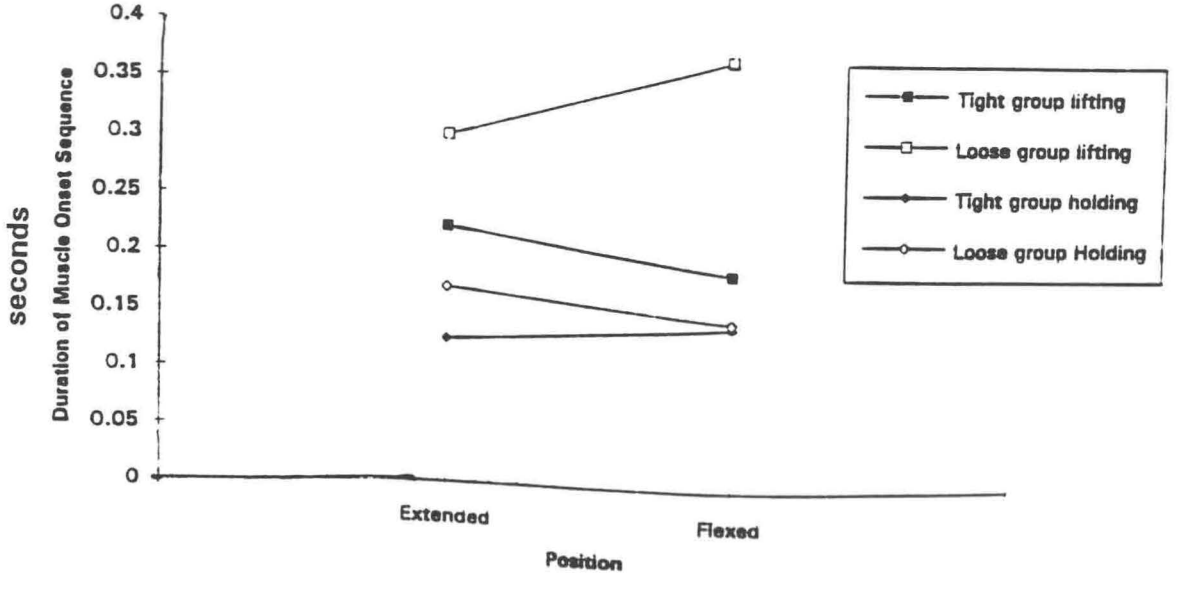
Table 5.2 below shows the statistical results for the DMOS variable. Significant differences are in bold type accompanied by an asterisk.

A significant three-way interaction was found for the DMOS variable. A graph of this interaction is shown in figure 5.1, below.

Table 5.2 Results Table for DMOS (n = 32; 16 Tight vs. 16 Loose)

	F value	df	p value
Main Effects			
Hip type	**F = 10.47***	**1,30**	**p = .003**
Position	F = .25	1,30	p = .618
Movement	**F = 36.47***	**1,30**	**p = .000**
Two Way Interactions			
Hip type by Position	F = 1.27	1,30	p = .269
Hip type by Movement	**F = 6.88***	**1,30**	**p = .014**
Movement by Position	F = .71	1,30	p = .407
Three-way Interaction			
Hip by Position by Movement	**F = 8.19***	**1,30**	**p = .008**

Figure 5.1 DMOS (in seconds) for tight vs. loose groups lifting and holding in extended and flexed positions.



Follow-up t-tests were conducted to analyze the three-way interaction. There were the four comparisons related to the major hypothesis. Since four different t-tests were conducted, the significance level was reduced to .0125 according to the Bonferroni adjustment (.05 divided by 4). Table 5.3, below, shows the results of the follow-up comparisons for the three-way interaction. Only those comparisons necessary for testing the major hypothesis are shown.

Table 5.3 Follow-up comparisons for the three-way DMOS interaction. (Four tests conducted at p = .0125 level).

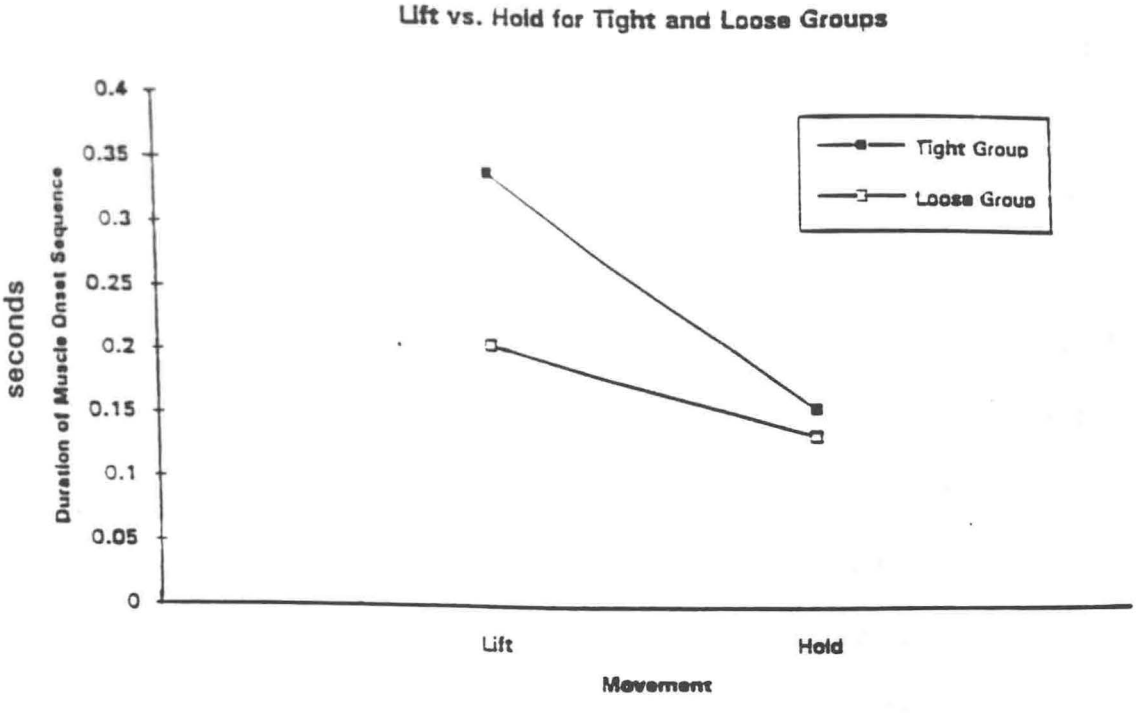
Comparisons- Major question	t value	df	p value
Loose/Lift/Extend vs. Tight/Lift/Extend	1.793	30	.0415
Loose/Lift/Flexed vs. Tight/Lift/Flexed	**3.769**	**30**	**.0004***
Loose/Hold/Extend vs. Tight/Hold/Extend	1.762	30	.0442
Loose/Hold/Flexed vs. Tight/Hold/Flexed	0.111	30	.4561

Regarding the major hypothesis, the tight group lifting in flexion vs. loose group lifting in flexion difference (TLiF vs. LoLiF) was significant (t = 3.769; p = .0004). The mean for the loose group lifting in flexion (.373) was significantly greater than the mean for tight group lifting in flexion (.187; difference = .186). None of the other four comparisons was significantly different.

Table 5.2 shows there is a significant two-way hip type by movement interaction (F = 6.88; p = .014). Usually, if a three-way interaction is found, it is inappropriate to interpret either a two-way interaction or main

effects. In some unusual circumstances, it is appropriate. This occurs when collapsing over variables does not result in loss of information.

Figure 5.2 Two-way interaction for DMOS (in seconds).



The two-way interaction is hip type by movement. Therefore, to see if this is one of those unusual cases where the two-way interaction should be interpreted, collapsing over the position variable is necessary. Figure 5.2 is a graph of this two-way interaction.

It would be appropriate to interpret the two-way interaction in this case only if the tight-loose comparison above lift and tight-loose comparison

above hold were significant. For economy, the closest comparison is tested. This is the comparison above hold: the tight holding vs. the loose holding comparison. This comparison was not significant, ($t = .9365$, $p = .250 > p = .05$, $df = 62$). Therefore, it is inappropriate to interpret the two-way movement by hip type interaction.

Table 5.2 also shows a significant main effect for hip type ($F = 10.47$, $p = .003$), and a significant main effect for movement ($F = 36.47$, $p = .000$). Because of the outcome of the three-way interaction, it is inappropriate to interpret either of these.

Table 5.4 is the results table to test the minor hypothesis. It shows 10 comparisons were significant at the $p < .0018$ level. Nine of those found significant were external comparisons. Four of the 9 external comparisons were those that had a weighting difference greater than 2 and therefore were expected to be significant. Five of the 9 were comparisons with a weighting difference less than 2 and were not expected to be different. Three external comparisons found significant were in the external only group. Comparison in this group had weighting differences of less than one and were not expected to be significant.

Only one of the internal comparisons was found significant. None of the internal comparisons expected to be significant were significant. The one internal comparison found significant was an internal X 1 comparison that was not expected to be different.

