THE MECHANICAL AND NEUROPHYSIOLOGICAL CHANGES THAT ACCOMPANY EXERCISE-INDUCED MUSCLE INJURY

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ABSTRACT

Injuries to finger and wrist muscles were examined in two separate studies. In the first study, 10 subjects were tested pre- and 24 hours after exercise-induced injury to the first dorsal interosseus (FDI) muscle. The lack of any significant change in active joint stiffness or its components at low torque suggests that the muscle injury produced little effect on the mechanical properties of small slow-twitch motor units. The increase in stimulated intrinsic joint stiffness and significantly lower joint stiffness at high torque suggests that there were greater effects on the mechanical properties of the large fast-twitch motor units. M-wave potentiation 24 hours after muscle injury suggests there were long-lasting changes to membrane properties. Compensation for the muscle injury included an increase in mean rectified EMG (MEMG) during submaximal contractions and a shift in the median frequency (MF) to lower values. These changes suggest reduced activation of the large motor units. Subjects' ability to track torque and to stabilize the MP joint was impaired. The decline in MP flexion maximum voluntary contraction (MVC), which was greater than anticipated, suggests that activation of the flexor digitorum superficialis (FDS) muscle was affected by the injured FDI muscle. Compensation by the FDS muscle for the injury to the FDI muscle occurred during a sustained submaximal metacarpophalangeal (MP) flexion task.

In the second study, 10 subjects were repeatedly tested once prior to and four times following a bout of eccentric exercise with the wrist extensor muscles. There was reduced passive range of motion (ROM), without change in passive

or active wrist joint stiffness, which suggests that there were changes to the mechanical properties of non-contractile tissue or that swelling occurred which only influenced movement at the extremes of the ROM. There was an impairment in the ability to track and sustain torque and the ability to stabilize a mechanically unstable load. The response of all of the wrist extensors was similar, suggesting that they operated in a synergistic manner. Tests performed with the wrist flexors as agonists showed no effects of the injury to the wrist extensors. The greatest deficit in maximal voluntary force occurred on Day 1 and recovered by Day 10. Muscle soreness peaked on Day 2. Performance deficits were more closely linked to impaired muscle function than to sensation of soreness.

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"To thine own self be true"
(unknown)

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TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER ONE - INTRODUCTION

Introduction to Eccentric Exercise

The term eccentric refers to a mechanical condition in which the torque due to the load is greater than the muscle torque and as a consequence the muscle length increases (Enoka 1994). Contractions of this type are a common occurrence in everyday activity, as all movements are a result of some combination of concentric, isometric and eccentric muscle contraction (Evans and Cannon 1991). For example, when a weight or a body is slowly elevated, muscles shorten to produce the force and cause movement. When the same object or body is slowly lowered the same muscles produce force while lengthening. During eccentric contractions, the maximal force that can be developed may be up to twice that during an isometric contraction (Katz 1939; Singh and Karpovich 1966; Komi 1973; Edman et al. 1978; Van Atteveldt and Crowe 1980; Westing et al. 1990), even though the number of attached cross-bridges is only approximately 10% greater (Lombardi and Piazzesi 1990; Faulkner et al. 1993). The central nervous system (CNS), in planning movements, seems to take advantage of this property. It has been demonstrated that the electrical activity of a muscle working eccentrically against a load is less than its activity when working concentrically against the same load (Asmussen 1953; Bigland and Lippold 1954; Basmajin 1967; Bigland-Ritchie and Wood 1976; Nakazawa et al. 1993). From cross-bridge theory of active force generation, the increase in force producing ability of stretched muscles is

thought to arise from the stretching of elastic cross-bridges, resulting in greater average force during attachment than when the muscle is isometric or is shortening (Huxley 1957, 1969; Harry et al. 1990; Wood et al. 1993).

After unaccustomed activities that involve multiple or high intensity eccentric contractions there is often a sensation of soreness the following day. This discomfort is thought to arise from damage to the active muscles, and is referred to as an exercise-induced muscle injury. Although injury to skeletal muscle is possible during concentric and isometric contractions, the probability of injury is highest during exercise involving eccentric contractions (Newham et al. 1983; Clarkson et al. 1986; Jones et al. 1989; Berry et al. 1990; Faulkner et al. 1993; Warren et al. 1993b; McCully and Faulkner 1986). Eccentric exercise provides a model for experimental muscle damage. Studies in this area have focused on the causes of muscle injury, the types of tissues damaged, the consequences and time course of recovery, as well as the adaptations that occur.

Mechanisms of Exercise-Induced Muscle Injury

There has been considerable interest in the sequence of events associated with muscle injury during and following eccentric exercise. The muscle injury is thought to be due to an initial mechanical event, which is further aggravated by a secondary injury involving metabolic changes (Stauber 1989; Armstrong 1990; Ebbeling and Clarkson 1990; Clarkson 1992; Warren et al. 1993b, c). These ideas are based on considerations of the high levels of force

developed during eccentric contractions, the evidence of swelling and the delayed onset of the muscle soreness.

Jubrias and Klug (1993) and others (McCully and Faulkner 1993; Warren et al. 1993a, b, c) suggested that the initial injury to the muscle is due to the high force during eccentric contraction. Early work of Abbot et al. (1952) and others (Asmussen 1953; Bigland and Lippold 1954; Knuttgen et al. 1971; Aura and Komi 1986; Knuttgen 1986) found that for a given level of force development there is less electrical activity and metabolic cost when a muscle is working eccentrically. This would suggest that for a given level of force there are fewer muscle fibers recruited, and thus a greater amount of force per individual muscle fiber during stretch. (Katz 1939; Curtin and Davies 1973). McCully and Faulkner (1986) have further suggested that as the peak force increases so does the extent of injury. Warren et al. (1993b) implicated materials fatigue, from high tensile stresses at such force levels, as the cause of damage to the muscle.

Others have suggested that the mechanical mechanism of injury is not related to the peak tension, but rather to a tension imbalance that occurs between adjacent sarcomeres (Lieber and Baskin 1983; Fridén and Lieber 1992; Faulkner et al. 1993; Wood et al. 1993). Within a given muscle fiber there exist populations of sarcomeres which have slightly different lengths (Huxley and Peachy 1961; Lieber and Baskin 1983) and sarcomeres which change length with slightly different velocities (Gordon et al. 1966; Edman 1979). It has been suggested that during a lengthening contraction the force in adjacent

sarcomeres could vary by more than 50% of maximum isometric force. This is due to the steepness of the slope of the lengthening portion of the force-velocity relationship (Fridén and Lieber 1992) and the resultant tension imbalance would lead to sarcomere damage. Wood et al. (1993) also endorsed the "non-uniform sarcomere hypothesis" based on their work with the frog sartorius muscle. They found that lengthening a muscle, which was already at a long length, resulted in non-uniform distribution of the length changes among sarcomeres - some sarcomeres stretched beyond minimum overlap while others stretched very little. With repeated lengthening contractions the weaker sarcomeres, due to being at longer lengths and having less filament overlap, were stretched by adjacent sarcomeres which led to sarcomere damage. These findings are supported by computer simulations performed by Morgan (1990).

Lieber et al. (1991) and others (Fridén and Lieber 1992) have also suggested that the cause of muscle damage during repeated eccentric contractions was due to fatigue and consequent injury of weakened fast glycolitic (FG) muscle fibers. Lieber and Fridén (1988) further speculated that during eccentric exercise the FG fibers may fatigue, due to their inability to generate sufficient ATP, and enter a state of rigor. Stretching fibers which are in this high stiffness state could lead to myofibrillar damage. There is some evidence that selective recruitment of fast fibers and selective derecruitment of slow fibers occurs during eccentric contractions. Nardone and Schieppati (1988) found that during voluntary triceps surae lengthening contractions there was derecruitment of the soleus muscle and slow-twitch motor units,

accompanied by selective activation of the fast gastrocnemius muscle and large fast-twitch motor units.

Other factors that have been implicated in inducing the initial injury include: the amount of strain in the muscle (Lieber and Fridén 1993) and metabolic factors such as high temperature, insufficient mitochondrial respiration, oxygen free radical production, and lower pH (Armstrong 1990).

Whatever the mechanism for the initial muscle injury, there appears to be strong evidence for secondary injury. The initial structural damage is thought to precipitate further degradation in the ensuing hours and to last for days. These subsequent events are thought to give rise to muscle soreness and decrement in force producing capability. Calcium (Ca^{++}), lysosomes, and myofibillar proteins with associated phagocytic activity have been suggested as mediators of the subsequent damage and repair (Ebbeling and Clarkson 1990; Clarkson 1992; Faulkner et al. 1993).

Faulkner et al. (1993) proposed that secondary injury to the muscle occurs by a biochemical process which involves phagocytic activity at the site of the original damage. Neutrophils and monocytes which release oxygen radicals cause further degradation of the muscle. The presence of macrophages, indicative of this process, has been shown in animal studies (Armstrong et al. 1983), but the results of human morphological studies are less clear (Jones et al. 1986). More recently MacIntyre et al. (1996) used technetium-99m labeled white blood cells to detect the early presence of inflammatory cells in the exercised muscle. They reported significantly more inflammatory cells in the

exercised muscle than the contralateral non-exercised muscle. These counts remained elevated up to and including 24 hours post-exercise.

Armstrong (1990) suggested that following the initial mechanical events there is an autogenic phase in the development of exercise-induced muscle injury. It is during this phase that abnormally high levels of Ca^{++} in the muscle trigger a series of events leading to necrosis. The accumulation or loss of Ca^{++} homeostasis during this phase results from damage to the sarcoplasmic reticulum (SR), failure of the SR to take up Ca^{++}, and/or diffusion of extracellular Ca^{++} through the damaged sarcolemma (Armstrong 1990; Ebbeling and Clarkson 1990; Evans and Cannon 1991; Byrd 1992). The increase in Ca^{++} may cause mitochondrial swelling, and/or activate proteases and phospholipases which can cause further myofibrillar degradation. According to Armstrong, the next stage is the phagocytic stage which involves the typical inflammatory response. This is followed by the final repair stage, the regeneration phase. Although the specific mechanisms are not known, it is possible that an initial event results in a loss of intracellular Ca^{++} homeostasis, which could play a primary role in further degradation.

Indicators of Exercise-Induced Muscle Injury

Muscle injury following eccentric exercise has been evaluated by direct and indirect indicators. The most direct evidence is provided by either histological or ultrastructural analysis. Electron microscopic (EM) analysis of muscle samples has shown broadening, streaming, and in places, total

disruption of Z discs (Fridén et al. 1983a). Further structural alterations that have been observed include myofibrillar and sarcolemmal disruptions, widening of the A and I bands, increased mitochondrial volume, cytoskeletal changes and increased cellular infiltration of monocytes and/or macrophages (Armstrong et al. 1983; Newham et al. 1983; McCully and Faulkner 1986; Ebbeling and Clarkson 1990; Stauber et al. 1990; Lieber et al. 1991; Byrd 1992).

Direct indicators have provided insight into the selectivity of muscle damage. Lieber and Fridén (1988) examined 231 fibers from six rabbit tibialis anterior muscles following lengthening while stimulated electrically and reported that the damaged fibers were exclusively of the FG type. The damage may also be predominantly to fast fibers in humans (Fridén et al. 1983a; Ebbeling and Clarkson 1990; Fridén and Lieber 1992). For example, biopsies by Jones et al. (1986) of the gastrocnemius muscle, exercised by walking backwards on an inclined treadmill, indicated that the type II muscle fibers were more severely affected than type I muscle fibers. Fridén et al. (1983a) reported a type II:type I muscle fiber ratio in randomly chosen micrographs containing Z-band disturbances of 2.8:1 immediately after and 3.0:1 three days after eccentric exercise of the vastus lateralis muscle on a bicycle ergometer. In a morphological study of runners Fridén et al. (1988) reported that 36% of the fibers examined in the vastus lateralis demonstrated abnormalities two hours following repetitive bouts of sprint running. Eighty percent of the abnormalities were found in fibers with narrow Z-bands, the type IIB fibers.

Studies have also provided some insight into the timing and magnitude of exercise-induced damage. Histological studies by McCully and Faulkner (1986) and others (Fridén et al. 1983a; Stauber et al. 1990) have demonstrated that there is minimal damage immediately after the exercise, and that ultrastructural disruption peaks 2 days or more after the initial injury. McCully and Faulkner also attempted to quantify the magnitude of damage 3 days after the initial injury using light microscopic techniques. In a single cross section through the belly of the muscle they reported injury involving 34% of the total muscle fiber cross-sectional area. Furthermore, there was a high correlation between the histological appearance of injury and decrease in peak force levels. Fridén et al. (1983a) have also reported ultrastructural changes in up to 52% of the muscle fibers observed. However, traditionally this type of evaluation is only done on one occasion following the injury. Due to the focal nature and time-course of exercise-induced muscle injury it is difficult to quantify and evaluate changes over time with direct indicators.

Direct indicators provide the most conclusive evidence of exercise-induced muscle injury. Direct indicators of damage can also be correlated with indirect indicators, which are often easier to measure and can be repeated over a period of time. Indirect indicators have included presence of muscle proteins in the blood, soreness, stiffness, and reduction in muscle strength and range of motion. Levels of muscle enzymes such as creatine kinase (CK), lactate dehydroginase (LDH) and myoglobin have been measured. The most commonly studied muscle enzyme is CK, elevated levels of which are taken to

reflect changes in the integrity of the muscle membrane. Numerous studies have shown dramatic increases in serum CK after eccentric exercise (Tiidus and Ianuzzo 1983; Byrnes et al. 1985; Jones et al. 1986; Newham et al. 1987; Clarkson and Tremblay 1988; Ebbeling and Clarkson 1990). Elevated levels are not found until 24 - 72 hours after exercise and can peak 1 to 7 days later. The time course, as well as the magnitude of change in CK levels, varies greatly among individuals and different exercise protocols and is, therefore, felt not to be an accurate indicator of the extent of muscle damage (Clarkson et al. 1992). Furthermore, levels of such enzymes have shown little or no relationship to other indicators of muscle injury (Newham 1988a; Clarkson et al. 1992; Rodenburg et al. 1993).

The most obvious and most common indirect indicators of exercise-induced muscle injury have been soreness and the sensation of stiffness, decreased range of movement and weakness. In an attempt to understand the mechanisms, the time course for recovery, and to aid in diagnosis and treatment, there have been many studies which have focused on these more functionally related indicators. Each of these indicators has been found to change with an unique time course during recovery, with variability in onset, duration of effect, and peak intensity (Jones et al. 1986; Stauber et al. 1990; Howell et al. 1993; Crenshaw et al. 1994; MacIntyre 1994). For example, Clarkson et al. (1992) reported, that following repeated eccentric exercise of the forearm flexor muscles, soreness was most severe 2-3 days post-exercise, while maximal strength and ability to fully flex the arm showed the greatest

decrement immediately following the exercise. Other studies have also investigated the relationship between indirect indicators (Tiidus and Ianuzzo 1983; Jones et al. 1986; Stauber et al. 1990; Howell et al. 1993; Crenshaw et al. 1994; MacIntyre et al. 1996). Rondenburg et al. (1993) suggested that large changes in one indicator corresponded with large changes in another indicator. However, they further concluded that it was difficult to predict the time courses of all changes from one indicator alone. The variability, and apparently weak correlation between the indirect indicators reported thus far, point out the need for further study to relate the changes in indicators of exercise-induced muscle injury to function.

Delayed Onset Muscle Soreness

Perhaps the most common indirect indicator of post-exercise injury is the reported feeling of muscle soreness. Delayed onset muscle soreness (DOMS) refers to the sensation or discomfort within the muscle during palpation or movement. Although it has been shown that muscle injury occurs during the eccentric exercise, the discomfort associated with the injury is not manifested until 24 - 72 hours later (Armstrong 1984; Clarkson et al. 1986; Jones et al. 1987a; Newham 1988; Rodenburg et al. 1993; MacIntyre et al. 1996). The discomfort gradually subsides then disappears within 5 to 7 days (Armstrong 1984; Jones et al. 1987a). Two major hypotheses have been proposed as to the cause of DOMS. Crenshaw et al. (1994) suggested that DOMS may be related to an increase in intramuscular pressure (IMP). When IMP was measured via catheterization of the vastus lateralis muscle two days after repetitive eccentric

exercise, elevated IMP and extensive intracellular swelling were evident and found to be correlated with the reported DOMS. Other researchers (Newham and Jones 1985; Fridén et al. 1986) have studied IMP and soreness and suggested that the amount of pressure is dependent upon the compliance of the muscle compartment being exercised. In muscles with compliant compartments there may not be any elevation of pressure, but they may still experience soreness, suggesting that IMP may not be directly associated with DOMS.

Smith (1991), in her review, suggested that muscle soreness was related to inflammation, as it exhibited signs typically associated with acute inflammation. These included increased levels of circulating neutrophils and monocytes, which synthesize large amounts of prostaglandin. Prostaglandin then sensitizes free nerve endings (type III and IV) in the muscle such that increased pressure due to applied forces or contraction leads to the generation of action potentials in these nerve endings. The delayed nature of the soreness was thought to be related to the delayed macrophage entry into the injured area. However, a study by Kuipers et al. (1985), in which administration of an anti-inflammatory drug had no significant effect on soreness suggests that this mechanism may not be involved in DOMS.

Although the specific mechanisms underlying muscle soreness have remained unclear, the effect of this pain on other indicators of muscle function has been of particular interest. Smith (1991), in her review, suggested that the ability to generate force may be influenced by acute inflammation. She stated, that it is possible that lower maximal force may be due to mechanical reflex

inhibition of the muscles experiencing pain. However, there have been studies that suggest otherwise. Newham et al. (1987) superimposed electrical stimulation during maximal voluntary contraction (MVC) to determine if subjects were performing maximally. They were unable to elicit any further force and concluded that subjects were contracting maximally in spite of the pain. Other investigators (Clarkson and Tremblay 1988; Newham 1988a; Rodenburg et al. 1993) have reported a poor correlation between the time course of the force decrement and reported muscle soreness.

There has been some speculation that pain could cause changes in muscle recruitment. Crenshaw et al. (1994), in order to explain a lack of elevation in their IMP/torque ratio, speculated that there could have been a shift in contribution by the quadriceps muscle in such a way that the sore vastus lateralis contributed less but overall torque remained the same. However, because they did not record electromyographic (EMG) activity in their study they could not test this hypothesis. Although there have been extensive studies on muscle soreness, they have been predominantly focused on the causes and time course of the discomfort. There has been limited investigation of the implications of the pain on motor control parameters, such as activation and recruitment patterns and how these changes might affect performance.

Stiffness

Another common indicator of exercise-induced muscle injury is the sensation of stiffness. Although commonly reported by subjects, this parameter has been studied much less extensively than others. The sensation of stiffness

has been described as a reluctance to stretch the affected muscle and has been most commonly evaluated by measuring the resting position of the joint post-exercise. As an example, work by Stauber et al. (1990) and others (Howell et al. 1985; Jones et al. 1987a) has shown that the elbow angle of the relaxed arm in standing subjects becomes more acute following eccentric exercise of the elbow flexors. Immediately after exercise, this angle begins to decrease and continues to decrease until the third day. The resting angle then gradually increases over the next week (Clarkson et al. 1992). Other attempts to quantify stiffness have involved determining the amount of force required to stretch the injured muscle. Jones et al. (1987a) assessed the amount of force required to extend the elbow through full range of motion following a protocol of eccentric exercise. They reported that post-exercise, the force required for movement increased in parallel with discomfort. Howell et al. (1993) estimated passive stiffness of the elbow flexors over the intermediate range of elbow angles from the slope of the relationship between static torque and elbow angle. They reported that passive stiffness more than doubled immediately after exercise and remained elevated for about four days.

Various theories have been proposed to account for the increased stiffness. Initially, it was believed that the sensation of stiffness was due to increased electrical activity in the muscle. EMG recordings from the sore muscles during passive extension have since shown that this is not the case (Bobbert et al. 1986; Jones et al. 1987a). Clarkson et al. (Ebbling and Clarkson 1990; Clarkson et al. 1992) proposed that an influx or accumulation of Ca++

could activate specific enzymes and cause excessive contractures in the damaged fibers. Howell et al. (1985) felt that the restriction of motion and apparent decrease in resting length of the muscles was due to edematous changes in the perimuscular connective tissue. Stauber et al. (1990) concurred and proposed that the swollen tissue pushing against the fascia could shorten the muscles passively. However, the relationship between the time courses of swelling and of changes in resting arm angle have not supported this theory (Clarkson et al. 1992).

Warren et al. (1993b) examined the role of contractile elements and activation in the force deficit exhibited following exercise-induced muscle injury. By stimulating the rat soleus muscle to tetanus and then rapidly stretching the muscle at constant velocity for 20 ms, they were able to measure the resultant resistance and thus, calculate the active stiffness of the muscle. By comparing the stiffness prior to, and following eccentric contractions, they showed a decrease in active muscle stiffness post-exercise. A comparison between the active muscle stiffness and the maximum tension developed allowed the investigators to speculate on the site and nature of the injury in the muscle. For example, had they found a proportionately greater loss of maximal tension than stiffness they could have interpreted this as indicating that cross-bridges were still attached but not producing active force. However, because the tension and stiffness showed similar patterns of change, the investigators suggested that the force deficit was the result of an inability to activate intact contractile proteins and not due to a loss of contractile elements.

Functional Performance

Following a bout of eccentric exercise there is a distinct decrease in force producing capability. Most commonly this decrement is quantified by the decrease in maximal isometric force. Clarkson et al. (1992) reported an immediate loss of over 50% isometric strength in forearm flexors, followed by gradual improvement over the next ten days. Other researchers have reported that subjects may not return to initial maximal isometric strength for up to three to five weeks (Newham et al. 1987; Ebbeling and Clarkson 1990; Howell et al. 1993). Other tests of strength capabilities have also shown similar decrements. Hasson et al. (1993) quantified peak torque for both concentric and eccentric contractions of the knee extensors at an angular velocity of 60°/s. They reported, that at 24 and 48 hours post-exercise, and for both types of contractions, values were significantly less than baseline. Fridén et al. (1983a) tested knee extension at different angular velocities after eccentric exercise and reported that the greatest decrement in force producing capability occurred at the highest angular velocity (300°/s). This trend remained for tests performed 20 minutes, 3 days and 6 days post-exercise. The authors speculated that the velocity related decrement in force occurred due to the selective nature of the muscle injury to type II muscle fibers.

Various hypotheses have been advanced for the large immediate and the prolonged strength loss. One possibility is that some sarcomeres are lengthened by performance of lengthening contractions (Katz 1939). Wood et al. (1993) suggested that as some sarcomeres were lengthened, at a fixed

muscle length other active sarcomeres would compensate by adopting shorter lengths, and there would be a resultant shift to longer lengths of the force-tension relationship. Recent work by Brown and Loeb (1994) supports this theory, as they found a dramatic narrowing and shifting of the isometric force-length curve in the cat caudofemoralis muscle after as few as two eccentric contractions such that force at short lengths was reduced by as much as 90%, while force at long lengths remained constant. Saxton and Donnelly (1994b) reported that, after subjects performed eccentric exercise of the forearm flexors, there was a disproportionately greater loss in maximum torque at small angles (short muscle lengths) compared to large angles (long muscle lengths). This trend persisted for up to four days but slowly equalized over ten days of recovery. These authors also suggested that a shift in the length-tension relationship was responsible for the greater loss in force production at more acute angles

Some researchers attribute the deficit in maximal force production to structural changes in the connective tissue. Damage to elements in series with the sarcomeres would contribute to an inability to transmit the tension between adjacent sarcomeres (or fibers). Histological evidence of damage to the A band and Z line streaming or complete disruption following exercise supports this theory (Fridén et al. 1983a; Fridén and Lieber 1992). Warren et al. (1993b) compared changes in stiffness of stimulated muscle to changes in maximum tension and speculated that the similar pattern of decline in tension and muscle

stiffness which they observed might indicate an impairment in cross-bridge formation or a specific loss of series elastic components .

Other researchers have suggested that the decrement in force producing capability is of a neurological origin. Although there is a poor relationship between the time course of the force decrement and soreness, there has been some suggestion that a change in neural activation patterns avoids recruiting the damaged fibers in order to facilitate their recovery. There has been some evidence that patterns of muscle activation are altered after exercise. Hasson et al. (1993) reported significantly reduced root mean squared (RMS) EMG of the vastus lateralis muscle 24 and 48 hours after exercise. This muscle was also reported to be the most sore. On the other hand, the rectus femoris muscle showed no change in RMS EMG and little soreness. In contrast, Newham et al. (1983) examined activation patterns and integrated EMG (IEMG) of the quadriceps muscles during a two second submaximal isometric knee extension task and saw no significant change in the ratio of the contributions of the three muscles to the total recorded quadriceps activity. However, these findings should be interpreted in light of the fact that the decrement in maximal voluntary force recovered fully 24 hours after the exercise bout, suggesting minimal, if any, damage as result of exercise.

The approach used by Warren at al. (1993a) to investigate the force deficit highlighted the inability to activate contractile proteins. They suggested that the force deficit was not related to damage of specific contractile elements themselves, but rather from failure of the excitation process prior to the release

of Ca^{++}, resulting in a decrease in the number of attached (activated) cross-bridges. They speculated that this was the result of an activation failure, due to structural damage to the sarcolemma, causing loss of the normal ion distribution across the membrane and thus impairing action potential conduction.

In addition to evaluating maximal force production, some investigators have assessed the relationship of force to excitation frequency in muscle post-exercise (Edwards 1981; Newham et al. 1983, 1987; Sergeant and Dolan 1987; Jones et al. 1989; Balvane and Thompson 1993). This relationship is derived by comparing the force generation observed for different frequencies of electrical stimulation. Following eccentric exercise muscles exhibit alterations in the contractile properties such that the force-frequency relationship is shifted in the direction of higher frequencies, i.e., electrical stimulation at low frequencies (1-20 Hz) results in less force than before exercise (Newham et al. 1983). This is referred to as low frequency fatigue. Fatigue of this nature is apparent immediately after exercise and may recover in as little as three to four days (Jones et al. 1989), or can take more than a week to recover (Newham et al. 1988). It has been suggested that this type of fatigue is not a consequence of the metabolic costs of the exercise (Jones et al. 1989), but rather the result of mechanical damage (Newham et al. 1983). However, the specific mechanism remains unclear.

Balvane and Thompson (1993) reported the response of post-exercised muscle to repeated 20 Hz trains of electrical stimulation lasting 300 ms, performed every second for 120 seconds. By recording the average force

produced during each successive period of stimulation and plotting this against time a fatigue index was determined. Although, the muscles exhibited typical low frequency fatigue, they were actually less fatiguable compared to pre-exercise. Balvane and Thompson attributed this to a lower initial force and reduced contribution of type II muscle fibers to the post-exercise fatigue index due to predominantly type II fiber damage during exercise.

Kroon and Naeije (1991) investigated the ability of the human biceps to sustain a submaximal isometric contraction following eccentric exercise. They found that the endurance time for a contraction at 40% of pre-exercise MVC was significantly shorter for up to 7 days after exercise. They also reported a higher initial RMS EMG and rate of shift of the EMG mean power frequency (MPF) over the first 30 seconds of the sustained contraction.

Komi and Viitasalo (1977) were concerned with neuromuscular performance. They measured IEMG and average motor unit potential (AMUP) of the lower limb extensors during submaximal isometric contractions and recorded force vs. time while subjects produced maximum force as quickly as possible. Two days following repeated maximal eccentric contractions, they reported greater IEMG and larger AMUP for a 250 N load, compared to pre-exercise. In addition, the maximum force and the rate of rise of force was lower compared to pre-exercise. Komi and Viitasalo speculated that the reduced neuromuscular performance, manifested as greater neural activation for a given load, was due to the soreness in the exercised muscle. Berry et al. (1990) more recently reported increased RMS EMG in the quadriceps muscles 1, 12, 24 and

48 hours after eccentric exercise while subjects held the leg slightly off the ground. However, there was no correlation between muscle soreness and increased EMG.

Bulbulian and Bowles (1992) provided further insight into the effects of eccentric exercise on the neuromuscular system. They evaluated motoneuron excitability by determining the Hoffman reflex (H-wave) expressed as a ratio of the maximal electrically stimulated muscle action potential (M-wave) from the gastrocnemius muscle. They reported a reduction of 24.7% immediately following downhill running for 20 minutes, compared to baseline. This reduction, however, was not evident 24 hours later. The investigators suggested that the reduction immediately following the exercise was indicative of selective type IIb motor unit recruitment and injury during eccentric exercise produced afferent feedback to the CNS, which inhibited type I motor units and reduced the H/M ratio. They also suggested that there was no apparent central mechanism which played a role in DOMS, as evidenced by the normal H/M ratios 24 hours after exercise. Instead, Bulbulian and Bowles speculated that soreness might be associated with muscle spindle sensitivity, and suggested that this could be investigated by evaluation of the stretch reflex in voluntarily activated muscles, rather than muscles activated by electrical stimulation.

Others have speculated that there are changes in the sensitivity of stretch receptors following eccentric exercise. Jones et al. (1987) proposed that increased mechanical sensitivity to stretch may give rise to pain as a consequence of muscle stiffness. Korr (1976) suggested that the restricted

motion often associated with post exercise muscle soreness might be the result of resetting of the myotatic reflex, but no evidence was provided.

Miles et al. (1993) studied the effects of eccentric exercise on neuromuscular control of maximal velocity and goal directed elbow flexion. They reported decreased peak velocity 24 and 48 hours after exercise. Movement time, time to peak velocity, time from onset of agonist EMG to movement onset and the time between agonist and antagonist EMG bursts all increased as a result of eccentric exercise.

