# RECOVERY FROM NEUROMUSCULAR FATIGUE FOLLOWING PLYOMETRIC AND RESISTED SQUAT EXERCISES TO EXHAUSTION

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# Recovery from Neuromuscular Fatigue following Plyometric and Resisted Squat Exercises to Exhaustion

By

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# ABSTRACT

The purpose of this study was to investigate the recovery from neuromuscular fatigue following plyometric and resisted squat exercises performed to exhaustion. Previous research has shown that recovery from neuromuscular fatigue is dependent on training status of the subject and the time, type and intensity of the exercise performed. In spite of this finding, the recovery period prescribed for plyometric training is similar to that of slow movement resisted exercise training. Plyometric and resisted squat exercises utilize a unique yet natural movement known as the stretch-shortening cycle (SSC), however due to the differing movement velocities of the exercises, the nature of the SSC performed is very different. This difference is believed to impose distinctive mechanical and neural stresses on the musculoskeletal system. Ten moderately trained subjects and four highly trained subjects completed two SSC trials to exhaustion. Subjects performed continuous drop jumps from a 30 cm platform during the plyometric trial and continuous squats with a load of 65% of their one-repetition maximum during the resisted squat trial. There was no difference in the neuromuscular recovery response following plyometric and resisted squat exercises performed to exhaustion. However, the duration of the plyometric trial was dependent on the training status of the subject; highly trained (HT) subjects (athletes who trained for their sport for 6 or more hours a week) (68.2%) than the moderately (MT) subjects (individuals who participated in 3 or more hours of physical activity a week) and the duration of the plyometric trial was significantly longer (67.1%) than resisted squat trial for the HT subjects. The duration of the trials did not differ for the MT subjects. Changes in neuromuscular properties following plyometric and resisted squat trials to exhaustion are independent of the type of exercise, duration of the trial and training status of the subject.

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| | |
|---|---|
| A | Ampere |
| BPM | Beats Per Minute |
| CrP | Creatine Phosphate |
| $Ca^{+2}$ | Calcium |
| cm | Centimeter |
| CNS | Central Nervous System |
| DHP | dihyropyridine |
| EC | Excitation Coupling |
| EMG | Electromyography |
| HFF | High Frequency Fatigue |
| HR | Heart Rate |
| H-reflex | Hoffman Reflex |
| HT | Highly Trained |
| Hz | Hertz |
| iEMG | Integrated Electromyography |
| $K^+$ | Potassium |
| kP | Kilopascal |
| Kg | Kilograms |
| LFF | Low Frequency Fatigue |
| min | Minute |
| ms | Millisecond |
| MT | Moderately Trained |
| M Wave | Compound Muscle Action Potential |
| mV | Millivolts |
| MVC | Maximal Voluntary Contraction |
| N | Newton's |
| $Na^+$ | Sodium |
| PAR-Q | Physical Activity Readiness Questionnaire |
| Pi | Inorganic Phosphate |
| PT | Plymetric Training |
| rpm | Revolutions Per Minute |
| RM | Repetition Maximum |
| RMS | Root Mean Square |
| RT | Resistance Training |
| s | Seconds |
| SSC | Stretch-Shortening Cycle |
| SD | Standard Deviation |
| SE | Standard Error |
| SR | Sacroplasmic reticulum |

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# 1 INTRODUCTION

## 1.1 BACKGROUND OF STUDY

Plyometric training has become a popular training method for improving athletes' explosive muscular power. The extensive research on the effects of plyometric training has proven it to be a widely accepted training method to enhance athletes' dynamic performance (8). Plyometric training adheres speed and strength exercises that stimulate the natural movement required in most sport settings (9). The exercises involve an eccentric component that utilizes an active pre-stretch followed by a concentric component that actively shortens the muscle. This movement is known as the stretch-shortening cycle (SSC). The quick pre-stretch during the eccentric phase has been attributed to the enhancement of power due to the storage of elastic energy in the muscle-tendon unit that is released during the concentric phase and the utilization of the stretch-reflex in the muscle fibers (6). For these reasons this form of training differs greatly from slow movement weighted exercises found in resistance training as the rapid and dynamic countermovement activates the reflex and central nervous system pathways, which overload the muscular system (9). Plyometrics (high impact SSC movements) may procure different mechanisms of fatigue due to alteration in reflex activity and inhibitory neural mechanisms in the muscle-tendon unit (12), which may result in acute and delayed decreases in neuromuscular performance therefore leading to variations in the recovery pattern and duration compared to low impact SSC activities (9). Thus there is a great importance for athletes to follow a highly specified plyometric training regime with an optimal recovery duration in order to benefit from the neuromuscular adaptations that result from SSC exercises, such as improved muscle force and contraction velocity (7).

Extensive literature has been published in the area of plyometric training with regards to sport specific SSC exercises, frequency, volume and intensity of training, and the physiological benefits, however little remains known about the optimal recovery period following bouts of plyometric training. Some researchers have proposed recovery periods similar to that of resistance training but as discussed later this seems inaccurate as the mechanisms of fatigue differ. Donald Chu has used anecdotal information and research from traditional resistance training to determine ranges of work to rest ratios between sets of differing intensity SSC exercises (25). Also, due to the involvement of unique mechanical and reflexive mechanisms, extensive research has been devoted to the study of neuromuscular fatigue through fatigue protocols of submaximal and maximal SSC exercises (11, 10, 5).

## 1.2 PURPOSE OF STUDY

As discussed, in depth information exists about the fatigue response and bimodal recovery pattern following SSC exercises such as hopping or jumping, however there appears to be a gap in the literature comparing distinctive forms of SSC involved in different types of athletic training (9). The drop jump is a commonly used exercise involved in a plyometric training program and it has been shown to alter motor control strategies and improve athletic performance measures (2). The drop jump induces a high stretch on the lower extremities from the extremely high impact loads placed on the body, which requires the neuromuscular system to activate different neural mechanisms compared to low impact movements (1). In order to utilize the rapid SSC movement, the goal of the drop jump is to perform the exercise with a high stretch velocity during the lengthening phase and minimize the amount of time to transition between the eccentric and concentric phases. These factors have been reported to enhance jump performance

due to an increase in power absorption and production (4). The resisted squat exercise is a dynamic movement that is functionally appropriate for athletic use as it allows the athlete to assume a position involved in many types of sport (3). The resisted squat is performed with a low stretch velocity during the lengthening phase to control the downward motion. A slight pause between the eccentric and concentric phases is implemented to ensure minimal involvement of passive elastic elements and greater contribution of the contractile elements for force production during the concentric phase (4). The protocols involved during the execution of the plyometric and resisted squat trials are believed to utilize different neural mechanisms and induce distinctive mechanical stresses on the contractile apparatus. The present study will investigate whether the nature of the SSC movement involved in exercise leads to differences in central and peripheral fatigue due to differing mechanisms of stress placed on the neuromuscular system. Thus, the purpose of this study is to compare the neuromuscular fatigue response and recovery profile following plyometric and resistance training exercises to exhaustion.

## 1.3 SIGNIFICANCE OF STUDY

Information accrued from this research will help to verify whether the current recommended recovery periods in plyometric training programs, which are based on traditional resistance training programs, have been accurately prescribed for this very unique form of exercise (3). The present study will provide physiological evidence to support or refute the ability to replicate the recovery periods from resistance to plyometric training programs. This information will help to ensure athletic training is performed to an optimal level by minimizing the possibility of over or under training and decrease the chance of overuse injuries. This study

will also provide mechanistic information regarding neuromuscular functioning following stretch-shortening exercises involving rapid and slow movements.

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# 2 REVIEW OF LITERATURE

## 2.1 INTRODUCTION

In forms of human locomotion, such as running, walking and hopping, the musculoskeletal system is naturally subjected to stretch forces that result in muscle lengthening (4). During these forms of locomotion, muscles perform a sequence of phases that involve the stretch phase of a pre-activated muscle (eccentric action) as the body makes contact with the ground followed by the transition phase as the muscle transcends into the shortening phase (concentric action) that propels the body in the plane of intent. The combination of these actions is known as the stretch-shortening cycle (SSC) (57). The role of the SSC is to enhance the performance of the shortening phase during the concentric action, which is not observed during a pure concentric action. However the precise mechanisms for the force and power potentiation during SSC are still uncertain and thus SSC research has become an increasingly popularized area (74). The unique yet natural loading of the musculoskeletal system during the pre-activation phase is believed to cause mechanical and structural modifications as well as activate reflex and central nervous system pathways that differ greatly from pure concentric and eccentric movements not found in typical human locomotion and other movement activities (74). The purpose of this review is to discuss the unique characteristics of the SSC, focusing on its involvement in a specialized type of athletic training known as plyometrics, and propose how the recovery from neuromuscular fatigue may differ following exercise involving the SSC.

## 2.2 STRETCH-SHORTENING CYCLE

### 2.2.1 STORAGE OF ELASTIC ENERGY IN SSC

Originally theorists believed that the enhanced power output and efficiency during the concentric action of a SSC movement was the result of stored elastic energy in the muscle-tendon units of the body (48). Cavagna et al. (21) were the first to conceptualize this theory, as they believed that the high work efficiency measured in the external and internal mechanical work during bouts of running was attributed to the substantial amount of energy from elastic recoil. The stretching phase (eccentric action) that occurs prior to the shortening phase (concentric action) was believed to produce elastic recoil of the muscle-tendon unit resulting in about half the energy spent in running (21). The concentric action corresponded to the positive work production of the muscle, actively shortening to produce work and the eccentric action corresponded the negative work, actively lengthening to produce work (96). Studies using the animal model proposed that the elastic recoil of a muscle's tendon could reduce the amount of work by approximately 30-50% of other muscles to produce equivalent workloads if stored elastic energy was absent (14). The ability for the muscle-tendon unit to store mechanical work as elastic energy during the eccentric action for subsequent use is believed to be a crucial characteristic of the musculoskeletal system in order to perform highly efficient locomotion (35).

Changes within the muscle-tendon unit have been shown to produce spring-like actions contributing to elastic energy (38). Tendons that have undergone stretch can recoil elastically much faster than any muscle can shorten. Thus in the early stages of takeoff during jumping, tendons act as power amplifiers that can build up force by storing strain energy and returning it the later stages as energy by elastic recoil (65). Kawakami et al. (56) measured fascicle length in the ankle plantar flexors and observed that muscle fibers optimally work in an isometric manner

and the storage and release of elastic energy for greater performance during a counter-movement action occurs in the tendon. In accordance, Ishikawa et al. (49) examined the interaction between fascicle and tendinous tissue of the vastus lateralis in humans during drop and squat jumps from constant dropping height but different rebound heights. During the drop jumps, when the vastus lateralis underwent a SSC action, a correlation between increased jump performance and rebound intensity was observed to occur. This was attributed to an effective fascicle length that allowed for tendon recoil in the shortening phase of the drop jump. When the fascicle length surpasses the effective length during a low intensity rebound jump no additional forces were produced and the passive recoil of tendinous tissue recovered less elastic energy (49).

Another approach to study the effects on tendon recoil and rebound performance is by manipulating the drop jump height. A positive correlation between increasing tendon recoil and drop jump height during countermovement has been reported to occur (56). In addition, rebound performance has been shown to be dependent upon the preceding height of the drop whereby performance improves as drop height increases (49). This increase in tendon recoil and rebound performance continues until the drop height becomes too great and protective mechanisms are activated (50). Ishikawa et al. (49) reported that the optimal drop height is individualized and is based on a drop height that allows for maximal lengthening of the tendious tissues during the contact phase. The inability of the tendon to lengthen once a certain drop jump height is attained is suggested to be due to inhibitory mechanisms that prevent further increases of the stretch load because the maximal drop condition is too high for the individual (49).

Ishikawa et al. (50) studied tendon recoil during short contact SSC movements performed at three different drop jump heights. The results of the study showed tendon recoil were optimized during short contact for drop jumps but can be limited by increasing drop height. The elastic recoil of the mechanical energy during SSC movements is decreased if the coupling time

(the duration of the transition phase that occurs between the stretch and the shortening of the muscle) is as long as observed during low drop height jumps (48). The coupling time plays a key role in the economy of muscular work and a shorter coupling time has been observed to produce a greater mechanical efficiency that is defined as the work done as a proportion of metabolic cost during SSC movements (17).

Two distinct processes appear to contribute to work performed by the muscle, which include the conversion of metabolic energy into mechanical work and the storage and re-utilization of elastic energy (35). Ettema (35) used the in situ rat model to analyze the contribution of these two processes on mechanical efficiency of the gastrocnemius during various stimulated SSC. It was discovered that in an extremely stiff muscle the mechanisms of the series elastic element did not produce the reutilization of recoiled elastic energy and the metabolic efficiency of the muscle equaled the mechanical efficiency. However, in muscles with a more complaint series elastic component an observed increase in elastic energy efficiency was revealed resulting from a more effective storage and release of elastic energy from the muscle-tendon unit (35). Mechanical efficiency has also been investigated in SSC movements in the human model and is optimized when angular velocity and ground reaction forces during the braking phases are increased (3).

The metabolic energy consumption of a muscle that is shortening is the sum of a cost of generating force and a cost of doing work; if an active muscle is forcibly stretched it does negative work, degrading mechanical energy to heat (65). McCaulley et al. (64) compared mechanical efficiency during various jumping protocols to make an indirect comparison of the elastic contribution to the concentric action by determining the aerobic and anaerobic energy expenditure as well as the assessment of negative and positive work. The authors concluded that negative work is optimized when the time allotted to complete the loading phase is decreased,

which leads to an increase in muscle activity and braking phase force and yields optimal muscle-tendon kinetics. When the negative work is optimized the mechanisms contribute to an increased concentric action and thus performance (64). The greatest increase in the braking force was associated with a significant improvement in mechanical efficiency, which was observed during the drop jump performed at 125% of the maximum countermovement jump height (64).

Since the original proposed theory, the topic of re-utilization of elastic energy on the efficiency of movement has been contested (48). Based on thermodynamics, the reutilization of elastic energy is not capable of enhancing mechanical efficiency because it is not believed to contribute to the conversion of metabolic energy into mechanical work (89). However, no convincing findings have been established to refute the storage and re-utilization of elastic energy as an important mechanism of enhanced force production (58).

### 2.2.2 STRETCH REFLEX IN SSC

Current research is exploring the idea that SSC movements found in forms of locomotion such as running and hopping are controlled by the same combination of central programming and spinal reflex mechanisms involved in cyclic movements (90). The effectiveness of the SSC to enhance force production is dependent on the rapid transfer from the pre-activation eccentric action of the stretch phase to the concentric action of the shortening phase (84). Myoelectric potentiation originating from stretch reflexes have been proposed to aid in the force production as a consequence of the high stretch velocity and pre-activity at the moment of impact during SSC movements (17, 29). Studies have revealed a consistent electromyogram (EMG) burst starting about 45 ms after the moment of impact during hopping (90, 39). This response was defined as a short latency response. It is suggested to be a stretch reflex response superimposed

on supra-spinal activity (96). The short latency stretch response can be easily reproduced and is visible in all lower extremity muscles (58). Central nervous system control of muscular activation during the short latency response of SSC movements is primarily attributed to spinal rather than supra-spinal sources (86).