Table 5.4 DMOS results table to test the minor hypothesis.
Significance = p< .0018

Comparison	t value, p value	Expected	Observed
External Resistance	**Expected = 0/4; Observed = 3/4**		
External Only			
1. THE v. TLiE	t = 3.646, p = .0005	N	Y
2. THF v. TLiF	t = 1.80, p = .041	N	N
3. LoHE v. LoLiE	t = 3.237, p = .0015	N	Y
4. LoHF v. LoLiF	t = 4.64, p = .00003	N	Y
		Expected = 0/4;	Observed = 3/4
Combined Resistance	**Expected = 6/12; Observed = 6/12**		
External/Internal X 2			
5. THE v. LoLiF	t = 5.206, p = .000007	Y	Y
6. TLiE v. LoHF	t = 2.632, p = .0066	Y	N
7. THF v. LoLiE	t = 3.839, p = .0003	N	Y
8. LoHE v. TLiF	t = .8300, p = .2065	N	N
		Expected = 2/4;	Observed = 2/4
External/Internal X 1			
9. THE v. LoLiE	t = 4.397, p = .0001	Y	Y
10. THE v. TLiF	t = 2.702, p = .0056	Y	N
11. THF v. LoLiF	t = 4.749, p = .00002	Y	Y
12. LoHE v. LoLiF	t = 4.214, p = .0001	Y	Y
13. TLiE v. LoHE	t = 3.647, p = .0005	N	Y
14. TLiE vs. THF	t = 2.822, p = .0042	N	N
15. TLiF v. LoHF	t = 1.616, p = .0583	N	N
16. LoLiE v. LoHF	t = 3.729, p = .004	N	N
		Expected = 4/8;	Observed = 4/8
Internal Resistance	**Expected = 6/12;Observed = 1/12**		
Internal X 2			
17. THE v. LoHF	t = .8862, p = .1913	Y	N
18. TLiE v. LoLiF	t = 2.950, p = .0031	Y	N
19. THF v. LoHE	t = .9271, p = .1806	N	N
20. TLiF v LoLiE	t = 2.717, p = .0054	N	N
		Expected = 2/4;	Observed = 0/4
Internal X 1			
21. THE vs. THF	t = .814, p = .2111	Y	N
22. THE v. LoHE	t = 1.762, p = .089	Y	N
23. TLiE v. LoLiE	t = 1.793, p = .042	Y	N
24. TLiE v. TLiF	t = 1.928, p = .0317	Y	N
25. THF v. LoHF	t = .1110, p = .4562	N	N
26. LoHE v. LoHF	t = .7808, p = .2205	N	N
27. TLiF v. LoLiF	t = 3.770, p = .0007	N	Y
28. LoLiE v. LoLiF	t = 2.717, p = .0054	N	N
		Expected = 4/8;	Observed = 1/8

Table 5.5 Mean LES latency (in seconds) for each experimental condition.

Condition	Mean sec.	Stand.Dev.
Loose lifting in extension (LoLiE)	.2217	.1614
Tight lifting in extension (TLiE)	.1382	.2153
Loose lifting in flexion (LoLiF)	.2126	.1674
Tight lifting in flexion (TLiF)	.1654	.1878
Loose holding in extension (LoHE)	.1511	.1493
Tight holding in extension (THE)	.1174	.1595
Loose holding in flexion (LoHF)	.1411	.1659
Tight holding in flexion (THF)	.1144	.1402

Table 5.6 Results Table for LES Latency (n=32; 16 Tight vs. 16 Loose)

	F value	df	p value
Main Effects			
Hip type	F=.78	1,30	p=.384
Position	F=.01	1,30	p=.917
Movement	**F=7.51***	**1,30**	**p=.01**
Two-way Interactions			
Hip type by Position	F=.82	1,30	p=.371
Hip type by Movement	F=.81	1,30	p=.375
Movement by Position	F=.42	1,30	p=.520
Three-way interaction			
Hip type by Position by Movement	F=.38	1,30	p=.544

Table 5.7 LES latency results table to test the minor hypothesis.
Significance = p< .0018 (.05/28)

Comparison	t value, p value	Expected	Observed
External Resistance	**Expected = 0/4;Observed = 0/16**		
External Only			
1. THE v. TLiE	t=.3109, p=.3790	N	N
2. THF v. TLiF	t=2.0073, p=.0269	N	N
3. LoHE v. LoLiE	t=.0579, p=.4771	N	N
4. LoHF v. LoLiF	t=1.2129, p=.1173	N	N
	Expected = 0/4; Observed = 0/4		
Combined Resistance	**Expected 6/12; Observed = 0/12**		
External/Internal X 2			
5. THE v. LoLiF	t=1.646, p=.0551	Y	N
6. TLiE v. LoHF	t=.1988, p=.4219	Y	N
7. THF v. LoLiE	t=.9096, p=.1852	N	N
8. LoHE v. TLiF	t=.3110, p=.3790	N	N
	Expected = 2/4; Observed = 0/4		
External/Internal X 1			
9. THE v. LoLiE	t=1.838, p=.038	Y	N
10. THE v. TLiF	t=.7781, p=.2213	Y	N
11. THF v. LoLiF	t=.7781, p=.2213	Y	N
12. LoHE v. LoLiF	t=.7781, p=.2213	Y	N
13. TLiE v. LoHE	t=.0437, p=.4827	N	N
14. TLiE vs. THF	t=.3726, p=.3561	N	N
15. TLiF v. LoHF	t=.3875, p=.3506	N	N
16. LoLiE v. LoHF	t=4.393, p=.0870	N	N
	Expected = 4/8; Observed = 0/8		
Internal Resistance	**Expected = 6/12;Observed = 0/12**		
Internal X 2			
17. THE v. LoHF	t=.4112, p=.3419	Y	N
18. TLiE v. LoLiF	t=1.098, p=.1405	Y	N
19. THF v. LoHE	t=.7170, p=.2395	N	N
20. TLiF v LoLiE	t=.3163, p=.3845	N	N
	Expected = 2/4; Observed = 0/4		
Internal X 1			
21. THE vs. THF	t=. 0579, p=.4771	Y	N
22. THE v. LoHE	t=.6161, p=.2712	Y	N
23. TLiE v. LoLiE	t=1.249, p=.1107	Y	N
24. TLiE v. TLiF	t=.3831, p=.3522	Y	N
25. THF v. LoHF	t=1.7984, p=.0411	N	N
26. LoHE v. LoHF	t=.7781, p=.2213	N	N
27. TLiF v. LoLiF	t=.7504, p=.2294	N	N
28. LoLiE v. LoLiF	t=.1567, p=.4383	N	N
	Expected = 4/8; Observed = 0/8		

Table 5.5 above shows the means and standard deviations for left erector spinae latency for the eight experimental conditions. The means are grouped in the table according to the major hypothesis. Table 5.6, above, shows the MANOVA test results for the left erector spinae latency. The only significant result was a main effect for movement ($F = 7.51$, $p = .01 < p = .05$). The lift movement showed greater latency than the hold movement.

Table 5.7 shows the results table for LES latency to test the minor hypothesis. No comparisons were found significant.

Table 5.8 shows the means and standard deviations for RES latency. There were no significant effects of any type for right erector as shown in table 5.9 below.

Table 5.8 Mean RES latency (in seconds) for each experimental condition.

Condition	Mean sec.	Stand.Dev.
Loose lifting in extension (LoLiE)	.2197	.1621
Tight lifting in extension (TLiE)	.1605	.1965
Loose lifting in flexion (LoLiF)	.2223	.1303
Tight lifting in flexion (TLiF)	.1677	.1645
Loose holding in extension (LoHE)	.2061	.1495
Tight holding in extension (THE)	.1431	.1515
Loose holding in flexion (LoHF)	.1765	.1677
Tight holding in flexion (THF)	.1335	.1439

Table 5.9 Results table for RES latency (n=32; 16 Tight vs. 16 Loose)

	F value	df	p value
Main Effects			
Hip Type	F=.91	1,30	p=.348
Position	F=.00	1,30	p=.986
Movement	F=1.21	1,30	p=.280
Two Way Interactions			
Hip type by Position	F=.00	1.30	p=.968
Hip type by Movement	F=.03	1,30	p=.854
Movement by Position	F=1.68	1,30	p=.205
Three-way Interaction			
Hip type by Position by Movement	F=.40	1,30	p=.534

Table 5.10 shows the 28 comparisons for RES latency to test the minor hypothesis. This variable had no comparisons that were significantly different.

For the previous dependent variables, (DMOS, LES latency, and RES latency), data were taken on 32 subjects. For the last two variables (LES EAC and RES EAC), data were available on only 26 subjects. Due to a computer failure, data were not available for 3 subjects from each group.

Table 5.10 RES latency results table to test the minor hypothesis.
Significance = p< .0018 (.05/28)

Comparison	t value, p value	Expected	Observed
External Resistance	**Expected = 0/4;Observed = 0/4**		
External Only			
1. THE v. TLiE	t = 2805, p = .3905	N	N
2. THF v. TLiF	t = .6246, p = .2685	N	N
3. LoHE v. LoLiE	t = .2466, p = .4034	N	N
4. LoHF v. LoLiF	t = .8625, p = .1875	N	N
	Expected = 0/4; Observed = 0/4		
Combined Resistance	**Expected = 6/12; Observed = 0/12**		
External/Internal X 2			
5. THE v. LoLiF	t = 1.5858, p = .0616	Y	N
6. TLiE v. LoHF	t = .2481, p = .4029	Y	N
7. THF v. LoLiE	t = 1.5891, p = .0613	N	N
8. LoHE v. TLiF	t = .6910, p = .2474	N	N
	Expected = 2/4; Observed = 0/4		
External/Internal X 1			
9. THE v. LoLiE	t = 1.381, p = .0888	Y	N
10. THE v. TLiF	t = .4398, p = .3316	Y	N
11. THF v. LoLiF	t = 1.829, p = .0387	Y	N
12. LoHE v. LoLiF	t = .3274, p = .3728	Y	N
13. TLiE v. LoHE	t = .7384, p = .2330	N	N
14. TLiE vs. THF	t = .4423, p = .3307	N	N
15. TLiF v. LoHF	t = .1505, p = .4407	N	N
16. LoLiE v. LoHF	t = .7401, p = .2325	N	N
	Expected = 4/8; Observed = 0/8		
Internal Resistance	**Expected = 6/12;Observed = 0/12**		
Internal X 2			
17. THE v. LoHF	t = .5917, p = .2793	Y	N
18. TLiE v. LoLiF	t = 1.0487, p = .1514	Y	N
19. THF v. LoHE	t = .1505, p = .4407	N	N
20. TLiF v LoLiE	t = .9005, p = .1875	N	N
	Expected = 2/4; Observed = 0/4		
Internal X 1			
21. THE vs. THF	t = .1825, p = .4282	Y	N
22. THE v. LoHE	t = 1.1837, p = .1229	Y	N
23. TLiE v. LoLiE	t = .9292, p = .1801	Y	N
24. TLiE v. TLiF	t = .1222, p = .4557	Y	N
25. THF v. LoHF	t = 1.7777, p = .2214	N	N
26. LoHE v. LoHF	t = .5263, p = .3013	N	N
27. TLiF v. LoLiF	t = 1.0414, p = .1530	N	N
28. LoLiE v. LoLiF	t = .0506, p = .4800	N	N
	Expected = 4/8;Observed = 0/8		

Table 5.11 below shows the means and standard deviations for the left erector spinae early amplitude change (LES EAC) variable. Values are in percent maximum voluntary contraction.