More recently, Saxton et al. (1994a, 1995) examined tremor, ability to match force and joint position sense following maximum voluntary eccentric exercise by the forearm flexors. They reported increased tremor amplitude until 48 hours after exercise. Impaired proprioceptive function was also demonstrated as perception of force and joint position were significantly impaired for up to 5 days after exercise. Subjects consistently overestimated level of force and underestimated elbow joint position. These findings suggest significant impairment of neuromuscular function and warrant further investigation of neuromuscular control following muscle injury.

Adaptation

The fact that athletes do not become dysfunctional as a result of long term training suggests that a muscle which is damaged during exercise has the capacity for repair and adaptation. The effect of a single bout of eccentric exercise on the response to a subsequent bout of exercise has provided some insight into these adaptations. Clarkson et al. (1992) found that after one bout of

high-force eccentric exercise the forearm flexor muscles were more resistant to damage, evaluated by changes in MVC, soreness, CK levels, swelling and relaxed joint angle, when the same exercise was performed as soon as 5 days later. Even though the muscles were not fully recovered after 5 days, they still showed less damage from the second bout of exercise, as well as an improvement in recovery time. This effect has also been shown following several repeated bouts of exercise. Newham et al. (1987) conducted a study in which subjects performed maximal eccentric contractions of the elbow flexors on three occasions, two weeks apart. They reported fewer complaints and faster recovery with regard to muscle pain and stiffness, respectively, after the second and third bouts compared to the first.

Several hypotheses have been put forward as possible explanations for the adaptations following an initial bout of eccentric exercise. These include a change in the pattern of motor unit recruitment, such that susceptible fibers are not recruited or that more fibers are recruited during the subsequent bout of activity. Studies by Kuipers et al. (1985) found resistance to subsequent injury three weeks after the initial exercise bout. They concluded that because the effect was present after such a long time, the adaptation must have taken place at a neuromuscular level. This was supported by significantly lower mean heart rate, mean O_2 consumption and mean blood lactate concentrations in the second eccentric trial compared to the first. Clarkson et al. (1992) suggested that an altered recruitment pattern of motor units could explain the repeated bout effect. During eccentric muscle actions, over an entire range of motion

different motor units are recruited at different points in time. They suggested that an adaptation in the motor unit recruitment pattern over the range of motion such that less force was produced by the active fibers at any point in time, would reduce the likelihood of an injury to the fibers. They also concurred, that such a long lasting effect reflects a neurological influence. Evidence for a peripheral origin of these neurological changes, rather than a central one, comes from a study in which subjects performed eccentric exercise with one leg, then subsequent exercise with either the same limb or the contralateral limb. Resistance to further injury was only found on the ipsilateral side (Clarkson et al. 1987).

It has also been suggested that the mechanism responsible for the repeated bout effect may be associated with preferential damage to a particular group of susceptible fibers. These fibers were thought to be more susceptible because they were weaker and at the end of their cycle of growth and replacement. Thus, there would be a decrement in force after the first bout of exercise during which these fibers were damaged, which would be absent after subsequent bouts of exercise. In a study by Newham et al. (1987) it was shown that the repeated bout effect was present even after the performance of maximal contractions. Since maximal contractions required the activation of all motor units, they suggested there would be no possibility of change in the extent or order of recruitment. Instead they suggested the effect might be due to the removal of contractile material that was susceptible to damage.

Others have suggested that the resistance to damage during subsequent bouts of exercise is due to changes that occurred in the connective tissue. Ebbling and Clarkson (1990) performed a study in which subjects repeated the exercise bout 5 days after the initial session. Less damage was produced with the second bout and recovery times were faster, even though the muscles still exhibited indications of muscle damage from the first bout. They suggested that complete recovery and repair, as indicated by measures of isometric strength and ability to move, was not required for subsequent resistance to damage. They also speculated that changes must have occurred in the connective tissue rather than the contractile components for this to be possible.

Training effects from long term repeated eccentric exercise have also been reported. For example, individuals who are involved in repeated training report gradual disappearance of feelings of soreness. These observations are further supported by studies on strength development, work capacity and other indicators of performance. Evidence for long term changes comes from the reported decreased oxygen consumption required to perform eccentric contractions after training (Klausen and Knuttgen 1971; Davies and Barnes 1972). Strength training studies have compared isometric, concentric and eccentric protocols and reported the greatest improvement in isometric strength after training with eccentric exercise (Jones and Rutherford 1987b; Pearson and Costill 1988; Pousson et al. 1990). Pearson and Costill reported a greater increase in fiber size in leg muscles exercised with an eccentric component. Fridén et al. (1983b) reported improvements of 375% in eccentric work capacity

following 8 weeks of eccentric training. An increased number of type IIC fibers was also reported in this study. These authors suggested that training with maximal eccentric work over 8 weeks caused a preferential recruitment of type IIB muscle fibers, which in turn caused reorganization of the contractile apparatus and improved coordination of motor unit recruitment to perform maximally.

Other changes considered less desirable, or maladaptations, have also been documented. Anterior knee pain in runners is thought to be cause by a maladaptation to repetitive eccentric muscle actions. The deceleratory eccentric muscle action serves an important role in running and a deficiency in this could lead to other more serious knee joint problems. A specific force deficit during isokinetic testing of the eccentric strength capabilities in runners experiencing knee pain supports this suggestion (Bennett and Stauber 1986). Others have hypothesized that with running the muscles of the lower extremities become less compliant, which leads to changes in the lower extremity biomechanics and gait pattern and perhaps increased forces at the joints (Wilcox 1989; Hamill et al. 1990; Harris et al. 1990; Hone et al. 1990). Chronic eccentric training has also been implicated in other overuse type musculoskelatal disorders such as tendonitis (Curwin and Stanish 1984).

The commonality of exercise-induced muscle injury and the fact that eccentric exercise is often used for training and in rehabilitation illustrates the need for a greater understanding by researchers and clinicians. However, the mechanisms and full implications of such exercise remains unclear.

CHAPTER TWO - PURPOSE

The purpose of this thesis is to study the mechanical and neurophysiological changes following eccentric exercise-induced muscle injury. These include changes in muscle mechanical properties, joint mechanical properties, reflex function, recruitment and activation patterns of synergist and antagonist muscles that affect motor performance.

Mechanical stiffness, particularly the component associated with active muscle force is recognized as being important in normal control of posture and movement in humans (Hogan 1984; Kearney and Hunter 1990; DeSerres and Milner 1991; Bennett, 1993; Milner and Cloutier 1993; Milner et al. 1995). The sensation of increased muscle stiffness has been associated with exercise-induced muscle injury. However, it is unclear whether this sensation of stiffness is in any way related to mechanical stiffness. The mechanical stiffness of a joint is defined as the ratio of the change in joint torque to change in joint angle. It is composed of three components which have different origins and can vary independently of one another. They are the elastic properties of non-contractile tissue (passive stiffness), the elastic properties of attached cross-bridges (intrinsic stiffness) and reflex activation of a muscle following a change in length (reflex stiffness) (Nichols and Houk 1976; Hoffer and Andreasson 1981; Sinkjaer et al. 1988; Carter et al. 1990; Toft et al. 1991).

Quantification of muscle stiffness following exercise-induced injury has predominantly involved evaluation of resting joint angle or amount of force required to move a joint through its full range (Howell et al. 1985; Jones et al. 1987a; Stauber et al. 1990; Clarkson 1992). Howell et al.(1993) quantified the stiffness of relaxed muscle (passive stiffness) by determining the relationship

between static torque and elbow angle from the middle to the end of the range of motion. This procedure revealed an increase in passive stiffness which was quantified as an average value over the range of motion tested. Because the effects have tended to be greatest at the extremes of the range of motion, it is not clear whether the stiffness of relaxed muscle also increases significantly in the mid-range of motion where the joint is most frequently positioned during normal activities. Based on previous work, the following hypothesis was formulated.

General Hypothesis 1:

Passive joint stiffness measured in the mid-range of joint motion will increase as a result of eccentric exercise.

Warren et al. (1993b), in an attempt to separate the contribution of contractile elements and activation to the force deficit following eccentric exercise, calculated total stiffness of the rat soleus muscle stimulated to tetanus. They showed that there was a similar pattern of decline in tension and muscle stiffness. This would suggest that the force deficit is due to fewer attached cross-bridges. However, the joint stiffness of a voluntarily activated muscle has not been measured after exercise-induced muscle injury to corroborate this finding under conditions where motor units are recruited in their normal order or at force levels below MVC. From histological evidence, it appears that type II muscle fibers are more susceptible to injury than type I fibers during eccentric exercise (Fridén et al. 1983a; McCully and Faulkner et al. 1986; Stauber et al. 1990). If type I muscle fibers are much less affected by eccentric exercise than type II muscle fibers, it is also likely that intrinsic muscle stiffness at low levels of muscle force will not change as a result of eccentric exercise.

General Hypothesis 2:

Intrinsic joint stiffness measured during voluntary contraction at low torque levels will not change as a result of eccentric exercise.

Bulbian and Bowles (1992) determined the H-wave from the gastrocnemius muscle and expressed it as a ratio of the M-wave. They reported a decline of 24.6% in this ratio immediately following a bout of downhill running, which by 24 hours post-exercise was back to normal. They suggested that the mechanical disruption of type II muscle fibers produced afferent feedback to the CNS, which reduced motoneuron excitability. No indicators of injury to the gastrocnemius were provided in this study. The recovery in the H/M ratio 24 hours after the exercise bout may indicate that there was minimal injury to the gastrocnemius muscle, and as such would not provide a clear indication of the effects of muscle injury on motoneuron excitability. As the H-reflex bypasses the spindle apparatus, this study provided no means of judging whether there were changes in spindle sensitivity as a result of exercise-induced muscle injury. However, the findings do indicate that reflex pathways may be influenced by eccentric exercise. Korr (1976) suggested that resetting of the myotatic reflex in response to muscle soreness may explain the restriction of motion associated with exercise-induced muscle injury. However, this theory was not supported by any experimental evidence. There has been no previous study of the effects of exercise-induced muscle injury on reflex activation in response to muscle stretch. Examination of the stretch reflex could reveal effects of exercise-induced muscle injury on afferent feedback mechanisms and pathways which may exert central influences that affect normal control of posture and movement.

General Hypothesis 3:

Following exercise-induced muscle injury, there will be a decrease in reflex excitation in response to muscle stretch only in situations where activation of large motor units is required. Consequently, there will be no change in short latency reflex EMG or reflex stiffness in tasks requiring low levels of torque, but there will be a reduction in both short latency reflex EMG and reflex stiffness in tasks requiring high torque.

Various hypotheses have been advanced to explain the selectivity of injury to large muscle fibers but the implications of the injury on the ability to move and perform functionally have received minimal attention. It is clear that exercise-induced muscle injury affects force-producing capabilities, but thus far this has been predominantly evaluated by testing maximal strength. Fridén et al. (1983a) tested maximum knee extensor torque at different angular velocities and reported that the greatest decrement in force producing capability occurred at the highest angular velocity. They speculated that the velocity related decrement was due to selective injury of type II muscle fibers. There have been few studies which have evaluated the effects of muscle injury on the ability to perform tasks requiring sub-maximal activation. Endurance time (Kroon and Naeije 1991) and EMG activity required for sub-maximal loads (Komi and Viitasalo 1977; Newham et al. 1983; Berry et al. 1990) have been examined, but this has provided little indication of the whether motor function is impaired. Since most activities throughout the course of daily living require less than maximal activation of muscles it is more appropriate to investigate the effects of muscle injury on the ability to perform tasks at sub-maximal levels of activation than at maximal activation.

General Hypothesis 4:

Performance in experiments designed to quantify motor function during sub-maximal muscle activation will be impaired following exercise-induced injury of the muscle, but only if the task requires the recruitment of large motor units.

Several studies have reported an increased emg:force ratio following eccentric exercise (Komi and Viitasalo 1977; Berry et al. 1990; Kroon and Naeije 1991). This ratio suggests that more neural activity is required to produce a given force after eccentric exercise. This may be the result of injury to large muscle fibers. If these fibers cannot generate force because they are damaged additional motor units may have to be recruited to compensate. Some researchers have suggested that there is an altered motor unit recruitment pattern following the initial bout of eccentric exercise which explains the resistance to injury during subsequent exercise bouts (Kuipers et al. 1985; Clarkson et al. 1987, 1992). So far this hypothesis has not been directly tested.

General Hypothesis 5:

Eccentric exercise will result in injury to large muscle fibers rendering them less able to produce force. At force levels where large motor units are normally required, the reduced contribution of injured large muscle fibers will be compensated by increased activation of slow motor units.

There are no reports of the effects of exercise-induced muscle injury on the action of an uninjured synergist muscle. Altered afferent feedback from the injured muscle could influence α-motoneuron excitability of a close synergist through heteronymous reflex actions. Since synergist muscles are normally activated together, injury to one muscle of the synergy could influence the

activation of an uninjured muscle. In particular, if the injured muscle is inhibited, the synergist could also be inhibited.

General Hypothesis 6:

Reflex inhibition of an uninjured synergist muscle will occur whenever that muscle is coactivated with a muscle injured by eccentric exercise, at levels high enough to recruit injured muscle fibers.

Changes in the pattern of activation of injured synergist muscles has been reported following exercise-induced muscle injury. Hasson et al. (1993) reported that following a bout of eccentric exercise involving the quadriceps muscle, the muscle which was reported to be the most sore showed the greatest reduction in RMS EMG during concentric and eccentric knee extension at 60^0/sec, while the muscle reported to be least sore showed no change. Crenshaw et al. (1994) speculated that there could be a shift in the activation of the quadriceps muscles in such a way that the muscle which was the most sore would make a smaller contribution to joint torque following injury, but the total joint torque would remain the same. However, they did not provide support for this theory with experimental data. In contrast, the findings of Newham et al. (1983) showed no significant change in the ratio of contribution for the three knee extensor muscles to the total recorded quadriceps activity. However, they collected EMG data from only 2 of 4 subjects, and reported a decline in force only at 2 and 10 minutes following exercise. Full recovery of force was reported 24 hours after the exercise bout suggesting minimal injury, if any, as a result of the exercise bout. For this reason their findings do not rule out the possibility of changes in recruitment patterns of injured synergists. Most studies of exercise-induced muscle injury have grouped the synergist muscles acting about a joint into a single unit. Therefore, it has not

been possible to determine whether there are changes in the relative force contributions or in the recruitment patterns of the individual muscles.

General Hypothesis 7:

Following exercise-induced injury to a group of synergist muscles, the pattern of activation of the injured muscles will not change. Activation of all injured muscles will be affected similarly.

There have also been no reports of the effects of exercise-induced muscle injury on the actions of uninjured antagonist muscles. Increased afferent activity from an injured muscle which inhibits synergists might be expected to reduce the reciprocal inhibition of antagonists. This could increase the amount of activity seen in the antagonist muscles whenever the injured muscle is activated.

General Hypothesis 8:

Exercise-induced injury will result in increased cocontraction of antagonist muscles whenever the injured muscle is reciprocally activated at levels high enough to recruit injured motor units.

The effect of pain on other indicators of muscle function has been of particular interest. It has been suggested that pain may inhibit the ability to generate force (Smith 1991) which may result in greater neural activation for a given load (Komi and Viitasalo 1977). However, the correlation between muscle soreness and increased EMG has not supported this theory (Berry et al. 1990). Furthermore, there has been a poor correlation between the time course of decrement in maximal force producing capabilities and muscle soreness following exercise-induced muscle injury (Clarkson and Tremblay 1988; Newham 1988a; Rondenburg et al. 1993). Most functional activities of daily living require less than maximal activation of muscles. Clinicians are most concerned with the

ability to perform tasks throughout the day rather than the ability to produce maximal force. No studies have investigated motor performance, maximal force producing capability and soreness, during recovery from injury. It is important to understand how these aspects of injury relate to one another to know whether to focus treatment on reducing pain or on maintaining or improving the ability to produce force so as to promote recovery for optimal performance of everyday tasks.

General Hypothesis 9:

Performance in tests designed to quantify motor function will be impaired following exercise-induced muscle injury. Decrements in performance will be better correlated with decrements in maximal force than with ratings of muscle soreness.

Hypotheses 1, 2 and 3 are related to the changes in mechanical parameters that occur following exercise-induced muscle injury, while hypotheses 3, 5, 6, 7 and 8 focus on the neurophysiological changes that accompany injury. Finally, hypotheses 4 and 9 deal with the changes in motor performance and ability to function following exercise-induced muscle injury.

Two comprehensive studies were conducted to test the hypotheses proposed above. In the first study, a single muscle was examined while operating in isolation. Subjects were tested pre-injury and 24 hours after eccentric exercise. Joint stiffness was measured under passive and active conditions for low and high levels of muscle activation. In the case of low levels of activation, it was decomposed into separate components. Reflex responses at low and high torque were also evaluated. Maximal M-wave was measured pre-, post- and 24 hours after exercise-induced muscle injury. A variety of functional tests including the ability to produce maximal force, maximal joint velocity, maximal joint stability,

ability to sustain constant torque and ability to track a changing torque were conducted. The effect of the muscle injury on a uninjured synergist muscle was also evaluated. This involved evaluating functional tests in which activation of both the injured muscle and its synergist was required.

In the second study subjects were repeatedly tested over a period of 11 days, once prior to and four times following a bout of eccentric exercise utilizing a group of synergist muscles. The purpose of this study was to investigate the effects of exercise-induced muscle injury on individual synergist and antagonist muscles. The effect of the injury on passive and active joint stiffness was examined. Passive range of motion and muscle soreness were also assessed. A variety of functional tests, including the ability to produce maximal force, maximum joint velocity, maximum joint stability, ability to sustain constant torque, and ability to track a changing torque were conducted. Both the injured muscles and their antagonists were tested throughout the period of recovery.

Comprehensive knowledge of the consequences of exercise-induced muscle injury and understanding of the underlying mechanisms is important for researchers and clinicians in interpreting adaptations to injury, some of which may be positive and should be reinforced or others that are negative and should be prevented because they may lead to other musculoskeletal disorders. The results and discussions presented in this thesis will attempt to address this need, as well as suggest directions for future investigations.

CHAPTER THREE - EXERCISE-INDUCED INJURY OF THE FIRST DORSAL INTEROSSEUS MUSCLE

Introduction

To date, studies of exercised-induced injury in humans have evaluated groups of muscles acting about a joint. This has made it difficult to determine the amount of damage that has occurred in any one muscle. Furthermore, careful analysis of changes in recruitment of motor units within an individual muscle or within synergistic uninjured muscles has not been performed. Often several muscles are activated by the central nervous system to perform a particular movement. Injury to one of these muscles could influence the recruitment or ability of other muscles to function. To investigate these ideas, specific injury to a single muscle and simultaneous collection of EMG activity from the injured muscle and surrounding musculature is necessary.

The purpose of this study was to investigate an exercise-induced muscle injury specific to a single muscle acting about a single joint. The first dorsal interosseus (FDI) muscle is the primary muscle acting to abduct the index finger. It is also one of several muscles that flexes the metacarpophalangeal (MP) joint. The FDI muscle was chosen for this study because it permitted quantification and analysis of the mechanical and neurophysiological changes accompanying exercise-induced injury to a single muscle. This analysis included separation of joint stiffness into its constituents components, similar to the work of Carter et al. (1990) for an uninjured muscle. By inducing injury of the FDI muscle, changes

in central nervous control, including reflex responses and recruitment strategies, were investigated. Comprehensive testing paradigms were designed to evaluate functional capabilities following exercise-induced muscle injury. In addition, to evaluate the effects of the injured FDI muscle on an uninjured synergist muscle, EMG was collected from the flexor digitorum superficialis (FDS) muscle and performance outcomes were determined for tasks requiring MP flexion.

The following hypotheses from those developed in Chapter 2 were addressed in this study:

1. Passive MP joint stiffness measured in the mid-range of MP joint motion will increase as a result of exercise-induced injury to the FDI muscle.
2. Intrinsic MP joint stiffness at low MP abduction torque will not change as a result of exercise-induced injury to the FDI muscle.
3. Following exercise-induced injury to the FDI muscle, there will be a decrease in reflex excitation in the response to muscle stretch, only in situations where activation of large motor units is required. Consequently, there will be no change in short latency EMG or reflex stiffness in tasks requiring low levels of MP abduction torque, but there will be a reduction in both short latency reflex EMG and reflex stiffness in tasks requiring high MP abduction torque.
4. Performance in tasks designed to quantify motor function during sub-maximal FDI activation will be impaired following exercise-induced injury to the FDI muscle, but only if the tasks require the recruitment of large motor units.

5. Eccentric exercise of the FDI muscle will result in injury to large muscle fibers rendering them less able to produce force. At force levels where large motor units are normally required, the reduced contribution of the injured large muscle fibers will be compensated by increased activation of small motor units.
6. Inhibition of the uninjured FDS muscle will occur whenever it is coactivated with the injured FDI muscle at levels high enough to recruit injured FDI muscle fibers.

Materials and Methods

Subjects

Ten normal male subjects participated in this study (age range 22 - 31). Nine of the subjects were right-handed, the other left-handed. None of the subjects reported any previous history of neuromuscular disorders. Each gave written informed consent to participate prior to the experiment (Appendix A). None had previously participated in any studies involving eccentric exercise. Most were familiar with the testing apparatus. Subjects were asked not to participate in any weight-training activities, specifically for the upper extremities, for the duration of the study. The experiment was approved by the University Research Ethics Review Committee at Simon Fraser University (Appendix A).

General Design

The FDI was chosen for this experiment for several reasons: 1) it is the primary muscle to abduct the index finger and one of several muscles that

flexes the MP joint, 2) its muscle belly is distinct and it is easy to record EMG with surface electrodes without interference from neighboring muscles, 3) its simple mechanical action makes it easy to record the abduction forces it exerts, 4) it is innervated by the ulnar nerve which is easy to activate by transcutaneous electrical stimulation, and 5) it has approximately 120 motor units (Feinstein et al. 1955) and a near balanced composition of type I (57%) and type II fibers (Johnson et al. 1973).

Subjects were tested on two separate occasions, previous to an exercise-induced muscle injury (pre), and 24 hours after muscle injury (24 hours). To ensure reproducible placement of EMG and stimulating electrodes on both days, an indelible pen was used to mark the skin. To ensure accurate positioning in the testing apparatus, the subject's forearm and hand were supported and strapped in a molded elastoplast splint, which was then clamped to the apparatus. The index finger was also splinted and clamped to the manipulandum to ensure identical positioning and to avoid any flexion-extension movement at the proximal interphalangeal (PIP) or distal phalangeal (DIP) joints or movement at the MP joint in an off-axis direction. Subjects were first positioned to test MP abduction, followed by MP flexion. All testing was performed on the left hand.

Apparatus

All testing, except for maximal M-wave, was conducted on a manipulandum which allowed positioning of the hand such that the axis of rotation was directly through the MP joint and movement occurred only in the

adduction-abduction, or flexion-extension direction. A torque motor (PMI U16M4) was used to generate loads which were computer controlled. The motor was used to produce offset torque during an initial holding period, mechanical behavior equivalent to a negative spring, and a servo-controlled ramp and hold angular displacement. The maximum torque that could be produced by the torque motor was 5 Nm.

Position and velocity were measured by a potentiometer and tachometer, respectively. The torque was measured by a linear strain gauge mounted on a cylinder, coupling the motor shaft to the manipulandum (Figure 3.1). EMG for tests performed with the manipulandum was recorded using active, bipolar, stainless steel, surface electrodes (Liberty Mutual MYO 111) with electrode contacts 4 mm in diameter and 13 mm apart. Muscle stimulation during joint stiffness trials was produced by a constant-current stimulator (Mikrofes) using a current pulse width of 0.16 ms and frequency of 43.5 Hz. Surface electrodes (Dermatrode EMG, North American Distributors), 1 cm in diameter were placed 2 cm apart over the muscle belly. Amplitude was adjusted to produce the desired torque level.

Transcutaneous electrical stimulation for determination of maximum M-wave was produced with a stimulator (Grass S88) fed through an isolation unit (Grass SIU5), using a pulse duration of 0.30 ms and frequency of 0.1 Hz. Stimulus intensity was adjusted to supramaximal levels to ensure maximal M-wave amplitude. The stimulating electrode consisted of saline soaked stimulating pads with a separation of 2 cm center to center. It was placed such

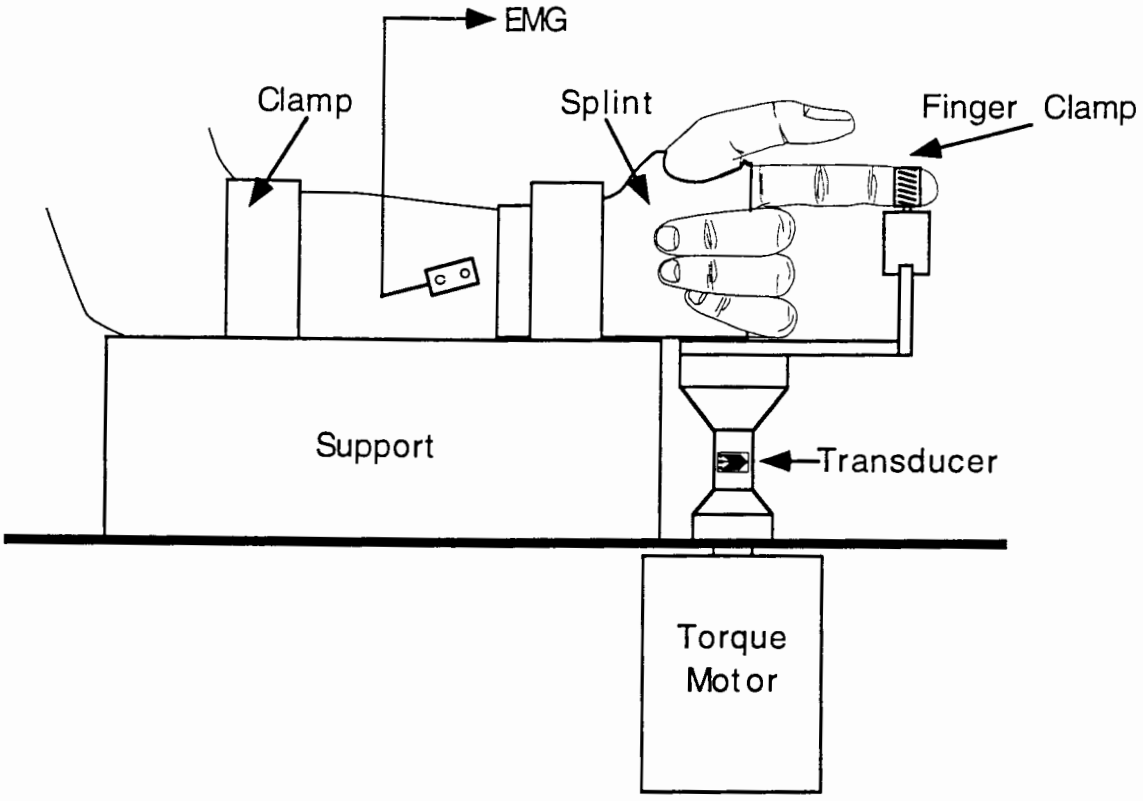

Figure 3.1: Experimental setup for MP flexion.

The left wrist was placed in a molded elastoplast splint and clamped to the manipulandum. A torque transducer measured motor shaft torque. Angular position was measured with a potentiometer in line with the torque motor shaft.

that the cathode was over the ulnar nerve approximately 10 cm proximal to the wrist. Surface EMG was recorded with a pair of Ag-AgCl discs, 9 mm in diameter, placed over the FDI muscle belly.

Recording

Manipulandum Tests

EMG activity was recorded from three muscles: first dorsal interosseus (FDI), flexor digitorum superficialis (FDS) and extensor digitorum indicis (EDI). Optimal placement of the electrodes for pre- and 24 hours after injury was determined by observing the EMG activity during brisk test movements. These included index finger movements of MP abduction, MP flexion, and MP extension. Signals were bandpass filtered from 45 - 550 Hz, amplified, digitized at 2 KHz and stored on disk for later analysis. Position, velocity and torque of the index finger were simultaneously recorded, amplified and digitized at 2 KHz and stored on disk.

Maximal M-Wave

EMG activity was preamplified (Grass P15, high pass at 30 Hz and low pass at 3 KHz) then further directed to an AC conditioning amplifier (15 Hz high pass and 30 KHz low pass). The resultant data were stored on a Vetter PCM recorder for later analysis. Calibration signals were recorded for each subject following collection of M-wave data.

Exercise Protocol

The exercise protocol was performed on the manipulandum. Subjects performed two sets of 50 maximal eccentric contractions with the FDI muscle in adduction-abduction. During each repetition, subjects began by performing a maximal isometric contraction, then resisted maximally as a 20^0 stretch was imposed in the adduction direction by the manipulandum over a period of 3 seconds. There was a 3 second rest between each repetition and 5 minutes between each set. Subjects were provided with visual feedback of their torque level and encouraged to produce maximum effort.

