Neuromuscular Activation and the Load Sharing Concept

by

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A Thesis
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in Partial Fulfillment of the Requirements
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NEUROMUSCULAR ACTIVATION AND THE LOAD SHARING CONCEPT

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A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirements of the degree of

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III. NOMENCLATURE

f	twitch response of a motor unit (arbitrary units)
F_n	total force output of a motor unit (arbitrary units)
F_F	force measured by the dynamometer's force transducer (N)
F_T	total force output of a motor unit pool (arbitrary units)
G	gain of the nerve-muscle system
I	mass moment of inertia of a solid body about its rotational axis
I_K	mass moment of inertia of the shank, foot and resistance pad about the transverse axis of the knee joint ($km \cdot m^2$)
I_{SF}	mass moment of inertia of the shank and foot about the transverse axis of the knee joint ($kg \cdot m^2$)
I_R	mass moment of inertia of the resistance pad about the transverse axis of the knee joint ($km \cdot m^2$)
k_S	radius of gyration of the shank about the transverse axis of the knee joint as a function shank length.
k_F	radius of gyration of the foot about the transverse axis of the ankle joint as a function of foot length
L_D	distance between the line of action of F_F and the transverse axis of the knee joint (m)
L_{cm}	perpendicular distance between the center of gravity of the shank-foot complex and the transverse axis of the knee joint (m)
L_S	shank length measured between the lateral malleolus of the ankle and the lateral femoral condyl of the knee (m)
L_F	foot length measured as the distance from posterior surface of the calcaneus to the first distal phalanx (m)
ΣM	sum of the moments about the a specific rotational axis
ΣM_k	sum of the moments about the transverse axis of the knee joint (Nm)
m_{SF}	mass of the shank and foot (kg)
m_F	mass of the foot (kg)
m_R	mass of the resistance pad (kg)
m_S	mass of the shank (kg)
P	peak twitch force of a motor unit
R	rise time of a motor unit twitch
R_L	longest duration twitch rise time for the motor unit pool
R_n	rise time of the n^{th} motor unit
SR	stimulus rate of the motor unit (Hz)
SR_n	normalised stimulus rate to the motor unit
θ_K	angle of knee flexion (rad)
α	angular acceleration of a solid body
α_S	angular acceleration of the shank (rad/s^2).

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I. Abstract

Purpose: The complex interaction between the multiple neural, mechanical and intrinsic muscular factors responsible for muscle damage during voluntary movements has not been well elucidated. Specifically, the association between neural recruitment strategies used to activate muscles during voluntary exercise and the occurrence of muscle damage has not been examined. This purpose of this study was to explore the role of motor unit activation strategies in the initiation of exercise induced muscle damage. In addition, this study was undertaken to examine electromyographic signals (EMG) from the knee extensor muscles during concentric, eccentric and isometric contraction in order to examine possible differences in neural activation strategies between contraction types and contraction velocities.

Methods: Isovelocity knee extensor strength tests were performed before and 24 hours after low level electrically evoked eccentric exercise of the quadriceps muscle (n = 6). Surface EMG was measured from the knee extensor muscle during maximum voluntary isovelocity knee extensor strength tests (3 repetitions, ±50, 100, 150, 200, 250°/s) for 8 subjects.

Neuromuscular Model: Isometric force and muscle fiber loading resulting from different neural activation strategies was simulated using a model based on human motor unit twitch properties.

Results and Conclusions

Indirect evidence of contraction-induced injuries, as shown by a decrease in voluntary moment generation and an increase in pain intensity, were found 24 hours after the low level electrical stimulation . These data support the hypothesis that the occurrence of contraction-induced damage resulting from eccentric contractions is related to the neural strategy used to activate the involved muscle(s).

EMG/angle/angular relationship displayed regional deficits with the most substantial decrease occurring during eccentric contractions. These data support the hypothesis that not only does a neural regulatory mechanism exists which limits full activation of the knee extensor muscles during isovelocity exercises, but that this mechanism is velocity, contraction type and angle dependent.

The neuromuscular model demonstrated that given the same external load across a muscle substantially fiber loading could arise given physiological acceptable activation patterns.

Taken together, these findings would be consistent with an adaptive load sharing neuronal mechanism where different motor units may be recruited under similar external loading conditions to reduce fibre and related ultrastructure stress.

1.0 INTRODUCTION

The complex interaction between the multiple neural, mechanical and intrinsic muscular factors responsible for muscle damage during voluntary movements has not been well elucidated. Specifically, the association between neural recruitment strategies used to activate muscles during voluntary exercise and the occurrence of muscle damage has not been examined. This investigation was undertaken in order to explore the role of motor unit activation in the initiation of exercise induced muscle damage. In addition, this study examines the EMG/angle/angular velocity relationship of the knee extensor muscles in order to examine possible differences in neural activation between contraction types, contraction velocities and range of motion during maximal voluntary contractions.

For the sake of convenience in presentation, the review of literature is divided into three sections: 2.0 Review of Muscle Physiology, 3.0 Neuromuscular Modeling, and 4.0 Review of Isokinetic Dynamometry.

The review of muscle physiology section provides a brief synopsis of the relevant literature associated with exercise induced muscle damage. In addition, this section provides a review of the literature on the various neural, mechanical and intrinsic muscular factors responsible for moment generation in humans.

The neuromuscular modeling section provides a review of literature on the force response of human and animal motor units. Within this section, a neuromuscular model is developed which was employed in this study to explore the loading of motor units under various neural activation strategies.

Isokinetic dynamometers are a measurement device used to estimate the resultant moment about a human joint during voluntary contractions. This device is commonly used to quantify the impairment in voluntary moment generation resulting from exercise induced muscle damage. The review of dynamometry section provides a short review of the literature on the errors associated with dynamometry measures.

2.0 REVIEW OF MUSCLE PHYSIOLOGY

Skeletal Muscle

Skeletal muscle is comprised of contractile proteins and numerous non-contractile molecules, which are responsible for the function and structural integrity of a muscle. The contractile proteins, myosin (thick filament) and actin (thin filament) are large polymerized protein molecules which are longitudinally arranged and interdigitate to form a hexagonal lattice within the sarcomere. Creation of force by a muscle is accompanied by the sliding of these thick and thin filaments past one another (Huxely and Hanson 1954). The generally accepted theory to explain the sliding of the filaments is the cross-bridge theory of muscle contraction (Huxely 1957). This theory suggests that during a contraction crossbridges extending from the thick filament attach to the thin filament and undergo a structural transformation which exerts a tensile force in both the thick and thin filaments. Following the transition and binding of adenosine triphosphate (ATP), the crossbridges detach and are free to repeat the cycle (Lieber 1992).

The central region of the thick filaments are linked together by the M band. It has been suggested that the M bands are responsible for holding the thick filaments in a regular array and provide a strong anchoring point for the structural protein titin (Billeter and Hoppeler 1992). The Z band, composed of the non-contractile proteins a-actinin and desmin is proposed to provide structural support for one end of the thin filament and holds the filaments at regular spacing intervals (Billeter and Hoppeler 1992).

The longitudinally arranged proteins nebulin and titin are generally referred to as the "endosarcomeric cytoskeleton" (Enoka 1994, Waterman-Storer 1991). Titin has been suggested to run the length of the 1/2 sarcomere with connections to Z band, M band and myosin filament in A-band region (Horowits and Podolsky 1987). Titin

is proposed to be responsible for maintaining the central location of thick filaments in relaxed muscle and has been found to be a compliant elastic filament (Horowits and Podolsky 1987). Wang and Wright (1988) postulated that nebulin plays a role in thin filament packing geometry, and/or acts to help actin filaments interdigitate with myosin during stretching of the sarcomere. Although nebulin has been formally identified as a compliant filament, work by Maruyama and colleagues (1989) showed that nebulin lacked extensibility even in highly stretch sarcomeres. The exact role of the "endosarcomeric cytoskeleton" in muscle fiber force generation is still not well understood (Lieber et al.1996).

Sarcomeres added in series, Z band to Z band, construct a myofibril. A human myofibril may contain several thousand sarcomeres in series (Huijing 1985). Each muscle fiber contains myofibrils added in parallel, each approximately 1-2 um in diameter often extending the length of the fiber (Billeter et al. 1992,). The shortening of a myofibril is thus equal to the summed shortening of all the sarcomeres (Huijing 1992). Encircling each set of myofibrils that comprises a muscle fiber is the sarcolemma. The sarcolemma consists of a cell membrane, called the plasma membrane, and an outer coat made up of a thin layer of polysaccharide material containing numerous thin collagen fibrils. Muscle fibers can vary from 10 to 60 um in diameter and 1 to 400 mm in length and may contain thousands of myofibrils arranged in parallel (Enoka 1994).

Myofibrils are embedded in a complex "exosarcomeric cytoskeleton" framework consisting of intermediate filaments (Enoka 1994, Waterman-Storer 1991). The intermediate filaments, including desmin, vimentin, and synemin are localized to the periphery of the Z band and form a honeycomb like net within the plane of the Z band (Waterman-Storer 1991). These longitudinal and transverse filaments interconnect Z bands and link to the sarcolemma, mitochondria, and nucleus within the muscle fiber (Lieber et al. 1996). The exosarcomeric cytoskeleton is connected to the sarcolemma and extracellular matrix by various connection proteins including talin, vinculin, a-actinin and intergrin (Tidball 1991). The exosarcomeric

cytoskeleton and various connection proteins exact role in force generation is still unknown (Lieber et al. 1996). However, researchers have speculated that these structures act together to transmit force from the sarcomere to the extracellular matrix (Lieber et al. 1996, Street and Ramsey 1965, Street 1983). Lazarides (1980) also suggested that the intermediate filaments have a role in maintenance of the longitudinal Z band spacing.

A three-level network of collangenous connective tissue links the muscle fibers together. Surrounding each muscle fiber is the endomysium, followed by the perimysium which collects bundles of fibers into fascicles, and finally the epimysium which ensheathes the entire muscle.

The Motor Unit

In 1925, E.G.T. Liddel and Charles Sherrington introduced the term motor unit to describe the smallest functional element that can be controlled by the nervous system (c.f. Carew and Ghez 1985). A motor unit is defined as the cell body and dendrites of a α-motoneuron, the multiple branches of its axon, and the numerous muscle fibers that it innervates. The α-motoneuron's cell body is located in the anterior horn of the spinal grey matter. The average number of fibers innervated by a single motoneuron varies dramatically between muscles, ranging from several thousand fibers per motoneuron for large powerful muscles to less than twenty fibers for the very small muscles such as the extraocular muscle of the eye (Burke 1981, Enoka 1995).

When a α-motoneuron is excited and generates an action potential, normally all of the muscle fibers in the motor unit are activated. As the discharge rate of the α-motoneuron increases the force output from the muscle fibers innervated by the α-motoneuron generally increases. By this arrangement, the central nervous system can control three parameters of motor unit activation in changing the force output of a single muscle: 1) the recruitment of additional motor units, 2) the modulation of the firing rate (rate coding) of the motor units that are already activated, and 3) the pattern

of discharge. (Burke 1981, De Luca et al. 1982, Freund et al. 1975, Kukulka and Clamann 1981, Milner-Brown et al. 1973a).

There has been an extensive amount of literature devoted to investigating the various properties of a motor unit. These studies have demonstrated that the biochemical, histochemical, morphological and physiological properties of motor units vary across the motor unit pool of a muscle (Burke 1981, Enoka 1995, Henneman and Mendell 1981). In addition, these studies have found that the properties of the motor unit pool vary dramatically between different muscles. However, the muscle fibers innervated by each α-motoneuron have been shown to generally possess the same properties (Burke 1981, Nemeth et al. 1986). The fibers of a motor unit are scattered throughout a broad region of muscle and interdigitate with fibers belonging to many other motor units (Bodine et al. 1987, Burke 1981).

In various vertebrate skeletal muscles, it has been observed that fibers often do not extend the full length of the fascicle (Barret 1962, Ounjian et al. 1991, Roy and Edgerton 1992, Trotter 1990). Barret (1962) observed that in the human sartorius and biceps femoris muscles, numerous muscle fibers tapered within the muscle belly. Barret suggested that many of these fibers may be arranged end to end in-series with one or more fibers (Barret 1962). Ounjian and colleagues (1991) showed that for the cat tibilalis anterior muscle the muscle fibers for a single motor unit can be distributed along the entire length of the muscle and that at least one end terminates within the fascicle. These authors also demonstrated that the fibers terminating mid-fascicularly tapered along their length often to a fine point with no apparent extension distally into a connective tissue interface. Based on the observations of tapered fibers and the various structural proteins connecting the sarcomere to the endomysium, several investigators (Ounjian et al. 1991, Street 1983, Trotter 1990, Trotter and Purslow 1992) have postulated the force transmission in skeletal muscles fibers occurs not only at the muscle-tendon junction but also along shear forces along the surface of the fiber that is in contact with the endomysial connective tissue matrix. However,

further work is required to clarify the role of the various extracellular proteins in force transmission.

Motor units are generally typed according to the various biochemical and/or histochemical measurements. The most common typing scheme for motor units, as proposed by Burke (1981), is to subdivide the motor unit pool into population that are slow-contracting and fatigue resistant (type S), fast contacting and fatigue resistance (type FR) and fast contraction and fast to fatigue (FF). In general, type S motor units are comprised of type I muscle fibers, type FR motor units are comprised of type IIa muscle fibers, and type FF motor units are comprised of type IIb muscle fibers (Burke 1981, Enoka 1995). Although motor units and fibers are generally classified into specific groups it should be noted that when any single motor unit or muscle fiber is measured, the value typically extend along a continuum (Burke 1981).

Motor Unit Recruitment

It has been well documented that the functions of motoneurons are highly correlated with their size. Specifically, there is a relationship between the size of a motoneuron and 1) its susceptibility to discharge by excitatory inputs, 2) the suppression of its firing by inhibitory inputs, and 3) the properties of the muscle fibers it supplies (Henneman and Mendell 1981). This organization promoted Henneman (1957) to conclude that there was a "size principle" underlying these correlations. According to the "size principle" put forth by Henneman, motor units are recruited in the order of increasing motoneuron size. However, the recruitment of motor does not solely depend on motoneuron size; it is also influenced by other motoneuron characteristics and by the organization of the synaptic input on the dendrites and soma (Burke 1981).

Most studies of motor unit behavior in humans have been performed during ramped isometric contractions. (De Luca et al. 1982, Masakado et al. 1995, Milner-Brown et al. 1973a, Monster and Chan 1977, Riek and Bawa 1992). These studies have generally found that motor units are activated in an orderly sequence, from

motor units that exert the smallest forces to those that exert the largest forces. In addition, many of these studies have demonstrated that higher threshold motoneurons generally have a lower firing rate than low threshold motoneurons (De Luca et al. 1982, Masakado et al 1995). However, several studies in humans have found that during dynamic movement, the nervous system appears to depart from the previously reported recruitment order observed during isometric contractions (Howell et al. 1995, Moritani et al. 1988, Nardone et al. 1989).

It has been demonstrated that the input-output relation of the motoneuron pool can by altered by varying the sources of synaptic input and by neuromodulatory agents that can change membrane conductance (as reviewed by Enoka 1995). It has been proposed by several authors (Enoka 1995, Heckman and Binder 1993, Kernell and Hultborn 1990) that variable inputs to the motor unit pool allow the nervous system to vary the rate coding and recruitment strategies used during any specific task. In a review of literature on motor unit recruitment Enoka (1996) concluded that compared to concentric and isometric contractions the rate coding and recruitment strategy used during eccentric contractions was unique. Based on single unit recordings, Nardone and colleagues (1989) proposed that high-threshold motor units in the gastrocnemius muscle were selectively activated when the plantar flexor muscles performed eccentric contractions compared with concentric contractions at moderate to high velocities. Several investigators have also proposed that the rate coding and recruitment strategies used during a specific task can be altered with training (Bernardi, et al. 1996, see reviews Enoka 1997 and Sale 1988).

Force/Length — Force/Velocity Relationship

As the length of a muscle fiber changes, and the thick and thin filaments slide past one another, the number of thin filament binding sites available for crossbridges is changed (Huxely 1957). Since the active force generation of a muscle fiber is considered to be a function of the number of attached crossbridges, the maximum active force generation of a muscle fiber changes with length (Huxely 1957, Partridge

and Benton 1981). The force/length relationship of whole muscle has been well demonstrated in numerous animal and human muscles (Partridge and Benton 1981).

In 1938, A.V. Hill demonstrated that the inverse relationship between muscle force and velocity of shortening of a maximally activated frog's sarorius muscle can be represented by a single rectangular hyperbola (Hill 1938). Hill's hyerbolic equation states that as the force across the muscle increases, the shortening velocity decreased until a local maximum force is attained at zero velocity. However, work on single isolated frog fiber has demonstrated that the Hill's hyperbolic equation is appropriate to about 78% of maximal isometric force, after which a deviation appears upon which a second hyperbolic path is fit (Edman et al. 1978, Edman 1988).

The maximum shortening velocity of a muscle fiber has been found to correlate well with the maximum rate of hydrolysis of ATP within the contractile system (Partridge and Benton 1981). On average, fast-contracting muscle fibers shorten approximately two to three times faster than slow-contracting fibers at their maximum velocity (Close 1972, Edman 1988). Since motor units are fiber type specific, types FF and FR motor units are generally capable of generating larger forces at high velocities compared to type S motor units (Close 1972).

Investigations of frog, mouse and cat whole muscle has revealed that for lengthening contraction force generation increases with increasing velocity of lengthening up to approximately 1.5 times maximal isometric force followed by a plateau region where force remains relatively constant with any additional increases in velocity of lengthening (Flintney and Hirst 1978, Harry et al. 1990, Katz 1939). In single isolated frog fibers, Edman and colleagues (1978) and Haugen (1991) found that the maximal force generated during lengthening contractions was approximately 1.7-1.9 greater than the force generated during a tetanized isometric contraction. Several studies have indicated that the increase in force production during lengthening contractions likely results from the elastic components resident in the muscle sarcomere (Huxley and Simmons 1971, Morgan 1977, Webber 1996)

Moment/Angle/Angular Velocity Relationship

Human motions involve the rotation or control of rotations of body segments. In the musculoskeletal system, the force generated by a muscle is transmitted to bone via tendons. As a result of this rotary system and the elastic nature of the tendon, during constant angular velocity movements the linear velocity of the involved muscle is rarely constant (Zajac 1989). The moment arm of the tendon/muscle system varies substantially throughout the range of motion of the joint (An et al. 1981, Grood et al. 1984). The moment generated about a joint resulting from a single muscle can be calculated as the vector cross product of the positional vector locating the tendon relative the joint and the force in the tendon (Lieber 1992). The principle components that dictate the moment/angle/angular velocity relationship are the moment arm/angle, force/velocity and force/length relationships of a single muscle and the tendon properties attaching the muscle to the bone. Unfortunately, the detailed connections between these various relationships have not been thoroughly investigated (Lieber 1992).

There are numerous muscles acting about the joint each having their own moment arm/angle, force/velocity and force/length relationships. In addition, there are various passive structures which also produce moments about the joint. Resultant joint moment (RJM) represents the sum total of all moments acting about the instantaneous axis of rotation of a joint resulting from muscular, tendinous, ligamentous, cartilaginous, capsular, and bone-on-bone forces (Hay 1992). In general, the passive structures tend to generate larger moment near the end ranges of motion of the joint (Partridge and Benton 1981). Due to the low coefficient of friction between articulation surfaces in none pathological joints, i.e. 0.001-0.005 (Andersson and Schultz 1979) the contribution of bone-on-bone forces to the change in RJM is minimal.

STATUS OF NEURAL ACTIVATION

Studies by various investigators have demonstrated that the moments generated during maximal voluntary concentric decrease with increasing angular velocity (Bobbert and Harlaar 1992, Caldwell et al. 1993, Griffin 1987, Hortobagyi and Katch 1990, Perrine and Edgerton 1978, Westing 1991, Westing et al. 1988). It has also been demonstrated that the moments generated during maximal voluntary isometric contractions exceed moments generated during maximal voluntary concentric contractions (Caldwell et al. 1993, Griffin 1987, Hortobagyi and Katch 1990, Perrine and Edgerton 1978, Westing et al. 1988).

Several authors have suggested that humans are unable to fully activate all of their motor units during maximal voluntary isometric and concentric contractions (Enoka 1995, Enoka 1997, Fuglevand et al. 1993, Perrine and Edgerton 1978). Perrine and Edgerton (1978) have postulated that a neural regulatory mechanism exist which may limit the maximum voluntary moments produced during isometric and low velocity concentric to 50% of the levels predicted by in vitro studies. In contrast, Sale (1988) in a review article stated that many healthy, well-motivated individuals are able to fully activate all motor units during voluntary isometric contractions.

It has been suggested that the twitch interpolation technique can be used to determine the status of maximal activation (Merton 1954). In this procedure, an electric stimulus is delivered to the nerve of the muscle(s) engaged in a maximum voluntary contraction. If the electric pulse elicits additional force from the muscle then the normal interpretation is that not all motor units were fully activated (achieved tetanus) by the voluntary contraction. Allen and colleagues (1995) used this technique to study subject's abilities to maximally activate their elbow flexor muscles during isometric contractions. Based on their results these authors concluded that subjects were able to activate their muscles between 90.3-99.8% of maximum. In a recent study, Suter and colleagues (1996) used twitch interpolation to study the ability of normal healthy subjects to activate their quadriceps during isometric knee extension. These authors found that an additional average moment of 4.3% was

generated when a twitch was superimposed on a maximal contraction. Based on these observations they concluded that healthy subjects are able to nearly fully activate the quadriceps during an isometric contraction. However, in many subjects the evoked interpolated twitch torque was found to be zero for contractions levels as low as 70% of the subjects maximum moment which suggests that there may be errors in the twitch interpolation technique for assessing the activation level of the muscle.

An indirect way of determining the status of motor unit activation is to compare firing rates of motor units during maximal voluntary contractions to those expected to be needed to produce maximum tetanic contractions of the motor units (Sale 1988). Numerous studies have measured the discharges rate of human motor units during maximum or near maximum voluntary isometric contractions and found that the maximum firing rate of the measured motor units averaged around 20-35 Hz (Bigland-Ritchie et al. 1983, De Luca 1982, Freund et al. 1975, Kukulka and Clamann 1981), although there are rare exceptions in which higher firing rates have been identified (Edstrom and Grimby 1986). However, several studies have demonstrated that muscles require artificial stimulus rates between 50-120 Hz to achieve maximum force during isometric contractions (Davies et al. 1985, Macefield et al. 1996, Binder-Macleod et al 1995, Thomas et al. 1991a). This finding suggests that individuals are unable to fully activate their muscles during isometric contractions.

Another way to explore the activation of muscles is to examine the surface EMG from a muscle during maximal voluntary contractions at different angular velocities (Westing et al. 1991). Surface EMG cannot be used directly to measure the activation level of a voluntary contraction (Basmajian and Deluca 1985, Winter 1990). However, if individuals are able to fully activate their muscles during concentric contractions it would be expected that the level of EMG would not change with increases in angular velocity, over different angles or during different contraction types (Gerdle and Langstrom 1987).

Barnes (1980) measured the surface EMG from the elbow flexor muscles of 6 male subjects during maximal voluntary concentric contractions at angular speeds of 60, 120, 180, 240 and 300°/s. Barnes found an inverse linear relationship between mean integrated EMG and angular velocity ($r = -0.99$) with the mean integrated EMG decreasing 33.3 percent from 60 to 300°/s. Nelson and associates (1973) measured the surface EMG from the tibialis anterior and soleus muscle during maximal voluntary concentric contractions. These authors found that as the velocity increased from 24°/s to 216°/s there was a non-significant decrease in the mean integrated EMG. Gerdle and Langstrom (1987) found a small (~ 4%) non-significant decrease in the mean integrated EMG of the medial triceps surae muscle as concentric contraction velocity increased from 30°/s to 180°/s. In contrast to these studies, several investigators have demonstrated that the surface EMG from the knee extensors during maximal concentric contractions increases with increasing angular velocities (Komi et al. 1987, Seger and Thorstensson 1994, Westing et al. 1991). These studies will be discussed in detail in the following section.

Most studies of human strength have generally found that maximum eccentric moments do not exceed maximum isometric moments and do not increase with increasing velocities (Dudley et al. 1990, Mayer et al. 1994, Webber 1996, Westing 1988). For example, Westing and colleagues (1988) found that peak eccentric knee extensor moments remained constant over the range of angular velocities of 30 to 270°/s. In addition, these authors found that the peak eccentric moments were not significantly greater than the maximum isometric moment. This relationship would not be expected based on the in vivo force/velocity relationship of muscle.

There is generally agreement in the literature that individuals are unable to maximally activate their muscles during an eccentric contraction (Enoka 1996, Gibala et al. 1995, Komi et al. 1987, Seger and Thorstensson 1994, Tesch et al. 1990, Webber 1996, Westing et al. 1991, Westing et al. 1988). It has been suggested that this tension-limiting mechanism may protect the musculoskeletal system from damage that could result if the muscles were allowed to become fully activated during

an eccentric contraction (Stauber 1989, Westing et al. 1988, Westing et al. 1991). There is currently no direct physiological evidence of a neural regulatory mechanism in humans. However, it has been postulated that a neural regulatory mechanism may result from complex interactions among several afferent and efferent signals, possibly including feedback from free nerve endings in muscles, Golgi tendon organs, joint receptors, cutaneous receptors and pain receptors (Westing et al. 1991).

Some methods have been used to explore the amount of neural regulation that may exist during eccentric contractions (Webber 1996, Westing et al. 1990). Westing and colleagues (1990) found that angle-specific eccentric moments increased by 21-24% above maximal voluntary contraction levels when electrical stimulation was superimposed during maximal eccentric contractions. Webber (1996) measured the moment generated by the knee extensors when eccentric contractions (100°/s, 12° displacement) were forced upon isometric contractions of varying strength. It was found that the maximum imposed eccentric moments were double (204%) the angle matched eccentric moments observed during voluntary maximal contractions at 100°/s.

Studies by various investigators have generally found that the EMG recorded from a muscle during maximal eccentric contractions is lower than the EMG recorded from the muscle during velocity matched maximum concentric contractions (Komi et al. 1987, Segar and Thorstensson 1994, Tesch et al. 1990, Westing et al. 1991)

Westing and colleagues (1991) measured the surface EMG from the vastus lateralis, vastus medialis and rectus femoris muscles of 14 male subjects during maximal concentric and eccentric contractions of the knee extensors (45, 90, 180, and 360°/s, 10-90° of knee flexion). For each subject the mean values of the full-wave rectified EMG signal between 30-70° of knee flexion were calculated and divided by the subject's average concentric EMG value at 45°/s. They found that the normalized EMG increased significantly as the angular velocity increased from 45 to 360°/s. From 45 to 360°/s the normalized EMG was found to increase ~40% in vastus lateralis, ~40% in vastus medialis and ~20% in the rectus femoris. For eccentric

contractions, the level of EMG was found to remain essentially constant with changes in angular velocity for vastus lateralis, vastus medialis and rectus femoris. With the exception of eccentric EMG from the vastus lateralis at 360°/s, the eccentric EMG from each muscle was found to be lower (range 1-30%) than the concentric EMG of each muscle measured at 45°/s. In addition, they reported that eccentric EMG was always significantly less than the velocity matched concentric EMG.

Segar and Thorstensson (1994) measured the surface EMG from the astus lateralis and vastus medialis muscles of 40 subjects during maximal concentric and eccentric contractions of the knee extensors (45, 90, 135 and 180°/s, 10-90° of knee flexion). The mean values of the full-wave rectified EMG signal between 30-70° of knee flexion were calculated for each subject and reported in uV (not normalized). These authors found that average EMG increased 8-25% as angular velocity increased from 45 to 180°/s during concentric contractions. The EMG measured during eccentric was observed to remain constant with increasing angular velocities and was 5-11% lower than the concentric EMG measured at 45°/s. No statistics were performed on this data.

Tesch et al. (1990) found that average integrated EMG from vastus lateralis and rectus femoris muscles were significantly less during eccentric compared to concentric maximal contractions at 180°/s.

Exercise Associated Damage to Muscle

Skeletal muscles are repeatedly damaged and repaired throughout life. During the past several decades, an extensive volume of literature has accumulated that also suggests that skeletal muscles can be damaged by mechanical stresses intrinsic to a muscle during various forms of voluntary exercise (Armstrong et al.1983, Friden et al. 1988, Gibala et al 1995, Mair et al 1992, Newham et al. 1983, Stauber et al. 1990). Although there is an abundance of literature on this subject, the mechanism(s) underlying the initial damage remains the subject of intense debate.

Observations that are often presented as evidence that a particular voluntary exercise has caused contraction-induced damage include impairment in the voluntary and/or electrical evoked force production of the exercised muscle(s) for periods greater than 24 hours after the exercise session; soreness in the exercised muscle which peaks in intensity 24-72 hours after the exercise session; and increased levels of intramuscular proteins in the blood 24-48 hours after the initial damage (Armstrong et al. 1990, Gibala et al 1995, MacIntyre et al. 1995, Newham et al. 1988, Stauber et al. 1988). However direct evidence that a particular protocol of contractions has produced damage to a muscle requires either histological, light microscopic or electron microscopic sections that show damage to the muscle (Faulkner et al. 1993). Direct evidence of contraction-induced damage in skeletal muscles after various forms of voluntary exercise have been identified in both humans (Friden et al. 1983b, Friden et al. 1988, Gibala et al 1995, Jones et al. 1986, Newham et al 1983, Stauber et al. 1989) and animals (Armstrong et al.1983, McNeil and Khakee 1992, Ogilvie et al. 1988).

The most commonly reported voluntary contraction-induced damage are small, focal areas of damage within the fibers and/or connective tissue of the muscle. (Friden et al. 1983b, Friden et al. 1988, Newham et al. 1983). The percentage of fibers displaying one or more focal areas of damage after exercise has been shown to vary dramatically from less than 1% to greater than 80% of the exercised muscle (Friden et al. 1983b, Gibala et al. 1995, Newham et al. 1983). Reports of voluntary contraction-induced damage in humans which are severe enough to lead to muscle fiber necrosis are relatively scarce. In the few studies that have reported necrotic fibers as the result of voluntary contraction-induced damage, the number of necrotic fibers observed varies widely between subjects but usually involves less than 2% of the fibers (Jones et al. 1986, Stauber et al. 1990).