Table 5.11 Mean LES early amplitude change (EAC) for each experimental condition. Values in percent MVC for .5 seconds (including .1second just prior to and .4 second after muscle onset)

Condition	Mean %change	Stand.Dev.
Loose lifting in extension (LoLiE)	13.9	9.3
Tight lifting in extension (TLiE)	13.8	12.1
Loose lifting in flexion (LoLiF)	10.2	8.1
Tight lifting in flexion (TLiF)	10.9	7.4
Loose holding in extension (LoHE)	19.7	12.5
Tight holding in extension (THE)	15.1	9.4
Loose holding in flexion (LoHF)	20.6	16.9
Tight holding in flexion (THF)	16.2	10.1

Table 5.12 below shows the statistical test results for the left erector spinae early amplitude change (LES EAC) variable. For this variable, the two-way interaction movement by position was significant ($F = 6.77$, $p = .016 < p. = .05$).

Table 5.13 shows the means and standard deviations for the two-way movement by position conditions for LES EAC found to be significant. Figure 5.3 is a graph of this significant two way interaction.

Table 5.12 Results table for LES EAC (n=26; 13 Tight vs. 13 Loose)

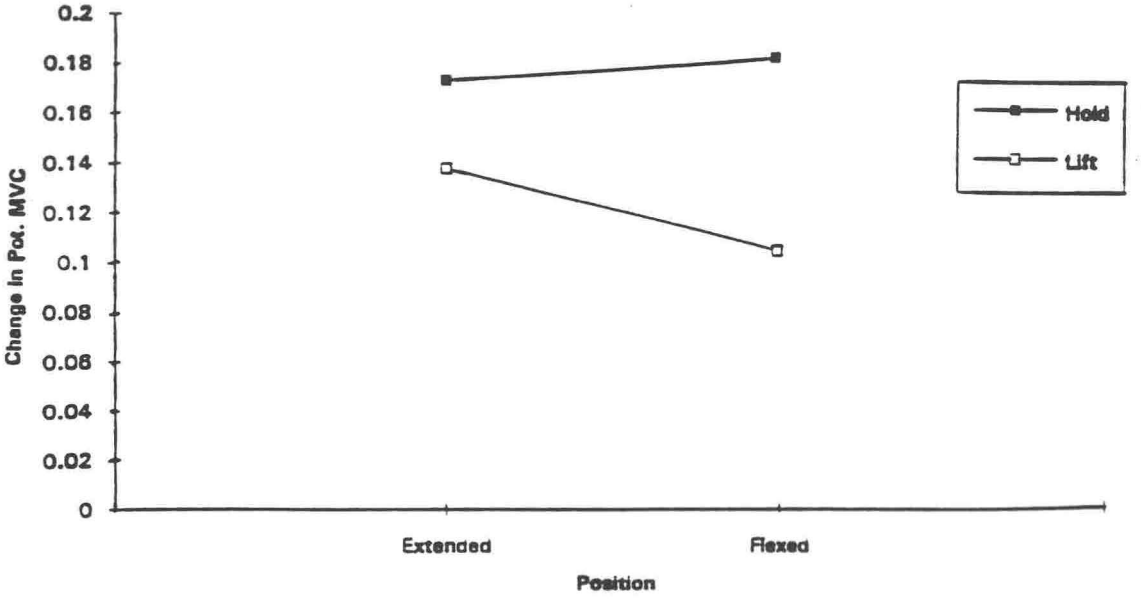
	F value	df	p value
Main Effects			
Hip type	F=0.00	1,26	p=.955
Position	F=3.15	1,26	p=.088
Movement	**F=18.72***	**1,26**	**p=.000**
Two-Way Interactions			
Hip type by Position	F=0.00	1,26	p=.974
Movement by Position	**F=6.77***	**1,26**	**p=.016**
Hip type by Movement	F=3.00	1,26	p=.096
Three-way Interaction			
Hip type by Position by Movement	F=.08	1,26	p=.784

Three follow up t-tests were conducted to analyze this two-way movement by position interaction. Accordingly, the tests were performed at .017 level of significance (.05/3 = .017). The results are shown in table 5.14. The first two tests were conducted because they are related to the major hypothesis. In these comparisons the extended vs. flexed difference was tested for the lift movement. The same test was conducted for the hold movement. Neither of these comparisons were significant. Therefore these

Table 5.13 Mean and standard deviation LES EAC. Values in percent MVC for .5 seconds (including .1second just prior to and .4 second after muscle onset) Two-way movement by position comparisons regardless of hip type.

Condition	Mean %change	SD
Lifting in Extended	13.8	10.5
Lifting in Flexed	10.5	7.6
Holding in Extended	17.4	11.1
Holding in Flexed	18.4	13.8

Figure 5.3 LES EAC Two-way movement by position comparisons. Lift extended vs. hold extended and lift flexed vs. hold flexed.



comparisons did not yield any information that was relevant to the major hypothesis.

A significant main effect for movement was also found ($F = 18.72$, $p = .000 < p = .05$). Since a two-way interaction for movement by position was found, it had to be decided whether it was appropriate to interpret this main effect. To do so, it had to be determined whether all two-way regardless of position, were significant. If so, it would be appropriate to interpret the main effect.

It was decided to test the lift extended vs. hold extended comparison to make the decision (see table 5.13 and figure 5.3 above). The means for these two conditions showed the smallest difference. This test was conducted and found to be not significant ($t = 1.189$, $p = .1201$). Therefore,

Table 5.14 Tests of significance to interpret the LES EAC movement by position interaction. (Tests conducted at the .017 level)

Tests to interpret the major hypothesis

Comparison	t value	df	p value
LE vs. LF	1.294	50	.101
HE vs. HF	0.279	50	.391

Test to decide regarding interpreting the main effect

HE vs. LiE	$t = 1.189$	50	.120

*significant at the .017 level

Table 5.15 LES EAC results table for testing the minor hypothesis.
significance = p< .0018 (.05/28)

Comparison	t value, p value		
External Resistance	**Expected = 0/4;Observed = 0/14**		
		Expected	Observed
External Only			
1. THE v. TLiE	t = .3279, p = .3729	N	N
2. THF v. TLiF	t = 1.5221, p = .0705	N	N
3. LoHE v. LoLiE	t = 1.3294, p = .0981	N	N
4. LoHF v. LoLiF	t = 1.9967, p = .0287	N	N
	Expected = 0/4; Observed = 0/4		
Combined Resistance	**Expected = 6/12;Observed = 0/12**		
External/Internal X 2			
5. THE v. LoLiF	t = 1.4439, p = .0809	Y	N
6. TLiE v. LoHF	t = 1.1836, p = .1241	Y	N
7. THF v. LoLiE	t = .5904, p = .2802	N	N
8. LoHE v. TLiF	t = 2.1755, p = .0198	N	N
	Expected = 2/4; Observed = 0/4		
External/Internal X 1			
9. THE v. LoLiE	t = .3283, p = .3728	Y	N
10. THE v. TLiF	t = 1.2755 p = .1072	Y	N
11. THF v. LoLiF	t = 1.6775, p = .0532	Y	N
12. LoHE v. LoLiF	t = 2.2997, p = .0152	Y	N
13. TLiE v. LoHE	t = 1.0474, p = .1527	N	N
14. TLiE vs. THF	t = .5676, p = .2912	N	N
15. TLiF v. LoHF	t = 1.8844, p = .3589	N	N
16. LoLiE v. LoHF	t = 1.2391, p = .1136	N	N
	Expected = 4/8; Observed = 0/8		
Internal Resistance	**Expected = 6/12;Observed = 0/12**		
Internal X 2			
17. THE v. LoHF	t = 1.0126 p = .1607	Y	N
18. TLiE v. LoLiF	t = .8875, p = .1918	Y	N
19. THF v. LoHE	t = .7880, p = .2192	N	N
20. TLiF v LoLiE	t = .919, p = .1836	N	N
	Expected = 2/4; Observed = 0/4		
Internal X 1			
21. THE vs. THF	t = .2722, p = .3939	Y	N
22. THE v. LoHE	t = 1.1837, p = .1229	Y	N
23. TLiE v. LoLiE	t = .0446, p = .4824	Y	N
24. TLiE v. TLiF	t = .7234, p = .2382	Y	N
25. THF v. LoHF	t = .8062, p = .2140	N	N
26. LoHE v. LoHF	t = .1545 p = .4392	N	N
27. TLiF v. LoLiF	t = .2420, p = .4054	N	N
28. LoLiE v. LoLiF	t = 1.1012, p = .1409	N	N
	Expected = 4/8;Observed = 0/8		

it was inappropriate to interpret the main effect for movement as significant since it can not be said that in all cases lift is significantly different from hold.

Table 5.15 above shows the 28 comparisons to test the minor hypothesis for LES early amplitude change. This variable had no comparisons that were found significant.