Testing Procedures

Following thorough cleaning with alcohol and mild abrasion of the skin, EMG electrodes were placed over the designated muscles. Each subject was seated comfortably in a chair with the left forearm and hand splinted and supported in a comfortable position. For experiments performed in adduction-abduction, the forearm and wrist were orientated in pronation and immobilized. The index finger was positioned in the manipulandum and secured in such a way as to prevent MP flexion with the axis of rotation about the MP joint directly over the shaft of the torque motor (Figure 3.2). For experiments performed during MP flexion, the forearm was orientated midway between pronation and supination, the wrist, hand and three remaining fingers were splinted to allow only movement of the index finger in flexion - extension. The hand was positioned such that the axis of rotation of the MP joint was directly over the shaft of the motor. The index finger was clamped to the manipulandum at the

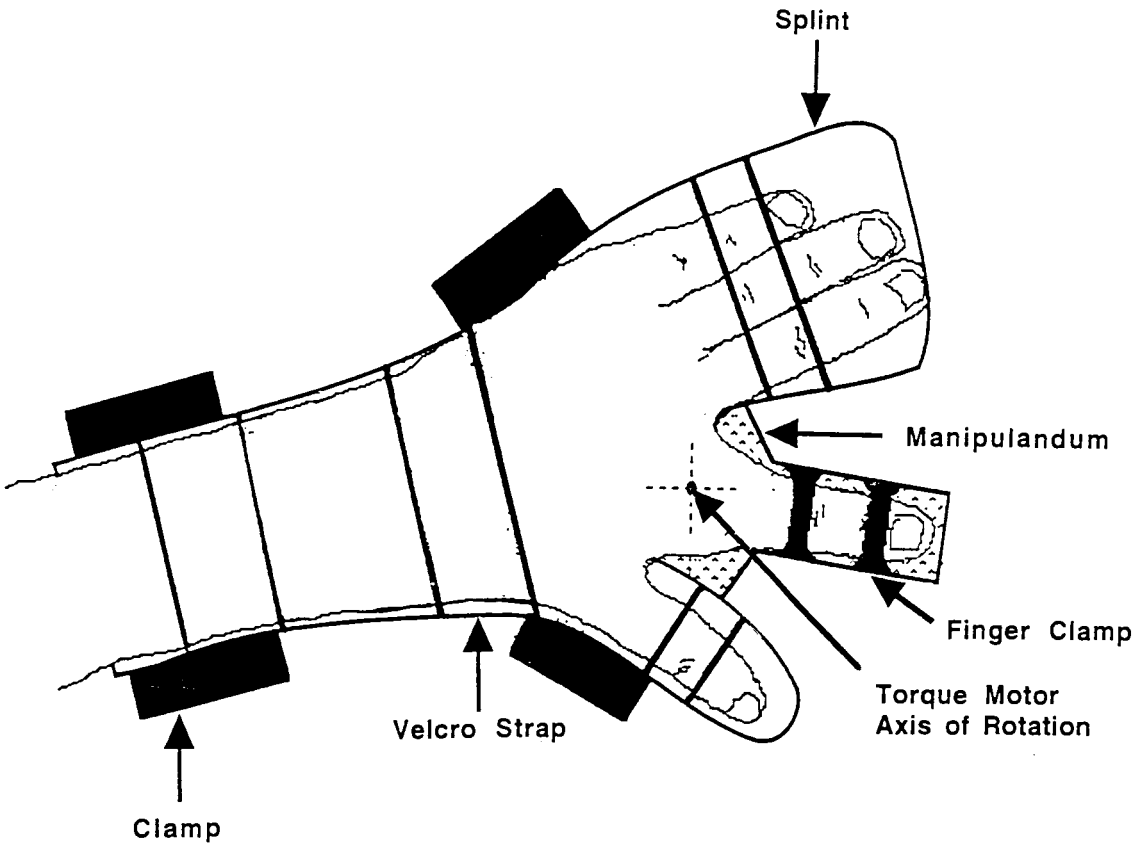

Figure 3.2: Schematic view of the experimental arrangement for MP abduction.

The left forearm and hand were placed in a elastoplast splint. The index finger was fitted with a splint to prevent flexion of the interphalangeal joints and secured to the manipulandum. The axis of rotation about the MP joint was directly over the shaft of the manipulandum.

distal phalanx. The finger was taped to prevent any movement of the PIP and DIP joints (Figure 3.1).

Most subjects were familiar with the tasks performed on the manipulandum. Those subjects who were not familiar with a particular task were given the opportunity to perform practice trials prior to data collection. To avoid fatigue during the testing session, subjects were given at least 30 seconds between each trial and up to 5 minutes between individual tasks.

The experiments were performed in the following sequence:

MP Abduction

1. *Stimulated joint stiffness.* High negative position feedback gain to the torque motor was used to hold the manipulandum at the neutral position of the finger (determined for each subject pre-injury and used for subsequent tasks and for tests performed 24 hours later). Subjects controlled the stimulator to slowly increase contraction of the FDI muscle. The amount of torque produced by the contraction was displayed as a cursor on the computer screen. The intensity of the stimulation was gradually increased until the level of torque produced was within the target window on the computer screen, equal to 15% of MP abduction MVC pre-injury ± 0.75%. After having reached the desired torque level and having remained within the target window for a period of time, between 1 - 2 seconds, the experimenter triggered a ramp angular displacement of 3° with a duration of 30 ms which stretched the FDI muscle. Subjects were instructed to relax completely throughout the entire

trial. The stimulator was turned off approximately 1 - 2 seconds after completion of the displacement. Adequate rest was provided between trials. Ten trials were saved on each day of testing. Evaluation of stimulated joint stiffness enabled decomposition of the total active joint stiffness into intrinsic and reflex components.

2. *Passive joint stiffness.* The manipulandum was set at the neutral position of the finger and the subject was instructed to relax completely. Once the subject was relaxed, the experimenter triggered a ramp displacement, as above. Any trials in which there was evidence of EMG activity or torque prior to the displacement were not accepted. Ten acceptable trials were saved.
3. *Active joint stiffness.* The manipulandum was set at the neutral position of the finger and the target window on the computer screen was set to one of two torque levels, 15% of MP abduction MVC pre-injury ± 0.75% or 65% of MP abduction MVC pre-injury ± 3.25%. The subject was instructed to move a cursor representing torque into the target window on the computer screen. Once the subject had held the cursor in the target window for a random period of time ranging from 1 - 2 seconds, the experimenter triggered a ramp displacement, as above. The subject was instructed not to respond to the displacement. Trials in which there was evidence of voluntary intervention were not accepted. Ten acceptable trials were saved.
4. *Maximum velocity.* A target window which represented the neutral position of the finger was displayed on the computer screen. The subject was instructed to move a cursor representing the position of the manipulandum into the

target window. Once in the target window for more than one second, the cursor changed colour and the subject was instructed to move the finger as rapidly as possible. The movement was stopped by mechanical stops placed at the end of the range of movement. The subject was encouraged to accelerate throughout the entire movement. The subject was provided with feedback of his performance following each trial. Five trials were saved. This experiment was performed to evaluate the ability to recruit motor units rapidly.

5. *Maximum instability.* In order to determine the limit of stability of the MP joint, the subject was required to move the manipulandum and hold it in a 1° target window at the neutral position of the finger while the torque motor produced a negative spring load, which pushed the manipulandum away from the neutral position in either direction. The limit of stability was determined by systematically increasing the negative stiffness of the load. A trial was considered successful if the manipulandum was brought into the target window within 20 seconds and held there for a least one second. If successful, the negative stiffness of the load was incremented by -0.01 Nm/deg for the next trial. The limit of stability was defined as the largest negative stiffness that the subject could successfully stabilize. This experiment was performed to evaluate the ability to stabilize the MP joint by cocontraction.
6. *Maximum voluntary contraction (MVC).* The manipulandum was locked at the neutral position of the finger and the subject performed a sustained (up to 3

seconds) maximal isometric contraction. There was a warm-up period prior to data collection which involved a series of submaximal contractions. Three trials at maximum effort were stored for later analysis.

7. *Torque tracking.* The manipulandum was locked at the neutral position of the finger and the subject was required to contract the FDI muscle isometrically to produce MP abduction torque. The target torque began at zero and increased to 65% of pre-injury MVC at a constant rate over a period of five seconds. The subject was provided with a display of his torque and the target torque on an oscilloscope. The target torque increased at a constant rate and the subject was instructed to match it. Five trials were collected. This experiment was performed to evaluate the ability to control torque output.
8. *Sustained contraction.* The manipulandum was locked at the neutral position of the finger and the subject was required to maintain a constant isometric MP abduction torque for 60 seconds, equal to 50% pre-injury MVC. The subject was given visual feedback of his torque and the target torque on the oscilloscope. Only one trial was performed. This experiment was performed to evaluate the ability to sustain torque output and the fatiguability of the injured muscle.

MP Flexion

After completion of the tasks for MP abduction, modifications in the experimental setup were made so that the following tasks for MP flexion could be performed:

9. *Maximum Voluntary Contraction (MVC).* The same protocol was used as described above, but now in MP flexion.
10. *Sustained Contraction.* The same protocol was used as described above, but now in MP flexion torque.
11. *Maximum Velocity.* The same protocol was used as described above, but now in MP flexion.

Maximal M-Wave.

Each subject was seated such that the arm was in a comfortable position supported in front of the body. Following thorough cleaning of the skin with alcohol and placement of the EMG electrodes, the location of the optimum stimulating site was determined by applying repeated stimulus pulses at several locations over the path of the ulnar nerve to elicit the largest compound action potential. The stimulus intensity was slowly increased until no further increase in M-wave amplitude observed. The maximal M-wave was recorded for 9 subjects prior to and 24 hours after eccentric exercise. In 7 subjects maximal M-wave was also recorded immediately after the exercise bout (post). Maximal M-wave was recorded to evaluate fatigue in the muscle after the exercise bout and to differentiate between the ability of the subject to voluntarily activate the muscle and the ability of the muscle to be activated.

Analysis

Joint Stiffness

To quantify joint stiffness, five of the ten recorded trials for each task were selected for further analysis. Trials were selected to ensure similar background torque levels and displacement amplitudes between tasks and days of testing. This was essential for comparison from day to day and to allow decomposition of the total stiffness into its respective components. EMG was rectified and the selected trials were then averaged for use in subsequent comparisons. From the averaged data, mean values of position and torque were calculated for the interval 0 - 125 ms before the onset of displacement and for the interval 100 - 200 ms after the onset of displacement. These intervals were chosen based on the observation that during these periods the torque and position were relatively constant. The joint stiffness was calculated as the ratio of the change in mean torque divided by the change in mean position.

The mean rectified EMG (MEMG) was calculated, using an interval 0 - 125 ms before the displacement as a measure of background activity. The reflex EMG was separated into phasic and tonic responses. The latency and duration of the phasic response was determined for each subject separately (approximately 50 ms - 120 ms following the onset of displacement). Tonic reflex EMG activity was calculated over the interval from 120 - 220 ms following the onset of the displacement. Background EMG was expressed as an absolute value and as a percentage of the EMG recorded during maximal MP abduction.

Reflex EMG was expressed as the percentage change with respect to background (pre-displacement) EMG.

The total stiffness at low torque (15% of pre-injury MP abduction MVC) was decomposed into its constituents as shown in Figure 3.3. The passive response has been shifted vertically for the purpose of illustration. The torque change in response to displacement of the joint was separated into passive and active components. The active component was further separated into intrinsic and reflex components. Little or no reflex activation of the muscles occurred when the subject was completely relaxed, i.e., during passive trials. It was assumed that this was also the case during trials in which the FDI was stimulated electrically. The change in active torque when the muscle was activated voluntarily or when it was activated by electrical stimulation was obtained by subtracting the change in passive torque. Stiffness was calculated by dividing the change in torque by the measured MP joint displacement. A distinction was made between the intrinsic stiffness during voluntary activation of the muscle and during electrical stimulation of the muscle. The former will be referred to as natural stiffness and the latter as stimulated intrinsic stiffness. The natural intrinsic stiffness was set equal to the average stiffness during the interval 40 - 70 ms after the onset of the displacement, i.e., prior to any reflex contribution to stiffness. The stimulated intrinsic stiffness was scaled to match the natural intrinsic stiffness and then subtracted from the active stiffness to obtain reflex stiffness.

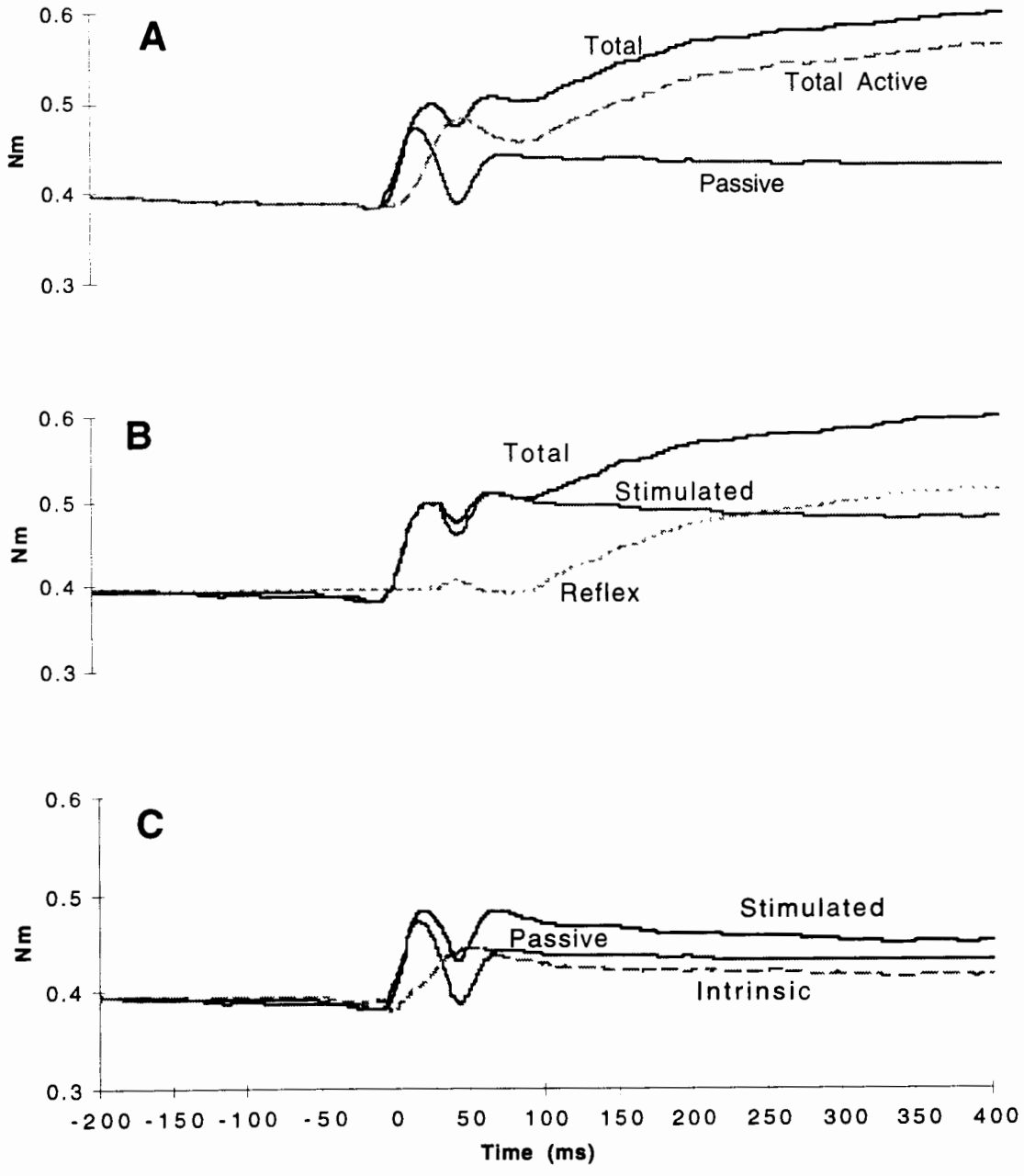

Figure 3.3: Decomposition of the total torque response at low torque.

A: The passive response has been shifted vertically to match the offset torque of the total response. When the passive response was subtracted from the total response the result was the total active response.
B: The stimulated response (scaled) was subtracted from the total response to obtain the reflex response.
C: Stimulated intrinsic response was obtained by subtracting the passive response from the stimulated response.

Maximum Velocity

Trials for each subject were analyzed individually to obtain maximum velocity, time of maximum velocity, time of onset of EMG activity with respect to movement onset, duration of EMG bursts, and IEMG of bursts. Movement onset was defined as the time when velocity exceeded 5 deg/s. The values for each of the above parameters were then averaged for the 5 selected trials to produce a mean value for each subject.

Maximum Instability

The highest negative load stiffness for which a subject successfully completed the task was taken as the limit of stability. MEMG was determined for each muscle in a 250 ms interval while the subject was stable in the target window.

Torque Tracking

The 5 trials for each subject were individually analyzed to determine median frequency (MF) and MEMG during each one second interval of the 5 seconds of increasing torque. For MF a 256 point spectrum was computed over an interval of 0.896 s, which allowed 3 spectra to be averaged. The variance about the best-fit straight line from beginning to end of the torque tracking was also determined (expressed in Nm^2). The mean parameters for the 5 trials were then calculated.

Sustained Contraction

During the 60 second contraction, the MF of the EMG signal for each muscle was computed at 5 second intervals from the average of nine 512 point spectra computed over an interval of 4.864 s. The slope of the MF plotted against time was computed, as well as MEMG for the first and last 5 s intervals.

MVC

The highest average torque over an interval of 125 ms and the corresponding MEMG over an interval of the same duration advanced by 40 ms were determined from the 3 trials for each subject for all three muscles. Previous studies of exercise-induced muscle injury have shown histological evidence of muscle fiber damage (Fridén et al. 1983a) or demonstrated inflammation in the muscle indicative of damage (MacIntyre et al. 1996) accompanied by a decline in maximal force. For these reasons, a decline in MVC 24 hours after the exercise bout was used as a criterion for muscle injury.

Maximal M - Wave

Data were reviewed and maximal responses were selected with SIGAVG (Cambridge Electronics, UK). Individual M-wave responses were calibrated using known voltage inputs. Peak to peak amplitude, time from first peak to second peak, and delay from stimulus to start of first peak (first evidence of deviation away from baseline) were determined (Figure 3.4).

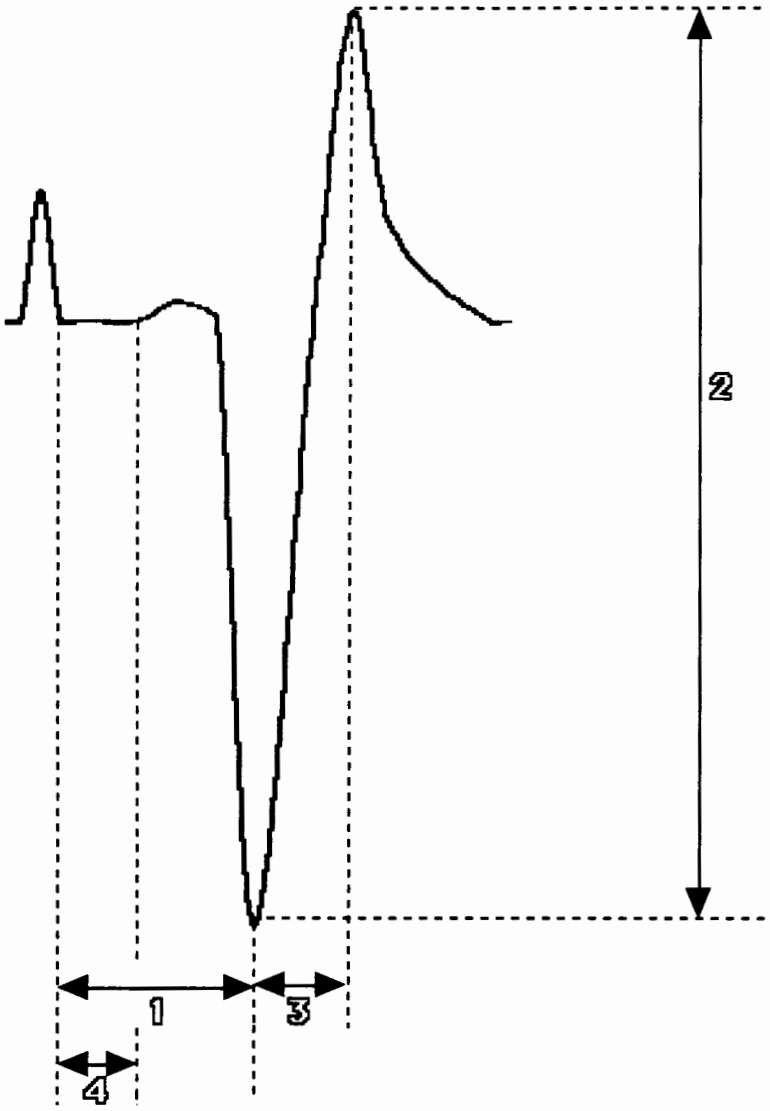

Figure 3.4: The components of maximal M-wave measured.

1: latency from the stimulus to the first M-wave peak
2: M-wave peak-to-peak amplitude
3: M-wave peak-to-peak duration
4. delay from stimulus to start of first peak

Experiment 2 was used to test hypothesis 1. Experiments 1, 2 and 3 were used to decompose total joint stiffness and to test hypotheses 2 and 3. Hypothesis 4 was tested with experiments 4, 5, 7 and 8. Experiments 3, 4, 5, 7 and 8 were used to test hypothesis 5. Hypothesis 6 was tested with experiments 9, 10 and 11.

Statistical Analysis

To evaluate the effects of exercise-induced muscle injury, parameter values pre- and 24 hours after injury were compared using a paired t-test. The test of statistical significance was set at 0.05.

Results

It was important to establish that the exercise protocol described in the methods produced injury to the FDI muscle. Although no direct indicators of muscle injury were evaluated, the severity of the exercise protocol (100 maximal eccentric contractions) was felt to be sufficient to produce muscle fiber damage. Other studies which have used less severe protocols have provided direct evidence of muscle injury. Stauber et al. (1990) had subjects perform 70 maximal eccentric muscle actions with the elbow flexors. Biopsies of the biceps brachii 48 hours after the exercise bout revealed myofiber and extracellular matrix disruption. In a study by Newham et al. (1987), very high levels of CK following a bout of 70 maximal eccentric contractions of the elbow flexors, was used as an index of the extent and the time course of the damage following the exercise bout. Jones et al. (1986) found evidence of an inflammatory response

with cellular infiltration in needle biopses taken from the biceps brachii after 20 maximal eccentric contractions.

Table 3.1 presents the means and standard deviations for MP abduction torque and the FDI MEMG pre- and 24 hours after injury. The pre-injury torque was reduced by 19% 24 hours after injury ($p<0.001$, $n=10$). The torque for each subject is shown in Figure 3.5. The FDI MEMG was also significantly reduced 24 hours after injury (75.2 μV pre-injury compared to 61.7 μV , $p<0.05$, $n=10$).

The compound action potentials evoked by supramaximal electrical stimulation of the ulnar nerve showed that the peak to peak amplitude and peak to peak duration decreased significantly from pre- to post-injury by 22.3% and 8.9%, respectively ($p<0.05$, $n=7$). Post-injury refers to immediately after the exercise bout. These findings suggest alterations of muscle membrane excitation and impulse propagation. For M-wave measured 24 hours after injury the peak to peak amplitude was found to have increased by 36% over pre-injury values ($p<0.01$, $n=9$). No significant differences were found in peak to peak duration or delay from stimulus to M-wave onset (Table 3.2). Maximal M-wave responses for one subject are plotted in Figure 3.6. M-wave potentiation suggests that fatigue that was not a factor 24 hours after the exercise bout.

Joint Stiffness

There was no significant difference in the passive joint stiffness or total joint stiffness during voluntary contraction at 15 % MVC 24 hours after exercise compared to pre-exercise (Table 3.3, Figures 3.7 and 3.8). Decomposition of

TABLE 3.1: MP abduction MVC.

	Torque (Nm)	FDI MEMG (μV)
Pre	2.15[a]	75.2
	(0.35)[b]	35.7
24 hours	1.74	61.7
	0.28	22.5
% change	19	
	18.6	
	p<0.001	p<0.05

[a] - mean

[b] - standard deviation

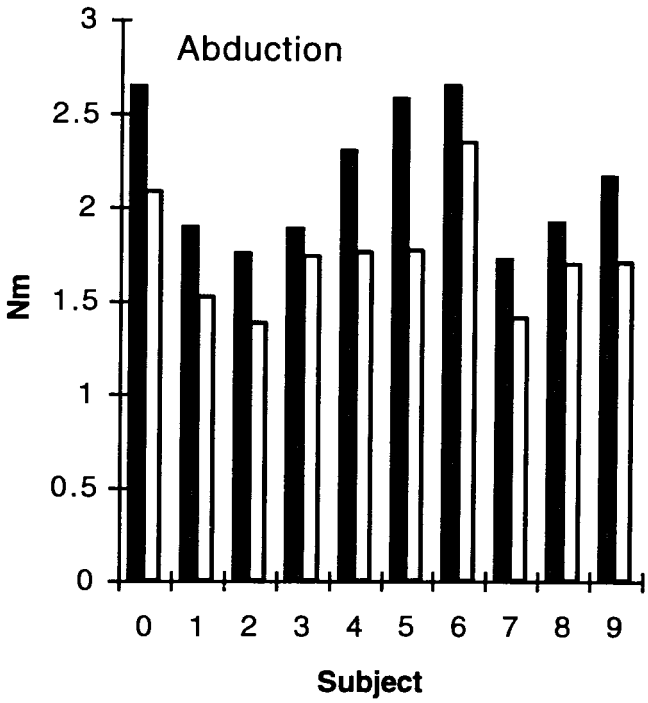

Figure 3.5: MP abduction torque for each subject.

pre-injury ⇒ shaded

24 hours after injury ⇒ clear

TABLE 3.2: Means and standard deviations of the measured M-wave parameters.

	Maximal M-Wave			
	Peak - Peak Amplitude (mV)	Peak - Peak Duration (ms)	Stimulus - Peak Duration (ms)	Delay (ms)
Pre	19.14[a]	4.47	7.89	3.09
(n=9)	(4.38)[b]	0.38	0.28	0.39
Post	14.87*	4.07*	7.54	2.96
(n=7)	4.09	0.55	0.65	0.38
24 hrs	26.03**	4.19	7.61	3.3
(n=9)	4.06	0.63	0.83	0.54
	* p<0.05 **p<0.01	• p<0.05		

[a] - mean

[b] - standard deviation

significant differences compared to Pre

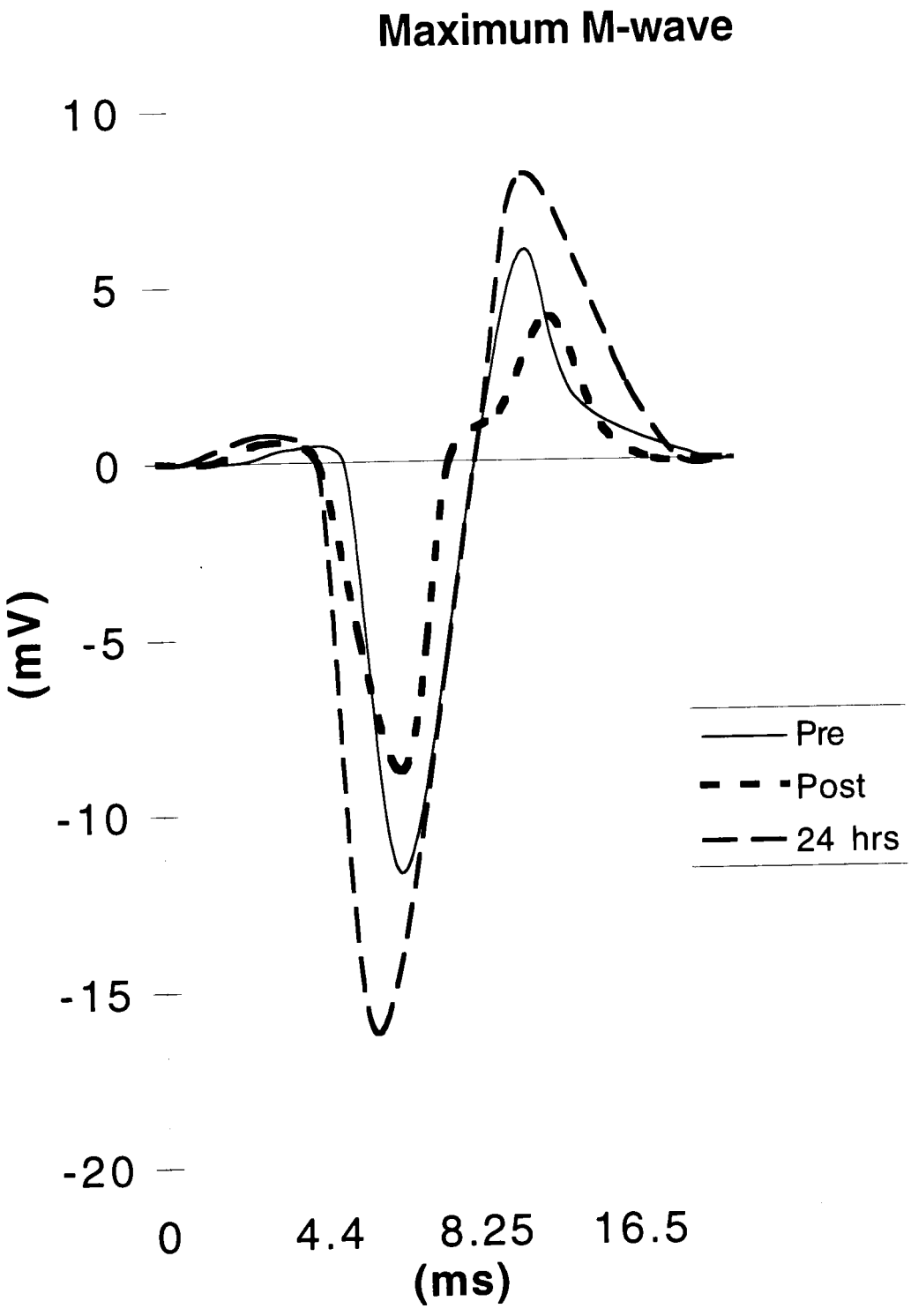

Figure 3.6: The maximal M-wave recorded from the FDI of one subject is shown pre-, post- and 24 hours after eccentric exercise.

TABLE 3.3: Components of joint stiffness at 15% MVC.