The majority of focal damage reported after a voluntary contraction-induced damage is in the myofibrils of the injured muscle (Armstrong et al. 1983, Friden et al. 1983b, Friden et al 1988, Gibala et al. 1995, Jones et al. 1986). These authors report

that the damage in the myofibrils originates from the Z-bands, which shows broading, smearing, or even total disruption. Z-bands in sarcomeres adjacent to the disrupted myofibrils also regularly appear out of register and normally follow a zigzag course. The amount of damage in a given area varies dramatically from only a single disrupted sarcomere to several hundred adjacent and continuous disrupted sarcomeres. Other types of focal damage that have been reported after various forms of voluntary exercise include extracellular matrix disruption (Stauber et al. 1990), sarcolemma disruption (Armstrong et al. 1983, McNeil and Kahkee 1992), swelling and disruption of the sarcotubular system (Armstrong et al. 1983), disruption of the cytoskeleton (Friden et al. 1983b, Armstrong et al. 1983), severe swelling and/or disruption of the mitochondria (Friden et al. 1983b, Friden et al 1988) and disruption of the central nuclei (Gibala et al. 1995).

Strenuous physical exercise of the limb muscles commonly result in contraction-induced damage, especially when the exercise is intense, fatiguing or involves a novel bout of eccentric contractions (Brown et al 1996, Friden et al. 1983a., Friden et al. 1983b., Golden and Dudley 1992, Jones et al. 1986, MacIntyre et al. 1996, Newham et al. 1987, Stauber et al. 1990). Indirect and direct evidence of contraction-induced damage in humans has been reported after strenuous voluntary exercise bouts of sprint running (Friden et al. 1988), eccentrically biased bicycling (Friden et al. 1983a, Friden et al. 1983b), downhill running (Byrnes et al. 1985, Schwane and Williams1987), downhill walking (Jones et al. 1986), marathon running (Hikida et al. 1983), stair stepping (Newham et al. 1983), and flexion and/or extensions exercises of the elbow (Jones et al. 1986, Gibala et al 1995, Newham et al. 1987, Stauber et al. 1990), knee (Brown et al 1996, Golden and Dudley 1992, MacIntyre et al. 1996) and ankle (Friden et al. 1986, Jones et al 1986).

During voluntary exercise the motions produced by muscle contractions can be broken down into concentric, eccentric, and isometric phases. There is an extensive amount of literature devoted to investigating which type of contraction (eccentric, concentric or isometric) is responsible for contraction-induced damage. Several

authors have provided both direct and indirect evidence that contraction-induced damage can occur during any type of voluntary contraction (Clarkson et al. 1986, Gibala et al. 1995, Newham et al. 1983). In spite of the potential for damage during any type of contraction, the probability of damage is generally considered to be the greatest during eccentric contractions (see reviews Faulkner et al. 1993, MacIntyre et al. 1995, Newham 1988). For example, it has been found that the probability of sustaining a contraction-induced damage is increased when the exercise involves a substantial amount of eccentric contractions (e.g. downhill walking or running). For additional information see reviews Armstrong et al. 1990 and Faulkner et al. 1993. In addition, several studies have found that when voluntary eccentric and concentric contractions are performed at similar moment levels the voluntary eccentric contractions have resulted in more severe contraction-induced damage (Gibala et al. 1995, Newham et al. 1983).

Newham et al (1983) investigated differences between voluntary concentric and eccentric contractions in 4 healthy subjects using stepping exercises. During the stepping exercise protocol the quadriceps muscle of the leg which stepped up contracted mostly concentrically, while the contralateral muscle which controlled the down step contracted mostly eccentrically. Biopsies were taken immediately after 20 minutes of the stepping exercise and examined using light microscopy. Biopsies taken from the concentrically exercised leg showed no abnormalities. However, an average of 8% of the fibers in the biopsies taken from the eccentrically exercised leg had focal areas of damage which affect more than two adjacent sarcomeres and more than two adjacent myofibrils.

Recently, Gibala and coworkers (1995) examined changes in muscle ultrastructure in 8 untrained male subjects after eccentric and concentric exercise of the forearm flexors. Subjects performed one bout (8 sets of 8 reps, 150° range of motion) of concentric bicep curls with one arm, and eccentric bicep curls with the contralateral using a free weight equal to 80% of pre-exercise concentric maximum. Bioposies taken immediately after the exercise session revealed that an average of

82% of the fibers in the eccentrically exercised arm of subjects had at least one myofibril disruption. Only an average of 33% of the fibers in the concentrically trained arm had at least one myofibril disruption.

The multiple neural, mechanical and intrinsic factors that influence moment generation during voluntary exercise make identification of the mechanism(s) underlying the initial contraction-induced damage extremely difficult. In order to gain better insight into some of the mechanisms responsible for voluntary contraction-induced damage numerous investigators have used various rat, mice and rabbit in situ (Fritz and Stauber 1988, Lieber et al. 1996, McCully and Faulkner 1985, Warren et al. 1994) and in vitro (Brooks et al. 1995, Jones et al. 1997, Warren et al.1993a) muscle preparations. These animal muscle preparations allow for greater control of variables such as the muscle load, muscle velocity and the recruitment.

McCully and Faulkner (1985) investigated the damaging effects of repetitive isometric and velocity matched concentric and eccentric contractions using an in situ mouse EDL muscle preparation. Three days after ~500 maximal electrically evoked isometric or concentric contractions there was no evidence of muscle damage. There was, however, extensive histological damage in muscles three days after ~500 maximal electrically evoked eccentric contractions.

The specific event that serves to initiate a contraction-induced damage is not known. It is generally recognized in the literature that contraction-induced damage is closely associated with eccentric contractions (Armstrong 1984, Ebbeling and Clarkson 1989, Newham et al. 1983, Stauber et al. 1990, Warren et al 1993b).

This has lead some authors to hypothesize that one or more mechanical aspects of the eccentric contraction that distinguishes it from isometric or concentric contractions may be responsible for initiation of the damage (Armstrong 1990, Golden and Dudley 1992, Newham et al.1983, Warren et al 1993a and 1993b).

During any given contraction, a structural component within a muscle will fail if the tensile stress borne by the component exceeds the component's tensile strength. Alternatively, if a muscle is subjected to repeated tensile stresses, a structural

component within the muscle may undergo materials fatigue (Warren et al.1993a). It has been well demonstrated that eccentric force can reach 1.5 to 1.9 times the maximum isometric force when stretch is applied in vitro (Flintney and Hirst 1978, Harry et al. 1990, Katz 1939, Edman et al.1978, Haugen 1991). This has lead many investigators to propose that contraction-induced damage is directly related to the forces produced during eccentric contractions (Katz 1939, McCully and Faulkner 1986, Newham et al. 1983, Warren et al. 1993). Several investigators have also proposed that multiple factors, including lengthening velocity, total length change, contraction number, and initial muscle length prior to the start of the contraction may also interact to result in contraction-induced damage during eccentric contractions (Brooks et al. 1995, Lieber and Friden 1993, Newham et al. 1988, Warren et al. 1993a and 1993b).

In several animal models, structural component within the muscle have been shown to fail after a single eccentric contraction (Brooks et al. 1995, Faulkner et al. 1993). Brooks et al (1995) investigated the effects of a single lengthening contraction on whole skeletal muscles of mice in situ. Maximally activated muscles were exposed to single stretches of 10, 20, 30, 50 or 60% strain magnitude, relative to L_O, at a rate of 2 L_O/s. It was found that a single stretch of strain magnitude of 30% L_O and greater produced significant isometric tetanic force deficits in the muscle one minute after the stretch. The isometric tetanic force deficit increased dramatically from a ~10% decrease in isometric tetanic force after the single stretch at 30% L_O strain magnitude to ~60% decrease in isometric tetanic force after the single stretch at with 60% L_O strain magnitude. Faulkner and coworkers (1993) also found that single lengthening contractions of more than 140% of muscle length during maximal contractions can results in significant damage to the muscle. The strain magnitudes which resulted in damage in these studies are greater than the strain magnitudes normal observed in human muscles in vitro during movements (Zajac 1989).

All investigations of contraction-induced damage in human have used multiple repetitions of the muscle to induce muscle damage. Several animal models have been

used to observe the effects of repeated loading on muscle fiber damage (Lieber and Friden 1993, McCully and Faulkner 1986 and 1985, Warren et al. 1993a). Lieber and Friden (1993) using an in situ rabbit tibialis anterior muscle preparation to observe the effects of 900 repeated stretches (lengthening contractions) with strain magnitudes of 12.5%L_O and 25%L_O at strain rates of 0.83L_O/s using muscle stimulation rates of 40Hz. These authors found that the fibers receiving 25%L_O strain had a significantly greater loss in maximum isometric tetanic force generation compared to the fibers strained at 12.5%L_O. In a second part of the study, they varied the timing of the imposed length change relatively to muscle activation between two groups receiving identical strain rates. This resulted in one group generating larger forces during the strain. They found no significant differences in the maximum isometric tetanic force generation between the two groups after the stretching protocols (900 repeating, 25%L_O or 12.5%L_O, 0.83L_O/s). Based on these observations, these authors suggested that it is not high force per se that causes muscle damage after eccentric contraction but the magnitude of the active strain.

Warren et al (1993a) varied the number of maximum eccentric contractions performed by an in vitro rat soleus muscle preparation from 1 to 10 (starting length .9L_O, strain 25%L_O, speed 2L_O/s). When the muscle performed eight or less eccentric contractions, there was no change in the maximal isometric tetanic force the fiber could produce compared to pre-exercise values. However, when the muscle performed greater than eight contractions there was a significant drop in the isometric tetanic force generating capacity of the fiber. Based on these observations, these authors proposed that the initiation of a contraction-induced damage is the cumulative result of multiple eccentric contractions. The findings of Warren et al. (1993a) are consist with the in situ mouse EDL study performed by McCully and Faulkner (1986) which demonstrated a linear decrease in the maximum isometric tetanic force measured 3 days after the damage protocol as the number of eccentric contractions increased from 15 to 150.

In humans, cumulative damage has also been observed during voluntary contractions (Brown et al. 1995). Brown et al (1995) had subjects perform 10, 30, or 50 voluntary maximal eccentric contractions of the knee extensors (60°/s, 100° range of motion). These authors found that the indirect indices of muscle damage measured 1,2 and 3 days after the exercises session significantly increased with the number of contractions performed.

Warren et al. (1993b) performed a systematic evaluation of the role of multiple factors (peak eccentric force, lengthening velocity, length change, and initial muscle length) in inducing muscle damaging using an in vitro rat soleus muscle preparation. In contrast to the study by Lieber and Friden (1993), these authors demonstrated that peak eccentric force, independent of lengthening velocity, initial (or final length) or length change, is associated with initiating muscle damage. McCully and Faulkner (1986) also demonstrated using an in situ mouse EDL preparation that the degree of damage resulting from eccentric contractions was directly related to the forces produced during the contractions.

The inconsistency in the findings of these studies may be the result of differences in experimental models and protocols, in particular, the fiber type compositions of the muscles studied, the use of both in vitro and in situ preparations, and the number and frequency of repeated contractions (Lieber and Friden 1993, McCully and Faulkner 1986, Warren et al. 1993a,). In 1902, Hough observed that the probability of sustaining a contraction-induced damage during novel voluntary eccentric exercise was directly related to the peak force generated during the exercise. Since Hough's seminal study it has been generally noted that there is a positive relationship between the probability of sustaining a contraction-induced damage and the moment generated during a novel voluntary eccentric exercise bout (see review Armstrong 1990).

It has been well demonstrated that the EMG recorded from a muscle during voluntary eccentric contractions is lower than the EMG recorded from the muscle during velocity match voluntary concentric contractions when the same moments are

generated (Basmajian and Deluca 1985, Bigland-Ritchie and Woods 1976, Bigland and Lippold 1954, Gibala et al. 1995, Nakazawa et al. 1993). For example, Gibala et al. (1995) recorded the surface EMG from the elbow flexors during slow (~50°/s) voluntary concentric and eccentric contractions using a load equal to 80 percent of the concentric maximum. They demonstrated that the average full wave rectified EMG was approximately 40 percent less during eccentric contractions. Based on the observations of reduced EMG levels during eccentric contractions several authors have speculated that fewer motor units are activated during eccentric contractions which results in *"high tensions"* being generated in the active motor units (Armstrong 1990 and 1984, MacIntyre et al. 1996, Newham 1983, Gibala et al. 1995, Golden and Dudley 1992). Furthermore, these authors have proposed that the *"high tensions"* generated in the active motor units result in mechanical disruption of structural elements in the active motor units or in the connective tissue that is in series with the contractile elements (Armstrong 1990 and 1984, MacIntyre et al. 1996, Newham 1983, Gibala et al. 1995, Golden and Dudley1992). Newham (1983) stated "if IEMG [intergrated electromyogram] is taken to reflect the total fiber activity, then during negative work about half the number of fibers are being activated to generate a given tension and therefore the tension per active fiber is doubled". Since surface EMG arises from the temporal summation of action potentials emanating from active motor units, it cannot be used as a direct indicator of the number of motor units recruited (Basmajian and Deluca 1985, Bernardi et al. 1996, Fuglevand et al. 1993). In other words identical levels of surface EMG can be obtained from a few motor units firing at high rates compared to numerous motor units firing at low rates.

Several investigators have proposed that compared to concentric and isometric contractions, the rate coding, pattern and recruitment strategy used during eccentric contractions may be unique (Enoka 1997, Howell et al. 1995, Moritani et al. 1988, Nardone et al. 1989). Howell et al. (1995) studied motor unit activity in the human first dorsal interosseus muscle using intramuscular fine wire electrodes. They found that during identical low force concentric and eccentric contractions, a larger number

of motor units were active during the concentric contraction. However, it remains unclear if the reduction in EMG levels observed in eccentrics compared to concentric contractions at identical high moment levels is the result of less motor units being active or the same number of motor units being activated at lower firing rates.

Transcutaneous application of electrical stimulation can be used to activate large numbers of motor units within a muscle at any specified firing rate within the motor unit's physiological capabilities (Morrissey 1988). It has been traditionally assumed that the recruitment order during nerve stimulation is the reverse of that seen during voluntary isometric contractions (Henneman and Mendell 1981). That is, electrical stimulation should recruit the largest and fastest conducting motor units with lowest intensities of stimulation and recruit the smallest and slowest conducting motor units at higher intensities. Reverse size order recruitment observed with nerve stimulation may not necessarily be extrapolated to transcut stim due to the difference in spatial orientation of the stimulating electrodes relative the nerves. Binder-Macleod et al. (1995) investigated the effects of various intensities and frequencies of NMES on the moment generation of the quadriceps during fatiguing isometric contractions. Based on the observations of minimal shifts in the force-frequency relationship during contractions at 20, 50 and 80% of maximum and variable fatigue at each intensity level, these authors concluded that the recruitment order observed during NMES is in marked contrast to the orderly recruitment that would be anticipated with volitional contractions.

Brown and colleagues (1996) studied the effects of imposed NMES on the knee extensors during 70 imposed movements as the subjects relaxed resulting in eccentric contraction (60°/s, 90° range of motion). Maximum isometric moment was measured 1,2,3,7 and 9 days post-exercise with and without superimposed electrical stimulation. Before the exercise bout, the intensity of the NMES was adjusted to evoke an isometric contraction equal to 50% of maximal isometric moment at a stimulation rate of 100 Hz. This stimulation setting was then used for the eccentric exercise session.

These authors found that maximum voluntary isometric moment generation was reduced 40-55% post-exercise. When electrical stimulation was superimposed on these maximal contractions the moment generation was observed to increase by ~10% at each testing day. However, when electrical stimulation was superimposed on a maximal contraction before the exercise no increase in moment generation was observed. Based on this observation, these authors proposed that there was a central limiting mechanism which restricted recruitment of partially damaged motor units.

Several reports in the literature refer to apparent differences in susceptibility of different muscle fiber types to voluntary eccentrically biased exercise. In animal muscle, it is equivocal whether voluntary eccentrically biased exercise causes damage preferentially to type I or type II fiber types (Armstrong et al.1983, Ogilvie et al. 1988). However, in studies involving humans the work of Friden et al. (1983b), Friden et al (1988) and Jones et al. (1986) are normally cited as direct evidence that type II fibers are preferentially damaged during voluntary eccentrically biased exercise.

Friden and colleagues (1983b) took biopsies from the vastus lateralis muscle of 9 subjects after 30 minutes of eccentric contraction-biased cycling. They found that 6 out of the 9 subjects had a greater percentage of Z-band streaming in type IIB fibers compared to type I fibers. Of the 3 subjects that did not show preferential type IIB damage, 1 subject had no damage in any fibers and 2 subjects had a higher percentage of type I fiber damage compared to type IIB. Jones and associates (1986) reported a greater degree of histological damage in type II fibers compared to type I fibers in the gastrocnemius muscle of only one subject after 90 minutes of walking backwards on a treadmill. Friden and coworkers (1988) took a total of 118 biopsies from the right vastus lateralis of 6 elite sprinters two hours of repeated bouts of sprint running. Of the 118 biopsies, 36% showed various degrees of focal damage. Of the fibers identified as damaged, 80% were identified as type IIB fibers. However since all biopsies were group together it is impossible to make any conclusion on how many of the subjects actually had preferential damage of their type II fibers.

It is difficult to interpret the human studies described above since they all involve voluntary movements. Warren et al (1994) suggested that altered recruitment patterns during different types of voluntary exercise may explain the varied results between these studies. These authors state that during voluntary movements "preferential damage to one fiber type may simply result from less recruitment of other fiber types". It remains unclear from the human studies if any preferential damage to type II fibers reported in the studies was the result of different loading in type II fibers during the exercise or due to greater intrinsic susceptibility to damage.

The work of Lieber and colleagues (1991, 1988) is often cited as direct evidence that type II fibers are preferentially injured by eccentric contractions. In their studies they utilized an in situ rabbit tibialis anterior muscle preparation to showed that after 1800 eccentric contractions over a 30 minute period only type II fibers demonstrated histological abnormalities. Lieber and colleagues (1991) suggested that the type IIB fibers may be have been more susceptible damage to fatiguing contractions compared to type I and type IIA fibers, based on their reduced ability to regenerate ATP after several minutes of contractions. These authors suggest that during a fatiguing contraction, type IIB would enter into a rigor or high-stiffness state due to a lack of ATP. They further postulate that subsequent stretch of the stiff fibers would mechanically disrupt the fibers, resulting in the cytoskeletal or myofibillar damage. However, in these studies all fibers within the muscle were activated at 40 Hz via peroneal nerve stimulation, which would result in type II fibers developing greater specific tensions than the type I fibers based on there force frequency relationship (Kernell et al. 1983).

The only study found that compares damage to type I and type II for similar specific tension loading profiles is that of Warren and colleagues (1994). These researchers compared the magnitude of damage in the mouse extensor digitorum longus (EDL) muscle which is predominantly fast-twitch (42%-type IIb, 56%-typeIIa, 2%-typeI) to the mouse soleus muscle which is predominantly slow-twitch (0%-typeIIb, 45%-typeIIa, 55%-typeI) after eccentric exercise using an in vitro muscle

preparations. Stimulation frequency was varied so the forces across the two muscles were identical during the eccentric contractions. Using light-microscopic histological analysis, they found significantly more damage in the EDL muscles compared to the soleus muscle 1 hour after the loading session. These observations would seem to support the previous voluntary exercise studies that have shown that fast-twitch muscle fibers are more susceptible than slow-twitch fibers to eccentric contraction-induced damage. However, Warren and colleagues noted that the soleus muscle of the mouse is recruited far more often than the EDL muscle of the mouse during normal daily activities. In a second part to their study, they unloaded the EDL and soleus muscles for 14 days before the eccentric exercise. They found that there was almost twice as much damage in the unloading soleus muscles compared to the normal soleus muscle 1 hour after identical eccentric loading. However there was no apparent difference in the level of damage between the normal and unloaded EDL muscles after the loading session. Based on this observation they suggested that for an given muscle fiber "it is not the fiber type composition per se that dictates its vulnerability to eccentric contraction-induced damage but its recent contractile history".

Differences in damage between type I and type II muscle fibers for identical loading profiles may result from ultrastructural differences between the two fiber types (Friden and Lieber 1992). The M band of type I fibers has been shown to have five distinct bridges, while type II fibers only have three bridges (Sjostrom et al., 1982). Terracio et al. (1989) found approximately three times as much spectrin in type I fibers compared to type II fibers. Friden and Lieber (1992) suggested that the decreased Z-band thickness in type II fibers would result in type II fibers being more susceptible to repetitive, high tension stresses compared to type I fibers

Adaptation to Eccentric Exercise

A number of investigators (Clarkson and Tremblay 1988, Golden and Dudley 1992, Mair et al. 1995, Newham et al 1987, Nosaka and Clarkson 1995, Nosaka et al

1992) have shown that the performance of a single bout of strenuous voluntary eccentrically biased exercise produces an adaptation such that many indirect indicators of muscle damage are significantly reduced when a second bout of strenuous voluntary eccentrically biased exercise is performed. Specifically, the recovery of 1) maximal voluntary isovelocity and isometric moment, 2) isometric moment during low frequency stimulation and 3) range of motion are significantly faster than that found after the first exercise bout. In addition, DOMS is less developed and intramuscular muscle protein release into the blood is extremely blunted (Clarkson et al. 1992, Golden and Dudley 1992, Newham et al. 1987, Newham et al. 1987, Nosaka and Clarkson 1995). Although no direct evidence of reduced muscle damage after a single bout of strenuous voluntary exercise could be found in the literature, Friden and coworkers (1983a, 1983b) reported that after 8-12 session of eccentrically biased cycling the frequency of Z-band damage was dramatically reduced compared to damage observed after only one session.

This adaptation has been shown to last from several weeks to several months after the initial exercise session (Newham et al. 1987, Nosaka and Clarkson 1995, Nosaka et al 1992). Nosaka and colleagues (1992) investigated the time course of the adaptation process. They had subjects perform identical bouts of maximum voluntary eccentric contractions (70 reps) of the forearm flexors muscles 6 weeks and 10 weeks apart and found that at 10 weeks there was still a significant reduction in indirect indicators of muscle damage although this reduction was less than that measured at 6 weeks. When subjects performed the same eccentric exercise bout 6 months later only creatine kinase levels in the blood stilled showed adaptation although this adaptation was greatly reduced compared to the adaptation at 6 and 10 weeks. Byrness and coworkers (1985) reported that a single exposure to 30 minutes of downhill running provided protection from both enzyme release and muscle soreness following a similar run 6 weeks later. In contrast, Schwane and Williams (1987) reported no change in the magnitude of enzyme release and little reduction in muscle

soreness after a 45 minutes downhill run following 2 weeks of downhill running for 5 to 15 minutes 5 consecutive days each week.

A number of authors (Armstrong et al. 1983, Armstrong 1984, Newham et al. 1987) have postulated that the adaptation process is the result of the first exercise bout causing damage and destruction to a population of susceptible fibers, possibly near the end of their life cycle. They suggest that the damage fibers undergo degeneration and release soluble enzymes into the blood stream and that only more resilient fibers remain which are better able to withstand the effects of eccentric exercise. However studies have shown that performing a bout of eccentric exercise that results in little or no release of creatine kinase can still result in an adaptation such that when an identical more strenuous second bout of eccentric exercise is performed many indirect indicators of muscle damage are significantly reduced (Schwane and Armstrong 1983).

Several authors (Newham et al. 1987, Schwane and Armstrong 1983) have suggested that the first bout of exercise causes an adaptation such that muscle fibers becoming more resistant to the fatiguing and damaging effects of eccentric exercise. Investigators have examined the effects of performing a second voluntary eccentric exercise session 2-5 days after an initial damaging voluntary eccentric exercise session (Ebbeling and Clarkson 1989, Ebbeling and Clarkson 1990, Nosaka and Clarkson 1995, Smith et al. 1994). Based on observation that the second exercise bout did not influence the time course of DOMS, CK levels in the blood, and strength recovery these authors concluded that the second exercise session did not exacerbate the damage caused by the first exercise session. However, the regeneration of muscle fibers in humans appears to begin around 4 days after damage (Armstrong 1990) and muscle biopsies normal show disruption for several weeks after the initial exercise session (Newham 1988, Jones et al. 1986).

Several authors (Golden and Dudley 1992, Clarkson et al. 1992, Friden et al. 1983a) have postulated that the adaptation from eccentric contractions is the result of a change in neural control strategy. Clarkson et al. (1992) hypothezied that "the first

bout of exercise produces an adaptation in the motor unit recruitment pattern over the range of motion such that less force is distributed among the fibers at any one point in time" which may "reduce the likelihood of severe or lethal damage to muscle fibers". There is evidence that the rate coding and recruitment strategies used during a specific task can be altered with training (Bernardi et al. 1996).

Delayed Onset Muscle Soreness

Exercise can result in the sensation of pain in skeletal muscle. Traumatic damage to muscle fibers or the surrounding connective tissues during exercise normally results in the sensation of pain at the instance of damage, with the pain often persisting for several days to weeks after the damage. Metabolic depletion and ischaemia during exercise can also result in the sensation of pain in skeletal muscle during the exercise, which disappears very rapidly after the exercise ends (Mills et al. 1982, Rodbard and Pragay 1968). There is another type of muscle pain which is caused by exercise and which has a very different and distinctive time course; it is not felt for a number of hours after the exercise and then persists for a number of days and is referred to as delayed onset muscle soreness (DOMS).

In 1902, Theodore Hough published the first report of delayed onset muscle soreness after exercise. Hough found that soreness developed in the flexor muscles of the middle finger 8-10 hours after performing rhythmical exercise. Hough postulated that this pain was most likely due to "some sort of rupture within the muscle". Since Hough's seminal paper an extensive volume of literature has accumulated on the occurrence of DOMS in humans after exercise. Reviews of this literature have been performed by several authors. (Armstrong 1984, Ebbeling and Clarkson 1989). The consensus of these reviews is that muscle soreness is felt between 8-24 hours after exercise and manifests as a dull, aching pain combined with tenderness and stiffness. The soreness peaks in intensity between 24 and 72 hours after exercise and usually disappears within 5-7 days. The soreness is more pronounced when the muscle is

palpated or during movements and there is no apparent correlation between the intensity or type of exercise and the time course of soreness development.

To date there has been no general agreement about the pathophysiological mechanisms responsible for DOMS. However, there is general agreement in the literature that DOMS is associated with contraction-induced damage (Armstrong 1984, Ebbeling and Clarkson 1989, Gibala et al. 1995, Smith 1991).

Several authors have proposed that afferent input from group IV and possibly group III receptors are responsible for sensation of DOMS (Ebbeling and Clarkson 1989, Friden et al. 1986, Smith 1991). It is known that group III and IV sensory neurons terminate in free nerve endings that are distributed primarily in the muscle connective tissue between fibers (Ebbeling and Clarkson 1989, Mense and Schmidt 1974). It has been also been established that group IV receptors respond to mechanical, chemical and noxious stimuli (Ebbeling and Clarkson 1989). However, there is little agreement on which type of stimuli(s) is responsible for the sensation of DOMS (Ebbeling and Clarkson 1989, Friden et al. 1986, Smith 1991).

After an extensive review of the literature, Smith (1991) suggested that the DOMS may be associated with a acute inflammation response. Smith suggested that white bloods cells start to migrate to the site of damage within a few hours of exercise which results in sensitization of type III and type IV afferent. Smith further postulated that small changes in pressure within the muscle would provide a mechanical stimulus and result in the sensation of soreness. Smith's (1991) hypothesis is partially supported by various studies in humans and rodents, which have shown increases in white blood cells in a muscle after repetitive voluntary or electrically evoked eccentric contractions. These studies have shown increases in the number of white blood cells in the muscle 4-24 hours post-exercise, with maximal levels 24-48 hours post-exercise which correlate well with the time course of DOMS (Armstrong et al. 1983, Jones et al. 1986, MacIntyre et al. 1996, McCully et al 1985). However, a few investigators (Friden et al. 1983b, Bobbert et al. 1986) have failed to

show a significant increase in white blood cells counts in human muscles after exercise although these muscles had significant DOMS.

Several authors have found significant increases in the resting intramuscular pressure of non-compartment for several days after eccentric exercise (Clarkson et al. 1992, Friden et al. 1986, Friden et al. 1988). However their is little correlation between muscle soreness which peaks 24-48 hours after exercise and compartment swelling which peaks 4-6 days after exercise (Clarkson et al. 1992, Friden et al. 1988).

Impairment in Moment Generation

Performance of voluntary concentric, isometric or eccentric contractions for an extended duration will lead to impairment in the muscles ability to produce maximal moments. The impairment in maximal moment production produced by isometric and concentric contractions is greatest immediately after the exercise and normally last for less than 2-4 hours. This impairment and recovery process has been well documented in both human (Gibala et al. 1995) and animal muscle (McCully and Faulkner 1985). However, in contrast to the moment generating impairment from concentric and isometric contractions, the impairment in maximal voluntary moment production after voluntary eccentric biased exercises has been reported to last up to 6 weeks (Howell et al. 1993, Newham et al. 1987, Clarkson et al. 1992). Isometric strength deficits of 45-85% have been shown to exist for several days following intense bouts of eccentric contractions (Clarkson and Tremblay 1988, Clarkson et al. 1992, Gibala et al. 1995, MacIntyre et al. 1996, Newham et al. 1987). These studies have generally found that voluntary isometric moment generation recovered to pre-exercise levels 5-10 days after the exercise.

Contraction-induced muscle damage are associated with a decrement in the ability to produce both voluntary and electrically stimulated forces (Gibala et al 1995, Warren et al. 1993b, Ogilvie et al. 1988, Friden et al. 1983b, Friden et al. 1988, Jones et al. 1986, Newham et al 1983, Stauber et al. 1990). The mechanism(s) responsible

for the marked reduction in strength are not well understood. It has been suggested that the strength deficit during voluntary contraction could be the result of the change in neural activation patterns that would "bypass" the more severely damaged fiber (Gibala et al. 1995, Clarkson et al. 1992). There is little or no relationship between the sensation of pain related to DOMS and the decrease in maximum voluntary isometric moment after eccentric exercise in highly motivated subjects (Ebbeling and Clarkson 1989, Clarkson et al. 1992, Newham et al 1987).