During hopping and drop jumping the Hoffman-reflex (H-reflex), which measures changes in muscle spindle afferent excitability (47), has been observed to be very low at takeoff, continue to remain low during the flight phase and increase before impact (90). Taube et al. (85) revealed that changes in reflex modulation could be attributed to changes in the Ia-afferent transmission into the muscle motoneuron pool. They proposed that the stretch reflex response contributes to the enhanced efficiency of the stretch-shortening cycle as a result of a significant enhancement of Ia-afferents transmission in the early stance phase. The response produces a reflex response superimposed upon muscle activation activity or by synchronizing muscular activation in the already active muscle at the time of impact (85). After impact, other sources appear to be predominant since the contribution of spinal reflexes to muscular activation during a drop jump progressively decline (85). Studies of spinal excitability clearly shown that if the muscle-tendon is stretched during the early impact phase to activate the afferent muscle spindles then spinal reflexes are a primary contributor to EMG activity during SSC movements (58, 96).

In order to deliver an effective SSC movement with enhanced force production, high muscle-tendon unit stiffness is required and the control of this stiffness is dependent upon the efficiency of the reflex system (73). It was found that muscle preactivity correlated with muscle stiffness during the initial contact phase during drop jumps and the short latency response contributes to a high series elastic stiffness, which is proposed to decrease contact time and increase the economy of work (6).

As SSC movements are involved in all forms of typical natural human locomotion, such as hopping and running, in a recent a review Komi (58) suggested that it would be highly unlikely that proprioceptive reflexes are possible mechanisms involved in SSC movements. Current research has now provided a significant amount of evidence to support the important role that stretch reflexes play in force production during SSC movements, which is not observed in pure isometric, eccentric or concentric movements (58).

## 2.3 PLYOMETRIC TRAINING

SSC movements are being increasingly incorporated into athletic training regimes due to the enhanced performance these exercises have on muscular power (62). Plyometric training uses SSC movements at high intensities that involve a rapid eccentric contraction followed by a powerful concentric contraction (62, 96). This form of training is used by power athletes in sporting activities and is considered in a special category of explosive training (91). The sudden impulse created by the rapid deceleration during the stretch phase and the almost immediate acceleration in the opposite direction during the shortening phase creates a potentiation mechanism that is dependent on the speed of the stretch phase (91). The speed of the stretch phase and sudden impulse puts vertical jumps in the fast countermovement category of explosive exercises while resistance squats are placed in the slow category, as resistance squat induced potentiation effects are nonexistent or minimal due to the prolonged eccentric and transition phases (50). In addition, the central nervous system command of contraction during slower resistance training differs from that during vertical jumping as supported by the finding that static overload training is less effective for increasing vertical jump performance than dynamic overload training (12). Plyometric training involves maximal SSC movements that induce greater

reaction forces, greater muscle activation and shorter exercise duration compared to SSC movements of natural locomotion (84).

Plyometric training is extremely complex due to the involvement of the SSC and as a result many unanswered questions remain in the area of exercise prescription with regards to frequency, volume, intensity of training, and recovery duration (8). Some researchers have proposed recovery periods similar to that of resistance training but according to Nicol et al. (74) this logic seems inaccurate, as the physiological mechanisms involved in the performance of SSC movement appear to be much different from pure isolated isometric, concentric and eccentric movements. Donald Chu has used anecdotal information and research from traditional resistance training to determine ranges of work to rest ratios between sets of differing intensity SSC exercises (25). Similar to resistance training, to develop muscular power D. Chu has suggested a work to rest ratio of 1:5 to 1:10 when performing plyometrics to ensure proper execution and intensity of exercise. To develop muscular strength and endurance exercise are performed in a continuous manner and less than two seconds of recovery time in a 12 to 20 minute workout is recommended (25). These work to rest ratio are generalized exercise prescription based on training goal, i.e. muscular power or endurance, however these recommendations may vary depending on several factors, such as the type of muscle action performed (94). Few studies have actually compared neuromuscular fatigue response and recovery patterns following plyometric and resistance training exercise to determine if similar work-to-rest ratios are appropriate based on neuromuscular fatigue responses.

Toumi et al. (88) compared measures of fatigue following consecutive sets of SSC exercises and isometric leg press performed in sets of 10 repetitions accompanied by 2 minutes of rest until the subjects could no longer perform the exercise at 50% of their MVC. Maximal isometric force and concentric power, muscle activation and kinematic data were analyzed

before and after fatiguing squat and drop jump trials to show changes in knee muscle tendon stiffness. The study hypothesized that only the SSC exercise would increase knee stiffness due to greater alternations in reflex sensitivity, however both fatiguing protocols had a similar effect on muscle stiffness and decreased jump performance, as shown by similar decreases in maximal isometric force, drop jump height, and the work of the knee joint during both squat and drop jumps as well as an increase in the duration of the eccentric and transition phases and hip flexion angle during the drop jump. Decreased jump performance was independent of the contraction type induced fatigue, thus the decreased jump performance was associated with a decreased stiffness of the knee extensor muscles (88). However, the SSC exercise performed on a sledge apparatus and the effects of high impact forces on the musculoskeletal system are reduced compared to a purely vertical movement such as a plyometric drop jump.

SSC movements present a complex model of fatigue that involve unique pathways that may not be utilized in isolated forms of movements (74). It is well known that the SSC heavily tax the skeletal muscles mechanically causing structural damage of the extra and intra-fusal muscle fibers influencing the reflex activation and inducing metabolic fatigue responses (74).

## 2.4 NEUROMUSCULAR FATIGUE

Neuromuscular fatigue was traditionally studied in isolated forms of either eccentric, concentric or isometric actions, however the current literature is substituting these models with forms of SSC movements as it is believed to be a more representative model of fatigue during natural human movement (74). One definition of fatigue is the inability to maintain or generate force following sustained or repeated muscle activation of moderate to high magnitude (32). Fatigue is caused by impairment at one or several sites along the neuromuscular chain of events

to initiate a muscular contraction (2). Sites of fatigue can be assessed in the central nervous system during and after a voluntary contraction and peripheral fatigue can be assessed by an electrical stimulation via the motor nerve of a muscle independent of volition (66). The previously discussed contributions of elastic energy and stretch reflexes to plyometric and related SSC actions may alter the state and site of fatigue when compared to non-SSC actions.

### 2.4.1 SSC AND NEUROMUSCULAR FATIGUE

An extensive review by Nicol et al. (74) discusses the well-documented bimodal recovery pattern that occurs following fatiguing SSC activities. It is a delayed process that involves a dramatic decline in muscular force immediately after (30 seconds) the SSC movement followed by a short-lasting recovery (30 minutes to 2 hours) and then a subsequent secondary drop (1-2 days) (30, 58). The recovery pattern following SSC movements is hypothesized to occur due to the unique rapid loading phase during the eccentric action that lengthens the muscle as it actively brakes prior to shortening (74). The continuous lengthening of the muscle during SSC exercises to fatigue results in an acute metabolic-induced response followed by a short-term recovery then a subsequent secondary reduction due to inflammatory and remodeling processes (76). The initial decline in force following SSC exercise is hypothesized to be the result of metabolic disturbances, which cause a reduction in reflex sensitivity. This mechanism is believed to correspond to blood lactate levels during high intensity SSC and a reduced muscle metaboreceptor-mediated response due to glycogen depletion during low intensity SSC (30, 7). In addition, the initial mechanical damage of individual muscle fibers (demonstrated by an observed increase in creatine kinase activity) following the SSC exercise results in decreased performance (36). The secondary reduction in performance is due to structural and chemical

damage that results in the infiltration of phagocytes and macrophages in the individual muscle fibers (36).

These changes are believed to alter afferent sensory pathways that contribute to the stretch-reflex thereby modifying joint and muscle stiffness regulation (46). The bimodal recovery pattern of muscular force parallels other neuromuscular properties that include activation level, muscle action compound potential (M Wave), H-reflex and stretch reflexes (30). It has been theorized that fatigue inducing SSC exercises alter the role of the muscle spindles located in the intrafusal fibers as a result of metabolic fatigue and/or muscle damage, which leads to modifications of viscous and elastic stiffness parameters of muscle (7, 46). Horita et al. (46) observed a decline in the short latency response following an exhaustive drop jump trial that was correlated to changes in stiffness regulation during the initial braking phase of the drop jump. Extra and intrafusal fibers damage resulted in reflex inhibition, due to the modulation of small diameter group III and IV afferents that are known to be activated during fatigue (41). It is suggested that pre-activation of the muscle during the eccentric action includes both $\gamma$ and $\alpha$-motoneurons, which allows for enhanced muscle spindle sensitivity (40). Horita et al. (46) observed an increase in pre-activation EMG after fatigue and they suggested central facilitation compensated for a decrease in muscle spindle sensitivity. Kutiunen et al. (59) found a similar response 2 hours post SSC exercise and hypothesized that a reflex loop that involved group III and IV muscle afferents, which activated $\gamma$-motoneurons lead to a subsequent increase in muscle spindle sensitivity and that $\alpha$-motoneuron activity would compensate temporarily for the contractile failure.

The long lasting recovery following SSC exercise is an interactive process that involves the deterioration of structural proteins in the intrafusal fibers, that directly reduced stretch-reflex responsiveness via Ia afferents and subsequently lead to a decreased regulation of stiffness in the

joint (58). Due to the long lasting recovery, SSC exercise performed 5 to 10 days subsequent to the first bout of exercise has been shown to produce a further delay in the recovery process (75). Thus, it is extremely important to determine the optimal recovery time between and following sets of plyometric exercises that utilize high intensity SSC movements to prevent declines in athletic performance and the possibility of overtraining.

### 2.4.2 LOW AND HIGH FREQUENCY FATIGUE

Impairment of excitation-contraction (EC) coupling and/or neuromuscular transmission contributes to a reduction in excitability of the muscle membrane resulting in neuromuscular fatigue (52, 32). EC impairment may be caused by failure of action potential propagation along the surface membrane and t-tubular system, a failure of the coupling mechanism between action potential and release of calcium, or a failure of calcium regulation at the level of the contractile elements (52). Failure of neuromuscular transmission is thought to occur due to an inactivation of presynaptic intramuscular nerve endings that act as a protective mechanism against damage to the muscle (37). To assess fatigue and induced contractile dysfunction evoked nerve stimulation at various frequencies and intensities provide a means for identifying sites of impairment resulting in neuromuscular fatigue (32).

Neuromuscular fatigue has been categorized into two types: (i) high frequency fatigue (HFF), which results in a preferential loss of force at high frequencies and a rapid recovery; and (ii) low frequency fatigue (LFF), which is characterized by long-term loss of force at low frequencies (68). Edwards et al. (34) were the first to document LFF in the human muscle as the selective loss of force at low stimulation frequencies despite recovery of the force generated at high stimulation frequencies. This type of fatigue is unique and was identified as a

distinguishable type of fatigue because of its long lasting presence that endures well after the cessation of the fatiguing exercise. LFF has been reported to persist for several hours and may take days to recover (52).

The factors that contribute to induction of LFF and HFF following exercise have been shown to be type, time and intensity dependent. The neuromuscular fatigue response following SSC exercises reveals that maximal intensity SSC exercises induce HFF following exercise to fatigue, which alters the electrical propagation subsequently leading to a reduction in muscle membrane excitability (51, 87); while submaximal intensity SSC induce LFF to a greater extent due to structural damage and impairment in EC coupling (84). Therefore, it is important to understand the extent of LFF induced by plyometric training, as the unique long lasting component of LFF may impede subsequent performance. During recovery from LFF there is a rightward shift in the force-frequency relationship where a relatively greater depression of force occurs at low compared to high frequencies (67). To perform a task that requires a firing frequency between 10-20 Hz following a fatiguing exercise that induces LFF, a larger number of motor units and/or an increase in the mean firing frequency of the motor nerves are required to complete the task successfully (34). The CNS must increase the firing rate or activate more motor units even though the muscle may show no decrement in maximal force (2). It is important to understand the different types of fatigue and their associated sites of impairment, as this will allow for an overall understanding of possible differences in the neuromuscular fatigue response and thus the recovery process following plyometric and resistance training.

#### 2.4.2.1 Low Frequency Fatigue during Exercise

LFF can be caused by a variety of exercises and a general observation has shown that there is a greater induction of LFF following exercises involving lengthening contractions of the active muscles such as eccentric- and SSC type exercises than in those fatigued by comparable shortening contractions (72, 63). Research examining differences in type of contractions have shown that eccentric contractions induce a greater amount of LFF fatigue compared to other isolated forms of movement (72). However, to our knowledge there are no studies that compare SSC type exercise to isolated forms of isometric, eccentric or concentric during an exercise of comparable time and/or intensity.

Dundon et al. (31) discovered concentric and eccentric contractions performed with the biceps brachii to a comparable percentage of an isometric MVC resulted in a similar induction of LFF, though a longer recovery of LFF after eccentric contractions was observed. However, in contrast to previous literature a follow up study examined possible differences in the response of flexors and extensors to LFF following concentric and eccentric contractions. In this study there was a similar level of LFF in the triceps brachii following both types of exercises to fatigue despite different contraction types (67). Meszaros et al. (67) concluded that the muscle group used to induce fatigue might contribute to differing results as the upper extremities muscles adapt differently than the lower extremities (which are continuously activated during gait) (Meszaros et al. 2010).

Baptista et al. (10) examined the intensity dependent relationship of LFF on the quadriceps following maximal concentric and eccentric contractions of the knee extensors and observed force impairment at low frequencies were non-significant in the induction of LFF. The study also examined LFF following submaximal eccentric and concentric exercise and discovered a relationship between the type of contraction at a submaximal intensity and

generation of LFF. There was an absence of LFF following submaximal eccentric exercise while LFF lasted for one-hour post recovery following submaximal concentric exercise (10). However, Davies & White (27) examined fatigue following submaximal box stepping, which involved lowering the body weight within each cycle (known as the negative component), which focuses on the generation of force through eccentric contractions. They discovered that the negative leg performing the eccentric work had a greater depression of LFF and HFF than in the positive leg performing the concentric contractions. Davies & Young (28) examined the effects of sustained and rhythmically performed isometric contractions on low and high frequency fatigue. They discovered a minimal effect of rhythmic isometric exercise on LFF and HFF while sustained isometric contractions resulted in statistical significant occurrence of LFF and HFF with a greatest reduction in force at low frequencies. The LFF intensity relationship for isometric contractions was observed to be the greatest at lower intensities of contractions where by 30% induced greater fatigue than at 60% or 100% MVC (27).

Ratkevicius et al. (78) demonstrated a time-dependence on the extent of LFF induced in individual muscle contractions during repetitive isometric exercise. The severity of LFF at a greater duration of contraction showed greater increases in EMG measures due to an increased recruitment of new motor units. This was proposed to explain the greater extent of LFF induced during the long duration contractions as the increased recruitment of faster contracting motor units are less resistant to LFF compared to earlier activated motor units (78). In accordance with this theory, Dundon et al. (31) observed an increase in EMG was correlated to an increase in LFF, which was strongest at low forces as well as after two hours following eccentric exercise. The response was attributed to muscle damage and neuromuscular adjustments.

SSC exercises engage neural control activating reflexive pathways, which reduce muscle activation and monosynaptic response following SSC exercise to fatigue (84). Research has

revealed an intensity-dependent relationship between SSC exercises and LFF. Strojnik & Komi (84) examined neuromuscular fatigue following short-lasting maximally intensive SSC exercises. The study demonstrated that maximal SSC resulted in both LFF and HFF, there was a rightward shift in the force-frequency relationship at all electrical stimulation intensities following maximal SSC. Similarly, subjects who performed 100 intermittent drop jumps of maximal intensity and 100 continuous jumps with maximal intensity showed a depressed force generation during recovery at all electrical stimulation frequencies (82). Intermittent jumps demonstrated the greatest extent of LFF immediately after exercise compared to the continuous jumps and a similar LFF rate of recovery was observed. However the intermittent jumps showed a persistent drop in force at low frequency stimulation for 24 hours post exercise (82).