	Stiffness (Nm/deg)					
	Passive	Total	Active	Reflex	Active Intrinsic	Stimulated Intrinsic
Pre	0.0148[a]	0.0529	0.0371	0.0138	0.0238	0.0148
	(0.0038)[b]	0.0112	0.0116	0.0096	0.0061	0.0095
24 hrs	0.0138	0.0513	0.0367	0.0176	0.0199	0.0268
	0.0043	0.0128	0.014	0.0157	0.0043	0.0091

$p<0.05$

[a] - mean

[b] - standard deviation

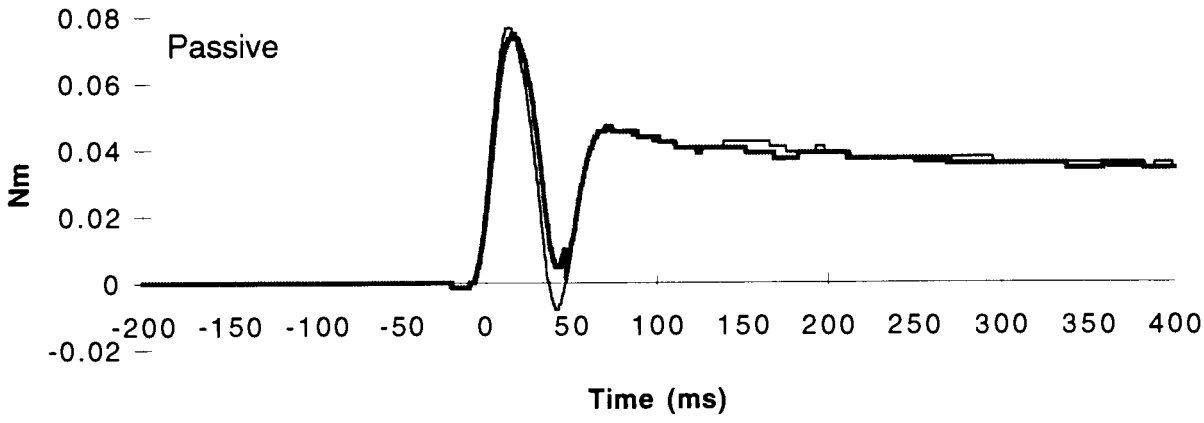

Figure 3.7: Passive torque responses for one subject pre- and 24 hours after eccentric exercise.

Passive joint stiffness was calculated for the interval 100 - 200 ms after onset of displacement.

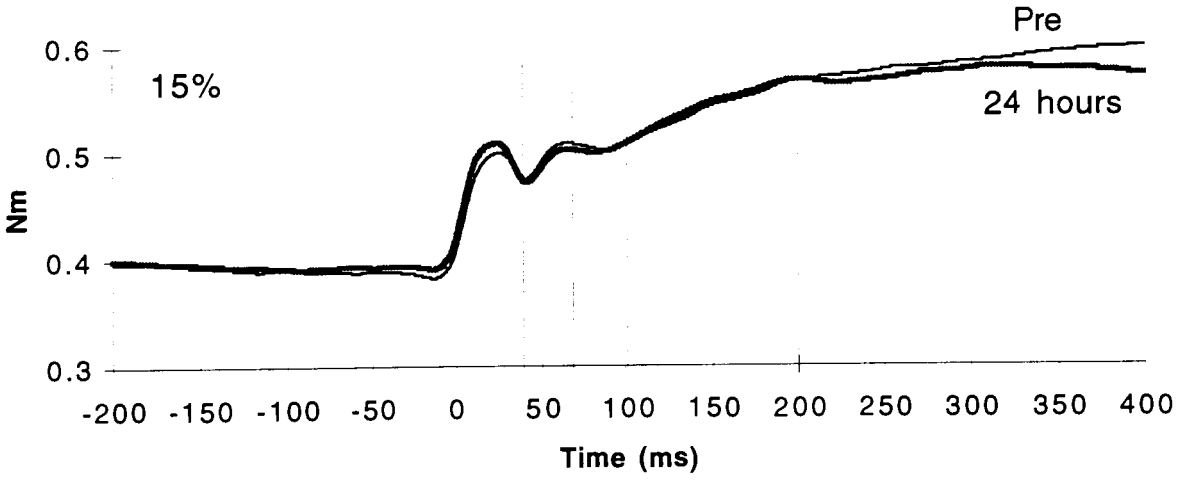

Figure 3.8: Torque responses for one subject during voluntary contraction to 15% of MVC pre- and 24 hours after eccentric exercise.

Total joint stiffness was calculated for the interval 100 - 200 ms after onset of displacement. The natural intrinsic stiffness was set equal to the average stiffness calculated for the interval 40 - 70 ms after the onset of displacement.

the total joint stiffness indicated that neither natural intrinsic nor reflex components changed. However, the stimulated intrinsic stiffness increased by 81% 24 hours after injury compared to pre-injury ($p<0.05$) and more stimulus current was required to produce the same abduction torque. While the stimulated intrinsic stiffness was less than the natural intrinsic stiffness pre-injury, it became greater than the natural intrinsic stiffness 24 hours after injury ($p<0.05$).

The background MEMG during 15% MVC was 66% greater 24 hours after injury compared to pre-injury ($p<0.001$). There were no significant differences in reflex or static MEMG at 15% MVC (Table 3.4). The response of one subject pre- and 24 hours after injury is shown in Figure 3.9.

The response to rapid displacement at 65% of pre-injury MP abduction MVC pre- and 24 hours after injury is shown in Figure 3.10. The joint stiffness determined 100 - 200 ms after the onset of the stretch decreased by 24.4% ($p<0.05$) (Table 3.5 and Figure 3.11). The FDI background MEMG did not change. However, it was a significantly higher percentage of MVC MEMG 24 hours after injury ($p<0.05$). The FDI reflex response declined as a percentage of background EMG ($p<0.01$). The tonic FDI reflex MEMG also declined significantly as a percentage of background EMG ($p<0.05$).

Functional Tests

The maximum velocity of MP abduction and latency from movement onset to peak velocity did not change significantly following injury. However, the

TABLE 3.4: FDI MEMG at 15% MVC.

	Background		Reflex	Static
	(μV)	(%MVC)	(%bckgrd)	(%bckgrd)
Pre	14.7[a]	20.6	121.4	46.3
	(11.2)[b]	8.3	35.9	28.7
24 hrs	24.4	52.3	92	33.5
	9	40.2	40.8	27.6
	p<0.001	p<0.05		

[a] - mean

[b] - standard deviation

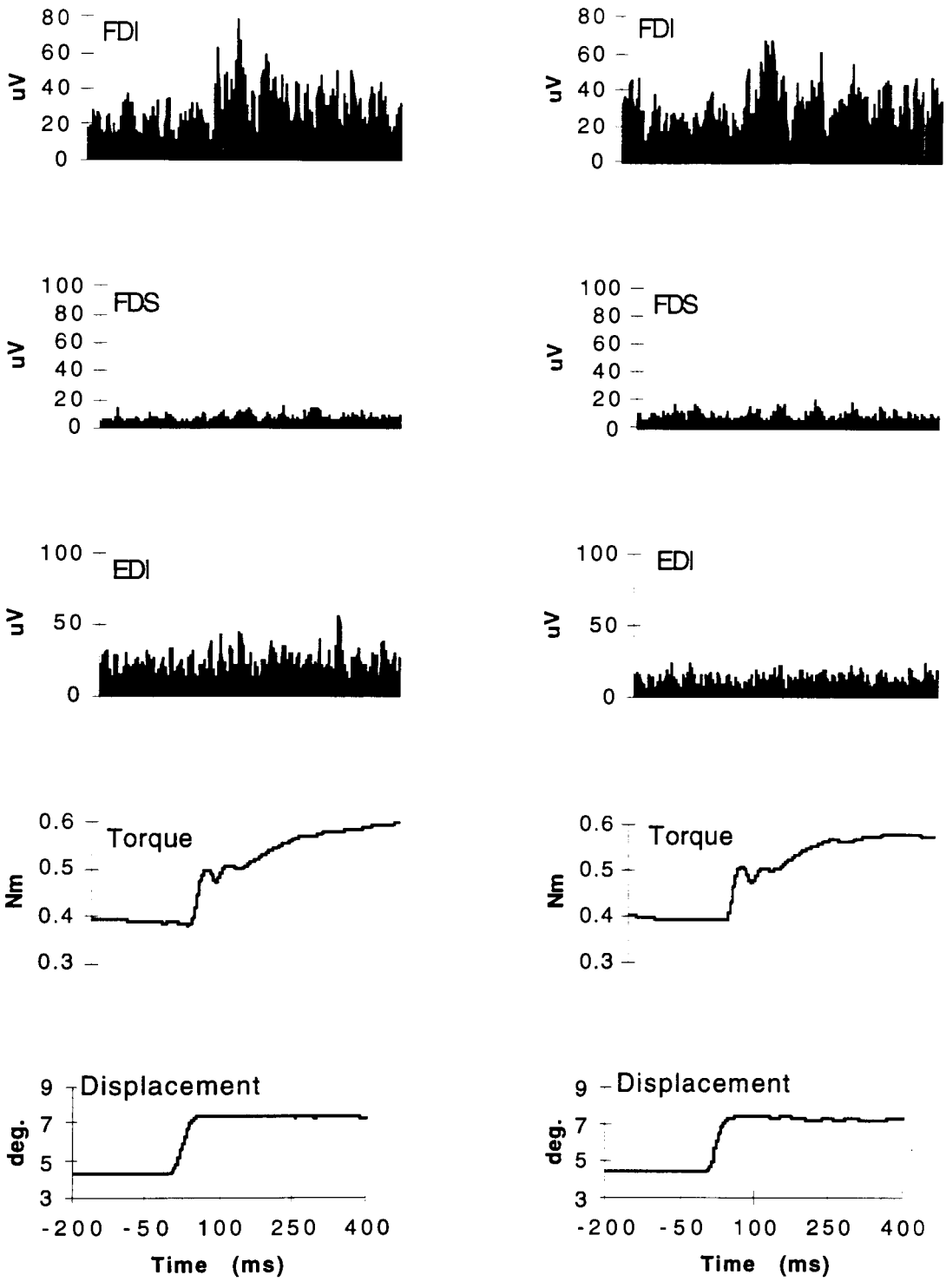

Figure 3.9: Response of one subject at 15% MVC pre- (L) and 24 hours (R) after eccentric exercise.

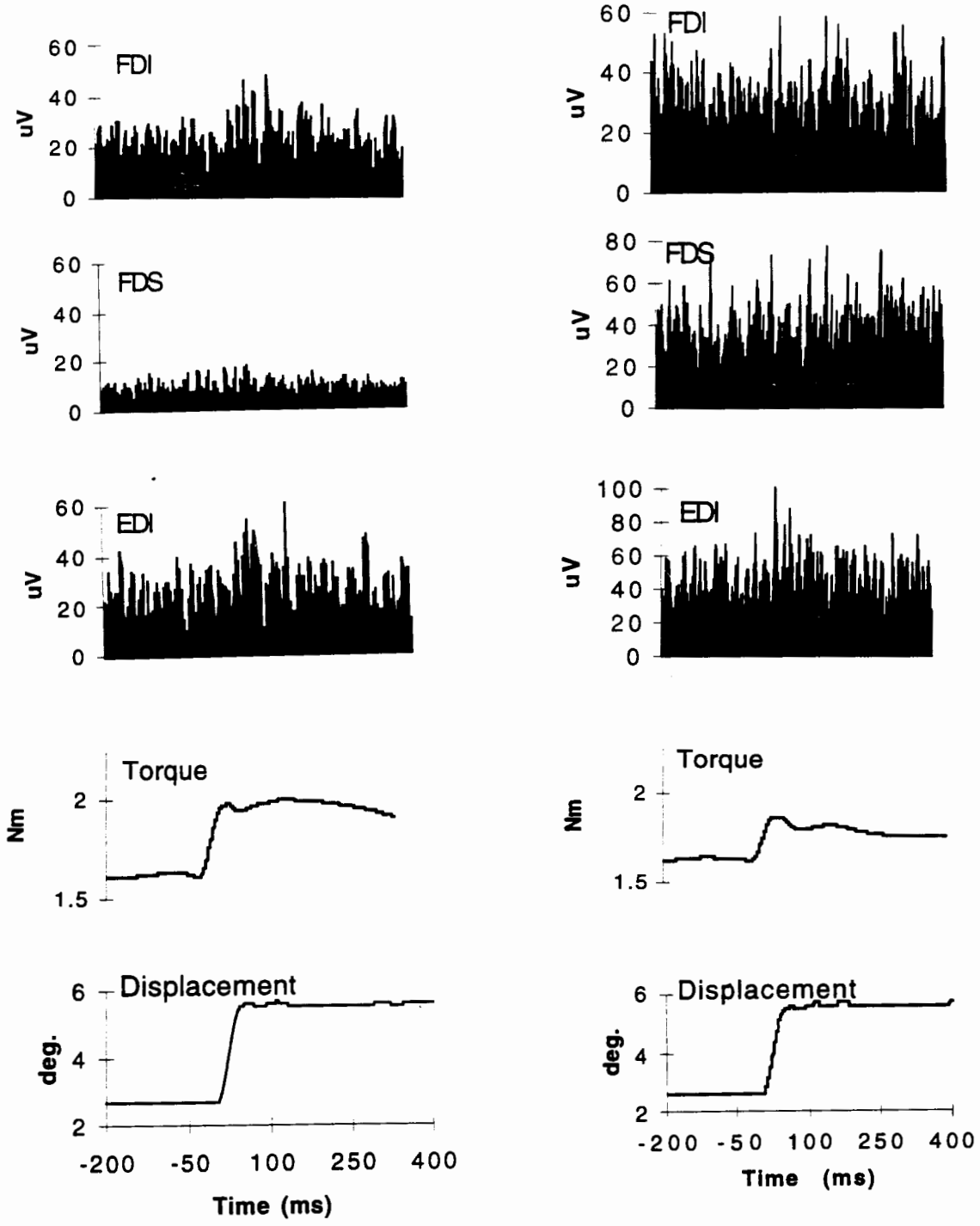

Figure 3.10: Response of one subject at 65% MVC pre- (L) and 24 hours (R) after eccentric exercise.

TABLE 3.5: Joint stiffness and FDI MEMG at 65% MVC.

	FDI MEMG				
	Background		Reflex	Static	Total stiffness
	(μV)	(%MVC)	(%bckgrd)	(%bckgrd)	(Nm/deg)
Pre	72.3[a]	93.4a	32.1	4.8	0.1001
	(48.3)[b]	(23.0)b	20.7	9.5	0.024
24 hrs	71.5	118.9	8.8	-7.4	0.0756
	32.4	32.3	17.6	10.8	0.021
		p<0.05	p<0.01	p<0.05	p<0.05

[a] - mean

[b] - standard deviation

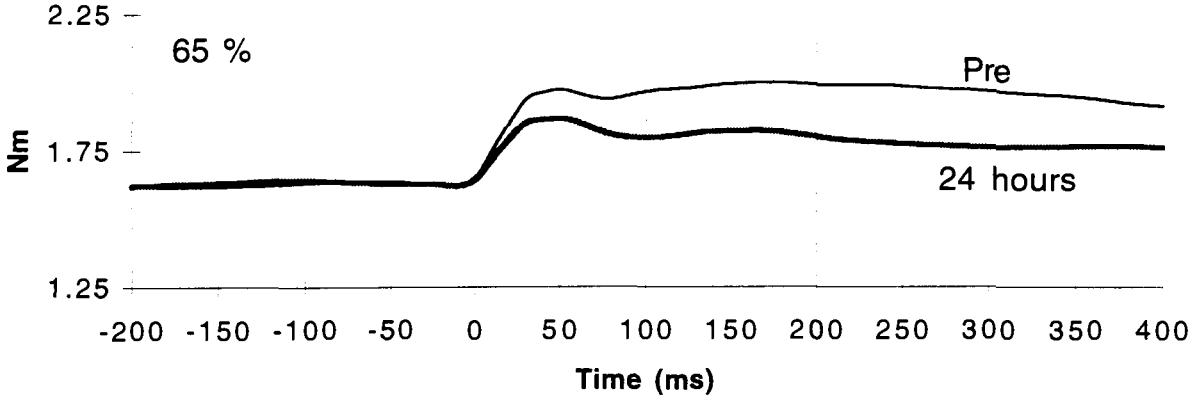

Figure 3.11: Torque responses for one subject during voluntary contraction to 65% of MVC.

Total joint stiffness was calculated for the interval 100 - 200 ms after onset of displacement.

onset of FDI EMG, measured as the time before movement onset, increased by 20.5% (Table 3.6, $p<0.05$). The duration of the FDI EMG burst also increased ($p<0.05$). There was no significant difference in the IEMG of the burst. Trials for one subject are compared in Figure 3.12.

The ability to stabilize an unstable load decreased after injury. The mean negative load stiffness that could be stabilized decreased by 14.4% ($p<0.001$) suggesting a corresponding decrease in joint stiffness (Table 3.7). The MEMG, expressed as a percentage of MEMG during maximal MP abduction, for the FDI, FDS and EDI did not change.

Variability in tracking a target torque, changing at a constant rate, was greater 24 hours after the injury compared to pre-injury (Table 3.8, $p<0.05$). The MF of the FDI EMG was similar pre- and 24 hours after injury during the first three seconds of contraction. However, it was significantly lower during the final two seconds 24 hours after injury ($p<0.05$ and $p<0.001$, respectively). The MEMG, on the other hand, was significantly greater during the first four seconds of contraction 24 hours after injury ($p<0.001$, $p<0.001$, $p<0.01$ and $p<0.05$, respectively). Figure 3.13 illustrates the higher FDI EMG and variance in torque, especially at higher levels, for one subject 24 hours after injury.

The similarity in slope of the MF during a 60 second contraction at 50% of MP abduction MVC suggests that the FDI muscle was not more fatiguable 24 hours after the injury than pre-injury (Table 3.9). However, the initial and the final values of MF 24 hours after injury were significantly lower ($p<0.05$) and the initial MEMG was significantly greater 24 hours after the injury ($p<0.05$).

TABLE 3.6: Means and standard deviations of the parameters measured during maximum velocity of MP abduction.

			FDI - EMG			
	velocity (deg/s)	time (ms)	onset (ms)	duration (ms)	IEMG (μV*s)	Normalized IEMG
Pre	513.4[a]	74.3	27.8	163.8	14.8	0.197
	(123)[b]	8.25	6.11	15.6	7.44	0.045
24 hours	487.1	75.9	33.5	179.9	13.3	0.22
	97.9	8.38	5.82	16.5	4.9	0.038
			$p<0.05$	$p<0.05$		

[a] - mean

[b] - standard deviation

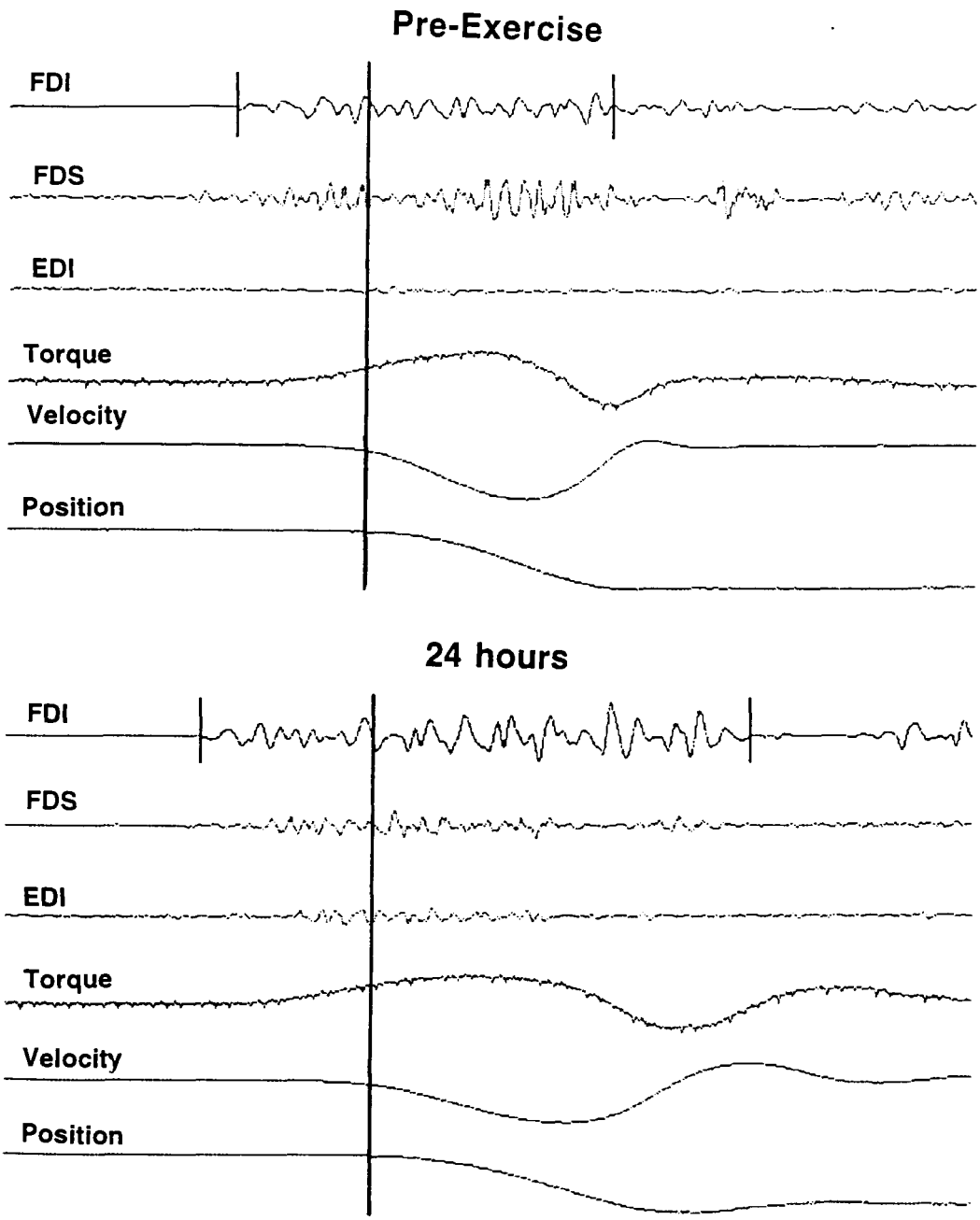

Figure 3.12: Trials for one subject for maximum velocity of MP abduction pre- and 24 hours after eccentric exercise.

TABLE 3.7: Means and standard deviations of the negative load stiffness and MEMG during the experiment to determine the ability to stabilize an unstable load.

	Maximal Instability						
		MEMG - FDI		MEMG - FDS		MEMG - EDI	
	Gain	(μV)	% of MVC	(μV)	% of MVC	(μV)	% of MVC
Pre	-0.118[a]	25	36.2	26.8	67.6	26.8	41.7
	(-0.017)[b]	15.7	15.3	20.3	64.4	20.3	26.9
24 hrs	-0.101	27.2	55.7	28.8	50.9	34	47.2
	-0.019	18.2	49.6	19.8	34.1	30.4	30.7
	p<0.001						

[a] - mean

[b] - standard deviation

TABLE 3.8: Means and standard deviations of (**A**) torque variance, FDI median frequency and (**B**) FDI MEMG for the torque tracking experiment.

A

Torque Tracking					
Variance (Nm^2)	Median Frequency - FDI (Hz) Time (sec)				
	1	2	3	4	5
19.2[a]	128.9	134.8	139.2	137	135.6
(12.6)[b]	21.9	24.8	22.3	24.9	26.3
27.4	132.3	134.2	129.8	123.8	117.4
16.5	22.1	23.9	21.9	27.6	25.9
p<0.05				p<0.05	p<0.001

B

FDI MEMG (μV) Time (sec)				
1	2	3	4	5
8.71	15.8	27.6	42.9	55.8
3.59	7.09	10.9	17	24.5
20.3	36.1	54	61.1	63.6
7.71	13.8	24.8	29.8	31.6
p<0.001	p<0.001	p<0.01	p<0.05	

[a] - mean

[b] - standard deviation

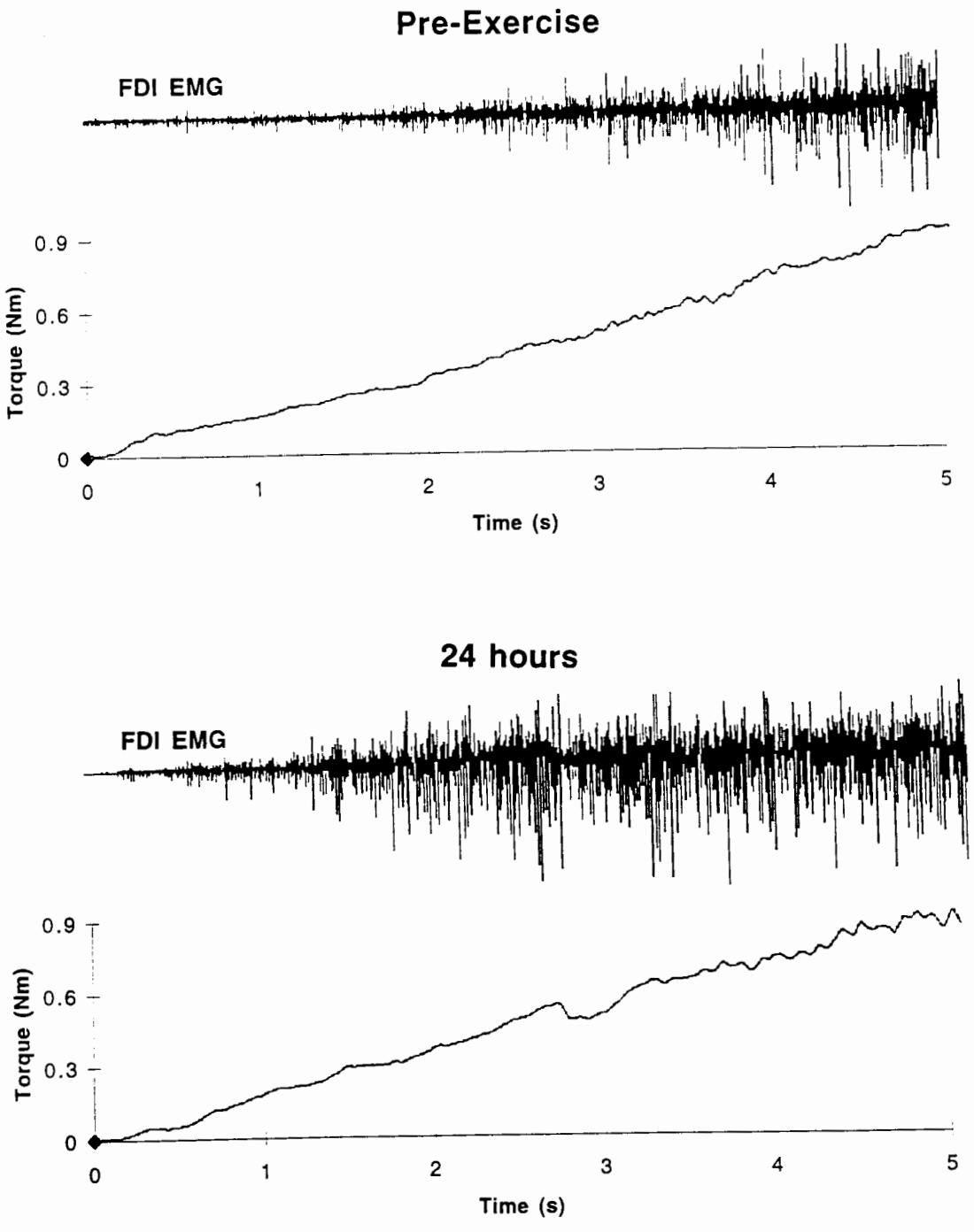

Figure 3.13: The FDI EMG and torque recorded for one subject while tracking torque changing at constant rate are shown pre- and 24 hours after eccentric exercise.

TABLE 3.9: Means and standard deviations of FDI median frequency and MEMG during a 60 second contraction at 50% MP abduction.

FDI Median Frequency (Hz)			FDI MEMG (μV)	
Slope	Initial	End	Initial	End
-0.83 [a]	126.4	83.8	43.2	35.8
(0.36)[b]	22.2	17.9	19.1	18.8
-0.707	113.2	74.9	56.4	29.7
0.34	27.8	13.1	27.6	9.8
	p<0.05	p<0.05	p<0.05	

[a] - mean

[b] - standard deviation

Effect on a Synergist Muscle

The maximal voluntary torque during MP flexion decreased by 15.2% 24 hours after injury ($p<0.001$), although FDI and FDS MEMG were not significantly different (Table 3.10). Based upon experiments employing muscle block with lidocaine, Ketchum et al. (1978) reported that the intrinsic muscles of the hand contributed 73% of the torque about the MP joint of the index finger, with the PIP and DIP joints held in extension. The FDI muscle contributed an average of 51% to this, or approximately 38.8% of the total MP flexion torque. Anatomical and biomechanical data presented by Zijdewind and Kernell (1994a) showed the FDI muscle moment potential was only 15% of the total contribution to MP flexion. Using the value reported by Ketchum et al., the 19% decrement in MP abduction torque found in this experiment would produce a decrement in MP flexion torque of 7.41%. Using the value suggested by Zijdewind and Kernell the expected decline would have been even lower.

The maximum velocity of MP flexion movement declined by 4.7% following injury (Table 3.11, $p<0.05$). The time following movement onset at which maximum velocity occurred increased, although it was not statistically significant ($p=0.055$). The time before movement onset at which the FDI and FDS bursts began increased ($p<0.05$ and $p<0.01$, respectively), as did the duration of the EMG burst for the FDI ($p<0.005$) and FDS ($p<0.001$). There were no changes in IEMG for either muscle. Trials for one subject pre- and 24 hours after injury are shown in Figure 3.14.