Golden and Dudley (1992) examined the moment/velocity relationship of the knee extensors of eight non-weight trained male subjects immediately after and 1, 4, 7 and 10 days after they had performed 100 repetitions of unilateral eccentric contractions of the knee extensors with a resistance equal to 85% of the post-exercise maximum. They measure angle specific isovelocity knee moment during concentric and eccentric maximum voluntary contractions at 60°/s and 180°/s over 0-90° of knee extension and maximum isometric moment. They found that 24 hours post-exercise the angle specific moments were significantly reduced by approximately the same percentage (~38%) at all velocities compared to pre-exercise values.

Mair and coworkers (1992) measured the peak isovelocity moment of the knee extensors during maximum concentric contractions at 30°/s, 90°/s and 180°/s in 6 non-weight trained subjects after 70 sub-maximal eccentric contractions. They found a larger decrease in maximum moment at 30°/s compared to 90°/s and 180°/s although this result was not significant.

Jones et al. (1985) measured the peak moment of the knee extensors during maximum isovelocity eccentric contractions at 90°/s, 180°/s and 300°/s and maximum isometric contraction at 80° of knee flexion in subjects after 30 minutes of eccentrically biased bicycling. Subjects (n=7) tested three days after the initial exercise bout showed a non-significant 9% decrease in peak moment at 90°/s and significant decreases in peak moment of 16% at 180°/s, 21% at 300°/s and 14% at 0°/s.

Gibala and coworkers (1995) measured the peak isovelocity moment of the forearm flexors during maximum voluntary concentric contractions at 30°/s and 180°/s and the peak isometric moment of the forearm flexors of 8 non-weight trained male subjects 0, 24, 48, 72, and 96 hours after sub-maximal eccentric exercise of the forearm flexors. They reported that at 0 and 24 hours post-exercise all three strength measures were significantly reduced by approximately the same percentage (~35%) compared to pre-exercise values. They reported that at 96 hours post-exercise only the peak isovelocity moment at 180°/s had recovered to post-exercise levels.

Shifts in Force/Length Relationship

Shifts in optimum muscle length for force production during isometric contractions have been reported for in vitro whole toad muscle preparations (Jones et al. 1997), for single frog fibers (Morgan 1990) and for electrically evoked contractions of human tricep surae muscle (Jones et al. 1997) for up to two days after repetitive eccentric contractions. Recently, Saxton and Donnelly (1996) have also shown that shifts in optimum muscle length occur during voluntary contractions after contraction-induced damage. They reported that the decline in maximum voluntary isometric moment of human elbow flexors, both with and without superimposed electrical stimulation, was significantly more pronounced at more acute elbow angles for up to 4 days after the initial exercise bout. The shift in the length tension curve in the studies by Jones et al (1997) and Saxton and Donnely (1996) recovered to pre-exercise values although there was still a significantly decrease in force production.

Morgan (1990) and Saxton and Donnely (1996) postulated that the shift in the length-tension curve is the unlikely the result of edema in the injured muscles since the shift is evident before any significant signs of edema. Morgan (1990) and Jones et al. (1997) have proposed that disruptions of numerous myofilaments in the injured muscle would leads to an increase in the series compliance of the injured muscle resulting in shifts of the length-tension curve towards longer length. Whereas, Saxton and Donnelly (1996) have postulated that the shift may be due to a

combination of over-extended sarcomeres and disruptions within tendinous attachments. However it is unclear how shifts in the length-tension curve recover so quickly since disrupted myofilaments and tendinous attachments would not be repaired within 4 days post-damage (Armstrong 1990).

3.0 NEUROMUSCULAR MODEL

Shifts in load sharing between motor units resulting from changes in motor unit recruitment and rate coding strategies have been difficult to verify experimentally largely because recruitment, rate coding and loading behaviors can only be monitored for a small fraction of the motor units participating in a voluntary contraction. Furthermore, investigations of the recruitment and rate coding strategies of motor units have generally been limited to low level voluntary contractions. Therefore in an attempt to quantitatively address how changes in recruitment and rate coding strategies can affect load sharing between motor units, a model was developed to predict the forces produced by individual motor units in a hypothetical motor unit pool during simulated voluntary contractions.

During the past century an large number of mathematical models have been developed to gain a better understanding of the various neuromuscular factors which result in the mechanical output of muscle. These models vary widely from models of single cross-bridge interactions to models of complex kinematic movements involving large numbers of muscles (Partridge and Benton 1981, Zajac 1989). A number of sophisticated neuromuscular models have been proposed in which the steady-state isometric force output of a muscle is based on the recruitment and rate coding of individual motor units from a hypothetical motor unit pool (Clamann 1993, Fuglevand et al. 1993, Heckman and Binder 1991, Kukulka and Clamann 1981). Fuglevand and colleagues (1993) and Heckman and Binder (1991) developed this form of neuromuscular model to investigate the steady-state system output of a muscle resulting from various recruitment and rate coding strategies.

The neuromuscular model developed by Heckman and Binder (1991) was comprised of 100 motor units based on properties derived experimentally for the cat medial gastrocnemius motoneuron pool and muscle. The maximum tetanic isometric force developed by the motor units in this model varied 40-fold across the motor unit pool with a skew towards small forces. The force developed by a motor unit at any

given fire rate was determined by the force-frequency relationship for the individual motor unit.

The neuromuscular model developed by Fuglevand and colleagues (1993) was comprised of 120 motor units based on experimental data from various animal and human motor unit studies. Motor unit twitch force was estimated as the impulse responses of a critically damped, second order system. The unfused twitch force amplitude of the individual motor units varied 100-fold across the motor unit pool and were inversely related to the rise time of the motor unit twitch. The force developed by a motor unit for a given firing rate was determined by motor unit twitch force summation taking into account nonlinear force-firing rate behavior of the motor units.

Both Heckman and Binder (1991) and Fuglevand and colleagues (1993) found that any steady-state force output from the modeled muscles could be achieved using several different recruitment and rate coding strategies that these authors proposed were physiologically possible. However these authors did not report on how the loading of individual motor units varied between these various strategies. No models examining load sharing between groups of motor units for different recruitment and rate coding strategies could be found in the literature.

The model developed in this study was adapted from the two neuromuscular models discussed above. Since this model was developed to investigate the load sharing phenomenon in humans, the properties of the hypothetical pool of motor units were based as closely as possible to the mechanical response seen experimentally in human motor units.

During the past 30 years there has been an extensive amount of literature devoted to the investigation of the mechanical response of individual motor units to different stimulation protocols under various experimental conditions. Assessment of the mechanical response to motor unit action potentials (MUAP) requires functional isolation of individual motor units (Burke 1981, Thomas et al. 1990a). To insure electrical stimulation of a single a motor unit it is necessary to either 1) dissect the fine filaments of centrally cut ventral roots so that electrical stimulation activates only

one motor axon or 2) penetrate the innervating motoneuron with an intracellular micropipette electrode which stimulates the motoneuron directly (Burke 1981). Once a motor unit is functional isolated the mechanical response to MUAPs can then be determined by measuring the force response across the two tendons of the whole muscle during the stimulation. Both methods of functional isolation are capable of sampling a relatively large proportion of the motor units with a motor unit pool and have been used to describe the mechanical response of individual motor units in a large number of mammalian (e.g. cat, rat, and rabbit) limb muscles (Burke 1981, Burke et al. 1976, Kernell et al. 1983, Stein et al. 1972).

Several techniques that have enable researchers to estimate the mechanical response of individual human motor units to single or multiple MUAPs through relatively non-invasive methods. The methods currently available for studying the mechanical response of human motor units to stimuli are spike-triggered averaging (STA), percutaneous nerve stimulation, intramuscular nerve stimulation, and intraneural stimulation (Garnett et al. 1979, Macefield et al. 1996, Nishizono et al. 1995, Stephens and Usherwood 1977, Thomas 1991). However, each one of these methods has technical limitations (Calancie and Bawa 1986, Lim et al 1995) which has resulted in these methods being used almost exclusively in small muscles during isometric contractions.

The STA method is one of the most widely used methods for the study of the single twitch response of motor units in muscles of intact humans (Lim et al. 1995). Initially developed by Buchtal and Schmalbruch (1970), this technique estimates the mechanical response of individual motor units from the force records of the whole muscle by synchronizing the force record to single unit EMG potentials during voluntary activation. The twitch response of a motor unit is then determined by averaging the force increment in response to 250-500 muscle action potentials from the same motor unit (Thomas et al. 1990a). With this method the twitch response of individual human motor units have been estimated for a number of muscles including the vastus lateralis (Nishizono et al. 1995), tibialis anterior (Feiereisen et al. 1997), as

well as for numerous small muscles of the hands, feet and forearms (Monster and Chan 1977, Milner-Brown et al. 1973b, Nishizono et al. 1990, Stephens and Usherwood 1977, Thomas et al. 1990a).

Synchronization between motor units can lead to errors in the estimation of the twitch properties using the STA method (Lim et al 1995). Since motor units are more likely to fire with some degree of temporal interdependence at higher force levels (De Luca et al. 1993), the STA method has generally been limited in large muscles to low to moderate contractions levels. Using the STA method during low level contractions may lead to a sampling bias of weaker motor units within the motor unit pool (Thomas et al. 1990b). This has limited the use of the STA method in the determination of the spectrum of twitch properties of the motor unit pool in large human muscles.

Several author have argued that single human motor units can be functionally isolated under some conditions using either intramuscular (Andreassen and Arendt-Nielsen 1987, Garnett et al. 1979, Stephens and Usherwood 1977) or intraneural (Macefield et al. 1996, Thomas et al. 1991a, Thomas et al. 1991b, Westing et al. 1990) microstimulation techniques. With these techniques, the motor axon of a single motor unit is electrical stimulated using microelectrodes inserted into the muscle or nerve. However, with the exception of the medial gastrocnemius and tibialis anterior muscles, these techniques have generally been limited to the small muscle of the hands and feet.

At present there are no single studies in the literature that provided sufficient information on all the necessary parameters needed to produce even a basic neuromuscular model of a large humans muscle (Zajac 1989). In addition, there is limited information about the mechanical response of motor units during dynamic contractions and/or fatiguing contractions. The hypothetical pool of human motor units modeled in this study was based on the existing data on the mechanical response of human and animal motor unit during non-fatiguing, isometric contractions.

Studies on both human and animals have demonstrated that there is a substantial range in the peak twitch and maximal tetanic forces that can be exerted by individual motor units within one muscle (Burke 1981, Kernell et al. 1983, Macefield et al. 1996, Thomas 1991). Experimental work on large muscles in the cat have typically found that the range of peak twitch forces of motor units in a muscle varied between 100-200 fold (Burke 1981, Burke et al. 1976, Kernell et al. 1983). Investigations of human muscles have found that the range of peak twitch forces of motor units within a muscle varies dramatically between muscles groups. Studies of the very small muscle of the fingers and toes have generally found a narrow range of peak twitch forces ranging from 10-20 fold (Macefield et al. 1996, Nishizono et al. 1990, Thomas et al. 1991a, Thomas et al. 1991b). The slightly larger extensor carpi radialis and extensor digitorum communis muscles in the forearm have shown larger ranges of peak twitch forces varying between 25 (Monster and Chan 1977) and 300 fold (Riek and Bawa 1992). The moderate sized tibialis anterior and medial gastrocnemius muscles have been shown to have ranges of peak twitch forces between 16 (Andreassen and Arendt-Nielsen 1987) and 150 fold (Feiereisen et al. 1997, Garnett et al. 1979). Although there have been no investigation of the complete range of peak twitch forces in large human muscles, Nishizono and coworkera (1995) showed using the STA method that during an isometric knee extension at only 3% MVC there was a 9 fold range in the peak twitch force of the motor units recorded from the vastus lateralis. Generally these human and animals studies have found that the frequency distribution of the peak twitch forces in the muscle is markedly skewed such that a large number of units produce small forces, whereas few units generate relatively large forces (Burke 1981, Burke et al. 1976, Feiereisen et al. 1997, Garnett et al. 1979, Monster and Chan 1977, Thomas et al. 1990a).

The rise times of motor units twitches in human muscles have been reported to extend along a continuum from 50 to 107 ms for the toe extensors (Macefield et al 1996), 35 to 85ms for the thenar muscles (Thomas et al. 1991a), 40 to 110 ms for the medial gastrocnemius (Garnett et al. 1978) and 21 to 82 ms for the tibialis anterior

(Friereisen et al. 1997). The relationship between rise time and peak twitch force in human muscles is unclear. In many of the small muscles there is no clear relationship between twitch force and rise time (Macefield et al. 1996, Nishizono et al. 1990, Thomas et al. 1991a, Thomas et al. 1991b, Westing et al. 1990). However in the tibialis anterior, medial gastrocnemius and extensor digitorum communis muscles there appears to be a clear relationship between rise time and peak twitch force such that motor units with faster rise times have larger peak twitch forces (Andreassen and Arendt-Nielsen 1987, Garnett et al. 1979, Monster and Chan 1977). This inverse relationship between rise time and peak twitch force has been shown in numerous animal muscles (Burke 1981, Burke et al. 1976)

Motor Unit Pool

The number of α-motoneurons innervating a large human limb muscle have been reported to vary from 700 to 1500 motor units (Enoka 1995). The motor unit pool for this model consisted of 21 motor units. In this model a single stimulus to the α-motoneuron resulted in the generation of a single MUAP. Each MUAP resulted in a all-or-none contraction of the muscle fibers innervated by the α-motoneuron. The propagation of the MUAP was considered instantaneous. The force response of the 21 motor units to single and multiple stimuli were varied to represent the ranges of force responses seen within motor unit pools experimentally. With fewer motor units there is less flexibility in the system for force generation.

The findings from the discussed studies on motor unit properties were incorporated into the model as follows:

1. The range of peak twitch forces across the 21 motor units was 100-fold. The smallest peak twitch force was given an arbitrary value of 0.01.
2. The rise times varied from 30 to 90 ms across the 21 motor units. Rise times were distributed evenly across the motor unit pool.

3. The relationship between peak twitch force (P) and rise time (R) was approximated as an inverse power function in the form

$$P(n) = \frac{\left(\frac{90}{R_n}\right)^{\log_3 100}}{100} \tag{3.1}$$

Where

$P(n)$ peak twitch force of the n^{th} motor unit (arbitrary units)

R_n rise time of the n^{th} motor unit

A plot of the peak twitch forces as a function of rise time for the hypothetical motor unit pool is shown in Figure 1. With this model 50 percent of the motor units had twitches with less than 0.1 units of force but had rise times that covered nearly two-thirds of the range. This distribution is similar to experimental data observed in the cat gastrocnemius muscle (Burke 1981)

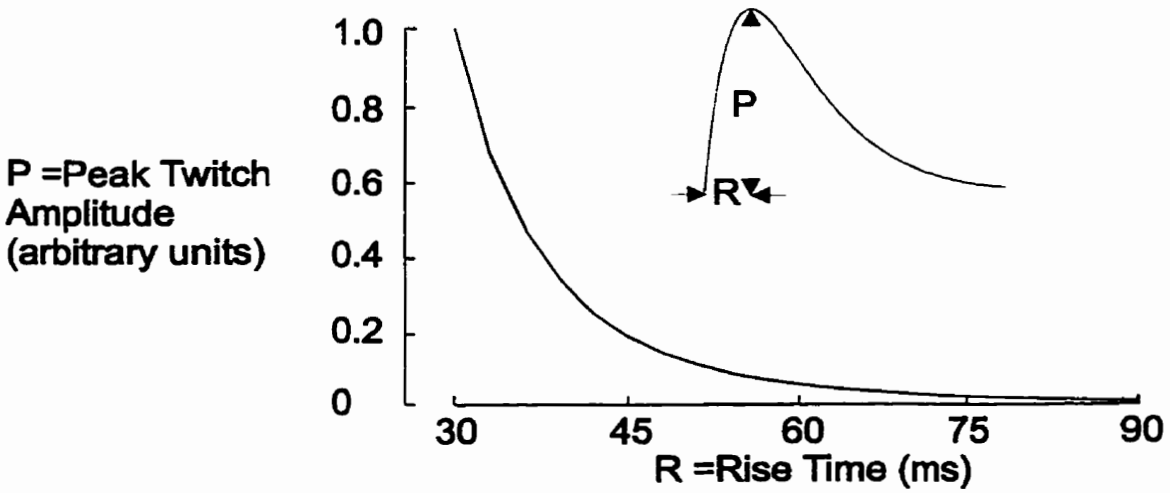

Figure 1. Relationship between peak twitch force (P) and rise time (R) for the hypothetical motor unit pool. A simulated isometric motor unit twitch is shown.

Motor Unit Twitch Response

The simplest way to predict the response of a linear system to signals of various frequencies is to take the Fourier transform of the response to a brief stimulus (Milner-Brown et al. 1973b). If motor unit force production is considered as a linear process, the motor unit twitch can be regarded as the impulse response of the motor unit system, and the Fourier transform of the twitch would give the frequency response of the system (Mannard and Stein 1973, Stein et al. 1972).

The frequency response of motor units have been studied in cats using sinusoidally (Rosenthal et al. 1970) and randomly (Stein et al. 1972, Mannard and Stein 1973) modulated stimulus trains and in the first dorsal interosseous muscle of humans during voluntary contractions (Milner-Brown et al. 1973b). These studies have demonstrated that for mean firing rates less than 10Hz, the Fourier transform of the twitch closely predicts the frequency response of the muscle. These author proposed that for low firing rates the motor units in these muscles are well approximated as a critically damped, second order system.

The impulse response, $f(t)$, of a second-order system with critical damping can by expressed as

$$f(t) = (A + Bt)e^{-t/R} \tag{3.2}$$

Where A,B and R are constants to be determined (Boyce and Diprima 1986).

The passive force developed by a motor unit at rest varies with muscle length (Zajac 1989). For this model it is assumed that the muscle is in a shortened position and the force across the motor unit at rest is negligible compared to the motor unit twitch force.

Substitution $f(0) = 0$ into equation (3.2) yields A = 0.

The peak twitch force occurs when the slope of *f(t)* is 0 for t>0. Differentiating equation (3.2) yields

$$\frac{df(t)}{dt} = Be^{-t/R}(1-t/R) = 0$$

(3.3)

Equation (3.3) will only be satisfied when t = R. This results in the constant R being equal to the rise time of the motor unit twitch.

The magnitude of the peak twitch force (P) developed by the motor unit can then be obtained by setting t = R in equation (3.2).

$$P = f(R) = \frac{BR}{e}$$

(3.4)

Solving for B in equation (3.4) yields

$$B = \frac{Pe}{R}$$

(3.5)

Substitution of equation (3.5) into equation (3.1) yields

$$f(t) = \frac{Pt}{R}e^{1-(t/R)}$$

(3.6)

Equation (3.6) was used to represent a motor unit twitch where

f(t) force response of the motor unit to a single stimuli at *t*=0

R rise time (ms)

P peak twitch force (arbitrary units)

Equation (3.7) was then used to represent the motor unit twitch responses for a pool containing n motor units by

$$f_n(t) = \frac{P_n \cdot t}{R_n} e^{1-(t/R_n)} \tag{3.7}$$

Nonlinearity of Twitch Summation

The summation of motor unit twitches during unfused tetanic contractions of mammalian muscle is highly nonlinear, with significant serial dependence between successive stimuli (Burke 1981). The gain of the nerve-muscle system can be considered as the area under the force-time curve resulting from each stimulus (Mannard and Stein 1973). As the stimulus rate to the muscle changes the gain of the nerve-muscle system is altered (Burke 1981, Mannard and Stein 1973). The sigmoid relationship between isometric force and stimulation rate (Macefield et al. 1996, Thomas 1991b) reflects the dependence of muscle gain on stimulus rate. The ratio of force to stimulation rate at any point on the force-stimulation rate curve is equal to the low-frequency gain (Kernell et al. 1983)

It has been well demonstrated that during low firing rates, motor units with slow rise times have faster force summation than motor units with fast rise times (Macefield et al. 1996, Thomas et al. 1991b). In addition, the slower motor units also obtain fused tetanus at lower firing rates then fast motor units. However, Kernell and colleagues (1983) found that when the firing rate is normalized as a function of the rise time of the motor unit, the force-firing rate curves are similar for most motor units in the cat hindlimb muscle.

Based on this finding the stimulus rate to the α-motoneuron was normalized to the rise time of the motor unit by:

$$SR_n = R \cdot SR \tag{3.8}$$

Where

SR_n normalized stimulus rate

R rise time of the motor unit (sec)

SR stimulus rate (1/sec)

Based on experimental evidence in cats, Burke and coworkers (1976) suggested that the greatest gain in motor unit force occurs when the time between successive MUAPs is equal to the rise time of the motor unit (SR_n=1). In addition, Burke and coworkers (1976) observed that the motor units in the cat displayed constant gain for normalized firing rates below approximately 0.4. Further evidence of constant gain is shown by the frequency response studies of motor units. These studies have generally observed that the gain of motor units below 10Hz is constant and similar in magnitude to an isolated twitch (Mannard and Stein 1973, Rosenthal et al. 1970, Stein et al. 1972). Burke and coworkers (1976) demonstrated that the maximum gain at SR_n=1 was approximately 3 times larger than the low stimulus rate gain.

This nonlinear force behavior was incorporated into the model by allowing the gain in motor unit force to change with stimulus rate. Motor units receiving normalized stimulus rates less than or equal to 0.4 were assumed to have a constant gain (G =1). The gain function for motor units with normalized stimulus rates greater than 0.4 was based on a model by Fuglevand et al (1993).

Fuglevand and coworkers (1993) suggested that the gain *(G)* of an isolated twitch for a normalized firing rate greater than 0.4 could be determined by:

$$G(n, SR_n) = \frac{1 - e^{-2(SR_n)^3}}{SR_n} \tag{3.9}$$

Where $G\ (n,\ SR_n\ )$ is the gain of the n^{th} motor unit receiving a normalized stimulus rate SR_n

Using this function, the peak gain occurs near the normalized stimulus rate of 1.0 and is approximately three times larger than the gain at normalized stimulus rates less than 0.4. This gain was then used to amplify the motor unit twitch. The twitch response of a motor was then represented as

$$f_{n,SR}(t) = G_{n,SR} \cdot \frac{P_n \cdot t}{R_n} e^{1-(t/R_n)} \tag{3.10}$$

The total force-time response of a motor unit $F_n(t)$ resulting from a series of stimuli was calculated as the sum of the individual twitch responses for the motor unit. The force-time response for the motor unit was represented as

$$F_{n,SR}(t) = \sum_{x=1}^{k} f_{n,SR,x}(t) \tag{3.11}$$

Where k is the total number of stimuli at time t

The predicted force output of a motor unit for a normalized firing rate of 1.0 is approximately 85 percent of the predicted maximum force. This force-frequency relationship is similar to the results of Kernell and colleagues (1983) that demonstrated that motor units of the peroneus longus muscle in the cat produced between 79-89 percent of its maximal force at a normalized stimulus rate of 1.0. The twitch response and force summation response of two motor units with fast and slow rise times is displayed in Figure 2. The profiles of force summation displayed in Figure 2. are visual similar to those seen experimentally in fast and slow motor units (Kernell et al. 1983).

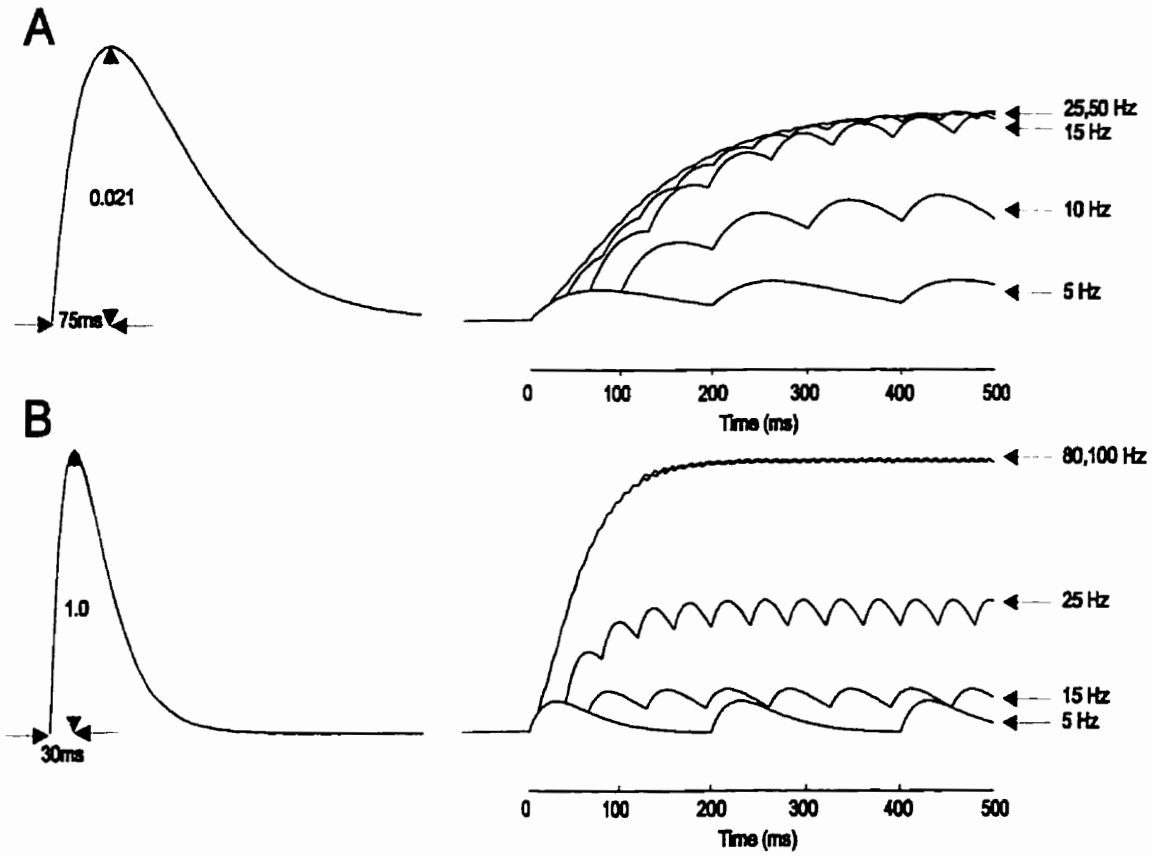

Figure 2. Simulated twitch response to a single stimuli and force summation response to stimulus trains for two motor units. A - motor unit with a slow rise time (75ms) with force summation shown for stimulus trains at 5, 10, 15, 25 and 50 Hz. B - motor unit with a fast rise time (30ms) with force summation for stimulus trains at 5, 15, 25, 80 and 100 Hz.

Total Force Output

The summation process for the mechanical output of motor units is uncertain. Experimental evidence suggests that the summation of motor unit force is a nonlinear process (Clamann and Schelhorn 1988, Morgan and Proske 1984).

Studies of summation of twitches produced by stimulating large groups of motor units have demonstrated that the twitch tension resulting from simultaneous stimulation of two or more groups of motor units is often less than the predicted linear sum of the tensions produced by each group (Brown and Matthews 1960, Hunt and Kuffler 1954). In contrast, several investigators have demonstrated that the combined tetanic force of 2-4 motor units is often greater than the algebraic sum of the forces produced by each unit individually (Clamman and Schelhorn 1988, Morgan and Proske 1984).

Systematic investigations of mechanical summation of multiple motor units over wide ranges of whole-muscle forces are not yet available (Heckman and Binder 1991). Due to a lack of knowledge about the summation rules over the entire force range, the total force output of the model was determined as the linear sum of the individual motor unit forces given by equation (3.12):

$$F_T(t) = \sum_{n=1}^{21} F_n(t)$$

The force output of the model for a simulated maximal voluntary contraction is shown in Figure 3. In accordance with the Size Principle (Henneman and Mendell 1981) motor units with smaller twitch forces were recruited earlier than motor units with larger twitch forces. The stimulus rates of the motor units were based on the rates observed on in human hand muscles during maximum voluntary isometric contractions (De Luca et al.1982, Bigland-Ritchie et al. 1983, Freund et al. 1975, Kukulka and Clamann 1981). These authors have found that the maximum firing rate of motor units typically range from 20-35 Hz and that small motor units normally achieve higher firing rates compared to larger motor units.

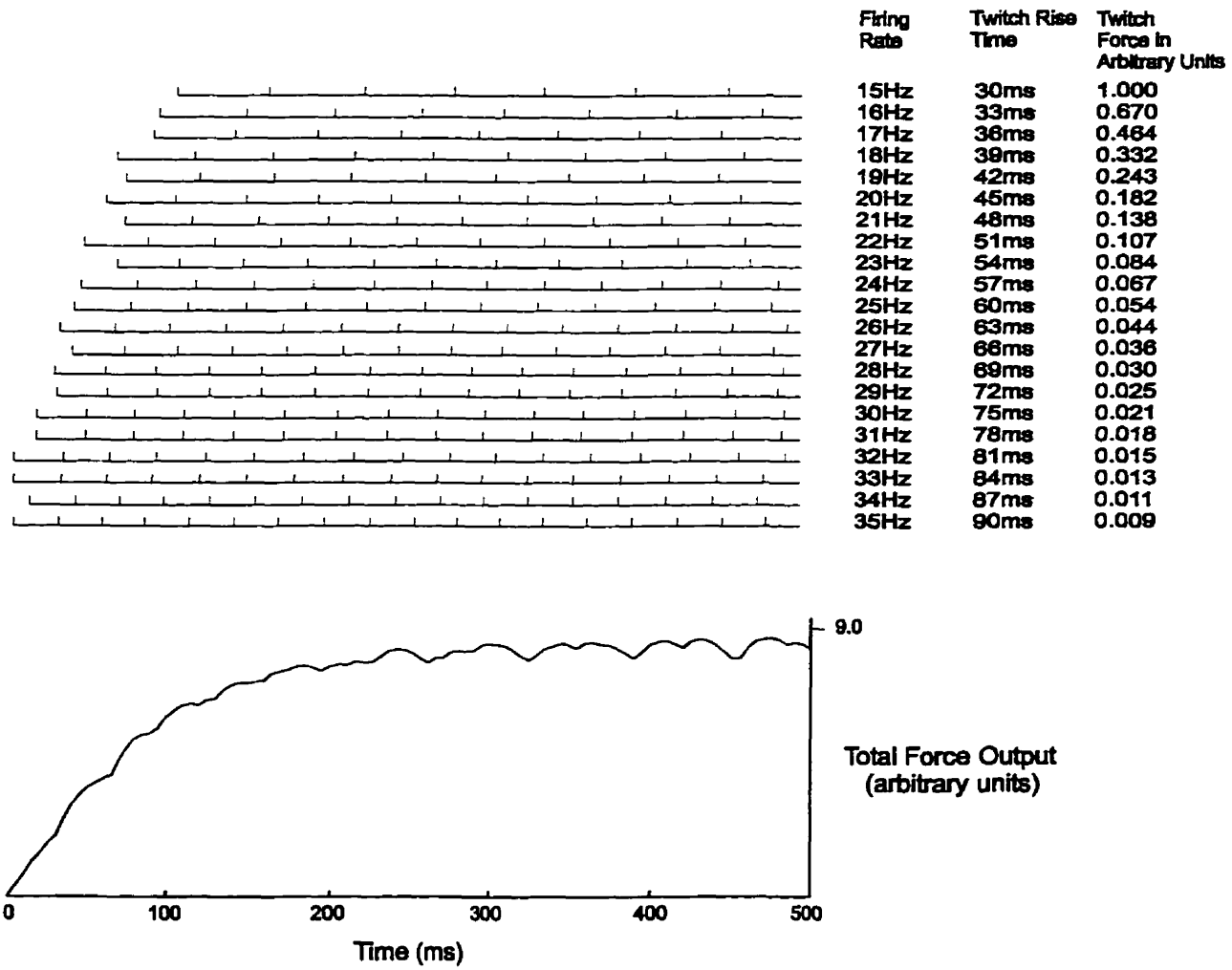

Figure 3. Force output of the model during a simulated maximally voluntary activated contraction of all motor units. Each tick marks correspond to a stimuli given to the motor unit. The twitch force, rise time and firing rate of each motor unit is shown on the right.