Current research has demonstrated the prevalence of LFF following isolated forms of exercise and following SSC exercises and this prevalence is dependent on the time and intensity of the exercise. However, with regards to exercise prescription, there appears to be a gap in the literature comparing SSC exercises to isolated forms of movement of comparable intensity and time. The importance of understanding the neuromuscular mechanisms involved in the response pattern would contribute to determining optimal recovery duration for plyometric training, plyometric training time may differ from slower resistance training because of the rapid SSC speed.

#### 2.4.2.2 Mechanisms of LFF

The slow recovery phase of LFF distinguishes this type of fatigue from others because it involves supplementary factors other than ionic and metabolic disturbances, which are known to recover within minutes following exercise (52, 34). LFF was hypothesized by Edwards et al. (34)

to be the result of an impairment of the process of EC coupling due to the characteristically long-lasting component; the possibility of ionic and metabolic alterations such as depletion of high-energy phosphate was disregarded. As well, a failure of the muscle action potential was not observed following LFF thus impairment of action potential propagation was disregarded. The EC coupling theory has since been proven to cause LFF. This has been demonstrated by the reduction of intracellular $Ca^{2+}$ at all stimulus frequencies resulting in a rightward shift in the force- $Ca^{2+}$ relationship (77, 23, 93). The reduction in $Ca^{2+}$ release will result in proportionately greater reduction in force at the low concentrations of $Ca^{2+}$ due to the steep curvature at low stimulus frequencies compared to high frequencies, where the curve reaches a plateau and moderate falls in $Ca^{2+}$ have little effect on force (92). As EC coupling involves many stages, research in LFF has been devoted to the understanding of the stages involved in the reduction of intracellular $Ca^{2+}$ during the slow recovery component of LFF.

The mechanisms responsible for the slow recovery of fatigue-induced LFF alterations seem to be directed towards $Ca^{2+}$ release and/or uptake at the structure level of the triad region, which is located between the t-tubule and the terminal cisternae of the sacroplasmic reticulum (SR) (19). The disturbance in $Ca^{2+}$ kinetics may contribute to a prolongation of contraction time and half relaxation time during electrical stimulation of the muscle observed during LFF (67). Skurvydas et al. (82) have shown a decrease in contraction and relaxation times after intermittent and continuous jumping exercise that induced LFF, which was suggested to be indirect evidence of a decreased $Ca^{2+}$ transient duration. The mechanism hypothesized to result in the reduction in $Ca^{2+}$ release from the SR is not altered by availability of $Ca^{2+}$ nor the result of an impairment in the channel itself but rather an impairment in the signaling pathway (19). As the action potential passes down the t-tubules, it causes a conformational change in the dihyropyridine (DHP) receptors located in the t-tubule wall, followed by a brief opening of the calcium channels

(ryanodine) of the terminal cisterane portion of the SR (19). It is believed that the alternation might occur in the signaling pathway between the DHP of the T-tubule and the ryanodine receptor, which may become inactive (23). Ryanodine receptors have also been influenced by calcium in the t-tubules in a biphasic manner; low levels of calcium open the ryanodine receptors while higher levels reduce or impair the opening (19).

Chin et al. (24) proposed the decrease in $Ca^{2+}$ release from sarcoplasmic reticulum associated with LFF has at least two components: (1) a metabolic component, which recovers within 1 hour and (2) a component dependent on the elevation of $Ca^{2+}$ time integral, which recovers more slowly. Maximal continuous and intermittent jumps induced LFF of the quadriceps muscle, with the greatest induction following the continuous jump. It was suggested that the LFF associated with the decrease in $Ca^{2+}$ release was the result of the metabolic disturbances following the intermittent jumps and the elevation of the $Ca^{2+}$ time integral associated with muscle damage following the continuous jump (82, 26). In addition, an increase in $Ca^{2+}$ levels in the cytosol has been shown to occur via stretch-activated channels but can be fully restored via the buffering of cytosolic calcium levels when long rest periods (2 minutes) are allocated between contractions (60). Exposure to an exercise that induces LFF without shorter rest times between contractions does not allow the muscle to buffer cytsolic calcium and high levels can have detrimental effects on the muscle (26).

Mechanical and metabolic (e.g. calcium efflux) factors due to overload injury, delayed onset muscle soreness and overtraining result in performance decrements following plyometric training that involved high intensity SSC actions (26). Results from several studies indicate that long lasting EC coupling impairment in skeletal muscle is due to disturbances of mechanical properties that follow LFF fatigue induced exercise (55, 19, 20). As discussed, eccentric and stretch shortening activities have been reported to induce LFF; these types of exercises contain a

component of muscle stretch, which can be responsible for muscle damage (2). LFF has been shown to have a greater prevalence when the muscle is under high forces generated by stretching a fully activated muscle and when work is performed during long rather than short muscle fiber lengths (71). Alterations in the structural configuration of the muscle following prolonged eccentric exercise is a result of disturbances to the myofibrillar structure including broadening and streaming of the Z-lines and total disruption of the sacromeres (80). Change in the force-length relationship is a possible theory that has been proposed to explain mechanisms related to structural alterations that induce LFF following exercise (55). The structural composition of sacromeres along the length of skeletal muscle fibers is arranged such that a fully lengthened muscle causes the shorter sacromeres located at the ends of the fibers to reach their maximum length capacity. This makes them vulnerable to structural damage possibly leading to a loss of force and changes in the force-frequency relationship resulting in LFF (55). In agreement with this theory is the "popping sacromere hypothesis" described by Morgan (70) as the non-uniform lengthening of sacromeres beyond their optimum length results in stretch-induced muscle damage that occurs during the lengthening of an active muscle. Shearing of myofibrils and deformations of the T-tubules is the result of non-uniform lengthening of the sacromeres (69). The structural damage that follows SSC and eccentric exercise is responsible for the induction of LFF. This is due to the incapability to preserve calcium homeostasis, which is the result of damage in the sarcoplasmic reticulum resulting in a decrease in $Ca^{2+}$ release (53). The recovery of neuromuscular fatigue induced by an activity that results in LFF involves a unique and long-lasting recovery process, which maybe due to metabolic and mechanical disturbances. However exercise that induces HFF involves a much quicker course as changes in membrane excitability revert back to rest level within minutes of recovery (2).

#### 2.4.2.3 High Frequency Fatigue

High frequency fatigue is characterized by as a rapid reduction in force following a high stimulation of a muscle to produce maximal force. To induce HFF in humans, a muscular contraction must exceed approximately 50% of maximum force output, which produces initial firing rates at high frequencies that rapidly declines over a minute followed by a quick recovery (2). Jones et al. (54) were the first to discover the unique neuromuscular responses from high levels of excitation frequencies on human skeletal muscle. Their study showed a number of novel findings that included; (i) to produce an equivalent force of an unfatigued MVC, a high-frequency maximal nerve stimulation is required; (ii) sustained high-frequency stimulation leads to a greater rate of force loss than is observed in an MVC; (iii) high frequency stimulation of a fatigued muscle suppresses force whereas reducing the frequency of stimulation results in an increase in force; (d) in a sustained MVC the slow rate of force loss is similarly observed in a high frequency nerve stimulation as long as the initial frequency is progressively reduced (54).

To further understand the suppression of force due to high frequency stimulation in a fatigued muscle, Edwards et al. (33) examined the electrical activity of the muscle via changes in firing frequency. They hypothesized that the continuous high frequency stimulation was the result of a failure of electrical propagation leading to the observed reduction in muscle membrane excitability. The decreased excitability was characterized by a total increase in the area of the action potential, which is a product of a broadening action potential and a minimal loss in amplitude. This change corresponds to an increased conduction time along the muscle fiber membrane (33). In accordance, SSC movements (proposed to mimic a closer representation of the natural model of fatigue during human movement) have been shown to induce HFF following maximal intensity exercise to fatigue due to impaired electrical propagation. This impairment is the result of a reduction in muscle membrane excitability and neuromuscular

fatigue (51, 87). There is minimal literature on HFF during SSC movements, however Jereb & Strojnik (51) studied fatigue following maximal hopping for 60 seconds and a Wingate test. An increase in force at low frequency stimulations was observed until 7 minutes post exercise following the hopping intervention and a decrease in force at low frequency stimulation until 3 minutes was observed following the Wingate intervention, with a potentiation at the fourth minute. Similarly, Tomazin (87) demonstrated that 60 seconds of hopping with maximum intensity induced greatest loss of force at high stimulation frequencies while the force at low frequency stimulation remain unchanged from pre to post intervention.

#### 2.4.2.4 Mechanisms of HFF

The slowing of the waveform and loss of amplitude of the action potential demonstrated to be involved in HFF was believed to be the result of a reduction in $Na^+$ and an increase in the extracellular fluid during activity (51). To further examine the theorized mechanisms involved in HFF, Metzger et al. (68) observed a significant reduction in force after high frequency stimulation that was believed to be related to $Ca^{2+}$ accumulation in the t-tubular lumen and a rapid recovery was due to the reversal of this phenomenon. It has been shown that the EC coupling may be affected to a higher degree in HFF compared to LFF due to the excess rate of $Ca^{2+}$ accumulation in the t-tubular lumen that is approximately 50 times faster after high vs. low frequency stimulation (13). Westerblad et al. (93) measured the distribution of intracellular calcium concentration in isolated single muscle fibers following high frequency stimulation. The results of the study showed (i) a complete recovery from HFF within 2-3 seconds, rejecting metabolic factors as a possible mechanism as metabolic recovery takes several minutes; and (ii) a significant presence of $Ca^{2+}$ gradients that rapidly disappeared after the stimulation frequency

was reduced. The study concluded that HFF was caused by a less effective release of calcium from the SR because of a failure of t-tubule conduction that is possibly caused by $K^+$ accumulation or $Na^+$ depletion in the t-tubules. In accordance with these findings, Balog et al. (9) demonstrated a recovery of $Na^+$ and $K^+$ to correspond with the fast recovery of force following high frequency fatigue stimulation in frog skeletal muscle. The decrease in $K^+$ and increase in $Na^+$ altered the ionic composition of the t-tubular lumen and decreased force following stimulation to induce HFF.

The alterations in the ionic composition of the t-tubule remain a persistent mechanism of HFF however the understanding of these findings is not completely understood. The increase in extracellular $K^+$ hypothesized to correspond with the decrease in muscle force observed in HFF contradicts the theoretical belief that an increase in levels of extracellular $K^+$ should lead to a depolarization in the resting membrane potential and therefore greater excitability (52). A possible explanation for this contradiction lies in the altered inward $Na^+$ current. $Na^+$ currents have been studied in human skeletal fibers of slow and fast twitch muscles by voltage clamp. Following high frequency stimulation of a muscle fiber, a slow inactivation of inward $Na^+$ current was predominantly observed in fast twitch fibers when the membrane potentials were negative (79). Fast twitch fibers are believed to have an impaired ability to remove K+ from the extracellular space and a steeper slope of slow inactivation-membrane potential relationship due to a lower $Na^+$-$K^+$ ATPase activity and a lower density of capillaries compared to slow twitch muscle (79).

Maximal rates of motor unit discharge in human skeletal muscle range between 10-50 Hz and mean rates during a maximal voluntary contraction typically have been recorded between 25-30 Hz (16). Thus speculations of the importance of evaluating HFF at high frequency stimulation in human skeletal muscle following SSC movements warrant questioning. Jones et

al. (52) believe HFF has an evolutionary significance as the skeletal muscle recognizes the need to offset the accumulation of $K^+$ by controlling the firing frequency and activity of the $Na^+$ in order to prevent extensive damage to the muscle.

### 2.4.3 CONCLUSION

LFF and HFF contribute to the induction of neuromuscular fatigue through differing physiological processes that have distinctive time courses of recovery. The extent of LFF and HFF induced in the musculoskeletal system is dependent on the time, type and intensity of SSC movements performed. The recovery of neuromuscular fatigue following SSC movements is dependent on the type and extent of fatigue that is induced in the muscle. Recovery of neuromuscular fatigue from SSC movements that primarily induce LFF from metabolic process occurs within 15- 30 minutes post exercise, while SSC movements that induce the slow mechanical component of LFF require a 60 minutes to at least a 24 hour recovery period (82, 93). SSC movements that induce neuromuscular fatigue from mainly HFF recover from evoked stimulations at high frequency relatively quickly; approximately 5 minutes post exercise (2).

## 2.5 SUMMARY

The SSC is involved in all human locomotion such as running, walking and hopping where muscles are actively stretched prior to shortening (42). According to a review by Nicol et al. (74), mechanisms proven to explain neuromuscular fatigue utilizing isolated forms of muscle actions (isometric, concentric or eccentric) cannot explain fatigue induced by the SSC due to the strong mechanical and reflex activation. This natural muscle function has a clear force and power

potentiation during the shortening phase of concentric muscle action, which is the resultant of the preceding eccentric muscle action during the lengthening phase (58). Plyometric training programs involve various types of body-weight jumping exercises, such as drop jumps, countermovement jumps and squat jumps, which utilize SSC movements at a high intensity. Plyometric training has been shown to improve strength, muscle power, and coordination, thus leading to an improvement in athletic performance as the rapid SSC exercises are found in all types of sport (1, 11, 18, 43). Due to the high intensity involvement of SSC actions, this unique form of training can cause reversible neural, structural and mechanical disturbances depending on the severity and duration of the prescribed exercises (74). Therefore, it is important to understand the physiological mechanisms that induce neuromuscular fatigue to gain further insight into the recovery pattern that follows plyometric training in order to optimize athletic performance at all training levels.

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# 3 CO-AUTHORSHIP STATEMENT

My contributions to this thesis:

i) The present research project is supported by Natural Sciences and Engineering Research Council (NSERC). Dr. David Behm and I discussed his original idea of a research project in neuromuscular fatigue and plyometric training. Together we developed the methodology for the current research. Based on our research idea and methodology, I submitted a proposal to NSERC. The proposal was approved and funding was provided by Master's level scholarship awarded to me in my first year of my Master's program at Memorial University.

ii) Dr. Behm and I developed the jumping apparatus and then contracted Memorial University Technical services to build the wooden boxes.

iii) I recruited all subjects and Kristina Sheridan and myself performed all research testing on each participant. The experimental methodology required Kristina Sheridan and myself present at all times.

iv) Raw data was collected by Kristina Sheridan and myself. Under the supervision of Dr. Behm, I performed all data analysis procedures.

v) I prepared the manuscript with the guidance of Dr. Behm.

# 4 STUDY

## 4.1 MATERIALS AND METHODS

### 4.1.1 *Subjects*

Fourteen healthy and physically active male subjects (25.28 ± 3.4 years, 179.58 ± 5.3 cm, 83.97 ± 10.7 kg) were recruited for this study. Ten subjects were categorized as moderately trained (MT) who had been physically active for one year or more and participated in a moderate level of physical activity per week (3 or more hours of activity per week). The MT group did not compete or train in sport at a competitive level. Four subjects were categorized as highly trained (HT) who had been physically active for one year or more and participated in a high level of physical activity per week (6 hours or more activity per week or more). The HT group included one rower, one cross country runner and two hockey players, all of which trained at highly competitive level in their sport. This categorization was based on the Canadian Society of Exercise Physiology physical activity readiness questionnaire (PAR-Q). Subjects were instructed to refrain from heavy exercise 24 hours before testing. All subjects read and signed a consent form prior to experimentation. Memorial University of Newfoundland's Human Investigation Committee gave ethical approval for the study.