The injury had no significant effect on the MF and MEMG of the FDI

TABLE 3.10: MP Flexion MVC.

	Torque (Nm)	FDI MEMG (μV)	FDS MEMG (μV)
Pre	4.84[a]	57.89	61.3
	(1.35)[b]	39.8	50.3
24 hrs	4.11	53.06	58.7
	1.24	29.6	52.6
% change	15.2		
	8.1		
	$p<0.001$		

[a] - mean

[b] - standard deviation

TABLE 3.11: **A**, **B**, **C**, Means and standard deviations of the parameters measured during maximum velocity of MP flexion.

A Maximum Velocity - MP Flex

	velocity (deg/s)	time (ms)
Pre	1040.9[a]	71.3
	(142.2)[b]	6.08
24 hours	992.4	74
	175.5	7.13
	p<0.05	p=0.055

B FDI - EMG

	onset (ms)	duration (ms)	IEMG (μV*s)	Normalized IEMG
Pre	19.9	154.2	8.34	0.152
	8.85	46	4.9	0.063
24 hours	23.6	183.2	7.85	0.159
	6.74	37.4	3.55	0.064
	p<0.05	p<0.005		

C FDS - EMG

	onset (ms)	duration (ms)	IEMG (μV*s)	Normalized IEMG
pre	15.77	108.9	4.84	0.096
	4.7	46.2	4.35	0.082
24 hours	21.1	135.1	6.59	0.122
	6.78	44.1	7.48	0.102
	p<0.01	p<0.001		

[a] - mean

[b] - standard deviation

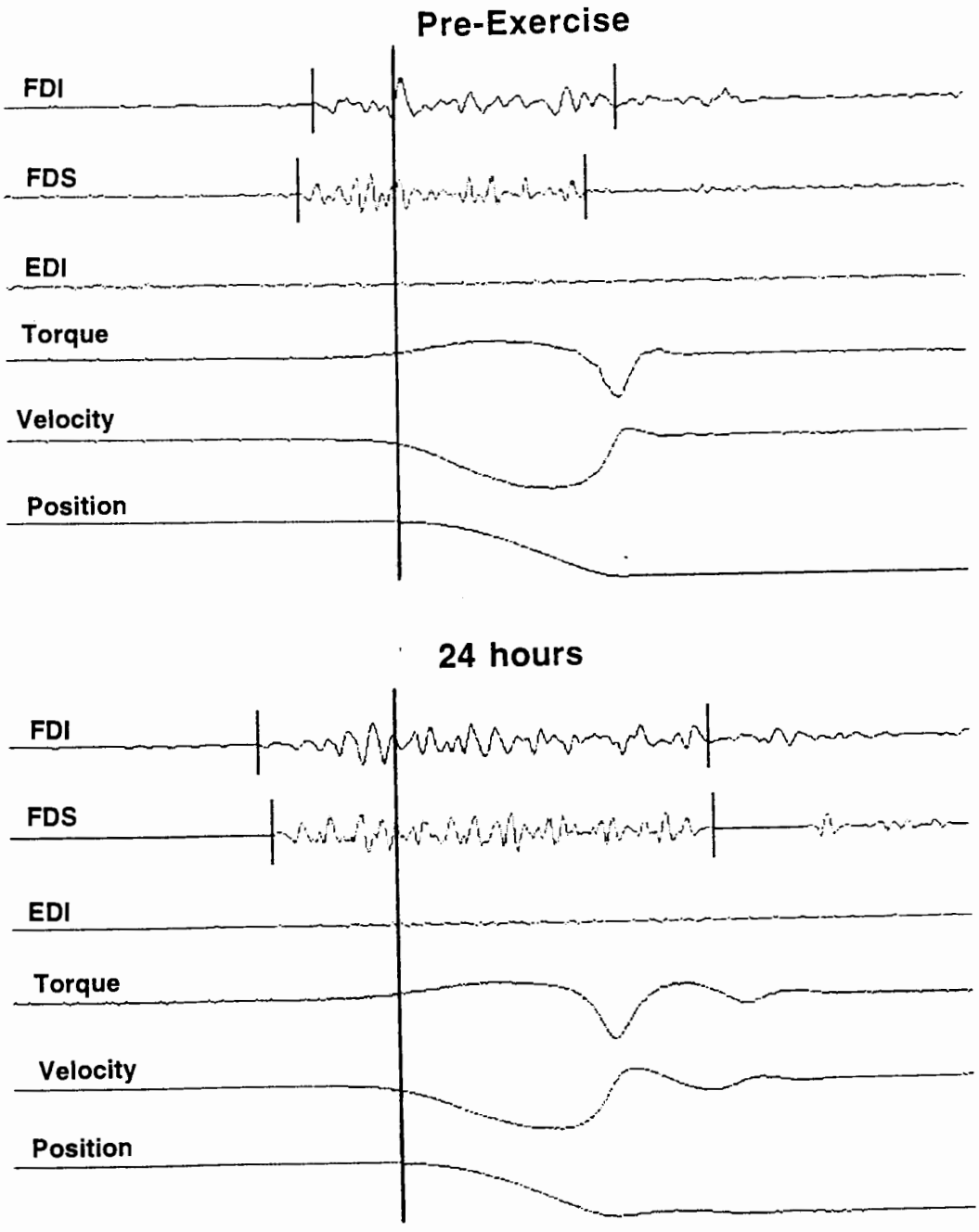

Figure 3.14: Trials for one subject for maximum velocity of MP flexion pre- and 24 hours after eccentric exercise.

during sustained MP flexion for 60 seconds (Table 3.12). The MEMG of the FDS was, however, greater during the final 5 s of the contraction following injury ($p<0.05$).

TABLE 3.12: Means and standard deviations of (**A**) FDI and (**B**) FDS median frequency and MEMG during a 60 second contraction at 50% MP flexion.

A FDI - EMG

Median Frequency (Hz)			MEMG (μV)	
Slope	Initial	End	Initial	End
(-0.646)[a]	132.1	98.4	29.3	35.3
(0.39)[b]	29.7	29.2	21.9	23.4
-0.734	127.2	89.2	32.4	35.8
0.37	22.3	31.6	17.6	26.7

B FDS - EMG

Median Frequency (Hz)			MEMG (μV)	
Slope	Initial	End	Initial	End
-0.249	180.1	168.3	15.9	25.2
0.14	27.4	24.3	17.1	15.9
-0.31	175.8	160.5	15.6	34
0.48	30.4	39.6	7.63	21.8

$p<0.05$

[a] - mean

[b] - standard deviation

Discussion

There was a reduction in maximum isometric torque of the FDI muscle 24 hours after the exercise bout which was unlikely to have been due to fatigue from the exercise bout because of the 24 hours allowed for recovery. Komi and Viitasalo (1977) had subjects perform repeated maximal eccentric and concentric contractions of the quadriceps muscle group. By the second day of a recovery period of four days the muscle tension and EMG had recovered completely from the concentric work, but not from the eccentric work. Davies and White (1981) studied the effects of eccentric and concentric work on the stimulated and voluntary isometric contraction of the triceps surae muscle. The results showed that following box stepping for an hour, only the leg which was constantly required to absorb the force of landing as the subject returned from the box to the floor (negative work) had long-lasting weakness. The electrically stimulated and voluntary forces were not fully recovered in the leg performing negative work 20 hours after exercise, whereas, the trailing leg (positive work) recovered within 2 - 4 hours.

Mechanical and neurophysiological changes that accompany muscle injury were examined by quantifying the effects of exercise-induced muscle injury to the FDI muscle. Although the injury may have been relatively mild, it did produce significant changes in joint mechanics and adaptations in neural control.

Mechanical Adaptation

It was hypothesized in the present study that injury to the FDI muscle would result in an increase in mid range passive MP joint stiffness. However, this turned out not to be the case. Howell et al. (1993) quantified elbow joint stiffness over a range from intermediate to end-range joint positions and reported a significant increase following eccentric exercise. They suggested that increased stiffness near the end-range of motion was due to swelling, and that the increased stiffness in the mid-range positions was due to muscle cell injury. Stauber et al. (1990) also suggested that sensations of stiffness arise from edematous changes in the muscle. The present study does not rule out the possibility of swelling causing increased joint stiffness at the end of ROM. It does, however, show that if swelling was present it was not sufficient to cause measurable changes in stiffness in the mid-range joint positions and thus does not affect the ability to move at these joint positions.

The lack of change in active joint stiffness for low torque suggests that the muscle injury did not affect the mechanics of small motor units required for low torques. Decomposition of active joint stiffness into the passive, natural intrinsic and reflex components did not show any changes either. The constituent values for active joint stiffness were similar to those reported by Carter et al. (1990) for the FDI muscle and are in support of hypotheses 2 and 4.

There was a difference between the natural intrinsic and stimulated intrinsic stiffness pre-injury. It is well accepted that during electrical stimulation of a muscle the recruitment order is reversed with respect to the size principle

and is controlled by the electrical excitability of the fibers and by the current density (Koester 1991). Thus, during stimulation of the FDI the large motor units must have been recruited first. The difference between the two measurements of intrinsic stiffness pre-injury may be explained by a difference in stiffness of different motor unit types. Slow-twitch motor units, despite developing smaller tetanic tensions than fast-twitch units, produce a greater stiffness per unit force (Petit et al. 1990). The higher stiffness in voluntarily activated muscle reflects recruitment of small slow-twitch motor units to produce the required torque, while the lower stiffness of stimulated muscle results from the activation and lower resistance to displacement of the larger faster twitch motor units.

Contrary to the findings for low torque, the active joint stiffness for high torque was significantly lower 24 hours after exercise-induced muscle injury. The FDI muscle is made up of 57% type I fibers (Johnston et al. 1973) and utilizes motor unit recruitment as the primary means to increase force up to 50% of MVC (De Luca et al. 1982). During contraction at 65% MVC most motor units should have been recruited, whereas, at 15% MVC predominantly small motor units would have been recruited. The decrease in stiffness for high torque may have been due, in part, to lower reflex stiffness, since the reflex EMG was reduced. However, the natural intrinsic stiffness also appeared to be lower for a number of subjects, since the torque was lower both during and following displacement of the finger. This would suggest that muscle fibers had a lower stiffness to force ratio following injury.

Injury to large fibers was also supported by the finding that during electrical stimulation of the muscle to produce a low level of torque, there was an increase in stimulated intrinsic joint stiffness 24 hours after muscle injury. The greater intensity of stimulation and resultant increase in the ratio of stiffness to force may have been due to injury to large fibers fast-twitch fibers and recruitment of smaller, slow twitch fibers which have a greater stiffness to force ratio.

Because the FDI also contributes torque to MP flexion it is possible that the increase in the stimulated intrinsic stiffness was due to a greater MP flexion component during electrical stimulation, which would have increased the number of attached cross-bridges but not contributed to abduction torque. However, the motor units in the FDI muscle are not arranged in particular task groups (Milner-Brown et al. 1973; Desmedt and Godaux 1981; Thomas et al. 1986; Flament et al. 1993; Jones et al. 1994) so any increase in electrical stimulation intensity should have resulted in an increase in torque and stiffness in both directions.

The presumed greater proportion of injury to the large motor units is in agreement with other studies which have provided morphological evidence of preferential damage of the large type II fibers (Fridén et al. 1983a; Jones et al. 1986; Fridén and Leiber 1992). Nardone et al. (1988) and others (Dick and Cavanagh 1987; Moritani et al. 1988) have suggested that this preferential involvement was due to different patterns of use during eccentric exercise. However, this does not preclude the possibility of greater intrinsic susceptibility

to damage of the type II fibers. The results of this study do not address this issue but they are consistent with a mechanical deficit in the large fibers following eccentric exercise.

Neural Adaptation

The decline in M-wave post-exercise, in this study, may have been the result of alterations of muscle membrane excitation and impulse propagation which has been reported to occur during fatiguing contractions. Studies of fatigue have shown a decrease in amplitude and an increase in duration of the M-wave during fatiguing concentric muscle contraction (Stephens and Taylor 1972; Bigland-Ritchie et al. 1982; Milner-Brown and Miller 1986; Bellemare and Garzaniti 1988). In contrast, in this study the amplitude and the duration decreased immediately post-exercise.

M-wave potentiation has been previously reported during, and immediately following, both voluntary and stimulated concentric contractions in humans (Duchateau and Hainaut 1985; Fitch and McComas 1985; Hicks and McComas 1989; Hicks et al. 1989, 1992; McComas et al. 1994; Nagata and Christianson 1995) but there is no evidence that it persists 24 hours after exercise. Potentiation following fatiguing concentric contractions has only been reported up to 3 minutes after exercise (Galea and McComas 1991). Increased M-wave amplitude 24 hours after the exercise bout in this study suggests changes in membrane properties produced either directly or indirectly as a result of eccentric exercise. The M-wave reflects the number of muscle fibers responding to stimulation, as well as the size of individual fiber potentials and

their degree of synchrony (Milner-Brown and Miller 1986; Bellmare and Garnzaniti 1988). M-wave potentiation is thought to arise from greater synchronization of muscle fiber action potentials or hyperpolarization of the resting membrane potential resulting in individual fiber action potentials of greater amplitude. It has been suggested that hyperpolarization is caused by increased activity of the electrogenic sodium pump (Hicks and McComas 1989). Some researchers have implicated calcium (Ca^{++}) in the secondary metabolic injury. Armstrong (1990) suggested that Ca^{++} homeostasis is disrupted as a result of damage to the sarcoplasmic reticulum and/or sarcolemma. Ca^{++} entry has been reported during action potentials in nerve and muscles. The role of Ca^{++} in the AP mechanism has been established in a variety of invertebrate preparations. Inward Ca^{++} current was observed by Fatt and Katz (1953) who reported that in the absence of sodium (Na^{+}), Ca^{++} spikes were evident in crab muscle fiber. Hagiwara et al. (1964) reported that the amplitude of the spike varied with the external concentration of Ca^{++}. An increase in Ca^{++} resulted in an increase in spike amplitude in the giant muscle fiber of the barnacle. Ca^{++} has also been shown to have an effect on potassium (K^{+}) current. In 1970, Meech and Strumwasser demonstrated that an injection of Ca^{++} ions into nerve cells caused an increase in K^{+} conductance. An increase in K^{+} conductance could lead to greater hyperpolarization and greater action potential amplitudes.

Since potentiation of the M-wave rather than decline was seen 24 hours after muscle injury the pathway for excitation of the muscle must have remained intact. This does not mean, however, that there should have been an

accompanying increase in force (Zijdewind and Kernell 1994b). In fact, in this study, the decline in MP abduction maximal torque and the decline in MEMG would suggest a decrease in the maximal neural drive to the motoneurons after injury. The decline in MEMG is consistent with the suggestions by Hasson et al. (1993) and others (Newham et al. 1987; Clarkson et al. 1987, 1992; Crenshaw et al. 1994) that there is selective inhibition of injured fibers so that they may go through the process of repair. However, no specific mechanism was proposed for this selective inhibition. Adaptation which has been reported in conjunction with the repeated bout effect has been postulated to be due to inhibition or sparing of the damaged fibers on the second and subsequent bouts of exercise (Kuipers et al. 1985; Newham et al. 1987; Clarkson et al. 1987, 1992). Studies of fatigue have shown declines in motoneuron discharge rates (Bigland-Ritchie et al. 1986a). The decline in motoneuron firing rates was suggested to be the result of a peripheral reflex from the fatigued muscle as any central effects were shown to recover within the time period studied. The authors speculated that the afferent limb of the reflex arose from receptors in the muscle sensitive to muscle contractile properties or the metabolic state of the muscle. Studies investigating the effects of immobilization and unloading of muscle have reported a similar decrease in neural drive to explain strength decrements (McComas 1994; Miles et al. 1994).

While there appeared to be a decline in neural drive, or inhibition, during maximal contraction, the amount of EMG increased for torque of less than 50% MVC. This finding is in agreement with the findings of Kroon and Naeije (1991)

and others (Komi and Viitasalo 1977; Berry et al. 1990) and may reflect higher firing rates and greater synchronization of the smaller motor units to compensate for damaged muscle fibers. Any inability of injured fibers to contribute to the force, whether due to inhibition or structural damage, would result in the need for increased contribution by other motor units. This may be analogous to the reported lower threshold and increased firing rates of the slow motor units and greater EMG during fatiguing contractions (Bigland-Ritchie et al. 1986b; Maton and Gamet 1989).

It has been suggested that resetting of the myotatic reflex may explain the increased muscle stiffness (Korr 1976). The lack of change in total joint stiffness or reflex stiffness at low torques in the present study suggests that this was not the case. Furthermore, there was no change in the reflex EMG at low torque. On the other hand, the reflex response was lower after injury when subjects were producing higher torque. Both phasic and tonic reflex EMG responses were significantly smaller 24 hours after the muscle injury. In fact, the tonic response was inhibited since it was less than the background activity prior to the stretch. The observed declines in reflex EMG could be explained by earlier saturation of the stretch reflex response after injury because of more complete recruitment of the motoneuron pool at lower torque or by reduced excitability of large motor units at high torque. This, once again, supports hypothesis 3 and would suggest a reduced ability to recruit or reduced excitation of the fast-twitch motor units under conditions in which they would normally be activated.

A decline in the short-latency stretch reflex response similar to that observed in this study has been reported after fatiguing contractions (Balestra et al. 1992; Duchateau and Hainaut 1993). It was suggested that the changes in the short latency response were related to peripheral mechanisms such as the inhibitory effects from group III and IV afferents and/or from pre-synaptic inhibition. The long-latency reflex response. However, showed no decrease during voluntary fatiguing contractions in contrast to the results of this study. Bulbulian and Bowles (1992) reported a reduced H-wave in the gastrocnemius muscle, expressed as a ratio of the M-wave, immediately after eccentric exercise of the lower extremity, however, it returned to baseline by 24 hours. They suggested that the decline was a result of type I motor unit inhibition by type II motor units utilized in the exercise. No indicators of injury to the gastrocnemius were provided in that study. The recovery in the H/M ratio 24 hours after the exercise bout is inconsistent with the results of the present study and may indicate that there had been minimal injury to the gastrocnemius muscle. Furthermore, as the H-reflex bypasses the spindle apparatus, changes in spindle sensitivity which might have resulted from exercise-induced muscle injury were not studied.

Evidence in support of hypothesis 5 and an indication of a change in activation patterns was provided by the EMG analysis during sustained contraction and torque tracking. In the task requiring sustained contraction of the FDI at 50% MVC, the initial and final MF were significantly lower after muscle injury. The power spectrum of the EMG signal describes the relationship

between signal amplitude and signal frequency. The MF is the mid-power point of the spectrum. The two principal factors affecting the frequency content are action potential shape and motor unit discharge rates. The shape of the action potential is very sensitive to conduction velocity. As the conduction velocity decreases, action potential duration increases and the constituent frequencies decrease. Most commonly, MF has been used for evaluation of fatiguing contractions during which a shift in the frequency content of the signal to the low end of the power spectrum reflects the fatigue of large fibers and reliance on the slow fatigue resistant fibers (Lindström et al. 1970; Bigland-Ritchie et al. 1981, 1986b; Hagberg and Ericson 1982; Duchateau et al. 1991; De Luca 1985).

Solomonow et al. (1990) and others (Bilodeau et al. 1995) have shown that an increase in MF was directly associated with the recruitment of large diameter fibers, whereas an increase in the firing rate did not affect MF. They concluded that average conduction velocity during motor unit recruitment was the major contributor to variations in MF. Muscles having a higher percentage of fast fibers would have a correspondingly greater initial MF (Komi and Tesch 1979; Moritani et al. 1982; Wretling et al. 1987; Gerdle et al. 1991; Kupa et al. 1995). The finding that the initial MF at 50% MVC was lower 24 hours after the injury suggests that slower motor units were used to initiate the sustained contraction. Initial MEMG was greater, consistent with higher activation of these units. These observations suggest that the muscle injury caused substantial changes in activation patterns. The lower final MF is consistent with reduced activation of larger fibers throughout the contraction. Berry et al. (1990)

suggested that if there was impaired function of the high threshold fast fibers following eccentric exercise one would expect the MF to shift to a lower value. However, in their study of the quadriceps muscle they found no shift in the mean power frequency of the EMG signal and concluded that motor units of similar activation characteristics were utilized before and following the exercise. The failure to find a shift of the mean power frequency may have been due to the level of contraction being too low to recruit fast motor units. Furthermore, they reported a single mean power frequency for all of the quadriceps muscles, which may have masked differences among the individual muscles.

During the torque tracking task, the MF did not begin to differ significantly between pre- and 24 hours after injury until the torque reached 30% MVC. At this point the MF dropped below the pre-injury value, again suggesting inhibition of the large fibers and reliance on the small fibers. The greater MEMG throughout the first four seconds suggests that the CNS increased the activity of the small fibers to compensate for injury to large fibers.

These findings provide the first evidence that there is a shift in activation of motor units after muscle injury. The finding of a decrease in MF in tasks requiring high torques is consistent with injury and reduced activation of the large fibers. The decline in MF was not related to a change in the shape of the AP due to fatigue as the duration of the M-wave did not change 24 hours after injury. The increase in MEMG also indicates that greater activation of the small fibers is needed to complete the tasks. No other studies have reported such findings. However, this was the first study to investigate exercise-induced

muscle injury in a single muscle. The focus on combined activity of several muscles operating about a joint may have masked these types of changes in previous studies.

Functional Adaptation

The decrease in maximal torque, as well as the increased MEMG required for submaximal torque levels seen in this study have been commonly reported after exercise-induced muscle injury. Such changes would suggest impaired functional ability. Damage to large fast-twitch fibers and greater reliance on small slow-twitch motor units would be expected to have definite functional implications. Miles et al. (1993) reported a decrease in maximum velocity of movement, which one might expect with injury to large fibers. However, contrary to the findings of that study and hypothesis 4, this was not found in the present study. Although there was no significant decline in maximum velocity, there was a significant increase in the time of EMG onset before movement and in the duration of the initial FDI burst. These adaptations in neural control were similar to the findings of Miles et al. (1993). The failure to find a change in maximum velocity may have been due to the fact that rapid finger abduction was a novel task, especially when performed with the left hand. Any decrement in velocity on the second day may have been offset by an increment in performance, due to practice. This explanation is supported by the significant decline in maximum MP flexion velocity, a much more familiar movement, following injury.

The shift of the MF to lower frequencies, reflecting an increased contribution of slow motor units, was evident in the task requiring a sustained contraction of 60 seconds. This task was used to investigate the fatiguability of the FDI after muscle injury. Other researchers have reported reduced endurance times and low frequency fatigue following injury. Jones et al. (1989) reported that it can take up to 48 hours for muscle force to return to pre-injury levels at low frequencies of electrical stimulation. Kroon and Naeije (1991) reported an increased rate of decline of the EMG mean power frequency over the first 30 seconds of a sustained contraction following eccentric exercise and suggested that this was due to increased fatiguability of the muscle. Using this index of fatiguability, the results of this study suggest that the FDI muscle was no more fatiguable after muscle injury, contrary to their findings. However, although the slope of the MF did not change during a 60 s contraction there was an overall shift of the MF to lower values, indicated by the lower initial and final MF. This would suggest greater reliance on slow motor units which would make the muscle less fatiguable. This interpretation is consistent with the conclusions of Balvane and Thompson (1993), who reported a similar finding in the quadriceps. Given that force may have been mainly produced by slower motor units, the duration of the task in this study may not have been sufficient to produce fatigue. Further studies of endurance times coupled with MF analysis would be useful to determine the ability of the small motor units to sustain force after muscle injury.

Following muscle injury, subjects were less able to track torque smoothly. This impairment was most evident at higher torque levels and thus supports hypothesis 4. The increase in torque variance may have resulted from decreased perception of muscle force, irregular jumps in force as the larger injured motor units were recruited, irregular changes in firing rate or tremor (Saxton et al. 1995). The shift in MF at the beginning of the trial illustrates that changes in neural control occurred even at low force levels. This is similar to the findings of Galganski et al. (1993), who studied an aged population. They suggested that the decline in the number of fast motor units that occurs with aging results in impairment of ability to control force.

Impaired motor control was also confirmed by the loss of the ability to stabilize the MP joint against loads which were highly unstable. Joint stiffness plays a prominent role in the control of posture and movement. Stability is achieved by cocontraction of agonists and antagonists which results in an increase in joint stiffness without a change in the net joint torque (Hogan 1984; DeSerres and Milner 1991; Milner and Cloutier 1993; Milner et al. 1995). An impairment. The FDI EMG during maximal cocontraction was approximately 50% of MVC EMG and did not change following muscle injury. This signifies that for a similar amount of EMG the same level of joint stiffness could not be achieved. Furthermore, the EMG during maximal cocontraction was approximately 50% of MVC EMG (Milner et al. 1995). The decline in control of joint stiffness by cocontraction and postural stability following exercise-induced muscle injury shown in this study has not been reported previously.

The observed decrements in performance, for the most part, support hypothesis 4 and are consistent with greater damage to large fast-twitch muscle fibers than small, slow-twitch fibers and greater reliance on slow-twitch motor units. Impairment of performance was generally only seen when high muscle activation was required.

Effect on a Synergist Muscle

The effect of an exercise-induced muscle injury on an uninjured synergist muscle has never previously been reported. The FDI and FDS muscles both contribute to MP Flexion. Several tasks were performed to determine if an injury to the FDI muscle would directly influence the FDS muscle or the ability to perform MP flexion.

Studies have confirmed that the order of recruitment of motor units in both MP flexion and abduction follows the size principle such that the control of individual units in the FDI is essentially independent of the direction in which the muscle exerts force (Thomas et al. 1986; Jones at al. 1994). Therefore, any injury to muscle fibers and decrement in torque in abduction should have had an effect on torque in flexion. The decline in flexion MVC, which was greater than predicted by the relative contribution of the FDI to flexion torque, supports hypothesis 6 and suggests that the injury to the FDI muscle had a strong effect on synergist muscles performing MP flexion.

Inhibition of close synergists has been reported during fatiguing contractions by Hayward et al. (1988). They suggested that there was increased heteronymous reflex inhibition during muscle fatigue. Bigland-Ritchie et al.

(1986a) reported a decline in motoneuron discharge rates following fatigue from maximal voluntary contraction. They attributed the decline to peripheral reflex inhibition because the central effects of the fatiguing contraction had recovered within the time period of study. They further speculated that the afferent limb of the reflex included group III and IV muscle afferents which were sensitive to fatigue-induced changes within the muscle. Hayward et al. (1991) reported fatigue-induced excitation of these afferents during muscle stretch, local pressure and muscle contraction.

It is unlikely that the FDS muscle was injured even if it had been active during finger abduction. This is because the FDS has an adduction moment at the MP joint and, therefore, would have been shortened rather than stretched by displacements imposed on the finger. However, these findings also do not rule out the possibility of greater cocontraction of antagonist muscles following muscle injury resulting in a decline in net torque.

Following muscle injury there was significant decline in the maximum velocity of MP flexion. This is in contrast to abduction and may be explained by the familiarity of this task compared to that of abduction. The EMG of the FDS muscle also indicates changes in the pattern of activation. Changes in onset and duration of EMG were similar to those found for the FDI muscle during rapid flexion and abduction. This would suggest that the ability to perform MP flexion by both the FDI and the FDS muscles was affected by the injury to the FDI muscle and lends further support to hypothesis 6.

Finally, the ability to sustain a contraction over 60 seconds revealed changes in the relative contributions of the FDI and FDS muscles. The difference in the FDI MF and MEMG that were present for the sustained MP abduction contraction were not evident in the MP flexion contraction. This suggests that an increase in activity of the FDS muscle rather than a change in the activation pattern of the FDI was used to compensate for the deficiency in the FDI. This compensation was most evident towards the end of the contraction. These changes illustrate the flexibility provided by the existence of the synergy performing the task and the effect that one muscle can have on another synergist muscle.

Summary

The lack of any significant change in active joint stiffness or its components at low torque suggests that the muscle injury produced little effect on the mechanical properties of small slow-twitch motor units. The increase in stimulated intrinsic joint stiffness and significantly lower joint stiffness at high torque suggests that there were greater effects on the mechanical properties of the large fast-twitch motor units. M-wave potentiation 24 hours after muscle injury suggests there were long-lasting changes to membrane properties. Compensation for the muscle injury included an increase in MEMG during submaximal contractions and a shift in the MF to lower values. These changes suggest reduced activation of the large motor units. Subjects' ability to track torque and to stabilize the MP joint was impaired. The decline in MP flexion MVC, which was greater than anticipated, suggests that activation of the FDS

muscle was affected by the injured FDI muscle. Compensation by the FDS muscle for the injury to the FDI muscle occurred during a sustained submaximal MP flexion task.

The findings of this study illustrate the mechanical changes that occur in a single muscle as a result of muscle injury. Changes in neural control, including reflex responses and recruitment were evident. The effect of these changes on the ability to control posture and movement were shown. The influence of an injured muscle on a synergist muscle was also apparent.

CHAPTER FOUR - EXERCISE-INDUCED INJURY OF THE WRIST EXTENSOR MUSCLES

Introduction

Most studies of exercise-induced injury in humans have focused on groups of muscles. This is reasonable as often several muscles contribute to a particular movement about a joint. However, it has been difficult to distinguish differential effects of injury on individual muscles. Given that several muscles are always simultaneously active during any task, a study of muscle injury which examines the effects on individual muscles could provide insight into the adaptation to injury. It is likely that muscle injury would affect the activation and performance of uninjured antagonist muscles, as well. So far, there have been no studies of antagonist muscle activity and its relationship to agonist muscle activity following exercise-induced muscle injury.