Table 5.16, below, shows the means and standard deviations for the movement by position early amplitude change for the right erector spinae muscle variable.

Table 5.16 Mean RES early amplitude change (EAC). Values in percent MVC for .5 seconds (including .1second just prior to and .4 second after muscle onset) .

Condition	Mean %change	Stand.Dev.
Loose lifting in extension (LoLiE)	13.5	7.4
Tight lifting in extension (TLiE)	9.3	4.7
Loose lifting in flexion (LoLiF)	9.7	6.1
Tight lifting in flexion (TLiF)	7.3	3.1
Loose holding in extension (LoHE)	22.9	13.8
Tight holding in extension (THE)	21.0	8.9
Loose holding in flexion (LoHF)	24.7	15.5
Tight holding in flexion (THF)	18.8	7.9

Table 5.17, below, shows the statistical results for this variable. A main effect of movement was found ($F = 35.10$, $p = .000 < p = .05$). EAC was found to be significantly greater for holding (21.7%) compared to lifting (9.95%). There were no other significant results for this variable.

Table 5.17 Results Table for RES EAC (n = 26; 13 Tight vs. 13 Loose)

	F value	df	p value
Main Effects			
Hip type	$F = 1.67$	1,26	$p = .209$
Position	$F = 3.02$	1,26	$p = 0.095$
Movement	**$F = 35.10$***	**1,26**	**$p = .000$**
Two-way Interactions			
Hip type by Position	$F = 0.40$	1,26	$p = .531$
Movement by Position	$F = 2.39$	1,26	$p = .135$
Hip type by Movement	$F = .02$	1,26	$p = .888$
Three-way Interaction			
Hip type by Position by Movement	$F = 2.68$	1,26	$p = .115$

Table 5.18 shows the results table for testing the minor hypothesis for the RES EAC variable. Ten comparisons were significant. All of these were external comparisons. Only three of the ten had weighting differences

Table 5.18 RES EAC results table for testing the minor hypothesis.
Significance = p< .0018 (.05/28)

Comparison	t value, p value	Expected	Observed
External Resistance	**Expected = 0/4;Observed = 3/4**		
External only			
1. THE v. TLiE	t = 4.1882, p = .00016	N	Y
2. THF v. TLiF	t = 4.9257, p = .00002	N	Y
3. LoHE v. LoLiE	t = 4.0131, p = .0003	N	Y
4. LoHF v. LoLiF	t = 3.1065, p = .0024	N	N
		Expected = 0/4; Observed = 3/4	
Combined Resistance	**Expected = 6/12;Observed = 7/12**		
External/ Internal X 2			
5. THE v. LoLiF	t = 3.7346, p = .0005	Y	Y
6. TLiE v. LoHF	t = 3.3121, p = .0015	Y	Y
7. THF v. LoLiE	t = .919, p = .1836	N	N
8. LoHE v. TLiF	t = 3.2564, p = .0017	N	Y
		Expected = 2/4; Observed = 3/4	
External/ Internal X 1			
9. THE v. LoLiE	t = 1.5617, p = .0657	Y	N
10. THE v. TLiF	t = 5.2466, p = .00001	Y	Y
11. THF v. LoLiF	t = 1.3897, p = .0887	Y	N
12. LoHE v. LoLiF	t = 3.1518, p = .0022	Y	N
13. TLiE v. LoHE	t = 3.3934, p = .0012	N	Y
14. TLiE v. THF	t = 3.7438, p = .0005	N	Y
15. TLiF v. LoHF	t = 3.8817, p = .0004	N	Y
16. LoLiE v. LoHF	t = 1.7557, p = .0460	N	N
		Expected = 4/8; Observed = 4/8	
Internal Resistance	**Expected = 6/12;Observed = 0/12**		
Internal X 2			
17. THE v. LoHF	t = .6628, p = .2569	Y	N
18. TLiE v. LoLiF	t = .2674, p = .3957	Y	N
19. THF v. LoHE	t = .9683, p = .1713	N	N
20. TLiF v LoLiE	t = 2.391, p = .0042	N	N
		Expected = 2/4; Observed = 0/4	
Internal X 1			
21. THE v. THF	t = .6711, p = .2543	Y	N
22. THE v. LoHE	t = .4520, p = .3277	Y	N
23. TLiE v. LoLiE	t = 2.2367, p = .0174	Y	N
24. THF v. LoHF	t = 1.1370, p = .1334	N	N
25. LoHE v. LoHF	t = .2149 p = .4158	N	N
26. TLiF v. LoLiF	t = .2420, p = .4054	N	N
27. TLiE v. TLiF	t = 1.3100, p = .1013	N	N
28. LoLiE v. LoLiF	t = 1.9056, p = .0344	N	N
		Expected = 4/8;Observed = 0/8	

of 2 or greater and were expected to be significant. Seven were comparison with less than 2 difference and were not expected to be significant. Three were external only comparison whereas none of these comparisons were expected to be significant. Three were external/internal X 2 comparisons and three were external/internal X 1 comparisons. None of the internal comparisons were significant.

CHAPTER 6

DISCUSSION

DMOS and the major hypothesis

The major hypotheses that DMOS would show a two-way hip type by position interaction was not supported. It was expected that there is a difference between tight and loose in the extended position but not in the flexed position and that pattern is the same for both lift and hold. Instead, a three-way interaction was found for DMOS. Follow-ups show no difference between loose and tight groups in the extended position but a difference in the flexed position for the lift movement. The pattern shown for the lift movement is opposite the hypothesis.

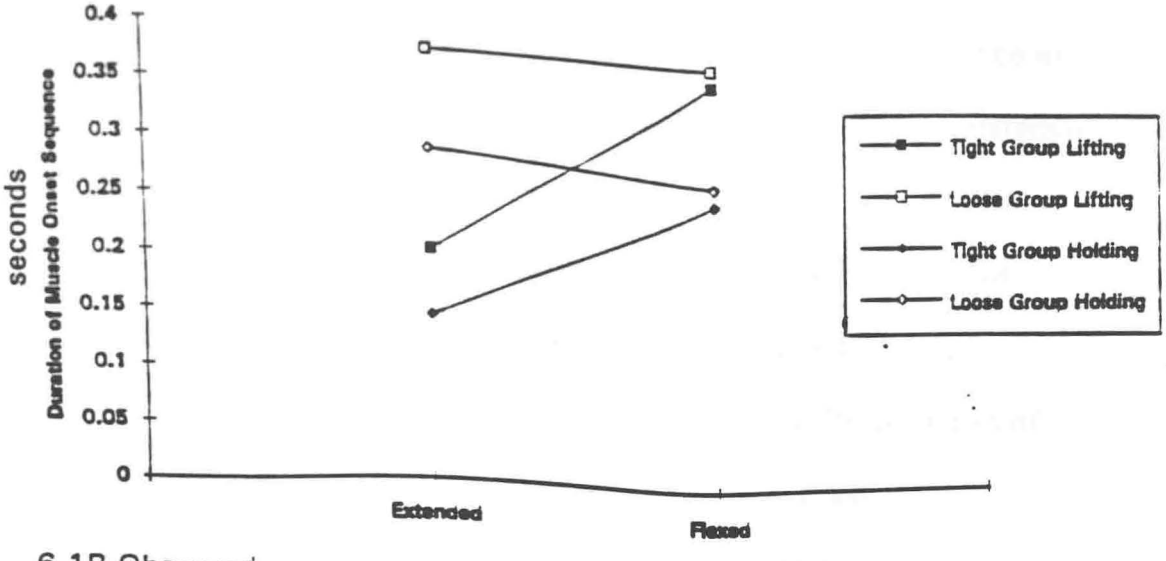
The loose-tight difference was found for the lift movement only. No significant differences in either extended or flexed position were found for the hold movement. In other words the tight-loose relationship was different for the two types of movement, instead of the same as was hypothesized. A comparison between the hypothesized differences and the observed differences are shown in figure 6.1 below.

This study was designed to take advantage of the assumption that manipulation of position would highlight already-existing differences between

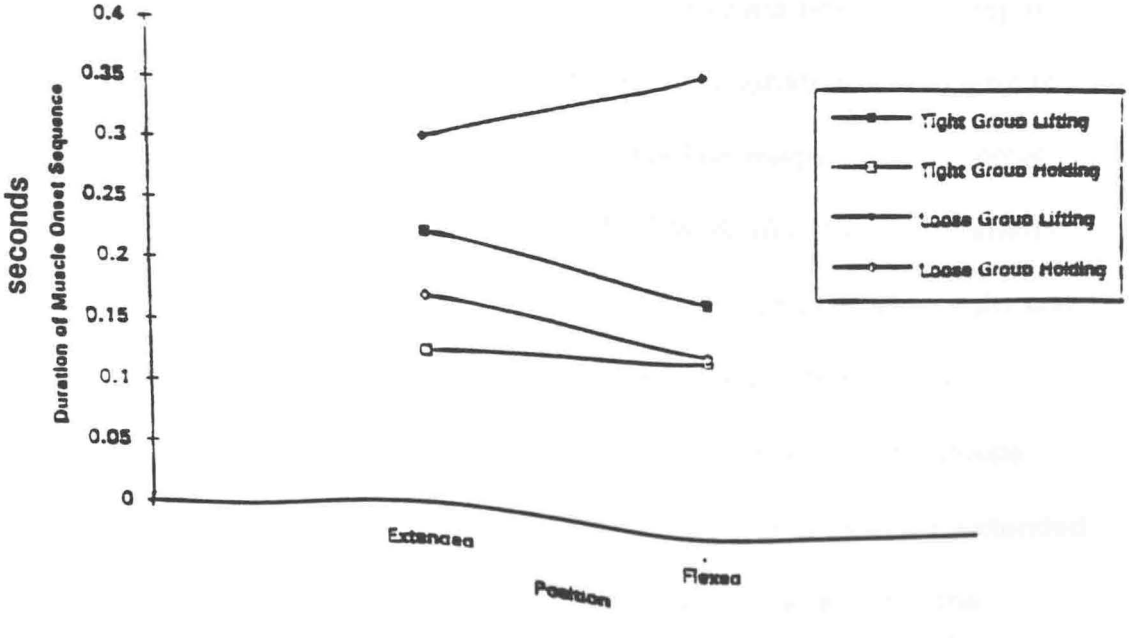
tight and loose subjects. Based on pilot study, an inverse relationship was expected between DMOS and internal resistance.