### 4.1.2 *Experimental Design*

Subjects partook in four experiment sessions that included one familiarization, two experimental trials, plyometric (PT) and resistance training (RT), and a control trial. These sessions were separated by a minimum of one-week period to ensure fatigue was not a

confounding factor. The PT and RT trials were randomized for each subject. Five moderately trained (MT) and 3 highly trained (HT) acted as control.

*Familiarization session.* On the first visit to the laboratory subjects completed a PAR-Q and consent form. Subjects' age, height, mass, training history, resting heart rate and blood pressure was recorded by experimenters. The session provided the opportunity for all subjects to become familiar with the testing protocol and equipment being used in the subsequent trials. In addition, the session was used to find each subject's one repetition maximum (RM) for a squat exercise. This information was needed to calculate the subject's 65% 1 RM required for the RT trial (Table 1). The mean 1 RM squat was 273.9 ± 63.9 kg and the mean 65% 1 RM was 177.1 ± 41.1 kg.

All subjects had previous experience performing a resisted squat. In order to ascertain the participants 1 RM squat load, the following procedure was followed. The subject performed a set of 5-10 squats without weight and the experimenters provided feedback to ensure that the correct technique was performed. The experimenters' increasingly added weight (10-20 kg) until approximately 75% of excepted 1 RM and 5 repetitions were performed at this weight. The subject was given a 5-minute rest period before the 1 RM squat test. The subject was asked to perform as many repetitions as possible at the estimated 95% of the excepted 1 RM. If the subject performed more then 5 repetitions the test was terminated and the subject was given a 5-minute rest period before performing 100% of their estimated 1 RM. If the subject was only able to perform 3 repetitions at 95% of their estimated 1 RM then the 1 RM was predicted from multiple-RM loads (3).

Subjects were also introduced to the plyometric protocol during the familiarization session. A demonstration of the drop jump was provided as well as verbal instructions were

given to the subjects to perform each jump with maximal effort. The jump was explained as a drop jump with maximal quickness and force once their feet contacted the ground. The goal of the drop jump was to achieve minimal contact time on force plate for each jump during the trial. No arm movement was allowed during the jump therefore subjects were instructed to place their hands on their hips. Subjects also maintained a jumping cadence of 70 beats per minute (bpm) (set by a metronome) to ensure similar jumping rates.

The subjects were introduced to and practiced performing isometric leg extension maximum voluntary contractions (MVC) to ensure they achieved a full MVC. They were directed to contract the right knee extensor as quickly and forcefully as possible. Subjects performed 5-6 repetitions with the goal of obtaining less than a 5% difference in force between two trials. A 3-minute rest period was allocated between each MVC. The subjects' were introduced to evoked stimulation at a submaximal voltage and amperage level of 100 volts and 50 amps for all stimulation protocols; twitch, 20 Hz and 100 Hz tetani. For the twitch, the stimulation level was progressively increased by an intensity of 20 milliamps until the maximal amperage of 1 amp was attained then the voltage was increased by 50 volts until the maximal stimulation was achieved. The stimulation level for the 100 Hz tetanus started at an initial stimulation of 1 amp and 100 volts followed by a 10 volts progressive increase in intensity until 30-40% of MVC force was observed. The identical stimulation intensity observed to give 30-40% of MVC during the 100 Hz tetanus was set for the 20 Hz tetanus.

*Testing Trials.* Blood pressure and resting heart rate were recorded prior to the start of the PT, RT and control sessions. Preceding experimental data collection, subjects underwent a 5-minute warm-up on a cycle ergometer at 75 rpm at a resistance of 1 kP. Heart rate and lactate were measured immediately following the completion of the warm-up. Subjects were seated on a

laboratory-designed apparatus used for testing leg extension force with hips and knees at 90°. The apparatus provided back support, chest and leg straps, hand grasp bars and a padded ankle strap that was attached to a strain gauge perpendicular to the lower limb to measure torque. The subjects' body was secured in this position to avoid extraneous movement that might affect signal recording.

*Plyometric trial.* Subjects began the trial standing with legs abducted and either foot situated on top of two 30 cm high custom designed plyometric boxes separated by a width of 40 cm (Figure 4.1 and 4.2). The metronome was set to 70 bpm and the subject was instructed to jump to the pace of the metronome making contact with the ground and boxes on every beat. Subjects were given verbal reminders to perform each jump with maximal quickness by allowing a minimal amount of contact time on the force plate as possible. A slight pause on the platform was required after the execution of each jump to ensure the subject stayed on pace with the metronome. The subject was given verbal warnings from the experimenter if they deviated from the pace of the metronome and the test was terminated if a third warning was given or if the subject self-terminated the trial.

*Resistance training trial.* The resisted squats required subjects to support an Olympic bar on their trapezius with 65% of their 1 RM in the "high-bar" position, with feet shoulder-width apart and knees slightly flexed. The heels remained in contact with the floor and the back in a neutral spine position (11). The hips and knees were flexed until a 90° angle at the knees was achieved, which was measured before the trial and a chair with additional height inserts was set to provide the subjects with a kinesthetic feedback of when 90° was achieved for each repetition. The metronome was set to 120 bpm, allowing two seconds for each eccentric and concentric

movement and the capability to maintain a slow continuous rhythm to complete one repetition. Three experimenters were required to act as spotters for each subject throughout the entire resisted squat trial. The subject was given verbal warnings from the experimenter if they deviated from the pace of the metronome and the test was terminated if a third warning was given or if the subject self-terminated the trial.

*Control Trial.* The subject passively rested for an individualized duration that was calculated from their mean time to exhaustion during the jumping and resistance training trials.

The pretest protocol involved the following procedure prior to the intervention. i) An evoked single pulse twitch on the right quadriceps was performed until a maximal force twitch was found; a plateau in twitch force indicated this. ii) The subjects passively rested for two minutes and were then instructed to perform 2 to 3 maximal voluntary contractions (MVC) of the right quadriceps during a unilateral knee extension for 3 seconds duration while receiving verbal encouragement. There was 3 minutes of passive rest allocated between each MVC and the completion of the third maximal contraction was dependent on a greater than 5% difference between the first two MVCs performed. iii) The subject passively rested for an additional three minutes and then an initial stimulation of 1 amp and 100 volts for a 100 Hz tetanus on the right quadriceps was performed followed by a 10 volt progressive increase in intensity until 30-40% of MVC force was observed. Three minutes of rest followed and the identical stimulation intensity observed to give 30-40% of MVC during the 100 Hz tetanus was set for the 20 Hz tetanus used to stimulate the quadriceps.

After the completion of the pre testing protocol the PT or RT trial was performed until subjects became exhausted. Subjects were given verbal encouragement throughout each testing

trial to ensure failure of the exercise was achieved. Heart rate was recorded immediately following exhaustion and the subject was set-up on the seated leg extension apparatus. The post-test protocol involved the delivery of a maximal evoked twitch, immediately followed by a 3 second MVC and 20 Hz and100 Hz tetanus (Figure 4.3, 4.5 and 4.6). This protocol was approximately 25 seconds in duration and was repeated during preset intervals of 1, 3, 5, 10, 20, 30, 40 and 60 minutes following exhaustion. Heart rate was recorded immediately post, and at 1, 3, 5 and 10 minutes and lactate was recorded at 3, 10, and 20 minutes following exhaustion.

### 4.1.3 *Measurements and Instruments*

*Heart Rate.* Heart rate was monitored via the S 802 Polar Edge heart rate monitor model (Polar Electro, Oy, Finland). Conduction gel (Signa Crème, Parker Laboratories, Fairfield, NJ) was used on the adjustable chest strap to reduce the resistance of the signal.

*Lactate.* Blood lactate was sampled from the right hand in a pronated position alternating between the index and middle fingers. The subject's finger was cleansed with a 70% isopropyl alcohol swab and allowed to air dry prior to collection. The finger was pierced with a sterilized lancet using a Accu-Chek® Softclix® (Laval, Quebec) needle pen. The first few drops were negated and wiped away with a sterilized gauze to avoid potential contamination. The next drop was collected and analyzed using an Arkray® Lactate Pro (Nakagyo-ku, Japan) blood lactate test meter.

*Electrical Stimulation.* Bipolar surface stimulating electrodes were prepared in the laboratory from paper surrounding aluminum foil and conduction gel (Signa Crème, Parker

Laboratories, Fairfield, NJ) dampened with water. The electrodes were 4-5 centimeter in width and individually constructed in length to ensure coverage of the width of the muscle belly. The positions of electrodes were marked and measured to guarantee that they were placed in the same location on subsequent visits. Stimulating electrodes were secured over the proximal (femoral triangle) and distal (immediately superior to the patella) portions of the quadriceps. Peak twitches and tetani were evoked with electrodes connected to a high-voltage stimulator (Digitimer Stimulator Model DS7H+, Hertfordshire, UK). The amperage (10 milliamps (mA)-1amp (A)) and duration (50 micro-seconds ($\mu$s)) of a 100-200 volt square-wave signal pulse until the evoked force output reached a plateau indicating a maximum twitch torque was attained. The tetanus used to represent high frequency fatigue was set at an individualized voltage and amperage that elicited 30-40% of MVC peak force during a 100 Hz square wave signal stimulation 300ms in duration (5). The voltage and amperage to elicit this force was also used for a 20 Hz square wave signal stimulation 700 ms long that is used to represent low frequency fatigue (5).

*EMG.* Two surface EMG recording electrodes (MediTrace Pellet Ag/AgCl electrodes, Graphic Controls Ltd., Buffalo, NY) were aligned parallel to the muscle fiber (dimensions 3 x 2 cm) at the midpoint of the anterior superior iliac spine and the patella on the mid-belly of the rectus femoris. A ground electrode was secured on the head of the fibula. EMG location sites were shaved, sanded (to remove dead epithelial cells), and cleansed with an alcohol pad to maximize EMG sensitivity. EMG signals were then monitored and stored on the computer. EMG activity was sampled at 2000 Hz, with a Blackman -61 dB band-pass filter between 10-500 Hz, amplified (bi-polar differential amplifier, input impedance = 2M$\Omega$, common mode rejection ratio $\geq$ 110 dB min (50/60 Hz), gain x 2000, noise $\geq$ 5 $\mu$V), and analog-to-digitally converted (12

bit) and stored on personal computer for further analysis. The EMG twitch property examined was amplitude of the muscle compound action potential (M Wave) of the rectus femoris (Figure 4.4). The EMG during the MVC was rectified and integrated during a preset interval of 500ms before and after the highest force amplitude of MVC of the rectus femoris. The iEMG was normalized to the peak RMS during the pre fatigue MVC test for every recovery period in each trial. The iEMG and M Wave measurements were calculated as a percentage of the pretest for all recovery periods during each trial.

*Torque.* Voluntary and evoked contractile force were measured via a high-tension wire clamped to a Wheatstone bridge configuration strain gauge (Omega Engineering Inc. LCCA 250, Don Mills, Ontario) perpendicular to the line of pull of the lower limb. Torques produced from muscular contractions were detected via a strain gauge, amplified (Biopac Systems Inc., Holliston, MA, USA, DA 100; analog to digital converter MP100WSW) and monitored on a computer. All data were stored on a computer at a sampling rate of 2000 Hertz (Hz). Data were recorded and analyzed with a commercially designed program (AcqKnowledge III, Biopac Systems Inc., Holliston, MA, USA). The twitch and MVC properties examined were peak-to-peak force (the baseline to highest force measurement). The 20 Hz and 100 Hz tetanus property examined was the mean torque during the last 50 ms of stimulation. The force measurements were calculated as a percentage of the pretest force for all recovery periods during each trial.

The EMG twitch property examined was amplitude of the muscle compound action potential (M Wave) of the rectus femoris. The EMG during the MVC was rectified and integrated during a preset interval of 500ms before and after the highest force amplitude of MVC of the rectus femoris. The iEMG was normalized to the peak RMS during the pre fatigue MVC

test for every recovery period in each trial. The iEMG and M Wave measurements were calculated as a percentage of the pretest for all recovery periods during each trial.

### 4.1.4 *Statistical Analysis*

A three-way ANOVA with repeated measures on the third factor was performed on all pre and post-interventions value (SPSS 18.0 for Macintosh). The three factors (2 x 3 x 9) included physical activity level (trained and moderately trained), condition (plyometric, resistance and control) and time (pre-fatigue and recovery periods of 1, 3, 5, 10, 20, 30, 40 and 60 minutes). Assumptions of sphericity were tested using Mauchley's test and degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity. *F*-ratios were considered statistical significant at the $p < 0.05$ levels. A Bonferroni post-hoc test and multiple *t*-test for paired samples were performed to test for significant differences between main effects and interactions, respectively. Descriptive statistics in text include means ± standard deviations (SD). Data in figures include means ± standard errors (SE).

## 4.2 RESULTS

Overall, the training status affected the subjects' duration of the PT trial. Highly trained (HT) subjects performed the PT trial for a longer duration than the moderately trained (MT) subjects during plyometric training until exhaustion. However, training status of the group did not affect the duration of the resisted squat trial. Subjects evoked contractile properties and voluntary activation showed similar patterns of recovery following both the PT and RT trial. The training status induced difference in the duration of the PT trial did not produce differences in

evoked contractile properties or voluntary activation. Heart rate and blood lactate measures to determine exercise intensity were higher following the PT trial compared to the RT trial; however changes were not training status dependent.

*Duration of plyometric and resistance training trials.* The PT trial duration was dependent on training status. The HT subjects jumped for a duration that was significantly ($p$ = 0.025) longer (68.2%) than the MT subjects. The HT subjects' PT duration was significantly ($p$ = 0.025) longer (67.1%) than the RT trial. In comparison, the duration of plyometric and resisted squat trial for the MT subjects was not significantly different (Figure 4.7).

*Heart rate and blood lactate measures during the plyometric and resistance training trials.* Heart rate and blood lactate levels attained during the PT and RT trials were not training status dependent. However, there was a significant interaction ($p < 0.001$) between the PT and RT trials for heart rate levels (Figure 4.8). At 1-minute recovery the mean heart rate level was significantly ($p < 0.001$) higher (13.4%) following the PT trial compared to the RT trial. There was also a significant interaction ($p = 0.025$) for blood lactate levels between the PT and RT trials (Figure 4.9). At 3 minutes recovery, the mean lactate level was significantly ($p = 0.011$) higher (25.6%) following the PT trial compared to the RT trial (Figure 4.9).

*Evoked twitch contractile properties.* Changes in peak twitch force during all recovery periods were similar following both the PT and RT trials and were not dependent on training status. However, there was a significant difference ($p < 0.000$) between the PT and RT trials and the control trial for the evoked contractile measures (Figure 4.10). At the 1 min recovery period, the peak twitch force was 29.7% and 31.6% significantly ($p < 0.05$) less than the control for both

the PT and RT trials, respectively. The mean peak twitch force began to increase towards a prefatigued state for both the exhaustive trials. At the 3 minutes of recovery the PT and RT trials increased to 95.4 ± 9.4 % and 94.27 ± 11.8 %, respectively, of the pre fatigue force. However, the peak twitch force following the control trial also increased above the pre testing force (111. 42 ± 15.0 %). Thus, the PT and RT trials remained significantly ($p < 0.05$) less than the control. After 5 minutes of recovery a secondary decrease in peak twitch force occurred for all trials. This reveals a methodological fatigue effect as peak twitch force also decreased for the control trial. However, the resisted squat trial did show a significantly ($p < 0.05$) greater decrease (14.6% and 14.8%, respectively) in force from the control trial at 40 and 60 minutes of recovery.