The purpose of this study was to investigate the effects of exercise-induced muscle injury on the control of synergist and antagonist muscles of the wrist. Flexion and extension of the wrist was chosen as a model system because the wrist could be easily displaced by the torque motor used in the previous study and because the activity of all of the important muscles involved in flexing and extending the wrist could be recorded with surface electrodes. Consequently, joint stiffness could be quantified and muscle activation patterns examined. Each of the three extensor muscles, extensor carpi ulnaris (ECU), extensor carpi radialis longus (ECRL) and extensor digitorum communis (EDC), was studied.

The effects of injury to the extensors on the control of the antagonist wrist flexor muscles, flexor carpi ulnaris (FCU), flexor carpi radialis (FCR), and flexor digitorum superficialis (FDS), were also evaluated. All tests were performed on five separate occasions, once before and four times following the exercise bout over a period of 10 days.

The following hypotheses were addressed:

1. Exercise-induced injury to the wrist extensor muscles will not affect the passive wrist joint stiffness measured in the mid-range of MP joint motion.
2. Following exercise-induced injury to the wrist extensor muscles, there will be a decrease in reflex excitation in the response to muscle stretch, only in situations where activation of large motor units is required. Consequently, there will be no change in short latency EMG or reflex stiffness in tasks requiring low levels of wrist extension torque, but there will be a reduction in both short latency reflex EMG and reflex stiffness in tasks requiring high wrist extension torque.
3. Performance in tasks designed to quantify motor function during sub-maximal wrist extensor activation will be impaired following exercise-induced injury to the wrist extensor muscles, but only if the tasks require the recruitment of large motor units.
4. Exercise-induced injury to the wrist extensor muscles will result in injury to large muscle fibers rendering them less able to produce force. At force levels where large fast-twitch motor units are normally required, the reduced

contribution of the injured large muscle fibers will be compensated by increased activation of small slow-twitch motor units.

5. Following exercise-induced injury to the wrist extensor muscles, activation of all the injured wrist extensor muscles will be affected similarly.
6. Exercise-induced injury to the wrist extensor muscles will result in increased activity of the antagonist wrist flexor muscles whenever the injured wrist extensor muscles are reciprocally activated at levels high enough to recruit injured muscle fibers.
7. Performance of tests designed to quantify motor function will be impaired following exercise-induced injury to the wrist extensor muscles. Decrements in performance will be better correlated with decrements in maximal wrist extensor force than ratings of wrist extensor muscle soreness.

Materials and Methods

Subjects

Ten normal male subjects participated in this study (age range 20 - 35). Eight of the subjects were right-handed, the other two left-handed. None of the subjects reported any previous history of neuromuscular disorders. Each gave written informed consent to participate prior to the experiment (Appendix A). None had previously participated in any studies involving eccentric exercise of the muscle of the forearm. Six had previously (10 weeks prior) been involved in the study of exercise-induced muscle injury of the FDI muscle. Most were familiar with the testing apparatus. Subjects were asked not to participate in any weight-

training activities, specifically for the upper extremities, for the duration of the study. The experiment was approved by the University Research Ethics Review Committee at Simon Fraser University (Appendix A).

General Design

Subjects were tested on five separate occasions, previous to exercise-induced muscle injury (pre), 24 hours (Day 1), 48 hours (Day 2), 96 hours (Day 4), and 240 hours (Day 10) after muscle injury. All testing was performed on the left hand.

Apparatus

A torque motor (PMI U16M4), coupled to the manipulandum, was used to generate loads which were computer controlled (Figure 4.1). In different tests, the torque motor was used to produce offset torque during an initial holding period, mechanical behavior equivalent to a negative spring, and a servo-controlled ramp and hold angular displacement. The maximum torque that could be produced by the torque motor was 5 Nm. In tasks which required torque greater than 5 Nm, a stiff spring (6.55 N/cm) was attached to the manipulandum. When the spring was stretched it produced a large torque opposing wrist extension.

Position and velocity were measured using a potentiometer and tachometer attached to the motor shaft. The torque was measured by a linear strain gauge mounted on a cylinder, coupling the motor shaft to the manipulandum (Figure 4.1). EMG was recorded using active, bipolar, stainless

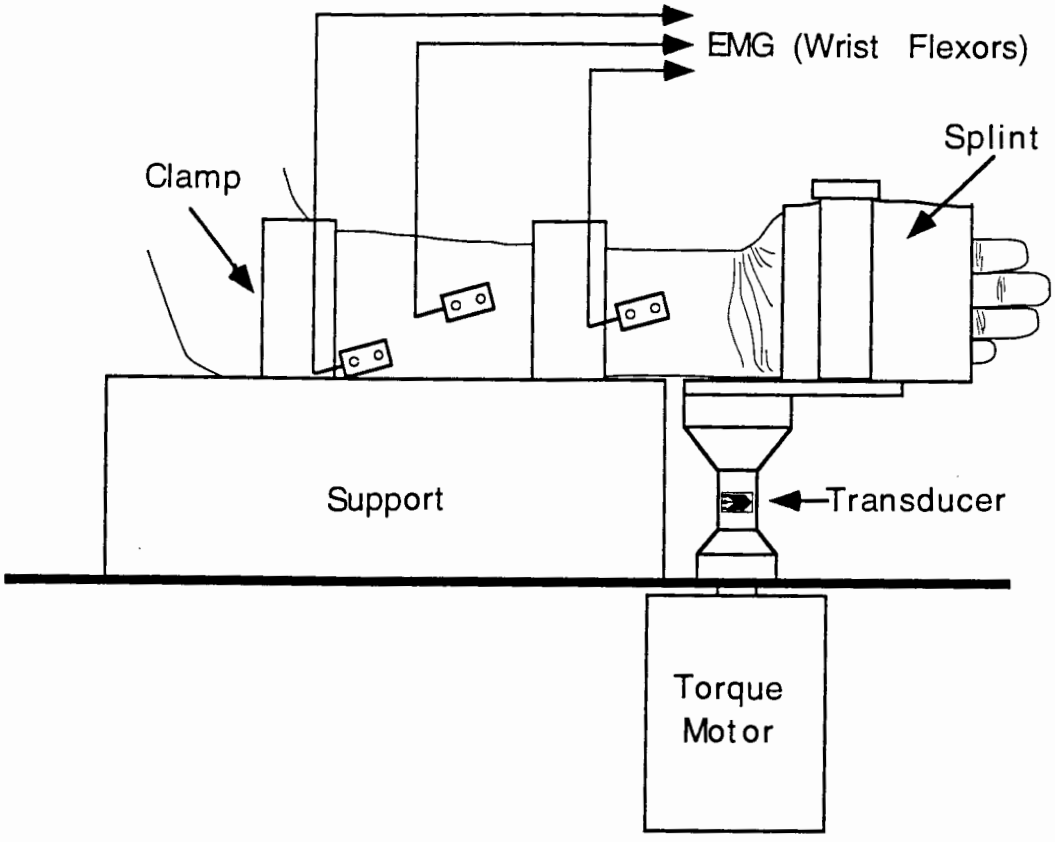

Figure 4.1: Experimental setup for the left wrist.

The hand and fingers were splinted and secured to the manipulandum such that the axis of rotation of the wrist joint was directly over the shaft of the torque motor. A torque transducer measured motor shaft torque. Angular position was measured with a potentiometer in line with the torque motor shaft.

steel, surface electrodes (Liberty Mutual MYO 111) with electrode contacts 3 mm in diameter and 13 mm apart.

Recording

EMG activity was recorded from six forearm muscles contributing to torque at the wrist: FCR, FDS, FCU, ECRL, EDC, and ECU. Optimal placements of the electrodes on the first day of testing (pre-injury) were determined by observing the EMG activity during brisk test movements. These included: ulnar deviation and wrist extension for ECU, ulnar deviation and wrist flexion for FCU. Finger flexion for FDS, finger extension for EDC, wrist flexion (fingers relaxed) for FCR and wrist extension (fingers relaxed) for ECRL. Each electrode was positioned to maximize the signal during the appropriate movement and minimize the signal during others. Electrode position on the skin was marked with indelible ink for reproducible positioning of electrodes on days subsequent to injury. Each day the recorded signal was tested by the above procedure. Active bipolar electrodes with high input impedance and small interelectrode distance were used which minimized the effects of electrode/tissue interface impedance, cross-talk from other muscles and pick-up from distant sources (de la Barrera and Milner 1994). Signals were bandpass filtered from 45 - 550 Hz, amplified and digitized at 2 KHz and stored on disk for later analysis. Position, velocity and torque of the wrist were amplified and digitized at 2 KHz and stored on disk.

Exercise Protocol

The exercise protocol for the wrist extensors was performed with free weights on a apparatus designed in the laboratory. The subject's forearm was supported on an inclined plane. Free weights were attached via a pulley system to the hand of the subject such that only the wrist extensors were used to support the weight. Subjects initially completed a regime of concentric-eccentric wrist extension exercises with 4.54, 3.41 and 2.27 kg, respectively. Repetitions were performed with each weight until subjects were unable to complete a cycle. This regime was repeated 3 times with 5 minutes of rest between each set. Following this series of exercises the subject performed a set of eccentric exercises only. The subject lowered weights of 5.68, 4.54, 3.41 and then 2.27 kg. One weight was used until the subject was unable to lower it in a controlled continuous manner. At this point, the weight was exchanged for the next smaller weight. Following each eccentric contraction, the weight was returned to the starting position by the experimenter. The subject was encouraged to move the wrist through the entire range of motion and perform as well as possible.

Testing Procedures

Following thorough cleaning with alcohol and mild abrasion of the skin, EMG electrodes were placed over the designated muscles. Each subject was seated comfortably in a chair with the left forearm secured and supported at comfortable height and elbow angle. The forearm and wrist were orientated midway between pronation and supination. The hand and fingers were splinted and the splint was rigidly fixed to the manipulandum. The manipulandum allowed

movement of the wrist in flexion-extension only. The hand was positioned such that the axis of rotation of the wrist joint was directly over the shaft of the motor.

Most subjects were familiar with the experiments performed on the manipulandum. Those subjects that were not familiar with a particular task were given the opportunity to practice prior to data collection. To avoid fatigue during the testing session subjects were given at least 30 seconds rest between each trial and up to 5 minutes between different tasks.

The experiments were performed in the following sequence:

1. *Muscle Soreness* . The subject was asked to rate the amount of soreness in the wrist extensors. Soreness was scored on a scale ranging from 0 to 6. Each number corresponding to a verbal description of soreness. The subject was allowed to score in half points. One score was reported while the subject pressed his wrist extensors and one while he moved the wrist; both scores were summed to obtain a single score between 0 and 12 for soreness (see Appendix B).
2. *Range of Motion (ROM).* The mid-range neutral position of the wrist was determined pre-injury and was used in subsequent experiments on each day of testing. The subject's wrist was passively moved into flexion and then extension on the manipulandum. The joint angle, measured from the neutral position, at which resistance to movement was 0.5 Nm was determined for both directions. The total ROM was the sum of the two angles. Three trials were performed.

3. (A) *Maximum voluntary contraction (MVC) - Extension.* The manipulandum was locked at the neutral position of the wrist and the subject performed a sustained (up to 3 seconds) maximal isometric contraction. There was a warm-up period prior to data collection which involved a series of submaximal contractions. Three trials at maximum effort were collected.

   (B) *MVC - Flexion.* As described above for wrist flexion.

4. (A) *Sustained contraction - Extension.* The manipulandum was locked at the neutral position of the wrist and the subject was required to maintain a constant isometric torque for 20 seconds. The torque levels were set at 10%, 30% and 50% of pre-injury wrist extension MVC. The subject was given visual feedback of his torque and the target torque on an oscilloscope. Only one trial was performed for each torque level with adequate rest between trials. This experiment was performed to evaluate the ability to sustain torque output.

   (B) *Sustained contraction - Flexion.* The same protocol was used as described above for extension, but now with torque levels of 10%, 30% and 50% of pre-injury wrist flexion MVC.

5. (A) *Torque tracking - Flexion.* The manipulandum was locked at the neutral position of the wrist and the subject was required to contract the wrist flexors isometrically to produce wrist flexion torque. The torque began at zero and increased to 50% of pre-injury MVC at a constant rate over a period of 5 seconds. The subject was provided with a display of his torque and the target torque on the oscilloscope. The target torque increased at a constant rate and

the subject was instructed to match it. Five trials were collected. This experiment was performed to evaluate the ability to control torque output.

(B) *Torque tracking - Extension.* The subject slowly contracted the wrist extensors over 5 seconds to a wrist extension torque level of 50% pre-injury MVC, tracking a torque target, as described above.

6. *Passive joint stiffness.* The manipulandum was set at the neutral position of the wrist and the subject was instructed to relax completely. Once relaxed, the experimenter triggered a small ramp displacement of 3° in 30 ms in the flexor direction. Any trials in which there was evidence of EMG activity or torque prior to the displacement were not accepted. Five acceptable trials were saved.
7. *Active joint stiffness.* The manipulandum was set at the neutral position of the wrist and the target window on the computer screen was set to one of two torque levels, 10% of pre-injury wrist extension MVC ± 0.5% or 50% of pre-injury wrist extension MVC ± 2.5%. The subject was instructed to contract the wrist extensors and move a cursor representing torque into the target window on the computer screen. Once the subject had held the cursor in the target window for a random period of time ranging from 1 - 2 seconds, the experimenter triggered a ramp displacement, as above. The subject was instructed not to respond to the displacement. Trials in which there was evidence of voluntary intervention were not accepted. Five acceptable trials per torque level were saved.
8. (A) *Maximum velocity - Flexion.* A 1° window representing the neutral position of the wrist was displayed on the computer screen. The subject was instructed

to move a cursor representing the position of the manipulandum into the window. Once in the window the subject was instructed to move the cursor from left to right on the computer screen as rapidly as possible by flexing the wrist. The subject was required to stop the movement within a 3^{o} target window on the right side of the computer screen. The center to center separation of the windows was 30^{o}. The subject was instructed to reduce the oscillations about the target position as quickly as possible. Only five successful trials were saved. For a trial to be successful the wrist had to be stationary within 0.5 degrees of the final position, before 375 ms had elapsed from movement onset. This experiment was performed to evaluate the ability to rapidly recruit motor units.

(B) *Maximum velocity - Extension.* The same protocol was used as described above, but now with movement occurring in the extension direction.

9. *Maximum instability.* In order to determine the limit of stability of the wrist joint, the subject was required to move the manipulandum and hold it in a 1^{o} target window at the neutral position of the wrist, while the torque motor produced a negative spring load, which pushed the manipulandum away from the neutral position in either direction. The limit of stability was determined by systematically increasing the negative stiffness of the load. A trial was considered successful if the manipulandum was brought into the target window within 20 seconds and held there for at least one second. If successful, the negative stiffness of the load was incremented by -0.05 Nm/deg for the next trial. The limit of stability was defined as the largest

negative stiffness that the subject could successfully stabilize. This experiment was performed to evaluate the ability to stabilize the wrist joint by cocontraction.

Analysis

ROM

The total ROM for each of three trials for each subject was averaged. A reduction in passive ROM has been a common finding following exercise-induced muscle injury. This experiment was performed to supplement decline in MVC as a criterion for muscle injury and to quantify changes that occur in muscle which may influence joint stiffness.

MVC

The highest average torque over an interval of 125 ms and corresponding MEMG over an interval of the same duration, advanced by 40 ms were determined from the 3 trials for each subject for all six muscles. Previous studies of exercise-induced muscle injury have shown histological evidence of muscle fiber damage (Fridén et al. 1983a) or demonstrated inflammation in the muscle indicative of damage (MacIntyre et al. 1996) accompanied by a decline in maximal force. For these reasons, a decline in MVC 24 hours after the exercise bout was used as a criterion for muscle injury.

Joint Stiffness

To quantify joint stiffness, the five recorded trials were averaged. From the averaged data, mean values of position and torque were calculated for the interval 0 - 125 ms before the onset of displacement and for the interval 100 - 200 ms after the onset of displacement. These intervals were chosen based on the observation that during these periods the torque and position were constant. The joint stiffness was calculated as the ratio of the change in mean torque divided by the change in mean position.

EMG for each of the three wrist extensor muscles was rectified and averaged for the five chosen trials in each task. The MEMG was calculated for the interval 0 - 125 ms before the stretch as a measure of background activity. Three consecutive intervals beginning 20 ms following the onset of displacement, at short latency (20 - 60 ms), intermediate latency (60 - 100 ms) and long latency (100 - 150 ms) were used for analysis of reflex responses. These intervals were chosen to represent time of occurrence and duration of the myotatic, late myotatic, and post myotatic reflex responses (Jaeger et al. 1982, DeSerres and Milner 1991; Milner et al. 1995). The change in MEMG following the displacement was computed for each interval by subtracting MEMG prior to displacement (background) from the MEMG over the reflex interval. This was expressed as a percentage change by dividing by the background value. The background MEMG was also determined for the wrist flexors.

Sustained Contraction

The median frequency (MF) and MEMG of the EMG signal for each muscle were computed during the first 5 seconds and the last 5 seconds of the 20 second contraction. The torque variance about the target torque was also determined over the entire 20 seconds.

Torque Tracking

The five trials for each subject were individually analyzed for MF and MEMG during each one second interval of the 5 seconds of increasing torque. For MF a 256 point spectrum was computed over an interval of 0.896 s, which allowed 3 spectra to be averaged. The variance about the best fit straight line from beginning to end of the torque tracking was also determined (expressed in $(Nm)^2$). The mean parameter values for the 5 trials were then calculated.

Maximum Velocity

Trials for each subject were analyzed individually to obtain maximum velocity, time of maximum velocity, and time to stabilize in the target window with respect to movement onset. The time to stabilize was determined from movement onset until the wrist was stationary within 0.5 degrees of the final position. The time of onset and time to peak EMG activity with respect to movement onset were determined for all six muscles. IEMG was calculated for the agonist from burst onset to time of peak velocity and for the antagonist from burst onset to time of maximum deceleration. Movement onset was defined as the point at which

velocity exceeded 5 deg/s. The values of each of the above parameters were then averaged for the 5 selected trials to produce a mean value for each subject.

Maximum Instability

The highest negative load stiffness for which a subject successfully completed the task was taken as the limit of stability. MEMG was determined for each muscle in a 250 ms interval while the subject was stable in the target window.

Experiments 2 and 6 were performed to test hypothesis 1. Hypothesis 2 was tested with experiment 7. Hypothesis 3 was tested with experiments 4A, 5B, 8B and 9. Hypothesis 4 was tested with experiments 4A, 5B, 7, 8B and 9. Hypothesis 5 was tested with experiments 3A, 4A, 5B, 7, 8B and 9. Hypothesis 6 was tested with experiments 3A, 3B, 4A, 4B, 5A, 5B, 7, 8A, 8B and 9. Hypothesis 7 was tested with experiments 1, 3, 4A, 5B, 7, 8B and 9.

Statistical Analysis

To test for significant differences over time individual parameters were analyzed using a repeated measures ANOVA design. The test of statistical significance for post-hoc comparisons was set at 0.05. Comparisons between pre-injury data and subsequent days were made using paired t-tests. Due to the number of interactions (4) the level of significance was set at 0.025 to avoid type I error.

Results

It was important to establish that the exercise protocol described in the methods produced injury to the wrist extensor muscles. Although no direct indicators of muscle injury were evaluated the severity of the exercise protocol was felt to be sufficient to produce muscle fiber damage. Studies of exercise-induced muscle injury where damage to muscle fibers has been confirmed by histological and ultrastructural analysis have used few as 20 maximal eccentric contractions (Jones et al. 1986) or involved submaximal eccentric work for 30 minutes (Fridén et al. 1983a). The exercise protocol in the present study involved repeated eccentric contractions by the wrist extensors with submaximal loads, performed until the subject was performing maximally and was unable to move in a controlled continuous manner. The exercise bout varied between subjects, involved approximately 150 repetitions and lasted 25 - 30 minutes. Therefore, the exercise protocol of this study was at least as strenuous as that used in the other studies where injury was verified by direct indicators.

Indirect Measures

Passive ROM was smaller on Day 1 than pre-injury. ROM increased Day 2, followed by continual increase from Day 4 to Day 10 (Figure 4.2). Soreness ratings on a scale of 12 were elevated on Day 1 to a score of 5.45 compared to zero, pre-injury, and reached a maximum of 6.45 on Day 2. Recovery then occurred until Day 10 (Figure 4.3). Wrist extension isometric MVC declined and recovered during the test period (Figure 4.4). The MVC declined by 24.5% from

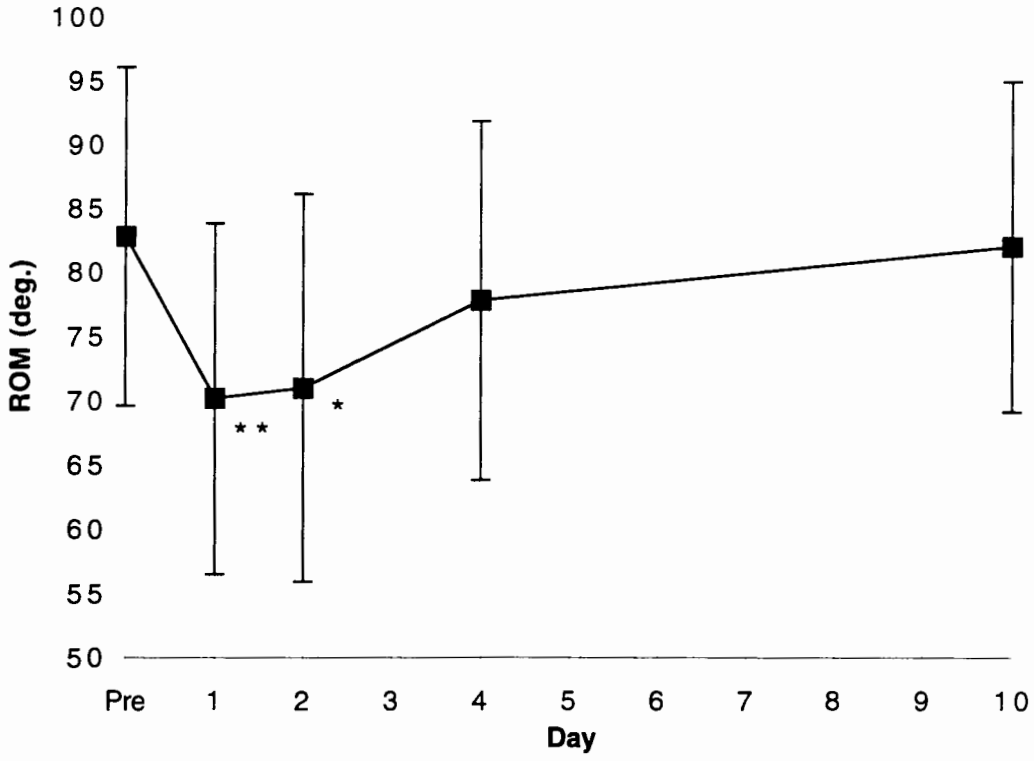

Figure 4.2: Passive wrist range of motion.

Values are means ± SD.
** significantly different than pre-injury $p<0.005$.
* significantly different than pre-injury $p<0.01$.

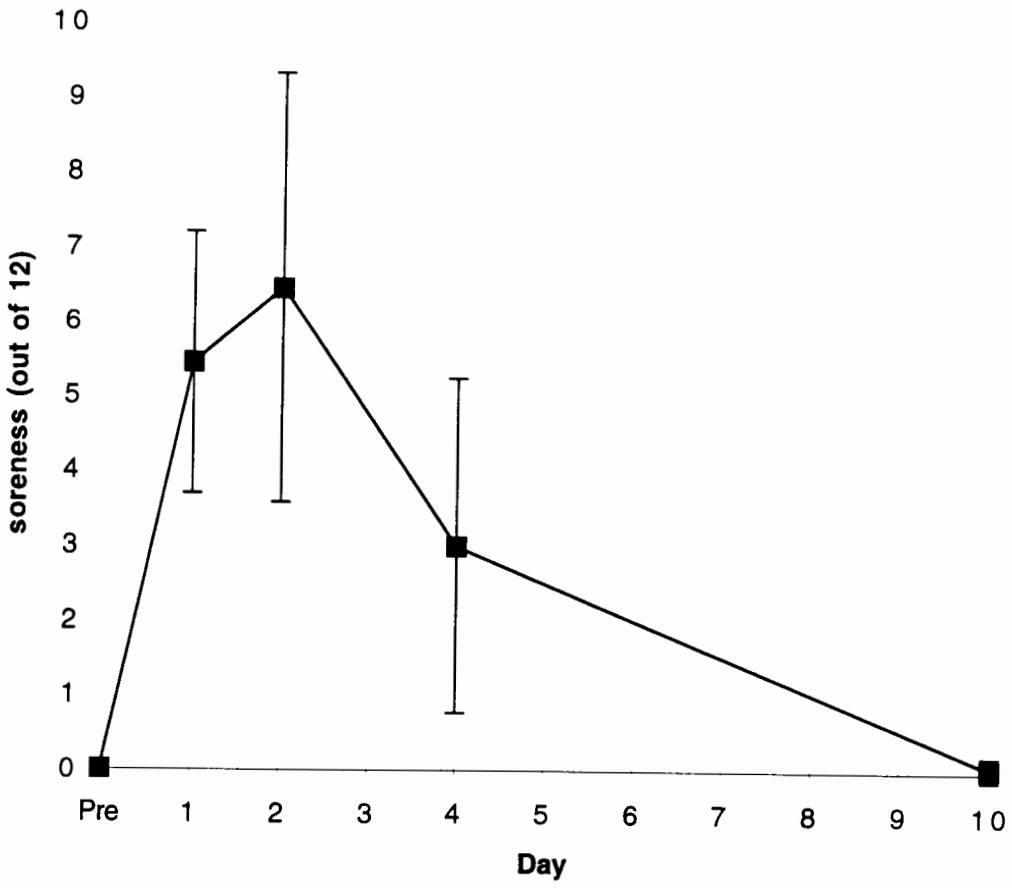

Figure 4.3: Ratings of wrist extensor muscle soreness.

Values are means ± SD.

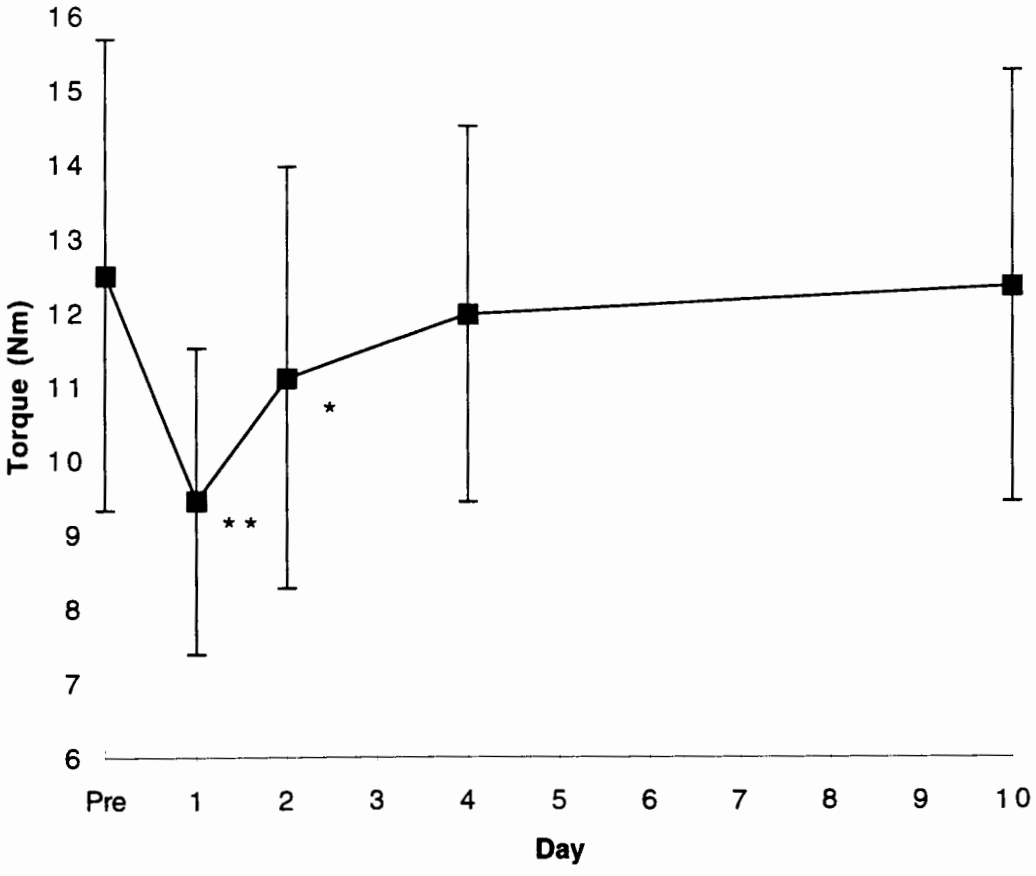

Figure 4.4: Wrist extension MVC.

Values are means ± SD.
** significantly different than pre-injury $p<0.001$.
* significantly different than pre-injury $p<0.005$.

pre-injury to Day 1. There was recovery over the remainder of the test period. MVC on Day 2 was still significantly lower than pre-injury, but by Day 4 was no longer significantly different from pre-injury. MEMG for the wrist extensors and flexors was never significantly different from pre-injury at any time during the period of testing (Table 4.1).