Figure 6.1 Hypothesized vs. Observed differences for DMOS

6.1A Hypothesized



6.1B Observed



Therefore, a decrease in DMOS was expected with greater internal resistance. This lead to the hypothesis that DMOS would be greater for the hip loose group in the extended position with no difference in the flexed position. However, a more general statement of the hypothesis is that there is a difference between tight and loose groups in one position and not in the other. In other words, the subjects were expected to show a difference in one position and not in the other position because of a "different difference" in internal resistance when subjects were compared in the two conditions. Therefore, any loose-tight difference observed for one position and not observed in the other could be taken as support for the notion that differences in muscle behaviors between subjects with different types of muscle imbalance might be due to differences in internal resistance.

The results for DMOS clearly suggest that if muscles in general are responding to differences in internal resistance, they are not responding in the manner hypothesized. There is an alternative explanation that seems to fit both the hypothesis that differences are based on magnitude of internal resistance and a inverse relationship between DMOS and internal resistance. It could be that internal resistance suppresses differences between tight and loose groups. It may be that the increased resistance in the extended position compelled muscles to onset more synchronously for both groups. Therefore, DMOS would tend to be the same for both groups in the extended position. In the flexed position, the loose group was released from the

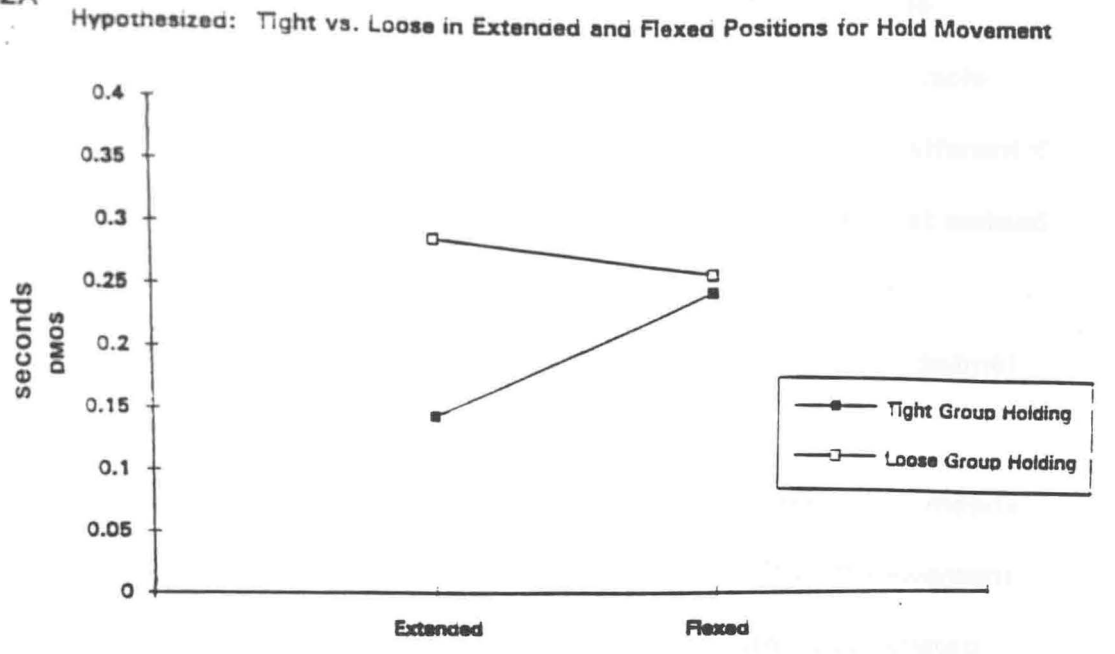
restriction to onset muscles synchronously. However, the tight group may be restricted in response in both extended and flexed position.

A principal part of the experimental design was the inclusion of two types of hip movement, i.e., lift and hold movements. The reason was to observe the extent to which changes in muscle behaviors are related to distinct types of imbalance and the extent to which those differences might be the same for the different movements. Visual inspection of figure 6.1B and table 5.3 reveals a possibly interesting pattern related to the comparison of results for the two movements.

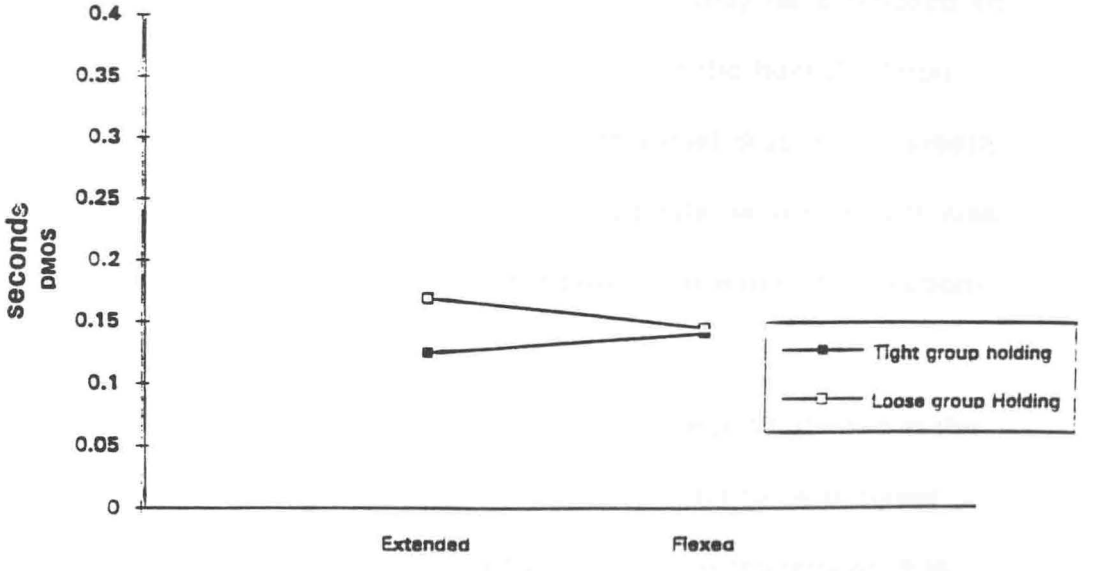
In the extended position the t-test for loose lift extend vs. tight lift extend was nearly significant ($t = 1.793$, $df = 30$, $p = .0415$; see table 5.3). Also in the extended position the loose hold extend vs. tight hold extend means were also nearly significant ($t = 1.762$, $df = 30$, $p = .0442$). Both would have been significant if tested at the $p = .05$ level. The interesting observation is that for the lift movement the means diverge with change from the extended to flexed position. For the hold movement the means seem to converge from extended and flexed. They behaved more as hypothesized (a difference expected in extended and not for flexed). If this were true, it leads to a slightly different interpretation of the findings.The comparison between hypothesized and observed is shown in figure 6.2 below.

Figure 6.2. Hypothesized vs. observed DMOS for hold movement

6.2A



6.2B



The three-way interaction found seems to lead to an interpretation that long-term difference in muscle imbalance causes distinct muscle behaviors for groups for voluntary movement but does not affect muscle behaviors for involuntary movements. The interpretation would be different if the findings for hold are considered significant. This would mean that instead of a distinct muscle behavior of loose and tight groups for voluntary movement and no difference for hold, the interpretation would be a distinct type of muscle behavior of groups for each type of movement.

One must be careful in this interpretation because none of the means for the hold movement are actually significant as tested. The lift movement is one in which the subject chose the time when movement was initiated. Subjects were instructed to track their movement visually by watching in a mirror and time the movement to a metronome. This may have included an element of skill that was not in the hold condition. In the hold condition subjects were not able to see when the platform would drop from beneath the leg. The drop time was variable (3, 5 or 7 seconds) so the subject was uncertain when it would occur. The subject performed more of a reaction-type movement in these conditions.

Pre-movement muscle behavior is said to be pre-programmed in the pre-motor and supplementary motor area. There are said to be different motor programs and sub-programs for different kinds of movement. It is conceivable the subjects may have developed different motor programs for

the two kinds of movement. The two types of long-standing muscle imbalance could have altered the two motor programs differently.Thus in the lift conditions the loose-tight difference was greater in the flexed position. In the hold conditions the loose-tight difference was greater in the extended position. However, a strict statistical interpretation is that the lift movement is a useful way to contrast loose and tight groups (and possibly, the effects of long-standing differences in muscle imbalance) while the hold movement is not.

Table 5.2 shows that there was a two-way movement by hip type interaction. When there is a three-way interaction it is usually, but not always, inappropriate to interpret any two-way interaction. Follow-up comparisons showed it was not appropriate to interpret this two-way interaction. These results probably simply confirm the fingings of the three-way interaction that there is a loose-tight difference for lift but not for hold.

Unlike the other dependent variables used in this study, DMOS was not specific to the erector spinae muscles. It was included to show any generalized effect of internal resistance on behavior of all the muscles involved in simple hip extension. The research strategy was to first see if there was an overall difference between tight and loose hip subjects in the timing behavior of stabilizing and prime muscles involved with hip extensor muscles. If an overall timing difference was found, this study was designed

to investigate how much difference in erector spinae latency has to do with the duration of muscle onset sequence.