M wave measurements were not affected by the training status of the group and there was no significant difference between the PT, RT and control trials during any of the recovery-testing periods. There was a significant main effect ($p < 0.000$) of time as a continuous decrease in the M wave occurred during the recovery period for all three trials. Data from each trial was collapsed to see the overall effect of recovery period on changes in M Wave. There was a significant ($p < 0.05$) difference in M wave amplitude for all recovery periods from the pretest force. An average decrease of 15.6% in M Wave amplitude occurred over the 60-minute recovery period for all three trials.

*Low and high frequency fatigue.* Changes in low and high frequency torques during the total duration of the recovery period were not different between the PT, RT and control trials. Changes in low and high frequency torques did not have a dependence on the training status of the group in this experiment.

*Voluntary activation and force.* MVC measures were not dependent on training status of

the group in this experiment. A significant main effect ($p < 0.000$) of time occurred during the recovery period for the PT, RT and control trials. The testing protocol appeared to induce fatigue, which was observed following the control trial, as the average MVC force during the 60-minute recovery period was 10.2% below the pretest force. Following the plyometric and resisted squat trials, the MVC torque decreased by 16.1% and 18.0% at the 1 min recovery and by 11.8% and 10.0%, respectively, at the 3 minutes of recovery, which was significantly ($p < 0.05$) different from the control trial. All trials followed a similar pattern of changes in MVC force from 5 to 60 minutes of recovery (Figure 4.11).

The iEMG was normalized to the peak RMS during the pre fatigue MVC test for every recovery period in each trial. The iEMG was calculated as a percentage of the pre fatigue test for all recovery testing periods. iEMG was not dependent on the training status of the group during the recovery period for the PT and RT trials in this experiment. At 1 min recovery both the plyometric and resisted squat trials were significantly ($p < 0.000$) less than the iEMG measure of control trial by 20.6% and 32.1%, respectively. The RT trial continued to remain significantly ($p < 0.04$) lower than the control during the 3 min recovery period. There were no significant differences in iEMG measures after the 3 minutes of recovery between the PT, RT and the control trials (Figure 4.12).

## 4.3 DISCUSSION

The most important finding in the study was that there was no difference in the neuromuscular recovery response following plyometric and resistance training trials performed to exhaustion. Furthermore, the present study revealed that the duration of the PT was dependent on the training status of the subject, HT subjects jumped significantly longer (68.2%) than the

MT subjects. In addition, the duration of the PT trial was significantly longer (67.1%) than RT trial for the HT subjects; however the duration of the trials did not differ for the MT subjects. Overall, the present study showed that changes in neuromuscular properties following plyometric and resisted squat trials to exhaustion are independent of the type of exercise or its duration and training status of the subject.

To our knowledge, this is the first study to reveal a similar recovery from neuromuscular fatigue of the quadriceps following PT and RT performed to exhaustion. The results from the present study have shown that when submaximal exercise is performed to a state of exhaustion the neuromuscular response is similar regardless of the velocity of the SSC movement. The resisted squat exercise utilized a slow SSC movement due to the long duration of the pre-stretch that is performed with a low velocity movement followed by a prolonged transition period between the eccentric and concentric phases. Benefits gained from the absorption of power and storage of elastic energy known to occur during the eccentric contraction is decreased during a slow SSC action (23). The long duration of the pre-stretch produces a stiffer muscle-tendon unit and lengthens the contractile properties leading to a loss of mechanical energy from the deattachment of cross-bridges (13). Slow SSC movements involve a minimal contribution of the passive elastic elements thus leading to a greater reliance on contractile properties of the muscle for force production, whereas force development during a rapid SSC movement has a greater dependence on the storage of elastic energy (11). During the plyometric exercise, the rapid SSC action allows the cross-bridges to remain attached during the stretch, aiding in the augmentation of force during the concentric phase (10). Although the velocity of the pre-stretch and the duration of the transition between phases differ between the exercises, potentially imposing different mechanical stresses on muscle, the neuromuscular responses behaved in a similar manner when both exercises were performed to a state of exhaustion.

Although no study has compared the neuromuscular responses following exercise involving slow and rapid SSC movements found in resistance training and plyometric exercises, respectively, other studies have compared different types of SSC actions. Nicol et al. (32) observed similar findings when examining the effects of different intensity and duration rapid SSC exercises on the neuromuscular fatigue response. The results of the study showed acute and delayed impairments in neuromuscular function, as measured by reflex EMG, twitch torque and active resistance to stretch, were independent of the type of exercise performed when comparing a maximal intensity 10 km run and a bout of exhaustive rebound exercise on a sledge apparatus. Similarly, Benson et al. (8) observed similar impairments in neuromuscular function following different intensity slow resisted SSC exercises. Three sets of 100% of their 10 repetition maximum (RM) load to failure and three sets of 90% of their 10 RM load with only the last set performed to failure (with 3 minutes of rest between each set for each trial) induced comparable decreases in MVC and iEMG. Not only has similar neuromuscular fatigue responses been observed to occur following different types of SSC movements but Toumi et al. (43) found a similar fatigue response following consecutive sets of 10 repetitions of SSC exercises on a sledge apparatus compared to successive sets of 10 repetitions of isometric contractions, with both exercises performed at 70% of subject's MVC with 2 minutes of rest between each set until exhaustion. Interestingly, the subjects were able to perform 3 more sets of the SSC exercise compared to the isometric exercise before complete exhaustion (43). The present study also observed that although the duration of both trials was similar for the MT subjects, the number of repetitions performed during the PT trial was greater due to the higher cadence of the exercise performed compared to the RT trial. Thus, the slow SSC movement utilized during the resisted squat trial may have decreased the ability to perform the same number of repetitions as the plyometric trial due to the greater reliance of the contractile properties of the muscle to produce

force. The rapid SSC movement during the PT trial utilizes a greater proportion of elastic energy from the muscle-tendon unit to enhance force, possibly aiding in a greater number of repetitions performed. Based on the findings from the above studies and the current study, it is believed that when an exercise is performed to a state of exhaustion a similar neuromuscular fatigue response occurs regardless of SSC utilized.

*Neuromuscular properties are not SSC speed dependent*

The acute neuromuscular fatigue response showed similar contractile properties following PT and RT exercise leading to impairments of the force-production capabilities of the muscle. Both types of slow and rapid SSC exercises, although never previously compared in a single study, have shown immediate reduction in maximal force production (45, 25, 17, 41), reduced electrically evoked force (45, 31, 36, 37) and reduced neural drive (9, 17, 41). In accordance with these findings, the present study revealed an acute impairment in the evoked and voluntary contractile properties of the muscle following PT and RT exercises to exhaustion.

The peak twitch force showed a biphasic recovery pattern following both the PT and RT trial, which is a typical fatigue response following exercise originally reported by Thompson et al. (42) in the frog model, commonly reported following SSC exercises in the human model (24). In the present study, the peak twitch force was depressed for the first 3 minutes of recovery and progressed towards a pre-fatigue state until a secondary decrease in force occurred during 40 to 60 minutes of recovery with the greatest depression observed following the resisted squat trial. Strojnik & Komi (40) also observed an acute decrease in peak twitch force approximately 2.5 minutes following maximal intensity drop jumps, which utilized rapid SSC movements until exhaustion, however no further measurements were taken after the 2.5 minute post-testing period. Similarly, Skurvydas et al. (37) revealed that both SSC fatiguing protocols, 100

intermittent drop jumps and 100 continuous drop jumps to exhaustion, induced an immediate decrease in peak twitch force at the 2 min recovery period and a secondary decrease at 20 minutes of recovery. Studies examining exercises involving slow SSC movements have also shown this biphasic recovery pattern. Raastad et al. (35) observed a decrease in electrically induced contractions between 5-20 min after strength exercise protocols that consisted squats and bilateral knee extensions. In the present study, peak twitch force decreased by 28% and 26% at 1 minute and 6% and 10% at 3 minutes of recovery post-fatigue for the squat and plyometric trials, respectively. Thus, the location of fatigue is partly of peripheral origin. The immediate changes in peak twitch force following both trials are believed to be the result of metabolic changes, which include a reduction in creatine phosphate (CrP) concentration, increased inorganic phosphate (Pi) and increased hydrogen ion concentrations (pH) (45). Metabolic changes have been shown to reach peak values within the first 5 minutes following exercise involving slow and rapid SSC movements as well as isometric contractions to exhaustion (35, 40, 4). Similarly, plyometric and resisted squat exercises to exhaustion showed a peak in blood lactate measures at 3 minutes of recovery and reached near-control values within 20 minutes following both exercises. The reduction in force could be associated with a reduced $Ca^{2+}$ release from the sacroplasmic reticulum and/or reduced capability of cross-bridges to form strong binding (30).

The secondary decrease in peak twitch force between the 40 and 60 minutes of recovery following the resisted squat trial is hypothesized to be the result of structural muscle changes. In the present study, the resisted squat trial induced a greater reduction in peak twitch force that could be the result of a decrease in $Ca^{2+}$ release due to disruptions of contractile proteins. Staron et al. (39) reported that unaccustomed high intensity resistance training exercises performed to fatigue induced myofibrillar disorganization due to the shear forces pulling at the Z-lines causing them to rupture. This disruption in contractile properties has been reported to occur following

pure concentric and eccentric strength exercise in untrained subjects (15). This also occurs in trained subjects following squats and bilateral contractions (35).

The M Wave measure showed a slight decrease over the total 60-minute duration of recovery following the both trials, however it was not significantly less than the control trial. Changes in the M wave indicate alterations in neuromuscular propagation and a decrease in the measure could be a reduction in the excitability of the muscle fiber membrane (2). The observed non-significant change in M wave amplitude suggests the neuromuscular excitability of the muscle was not impaired even when the exercises were performed to a state of exhaustion. This is possibly due to the dynamic nature of the exercises, which is in agreement with previous studies using dynamic contractions (22) or isokinetic movements (33), where long sustained isometric contractions (14) led to a decrease in M wave amplitude.

The MVC force was immediately depressed during the first 3 minutes following both PT and RT trials and slightly recovered pre-fatigue force level over the 60-minute recovery period. In the present study, MVC force decreased 18% and 17% at 1 minute and 11% and 12% at 3 minutes of recovery in MVC force following the PT and RT trials, respectively. Similar decreases in voluntary force have been reported by other studies regardless of the involvement of slow or rapid SSC movements utilized in exercises performed to exhaustion. A decrease in isometric MVC force between 15-25% has been found to occur immediately following resistance training exercises using a slow SSC movement (8, 22, 24) and a 13-30% decrease following rapid SSC movements (36, 40, 25, 1, 20). Following both the PT and RT trials the impairment in the voluntary force generating capability of the muscle was accompanied by a reduction in iEMG during the first 3 minutes of recovery. Thus central fatigue was also partly responsible for the loss of force production because there was a decrease in neural activation immediately following exercise involving slow and rapid SSC movements. In the present study iEMG was lower at 1

and 3 minutes compared to pre-fatigue iEMG, with no differences thereafter for both SSC trials. The recovery of central fatigue and the restoration of neural activation seem to occur within 5 minutes following different fatiguing exercises (6). The recovery of neural activation following exercises involving slow and rapid SSC movements to exhaustion in the current study appear to behave in a similar manner.

A minimal level of LFF was observed to occur following both the PT and RT trials, however the force did not decrease to a level that indicates LFF to be a major contributor to peripheral fatigue. HFF was not observed to occur following either PT or RT trials. Heart rate and blood lactate levels were higher following the PT trial than the RT trial for both HT and MT subjects. However, the greater blood lactate levels and cardiovascular stress induced by the plyometric trial was not large enough to produce a differing neuromuscular response during the 60 minute recovery period compared to the resisted squat trial.

*Training Status*

In the present study the duration of the PT trial was dependent on the training status of the subject. Although the HT subjects had minimal or no prior plyometric training experience, it is possible that their training included explosive type movements that involved rapid SSC actions to accelerate the body forward. Sprint training, which is typically involved in most sport, is an explosive movement that has been shown to be comparable or superior to plyometric training. It has been shown to improve leg extensor strength, power, sprint and agility performance due to increased activation of the leg musculature and involvement of both concentric and rapid SSC movements to generate force production (27).

Explosive type training has not only been shown to increase leg muscle power but different neural adaptation strategies during high impact SSC movement have been observed in

different groups of athletes due to training specificity of their sport (2). Exercise involving SSC movements has been suggested to modulate the reflex loop in the muscle, which is believed to alter the sensitivity of Ia afferents, changes in $\alpha$ - $\gamma$ motoneuron linkage, or inhibition from the Golgi tendon organ (17). In addition, changes in the reflex system have been closely linked to changes in the stiffness of the muscle-tendon unit during landing phase of SSC movement (19). A stiff muscle-tendon unit increases concentric power production during the SSC movement due an increased ability of the unit to store and release kinetic energy (18). Avela et al. (2), based on findings of different athletic populations, proposed that athletes with a greater exposure to high impact loading may have some structural adaptations thus preventing exercise induced reflex inhibition during high impact SSC movements. In addition, previous studies have demonstrated changes in reflex excitability following different types of training (endurance and hopping training) involving SSC movement (34, 44). In the present study, an increased exposure to rapid SSC movement during training and competition in high level sport may have aided the HT subjects to have superior stiffness control of the muscle-tendon unit due a more experienced and efficient reflex system. Therefore, the interaction between the reflex and stiffness control of the muscle-tendon unit would enable the HT subjects to jump 68% longer than the MT subjects during the PT trial in the present study.

In the present study, the maximal strength during the RT trial was not dependent on the training status. The average predicted 1 RM squat for the HT subjects was 124.1 kg compared to 124.2 kg for the MT subjects. Greater maximal strength is associated with greater power absorption during the eccentric phase of slow large-amplitude SSC movements for heavier loading intensity (80% 1 RM). It is also associated with better power production during the concentric phase for a lighter loading intensity (40% 1 RM) (11). The RT trial in the current study, which required subjects to perform 65% of their 1 RM squat is considered a moderate

loading intensity, thus maximal strength would influence both power absorption and production during the slow SSC movement. However, as the maximal strength did not different between the groups, the contribution of maximal strength on the power absorption and production during the slow SSC movement during the RT trial was similar. This was evident by the non-significant difference in the duration of the resisted squat between the HT and MT groups.