Wrist Joint Stiffness

Passive wrist joint stiffness did not change as a result of eccentric exercise-induced muscle injury (Figure 4.5), nor was there any significant change in the MEMG background activity of the wrist extensor muscles during the passive joint stiffness experiment (Table 4.2). Active wrist joint stiffness at 10% of pre-injury wrist extension MVC did not differ significantly on any day of testing compared to pre-injury (Figure 4.6; Table 4.3). The background MEMG of the ECRL muscle was greater on Day 1 compared to pre-injury ($p<0.005$), but the short latency reflex response in this muscle was lower on Day 1 than pre-injury ($p<0.0005$). The background MEMG of the EDC muscle was greater on Day 1 and Day 2 compared to pre-injury ($p<0.0001$ and $p<0.005$, respectively) as was the background MEMG of the ECU muscle ($p<0.025$). However, the reflex responses for the EDC or ECU muscle were not different following injury. All three wrist extensor muscles showed a similar pattern of change and recovery during the 10 days of testing (Figure 4.7).

Active wrist stiffness at 50% pre-injury wrist extension MVC declined from a pre-injury value of 0.314 Nm/deg to 0.258 Nm/deg on Day 1 ($p=0.05$). On all

TABLE 4.1: Torque and MEMG for wrist extension MVC.

	Pre	Day 1	Day 2	Day 4	Day 10
Torque (Nm)	12.51(3.18)	9.45(2.06)*	11.11(2.84)*	11.97(2.53)	12.35(2.91)
MEMG (μV)					
FCR	5.55(3.08)	4.41(2.11)	6.22(6.02)	4.93(3.17)	6.82(6.79)
FDS	29.01(27.6)	21.36(15.9)	25.92(21.6)	23.59(17.7)	26.56(16.0)
FCU	22.70(10.4)	33.43(55.9)	17.22(5.5)	22.07(7.2)	21.28(9.4)
ECRL	235.5(42.9)	280.1(138.7)	260.9(80.4)	242.2(62.6)	254.2 (56.8)
EDC	309.7(67.7)	333.6(90.8)	341.3(99.3)	321.2(98.3)	314.3(83.7)
ECU	253.2(63.4)	230.1(59.9)	234.4(61.7)	227.3(55.6)	224.0(62.6)

Means with Standard Deviations in ()

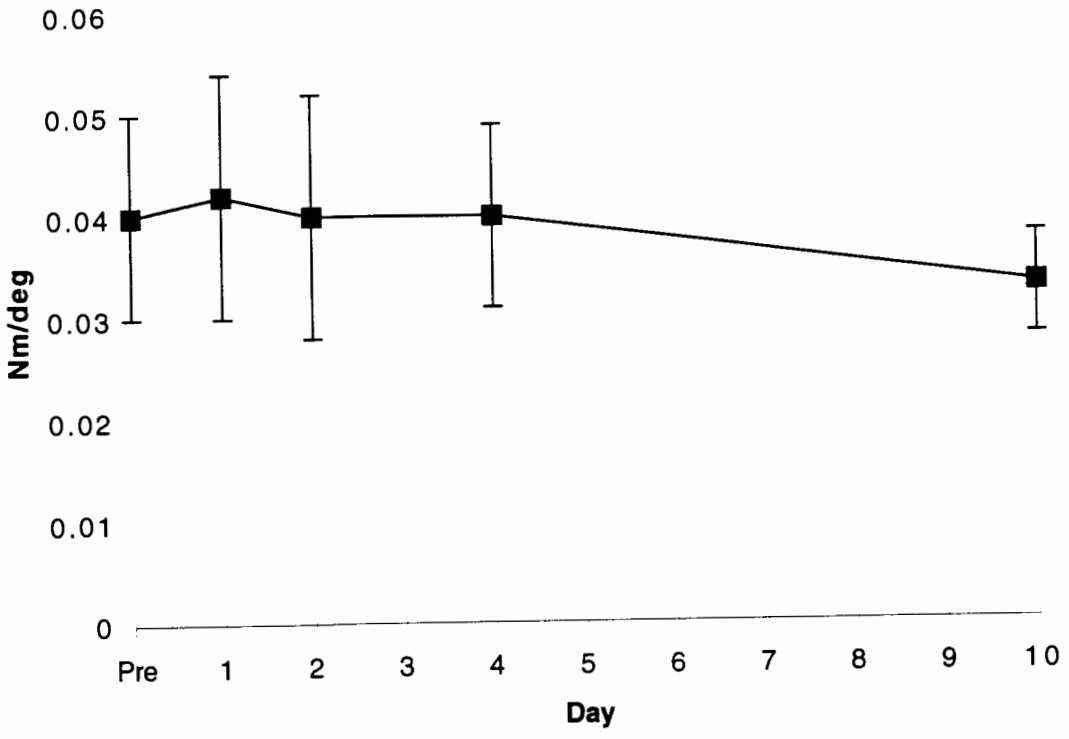

Figure 4.5: Passive wrist joint stiffness.

Values are means ± SD.

TABLE 4.2: MEMG background activity of the wrist extensor muscles during the passive joint stiffness experiment.

	Pre	Day 1	Day 2	Day 4	Day 10
Background MEMG (μV)					
ECRL	2.07(.75)	1.76(.33)	1.66(.36)	1.90(.78)	1.74(.37)
EDC	3.84(1.7)	3.11(.70)	3.03(.47)	3.07(.54)	2.87(.46)
ECU	2.75(.39)	2.77(.44)	2.64(.15)	2.68(.21)	2.64(.31)

Means with Standard Deviations in ()

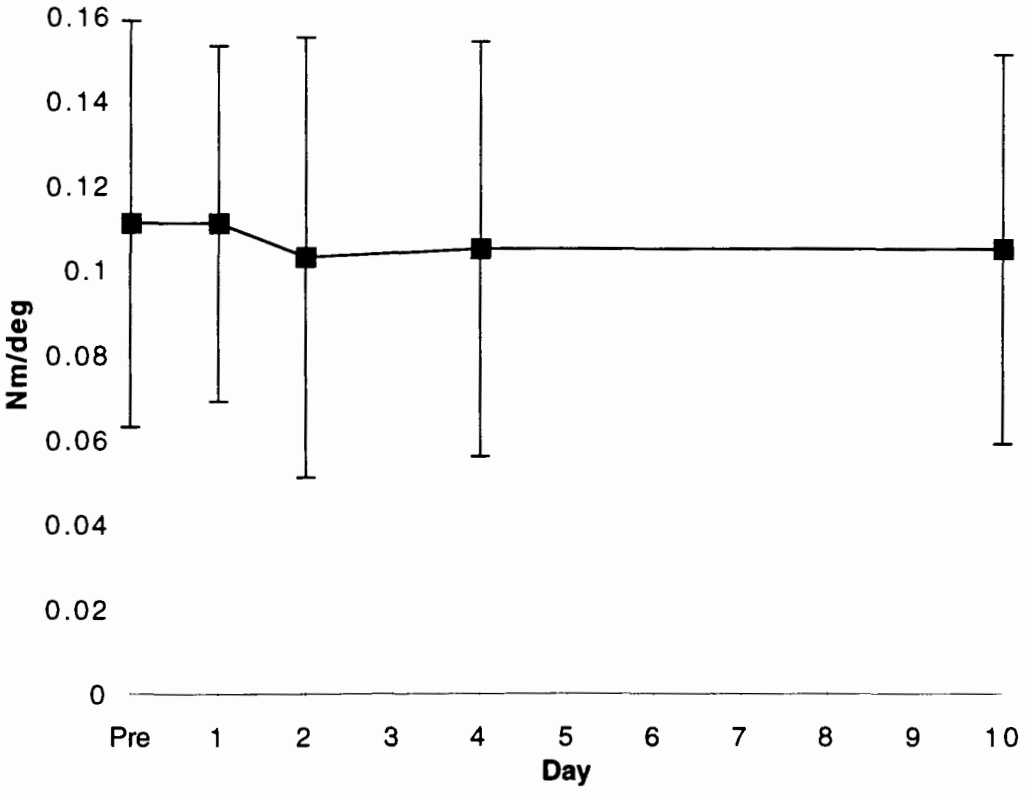

Figure 4.6: Active wrist joint stiffness at 10% of pre-injury wrist extension MVC. Values are means ± SD.

TABLE: 4.3: Wrist joint stiffness and wrist extensor muscles MEMG at 10% of pre-injury wrist extension MVC.

	Pre	Day 1	Day 2	Day 4	Day 10
Wrist Stiffness (Nm/deg)	.111(.05)	.111(.04)	.103(.05)	.105(.05)	.105(.05)
ECRL					
Background MEMG (μV)	41.10(20.9)	72.58(25.1)*	46.84(9.03)	44.34(13.2)	32.99(15.09)
Short Latency (20 - 60 ms)	55.4(14.7)	28.4(20.9)*	41.3(26.1)	60.0(31.6)	59.5(30.0)
Intermediate Latency (60 - 100 ms)	41.6(36.5)	35.1(33.6)	48.8(39.4)	38.9(48.2)	28.1(34.5)
Long Latency (100 - 150 ms)	18.3(25.0)	15.9(31.3)	13.4(25.1)	3.1(29.7)	8.6(19.0)
EDC					
Background MEMG	56.87(21.9)	97.27(34.6)*	71.26(26.9)*	58.55(29.6)	57.60(19.1)
Short Latency (20 - 60 ms)	25.6(14.2)	21.6(26.4)	23.3(15.2)	28.0(12.4)	16.3(12.0)
Intermediate Latency (60 - 100 ms)	42.9(19.2)	39.2(18.2)	43.7(28.7)	43.7(29.8)	31.1(26.0)
Long Latency (100 - 150 ms)	10.2(15.6)	15.0(15.4)	6.8(20.1)	3.3(21.1)	2.4(17.1)
ECU					
Background MEMG	48.08(20.2)	66.13(24.5)*	54.97(20.2)	49.18(17.5)	42.25(11.4)
Short Latency (20 - 60 ms)	23.1(18.0)	22.0(9.9)	18.8(22.8)	22.6(27.2)	17.6(15.9)
Intermediate Latency (60 - 100 ms)	27.1(25.6)	41.2(23.9)	33.7(21.4)	31.4(20.7)	31.5(12.7)
Long Latency (100 - 150 ms)	5.7(17.3)	9.8(15.6)	-1.0(21.9)	-1.2(15.3)	-3.6(15.3)

Means with Standard Deviations in ()

Reflex responses as a percentage change in Background MEMG

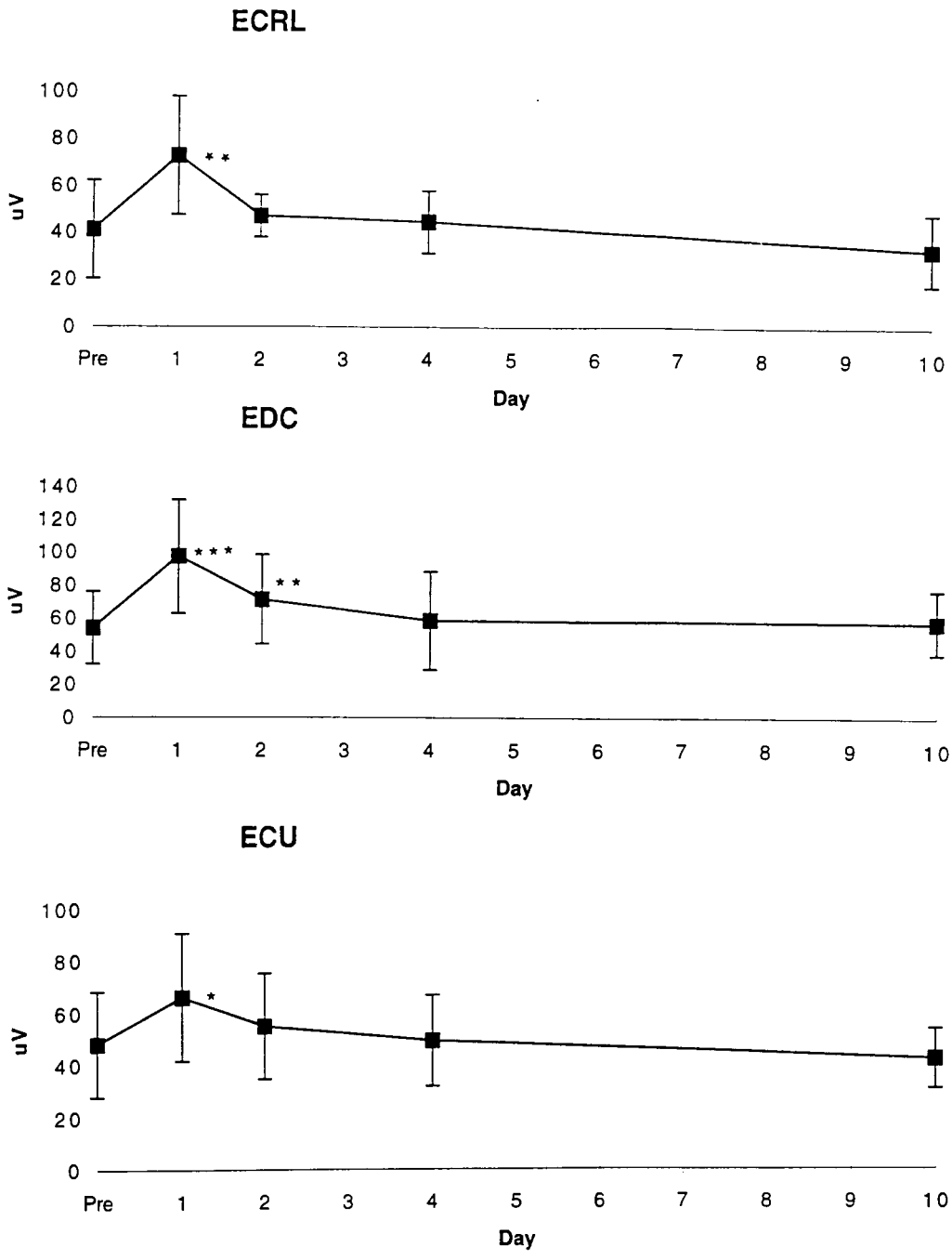

Figure 4.7: Background MEMG of the wrist extensor muscles at 10% of pre-injury wrist extension MVC.

Values are means ± SD.
*** significantly different than pre-injury $p<0.0001$.
** significantly different than pre-injury $p<0.005$.
* significantly different than pre-injury $p<0.025$.

other days of testing the values were no different from pre-injury (Figure 4.8). The background MEMG of the EDC muscle was significantly greater on Day 1 and Day 2 compared to pre-injury ($p<0.005$ and $p<0.01$, respectively; Table 4.4). The background MEMG of the ECRL and ECU muscles was also greater on Day 1. However, the difference was not significant. The pattern of increase in background MEMG and recovery during the period of testing was similar for all three wrist extensors (Figure 4.9). The background MEMG of all three wrist flexor muscles was also greater on Day 1 compared to pre-injury (Table 4.4) indicating more co-contraction of antagonist muscles.

The short latency reflex response of the EDC muscle was significantly lower on Day 1 compared to pre-injury ($p<0.025$). It was also lower in the other two wrist extensors, but the difference was not significant (Figure 4.10). There were no significant differences in any of the other reflex responses of the wrist extensors. The response to a rapid displacement of 3° at a background torque of 50% MVC for one subject pre-injury and on Day 1 is shown in Figure 4.11.

Wrist Extension Experiments

The torque variance during tracking of a target torque is shown in Figure 4.12. The variance on Day 1 was double that pre-injury ($p=0.0316$; Table 4.5), whereafter it declined. Figure 4.13 shows the torque traces for one subject pre-injury and on Day 1. It is apparent that the torque increased less smoothly on Day 1, and this impairment became more pronounced as the subject reached higher torques. The MEMG of the ECRL and EDC muscles during the first

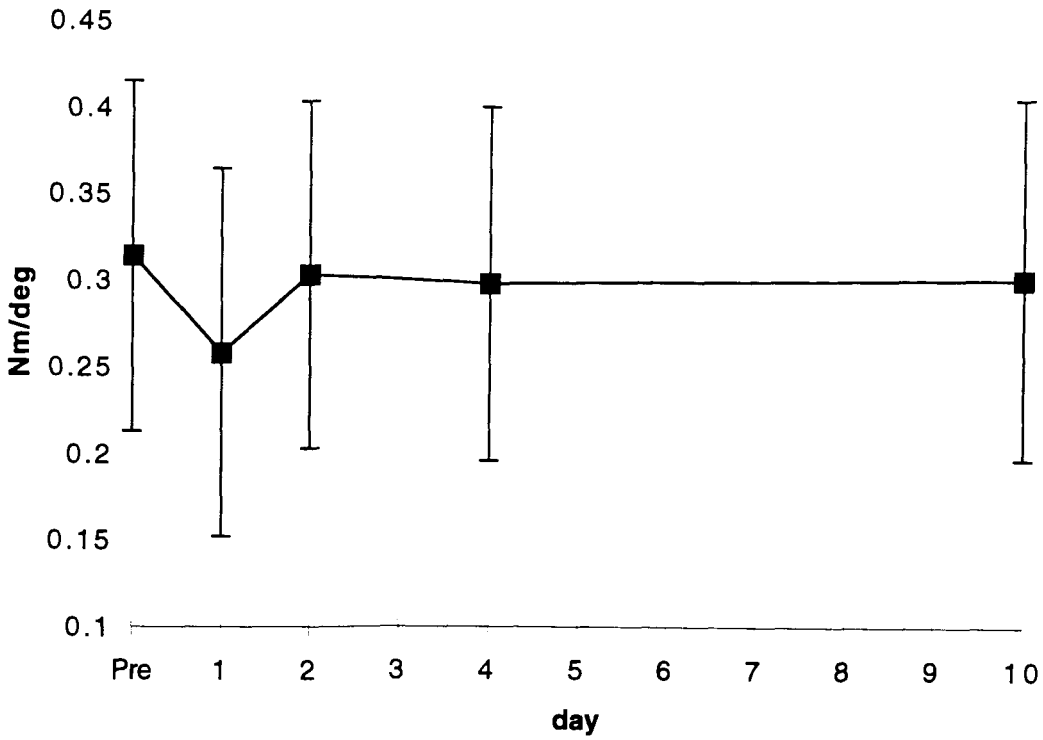

Figure 4.8: Active wrist joint stiffness at 50% of pre-injury wrist extension MVC. Values are means ± SD.

TABLE 4.4: Wrist joint stiffness and MEMG at 50% of pre-injury wrist extension MVC.

	Pre	Day 1	Day 2	Day 4	Day 10
Wrist Stiffness (Nm/deg)	.314(.10)	.258(.11)	.303(.10)	.298(.10)	.301(.10)
ECRL					
Background MEMG (μV)	143.6(80.3)	198.3(93.4)	175.5(42.4)	164.9(67.8)	118.7(72.9)
Short Latency (20 - 60 ms)	18.9(18.4)	13.0(23.7)	21.7(22.0)	21.6(18.2)	30.6(19.8)
Intermediate Latency (60 - 100 ms)	27.1(19.8)	29.3(18.5)	43.0(23.3)	31.0(25.9)	26.4(18.3)
Long Latency (100 - 150 ms)	8.7(13.4)	7.8(14.4)	16.7(15.4)	16.0(17.9)	27.3(23.0)
EDC					
Background MEMG	175.3(46.2)	291.6(85.8)*	242.3(63.4)*	214.6(68.5)	164.7(56.6)
Short Latency	14.1(7.6)	-1.3(12.9)*	7.9(8.4)	9.4(11.3)	12.2(13.2)
Intermediate Latency	27.4(18.0)	17.4(15.0)	19.4(12.9)	30.1(19.0)	28.3(18.5)
Long Latency	14.2(21.8)	10.5(11.7)	9.7(8.2)	18.1(18.3)	12.9(20.5)
ECU					
Background MEMG	148.2(25.4)	184.2(75.8)	165.5(38.7)	147.9(25.9)	123.2(25.6)
Short Latency	10.6(15.8)	5.0(17.1)	6.2(13.9)	11.6(14.6)	10.1(15.1)
Intermediate Latency	30.5(17.8)	18.2(19.2)	29.6(11.8)	32.2(20.4)	28.7(13.2)
Long Latency	7.3(16.6)	9.1(9.1)	10.3(13.5)	5.2(19.1)	11.7(20.6)
FCR					
Background MEMG	1.98(.45)	3.44(1.2)*	2.75(.74)	2.44(1.0)	1.85(.33)
FDS					
Background MEMG	6.76(7.1)	18.72(14.1)*	15.1(10.3)	13.2(9.6)	13.07(5.7)
FCU					
Background MEMG	10.55(2.9)	15.44(3.7)*	11.98(2.6)	11.17(2.6)	9.74(2.7)

Means with Standard Deviations in ()

Reflex responses as a percentage change in Background MEMG

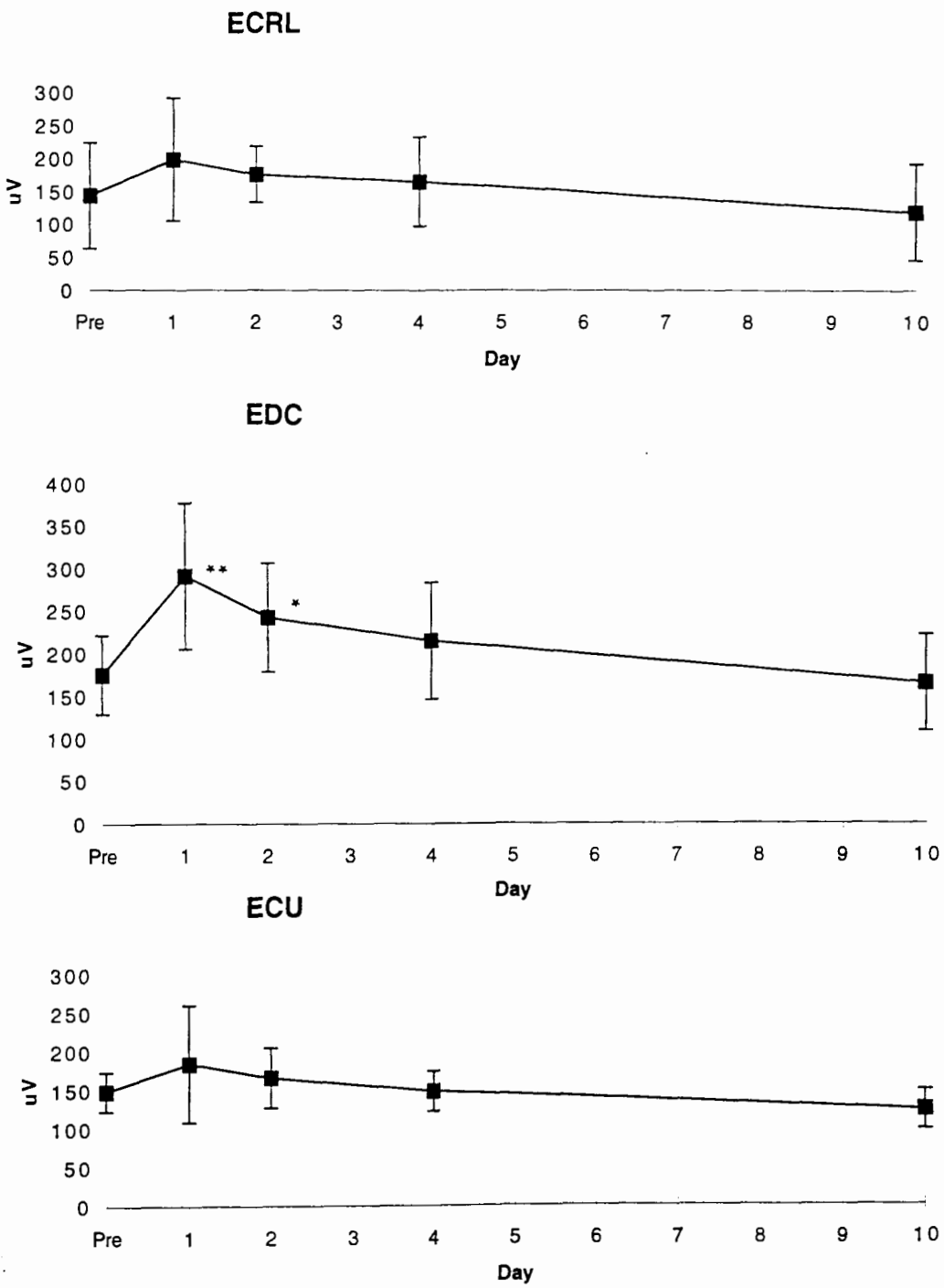

Figure 4.9: Background MEMG of the wrist extensor muscles at 50% of pre-injury wrist extension MVC.

Values are means ± SD.
** significantly different than pre-injury $p<0.001$.
* significantly different than pre-injury $p<0.01$.

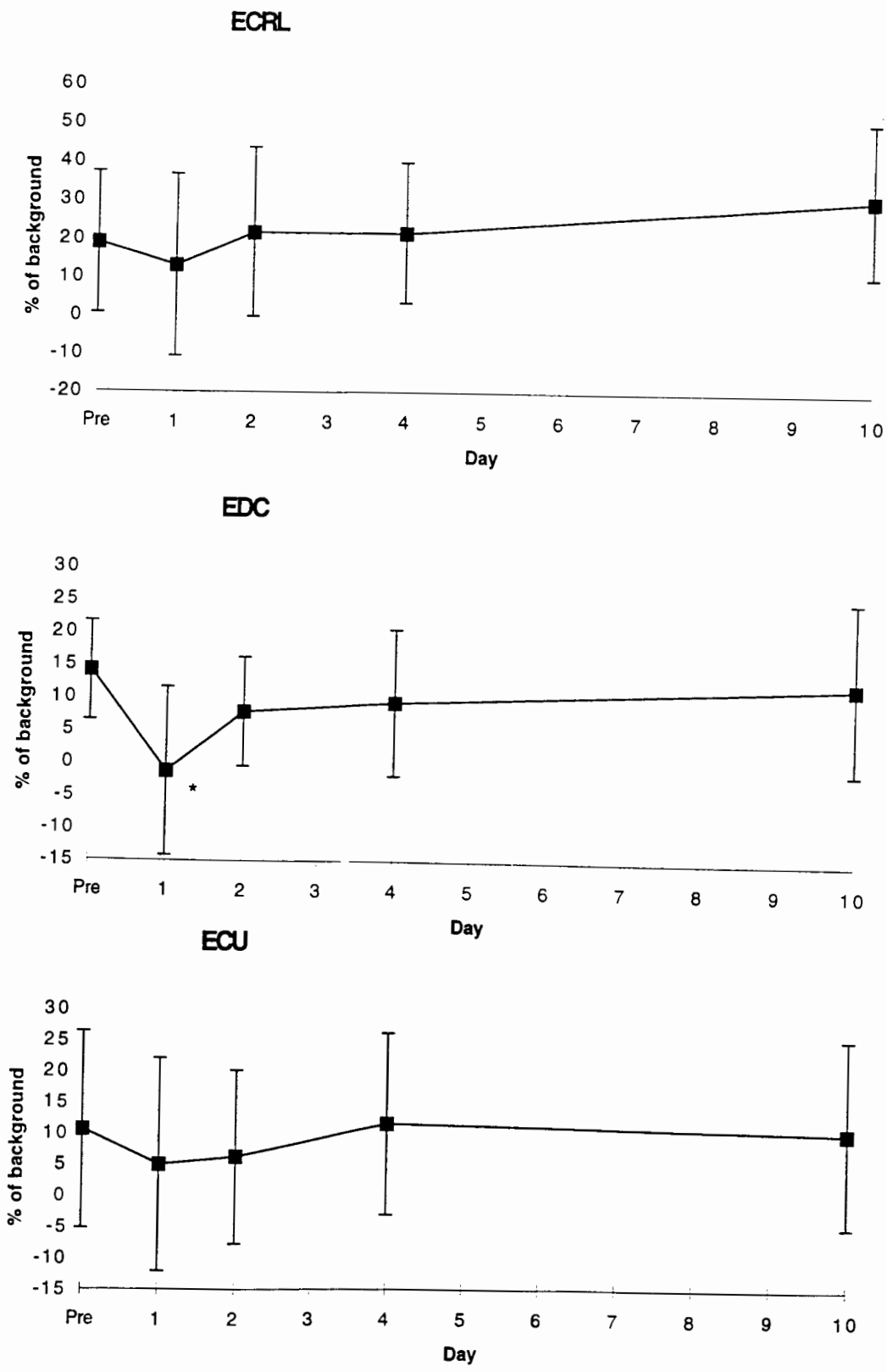

Figure 4.10: Short latency reflex response of the wrist extensor muscles at 50% of pre-injury wrist extension MVC.

Values are means ± SD.