Nonlinearities in the force response of motor units resulting from activation history were not included in this model. This includes effects resulting from post-tetanic potentiation (Thomas et al. 1991b, Sandercock and Heckman 1997), fatigue (Thomas et al. 1991b) and motor unit doublets (Burke et al. 1970, Sandercock and Heckman 1997).

4.0 REVIEW OF DYNAMOMETRY

Isovelocity Dynamometer

Isovelocity dynamometers are computer-controlled hydraulic or electrically driven devices used primarily by researchers and clinicians to estimate RJM. During standard dynamometer tests the limb of a subject is attached to the dynamometer's actuator arm (Appendix A). The axis of rotation of the actuator arm is aligned with the axis of rotation of the joint being tested enabling the attached limb and actuator arm to rotate together in the same plane of motion. In one brand of isokinetic dynamometers (Kincom) the forces applied to the actuator arm are measured using a force transducer which bridges the gap between the actuator arm and limb attachment pad. This force transducer is only sensitive to forces perpendicular to the long axis of the actuator arm in the same plane of motion as the actuator arm. The moment recorded by the dynamometer during testing is the product of force measure by force transducer (F_F) and the distance from the axis of rotation of the actuator arm to the middle line of the resistance pad (L_D).

Many researchers have assumed that the RJM is equal to the moment measured by the dynamometer (Perrine and Edgerton 1978, Thorstenson et al. 1976). However, it is been well established that the moments obtained using an isokinetic dynamometers are not equivalent to the RJM (Chow et al. 1997, Herzog 1988, Kaufman et al. 1995). Gravitational effects, inertial effects, non-rigidity of the actuator arm/human limb segment, axial misalignment, and body stabilization effects can all affect the measurement of moments by an isovelocity dynamometer (Chow et al. 1997, Herzog 1988, Kaufman et al. 1995, Rothstein et al. 1987, Winter et al. 1981).

Kaufman and colleagues (1995) used a body-mounted triaxial electrogoniometer to quantify the errors associated with dynamometer measurments of knee flexion and extension at 60°/s and 180°/s. These authors concluded that only

gravitational and inertial effects resulted in significant errors between the measured dynamometer moment and the RJM about the knee. Herzog (1988) accounted for many of the differences between RJM about the knee and the measured dynamometer moment during knee extensions at 0, 120°/s, 240°/s. Herzog concluded that in the "isovelocity" range of the movement errors resulting from axial misalignment were minimal if the knee joint axis and axis of rotation of the actuator arm were carefully aligned before each trial and the subjects leg and upper body were well stabilized.

5.0 Objectives and Hypotheses

Purpose

The complex interaction between the multiple neural, mechanical and intrinsic muscular factors responsible for muscle damage during voluntary movements has not been well elucidated. Specifically, the association between neural recruitment strategies used to activate muscles during voluntary exercise and the occurrence of muscle damage has not been examined. This investigation was undertaken in order to explore the role of motor unit activation in the initiation of exercise induced muscle damage. In addition, this study examines the EMG/angle/angular velocity relationship of the knee extensor muscles in order to examine possible differences in neural activation between contraction types, contraction velocities and range of motion during maximal voluntary contractions.

Objectives

DOMS Study

Studies Objective: To examine contraction-induced muscle damage in the knee extensor muscles 24 hours after electrically evoked eccentric exercise in order to gain insight into the possible association between different neural activity pattern and muscle damage.

1. To determine and compare voluntary moment generation of the knee extensors pre-exercise and 24 hours post-exercise through the analysis of the moment/angle/angular velocity relationships of the knee extensors.
2. To determine and compare soreness in the knee extensors pre-exercise and 24 hours post-exercise through examination of pain intensity scales.
3. To compare strength and soreness measures from the NMES group to a control group that does not perform the electrically evoked eccentric exercise session.

EMG Study

1. To examine the EMG/angle/angular velocity relationship of the knee extensor muscles in order to examine possible differences in neural activation between contraction types, contraction velocities and range of motion during maximal voluntary contractions.

Neuromuscular Model

1. To illustrate how changes in recruitment and rate coding strategies can affect the loading of motor unit's muscle fibers.

Hypotheses

DOMS Study

1. The control group will show no significant changes in maximal voluntary moment generation or soreness in their knee extensors between test 1 and a test 24 hours later.
2. Indirect evidence of contraction-induced muscle damage in the knee extensor muscles will be evident 24 hours after the electrically evoked eccentric exercise. Specifically:
    a) The maximum voluntary isometric moment will be lower 24 hours post-exercise compared to pre-exercise measures.
    b) The moment/angle/angular velocity relationship will show regional deficits 24 hours post-exercise compared to pre-exercise measures.
    c) The soreness in the knee extensor muscles will be significantly increased 24 hours post-exercise compared to pre-exercise measures.

EMG Study

1. A substantial degree of neural regulation will exist that limits EMG activation level during maximal voluntary contractions. The relative degree of neural regulation will be contraction type, contraction velocity and range of motion dependant.
   Specifically:

   a) The EMG magnitude during maximum voluntary eccentric contractions will be lower than the EMG magnitude during maximum voluntary concentric or isometric contractions.
   b) The EMG magnitude will increase with increasing angular velocities during maximum voluntary concentric contractions.
   c) The EMG magnitude during maximum voluntary isometric contractions will not exceed the EMG during high speed concentric contractions.

Neuromuscular Model

1. Different physiologically feasible recruitment patterns will result in substantially different loading patterns in the muscle fibers of a motor unit.

Delimitations

1. This study examined subjects 18-40 years of age.
2. Isovelocity measurements were delineated to those taken with the hip position assumed when the backrest was reclined 15° from vertical.
3. Isometric strength measures were delimited to testing at 70° of knee flexion and only knee extension was examined.
4. Velocity spectrum strength testing was delimited to the testing velocities of 50, 100, 150, 200, and 250°/s and examined knee extension movements.
5. Range of motion during strength testing was delimited to 5° to 95° of knee flexion.
6. Electromyography measures were delimited to the knee extensor and flexor muscles.

Limitations

1. Analysis of moments and electromyography was limited to single segment motion in this study which is not necessarily representative of neuromuscular activity during functional multi-segmental motion.
2. Analysis of electromyographic signals was limited to activation level analysis and may not represent the findings of single motor unit activity.
3. The spectrum of velocities tested in this study is only a portion of the velocity spectrum encountered during functional activities.
4. The measures taken were dependent on the subjects abilities to provide maximal effort.

6.0 METHODOLOGY

SUBJECTS

Two separate experiments were performed in this study. Two groups of 6 subjects were recruited for the DOMS section of the study. These groups were designated as the control group and the neuromuscular electrical stimulation (NMES) group. One group of 8 subjects, designated as the EMG group, was recruited for the EMG section of the study.

Inclusion Criteria

Subjects between the ages of 18 and 40 years were included in this study. Subjects were physically active, participating in self reported aerobic exercise of greater than 20 minutes at least twice/week.

Exclusion Criteria

Subjects were excluded if they had any history of serious damage to the dominant lower extremity, any self reported restriction in range of motion of dominant lower extremity, or a current damage or disease affecting the non-dominant lower extremity. Exclusion criteria also included those with known cardiovascular disease or any other medical conditions which might preclude involvement in the study (i.e. history of arthritis or other inflammatory conditions affecting the hip, ankle or foot). Subjects participating in the DOMS design section were excluded if they reported any substantive participation in lower extremity weight training.

Recruitment

A sample of convenience was recruited by word of mouth from the Bannatyne Campus and the Fort Garry Campus of the University of Manitoba.

Selection

After being recruited for the study, the appropriateness of the subject's inclusion into the study was determined (Appendix C).

Informed Consent

All subjects were required to sign an informed consent form prior to participation in this investigation (Appendix B). This study was approved by the University of Manitoba Faculty of Medicine Committee on the Use of Human Subjects in Research.

INSTRUMENTATION

Dynamometer

Isovelocity dynamometers have been shown to be capable of providing reproducible dynamometer moment measurements for various testing protocols using concentric, eccentric and isometric contractions of the knee extensors (Gleeson and Mercer 1992, Harding et al. 1988, Webber 1996).

A Kinetic-Communicator (Kin-Com 500H, Chattecx Corporation, Hixson, TN) isovelocity dynamometer (Appendix A) was used in this study to evaluate the knee flexor and extensor knee moments generated by the subjects during maximal voluntary contractions and to administer passive knee motion. The actuator arm for this device was hydraulically driven and servo-controlled by a microprocessor through a valve. By varying the voltage across the valve the angular velocity and direction of the actuator arm can be controlled. The actuator arm for this device could be programmed to rotate at a constant angular velocity (isovelocity) independent of the force applied to the actuator arm by the subject's limb.

The Kin-Com is equipped with a strain gauge force transducer which bridges the gap between the actuator arm and limb attachment pad. The force transducer uses

a 4 gauge bridge arrangement with two temperature compensation elements secured on the surface of a steel 'I' bar. Under normal ambient room temperatures this transducer had a resolution equal to 1N over the measurement range of 4kN (±2000N). The Kin-Com is also equipped with an AC electric generator type tachometer and a wire wound, linear type potentiometer which are attached to the actuator arm. The force transducer, tachometer and potentiometer were used to measure the force applied by the subject's limb to the actuator arm, the angular velocity of the subject's limb and angular position of the subject's limb respectively.

The mechanical reliability of the Kin-Com has been shown to be highly reliable for measurements of angular velocity, angular position and force measurements in both static and dynamic tests (Farrell and Richards 1986, Mayhew et al. 1994).

Estimation of Resultant Joint Moment

To estimate the resultant moment about the knee joint (RJM_K) from moments recorded on a dynamometer during knee extensions the lower extremities were modeled as a mechanical system composed of two rigid elements, 1) the leg and 2) the human shank-foot system. The body segments were assumed to be interconnected by a simple hinge joint (no translation). Although knee joint translation does occur in normal and pathological knee joints, its magnitude is relatively small (e.g. 2cm) and difficult to measure (Chao 1980). Several other assumptions are necessary for this model:

1. The transverse axis of the knee joint is initial aligned with the axis of rotation of the actuator arm and remains aligned throughout the movement.
2. Relative motion between the actuator arm and leg does not occur.
3. The moment of inertia of the shank, foot and resistance pad about the transverse axis of the knee joint remains constant throughout the movement.

4. All forces (excluding forces resulting from limb weight) applied by the subject's limb to the actuator arm are perpendicular to the long axis of the actuator arm in the same plane of motion as the actuator arm.

During ideal isovelocity motions (angular acceleration = 0) and isometric contractions the condition of static equilibrium is applicable ($\sum M = 0$). The RJM_k can be calculated during periods of static equilibrium by compensating the recorded dynamometer moment for the moment resulting from the weight of the leg, foot and resistance pad (Winter et al. 1981). The sum of the moments about the knee during ideal isovelocity motions and isometric contractions is represent by:

$$\sum M_k = RJM_k - (m_{SF}\ L_{cm} + m_R\ L_D)\cos\theta - F_F\ L_D = 0 \qquad (6.1)$$

Where

F_F	force measure at the dynamometer's force transducer (N)
L_D	distance between the line of action of F_F and the transverse axis of the knee joint (m)
L_{cm}	distance between the center of gravity of the shank-foot complex and the transverse axis of the knee joint
$\sum M_k$	sum of the moments about the transverse axis of the knee joint (Nm)
m_{SF}	mass of the shank and foot (kg)
m_R	mass of the resistance pad (kg)
RJM_k	resultant moment about the transverse axis of the knee
θ	angle of knee flexion (rad)

Rearranging equation (6.1) yields

$$RJM_k = F_F L_D + (m_{SF} L_{cm} + m_R L_D) \cos\theta \quad (6.2)$$

During normally "isovelocity" testing on a isokinetic dynamometer the actuator arm undergoes varying degrees of angular accelerations. The angular acceleration occurs throughtout the range of motion with the largest angular accelerations occurring near the end ranges of motion (Chow et al. 1997, Kaufman et al. 1995). During periods of angular acceleration the condition of static equilibrium validity is questionable and the dynamometer moment must be compensated for both inertial and moment of the weight effects (Chow et al. 1997, Herzog 1988, Kaufman et al. 1995). The compensation of the dynamometer moment is based on the mechanics of angular acceleration governed by equation (6.3)

$$\Sigma M = I\alpha \quad (6.3)$$

Where

- I mass moment of inertial of a solid body about its rotational axis
- α angular acceleration of the solid body
- ΣM sum of the moments about the rotational axis

The mass moment of inertial of the shank, foot and resistance pad about the transverse axis of the knee joint is represent by:

$$\Sigma I_K = I_R + I_{S\,F} \quad (6.4)$$

Where

- I_K mass moment of inertia of the shank, foot and resistance pad about the transverse axis of the knee joint (km·m2)

I_{SF} mass moment of inertia of the shank and foot about the transverse axis of the knee joint ($km \cdot m^2$)

I_R mass moment of inertia of the resistance pad about the transverse axis of the knee joint (km·m2)

Combining equations 6.1, 6.3, and 6.4 yields

$$(I_R + I_{SF})\,\alpha_L = RJM_k - (m_{SF}\,L_{cm} + m_R\,L_D)\cos\theta - F_F\,L_D \qquad (6.5)$$

Where α_D represents the angular acceleration of the shank (rad/s).

Rearranging equation 6.5 yields

$$RJM_k = (I_R + I_{SF})\,\alpha_L + (m_{SF}\,L_{cm} + m_R\,L_D)\cos\theta + F_F\,L_D \qquad (6.6)$$

Equation 6.6 is used to estimate the RJM_k based on the moments recorded by the dynamometer during knee extensions.

Estimation of Moment of Inertia

Data on the radius of gyration of individual human limbs about a transverse axis through the distal joint center of the limb as a function of the limb's length have been provided (Dempster 1955). Once the mass moment of inertia of a limb is determined, the parallel axis theorem can then be applied to determine the mass moment of inertia of the limb through any parallel axis (e.g. Enoka 1994). The mass moment of inertia of the shank and foot about the transverse axis of the knee joint can be represented as:

$$I_{SF} = m_S\,(k_S\,L_S)^2 + m_F\,(k_F\,L_F)^2 + m_F\,L_S^2 \qquad (6.7)$$

Where

L_S length of the shank along its long axis (m)

L_F length of the foot along its long axis (m)

m_S mass of the shank (kg)

m_F mass of the foot (kg)

k_S radius of gyration of the shank about the transverse axis of the knee joint as a function shank length.

k_F radius of gyration of the foot about the transverse axis of the ankle joint as a function of foot length

The mass moment of inertia of the resistance pad about an axis through the center of the resistance pad parallel to the transverse axis of the knee joint is negligible in comparison to RJM_k. I_R is can then be calculated using the parallel axis theorem to yield

$$I_R = m_R L_D^2 \tag{6.8}$$

Electromyography

A BioSys EMG recording system was used in this study to determine the electrical activity of the knee extensors and flexors during voluntary concentric, eccentric and isometric contractions of the knee extensors. In the BioSys EMG recording system surface electrodes are attached to a small battery powered differential preamplifiers which are optically coupled to the main amplifier. The preamplifiers have an input impedance of 44 Mohms and common mode rejection ratio greater than 85dB (95dB at 60Hz). The preamplifiers amplified the raw signal 100 times.

Neuromuscular Electrical Stimulator

Neuromuscular electric stimulators (NES) are commonly used by researchers and clinicians to produce involuntary muscle contractions of the knee extensors (Brown et al. 1996, Morrissey 1988). NES's have been shown to be capable of consistently evoking contractions within the same muscle mass (Brown et al. 1996)

A two channel, constant current stimulator (Medtronic Select) was used in this study to produce involuntary contractions of the knee extensor muscles. The stimulator was capable of delivering rectangular wave pulses of 300 usec in duration at stimulation rates varying from 1 to 80 Hz. The current amplitude of the rectangular wave pulse could be varied from 0-100 mA.

Visual Analog Scale

The visual analogue scale (VAS) is a measurement tool which is commonly used to assess pain intensity and perceived functional ability. The VAS consists of a straight line normally 10 centimeters long. At each end of the line are descriptive phrases relating to the extremes of the measurement. VAS's have been shown to be a valid and sensitive measure of pain intensity which can be treated as ratio data for the purposes of statistical analysis (Jensen and Karoly 1992).

Procedure - DOMS and EMG Design

Only the dominant leg of each subject was tested. The dominant leg was defined as the one the subject would use for kicking a ball (McLean and Tumilty 1993). Body mass (kg) was measured using a standard medical balance scale (Continental). Shank length (L_S) was measured between the lateral malleolus of the ankle and the lateral femoral condyl of the knee. Foot length (L_F) was measured as the distance from posterior surface of the calcaneus to the first distal phalanx.

DOMS Design

All subjects in the DOMS section of the study were given uniform instructions regarding the evaluation procedure prior to initiation of testing. All subjects performed two testing sessions (test 1 and test 2) separated by approximately 24 hours (23-26 hours range). During each testing session the subject performed two dynamometer tests, the maximum voluntary isometric contraction (MVIC) test followed by an isovelocity strength test. At the beginning of each testing session the subject performed a 5 minute warm-up on a Monarch cycle ergometer (50-70 rpm, braking resistance less than 10 N).

After completing the isovelocity test in test 1 subjects in the NMES group performed the NMES exercise session. Except for the NMES exercise session all other procedures and measurements were identical between the control and NMES group.

A few minutes before starting each testing session the subject completed three visual analog scales (VAS). The first VAS (VAS-R) required the subjects to assess the pain intensity in the quadriceps muscle group of their dominant leg while seated in a chair. The second VAS (VAS-S) required the subjects to assess the pain intensity in the quadriceps muscle group of their dominant leg during the descent phase of a slow squat from a standing position to 90° of knee flexion. The third VAS (VAS-F) required the subjects to assess how the pain in the quadriceps muscle group of their dominant leg would impair their function during normal athletic activities.

DOMS Testing Schedule

NMES Group	Control Group
Test 1	**Test 1**
A. MVIC Test	A. MVIC Test
B. Isovelocity Strength Test	B. Isovelocity Strength Test
Neuromuscular Electrical Stimulation of knee extensors	
Test 2	**Test 2**
Return 24 hours after Test A	Return 24 hours after Test A
Repeat tests A and B	Repeat tests A and B

EMG Design

All subjects in the EMG design section of the study were given uniform instructions regarding the evaluation procedure and equipment prior to initiation of testing. After performing a 5-minute warm-up on a Monarch cycle ergometer (50-70 rpm, light resistance) subjects performed two dynamometer tests, a MVIC test followed by an isovelocity strength test. During each dynamometer test the EMG signals from the vastus lateralis, vastus medialis, rectus femoris and biceps femoris muscle groups were recorded.

EMG Electrode Placement

All subject in the EMG group and 3 subjects in the NMES group had bipolar surface EMG electrodes (18 mm diameter, 3M Red Dot Ag/AgCl) attached to their dominant leg prior to the warm-up. Subjects in the NMES group kept the same electrodes attached to their leg until both test 1 and 2 were completed (see Appendix D for EMG results for NMES group). The electrodes were placed over the muscle bellies of the vastus lateralis, vastus medialis, rectus femoris and biceps femoris muscle groups with an inter-electrode distance of approximately 2 cm. The electrodes were oriented in a proximal-distal configuration parallel to the muscle fibers as

predicted by normal human anatomy. The ground electrode for each set of active electrodes was placed approximately 4 cm laterally of the midpoint between the two active electrodes. The position of each electrode was marked with a pen to insure that any electrode could be replaced in the proper position if accidentally removed.

Each of the set of electrodes was attached to one of four preamplifiers. The preamplifiers were secured to a waist strap around the subject. To reduce the risk of motion artifact, the wires leading from the preamplifier to the electrodes were looped and taped to the subjects leg and each electrode was further secured with adhesive tape.

Since the resistance of the skin is not a critical factor when using amplifiers with high input impedance (i.e.>10Mohms), no preparation to reduce skin resistance was required (Basmajian and Deluca 1985, Winter 1990). However, when necessary the attachment area for the electrodes was shaved and cleaned with rubbing alcohol to provided better electrode contact and adhesion.

The EMG signals from the vastus lateralis, vastus medialis, rectus femoris and biceps femoris muscle groups were band passed filtered (30 to 1000 Hz) in the main amplifier using a first order RC filter (roll-off of 6 dB per octave). The raw EMG signal was amplified 500-2000 times. This gain remained constant until all tests were completed for the subject

Positioning and Alignment on Dynamometer

Subjects were positioned on the dynamometer in the sitting position, leaning against the backrest with their non-dominant leg hanging over the edge of the seat and their shoes removed (Appendix A). The backrest was inclined 15° from vertical forming an angle of 100° to the seat pan. Subjects were stabilized on the dynamometer by straps secured around the thigh of their dominant leg and their waist. Subjects were instructed to firmly grasp the dynamometer seat and keep their back firmly against the backrest during testing to provide additional stabilization.

The subject's leg was securely fastened to the pad on the dynamometer's actuator arm using velcro straps at a comfortable position approximately 5 cm proximal to the medial malleolus. The axis of rotation of the dynamometer's actuator arm was visually aligned with the lateral femoral condyle of the dominant leg when the knee was flexed at 45° (full knee extension = 0°). The moment arm of the actuator arm (L_D) was recorded for each subject. The subjects remained secured on the dynamometer until all dynamometer tests were completed for that testing session. The seat and actuator arm position were kept constant for the between test 1 and 2 for each subjects.

Encouragement

During both the MVIC and isovelocity strength test all subjects were given consistent enthusiastic verbal encouragement.

MVIC Test

The leg was positioned at 45° of knee flexion and the force was recorded from the dynamometer's force transducer once a consistent value was maintained as the subject relaxed. The subject's leg was then positioned at 30° of knee flexion. Three sub-maximal contractions of the knee flexors were completed by the subject for familiarization purposes. After a 1 minute rest the subject was required to perform one MVIC of the knee flexors for a 5 seconds duration. The leg was then position at 70° of knee flexion and three sub-maximal contractions of the knee extensors were completed by the subject. After a 1 minute rest the subject was required to perform two MVICs of the knee extensors. Each MVIC of the knee extensors was for a 5 second duration and each repetition was separated by 3 minutes. A 5 minute rest was given between the MVIC and isovelocity strength test.

Isovelocity Strength Test

The start and stop angles on the dynamometer were adjusted to 5° and 95° of knee flexion respectively. The strength test was performed at the following speeds, 50, 100, 150, 200 and 250°/s. At each testing speed 3 maximal effort concentric and eccentric contractions were performed. A 10 second pause was provided between successive concentric and eccentric contractions at each velocity and a 2 minute rest was given between speeds. The actuator arm movement was not initiated until the subject produced at least 10% of the peak RJM_K produced during the MVIC test. At each testing speed the subject performed 3-6 sub-maximal concentric and eccentric repetitions for familiarization purposes. Subjects were not allowed to view the dynamometer screen during testing.

NMES

Subjects in the NMES group received NMES of the knee extensors 5 minutes after the completion of the isovelocity strength test in test 1.

Four thin rubber electrodes (10 cm by 3 cm) were secured to the quadriceps using elastic pro-wrap. Before the electrodes were secured, a thin coat of electrolyte gel was applied to the surface of the electrodes to increase conductance between the electrodes and the skin and provided a uniform current distribution. The two positive electrodes were placed 2/3 of the distance between the anterior superior iliac spine and the superior pole of the patella. The two negative electrodes were placed approximately 4 cm proximal to the superior pole of the patella. One set of positive and negative electrodes was placed medially on the quadriceps and the other positive and negative set was placed laterally. This electrode configuration ensured that fibers from the vastus lateralis, vastus medialis and rectus femoris muscle groups would be recruited during the NMES.

The subject's leg was positioned at 70° of knee flexion. The stimulation frequency was set at 30 Hz. During a 1 minute familiarization period the amplitude of the stimulus pulse amplitude was slowly increased and the electrically evoked RJM_K

in the extensor direction was measured by the dynamometer. The pulse amplitude was increased until the electrically evoked RJM_k in the extensor direction was equal to 15% of the average RJM_k produced during the MVIC test of the knee extensors. This pulse amplitude and the 30 Hz frequency setting were then used for the NMES exercise session ($NMES_{ecc}$). Identical settings were used on both channels of the NES unit.

NMES Exercise Session

The start and stop angles on the dynamometer were adjusted to 15° and 95° of knee flexion respectively. The passive mode on the dynamometer was set at 100°/s for knee flexion and 15°/s for knee extension. Each subject performed 20 knee flexion/extension repetitions and were instructed to relax completely during all movements. A 10 second pause was provided between successive flexion and extension movements.

Approximately 1 second before the start of each passively imposed knee flexion movement $NMES_{ecc}$ was applied to the knee extensors. The $NMES_{ecc}$ continued until the passive knee flexion movement was completed for that repetition. The subject's leg was then passively extended to the 15° starting position. This NMES exercise session resulted in an involuntary (electrically induced) eccentric contraction of the knee extensors.

Data Collection, Reduction and Analysis

NMES and Control Group

The analog signals from the dynamometer's force transducer, tachometer and potentiometer were digitized and collected during each of the MVIC and isovelocity strength test. The signals were digitized at 100 Hz using a 12 bit A/D converter and collected on-line using a 486 IBM computer. All raw data was then exported to a 486 IMB computer for further processing. Post-processing was performed using Quattro Pro 6.0 and Isomap Dynamometry Software (Isodyne Inc., Winnipeg, Manitoba).

The raw data was converted to dynamometer moment (Nm), angular position (°) and angular velocity (°/s). These data files were exported to Quattro Pro 6.0 for the calculation of RJM_k.

Calculation of RJM_k

RJM_k was calculated for all eccentric, concentric and isometric contraction using equation 6.6. The values of α_L, $(m_{SF}\ L_{cm} + m_R\ L_D)$, I_{SF} and I_R from equation 6.6 were estimated as follows:

1. Angular acceleration (α_L) was derived by numerically differentiating the unfiltered angular velocity waveform,
2. $(m_{SF}\ L_{cm} + m_R\ L_D)$ was estimated from the force (F_{45}) recorded as the subject relaxed with their the leg positioned at 45° of knee flexion during the MVIC test.

$$(m_{SF}\ L_{cm+}\ m_R\ L_D) = \frac{F_{45}}{\text{Cos}45°} \tag{6.9}$$

3. The radius of gyration as a proportion of segment length about the distal axis of the segment were provided by Dempster (1955) for the shank (r_S) and foot (r_S). The mass of the shank (m_S) and foot (m_F) were estimated using regression equations based on body mass provided by Chandler et al (1975). I_{SF} was then estimated using equation (6.7),

4. I_R was estimated using equation (6.8) with m_R = 1 kg.

To remove flatline data from the records an angular acceleration threshold was used to demarcate the start and end of eccentric and concentric repetition. The starting point was marked when α_L initial went above 200°/s^2 . This occurred within the first 10 ms of the movement. The end of the contraction was marked when $\alpha_L >$ -200°/s^2 during the final 20ms of the movement. Analysis of data was restricted to the region between the start and end of the contraction as defined by the angular acceleration threshold.

Analysis of RJM_k

All RJM_k were body massed normalized (RJM_k^{BN}) for comparison purposes. The peak RJM_k^{BN} (average of peak RJM_k^{BN} from 3 repetitions), average RJM_k^{BN} (average of the average RJM_k^{BN} from 3 repetitions) and angle of occurrence of peak RJM_k^{BN} (average of angle of occurrence at peak RJM_k^{BN} from 3 repetitions) were determined for all concentric and eccentric contractions at all speeds using Quattro Pro 6.0. In addition, the peak isometric RJM_k^{BN} (average of peak RJM_k^{BN} from 2 repetitions) was also determined for all isometric contractions of the knee extensors.

Strength Maps

Two strength maps (test 1 and test 2) were generated for each subject using ISOMAP Software. The 15 eccentric and 15 concentric RJM_k^{BN} /angle/angular velocity data calculated from a subject's isovelocity strength test were used to generate an individual strength map. ISOMAP uses a bicubic interpolating spline to

fit the RJM_k^{BN} /angle/angular velocity curves to a 50 X 50 velocity/angle matrix. This results in the three-dimensional RJM_k^{BN} /angle/angular velocity relationship being expressed in the form of a two-dimensional relief-type map, called a strength map.

The x-axes (angular velocity) ranged from +250°/s to -250°/s with the negative velocities representing eccentric contractions and positive velocities representing concentric contractions. The y-axes (knee joint angle) ranged from 5 to 95°. Each color level on the z-axes (RJM_k^{BN}) represents a ten percent increment relative to 98% of the maximum RJM_k^{BN} on the maps. The isometric RJM_k^{BN} from the MVIC test were not incorporated into the strength maps since they only represent RJM_k^{BN} at one angle.

The area under the RJM_k^{BN} /angle/angular velocity map was calculated for each subject's strength maps (volume in Joules) from test 1 and test 2. For each testing session, each groups individual strength maps were averaged together to generate an average strength map for the testing session. This resulted in an average strength map being generated both test 1 and test 2 for both the NMES and control groups. Difference maps were then generated for each group by subtracting the average strength map from test 2 from the average strength map from test 1. The difference maps were generated to allow for regional event detection analysis of the RJM_k^{BN} data in terms of angle and angular velocity.