The PT trial used an absolute drop jump height of 30 cm for all subjects as this height has been previously found to result in the greatest jump height and lowest contact time when compared to drop jump heights of 45 and 60 cm in MT subjects. However, the magnitude of these changes between drop jump heights of 30 to 60 cm was minimal (48). Studies have used relative drop jump heights based on percentages of maximum jump height when examining the effects of SSC exercises on fatigue (21). However unpublished data (46), has shown that the maximum jump height among HT subjects was 29.9 cm; we feel that a 30 cm drop jump height was within an appropriate range to ensure both MT and HT subjects were able to perform the SSC exercise over an extended period of time with maximum height and minimal contact time. In addition, MVC force has been associated with dynamic performances, including vertical jump (26). In this study, the MVC force was 791.69 N and 813.2 N for the HT and MT subjects, respectively. As jump height is dependent upon achieving high velocities and power output, the HT subjects did not have an advantage on the absolute drop jump height because the cadence of the jump was controlled and the force production was similar between training status groups (26). Therefore, we feel that the HT and MT subjects underwent similar mechanisms of fatigue during the PT trial. However, as the RT was performed using relative loading protocol future studies using relative loading during plyometric exercises may be of interest when comparing slow and fast SSC exercises.

Similar to the findings from this study, Skurvydas et al. (38) also found that the time-course of muscle force recovery was independent of the training status of the subject. There was no difference in muscle force recovery among sprinters, long distance runner and untrained persons following the performance of intermittent rapid SSC exercises with maximal intensity. The time recovery for neuromuscular properties following exercise has been shown to be dependent upon many factors including the degree of eccentric force production involved and the training status of the subjects (35), however the findings of this study concluded that when the subject's fatigue level reaches a state of exhaustion the subsequent recovery pattern is independent of any these variables.

## 4.4 CONCLUSION

The duration of the PT trial was dependent on the training status of the subject, whereas the recovery of all neuromuscular properties following the PT and RT trial were independent of the duration of the trial, the training status of the subject, or the type of SSC movement utilized during the exercise. The only exception was during 40- 60 minutes of recovery when the peak twitch force was slightly more depressed following the resisted squat trial. This is possible due to the increased stress on the contractile properties of the muscle during the slow SSC movement leading to greater disruptions of the contractile proteins in the muscle. Based on the findings from the present study, when submaximal exercise is performed to a state of exhaustion the recovery of neuromuscular properties behave in a similar manner regardless of the velocity of the prestretch of the SSC movement. Therefore, when developing a plyometric training program for elite or recreational athletes the duration of recovery following bouts of submaximal drop jumps to exhaustion may be similar to that prescribed in resistance training programs. Further research

should focus on recovery from sets of submaximal plyometric and resistance training exercises. After performing an initial fatiguing test, such as performed in the present study, a preset relative intensity level of a percentage of the number of repetitions performed until exhaustion to determine if recovery patterns continue to behave in a similar manner.

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## 4.6 FIGURE LEGEND

**Figure 4.1:** Illustrations of the plyometric training (PT) apparatus; pre-drop phase onto the ground from boxes.

**Figure 4.2:** Illustrations of the plyometric training (PT) apparatus; pre-jump phase onto 30 cm boxes from ground.

**Figure 4.3:** Figure is a data recording of quadriceps EMG (top) and force output (bottom) during an evoked twitched followed by an MVC.

**Figure 4.4:** A magnified example of an EMG recording from an evoked twitch stimulation representing an M Wave.

**Figure 4.5:** Figure is a data recording of EMG (top) and force output of the quadriceps during an evoked 20 Hz stimulation.

**Figure 4.6:** Figure is a data recording of EMG (top) and force output of the quadriceps during an evoked 100 Hz stimulation.

**Figure 4.7:** The duration of plyometric (PT), resistance training (RT) and control trials. An asterisk (*) indicates significant differences ($p < 0.05$) between training status. A star (★) indicates significant differences ($p < 0.05$) within training status.

**Figure 4.8:** Mean heart rate (± SE) during the testing periods (pre fatigue, after warm-up,

immediately post exercise, and 3, 5 and 10 minutes of recovery) for each trial. An asterisk (*) indicates significant differences ($p < 0.05$) between both the plyometric (PT) and resistance training (RT) trials from the control trial. A star (★) indicates significant differences ($p < 0.05$) between the PT and RT trials.

**Figure 4.9:** Mean blood lactate (± SE) during the testing periods (pre fatigue, after warm-up and 3 and 10 minutes post recovery) all three trials. An asterisk (*) indicates significant differences ($p < 0.05$) between both the plyometric (PT) and resistance training (RT) and trials from the control trial. A star (★) indicates significant differences ($p < 0.05$) between the PT and RT trials.

**Figure 4.10:** Mean percentage change of peak twitch force (± SE) during the testing periods (pre fatigue and 1, 3, 5, 10, 20, 30, 40 and 60 min post recovery) for all three trials. An asterisk (*) indicates significant differences ($p < 0.05$) between both the plyometric (PT) and resistance training (RT) trials from the control trial. A cross (†) indicates significant differences ($p < 0.05$) between the RT trial and control trial.

**Figure 4.11:** Mean percentage change of maximal voluntary contraction (MVC) (± SE) during the testing periods (pre fatigue and 1, 3, 5, 10, 20, 30, 40 and 60 min post recovery) for all three trials. An asterisk (*) indicates significant differences ($p < 0.05$) between both the plyometric (PT) and resistance training (RT) trials from the control trial.

**Figure 4.12:** Mean percentage change of integrated EMG (iEMG) (± SE) during the testing periods (pre fatigue and 1, 3, 5, 10, 20, 30, 40 and 60 min post recovery) for all three trials. An asterisk (*) indicates significant differences ($p < 0.05$) between both the plyometric (PT) and

resistance training (RT) trials from the control trial. A cross (†) indicates significant differences ($p < 0.05$) between the RT trial and control trial.

Table 4.1. Experimental Protocol:

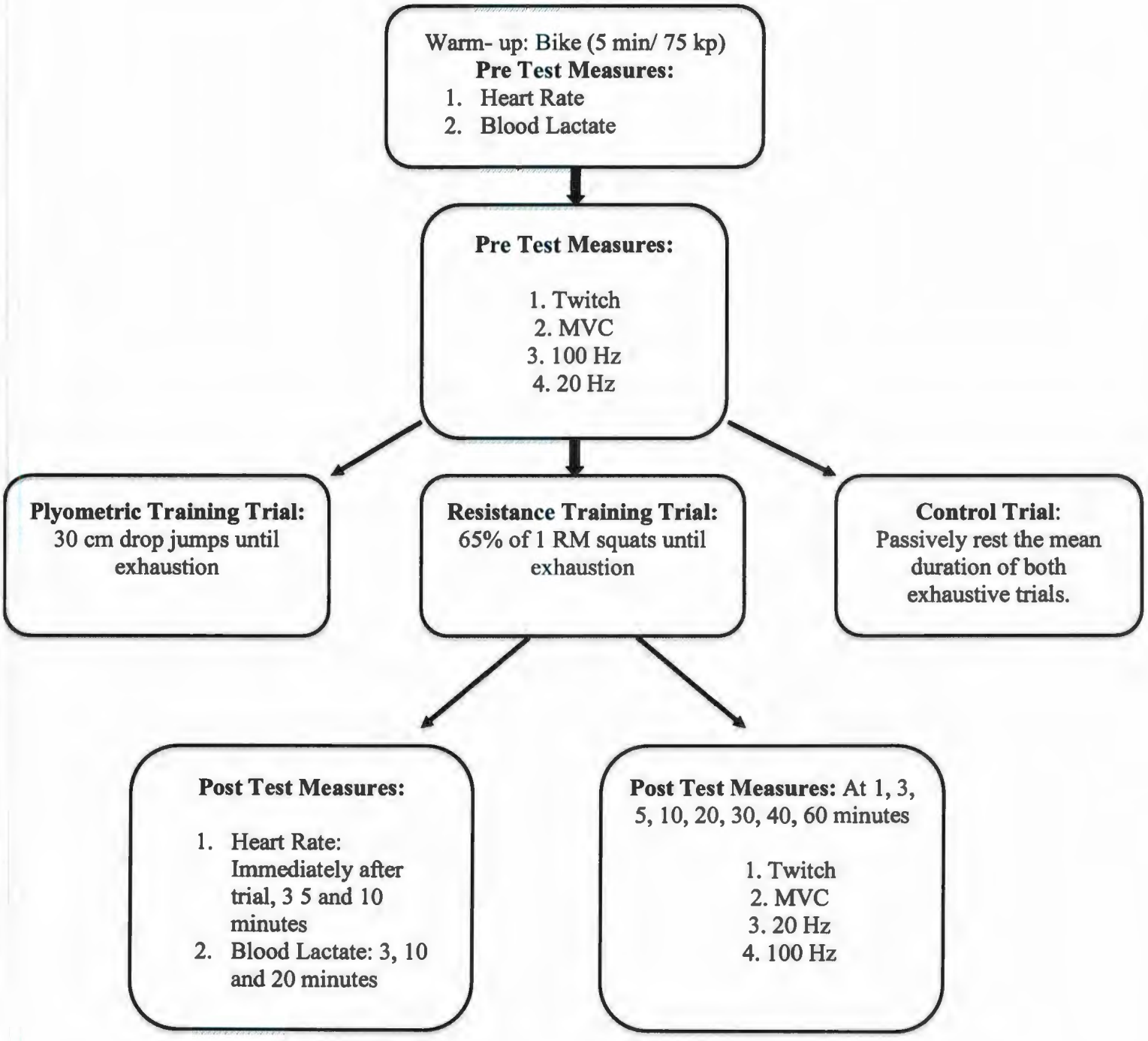


Table 4.2. Subject Information

| | Weight (kg) | Height (cm) | 1 RM Squat (kg) |
|---|---|---|---|
| Moderately Trained | 85.3 | 180.1 | 124.2 |
| Highly Trained | 78.4 | 177.5 | 124.1 |

## Table 4.3. Physiological Measures during pre-test and recovery

| Measures | Pretest | | | Post 1 min | | | Post 3 min | | |
|---|---|---|---|---|---|---|---|---|---|
| | Squat | Jump | Control | Squat | Jump | Control | Squat | Jump | Control |
| Heart Rate | 66.5 ± 11.42 | 66.6 ± 11.0 | 66.7 ± 9.54 | 139.5 ± 9.10 | 161.0 ± 24.9 | 71.5 ± 12.30 | 99.0 ± 15.45 | 109.2 ± 12.34 | 72.2 ± 16.15 |
| Lactate | 1.8 ± 0.472 | 1.7 ± 0.63 | 2.5 ± 1.12 | n/a | n/a | n/a | 9.1 ± 1.74 | 12.3 ± 2.78 | 2.0 ± 0.60 |
| *Maximal Twitch* | | | | | | | | | |
| M-Wave | 3.8 ± 1.70 | 4.6 ± 2.39 | 1.23 ± 0.58 | 3.13 ± 1.60 | 3.9 ± 1.69 | 1.17 ± 0.53 | 3.0 ± 1.22 | 3.7 ± 1.80 | 1.1 ± 0.51 |
| P-P Torque | 167.7 ± 39.69 | 151.0 ± 34.7 | 151.8 ± 31.81 | 119.5± 32.69 | 110.8 ± 28.78 | 157.2 ± 35.0 | 156.7 ± 28.15 | 136.3 ± 30.59 | 161.5 ± 31.92 |
| *MVC* | | | | | | | | | |
| iEMG | 0.25 ± 0.032 | 0.26 ± 0.015 | 0.30 ± 0.012 | 0.22 ± 0.02 | 0.19 ± 0.045 | 0.32 ± 0.027 | 0.23 ± 0.037 | 0.23 ± 0.058 | 0.30 ± 0.038 |
| P-P Torque | 810.2 ± 160.89 | 806.6 ± 156.4 | 774.4 ± 156.33 | 670.5 ± 175.10 | 676.1 ± 146.0 | 743.7 ± 153.05 | 727.1 ± 147.19 | 710.1 ± 137.28 | 734.3 ± 142.76 |
| *Tetanus* | | | | | | | | | |
| 20 Hz Torque | 232.4 ± 55.97 | 236.9 ± 65.61 | 235.1 ± 47.33 | 196.9 ± 22.7 | 214.3 ± 42.5 | 235.9 ± 55.79 | 209.8 ± 34.1 | 220.20 ± 51.46 | 258.5 ± 67.18 |
| 100 Hz Torque | 303.4 ± 26.51 | 295.4 ± 67.80 | 272.7 ± 68.65 | 231.8 ± 21.4 | 269.4 ± 56.1 | 268.3 ± 78.43 | 278.4 ± 41.25 | 279.9 ± 49.37 | 290.3 ± 97.52 |
| **Measures** | Post 5 min | | | Post 10 min | | | Post 20 min | | |
| | Squat | Jump | Control | Squat | Jump | Control | Squat | Jump | Control |
| Heart Rate | 90.0 ± 12.2 | 97.12 ± 11.03 | 70.3 ± 10.25 | 88.6 ± 15.32 | 89.6 ± 10.21 | 66.75 ± 9.43 | n/a | n/a | n/a |
| Lactate | n/a | n/a | n/a | 8.2 ± 3.55 | 10.3 ± 1.94 | 1.6 ± 0.30 | 5.0 ± 1.57 | 6.6 ± 1.80 | 1.6 ± 0.30 |
| *Maximal Twitch* | | | | | | | | | |
| M-Wave | 2.8 ± 1.22 | 3.5 ± 1.5 | 1.1 ± 0.52 | 2.7 ± 0.97 | 3.8 ± 1.9 | 1.12 ± 0.53 | 2.7 ± 0.86 | 3.4 ± 1.78 | 1.1 ± 0.46 |
| P-P Torque | 158.4 ± 30.87 | 143.4 ± 41.79 | 167.9 ± 38.38 | 147.3 ± 27.79 | 137.4 ± 43.62 | 152.1 ± 28.15 | 132.0 ± 25.09 | 130.1 ± 37.43 | 137.1 ± 25.78 |
| *MVC* | | | | | | | | | |
| iEMG | 0.24 ± 0.029 | 0.21 ± 0.065 | 0.27 ± 0.048 | 0.25 ± 0.042 | 0.23 ± 0.049 | 0.29 ± 0.042 | 0.24 ± 0.039 | 0.23 ± 0.061 | 0.28 ± 0.038 |
| P-P Torque | 730.0 ± 160.76 | 705.2 ± 143.26 | 716.2 ± 163.9 | 744.8 ± 147.64 | 722.0 ± 151.40 | 713.18 ± 143.33 | 710.2 ± 141.70 | 704.4± 149.58 | 682.9 ± 147.52 |
| *Tetanus* | | | | | | | | | |
| 20 Hz Torque | 202.8 ± 30.32 | 210.2 ± 57.58 | 251.6 ± 55.46 | 197.9 ± 27.8 | 213.1 ± 72.13 | 252.5 ± 56.89 | 184.8 ± 25.94 | 220.74 ± 36.7 | 246.29 ± 56.64 |
| 100 Hz Torque | 277.1 ± 24.99 | 276.9 ± 64.91 | 297.1 ± 76.81 | 265.0 ± 16.66 | 273.4 ± 79.95 | 282.9 ± 91.07 | 258.5 ± 35.17 | 253.3 ± 47.74 | 285.8 ± 87.08 |
| **Measures** | Post 30 min | | | Post 40 min | | | Post 60 min | | |
| | Squat | Jump | Control | Squat | Jump | Control | Squat | Jump | Control |
| Heart Rate | | | | | | | | | |
| Lactate | | | | | | | | | |
| *Maximal Twitch* | | | | | | | | | |
| M-Wave | 2.9 ± 0.92 | 3.4 ± 1.99 | 1.0 ± 0.40 | 2.6 ± 0.89 | 3.5 ± 2.04 | 1.03 ± 0.43 | 2.8 ± 0.81 | 3.3 ± 1.94 | 1.0 ± 0.39 |
| P-P Torque | 124.5 ± 24.59 | 122.5 ± 35.03 | 134.6 ± 24.34 | 124.6 ± 27.48 | 122.13 ± 31.84 | 132.0 ± 24.94 | 126.4 ± 28.50 | 119.8 ± 30.93 | 136.6 ± 28.62 |
| *MVC* | | | | | | | | | |
| iEMG | 0.23 ± 0.036 | 0.22 ± 0.063 | 0.28 ± 0.033 | 0.23 ± 0.050 | 0.23 ± 0.062 | 0.30 ± 0.038 | 0.22 ± 0.045 | 0.22± 0.065 | 0.26 ± 0.025 |
| P-P Torque | 706.6 ± 142.07 | 693.3 ± 134.83 | 670.6 ± 139.04 | 693.0 ± 140.19 | 692.6 ± 133.66 | 681.0 ± 150.37 | 708.5 ± 144.02 | 696.7 ± 151.12 | 693.8 ± 145.38 |
| *Tetanus* | | | | | | | | | |
| 20 Hz Torque | 177.10 ± 35.5 | 214.7 ± 48.6 | 262.1 ± 70.15 | 196.2 ± 29.76 | 219.5 ± 38.75 | 237.2 ± 70.29 | 211.0 ± 29.76 | 227.5 ± 52.0 | 242.4 ± 65.4 |
| 100 Hz Torque | 260.3 ± 26.9 | 253.9 ± 71.27 | 313.0 ± 103.85 | 272.1 ± 14.16 | 284.3 ± 50.62 | 255.6 ± 68.36 | 289.5 ± 17.88 | 278.5 ± 84.70 | 295.4 ± 84.66 |