Latency and the major hypothesis

This variable was included to test one part of the Janda postulation regarding hyperactivity of the erector spinae in the presence of tight hip flexors. One part of hyperactivity, it was deduced from Janda's writings and presentations, was muscle activity that is inappropriately early. This translated to the possibility of decreased latency. Both left and right latency were studied because the possibility existed that the two muscle groups could behave differently when only the right leg was lifted or held.

The hypothesis that tight hip subjects would show significantly shorter latency in the extended position but no difference in the flexed position for both lift and hold movements was not supported for both LES and RES latency. Instead a main effect for movement was shown for LES latency. No significant differences were observed for RES latency.

Latency was expected to be different for the hip tight subjects, especially in the extended position. The hip tight subjects had a much greater pelvic inclination measurement in prone lying. This meant that their lordotic curve would likely be greater. Increased lordosis would cause the erector spinae muscles to be more slack than for the hip loose subjects.

EMD studies have shown that a muscle's ability to generate force is always a product of its length (stretch force) and its contractile force (the result of muscle fibers shortening). Moreover, one of these elements may compensate for the other, that is, one can remain unchanged while the other is increased to compensate. Therefore, when there is increased resistance to a muscle's action or increased slack in the muscle, latency does not necessarily change. Latency may stay the same when there is increased resistance or when the muscle is slack if there is a simultaneous increase in amplitude. Therefore, no difference in latency across different conditions alone is not necessarily evidence that the research hypothesis is not supported. It could simply mean that hyperactivity of erector spinae muscles in hip tight subjects may be inappropriate amplitude only but not necessarily early inappropriate activity (decreased latency). However, if there are no changes in amplitude as well, the idea that persons with anterior hip tightness develop an increased excitability in erector spinae muscles that can be detected with simple prone hip extension should be questioned. This will be seen later when the early amplitude change variable, EAC, is considered.

This study set out to find if there is a difference in muscle behaviors between subjects with distinct types of muscle imbalance and to what extent those differences reflected hyperexcitability of erector spinae muscles for the tight group. The DMOS results seem to show there is a difference in muscle behaviors. The LES and RES latency results seem to suggest muscle

behavior differences reflected by DMOS have nothing to do with the timing of the erector spinae.

However, the timing of one or both of the other two muscles had to be responsible for the tight-loose DMOS differences with change in position. Bullock-Saxton was able to distinguished subjects with and without ankle pathology using DMOS for the same four muscles as this study. Her subjects performed a movement similar to the lift in extended movement in this study. She found that LES and RES latencies did not discriminate subjects with pathology and normal subjects, but gluteus maximus latencies did.[98]

Since latencies for all four muscles used in this study were available, it was tempting to see if a muscle other than the erector spinae discriminated between experimental conditions. Hamstring was randomly chosen. Two t-tests were performed comparing tight and loose in the extended position and tight and loose in the flexed position. Findings were similar to those for the DMOS variable. There was no tight-loose differences for the extended position but a tight-loose difference for lifting in the flexed position, (TLiF vs LoLiF). A more complete analysis of which muscle or muscles may be responsible for the DMOS difference must await another study. However, these results suggest that the hamstring latency is at least one of the muscles responsible for the difference in DMOS observed rather than the latency of erector spinae.

Early amplitude change and the major hypothesis

Erector spinae muscles are assumed to hypertrophy in persons with tight anterior hip structures. It is accepted that a major reason for this hypertrophy is the increased mechanical demand during standing and walking. The normal response of muscles to increased resistance applied over time is hypertrophy. Normally muscle hypertrophy results from muscles overcoming greater resistance. There is usually a direct relationship between a muscle meeting greater resistance and a greater magnitude of EMG amplitude from that muscle. For this study, it was assumed that muscles that are overcoming greater resistance would show behaviors suggesting they were overcoming greater resistance. This is an important assumption because there is another assumption that could be made. The alternative assumption is that muscles that hypertrophy in response to increased resistance over time (after hypertrophy is developed) may show no increase in amplitude when overcoming the same resistance. With increase muscle mass, the muscles may develop the ability to overcome the resistance with less relative contractile force. However, Janda suggests that muscles that appear to be hypertrophied show hyperexcitability. He studied this phenomenon only by looking at muscle timing. EMD studies have demonstrated that muscles meet increased resistance by different combinations of amplitude and latency. If hyperexcitability is due to increased internal resistance muscles behaviors should show some changes

in combined amplitude and latency. It has already been shown that there are no latency changes with changes in internal resistance. It could be that amplitude changes may be the only alterations that would explain Janda's observation of hyperexcitability of erector spinae muscles in subjects with tight anterior hip joint structures during the prone hip extension test.

The hypothesis that subjects with tight anterior hips would show greater EAC in the extended position but no difference in the flexed position for both lift and hold movements was not supported. Instead a two-way movement by position interaction was found for LES EAC. A main effect for movement was found for RES EAC. More significant to the main question of the study was that the two-way hip type by position interaction was not supported for any of the latency or amplitude measures of erector spinae behavior.

That no support for erector spinae hyperactivity in the pre-movement muscle behaviors EAC and latency may be due to either an improperly designed experiment or a faulty concept. On the experiment side, it may have been that the pre-movement period may have not been the correct period from which to sample. This period was chosen to more definitely capture pre-programmed muscle activity. It is well-known that there is a delay between formation of motor commands and their execution. It could be that the execution of the pre-programmed motor commands for amplitude occur later in the movement period. If this is true it would agree with Ross,

et. al. who found that with greater external resistance peak EMG activity occurred later in the movement period.[107]

On the other hand it could be that the idea that early inappropriate activity, i.e., hyperactivity, develops in subjects with tight anterior hip structures may be erroneous. If hypertrophy of erector spinae does develop in persons with tight hip flexors, it could be entirely an appropriate response to mechanical stimuli from erect stance and gait as Kendall and Kendall have suggested.

The Minor Hypothesis

Position and hip type varied the tightness of the anterior hip structures, an internal source of resistance. A pilot study showed that the hold movement had a requirement for a greater velocity of contraction. This indicated that the same weight (the weight of the right lower extremity) was accelerated more quickly downward in the hold condition. Thus the source of differences for the movement variable was considered to be external resistance with hold greater and lift less. It was possible to contrast conditions which differed only on internal resistance, which differed only on external resistance and which had combinations of internal and external resistance. The experimental hypothesis is that there is no difference between sources of resistance.

T -tests were conducted on all combinations of the eight experimental conditions. The significance level was $p < .0018$ level. Comparisons where

the internal and/or external resistance weighting of the two conditions differed by 2 or greater would be significant. Any comparisons where the internal/external resistance weighting for the two conditions was less than 2 would not be significant. The statistical hypothesis had two parts. First was that only comparisons with weighting of 2 or greater would be significant. Second was that this would be found regardless or whether the source of resistance was external, internal or a combination of internal and external. The test of the hypothesis was inspection of charts showing results of the 28 possible comparisons and number of agreements and disagreements with the hypothesis.

DMOS and the minor hypothesis

Table 5.4 shows that the hypothesis that comparison with a weighting difference of 2 or more would be significant and comparisons with a weighting difference of 1 or less would not be significant regardless of resistance source is not supported. Agreements and disagreements with the hypothesis were evenly split with 14 cases agreeing with the hypothesis and 14 in disagreement. By any statistical measures, a 50-50 result does not mean support for the hypothesis.

Ten of the 28 comparisons were found significant at the $p = .0018$ level. Only 4 were in agreement with the hypothesis. The other six cases were comparisons that were not expected to be significant but were found significant. An interesting pattern seemed to exist when an analysis is done

of what comparisons were significant. Of the ten significant comparison, nine were defined as having a difference in external resistance. Only one significant comparison was an internal pairing. This seems to indicate that external resistance had a much greater effect on muscle behaviors than internal resistance. The major reason for differences between conditions was movement rather than the hypothesized weightings on internal resistance.

One interpretation of these findings is that there is a difference between the response to internal and external resistance. To make this conclusion, one must assume that the magnitude of resistance of internal and external resistance was similar. For example the difference in magnitude of resistance between lift and hold must be assumed to be similar to the difference in magnitude between tight and loose or flexed and extended. If so, the greater response to lift versus hold only is support for the hypothesis that there is a greater response to external resistance than internal resistance. This could be expanded to the hypothesis that sole reason for hypertrophy of erector spinae muscles when hip flexors are tight may be external mechanical factors instead of the external resistance plus internal resistance.

Another interpretation is that the difference in resistance magnitude for lift and hold was much greater than the difference in magnitude between loose and tight or flexed and extended. Arguing against this point is the fact that comparison where there was a difference in two sources of internal

resistance were no different from those which differed on the basis of one source on internal resistance. On the other hand, there could have been an internal signal which dampened the effects internal resistance only. However, if this were so, there probably would have been a pattern indicating certain results for comparisons where there was a mixture between internal and external resistance and comparisons where the difference was only internal or external resistance. The strongest hypothesis is that muscle behaviors seem to alter with changes in external resistance and do not seem to alter with changes in internal resistance.

DMOS and the directional hypothesis

An examination of the last column of tables 5.1 and 5.4 seems to support the directional hypothesis that there is an inverse relationship between DMOS and resistance magnitude. In 9 of the 10 conditions found significant the condition with the greater resistance weighting had the shorter DMOS.

The inverse relationship between DMOS and internal resistance agrees with Ross, et. al., who studied the onset sequence of muscles (back extensors and abdominals) and found the duration decreased with each increase in level of external resistance. This inverse relationship held even when the resistance level changed from nominal to 30%. Other muscle performance variables such as amplitude sometimes did not change

significantly with percent increases in maximal voluntary contraction while DMOS always did.[107]

The pilot study contrasting 5 normal subjects with 7 hip-tight subjects also seemed to support an inverse relationship between magnitude of internal resistance and DMOS. In conditions where internal resistance was hypothesized to be high, duration of sequence was low and when internal resistance was hypothesized to be low, duration was greater.