Confidence maps were generated by performing 2500 velocity and angle matched 2-tailed independent t-tests between the average strength maps from test 1 and test 2. Confidence maps were generated for both the control and NMES groups and were used to show which areas on the difference maps were statistically different. Each velocity and angle matched 1 X 1 grid was accepted as being significantly different only if it was surrounded by at least 8 neighboring and contiguous grid squares which that were also identified as being significantly different. Since the likelihood of have 6 contiguous Type I errors is less than 0.006, errors resulting from

multiple comparison testing is highly unlikely and Bonferroni correction of the t-test values is unnecessary.

EMG Group

The analog signals from the dynamometer's force transducer, tachometer, and potentiometer the vastus lateralis, vastus medialis, rectus femoris and biceps femoris muscle groups were digitized and collected during the MVIC and isovelocity strength tests. Only maximum voluntary contractions were recorded. Every recording was 10 seconds in duration, starting 3-5 seconds before the beginning of each contraction, with at least 3 seconds of recording after the completion of the repetition. All signals were digitized at 500 Hz using a 8 channel, 12 bit A/D converter and collected on-line using a 486 IBM computer. After each repetition the record was viewed using a customized program (Capture, School of Medical Rehabilitation) to examine the integrity of the data. No repeated trials were necessary for any of the tests. A section of sample recording for a concentric contraction at 100°/s is displayed in Figure 4.

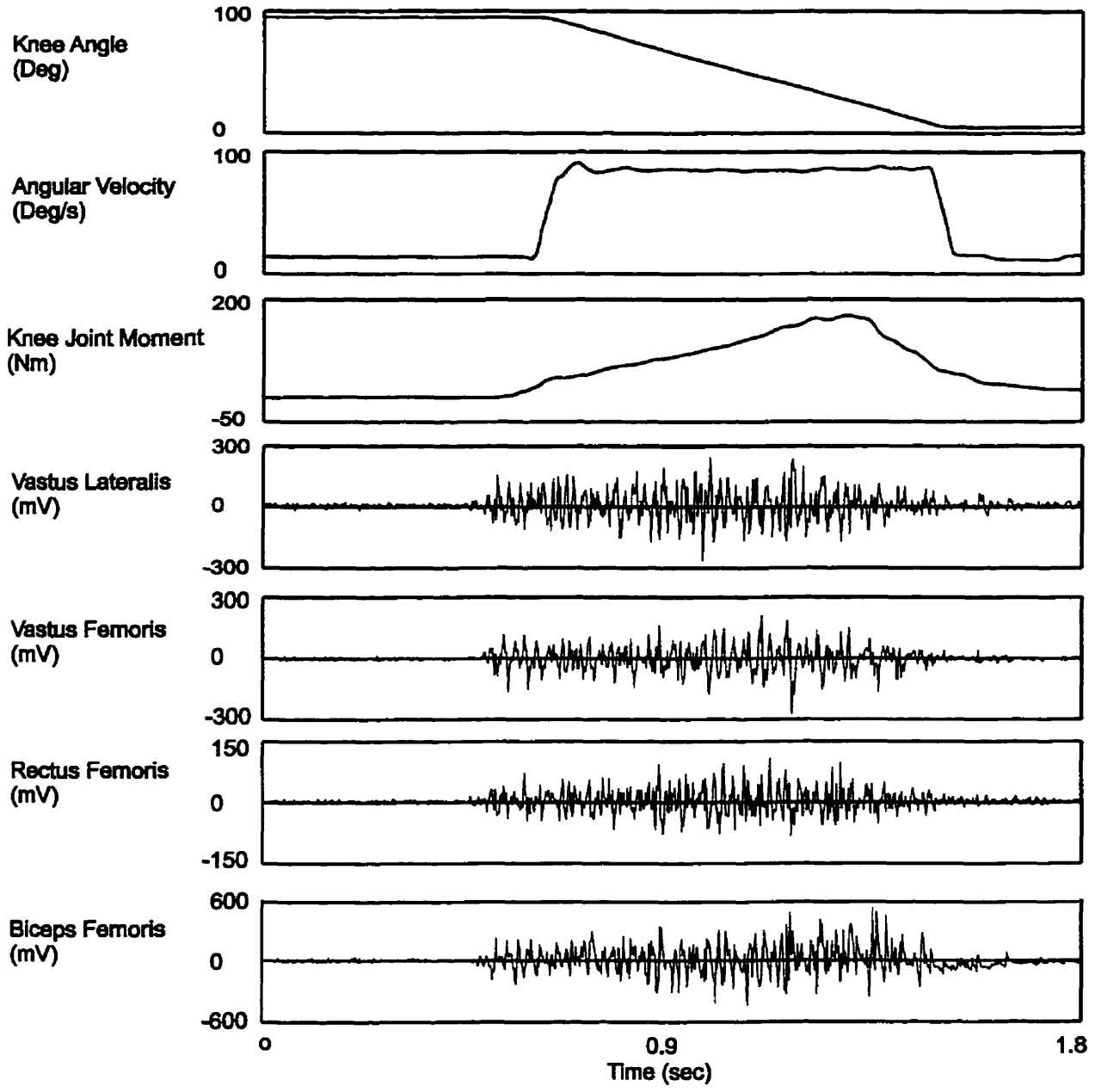

Figure 4. Example recording taken during a concentric knee extension at 100°/s. Knee angle, angular velocity and knee joint moment signals were obtained from the dynamometer. Raw EMG signals were band passed filtered (30 to 1000 Hz, -3db) and amplified (500-2000 times). All signals were sampled at 500 Hz.

RJM_k was then calculated for all eccentric, concentric and isometric contraction using the identical method discussed above for the NMES and control groups (see Calculation of RJM_k). All RJM_k were body massed normalized (RJM_k^{BN}) for comparison purposes.

Each of the EMG recordings were displayed separately on a VGA monitor using a customized program (Chart, School of Medical Rehabilitation 1994) and checked for unwanted signals. Although all precautions were taken to prevent unwanted signal, approximately eighty percent of the EMG recordings from the biceps femoris contained large motion artifacts throughout the records. The hamstring EMG was not used for statistical analysis. These motion artifacts resulted from the electrodes rubbing on the dynamometer seat during the movement. There were negligible motion artifacts in the EMG signals from the vastus lateralis, vastus medialis and rectus femoris in the range of motion from 20° and 80° of knee extension. There were some minimal motion artifacts in the first and last 15° of motions in approximately 20% of the records. However, these artifacts did not result in any significant changes in the EMG signal averaged across all subjects. All EMG records from the vastus lateralis, vastus medialis and rectus femoris were used for further analysis. There were negligible motion artifacts in the EMG signals recorded during the isometric contractions and all records were used for further analysis.

Post-processing was performed using Quattro Pro 6.0 and a customized program (Batch-analysis, School of Medical Rehabilitation). Using the batch-analysis program, all EMG records were full wave rectified and passed through a 2^{nd} order recursive Butterworth digital filter with a low pass set frequency of 15 Hz. This provided a linear envelope which retained temporal and amplitude information about the raw EMG signal.

Normalization of EMG signal

As a result of variations in signal gain, electrode placement, subcutaneous fat levels, muscle geometry and various other variables between subjects, EMG signals which are not normalized cannot be compared between muscles and between individuals (Basmajian and Deluca 1985, Duchene and Goubel 1993, Soderberg and Cook 1984, Winter 1990). Comparison can by made by normalizing the EMG signals with respect to the EMG signal recorded during a maximum voluntary isometric contraction (Basmajian and Deluca 1985, Duchene and Goubel 1993, Winter 1990).

For each MVIC of the knee extensors the start and stop times of a one second window were determined when the isometric RJM_k^{BN} was maximal and relatively constant. The average magnitude of the EMG linear envelope (IEMG) was calculated during this one second window for the vastus lateralis, vastus medialis and rectus femoris muscles for each subject. For each subject the EMG linear envelopes for concentric and eccentric contractions for each muscle group were normalized to the IEMG calculated for the subject corresponding to the muscle group.

The average magnitude of the normalized EMG linear envelope was calculated between 20° and 80° of knee flexion for each concentric and eccentric repetition. AEMG was calculated as the average of the average magnitude of the normalized EMG linear envelope from the three repetitions. AEMG provided an overall indicator of the magnitude of the electrical activity within the muscle during the contraction. Average RJM_k^{BN} (average of the average RJM_k^{BN} between 20° and 80° from three repetitions) was also calculated for each subject.

The window between 20° and 80° of knee extension was used because 1) the angular velocity is relatively constant (acceleration between $\pm 300°/s^2$) within this window, 2) most motion artifact occurs at the beginning and ends of the movement (Winter 1990), 3) Westing et al. (1991) suggested that possible artifacts in the surface EMG signal resulting from movement of the muscle under the skin would be mirrored within this window across velocity-matched eccentric and concentric tests, and 4) the

rate of rise of the EMG linear envelope was consistently observed to reached a general plateau by 20° of knee flexion for eccentric contractions and by 80° of knee flexion for concentric contractions at all angular velocities.

EMG and Strength Maps

For each subject, strength maps were generated from the RJM_k^{BN} data collected during the isovelocity strength tests using the ISOMAP software. The individual strength maps were then averaged together to yield an average strength map for the EMG group.

The ISOMAP software was used to generate EMG maps from the 15 eccentric and 15 concentric AEMG/angle/angular velocity curves recorded during a subject's isovelocity strength test. The EMG maps generated for the vastus lateralis, vastus medialis and rectus femoris muscles groups were averaged together to produce an average EMG map for each subject. The individual average EMG maps were averaged together to produce an average EMG map for the EMG group. An example EMG map for the vastus lateralis is shown in Figure 5. A RJM_k^{BN} / EMG map was also generated by dividing the average strength map for the EMG group by the average EMG rnap.

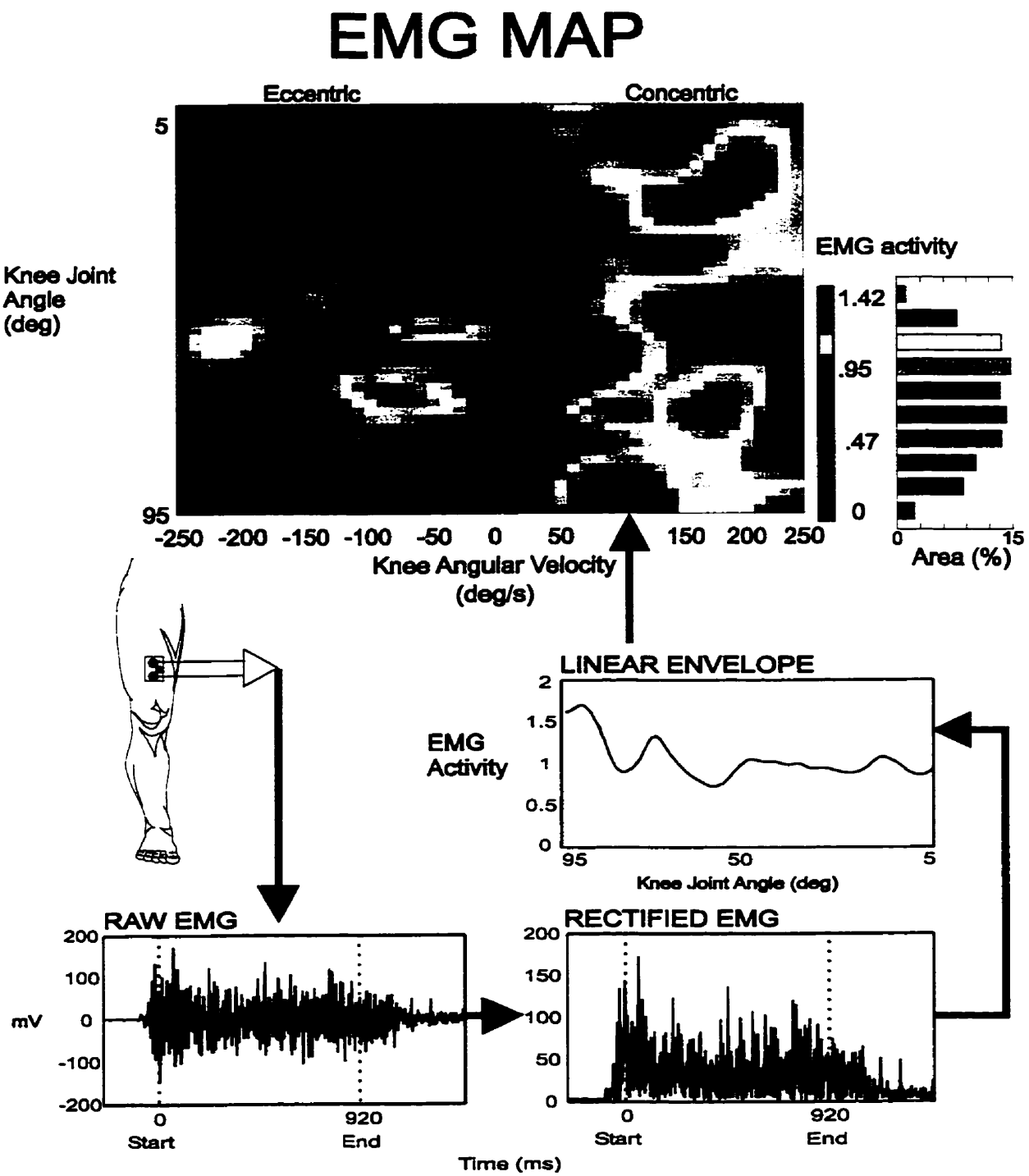

Figure 5. EMG map for the vastus lateralis. The x-axes represents knee angular velocity (°/s). The y-axes represent knee joint angle (°). Each color level on the z-axes represents a ten percent increment relative to 98% of the maximum EMG magnitude. The raw EMG is first rectified and low passed filtered at 30 Hz. The EMG is normalized to IEMG for the vastus lateralis.

Neuromuscular Model

The force generated across a muscle during isometric contractions was simulated in Quattro Pro 6.0 using the neuromuscular model developed in this study. A maximal voluntary contraction was simulated based on recruitment and firing rates observed in humans (Bigland-Ritchie et al. 1983, De Luca et al.1982, Freund et al. 1975, Kukulka and Clamann 1981). The recruitment and rate coding strategy used for the maximal voluntary contraction simulation is displayed in Figure 3. The average force generated during the steady state portion of the contraction (t >200ms) was recorded. The maximum force generation of a motor unit was determined by increasing the stimulus rate to the motor unit in 1 Hz increments until there was a force plateau or 100 Hz stimulation frequency was obtained. This was defined as tetanus for the motor unit. The maximum theoretical force output of the model was determined as the sum of the force generated by all motor units when stimulated at a rate to achieve tetanus.

Model of Load Sharing

The neuromuscular model was used to illustrate how changes in recruitment and rate coding strategies affect the loading of individual motor units. Since this model was used for illustration, a systematic evaluation of all parameters effecting load sharing was not performed. Two neural strategies were tested in the simulation, (narrow rate coding and broad rate coding). In the narrow rate coding strategy motor units were stimulated in a narrow range (8-16 Hz) after being recruited, whereas in the broad rate coding strategy motor units were stimulated over a large range (1-25Hz) after being recruited. Once a motor unit was recruited its stimulation rate remained constant. In both recruitment strategies motor units with smaller twitch forces were recruited earlier than motor units with larger twitch forces (Henneman and Mendell 1981). In addition, the firing rates of the smaller motor units were always larger than the firing rates of the larger motor units (Bigland-Ritchie et al. 1983, De

Luca et al.1982). Similar forms of the narrow and broad rate coding strategies have been observed in human muscles during sub-maximal voluntary contractions (Bigland-Ritchie et al. 1983, De Luca et al.1982, Kukulka and Clamann 1981, Milner-Brown et al. 1973c, Monster and Chan 1977).

For both the narrow and broad rate coding strategies the stimulation rate of individual motor units were adjusted to achieve an average force output equal to 70 percent of the steady state force achieved during the simulated maximal contraction. The peak force generated by each motor unit during the simulations was compared to the force generated by the motor unit during a simulated tetanus contraction (theoretical physiological maximum force output)

Statistical Analysis

1. Statistical analysis was performed using SYSTAT 5.0 for Windows, Quattro Pro 6.0 for Windows, and Isomap Software. The level of significance was established at an alpha level of 0.05

2. Differences in the VAS scores between pre-test, post-test and 24 hours post-test were analyzed using repeated measures analysis of variance (ANOVA). Each group (NMES and control) and type of VAS administered (VAS-S, VAS-R and VAS-F) were analyzed separately. Post-hoc analysis was performed using 1-tailed paired t-tests with Bonferonni correction.

3. The group (NMES and control) and test (test 1 and 2) effects on the peak isometric RJM_K and volume of the knee extensor map were analyzed using repeated measures two-way ANOVA. Post-hoc analysis was performed using 2-tailed paired t-tests.

4. The speed and test effects on the strength parameters peak RJM_K . average RJM_K and the angle of occurrence of peak RJM_K were analyzed by means of a series of repeated measures two-way ANOVA. Each group (NMES and control) and contraction type (concentric and eccentric) was analyzed separately. Post-hoc analysis was performed on the peak and average RJM_K using 1-tailed paired t-tests, with and without Bonferroni corrections. Post-hoc analysis was performed on the angle of occurrence of peak RJM_K using 2-tailed paired t-tests.

5. Regional differences in the knee extensor strength maps between test 1 and 2 were analyzed using confidence maps.

6. The speed and EMG location (vastus lateralis, vastus medialis and rectus femoris) effects on the AEMG were analyzed using repeated measures two-way ANOVA. Each group (NMES and control) and contraction type (concentric and eccentric) was analyzed separately. Post-hoc analysis was performed on the peak and average concentric and eccentric RJM_K using 1-tailed paired t-tests, with and without Bonferroni corrections. Post-hoc analysis was performed on the angle of occurrence of peak RJM_K using 2-tailed paired t-tests.

7.0 RESULTS

Subjects

DOMS study

A total of 12 subjects participated in the DOMS study; 6 in each of the NMES and control groups. DOMS group demographics are outlined in Table 1.

	Control	NMES
Number of subjects	6 (4 M / 2 F)	6 (2 M / 4 F)
Age (years)	28.6 ± 4.5	26.8 ± 5.5
Body Mass (kg)	70.7 ± 7.9	68.4 ± 15.7
Aerobics training bouts/week	3.5 ± 1.3	3.2 ± 0.74

Table 1. Demographics for the NMES and control groups. Mean ± SD values are shown where applicable.

No significant differences were observed between the groups for age, body mass, and self-reported frequency of aerobic training. One subject in the NMES group reported lower body weight training at a frequency greater than once every month during the previous 6 months. This subject reported 'light' lower body weight training once a week for the past year.

EMG Study

8 subjects participated in the EMG study. EMG group demographics are outlined in Table 2.

Number of subjects	8 (5 M / 3 F)
Age (years)	27.6 ± 4.6
Weight	68.9 ± 11.1
Aerobic training bouts per week	3.75 ± 0.97
Lower body weight training per week	0.5 ± 0.86

Table 2. Demographics for the EMG group. Mean ± SD values are shown where applicable.

DOMS experimental results

Pain related measures

Scores derived from the VAS from questions regarding the pain intensity at rest (VAS-R), peak pain intensity during the decent phase of a slow squat (VAS-S), as well as the effect of pain on the ability to perform functional tasks (VAS-F) for pre-test, post-test and 24 hours post-test are graphically displayed in Figure 6. There were no significant differences between pre-test, post-test and 24-hours post-test VAS scores for the control group in each of the three tests (repeated measures ANOVA). There was a significant difference between the pre-test, post-test and 24 hours post-test VAS scores for the VAS-R ($p<0.05$), VAS-S ($p<0.005$) and VAS-F ($p<0.05$) for the NMES group (repeated measures ANOVA). Bonferroni corrected 1-tailed t-tests revealed a significant increase in VAS scores post-test compared to pre-test for VAS-F ($p<0.016$). There was a significant increase in VAS scores 24 hours post-test compared to pre-test for the VAS-R ($p<0.05$), VAS-S ($p<0.05$), and VAS-F ($p<0.001$). There were no statistically significant increases in the VAS scores post-test compared to pre-test for the VAS-R and VAS-S test, however the p values for these tests ($p = 0.052$ and $p = 0.10$ respectively) suggest that the power of the study was likely to low to show these differences. One subject in the NMES group reported a higher VAS-F score of 4.11 24 hours post-test.

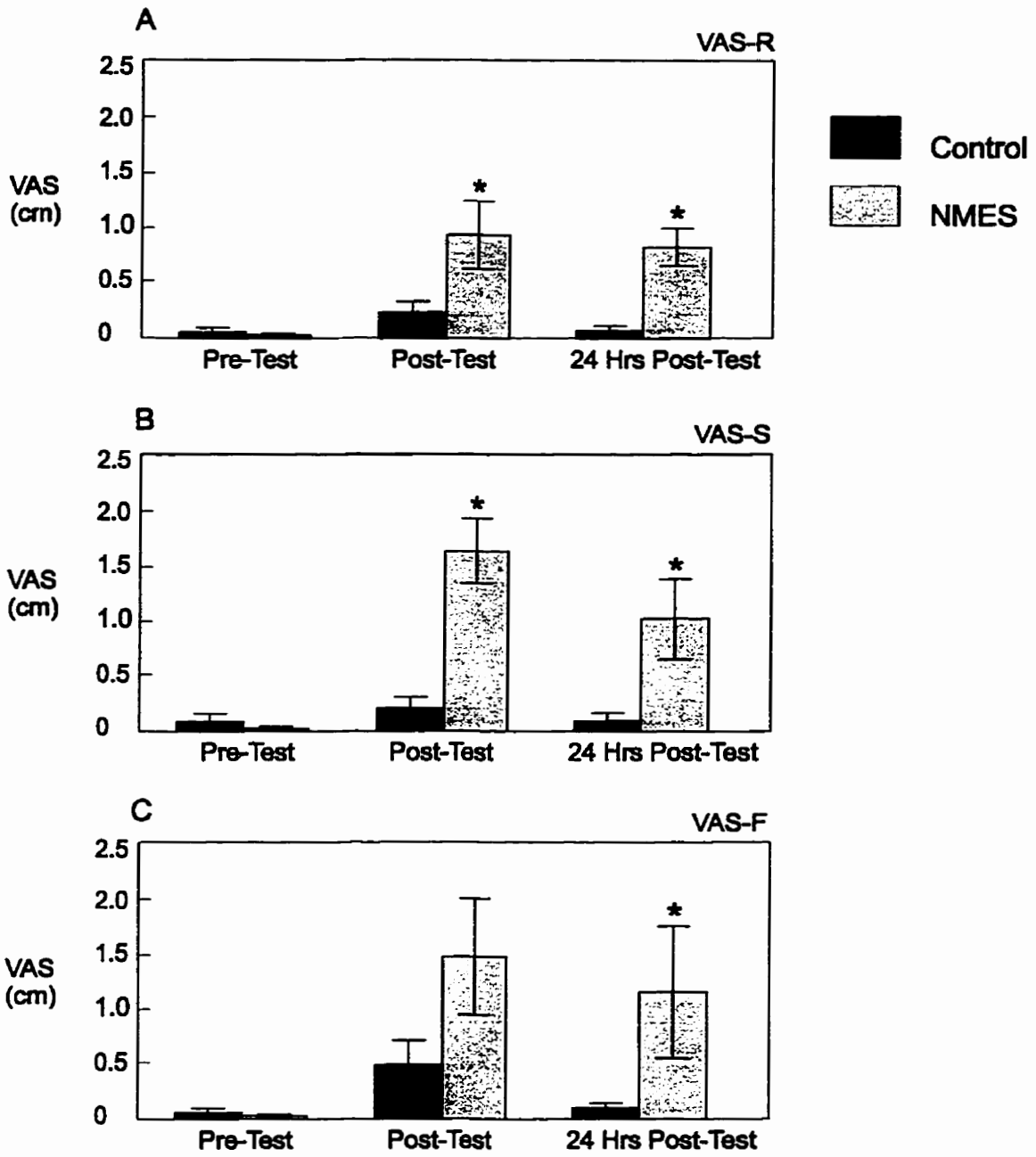

Figure 6. Scores derived from VAS pre-test, post-test and 24 hours post-test. A - pain intensity at rest (VAS-R). B - peak pain intensity during the decent phase of a slow squat (VAS-S). C - effects of pain on the ability to perform tasks (VAS-F). Mean and standard error bars are shown. *Significantly different from pre-test ($p<0.05$)

MVIC Test

Peak isometric RJM_k^{BN} for test 1 and test 2 are displayed graphically in Figure 7 for the NMES and control groups. The split-unit ANOVA table presenting the statistical analysis of the results of the peak isometric RJM_k^{BN} data is displayed in Table 3. As summarized in Table 3, there was a statistically significant decrease in the peak isometric RJM_k^{BN} in test 2 compared to test 1 ($p<0.001$). The peak isometric RJM_k^{BN} decreased 7.8% ($p < 0.04$) in the NMES group and 7.5% ($p < 0.01$) in the control group (2-tailed, paired t-tests). There were no significant differences in the peak isometric RJM_k^{BN} between the NMES and control group.

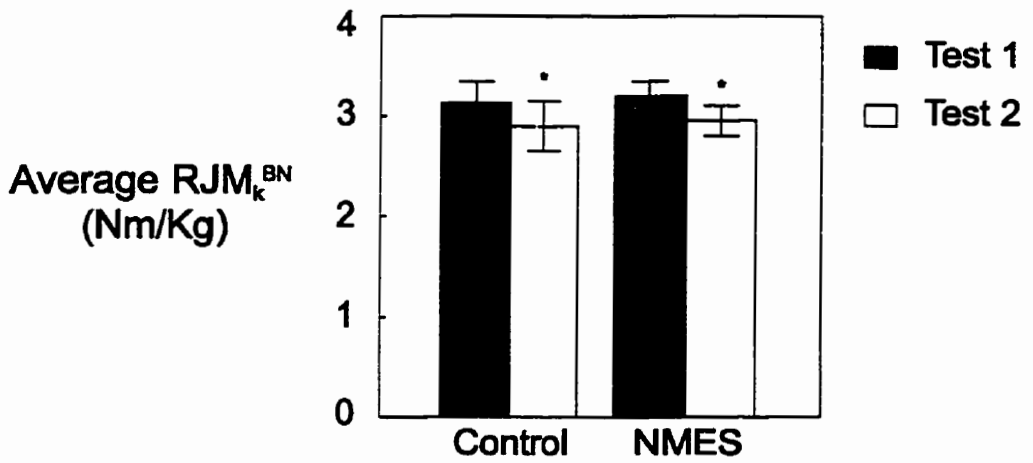

Figure 7. Peak isometric RJM_k^{BN} at 70° of knee flexion. Standard error bars are shown . *Significantly different from test 1 ($p<0.05$).

	SS	DF	MS	F	p
Between Subjects		11			
Between Groups	0.023	1	0.023	0.045	0.836
Error	5.174	10	0.517		
Within Subjects					
Between Tests	0.352	1	0.352	20.287	***0.001***
Group x Test	0.000	1	0.000	0.023	0.884
Error	0.173	10	0.017		

Table 3. Split-unit ANOVA table summarizing results for change in peak isometric RJM_k^{BN} between testing periods. Dependent - peak isometric RJM_k^{BN}, Independent - test, group

Isovelocity strength testing

The peak and average RJM_k^{BN} from isovelocity strength test 1 and 2 for the NMES and control groups are illustrated in Figure 8 and Figure 9, respectively.

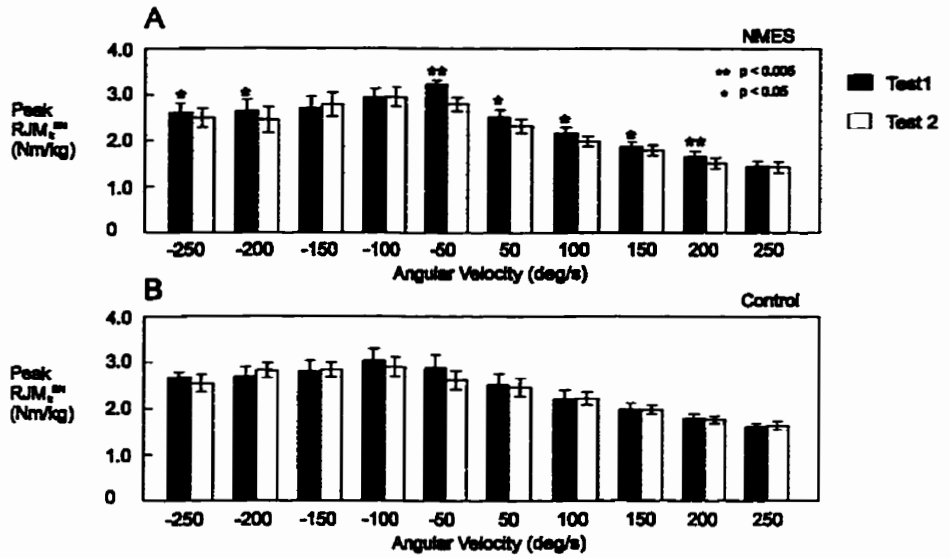

Figure 8. Peak RJM_k^{BN} / angular velocity relationships for test 1 and test 2. A - NMES group. B - Control group. p-values are results from paired t-tests at each velocity without Bonferroni correction. Standard error bars are plotted. Concentric velocities are designated as positive and eccentric velocities as negative

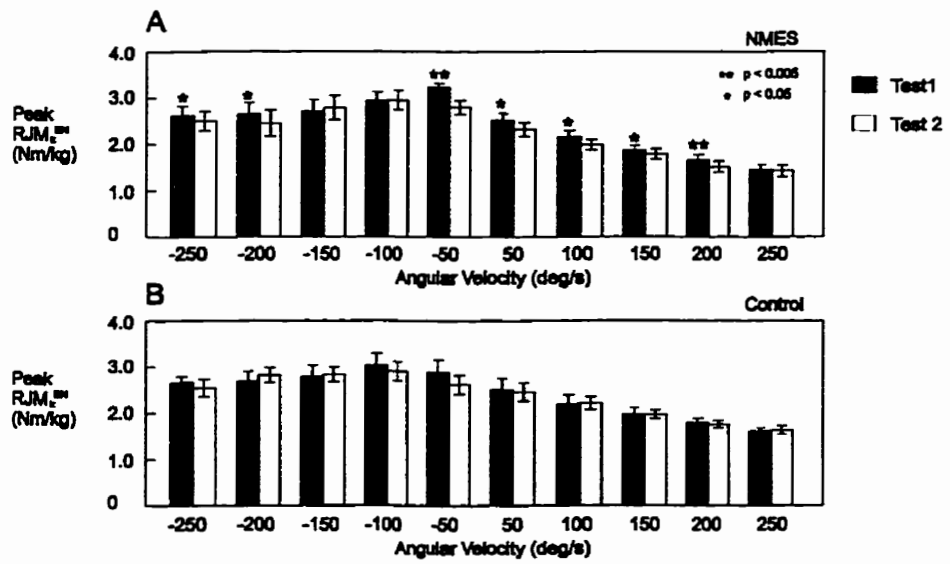

Figure 9. Average RJM_k^{BN} / angular velocity relationships for test 1 and test 2. A - NMES group. B - Control group. p-values are results from paired t-tests at each velocity without Bonferroni correction. Standard error bars are plotted. Average RJM_k^{BN} from 5° to 95° of knee flexion. Concentric velocities are designated as positive and eccentric velocities as negative

The repeated measures two-way ANOVA table presenting the statistical analysis of the results of the eccentric peak RJM_k^{BN} data from the test 1 and test 2 for the NMES group are displayed in Table 4. This repeated measures two-way ANOVA format was used in the analysis of the peak RJM_k^{BN}, average RJM_k^{BN} and angle of occurrence of peak RJM_k^{BN} . Each group (NMES and control) and contraction type (concentric and eccentric) was analyzed separately using this method.