Figure 4.1 Plyometric Training (Pre-drop Phase)

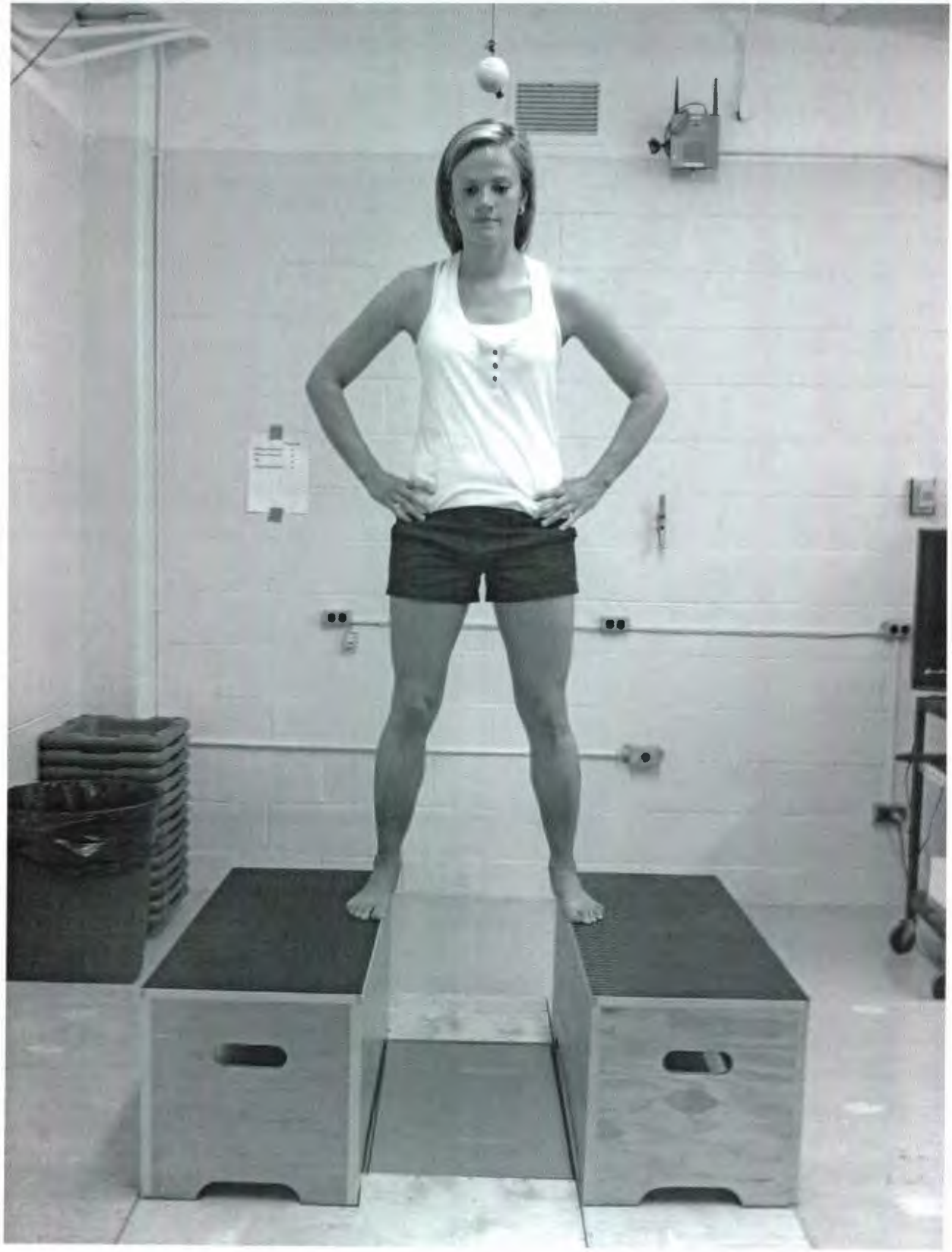


Figure 4.2 Plyometric Training (Pre-jump Phase)

Figure 4.3 Evoked Twitch and Maximal Voluntary Contraction (MVC)