Another indication of the inverse relationship between DMOS and hypothesized internal resistance comes from inspecting figure 5.1. The values appear to be stacked for both extended and flexed with tight holding, loose holding, tight lifting and loose lifting in successively greater order. The level weightings for each of these conditions is 6, 5, 5, and 4 respectively for the extended condition and 5, 4, 4, 3 for the hold condition. These conditions would not be expected to take this sequential order unless something like response to increased resistance were not operating.

Latency and the minor hypothesis

It was expected that LES and/or RES latency would show 12 of 28 comparisons with a weighting difference of 2 or greater would be significant. The remaining 16 comparisons with a weighting difference of less than 2 would not be significant. Significant differences are independent of type of resistance force. For both LES latency and RES latency the hypothesis was

not supported. None of the comparisons expected to show significance for either LES or RES occurred. This is shown in tables 5.7 and 5.10.

It was not entirely unexpected that the latency might not be different with change in position or hip type. The pelvis was expected to change its tilt with different conditions. In the extended position the pelvis was expected to be forwardly tilted especially for the extended position. In the flexed position the pelvis was expected to be tilted more posteriorly. With pelvic tilt the length of the these muscles was expected to change. In the flexed position the muscles were expected to be slightly longer. For the hip tight subjects the muscles might be more elongated because they may have developed adaptive shortening of these muscles. In the extended position, the erector spinae muscles were expected to be slightly more slack especially for the hip tight subjects. There were actually several possibilities for latency behaviors. This depended upon the amplitude behavior of the muscles.

EMD studies show that latency need not change with increased resistance. A muscle on slack and a muscle under stretch may show the same latency. However, the slack muscle usually has a greater amplitude if it shows the same latency as the elongated muscle. Therefore, it is possible that the latency could be unchanged in all experimental conditions. However, the amplitude behaviors of the muscles would be expected to show the requisite compensatory behavior.

It was expected that when the movement factor was added the difference in resistance in some of the comparisons might make some significant. The lift movement had a requirement for a slower speed of contraction. EMD studies have shown that eccentric contractions often have a shorter latency than concentric movements. It was expected that the hold comparisons might have a shorter latency. This is clearly not supported. The results add support to the hypothesis that if tight subjects have hyperexcitability of the erector spinae, it is not reflected by the latency of these muscles. The reason for apparent muscle hypertrophy seen clinically may be solely due to the mechanical demands of stance and walking.

Early amplitude change and the minor hypothesis

It was hypothesized that for LES and RES EAC the 12 of 28 comparisons with weighting differences of 2 or more would be significant. Comparisons with a weighting difference of 1 or less would not be significant. These comparisons would be significant or not significant regardless of type of resistance.

The results for LES EAC are shown in Table 5.13. None of the comparisons were significant for this variable. There are no amplitude differences even when the movement factor is included in comparisons. Coupled with the no difference in latency found for the same muscle, this

seems to reinforce the idea that hyperactivity differences between loose and tight subjects does not exist.

The results of the 28 comparisons for RES EAC are shown in Table 5.16. Ten of the 28 comparisons were significant. There were only three agreements with the hypothesis that comparisons with a weighting difference of 2 or greater would be significant. There were 8 disagreements with the same hypothesis. There were 12 disagreements with the second part of the hypothesis that comparisons with a weighting difference of less than 2 would not be significant. The hypothesis that comparisons are significant or non-significant regardless of type of resistance does not seem supported.

All ten of the significant comparison were classified as external of some type. Three of the ten were external only. Another three were external with two sources of internal resistance. The remaining four were external with one source of internal resistance difference. This seems to indicate that the source of external resistance was an overriding factor. The addition or subtraction of internal resistance seemed to have no effect on the likelihood of finding a significant comparison. When there was no source of external resistance, comparisons were not likely to be different. This seems to lead to the same conclusions as found with DMOS. Muscle behaviors seemed to be preferentially stimulated to perform differently based on external rather than internal resistance. This assumes the magnitude of resistance was similar for

internal and external sources. On the other hand the difference in magnitude of resistance may have been great for external and not different enough between internal conditions.

RES EAC and the directional hypothesis

A conclusion as to whether the idea that with greater resistance early amplitude change was greater can be arrived at by looking at table 5.14 and table 5.16. In only 6 of the 10 comparisons found significant did the condition with the greater weighting on internal/external resistance have the greater EAC. This number is considerably less than expected. It was assumed that when muscles overcome greater resistance, the contractile activity would be greater. The next factor after magnitude of resistance one would consider to explain why there was not an exact direct relationship between magnitude of resistance and magnitude of EAC would be muscle length. In exactly half of the significant comparisons was the position different. This indicates that muscle length is not a critical factor. The same 50-50 result came from looking at comparisons for lift and hold. This indicates that hip type was not the determinant of significance. It seems to suggest that there might have been a combination of when the EMG recordings were taken and a magnitude of difference on the basis of external resistance. The pre-movement period may have been a time when amplitude of muscle recruitment is most inconsistent. In addition to this, the difference

in resistance (same magnitude of resistance with greater acceleration) may not have been sufficient to make for a consistent relationship between lift and hold conditions.

Perhaps an early amplitude change difference based on the resistance levels used in this study should not have been expected. Ross, et. al., showed no significant difference in back extensor amplitude when the resistance level changes were as great as 20% MVC. In that study, it could have been that with the long effort arm of RA muscles relative to the effort arm of the erector spinae, the RA could actually elongate the erector spinae muscles prior to or simultaneous with their contraction. This would increase the effectiveness of the contractile portion of the extensor muscles relative to the resistance. EMD studies show that a muscle may be able to maintain its effectiveness against an increased resistance by elongation of the elastic portion and exerting the same effort with the contractile portion the same. This would explain why RA amplitude increased or stayed the same with greater levels of resistance to a back extension movement when it was expected its amplitude would decline. Therefore, it may explain why significant differences in back extensor EMG amplitudes were often not found with the increase in resistance of 20%.

The hamstring could have been performing the same function as in the Ross study. It could have elongated the back extensor more or less depending on its position. This would tend to keep the EAC of back

extensors the same. It may have been better to analyze all the muscles involved in the movement, as was done in the Ross study, rather than concentrating on the back extensor muscles.

A second reason could have been when the amplitude readings were taken. In this study, interest was in the pre-movement period. It was reasoned that the events in the pre-movement period give the best picture of any neurological programming that might have taken place because of long-standing differences in internal resistance due to hip type. The Ross study shows that with each increase in resistance level, the peak amplitude occurs later. By concentrating on the pre-movement recruitment rather that recruitment after movement had begun, this study may have missed the most important time for distinguishing between conditions on the basis of level of resistance.

The subjects were deliberately chosen to be as different as possible and still have a sample large enough for statistical power. In total they were at least 3 SD apart on their hip tightness measures. One would think that if internal resistance were a strong influence, DMOS (the seemingly most sensitive variable) would show a more consistent results.

The dependent variable measures (ability to discriminate)

DMOS and EAC seemed to be the most sensitive indicators in this study. These variables seemed to differentiate the conditions in the most

consistent manner. Differences in resistance conditions may not have great enough to make changes in latency. This suggests DMOS and EAC could be more sensitive measures when difference in resistance between conditions are small.

DMOS

Bullock-Saxton has used the DMOS variable to differentiate between normal and pathological groups. In that study latency did not differentiate groups while DMOS seemed to.[98] DMOS may have promise as a treatment outcome measure where a change in internal resistance is expected. For example, DMOS could be measured before and after stretching exercises to judge their effectiveness.

Erector spinae latency

LES and RES latency showed no ability to discriminate between conditions. This confirms what has been found in other studies. Norman and Komi studied the latencies of the triceps and biceps. As in this study, they altered the starting position so that both the triceps and biceps were at different lengths. They found no differences in latency based on starting position.[111] Bullock-Saxton found no significant differences between LES when right leg was lifted and LES when left leg was lifted. The same was

found for RES. No differences were found for both normal and injured subjects.[103]

It may seem odd to use a variable when other studies have shown it does not discriminate between conditions. However, there were good reasons for including this variable. The first is that EMD studies have shown that to interpret muscle responses to changes in resistance using EMG, one must have information on both timing and amplitude. This is especially true if there are comparisons between conditions in which muscle length is changed. In this study it was important to record the latency of the muscles even if changes were not expected. This leads to a fuller interpretation of any amplitude changes. EMG is only a measure of the contractile component of the muscle. Measures of the other component, the passive component must be inferred from timing and force data along with the EMG. Without timing data amplitude data leads to only limited interpretation. For example, in this study, if there were no changes in amplitude for different conditions, one would always be left to doubt whether this reason was that the length of muscle change with each condition (in which case the latency would be different for each condition but the amplitude would be the same).

The second reason was to define, if possible, Janda's concept of hyperactivity and inhibition of certain muscles with certain types of muscle imbalance. For several decades clinicians have noted that persons with diminished hip extension seem to have hypertrophied, stronger and tighter

back extensors. This relationship has been ascribed to the body mechanics during erect stance and the maintenance of trunk position during gait. Janda has suggested the notion that the erector spinae develop a hyperactive state in the erector spinae which may develop from the mechanical demands of erect posture and gait. Janda has demonstrated this hyperactive state in the timing behaviors of muscles (including the erector spinae) using a simple prone hip extension movement. Although it is not specifically stated, it is a fair interpretation that hyperactivity implies that erector spinae contractions are early and inappropriately greater amplitude in persons with tight hip flexors compared to normals.