	SS	DF	MS	F	p
Between speeds	5.679	4	0.541	0.876	0.492
Error	6.930	25	0.617		
Within Subjects					
Between Tests	0.263	1	0.263	7.867	*0.010*
Group x Test	0.479	4	0.120	3.580	*0.019*
Error	0.837	25	0.033		
Total					

Table 4. Example two-way ANOVA table summarizing results between eccentric Peak RJM_k^{BN} from the test 1 and test 2 for the NMES group

The p-values of the ANOVA results for the peak RJM_k^{BN}, average RJM_k^{BN} and angle of occurrence of the peak RJM_k^{BN} during concentric and eccentric contractions for the NMES and control group are listed in table 5

	NMES group		Control group	
	Concentric	Eccentric	Concentric	Eccentric
Peak RJM_k^{BN}				
Between tests	*0.001*	*0.010*	0.781	0.842
Test x angular velocity	0.204	*0.019*	0.961	0.851
Average RJM_k^{BN}				
Between tests	*0.001*	0.161	0.768	0.831
Test x angular velocity	0.584	*0.044*	0.970	0.835
Angle of peak RJM_k^{BN}				
Between tests	*0.010*	*0.001*	0.088	0.098
Test x angular velocity	0.447	*0.044*	0.161	0.186

Table 5. The p-value results of the two-way ANOVA for peak RJM_k^{BN}, average RJM_k^{BN} and angle of occurrence of the peak RJM_k^{BN} during concentric and eccentric contractions. Significant values ($p<0.05$) are italicized

As summarized in Table 5, the NMES group exhibited statistically significant differences between the two testing periods in peak RJM_k^{BN} and average RJM_k^{BN} for concentric contractions and in peak RJM_k^{BN} for eccentric contractions. The test by angular velocity interaction was also statistically significant for both the peak RJM_k^{BN} and average RJM_k^{BN} during eccentric contractions for the NMES group. The control group exhibited no significant differences between average RJM_k^{BN} or peak RJM_k^{BN} between the two testing periods, as well as no significant test by angular velocity interactions.

From Figure 8A and 9A it is observed that peak and average RJM_k^{BN} decrease in magnitude in 8 out of the 10 testing velocities in test 2 compared to test 1 for the NMES group. One tailed paired t-tests, with and without Bonferroni corrections, were used for post-hoc analysis of the significant angular velocity and test by angular velocity interaction effects. The NMES group displayed significantly lower peak knee moments during test 2 at angular velocities of -250°/s,-200°/s, -50°/s, 50°/s, 100°/s, 150°/s and 200°/s using t-tests without Bonferroni correction and at -50°/s and 200°/s using Bonferroni corrected t-tests. The average RJM_k^{BN} in the

NMES group was also significantly lower in second day of testing at angular velocities of -50°/s, 50°/s, 100°/s and 200°/s using uncorrected t-tests and at -50°/s, 50°/s and 100°/s using corrected t-tests.

For illustration purposes each individual subject's RJM_k^{BN} during an eccentric contraction at -50°/s from the test 1 and test 2 are shown in Figure 10. Both groups show an average decrease in peak RJM_k^{BN} in test 2 compared to test 1 at -50°/s as exhibited in Figure 9. However individuals in the control group exhibit higher variability in the change in peak RJM_k^{BN} from tests 1 to test 2. In the control group two individuals had an increase in peak RJM_k^{BN} in test 2 while four individual had a decrease in peak RJM_k^{BN} in test 2 resulting in a non-significant average decrease in peak RJM_k^{BN} of 9% from test 1 to test 2. Conversely all six individuals in the NMES group show a consistent loss in peak RJM_k^{BN} during test 2 resulting in a significant decrease in peak RJM_k^{BN} of 14% from test 1 to test 2.

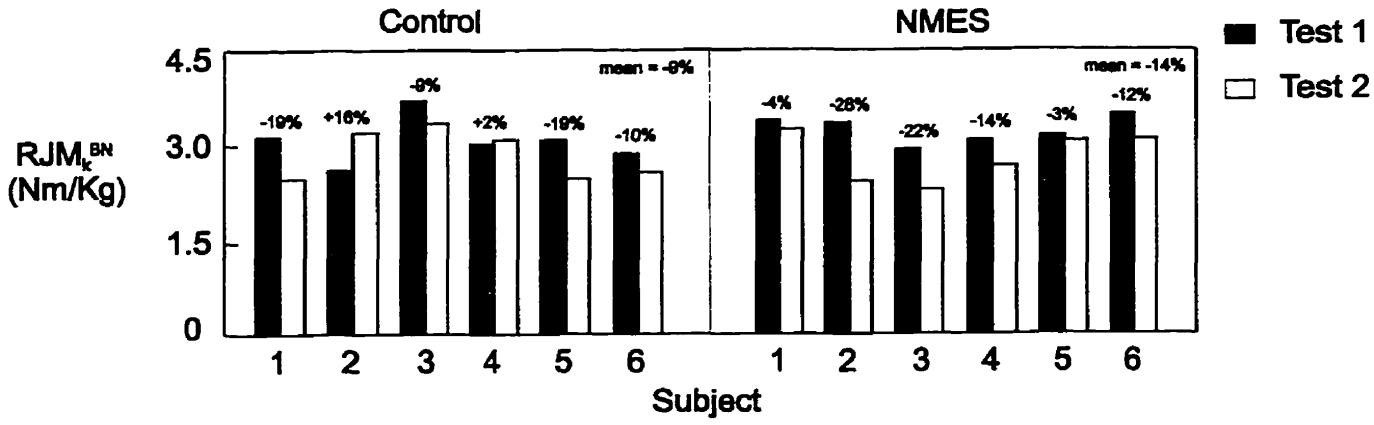

Figure 10. Peak RJM_k^{BN} during an eccentric contraction at 50°/s for individual subjects in the NMES and control groups. The percentage change in peak RJM_k^{BN} from test 1 to test 2 is shown for each subject.

The angle of occurrence of the peak RJM_k^{BN} for each angular velocity from test 1 and test 2 for the NMES and control groups is illustrated in Figure 11. The angle of occurrence is measured as knee flexion (0° = full knee extension). As summarized in Table 5, there was a significant difference in the angle of occurrence of peak RJM_k^{BN} between testing periods for the NMES group for both concentric ($p<0.010$) and eccentric ($p<0.001$) contractions. The test by angular velocity interaction was also statistically significant for eccentric contractions performed by the NMES group ($p<0.044$). The control group exhibited no significant differences between the angle of occurrence of peak RJM_k^{BN} between the two testing periods, as well as no significant test by angular velocity interactions. From Figure 11 it is observed that the angle of occurrence of peak RJM_k^{BN} increased in 9 out of the 10 testing velocities in test 2 compared to test 1 for the NMES group. Post-hoc analysis performed using two-tailed paired t-tests revealed that the increases in the average angle of occurrence of the peak RJM_k^{BN} for the NMES group during the second day of testing were statistically significant at -250°/s (63.1° to 70.1°, $p<0.037$), -200°/s (62.2° to 66.3°, $p<0.028$) and -150°/s (63.1° to 70.1°, $p<0.038$).

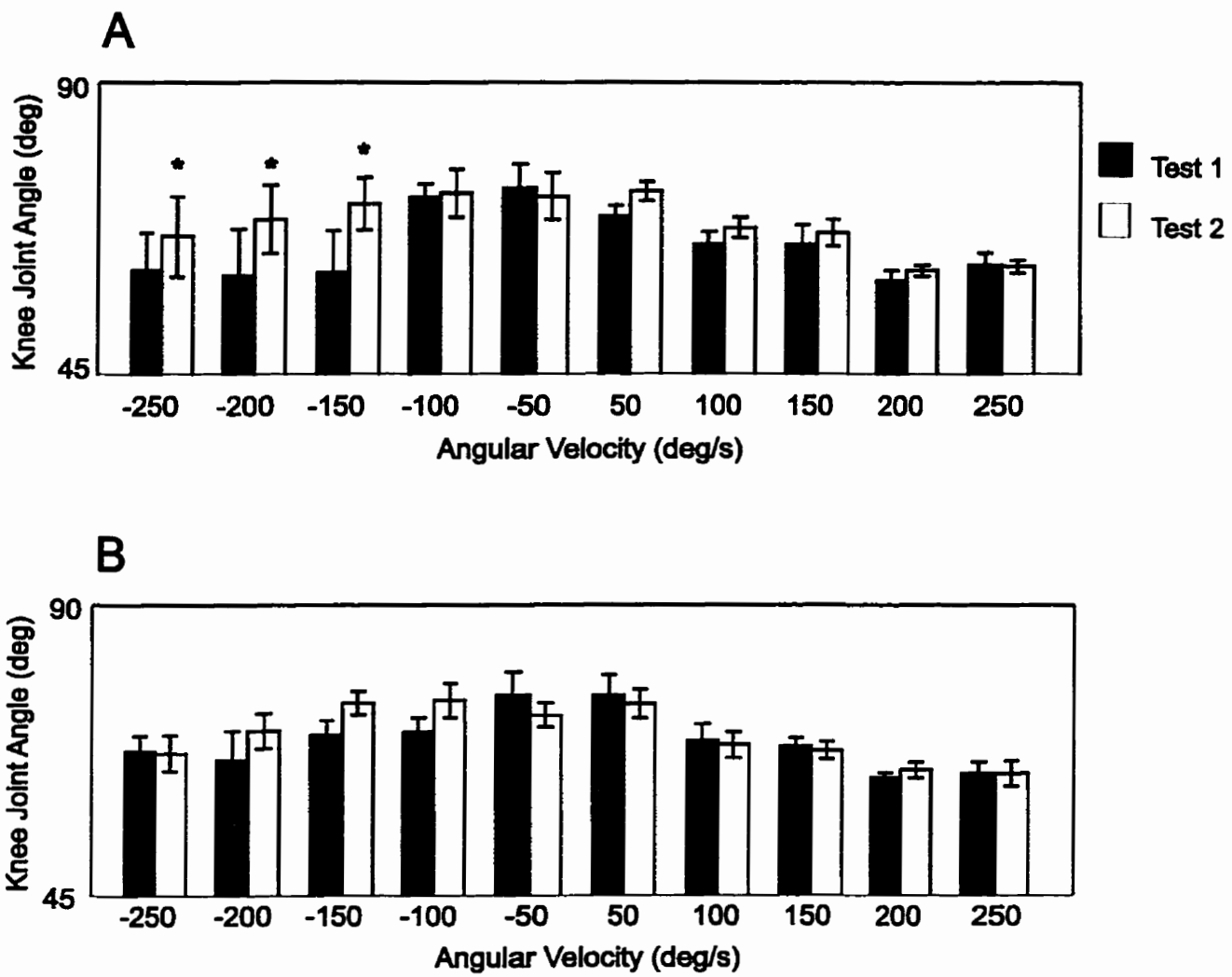

Figure 11. Angle of occurrence of peak RJM_k^{BN} for test 1 and test 2. A - NMES group. B - control group. Mean and standard error bars are plotted. *Significantly different from test 1 ($p<0.05$ - paired t-tests at each velocity without Bonferroni correction).

Strength maps

Body mass normalized strength maps were generated for each subject in the NMES and control groups for test 1 and test 2. For each testing period, the individual strength maps within a group were averaged together. The average strength maps from test 1 and test 2 for the NMES group are displayed in Figure 12. There were no obvious differences in the magnitudes of RJM_k^{BN} achieved and the shape of the map surface between the average strength maps for the control group and NMES group for test 1.

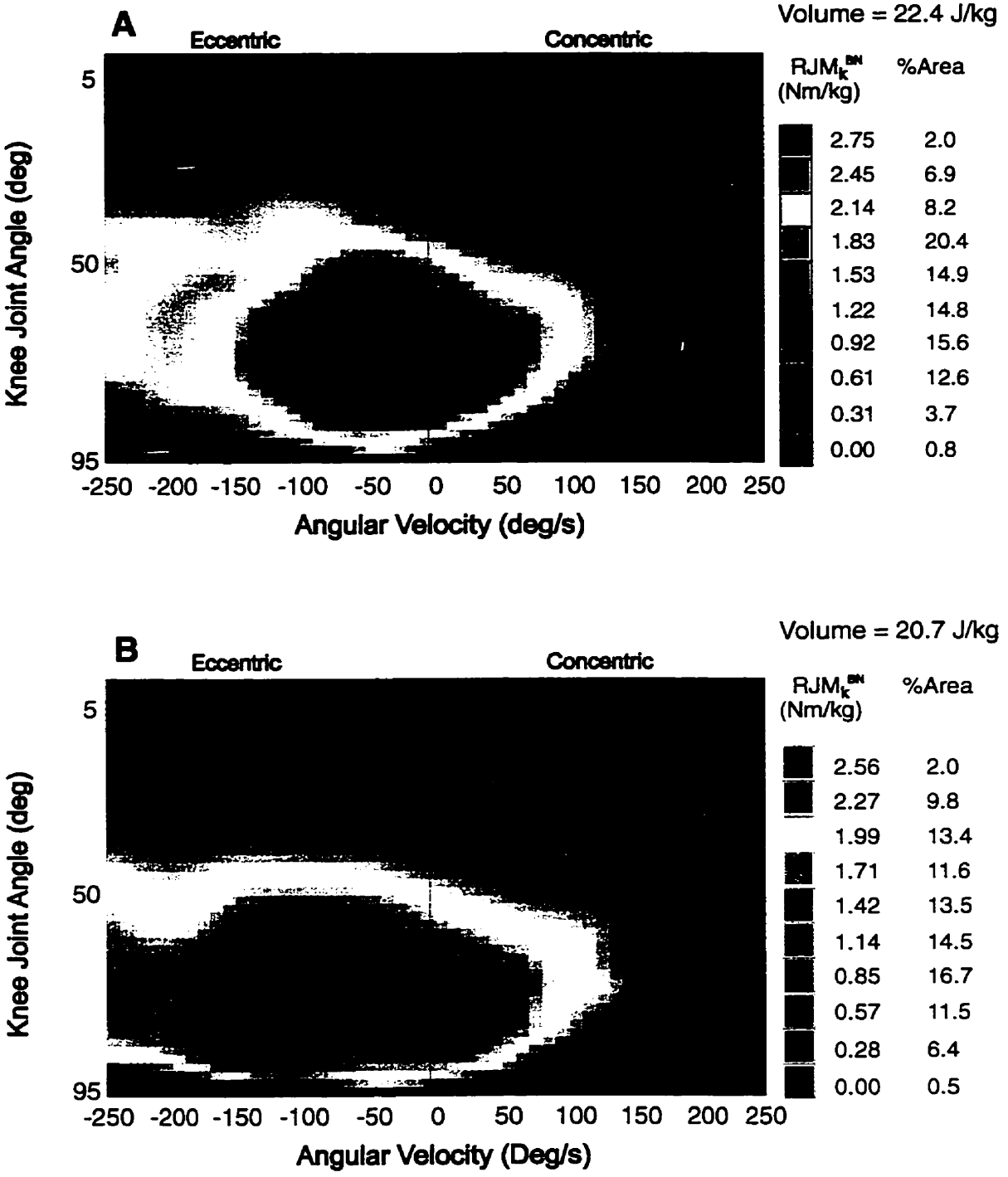

Figure 12. Average strength maps of the knee extensors for the NMES group A - test 1 B - test 2. The percentage area of the map associated with each color range of RJM_k^{BN} is shown to the right of the map. Volume represents the area under the strength map. The leg angular velocity is shown on the horizontal axes and the knee joint angle is shown on the vertical axis.

The volume of the average strength map for test 1 and test 2 for the control and NMES groups are displayed graphically in Figure 13. A repeated measures two-way ANOVA was used to compare the volumes between the two testing periods and groups. There was a significant test effect ($p<0.04$), as well as a significant group by test interaction effect ($p<0.05$). Post-hoc analysis using 2-tailed paired t-tests showed a significant 6.4% decrease ($p<0.05$) in the volume of the average strength map in test 2 for the NMES group. There was a non-significant decrease (0.91%) in volume of the average strength map for the control group.

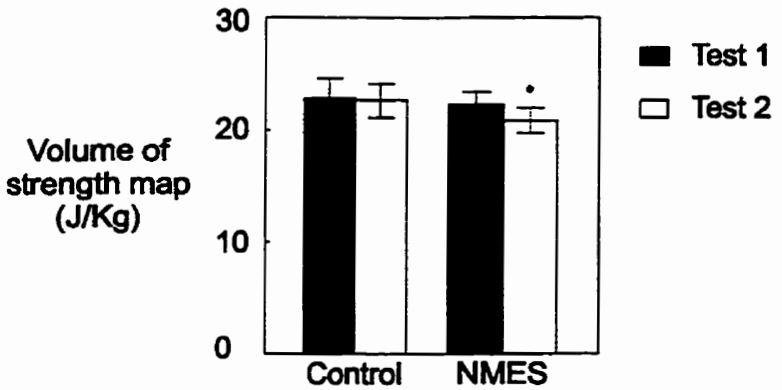

Figure 13. Volume of the average strength map. Mean and standard error bars are plotted. *Significantly different from test 1 ($p<0.05$)

For both the NMES and control groups difference maps were generated by subtracting the average strength map from test 2 from the average strength map from test 1. Confidence maps were generated to determine the statistical significance of each section of the difference maps. The difference and confidence maps from test 1 and test 2 for the NMES and control group are displayed in figure 14.

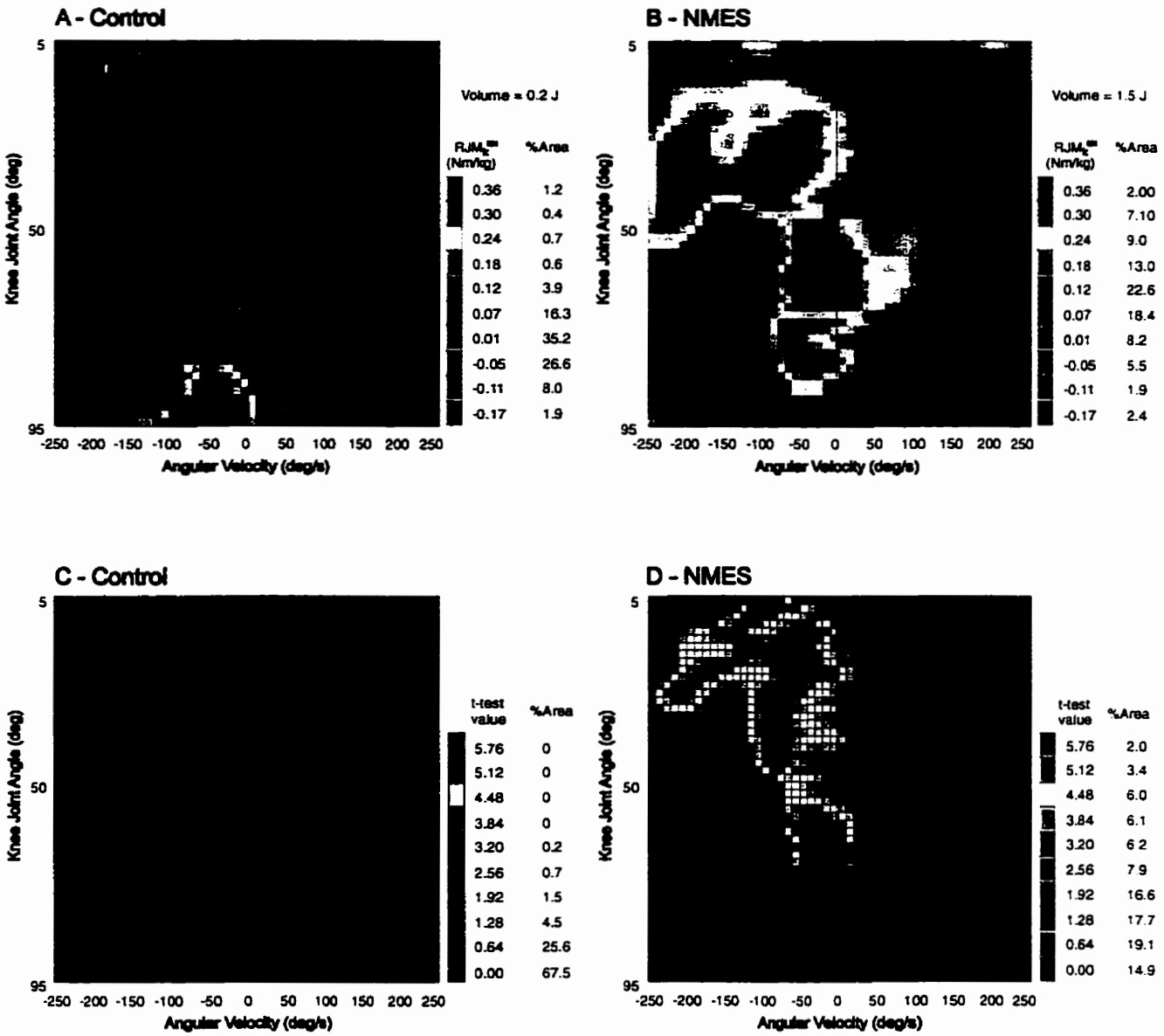

Figure 14. Differences maps derived from average strength maps (test 1 - test 2). A - Control group. B - NMES group. Confidence maps depicting the independent t-test values for each of the difference maps. C - Control group. D - NMES group. The areas highlighted by the overlay grid represent significant t-test values (t-test values > 2.28). Volume represents the total volume under the differences map.

Regional strength differences can be observed in the difference maps (Figure 14 A and B) for the NMES and control group. Regional analysis was performed using a threshold values of +0.15 Nm/kg for decreases in RJM_k^{BN} between test 1 and test 2 and -0.10 Nm/kg for increases in RJM_k^{BN} . The NMES group demonstrated a RJM_k^{BN} deficit greater than 0.15 Nm/kg over 31.7% of the map. This region of deficit was largely localized to two regions on the eccentric side of the map. One region of this deficit was in the 15-45° range of motion for angular velocities -250°/s to 0°/s. The second region of deficit was observed in the 15-90° range of motion for angular velocities -90°/s to 10°/s. The control group demonstrated a RJM_k^{BN} deficit greater than 0.15 Nm/kg over a small region (2.1%) of the map which occurred over the 82- 95° range of motion and -107°/s to -5°/s velocity range. Both the control and NMES group demonstrated an increase in RJM_k^{BN} generation during high speed eccentric contractions (-200°/s to -250°/s) over the range of motion of 50-95° corresponding to the lower left quadrant of the map. In this region, the NMES group and control group displayed an increase in RJM_k^{BN} generation greater than 0.10 Nm/kg for an area 9.4% and 4.2% of the total map respectively.

A confidence map was used to determine the statistical significance of the regional strength differences observed on the difference maps. The results from 2500 2-tailed independent t-tests are displayed in the confidence maps in Figure 14 (C and D). The small boxed areas on the confidence maps represent the area which is greater than the critical t-test value of 2.228 (df = 10, alpha level 0.05).

Using this critical value, 29.5% of the NMES difference map was found significantly different. This area was largely localized to the same region that showed a moment deficit greater than 0.15 Nm/kg in the NMES difference map. The confidence map for the control group showed a 0.4% area of significant difference in the 91- 95° range of motion and -56 to -15°/s velocity spectrum area of the map. The confidence maps found that the increases in the RJM_k^{BN} generation were non-significant.

EMG experimental results

Isovelocity strength test

AEMG / knee angular velocity relationship for the vastus lateralis, vastus medialis and rectus femoris muscles and the average RJM_K^{BN} / knee angular velocity relationship for the EMG group are graphically illustrated in Figure 15. The average isometric RJM_K^{BN} from the MVIC test was 2.85 Nm/kg ± 0.13 SE. As observed on Figure 15 the average isometric moment was significantly higher than the average RJM_K^{BN} for eccentric or concentric contractions.

A repeated measures two-way ANOVA was used to compare the AEMG between muscles and angular velocities. Analysis was performed separately for the eccentric and concentric velocities. During concentric contractions the AEMG increased with increasing velocities for each muscle ($p<0.001$). In contrast to concentric contractions, the AEMG during eccentric contractions was not observed to be velocity dependent (N.S). The AEMG showed statistically significant differences between muscles (concentric $p<0.008$, eccentric $p<0.001$). Post-hoc analysis using tukey's multiple comparison test demonstrated that the AEMG for the rectus femoris was significantly lower than the AEMG for the vastus lateralis and vastus medialis for both concentric and eccentric contractions. There were no statistically significant muscle by angular velocity interactions for either concentric or eccentric contractions (two-way ANOVA).

The AEMG was found to be greater than 1.0 during concentric contractions at 200°/s and 250°/s for the vastus lateralis and at 100°/s, 150°/s, 200°/s and 250°/s for the vastus medialis. The vastus medialis was found to have a maximum average AEMG value of 1.15 at 250°/s.

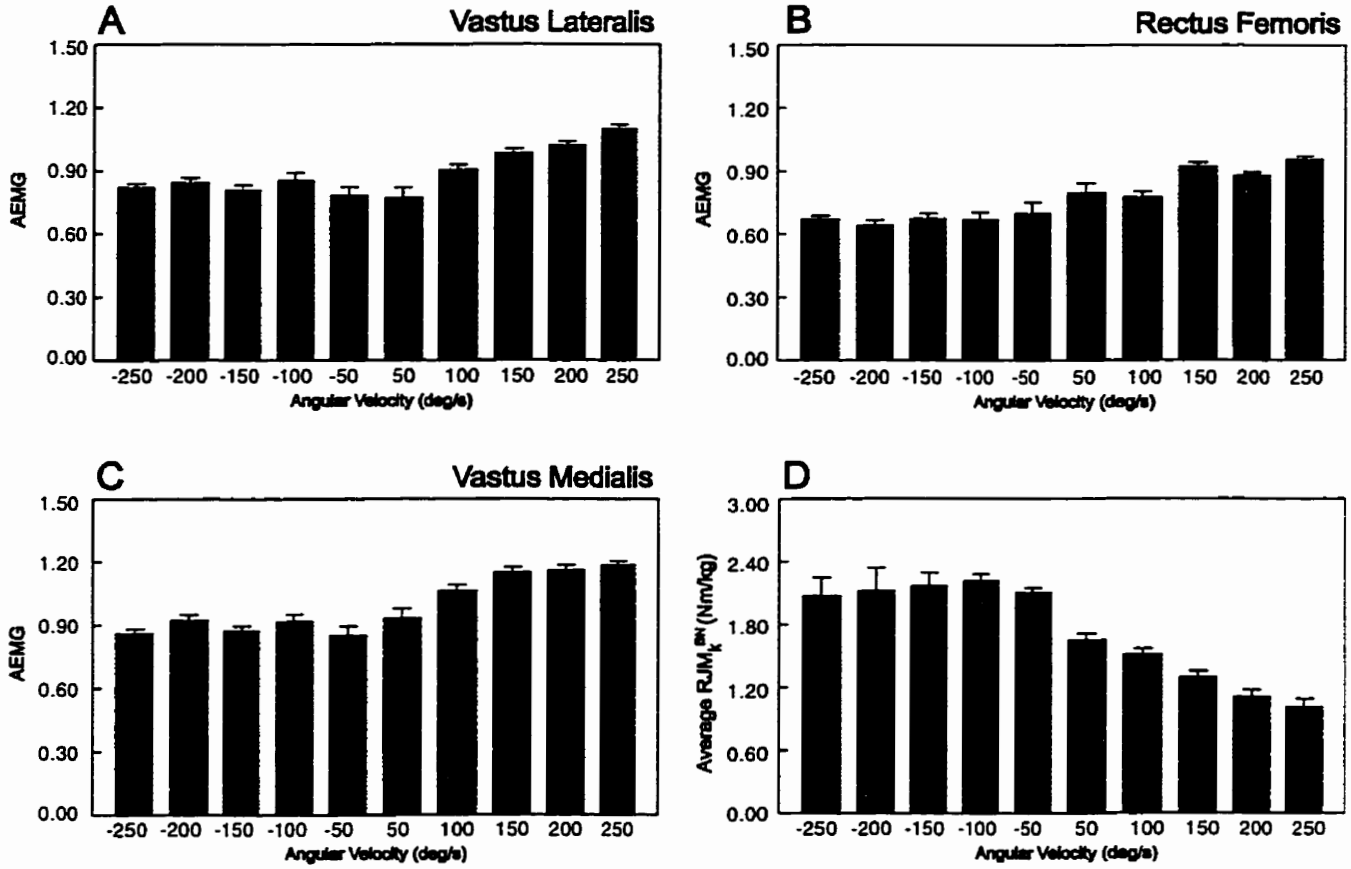

Figure 15. AEMG / angular velocity relationships for A - vastus lateralis, B - vastus medialis, and C - rectus femoris muscles. D - average RJM_k^{BN} / angular velocity relationship (D). AEMG represent the average EMG linear envelope from 20° to 80° normalized to the average EMG linear envelope during a MVIC of the knee extensors at 70°. Standard error bars are plotted.