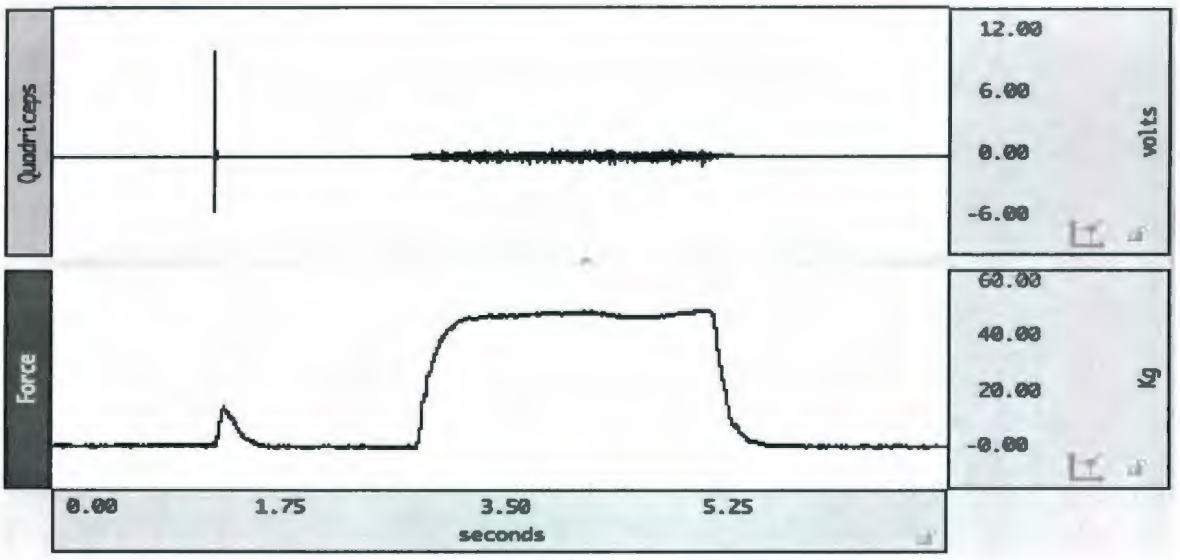



Figure 4.4 Compound Muscle Action Potential – M Wave

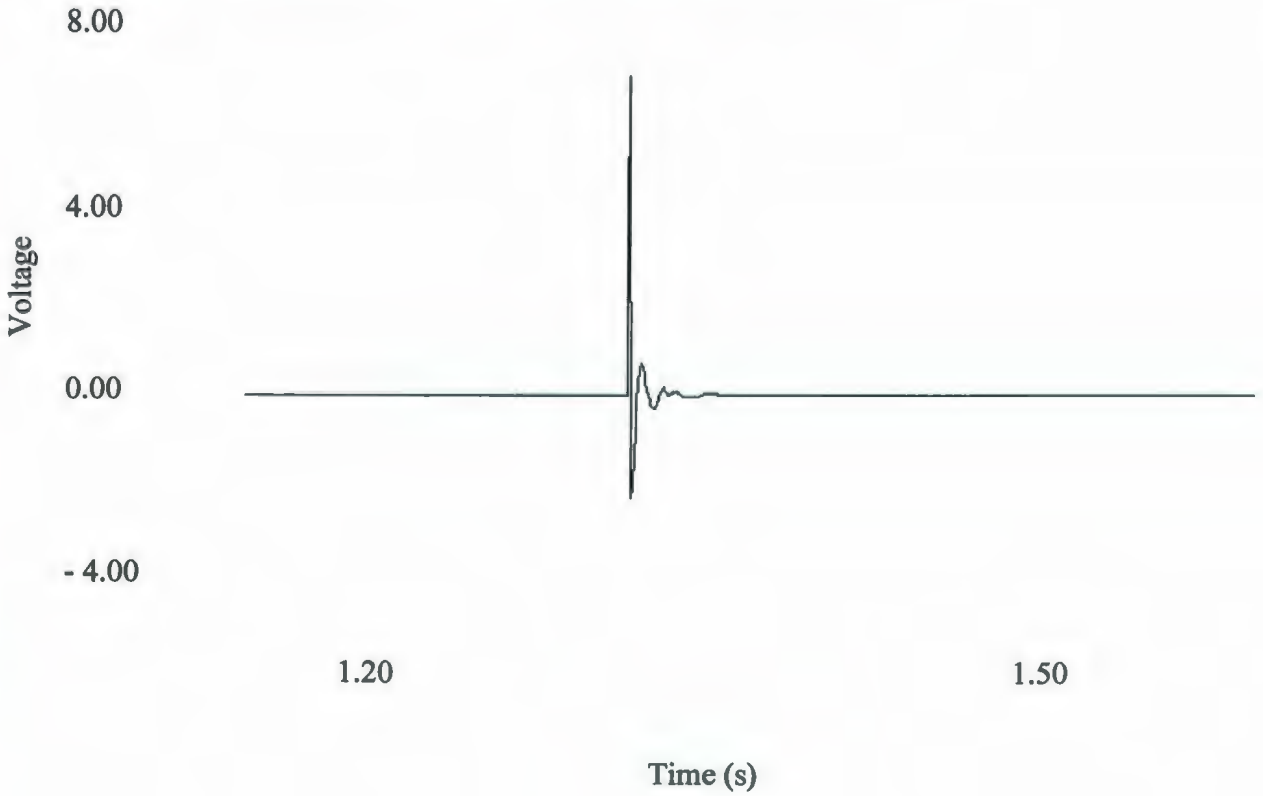


Figure 4.5 Low Frequency Tetanus – 20 Hz

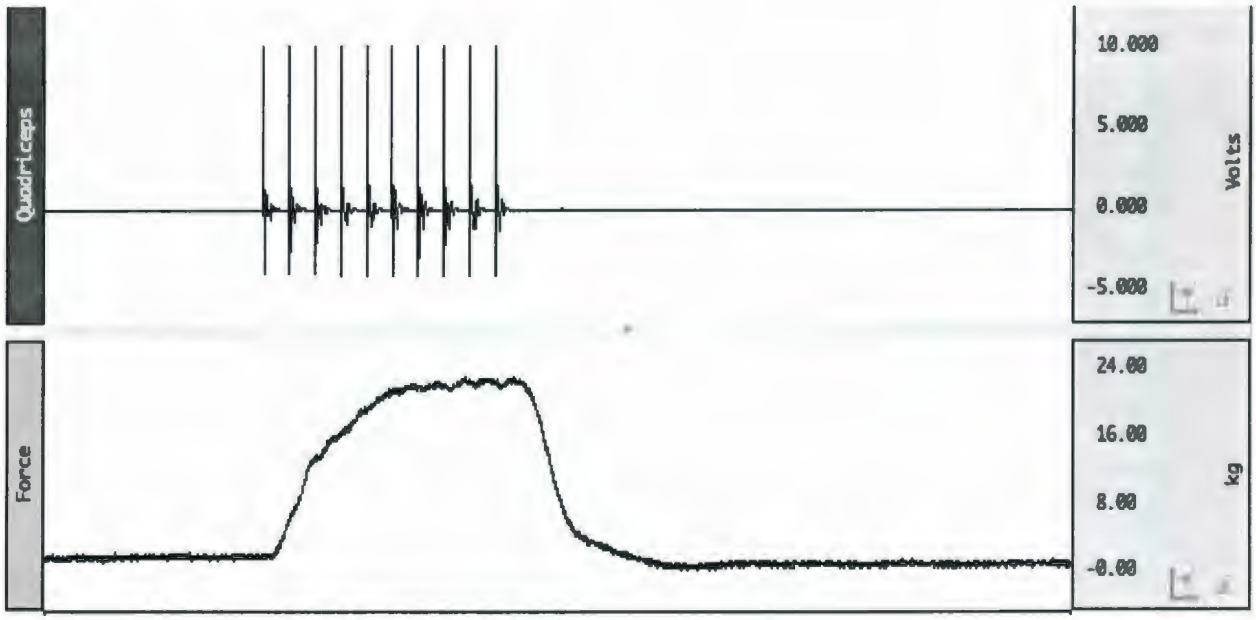



Figure 4.6 High Frequency Tetanus – 100 Hz

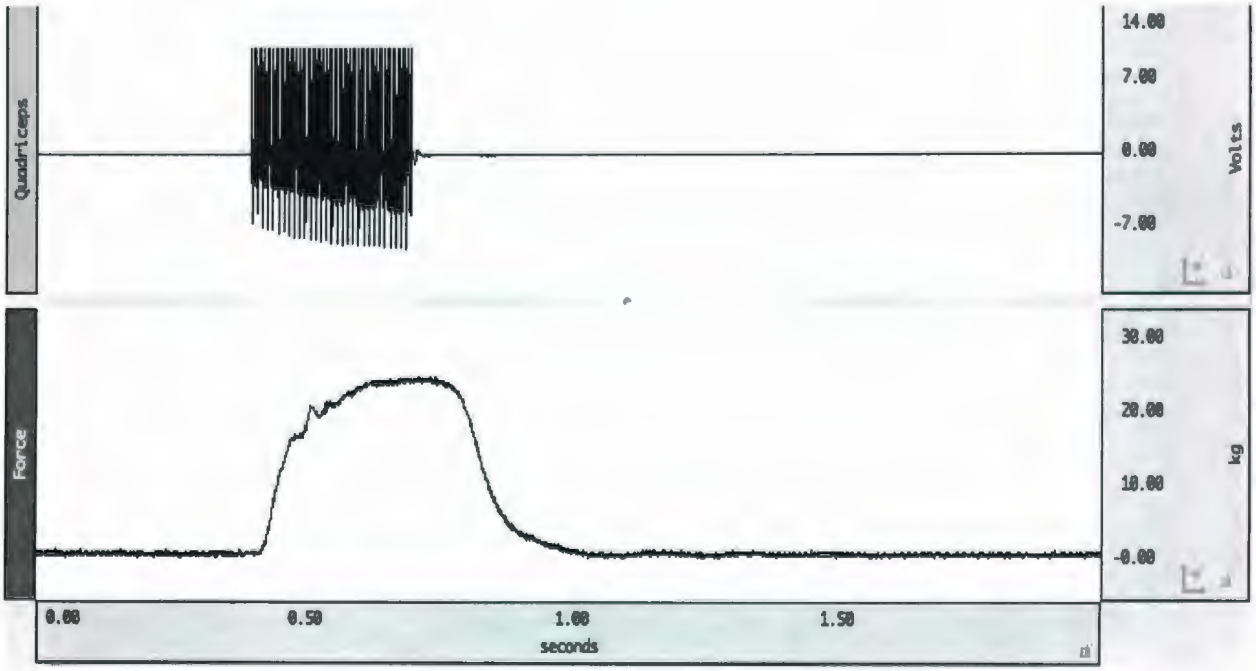


Figure 4.7

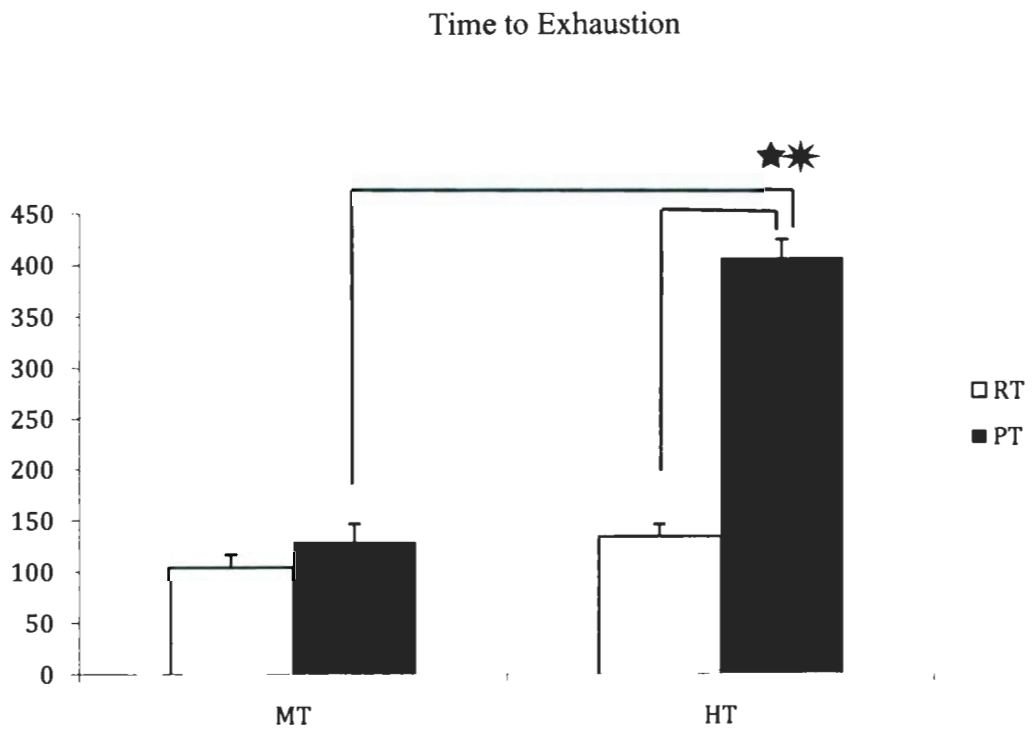



Figure 4.8

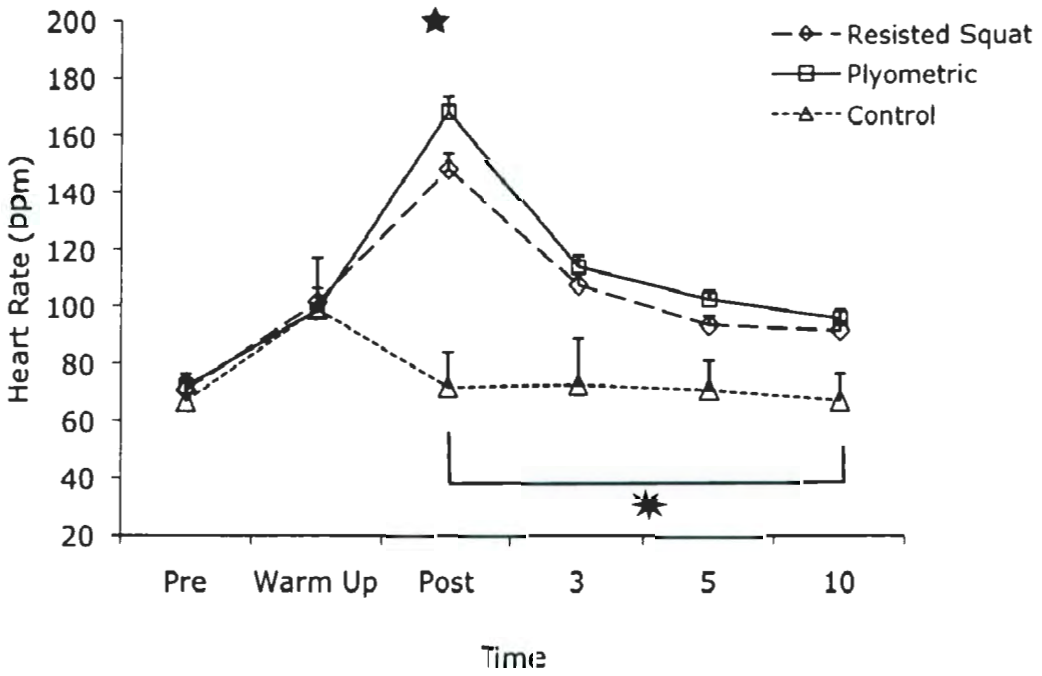


Figure 4. 9

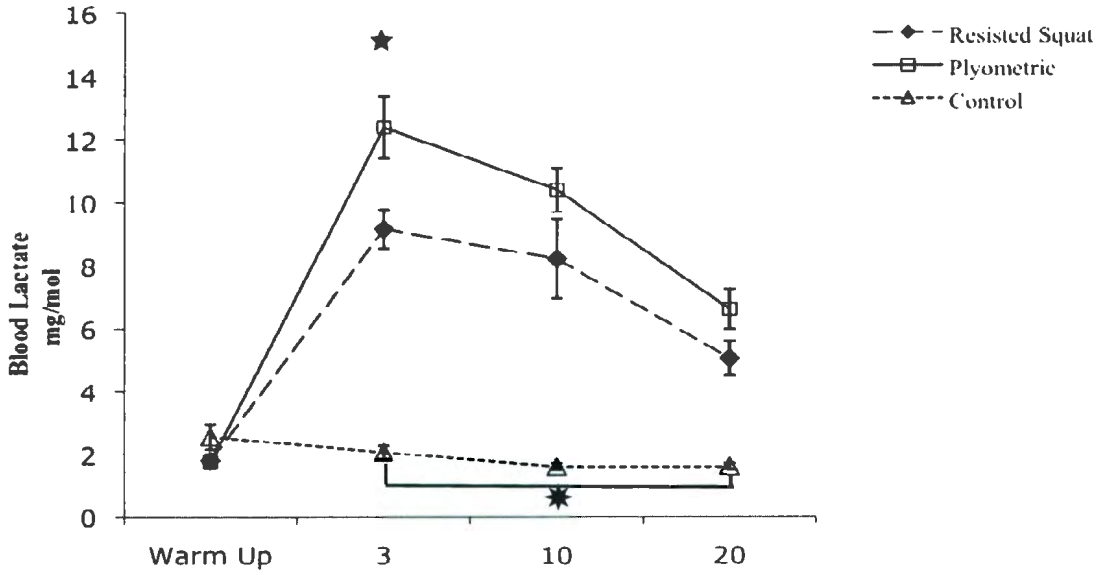



Figure 4. 10

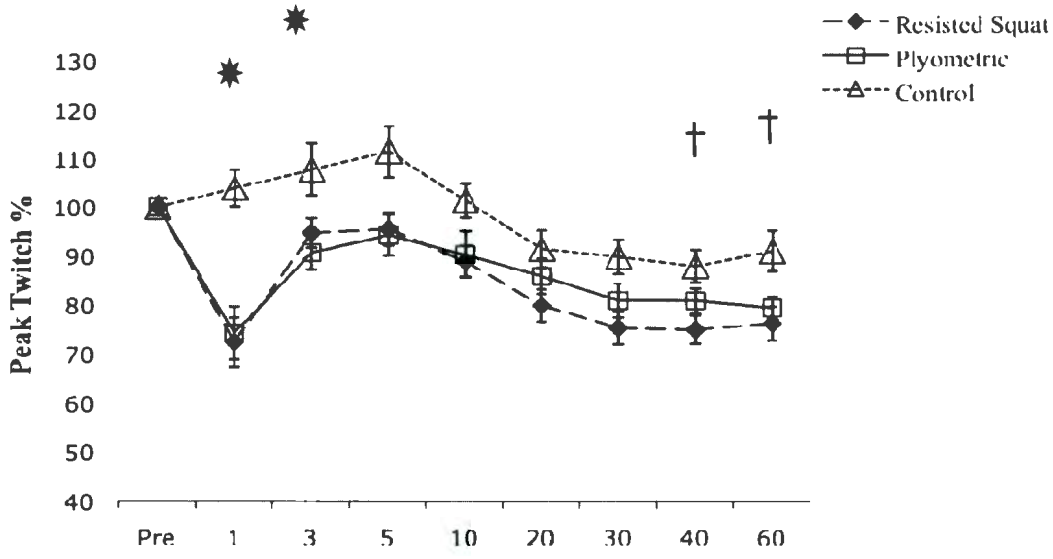


Figure 4. 11

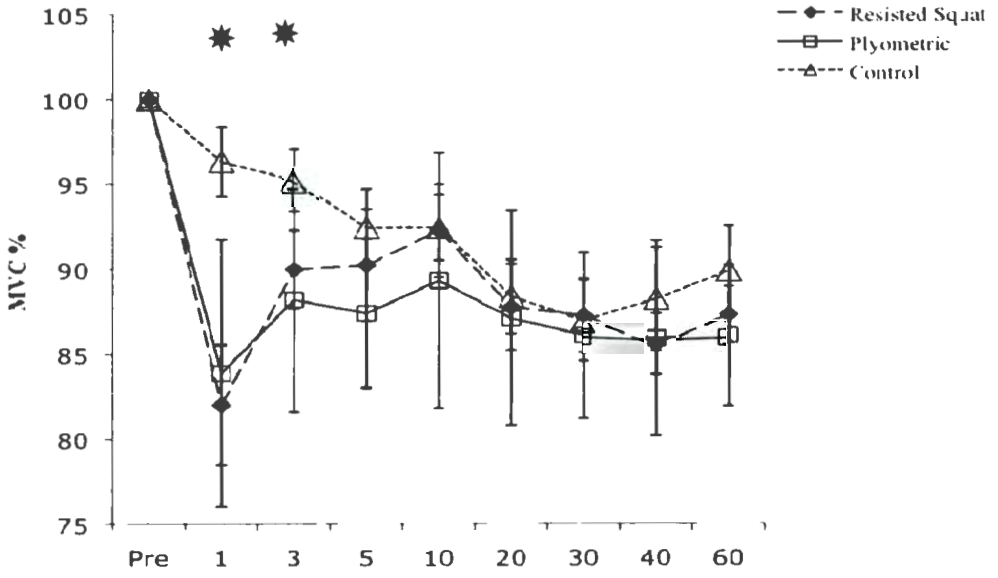
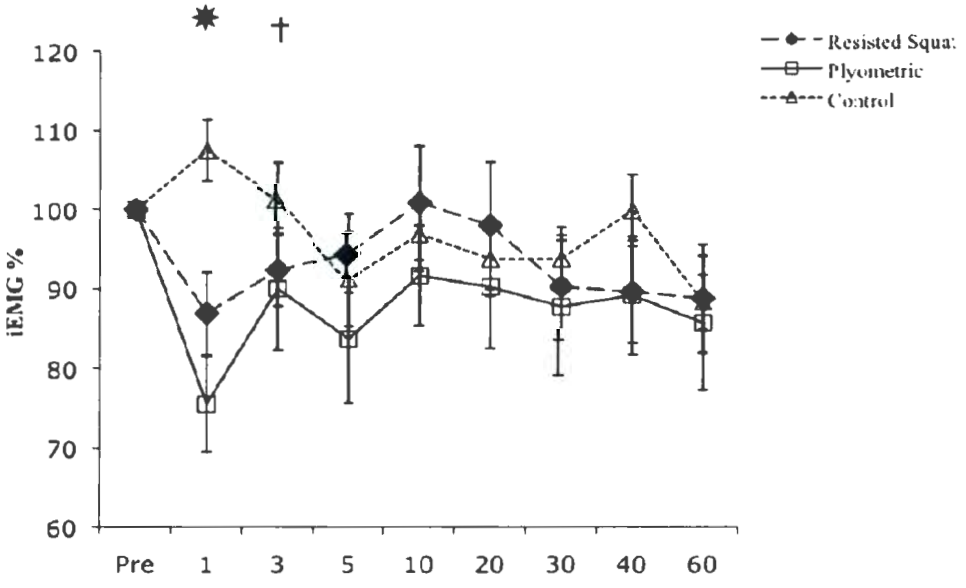



Figure 4. 12



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## Appendix 1:

### Experimental Subject Information Sheet

| Session: | | Name: | | Date: | |
|---|---|---|---|---|---|
| RHR | | RBP | | | |
| **Pretest** | | | | | |
| Heart Rate | | Blood Lactate | | MVC | |
| Twitch Level | | 100 Hz | | 30% MVC | |
| File Name: | Protocol | Torque | Stim Level | | Keep |
| 1 | | | | | |
| 2 | | | | | |
| 3 | | | | | |
| 4 | | | | | |
| 5 | | | | | |
| 6 | | | | | |
| 7 | | | | | |
| 8 | | | | | |
| 9 | | | | | |
| 10 | | | | | |
| 11 | | | | | |
| 12 | | | | | |
| 13 | | | | | |
| 14 | | | | | |
| 15 | | | | | |
| Posttest | | | | | |
| File Name: | Protocol | Time | Blood Lactate | Heart Rate | |
| | twitch/mvc | 1 | | | |
| | 20 Hz | 1 | | | |
| | 100 Hz | 1 | | | |
| | twitch/mvc | 3 | | | |
| | 20 Hz | 3 | | | |
| | 100 Hz | 3 | | | |
| | twitch/mvc | 5 | | | |
| | 20 Hz | 5 | | | |
| | 100 Hz | 5 | | | |
| | twitch/mvc | 10 | | | |
| | 20 Hz | 10 | | | |
| | 100 Hz | 10 | | | |
| | twitch/mvc | 20 | | | |
| | 20 Hz | 20 | | | |
| | 100 Hz | 20 | | | |
| | twitch/mvc | 30 | | | |
| | 20 Hz | 30 | | | |
| | 100 Hz | 30 | | | |
| | twitch/mvc | 40 | | | |
| | 20 Hz | 40 | | | |
| | 100 Hz | 40 | | | |
| | twitch/mvc | 60 | | | |
| | 20 Hz | 60 | | | |
| | 100 Hz | 60 | | | |