The latency recordings from this study do not support the notion of difference in onset for erector spinae muscles in tight hip subjects. It may be that hyperexcitability is revealed by some other timing phenomenon such as time to peak amplitude. However, this study was interested in seeing whether there were changes in timing and amplitude in the pre-movement period where changes are more likely be due to motor pre-programming.

Early Amplitude Change

RES EAC seemed to have some ability to discriminate. Like DMOS, it seemed to vary with differences in movement rather than on hip type or position. As with latency, this variable did not support the notion of hyperexcitability of erector spinae muscles in subjects with tight hip flexors.

However, it may be that hyperexcitability may be revealed by studying amplitude in another time period. This study concentrated on the pre-movement period where, it is assumed, muscle behaviors can be ascribed more exclusively to motor pre-programming influences. Peak amplitude after movement onset may reveal the hyperactivity in tight-hip subjects suggested by Janda's studies. Whether there is any support for the notion in another time frame may be answered in a subsequent study.

Conclusions

1. There is no evidence in the pre-movement period of differences in erector spinae amplitude and timing to support the notion of hyperexcitability of erector spinae muscles in tight-hip subjects.
2. Subjects with tight anterior hip structures and subjects with loose anterior hip subjects show a difference in the onset duration of four muscles involved in hip extension but only for voluntary concentric hip extension.
3. The type of movement had an effect on muscle behaviors in the pre-movement period while the relative tightness of opposing muscles had no effect.

Implications

1. This study shows reasons to question the notion that erector spinae muscles develop a state of hyperactivity in subjects with tight anterior hip

structures (tight hip flexors) as suggested by Janda. No differences were found between subjects with tight hips and subjects with extreme opposite measurements. However, this study collected data from the pre-movement period only. It could be that differences indicating hyperactivity may occur after movement onset. Similar data should be gathered from that period to furthur test the idea of hyperexcitability of these muscles in tight hip subjects.

2. Some differences between loose and tight subjects were found in the pre-movement period. This suggests the groups with different types of muscle imbalance may develop distinct motor pre-programming patterns for prone voluntary hip extension. The pattern does not seem to include a change in latency of the erector spinae muscle and the onset of muscles other than the erector spinae may be responsible for the changes observed.

3. There was a question whether there was a difference in the effects of external and internal resistance. Testing the major hypothesis involved comparing only conditions in which the internal resistance varied. Conditions with differences in external resistance were not compared. A tight-loose difference was found when position was manipulated. This suggests that internal resistance did cause a change in behaviors. However, in testing the minor hypothesis conditions where the external resistance was different seemed predominant in showing differences. This suggests that in the pre-movement period, external resistance had a much greater effect on muscle

behaviors than internal resistance. It may also suggest that external resistance differences have a strong enough effect to cancel the effects of differences in internal resistance.

The motor programming area may be more sensitive to pre-movement external mechanical input rather than mechanical information coming from internal sources. If there is a response to internal resistance it is weak and easily cancelled by the addition of external resistance.

Clinical Implications:

The results of this study seem to call into question that Janda's visual inspection test of muscle onset during a prone hip extension movement can distinguish tight hip subjects from normals. Since this test is the cornerstone of the notion of hyperactivity of erector spinae muscles in persons with tight hip flexors, that concept is also called into question. There is only scant evidence that subjects with much greater differences (tight hip subjects from loose hip subjects) can be differentiated using EMG, a more sensitive measure of muscle onset than visual inspection. However, the evidence is only from the pre-movement period. The test might find greater validation when studies of EMG activity in the after-onset period are done.

Most problems that need rehabilitation are multi-dimensional. The usual dimensions are inherited, congenital, environmental, acquired, mechanical, neurological, psycho-social, and cardio-respiratory. The therapist

wants to know to what part each dimension plays in the natural history of the problem in order to select interventions or accommodation procedures if interventions will be ineffective. This study was undertaken to try to observe whether an element in the neurological dimension (the development of a motor program) was involved in a well-accepted clinical phenomenon known as muscle imbalance. Relatively scant differences were observed in subjects with widely disparate types of muscle imbalance. This suggests that if neurological motor planning is involved in the development of different types of muscle imbalance, its effect is minimal. External mechanical factors seemed much more responsible for differences observed between conditions. This suggests that signals related to external environment may be more important than signals related to the internal environment in determining muscle behavior in the pre-movement period.

The duration of muscle onset seemed to differentiate most consistently between conditions in this study. DMOS may be a clinically useful measure for those who use EMG clinically to distinguish abnormal muscle behavior from normal. This duration variable may be useful in identifying pathology or studying the effects of different treatments.

Over 250 subjects were tested for hip flexor length using an inclinometer. Means and standard deviations were calculated for a non-symptomatic group of subjects in the 18-55 years age group. The

measurement produced means with high variability. These measurements may be clinically useful despite the high variability.

Suggestions for further study

This study concentrated on muscle behaviors in the pre-movement period. Muscle behaviors are more assuredly the result of motor pre-programming in this period. This study found no differences for latency and few differences in amplitude. The pre-movement period showed no evidence to indicate hyperexcitability of erector spinae in hip tight subjects. It may be that an interval other than the pre-movement period may show differences in timing and amplitude muscle behavior for groups with different hip muscle imbalances. Ross, et.al. found that when external resistance was changed for a back extension movement, peak EMG occurred later after the onset of movement. The data collected for this study could be used to conduct a study to see if there is any systematic variation in peak amplitude and timing of erector spinae after onset of movement. However, if differences are found a method must be found to discern whether these differences are due to motor pre-programming or differences in the mechanical requirements of the ongoing movement.

The influence on internal resistance seemed very weak. It could be that the differences between extended and flexed positions might not have

been great enough to provoke observable differences. A follow-up study using more extreme differences between positions might be useful.

The difference between lift and hold was assumed to be a difference in external resistance. This variation had the greatest influence in making comparisons between conditions significant. To confirm whether the differences observed for movement were due to external mechanical influences a study employing different external weights could be conducted. Normal subjects could lift and hold different magnitudes of weight in the two different positions. Comparisons could be done to see if different weights caused similar differences in duration, timing and amplitude as in this study. Weights could be graduated to see how much resistance difference is needed to show an effect. The effect of changing position on the effect of weight could be observed.

It has been mentioned several times that the erector spinae are assumed to hypertrophy because of external resistance caused by the distribution and acceleration of the trunk during stance and gait. This well accepted assumption has not been tested. Loose hip and tight hip subjects could be compared on the location of line of gravity during stance and muscle response to perturbations of stance. The same subjects could be studied during gait to see if there is a difference in erector spinae behavior during terminal stance in gait as is assumed.

The duration of muscle onset was different for loose and tight subjects in the lift movement. However, the latency of the erector spinae was not different. This meant that some other muscle is responsible for the difference in DMOS. The muscle whose timing is most responsible for differences in DMOS could be more clearly defined. It was found that the hamstring muscle was to some extent responsible for the differences in DMOS. It may be that the gluteus maximus may also be responsible.

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APPENDIX

Table A.1 Pilot study results; n = 12; 7 loose hip vs. 5 tight hip subjects.
Results in seconds for DMOS and latency
Results in percent MVC for EAC

	Group	Lift Ext.	Lift Flex	Hold Ext.	Hold Flex
DMOS	HT	.1457	.1654	.1173	.1444
secs.	HL	.2163	.2258	.1660	.1896
LES (LAT)	HT	.021	.067	.098	.165
secs.	HL	.118	.114	.078	.095
RES (LAT)	HT	.063	.098	.160	.209
secs	HL	.151	.148	.162	.156
EAC % %	HT	19.88	36.76	32.39	61.93
% change	HL	3.10	13.06	2.93	48.49

Table A.2. Pilot Study Results; n = 12; 5 Tight vs. 7 Loose Subjects

	DMOS	Les Lat	Res Lat	Res EAC
Main Effects				
Movement	F = 5.49	F = 1.59	F = 2.47	
	p = .241	p = .239	p = .147	
	Li > H[#]	H > Li[@]	H > Li[@]	H > Li[&]
Hip type	F = 2.47	F = .08	F = .36	
	p = .147	p = .748	p = .560	
	Lo > T[#]	Lo > T[@]	Lo > T[@]	T > L[&]
Position	F = 3.21	**F = 4.80***	F = 1.95	
	p = .104	**p = .053**	p = .193	
	Fl > Ex[#]	Fl > Ex[@]	Fl > Ex[@]	Fl > Ext
Two Way				
HipXMvt	F = .52	**F = 6.39***	**F = 7.19***	
	p = .488	**p = .030**	**p = .023**	
	LL > TL[#]	LL > TL[@]	LL > TL[@]	TL > LL[&]
	LH > TH[#]	TH > LH	LH > TH	TH > LH[&]
MvtXPos	F = .07	F = .21	F = .01	
	p = .802	p = .657	p = .921	
	LF > LE[#]	LF > LE[@]	LF > LE[@]	LF > LE
	HF > HE[#]	HF > HE[@]	HF > HE[@]	HF > HE
HipXPos	F = .08	F = 3.05	F = 1.85	
	p = .790	p = .112	p = .203	
	LF > TE[#]	LF > TE[@]	LF > TE[@]	LF > TE
Three Way				
MvtXHipXPos				
	F = .00	F = .001	F = .46	
	p = .955 [#]	p = .993	p = .513	
	LLE > TLE[#]	LLE > TLE[@]	LLE > TLE[@]	TLE > LLE[&]
	LLF > TLF	LLF > TLE[@]	LLF > TLF[@]	TLF > LLF[&]
	LHE > THE[#]	THE > LHE	LHE > THE[@]	THE > LHE[&]
	LHF > THF[#]	THF > LHF	THF > LHF	THF > LHF[&]

Increased Resistance =

[#]Decreased DMOS 12/12
[@]Decreased Latency 19/24
[&]Increased EAC 6/11