No angular velocity effect on average RJM_k^{BN} generation for eccentric contractions was observed (N.S., one-way ANOVA). The average RJM_k^{BN} during concentric contraction decreased significantly with increasing angular velocity ($p<0.001$, one-way ANOVA).

There were obvious differences in AEMG and average RJM_k^{BN} between concentric and eccentric contractions at different speeds as illustrated in Figure 15. These difference were explored using the ratio of eccentric to concentric AEMG and average RJM_k^{BN} at matching angular speeds (Table 6). No statistically significant differences were found between eccentric and concentric AEMG at 50°/s for the three muscle groups or for the vastus lateralis at 100°/s. With the exception of the vastus lateralis ratio at 50°/s, the AEMG during eccentric contractions was lower than the AEMG during concentric contractions at matching angular speeds. There was a significant drop in the eccentric to concentric AEMG ratio with increasing angular velocities (one-way ANOVA, $p<0.01$), with the eccentric AEMG decreasing to an average of 73.3% of concentric AEMG at 250°/s. As displayed in Table 6, the average RJM_k^{BN} was significantly higher in eccentric contractions compared to concentric contractions at all angular speeds. The eccentric to concentric average RJM_k^{BN} ratio increased with increasing velocities (one-way ANOVA $p<0.001$), with the average RJM_k^{BN} during eccentrics contractions being an average of 205% larger than velocity matched concentric contractions at 250°/s.

Angular Velocity (°/s)	Eccentric magnitude / concentric magnitude			
	Vastus Lateralis	Rectus Femoris	Vastus Medialis	Average RJM_k^{BN}
50	1.04±0.09 p=0.215	0.93±0.07 p=0.063	0.89±0.09 p=0.055	**1.28±0.05** **p=0.001**
100	0.94±0.05 p=0.103	**0.86±0.04** **p=0.004**	**0.87±0.08** **p=0.037**	**1.49±0.08** **p=0.001**
150	**0.83±0.09** **p=0.016**	**0.77±0.06** **p=0.005**	**0.74±0.07** **p=0.005**	**1.68±0.09** **p=0.001**
200	**0.83±0.08** **p=0.026**	**0.78±0.06** **p=0.005**	**0.72±0.08** **p=0.004**	**1.89±0.14** **p=0.001**
250	**0.76±0.08** **p=0.006**	**0.74±0.06** **p=0.001**	**0.70±0.05** **p=0.001**	**2.05±0.11** **p=0.001**

Table 6. Eccentric to concentric ratios ± SD for AEMG and average RJM_k^{BN} at matching angular speeds. Ratios where determined by averaging individual subject ratios. One-tailed, paired t-tests between the eccentric and concentric ratios at a given speed are shown.

Co-contraction

The magnitude of the AEMG of the biceps femoris muscle was observed to generally increase with increasing velocities for both concentric and eccentric contractions. The AEMG for the biceps femoris was normalized to the average EMG linear envelope during a MVIC of the knee flexors at 70°. The magnitude of the AEMG of the biceps femoris was found to range from 0.15 - 0.25 for slow velocities (50-150°/s) and from 0.20 - 0.35 for fast velocities (200-250°/s). Due to the large amount of discard records resulting from motion artifact (approximately 80% of the records) no statistics analysis were performed.

Regression Analysis

Linear regression and second-order polynomial regression analysis were used to explore the angular velocity effect on AEMG and average RJM_k^{BN} for concentric contractions. The results from these regressions are displayed in Table 7.

Although a very strong relationship was observed between the average RJM_k^{BN} and angular speed using both the linear and second-order polynomial fits ($r>0.994$ and $r>0.995$ respectively) only a weak relationship (r between 0.32-0.49) was found between AEMG and angular velocity. As illustrated in Table 7 there was a notable difference in the intercept (a0) and slope (a1) for the AEMG / angular velocity relationship for the three different muscles.

	Linear Regression			Second-order Polynomial Regression			
	a0	a1	R	a0	a1	a2	r
	$y=a0+a1x$			$y=a0+a1x+a2x^2$			
Average RJM_k^{BN}	1.875	0.1734	0.994	1.879	-.2218	0.009	0.995
AEMG:							
A. Vastus Lateralis	0.7220	0.0015	0.49	0.6468	0.0028	$-4.23*10^{-6}$	0.49
B. Vastus Medialis	0.9180	0.0012	0.35	0.7747	0.0037	$-8.18*10^{-6}$	0.38
C. Rectus Femoris	0.7371	0.0008	0.32	0.7401	0.0008	$1.65*10^{-7}$	0.32
Combined (A+B+C)/3	0.7924	0.0012	0.36	0.7209	0.0024	$-4.08*10^{-6}$	0.36

Table 7 Linear and second-order polynomial regressions between average RJM_k^{BN} (Nm/kg) and knee angular velocity (°/s) and between AEMG magnitude and knee angular velocity (°/s). Regressions were performed only for concentric contractions. All regressions were significant ($p < 0.05$).

EMG maps

For each subject the vastus medialis, vastus lateralis and rectus femoris EMG maps were generated. The RJM_k^{BN} generated during testing result largely from the combination of forces produced by the vastus medialis, vastus lateralis and rectus femoris muscles acting on the common patellar tendon. As a result of this fact the average vastus medialis, vastus lateralis and rectus femoris EMG maps were averaged together to generate an average EMG map which represents the general quadriceps muscle activity during knee extension (Figure 16). The average strength map for the EMG groups is also displayed in Figure 16. Quantitative analysis of the EMG map reveals that the maximum EMG magnitude for concentric contractions occurred in the

range of motion from 50° to 67° at angular velocities of 220 to 250°/s. The angle of occurrence of peak EMG magnitude was observed to decrease with increasing concentric angular velocities (95°/s at 50°/s to 60°/s at 250°/s) and increase with increasing eccentric angular velocities (5°/s at -50°/s to 17°/s at -250°/s) . The EMG magnitude for eccentric contractions was relatively equal over the range of motion of 10° to 85° and velocity spectrum of -50°/s to -250°/s. The EMG magnitude was observed to decrease near the end range of motion for both concentric (12° to 5°) and eccentric contractions (85° to 95°).

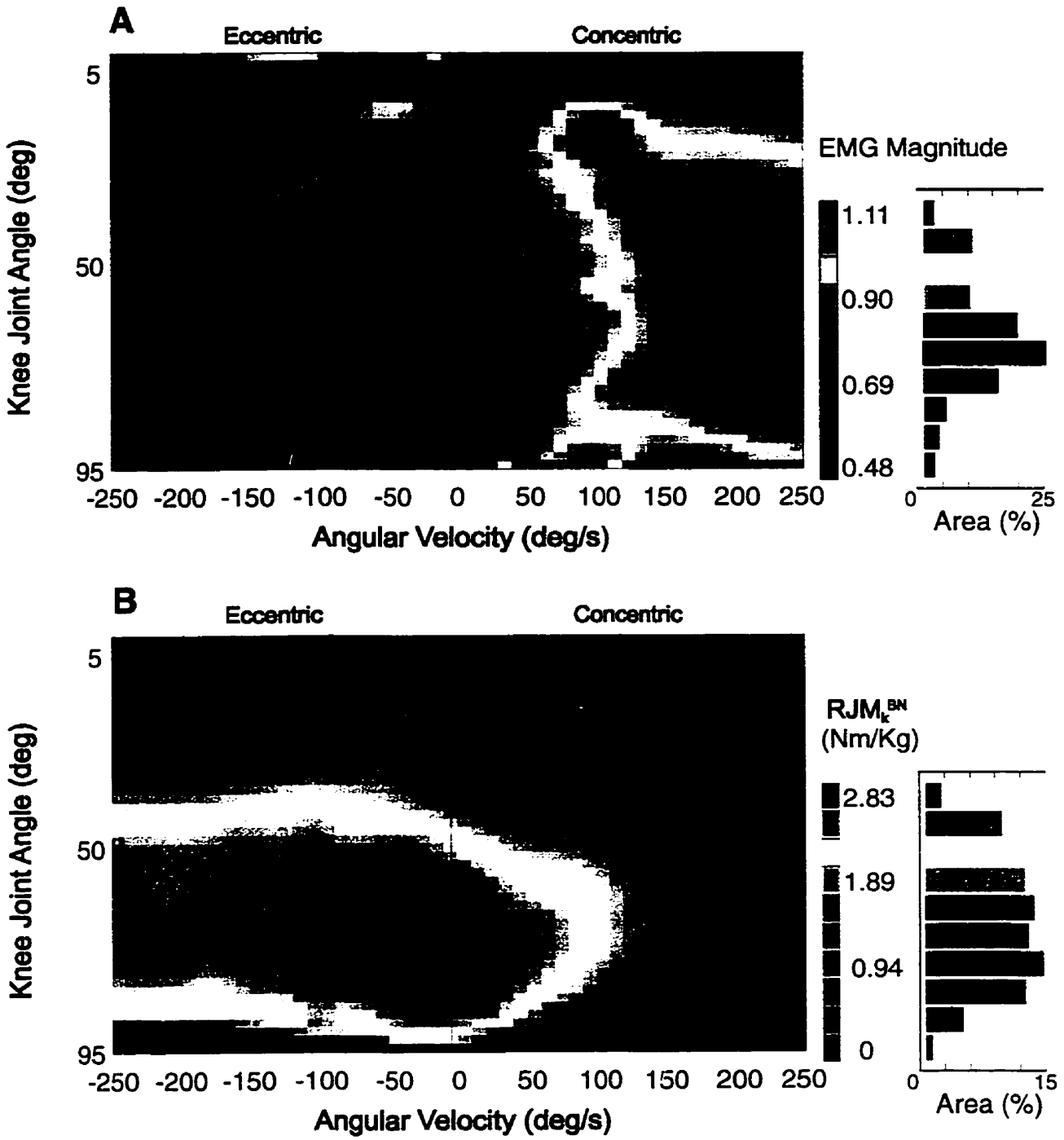

Figure 16. A- Average EMG map. B - Average strength map. The EMG map represent the average of the normalized vastus lateralis, vastus medialis and rectus femoris EMG maps. The percentage area of the map associated with each color range of RJM_K^{BN} and EMG is shown to the right of the map. The leg angular velocity is shown on the horizontal axes and the knee joint angle is shown on the vertical axis.

The average RJM_k^{BN} / AEMG relationship was generated by dividing the average RJM_k^{BN} from 20° to 80° by the velocity matched AEMG and is displayed in bar graph format in Figure 17A. The RJM_k^{BN} / EMG magnitude ratio map generated by dividing the average strength map for the EMG group by the average EMG map is displayed in Figure 17B. From the bar graph it is evident that the average RJM_k^{BN} / AEMG magnitude ratios are larger for eccentric contractions compared to concentric contractions, are relatively equal for eccentric contractions and decrease with increasing angular velocities for concentric contractions. As is evidenced by the standard deviation bars on Figure 17A there was substantially higher variability between subject in the average RJM_k^{BN} / EMG ratios during eccentric contractions compared to concentric contractions. The average RJM_k^{BN} / EMG ratio during the MVIC at 70°/s (Figure 17A) was found to be higher than the RJM_k^{BN} / EMG ratio at 70° for eccentric contractions as observed in Figure 17B.

Analysis of the RJM_k^{BN} /EMG activity ratio map demonstrates that the peak RJM_k^{BN} / EMG ratio occurs between a range of motion of 60° and 75° and a range of velocities of -190°/s to -30°/s. Of particular interest is the observation that although the angle of occurrence of peak RJM_k^{BN} and peak EMG magnitude change with increasing velocities the angle of occurrence of the peak RJM_k^{BN} / EMG ratio occurred at within a constant range of 73° to 77° throughout the range of velocities.

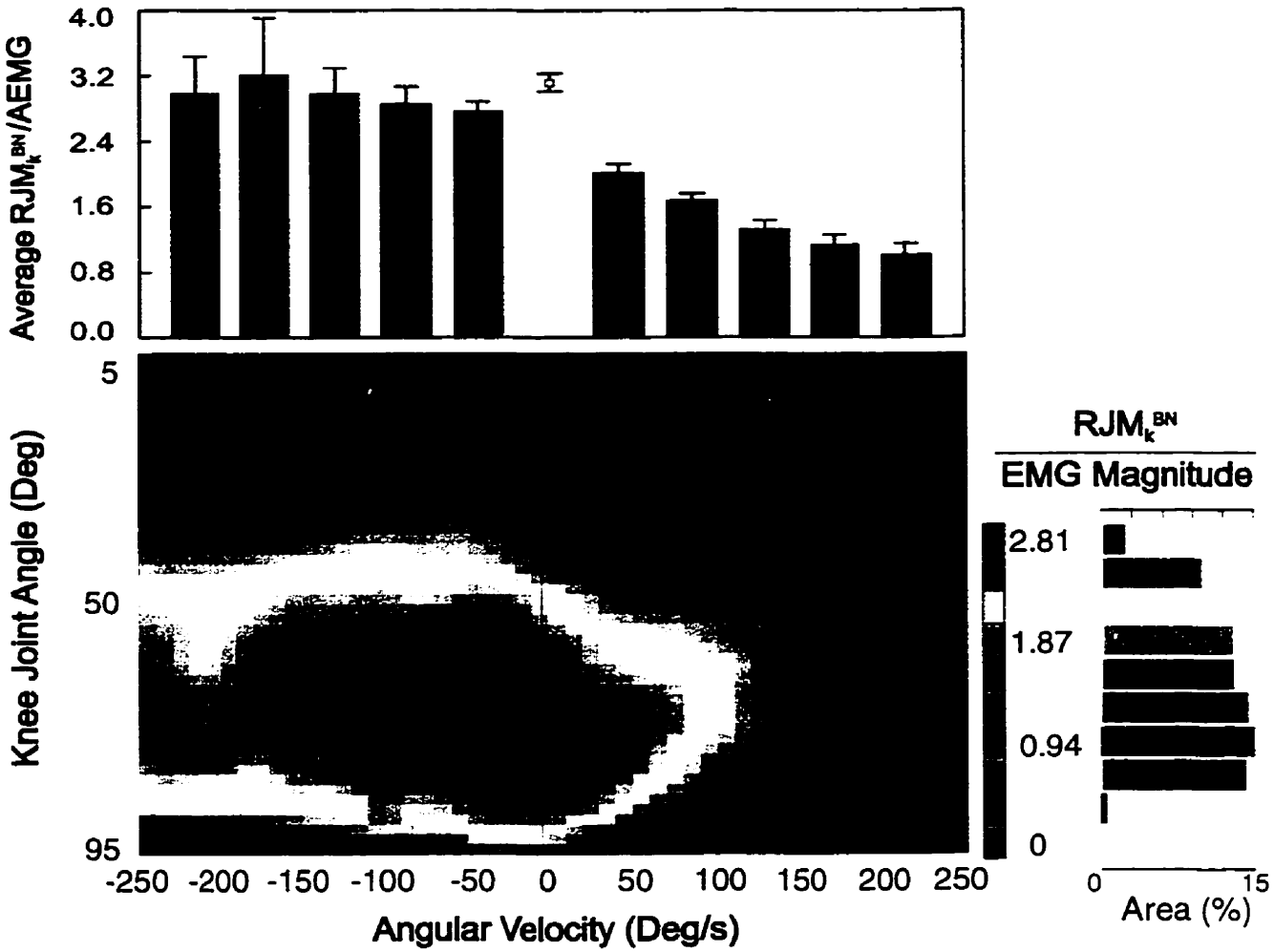

Figure 17. A - Average RJM_k^{BN} / AEMG relationship. B - RJM_k^{BN} / EMG magnitude ratio map. The percentage area of the map associated with each color range of the RJM_k^{BN} / AEMG ratio is shown to the right of the map. Standard error bars are plotted on the bar graph.

Neuromuscular Model

Firing Rate for Tetanus

The firing rate to achieve tetanus (maximum physiological force output) occurred between 22 and 80 Hz for all motor units. The average firing rate for maximum force output occurred at 54 Hz. The maximum force output was determined to be 170% greater the steady-state force output achieved for the simulation of maximum voluntary force (average firing rate 25 Hz) and 250% greater than the steady-state force output for the broad and narrow recruitment strategies.

Recruitment Pattern

The simulation results from narrow and broad rate coding strategies are shown in Figure 18. The average stimulus rate to the motor pool in the narrow rate coding strategy was 13.6 Hz, compared to 18.3 Hz for the broad rate coding strategy. From Figure 18B it is observed that the rate of rise of the force across the muscle during the first 120ms of the contraction was identical for the two recruitment patterns. In addition, the force across the muscle from 120ms to 500ms was observed to be very similar between the two recruitment patterns. The average force across the muscle from 120ms to 500ms was identical between recruitment patterns 1 and 2.

Muscle Fiber Loading

The peak force across each muscle fiber as a percentage of the peak tetanized tension for the narrow and broad rate coding strategies is shown in Figure 18C. As displayed in Figure 18C, there are obvious differences in the peak forces of matching fibers between the two strategies. The relative peak force was found to be higher in 16 out of 21 fibers using the narrow rate coding strategy. The 5 largest fast twitch fibers had higher relative peak forces using the broad rate coding strategy. The number of fibers having peak forces that were equal to their tetanized force was found to be 11 out of 21 for the narrow rate coding strategy and only 3 out of 21 for the broad rate coding strategy.

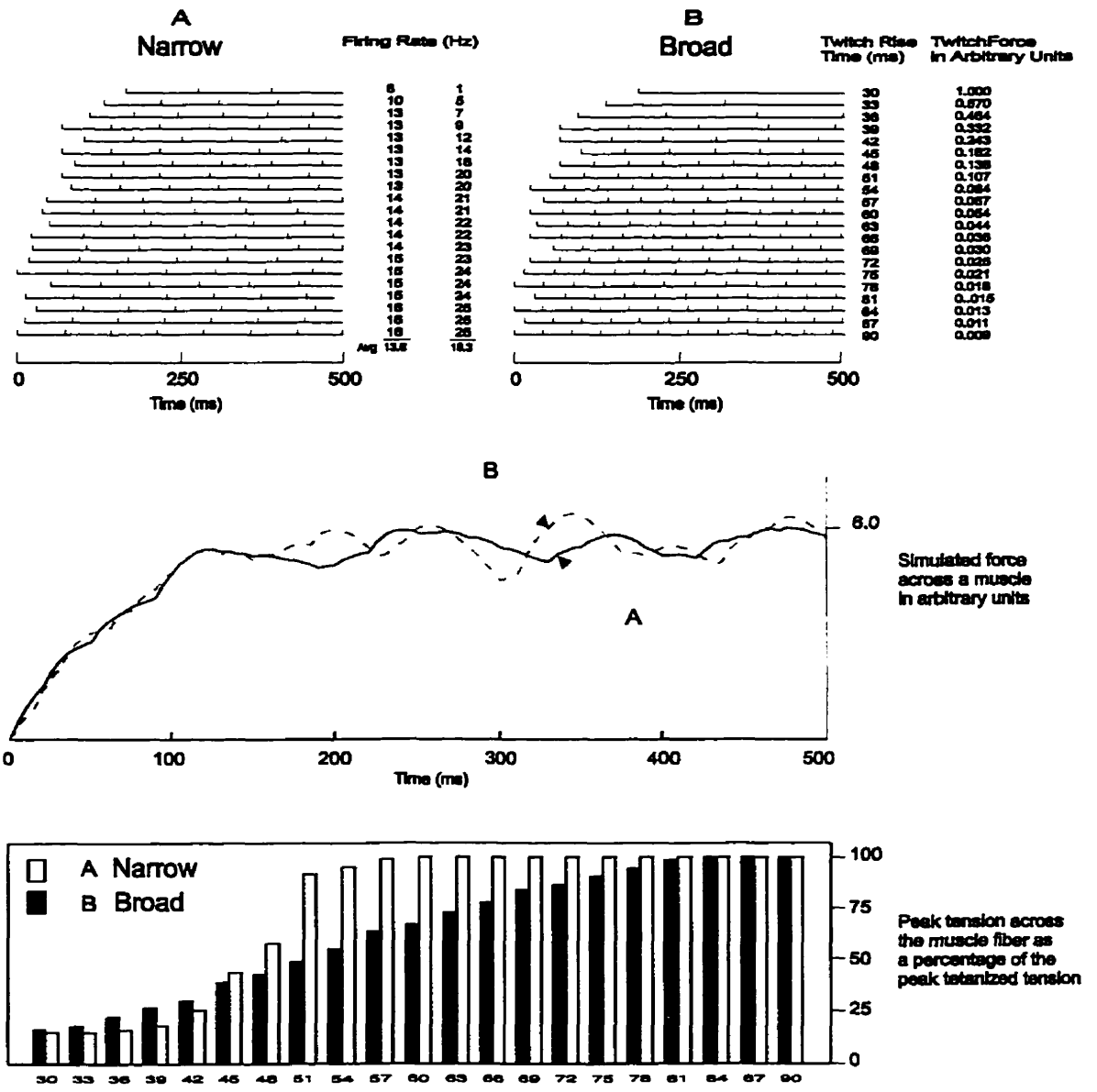

Figure 18. Simulation results from the narrow rate coding strategy (A) and broad rate coding strategy (B). Each tick on the top panel represents single stimuli to the motor unit. The middle panel is the total force response of each motor unit pool to the stimulus trains. The bottom panel is the peak tension development in each motor unit as a percentage it maximum physiological tension motor unit (tension at tetanus).

8.0 DISCUSSION

DOMS Study

Consistent with the hypotheses, it was found in this study that low level, electrical muscle stimulation resulted in significant increase in pain intensity and decreased strength of the individuals.

The multiple neural, mechanical and intrinsic factors that influence moment generation during voluntary exercise make identification of the mechanism(s) underlying the initial contraction-induced damage extremely difficult. Indirect measures of contraction-induced damage resulting from electrically evoked eccentric contractions of the knee extensor muscles were studied in order to gain insight into the neuromuscular factors responsible for the production of contraction-induced damage resulting from voluntary exercise.

The specific event(s) that serves to initiate a contraction-induced damage is not known. In spite of the potential for contraction-induced damage during any type of voluntary contraction, the probability of damage is generally considered to be the greatest during eccentric contractions. It has been well demonstrated that forces arising during lengthening contractions (eccentric) can reach 1.5 to 1.9 times the maximum isometric force in in vitro preparations (Edman et al.1978, Flintney and Hirst 1978, Harry et al. 1990, Haugen 1991, Katz 1939). In vivo and in situ animal muscle preparations have generally demonstrated that muscle activated near its maximum capacity is only damaged during lengthening contractions (Lieber et al. 1996, McCully and Faulkner 1985, Warren et al.1993c). It is important to note that, systematic evaluation of exercise or experimentally induced damage during isometric or concentric contractions has not been achieved. During voluntary contractions, EMG is lower for eccentric contractions compared to concentric contractions at the same force levels (Basmajian and Deluca 1985, Bigland and Lippold 1954, Bigland-Ritchie and Woods 1976, Gibala et al. 1995, Nakazawa et al. 1993). Based on this observation several authors have speculated that fewer motor units are activated

during eccentric contractions which results in "high tensions" being generated in the active motor units (Armstrong 1990 and 1984, Gibala et al. 1995, MacIntyre et al. 1996, Newham et al. 1983, Golden and Dudley 1992). It has been suggested that these "high tensions" result in mechanical disruption of structural elements in the active motor units or in the connective tissue that is in series with the contractile elements (Armstrong 1990 and 1984, MacIntyre et al. 1996, Newham et al.1983, Gibala et al. 1995, Golden et al.1992). However, in vivo and in situ animal muscle preparations have shown that other factors including lengthening velocity, total length change, contraction number, and initial muscle length prior to the start of the contraction also result in muscle damage, often independant of the force generated by the muscle (Brooks et al. 1995, Lieber and Friden 1993, Newham 1988, Warren et al. 1993a and 1993b).

It is proposed that the occurrence of contraction-induced damage during voluntary eccentric exercise may be dependent on the strategy used by the nervous system to activate the involved muscle(s). Specifically, recruitment, rate coding and discharge patterns used by the nervous system to activate the muscle(s) may result in damage when any of the following occur: 1) high tension development in muscle fibers, 2) recruitment of damage susceptible motor units, 3) substantial shearing between muscle fibers, and 4) repetitive loading of damage susceptible structures within the muscle. In this study, NMES was used to simulate an abnormal recruitment, rate coding and discharge pattern in the knee extensor muscles during eccentric contractions. With NMES type FF motor units comprised of type IIb muscle fibers are easily recruited as a result of their axonal properties (i.e. large axons resulting in higher excitability). The preferential recruitment of type FF is not seen in a normal physiological isometric recruitment (DeLuca et al. 1983).

In this study NMES was used on the quadriceps muscle group which resulted in an isometric RJM_K of only 15% of maximal isometric RJM_K. When the NMES is applied to the knee extensors during passively imposed knee flexion, electrically evoked contraction would result in a RJM_K equal to 20-30% of maximal voluntary

eccentric RJM_K and less than 15% of the eccentric RJM_K when the muscle is maximally physiologically activated (e.g. at 60-100 Hz). It is generally considered that low repetition low moment level (15-30% of maximum voluntary eccentric RJM_K) voluntary eccentric contractions at do not result in muscle damage. As a frame of reference, this loading would be similar to walking down a single flight of stairs with a small size backpack on.

Indicators which are normally used as evidence that contraction-induced damage has occurred after an eccentric exercise session are: 1) a decrease in isometric or isovelocity RJM generation 24 hours after the exercise session, or 2) the delayed development of soreness in the exercised muscle (Clarkson et al. 1992, Gibala et al. 1995, MacIntyre et al. 1996, Newham et al. 1987).

As hypothesized, the results from this study reveal that 24 hours after the NMES exercise session subject in the NMES group had a significant decrease in the peak isometric RJM_K^{BN}, a significant increase in soreness in the knee extensor muscles, and a significant decrease in angle and velocity specific RJM_K^{BN} measurements (31.7% of strength map). Subjects in the control group demonstrated no significant increases in soreness in the tested knee extensor muscle, and with the exception of a very small angle and velocity specific decrease in isovelocity RJM_K^{BN} (2.1% of strength map) had no significant decrease in isovelocity RJM_K^{BN} in test 2. Contrary to what was hypothesized, the subjects in the control group had a small (7.8%) decrease in peak isometric RJM_K^{BN} in test 2. Possible explanations for this decrease are 1) less motivation in the subjects when performing the isometric contraction in test 2, or 2) the initial strength test produced significant damage in the knee extensor muscles. It is very unlikely that the initial strength test produced significant damage in the knee extensor muscles of the control subjects. This is based on the observation that if significant damage had occurred as a result of the strength test in test 1 it would be expected that subjects would also experience soreness in the knee extensor and would demonstrate a decrease in isovelocity RJM_K^{BN} production in test 2.

Immediate Post-Exercise Soreness

Subjects in the NMES group demonstrated increased pain intensity levels in their knee extensors immediately post-exercise. It has generally been found that soreness after voluntary eccentric exercise is first felt between 8-24 hours post-exercise (Armstrong 1984, Ebbeling and Clarkson 1989, Friden et al. 1984, Gibala et al. 1995, Smith 1991). The plausible explanation for this observation may be related to the unusual situation that occurs with NMES where the muscle activation is dissociated from the descending commands from higher brain centres. This may result in subjects falsely reporting an unusual sensation as uncomfortable or painful. Further research into this result is warranted.

Isovelocity Strength Test

The results from this study demonstrate an angle, angular velocity and contraction type specific decrease in RJM_K^{BN}. This deficit in moment generation was observed largely during eccentric contractions at smaller knee flexion angles. Few studies have examined contraction type (isometric, concentric and eccentric) specific changes in moment generating capacity after exercise or experimental interventions.

One related study by Golden and Dudley (1992) examined the moment/velocity relationship of the knee extensors of eight non-weight trained male subjects 1 day after they had performed 100 repetitions of unilateral eccentric contractions of the knee extensors with a resistance equal to 85% of the pre-exercise maximum. They measured angle specific isovelocity knee moment during concentric and eccentric maximum voluntary contractions at 60°/s and 180°/s over 0-90° of knee flexion and maximum isometric moment. They found that 24 hours post-exercise the angle specific moments were significantly reduced by approximately the same percentage (–38%) at all velocities compared to pre-exercise values. However, it was unclear whether post-hoc multiple comparisons were performed to determine if specific differences existed for each contraction type.

It is well accepted that pain resulting from nocioceptive activation can alter neuromuscular activation patterns. It has also been suggested that the strength deficit observed during voluntary contraction could be the result of a change in neural activation patterns that would "bypass" the more severely damaged fiber (Gibala et al. 1995, Clarkson et al. 1992). During eccentric contractions, it is plausible that a pre-programmed activation strategy is employed to produce selective recruitment of undamaged motor units.

Angle of Occurrence of Peak RJM_K^{BN}

In addition to the soreness and RJM_K^{BN} measures it was also found that subjects in the NMES group had a significant shift toward flexion in the average angle of occurrence of peak RJM_K^{BN} at -250, -200, -150°/s in test 2. This finding is in agreement with previous studies which have demonstrated increases in the length at peak force or the increases in the angle of occurrence of peak moment after eccentric exercise (Jones et al. 1997, Morgan 1990). The control group demonstrated no significant changes in angle of occurrence of peak RJM_K^{BN} between test 1 and 2. It has been suggested that disruptions of numerous myofilaments in the injured muscle, or over-extended sarcomeres and disruptions within tendinous attachments lead to an increase in the series compliance of the injured muscle which results in shifts of the length-tension curve towards longer length (Jones et al. 1997, Morgan et al. 1990). In this study only a small percentage of the muscle fibers within the muscle were likely damaged. It is unlikely that this minor damage resulted in a significant change in the series compliance of the injured muscle. A change in neural activation pattern between test 1 and test 2 could wholly explain the difference in angle of occurrence of peak RJM_K^{BN} observed in this study.

As hypothesized, results from this study provide indirect evidence that the occurrence of contraction-induced damage resulting from eccentric contractions is related to neural strategies used to activate the involved muscle(s). In addition, these

results are consistent with the notion that it is not the force across the muscle, per se, that dictates the damage to the muscle during lengthening contraction but instead it is the loading of individual structures within the muscle during the lengthening contractions that dictate the occurrence of damage.

EMG Study

In this study the EMG/angle/angular velocity relationship was used to explore the status of activity in the knee extensor muscles during maximal voluntary contractions. Electromyographic signals cannot be used directly to measure the degree to which humans can fully activate their muscles. However, if individuals were able to fully activate their knee extensor muscles it would be expected that during maximal voluntary knee extensions the level of EMG measured from the knee extensor muscles would remain constant with changes in angular velocity, angle or contraction type.

As hypothesized, analysis of the data from the EMG/angle/angular velocity and AEMG/angular velocity relationships revealed that the EMG from the knee extensors increased with increasing concentric velocities. In addition, with the exception of the AEMG from the vastus lateralis at 50°/s, AEMG from the knee extensors was found to be less during eccentric contractions compared to concentric contractions and displayed no velocity dependency. Similar findings of eccentric to concentric EMG been reported in previous studies of the EMG activity from the knee extensors, although these studies did not report on isometric EMG magnitude (Komi et al. 1987, Segar and Thorstensson 1994, Tesch et al. 1990, Westing et al. 1991). The results from this study demonstrate that isometric AEMG is higher than eccentric AEMG. In addition, isometric AEMG was found to be lower than the concentric AEMG in the vastus lateralis (100, 150, 200 and 250°/s) and vastus medialis (100, 150, 200 and 250°/s) muscles. Interestingly, the AEMG during an isometric contraction was slightly higher (3%) than the AEMG of the rectus femoris muscle

during concentric contractions at 250°/s. Westing et al. (1991) reported that with increasing angular velocities from 45 to 360°/s the EMG was found to increase ~40% in vastus lateralis, ~40% in vastus medialis and only ~20% in the rectus femoris. However, Westing and colleagues did not compare their values to maximal voluntary isometric EMG levels. Based on this result and the work of Westing and colleagues it appear that subjects are able to activate their vastus lateralis and vastus medialis muscles to a greater extent at higher concentric velocities compared to the rectus femoris muscle.

Several investigators have speculated that a neural regulatory mechanism may limit the level of muscular activation during voluntary eccentric contractions to protect the musculoskeletal system from damage (Webber 1996, Westing et al. 1991, Westing et al. 1988). In addition, it has also been proposed that a neural regulatory mechanism may exist which limits muscular activation during voluntary isometric and low velocity concentric contractions (Perrine and Edgerton 1978). The results from this study indicate that subjects are not able to fully activate their knee extensor muscles during eccentric, isometric, and lower velocity concentric contractions. A putative neuroprotective mechanism may be releasing its effect as the concentric velocity increases and in combination with the fact that the force generating capacity diminishes with increasing velocity of contraction. These data support the hypothesis that not only does a neural regulatory mechanism exists which limits full activation of the knee extensor muscles during isovelocity exercises, but that this mechanism is velocity, contraction type and angle dependent. However, the extent to which this neural regulation mechanism is operational in other behaviors remains to be demonstrated.

It cannot be determined from these results if the subjects were able to fully activate their knee extensor muscles during high velocity concentric contraction. Since the isometric AEMG for the rectus femoris was always higher than the concentric AEMG for the rectus femoris at every speed, it is safe to assume that the rectus femoris was not fully activated at high concentric velocities. Several

investigators have demonstrated that short term strength training programs can result in increases in maximum RJM_K generation at high concentric velocities with little or no hypertrophy (Caiozzo et al. 1981, Kanehisa and Miyashita 1983). This suggests that untrained subjects may not be able to fully activate their knee extensors at high concentric velocities and that with training an adaptation of the neural activation pattern occurs.

Without a point of reference for maximal physiological activation it is impossible to quantify the restriction of neuromuscular activation during voluntary contractions. An eloquent and innovative study undertaken by Webber in 1996 to address this very point, demonstrated that RJM_K during voluntary eccentric contractions at 100°/s was approximately 50% lower than RJM_K that would be expected if the knee extensors were fully activated. In this study it was found that maximal voluntary eccentric EMG was 25 to 40% lower than the EMG observed during maximal voluntary concentric contractions at 250°/s. Due to the non-linearity in the EMG/ RJM_K relationship during dynamic contractions it is unclear if this 25 to 40% decrease in EMG would result in a 50% decrease in eccentric RJM_K compared to the RJM_K that would be expected during full activation. It is also possible that the subjects did not maximal activate their knee extensors during the maximal voluntary concentric contraction at 250°/s. If this was true than eccentric EMG would be reduced by more than 25 to 40% compared to the EMG level required for full activation.

Surface EMG arises from the temporal summation of action potentials emanating from active motor units (Basmajian and Deluca 1985, Bernardi et al. 1996, Fuglevand et al. 1993). If the nervous system used identical recruitment, rate coding and activation patterns during eccentric and isometric contractions it would be expected that for identical surface EMG levels the force generated during an eccentric contraction would be substantially higher than the force generated during an isometric contraction.

In this study it was found that the mean RJM_K /EMG ratio was 7 to 30% higher for the maximal voluntary isometric contraction compared to the angle specific RJM_K /EMG ratio at 70° of knee flexion during maximal voluntary eccentric contractions. This is consistent with a nervous system mechanism which used different neural strategies to produce RJM_K during eccentric contractions compared to isometric contractions. This is in agreement with several human studies in which it was found that during eccentric contractions, the nervous system appears to depart from the typically reported recruitment order observed during isometric contractions (Howell et al. 1995, Moritani et al. 1988, Nardone et al. 1989).

RJM_K^{BN}/angle/angular velocity

EMG and RJM_K^{BN}/angle/angular velocity relationships generated by the musculature about the knee during maximum voluntary contractions were examined in this investigation. As hypothesized, analysis of the RJM_K^{BN}/angle/angular velocity relationship revealed that eccentric RJM_K^{BN} values (peak RJM_K^{BN} and average RJM_K^{BN}) were greater than concentric RJM_K values. In addition, concentric RJM_K^{BN} values were found to decrease with increasing angular velocities, whereas eccentric RJM_K^{BN} values displayed no velocity dependency. Furthermore, the average isometric RJM_K^{BN} was found to be larger than the average eccentric RJM_K^{BN} . These findings are in agreement with the results of previous evaluations of knee strength (Griffen et al. 1993, Webber 1996, Westing et al. 1991, Westing et al. 1988)

The in vivo force/velocity relationship demonstrated in animal muscles during electrically evoked maximal contractions does not resemble the in vitro RJM_K^{BN}/angular velocity relationship observed in this study. The complex interactions between multiple mechanical, neural and muscular factors in the production of voluntary RJM can explain this difference. However, if the knee extensors were fully activated without corresponding co-contraction of the knee flexor muscles, one would expect that the eccentric to isometric RJM_K^{BN} ratios would be similar in magnitude with that observed in isolated muscle. In agreement with this

study, previous studies of human moment generation have generally found that maximum eccentric RJM do not exceed maximum isometric RJM (Dudley et al. 1990, Mayer et al. 1994, Webber 1996, Westing et al.1988). Although the role of co-contraction of the antagonist muscles in reducing RJM during maximum isometric and eccentric voluntary contractions is not well understood (Snow et al. 1993), it is difficult to envision that the decrease in maximal voluntary eccentric RJM compared to maximal voluntary isometric RJM solely results from increased co-contraction. The results would tend to suggest that humans are unable to fully activate their muscles during eccentric contractions.

NEUROMUSCULAR MODEL

Performance of a single bout of strenuous voluntary eccentric exercise produces an adaptation such that many indirect indicators of muscle damage are significantly reduced when a second similar bout of strenuous voluntary eccentric exercise is performed (Clarkson and Tremblay 1988, Golden and Dudley 1992, Mair et al. 1995, Newham et al 1987, Nosaka and Clarkson 1995, Nosaka et al 1991). It has been proposed that the adaptation from eccentric contractions is the result of a change in neural control strategy (Golden and Dudley 1992, Clarkson et al. 1992, Friden et al. 1983a). However, changes in the neural control strategies resulting have been difficult to verify experimentally largely because recruitment, rate coding and loading behaviors can only be monitored for a small fraction of the motor units participating in a voluntary contraction. Furthermore, investigations of the recruitment and rate coding strategies of motor units have generally been limited to low level voluntary contractions. Therefore in an attempt to illustrate how changes in recruitment and rate coding strategies may affect load sharing between motor units, a neuromuscular model was developed. This model was used to predict the forces produced by individual motor units in a hypothetical physiological motor unit pool during simulated voluntary contractions.

Several sophisticated neuromuscular models, similar to the one constructed in this study, have been developed to investigate the steady-state system output of a muscle resulting from various recruitment and rate coding strategies. (Fuglevand et al. 1993, Heckman and Binder 1991). With these models, investigators were able to demonstrate that any steady-state force output from the modeled muscles could be achieved using several different recruitment and rate coding strategies that were deemed physiologically possible. However, these models were limited to the investigation of the total system output and did not examine loading patterns of individual muscle fibers. In addition, these models were limited to investigations of the steady-state isometric force output and did not examine differences in initial force development between the different recruitment and rate coding strategies

The neuromuscular model developed in this study was comprised of 21 motor units with a physiological range of twitch properties. The twitch rise times were inversely related to twitch amplitude. Nonlinear force-firing rate behavior was simulated by varying motor unit gain as a function of firing rate and the force exerted by the system was computed as the sum of the forces from all active motor units.

The sigmoid force-frequency relationship for the modeled motor units as well as for the entire system output was similar to that regularly seen experimentally during isometric contractions (Kernell et al. 1983, Macefield et al. 1996, Thomas et al. 1991a). For example, Kernell and colleagues (1983) demonstrated that motor units of the peroneus longus muscle in the cat produced between 79-89 percent of its maximal force at a normalized stimulus rate of 1.0. In this model, the predicted force output of a motor unit for a normalized firing rate of 1.0 was approximately 85 percent of the predicted maximum force.

Although the force-frequency response of this model is similar to that seen experimental for isometric contractions, there are several limitations in this model. Due to the lack of experimental data available on the force response of motor units during eccentric and concentric contraction, the model was based on the force response of motor units observed during isometric contractions. This model is

therefore not suitable for simulations of eccentric and concentric contractions. In addition, nonlinearities in the force response of motor units resulting from activation history were not included in this model. This includes effects resulting from post-tetanic potentiation (Thomas et al. 1991b, Sandercock and Heckman 1997) and fatigue (Thomas et al. 1991b). This model is therefore not suitable for examining the force response of prolonged contractions.

In this model the mechanical interactions of motor units were assumed to be independent of one another, thus the total force output of the model was determined as the sum of the individual motor units forces. However experimental evidence has suggests that the summation of motor unit force is a nonlinear process (Clamann and Schelhorn 1988, Morgan and Proske 1984, Powers and Binder 1991). It is likely that this nonlinearity results largely from mechanical coupling between motor units due to the various proteins connecting motor units together. However, systematic investigations of mechanical coupling and nonlinear summation of multiple motor units over wide ranges of whole-muscle forces are not yet available. It therefore unknown what effect this nonlinearity of force summation would have on the force output of model during different recruitment patterns.

Loading Sharing Simulation

Two neural strategies were tested in the simulation (narrow rate coding and broad rate coding). Both neural strategies were considered physiologically feasible since similar forms of the narrow and broad rate coding strategies have been observed in human muscles during sub-maximal voluntary isometric contractions (Bigland-Ritchie et al. 1983, De Luca et al.1982, Kukulka and Clamann 1981, Milner-Brown et al. 1973c, Monster and Chan 1977). As hypothesized, the two rate coding patterns resulted in substantially different loading patterns in the muscle fibers.

In the narrow rate coding strategy the peak force was found to be higher in 16 out of 21 motor units compared to the broad rate strategy. In addition, the number of fibers having peak forces that were equal to their tetanized force (maximum force)

was found to be 11 out of 21 for the narrow rate coding strategy and only 3 out of 21 for the broad rate coding strategy. Based on this observation it may appear that the narrow rate coding strategy would be more damaging. However, it is impossible at this time to determine which type of strategy would result in the highest degree of damage. For example, the 5 fibers with higher loading in the broad rate coding strategy were all considered fast motor units. There is evidence to suggest that Type II fiber may be more susceptible to damage than type I fiber (Friden et al. 1983b, Friden et al 1988, Jones et al.1986). In addition, it is well known the fibers of a motor unit are scattered throughout a broad region of muscle and interdigitate with fibers belonging to many other motor units (Bodine et al. 1987, Burke 1981). It is plausible that one recruitment strategy resulted in higher shearing loads between motor units resulting in more damage.

This model illustrates the variability in fiber loading resulting from different neural activation strategies. The results from this model demonstrate that two neural strategies can be employed to obtain similar total muscle force output. However, the damage associated with each strategy remains unclear. These results suggest similar finding to those of the DOMS study. Specifically, it is not the force across the muscle, per se, that dictates the damage to the muscle during a contraction but instead it is the loading of individual structures within the muscle during the contractions that dictate the occurrence of damage.

9.0 CONCLUSION

1. Indirect evidence of contraction-induced injuries were found 24 hours after low level electrical stimulation. These data support the hypothesis that the occurrence of contraction-induced damage resulting from eccentric contractions is related to the neural strategy used to activate the involved muscle(s).

2. EMG/angle/angular relationship displayed region deficits with the most substantial decrease occurring during eccentric contractions. These data support the hypothesis that not only does a neural regulatory mechanism exists which limits full activation of the knee extensor muscles during isovelocity exercises, but that this mechanism is velocity, contraction type and angle dependent.

3. Taken together, these findings would be consistent with an adaptive load sharing neuronal mechanism where different motor units may be recruited under similar external loading conditions to reduce fibre and related ultrastructure stress.

10.0 REFERENCES

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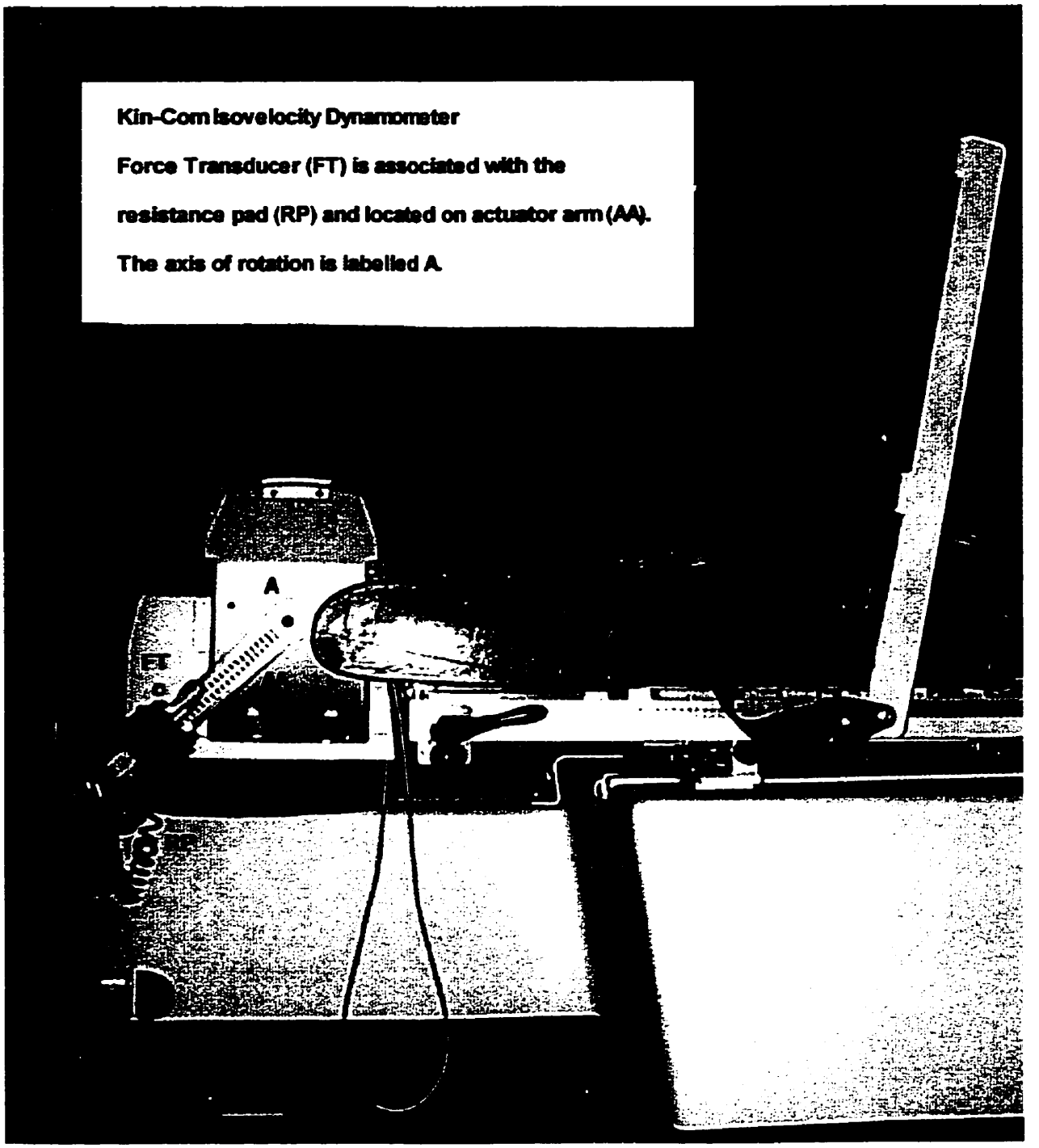
Appendix A – Isokinetic Dynamometer



Appendix B - Paraphrase and Informed Consent Form

The effect of eccentric contraction on knee extensor strength and quadriceps neuromuscular activation pattern
Paraphrase and Informed Consent Form
University of Manitoba 1996

Paraphrase #1

During functional activity and athletic pursuits muscles act by shortening, lengthening and maintaining their length to control movement and stabilize the body. Our understanding of the ability of muscles to produce force during lengthening contractions is limited. The relationships among muscle force generating capabilities and electrical activity of the muscles during lengthening and shortening contractions at different speeds and in maintaining a stationary position are not well understood. In addition, the effects of different forms of exercise on these parameters has not been well defined. This study is aimed at providing more information about human ability to produce force during contractions and the effects of different forms of lengthening exercise on this ability.

PROCEDURE

As a subject in this study you will be asked not to partake in any form of exercise other than your regular daily living activities on the day of the testing and for following 3 days. You will undergo a simple screening assessment. You will then be asked to warm-up for 5 minutes on a stationary exercise cycle. Then the strength of your muscles about your dominant knee (the one you kick a ball with) will be tested on a special device (isovelocity dynamometer). You will be required answer a few questions about your physical state before the test and 0, 2, 24 and 48 hours after the first test. The total time for testing will not exceed 4 hours.

Before the start of the first test, nine EMG electrodes will be placed on the front and back of your upper thigh to measure the electrical activity of your muscle during movements. The first test ('knee flexion isometric test') will require you to pull against a stationary dynamometer arm with your lower leg for approximately 4 seconds. The second test ('knee extension isometric test') will require you to push against a stationary dynamometer arm with your lower leg for approximately 4 seconds. The third test ('strength test') will require maximal effort knee flexion and extension (bending and straightening) at different speeds over a 90 degree range of motion. During these tests you will be asked to perform sub-maximal and maximal effort contractions. You may be asked to repeat these tests after 2 or 48 hours of rest.

The effect of eccentric contraction on knee extensor strength and quadriceps neuromuscular activation pattern
Paraphrase and Informed Consent Form
University of Manitoba 1996

Risks

Strength Test

The risks associated with the strength test are minimal including;
A. After maximal exertion you may experience some discomfort/stiffness in the muscles surrounding the knee joint which may last up to 96 hours after the test. This is a normal consequence of exercise and will resolve on its own. However, if obvious pain arises at any time during the test, the test will be discontinued.
B. Although there have not been any published reports of muscle damage during these tests, there is a remote possibility that a tear in the muscle may occur. Similar tests have been performed on athletes and untrained subjects about different joints, with and without pathology, and even after surgery without documented damage to the muscles.

Electrodes for Recording Muscle Activity

The only risk associated with having EMG electrodes on your leg is reddening of the skin due to irritation from the electrode adhesive. This is a minor side effect and should not last more than 2 hours.

You will not be identified in any published report of the results of this study. You are free to withdraw at any time without prejudice. You are not responsible for any costs directly related to the study.

If you have any questions or do not understand any aspect of this form, please contact.

Dr. Dean Kriellaars
School of Medical Rehabilitation
University of Manitoba
7876-2289

The effect of eccentric contraction on knee extensor strength and quadriceps neuromuscular activation pattern

Paraphrase and Informed Consent Form

University of Manitoba 1996

Consent Form

I have read the paraphrase and understand the nature of the study including the potential benefits and risks. I have satisfied any questions that I may have had with respect to this study. I agree to participate in this study and abide by the procedural requirements.

I understand that I may withdraw from the study at any time.

Subject _____ Date_____

Witness _____ Date_____

Investigator _____ Date_____

Appendix C - Screening Assessment for Subjects

1. Name _____

2. Date _____

3. Date of Birth _____

4. Height _____

5. Weight _____

6. Maximal circumference of thigh _____

7. Which leg would you kick a ball with? R or L

8. Have you participated in any elite/competitive sports in the past 5 years?

9. Have you participated in any lower extremity weight training greater than 2 times per week in the past 5 years?

10. Do you exercise regularly? If so, what type of activity do you participate in? How long are your workouts and how frequently do you exercise?

11. Have you ever injured the leg you kick a ball with? If yes, specify type of injury.

12. Do you have any restriction in movement of your "kicking" leg?

13. Do you have any cardiovascular problems (e.g. dizziness, high blood pressure, pain in chest) or any other medical conditions (e.g. arthritis) which might affect your ability to participate in the study?

14. Are you currently pregnant or breastfeeding?

15. Do you currently have any injury to your non-dominant leg?

16. Have you ever used performance enhancing drugs? If yes, please specify types of drugs used, dosage of drugs consumed and period of time spent using each drug?

Appendix D – Electromyography for NMES group

AEMG / knee angular velocity relationships for the vastus lateralis, vastus medialis and rectus femoris muscles during for test 1 and test 2 for three subjects in the NMES group are displayed graphically in the figure below. AEMG represents the normalized average magnitude of the EMG linear envelope from 20° to 80° of knee flexion. The AEMG was normalized to the average magnitude of the EMG linear envelope during the two MVIC of the knee extensors performed during test 1. The average RJM_k^{BN} is calculated from 20° to 80° of knee flexion.

No statistical analysis was performed on this data due to the small number of subjects. Of interest is the observation that although the average RJM_k^{BN} decreased in test 2 at every angular velocity the AEMG increased in the vast majority of angular velocities (21 of 30).

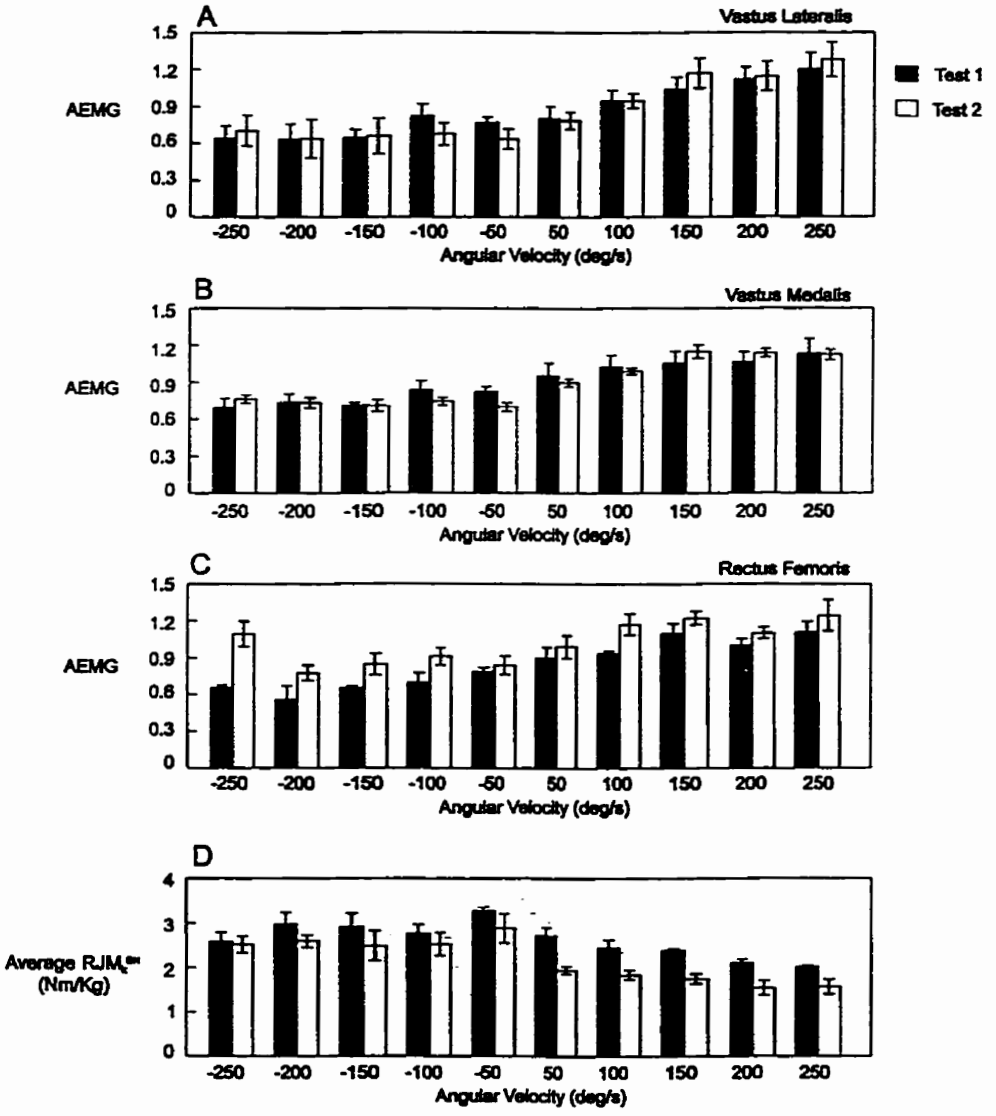
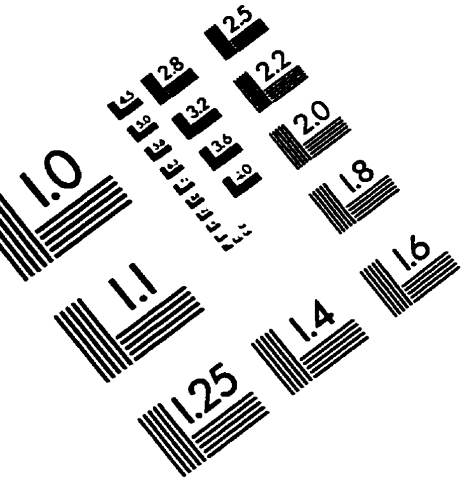
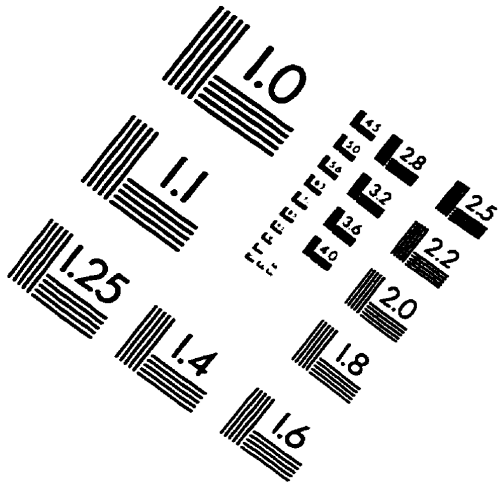
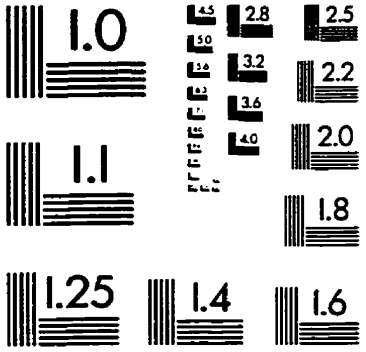
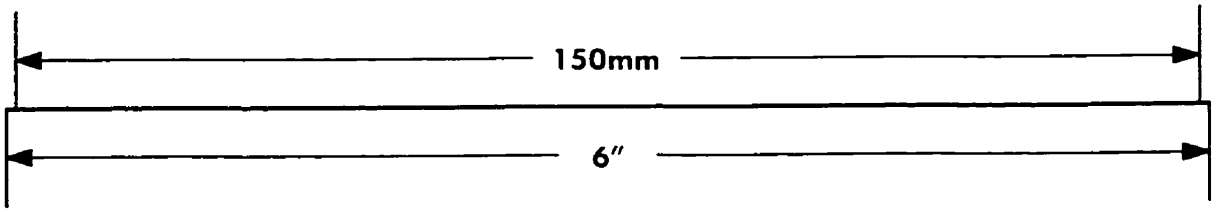


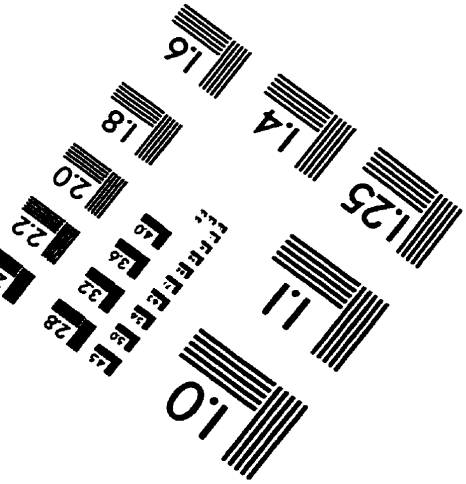
IMAGE EVALUATION
TEST TARGET (QA-3)

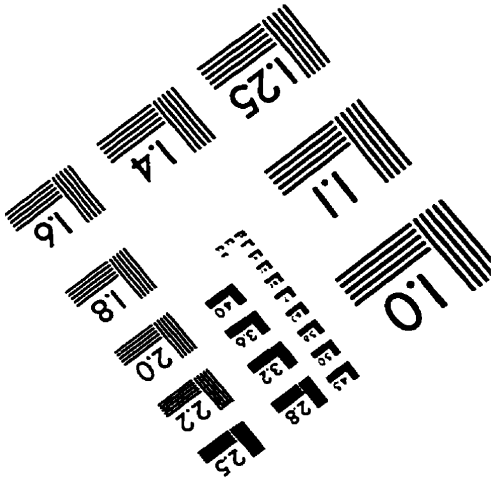












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