* significantly different than pre-injury $p<0.025$

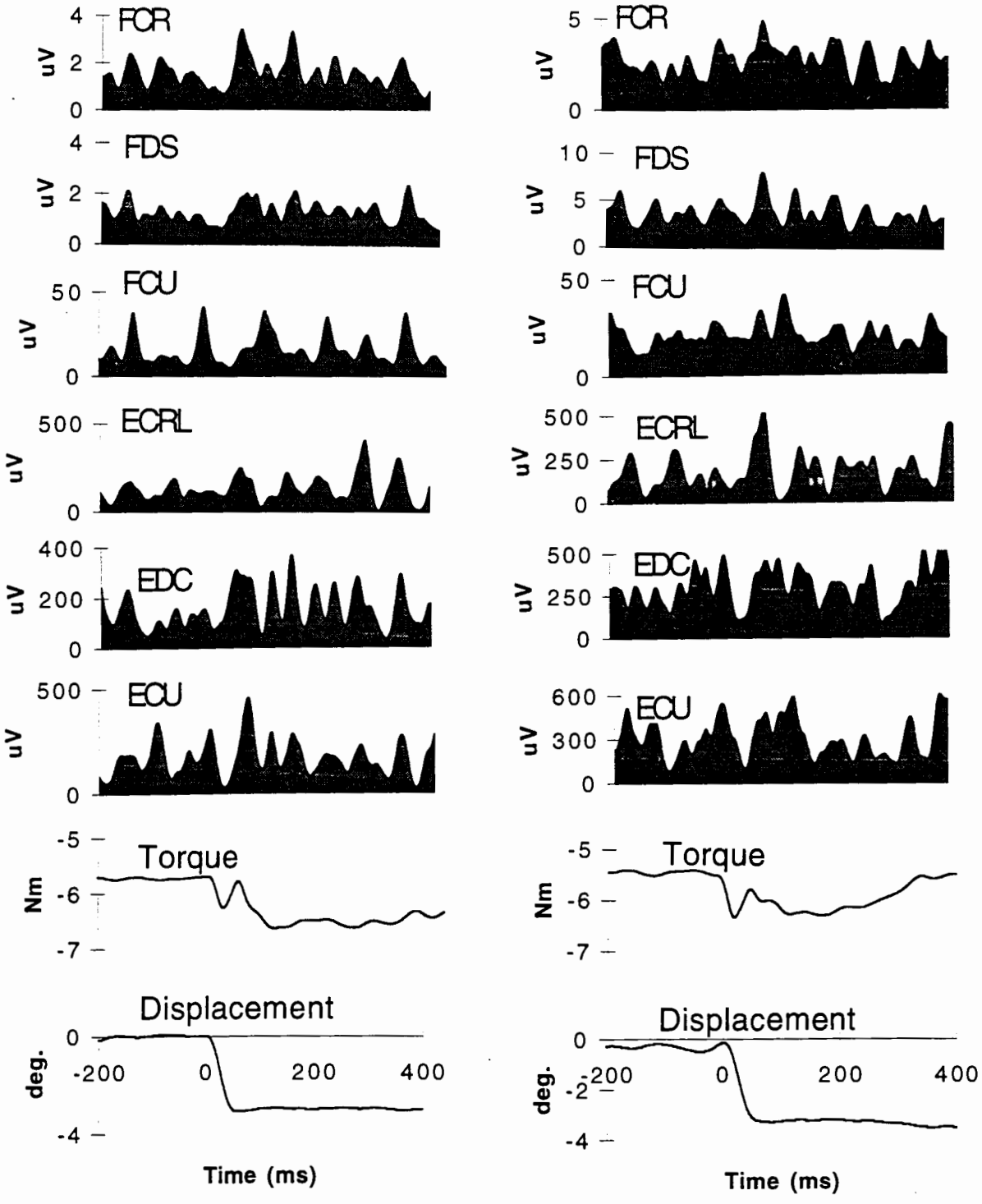

Figure 4.11: The response to rapid displacement at 50% of pre-injury wrist extension MVC of one subject Pre-injury (L) and Day 1 (R).

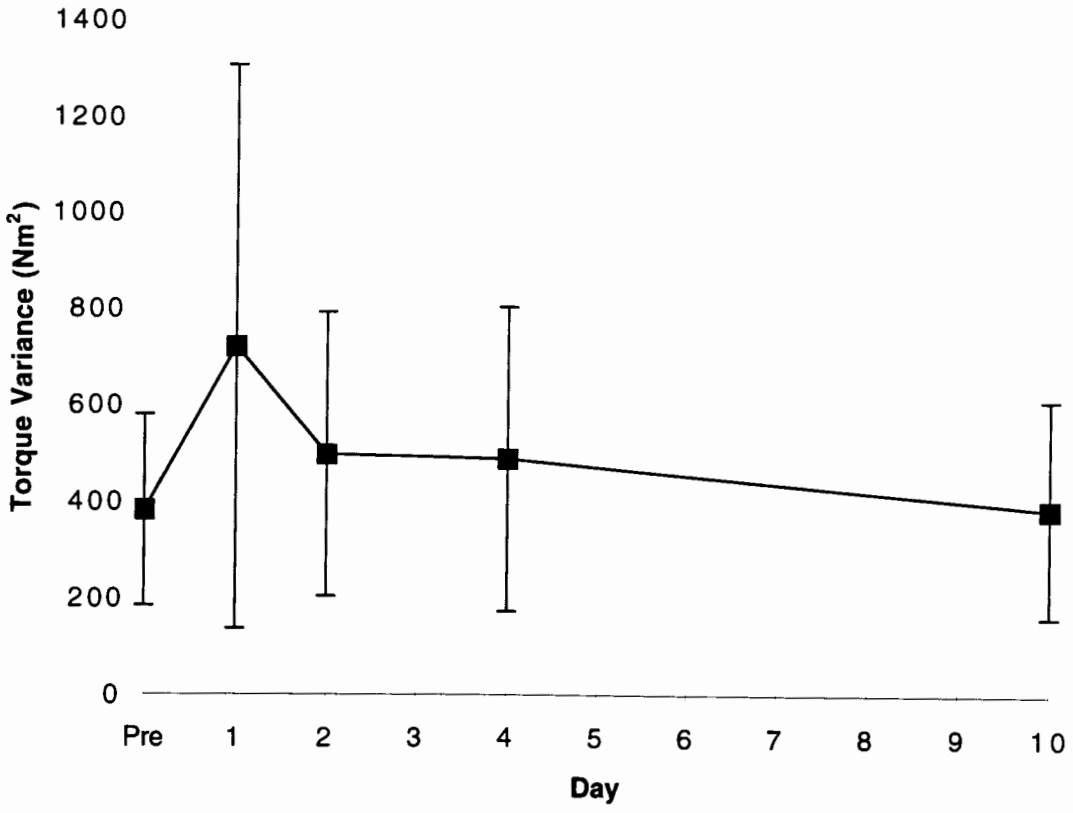

Figure 4.12: Torque variance while tracking extensor torque changing at constant rate.

Values are means ± SD.

TABLE 4.5: Torque variance, MEMG and median frequency while tracking extensor torque changing at constant rate.

	Pre	Day 1	Day 2	Day 4	Day 10
Variance (Nm^2)	382.3(197.8)	721.1(584.7)	498.1(294.1)	489.1(315.6)	386.7(225.0)
ECRL					
MEMG Initial (μV)	30.05(13.6)	51.77(20.6)*	39.22(15.7)	34.62(14.4)	27.61(13.8)
MEMG Final	139.7(88.0)	210.8(71.2)*	161.5(40.3)	144.2(36.0)	121.1(61.6)
MF Initial (Hz)	170.7(26.5)	169.6(16.8)	163.7(24.8)	162.5(17.0)	171.2(21.1)
MF Final	206.4(15.1)	199.6(14.9)	197.5(6.4)	195.9(18.0)	204.3(15.1)
EDC					
MEMG Initial	32.03(11.9)	73.41(26.3)*	55.18(23.3)*	43.85(18.6)	41.12(12.0)
MEMG Final	156.3(89.6)	274.9(89.4)*	197.7(45.8)	152.2(42.6)	149.2(52.4)
MF Initial	172.3(22.8)	165.8(16.8)	168.2(16.7)	169.8(18.0)	168.5(18.9)
MF Final	178.1(19.2)	171.6(24.9)	163.7(15.7)	171.9(26.3)	170.9(18.5)
ECU					
MEMG Initial	35.02(13.9)	48.82(26.2)	41.91(22.9)	33.49(20.2)	35.06(20.3)
MEMG Final	137.5(39.5)	175.2(63.9)	163.9(57.9)	142.6(43.4)	136.1(46.7)
MF Initial	204.3(16.8)	197.6(11.1)	185.7(11.9)*	196.4(15.1)	193.7(15.3)
MF Final	211.1(18.6)	203.4(14.3)	199.1(19.5)	202.9(18.6)	197.8(20.6)
FCR					
MEMG Initial	.531(.14)	.799(.29)	1.22(1.7)	.678(.28)	.671(.33)
MEMG Final	1.68(.28)	3.15(.95)*	2.99(1.5)	2.22(1.2)	2.05(1.1)
FDS					
MEMG Initial	2.83(2.5)	4.49(3.9)	4.08(3.15)	3.01(2.15)	3.41(1.7)
MEMG Final	9.76(10.7)	18.14(15.1)	14.7(11.3)	11.40(8.5)	13.77(4.6)
FCU					
MEMG Initial	4.43(.54)	4.62(.82)	5.70(4.4)	4.54(.74)	4.48(1.1)
MEMG Final	10.65(5.6)	12.67(2.4)	12.15(5.1)	8.95(1.7)	8.30(1.5)

Means with Standard Deviations in ()
Initial - first second, Final - fifth second

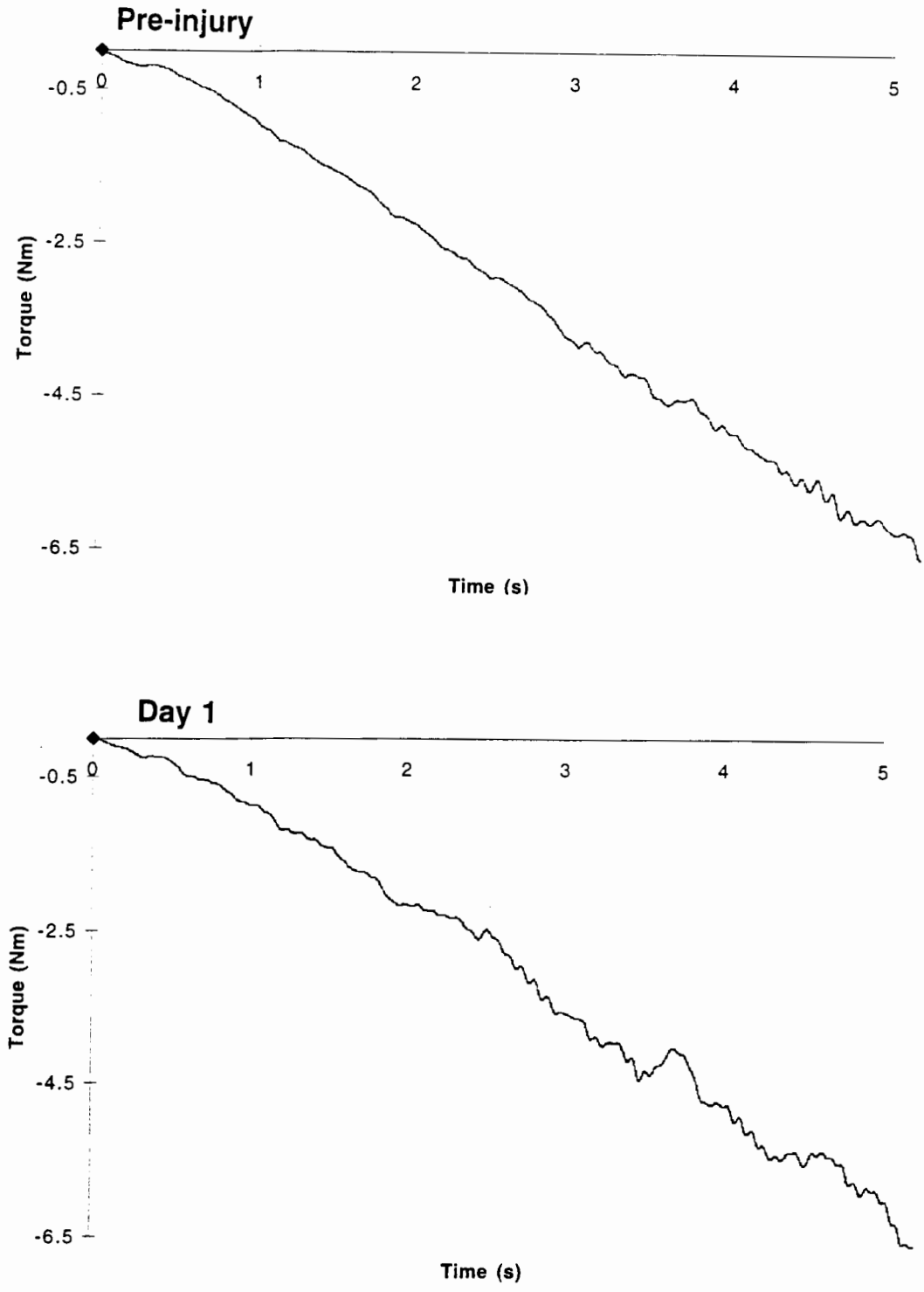

Figure 4.13: The torque recorded for one subject while tracking extensor torque changing at constant rate is shown Pre-injury and Day 1.

second of torque tracking (initial) was significantly greater on Day 1 than pre-injury ($p<0.01$ and $p<0.0005$, respectively).The initial MEMG of the EDC was also significantly greater than pre-injury on Day 2 ($p<0.01$). The MEMG of the ECRL and EDC muscles during the fifth second of torque tracking (final) was significantly greater on Day 1, as well ($p<0.005$, in both cases). The MEMG of the ECU muscle during the initial and final seconds was also greater on Day 1 than pre-injury but the difference was not significant. All three muscles demonstrated a similar time course of elevated activity and recovery over the ten days of testing. Among the flexor muscles, only the MEMG of the FCR muscle during the final second on Day 1 was significantly different than pre-injury ($p<0.005$) suggesting only a small effect on the antagonist muscles. The only significant effect on MF was a lower initial MF of the ECU on Day 2 compared to pre-injury ($p<0.005$).

The torque variances for sustained contractions at 10%, 30% and 50% of pre-injury MVC are shown in Figure 4.14. The amount of torque variance was significantly greater on Day 1 compared to pre-injury for all three levels of torque ($p<0.005$; Table 4.6) and returned to pre-injury levels by Day 4. Figure 4.15 compares the torque variance for the three torque levels between pre-injury and Day 1. It is evident that the torque variance on Day 1 becomes proportionately greater as the torque increases. The pre-injury and Day 1 torque traces for one subject at 10% and 50% of MVC are shown in Figure 4.16. The torque trace variation is most pronounced during the task requiring 50% MVC. Table 4.7 presents the MEMG and MF for the three wrist extensors at 10% and 50%

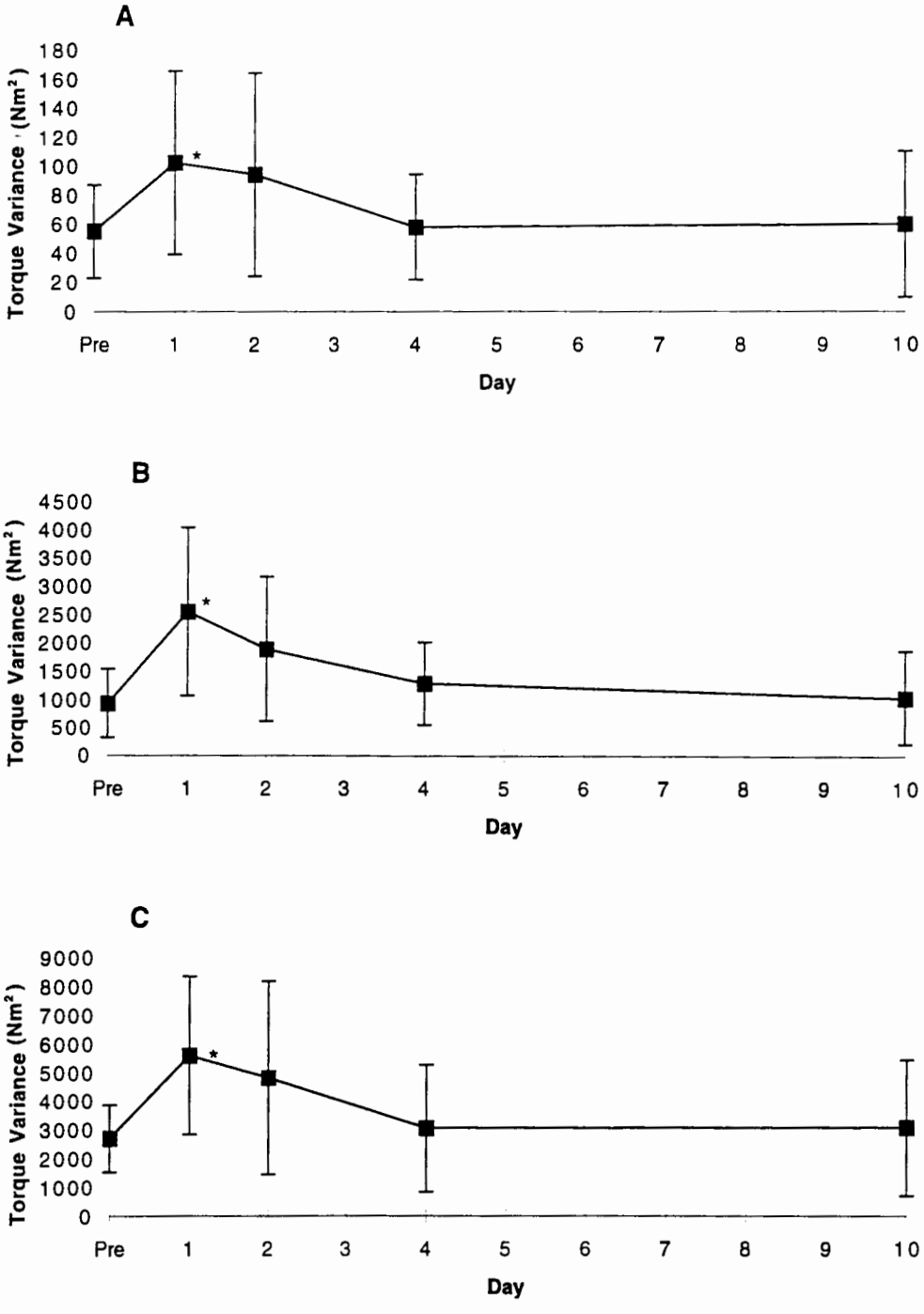

Figure 4.14: The torque variances for sustained contractions at (**A**) 10%, (**B**) 30% and (**C**) 50% of pre-injury wrist extension MVC.

Values are means ± SD.
* significantly different than pre-injury $p<0.005$.

TABLE 4.6: The torque variances for sustained contractions at 10%, 30% and 50% of pre-injury wrist extension MVC.

	Pre	Day 1	Day 2	Day 4	Day 10
Torque Variance (Nm^2)					
10% Extension MVC	54.98(32.1)	102.5(62.3)*	94.29(70.2)	58.17(36.5)	60.14(50.4)
30% Extension MVC	927.6(603.3)	2553.5(1485)*	1894.3(1279)	1288.9(731.7)	1043.5(825.5)
50% Extension MVC	2696.0(1159)	5588.9(2757)*	4802.1(3361)	3050.4(2217)	3070.1(2368)

Means with Standard Deviations in ()

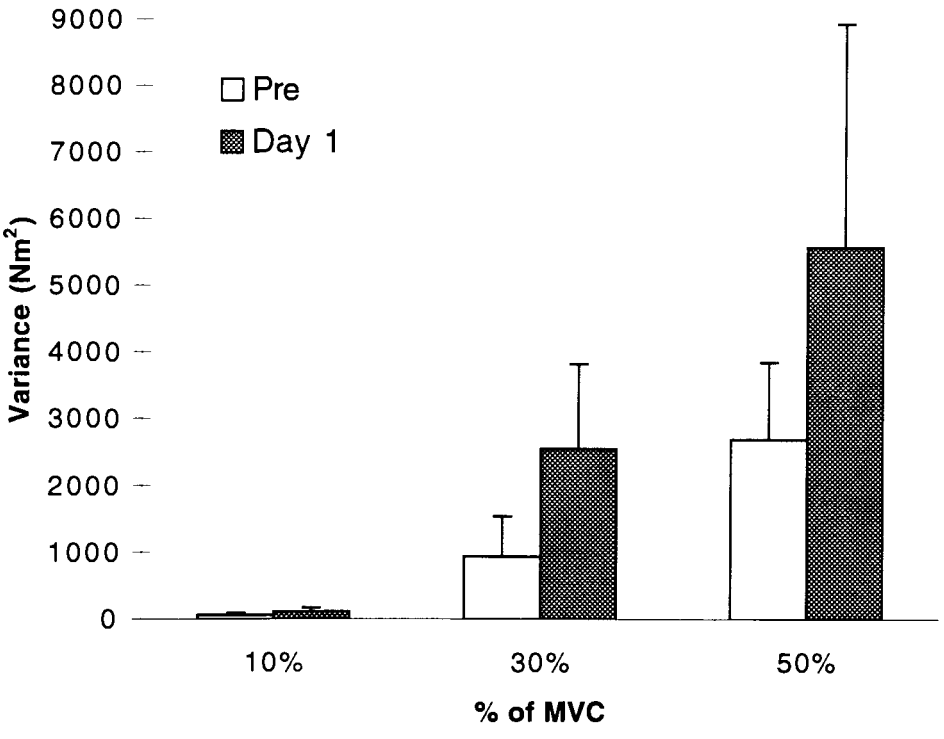

Figure 4.15: The torque variance for sustained contractions at 10%, 30% and 50% of pre-injury wrist extension MVC pre-injury and Day 1.

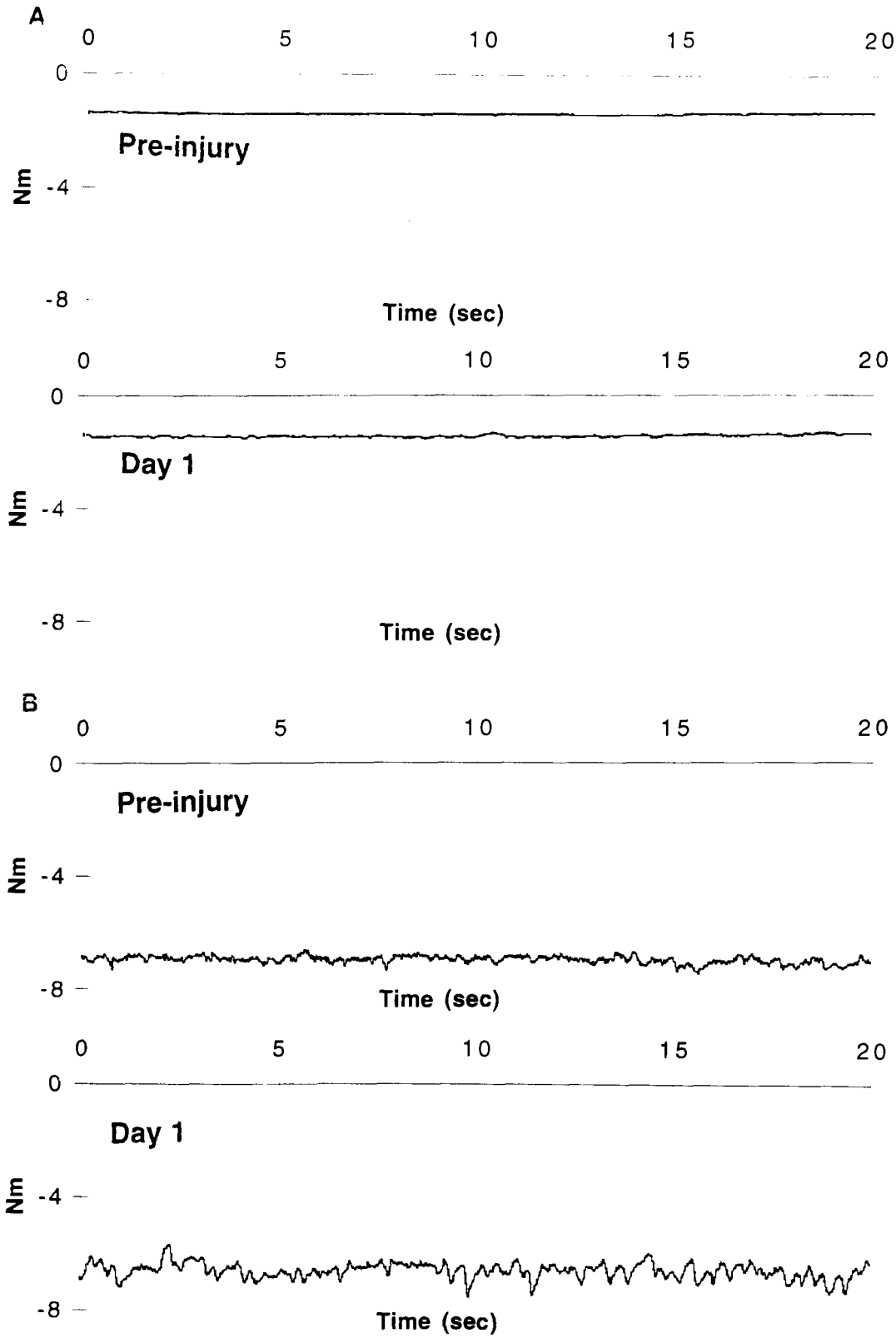

Figure 4.16: The pre-injury and Day 1 torque traces for one subject for a sustained contraction at (**A**) 10% and (**B**) 50% of pre-injury wrist extension MVC.

TABLE 4.7: The MEMG and MF of the wrist extensor muscles for a sustained contraction at 10% and 50% of pre-injury wrist extension MVC.

		Pre	Day 1	Day 2	Day 4	Day 10
ECRL						
10% Extension MVC						
	MEMG Initial (μV)	33.54(16.4)	53.05(14.3)*	45.53(15.4)*	41.51(13.8)	31.57(15.2)
	MEMG Final	31.27(14.4)	46.79(13.9)*	43.74(13.9)*	37.84(12.1)	30.29(11.8)
	MF Initial (Hz)	169.5(18.3)	162.3(23.8)	163.6(28.0)	165.4(18.7)	163.1(30.4)
	MF Final	164.4(18.5)	158.9(19.4)	157.3(25.3)	159.2(20.4)	159.8(26.0)
50% Extension MVC						
	MEMG Initial	128.3(84.4)	190.5(64.5)	163.8(46.1)	140.6(38.2)	117.8(47.4)
	MEMG Final	104.0(56.9)	145.6(54.4)	133.5(36.8)	117.6(34.7)	100.8(37.2)
	MF Initial	167.2(19.6)	159.6(21.5)	144.3(19.5)	157.2(23.1)	157.2(27.6)
	MF Final	155.3(22.8)	147.3(24.6)	140.6(23.4)	147.1(23.1)	155.8(27.3)
EDC						
10% Extension MVC						
	MEMG Initial	44.33(26.5)	81.14(29.5)*	60.51(33.1)	46.9(20.7)	48.29(18.76)
	MEMG Final	48.92(25.3)	82.29(35.0)*	62.87(28.9)	49.14(18.5)	48.49(18.0)
	MF Initial	180.3(20.2)	169.7(20.7)	155.8(13.2)*	167.6(29.5)*	160.7(31.7)
	MF Final	174.6(17.9)	167.6(22.9)	160.4(17.7)	166.0(21.7)	161.1(23.6)
50% Extension MVC						
	MEMG Initial	155.2(73.7)	275.5(86.2)*	210.0(57.1)*	171.6(59.7)	177.5(75.8)
	MEMG Final	159.9(52.5)	247.1(64.5)*	202.0(55.9)	183.2(56.6)	171.5(68.8)
	MF Initial	164.8(17.6)	160.3(20.7)	160.1(19.0)	157.6(24.0)	162.3(21.8)
	MF Final	150.8(23.4)	141.2(24.8)	142.8(18.9)	142.2(23.9)	141.2(27.9)
ECU						
10% Extension MVC						
	MEMG Initial	34.96(1104)	49.33(26.6)	47.21(26.2)	34.77(16.9)	34.4(16.8)
	MEMG Final	38.05(10.6)	48.89(22.8)	45.49(21.2)	36.72(17.0)	39.96(19.9)
	MF Initial	201.9(14.9)	196.5(19.2)	189.0(19.9)	189.3(14.1)	189.1(18.5)
	MF Final	194.7(19.0)	190.8(13.8)	188.6(19.5)	188.5(15.6)	183.4(22.9)
50% Extension MVC						
	MEMG Initial	120.8(33.0)	169.7(57.4)*	162.5(54.8)	127.1(23.8)	120.6(37.4)
	MEMG Final	130.9(38.0)	169.7(56.9)*	146.2(48.8)	134.3(26.5)	117.7(40.1)
	MF Initial	203.5(22.1)	202.1(15.1)	189.7(14.7)	192.0(14.8)	190.4(12.9)
	MF Final	192.4(20.4)	175.9(17.4)	171.0(15.2)	179.5(22.8)	174.2(12.9)*

Means with Standard Deviations in (); Initial - first second, Final - fifth second

MVC. For the sustained contraction at 10% MVC, the Day 1 initial MEMG of the ECRL and EDC muscles (first 5 seconds) was significantly greater than pre-injury ($p<0.005$ and $p<0.01$, respectively). The initial MEMG of the ECRL remained significantly higher on Day 2 ($p<0.025$). The final MEMG of the ECRL (last 5 seconds) was also significantly higher on Day 1 and 2 ($p<0.025$ and $p<0.01$) compared to pre-injury, while the MEMG of the EDC muscle was higher on Day 1 only ($p<0.005$). Although the changes in activity of the ECU muscle were never significantly different from pre-injury they did exhibit the same pattern of change over the ten days as the other two muscles (Figures 4.17 and 4.18). The initial MF of the EDC was lower on Day 2 and Day 4 for the sustained contraction at 10% of MVC compared to pre-injury. Otherwise, the initial and final MF of the three wrist extensors were not significantly different from pre-injury.

For the sustained contraction at 50% MVC, the Day 1 initial MEMG was significantly greater for the EDC and ECU muscles compared to pre-injury ($p<0.0001$ and $p<0.005$, respectively). The initial MEMG of the EDC remained significantly higher on Day 2 ($p<0.025$). The final MEMG was also significantly higher in the EDC and ECU muscles on Day 1 ($p<0.0005$ and $p<0.005$, respectively). Although the changes in activity of the ECRL muscle were never significantly different from pre-injury, they did show the same pattern over the ten days as the other two muscles (Figures 4.19 and 4.20). There were no significant changes in the initial or final MF of the three wrist extensors during the 10 days of testing. The initial and final MEMG of the FCR muscle were also significantly greater on Day 1 and 2 compared to pre-injury for sustained wrist extension at

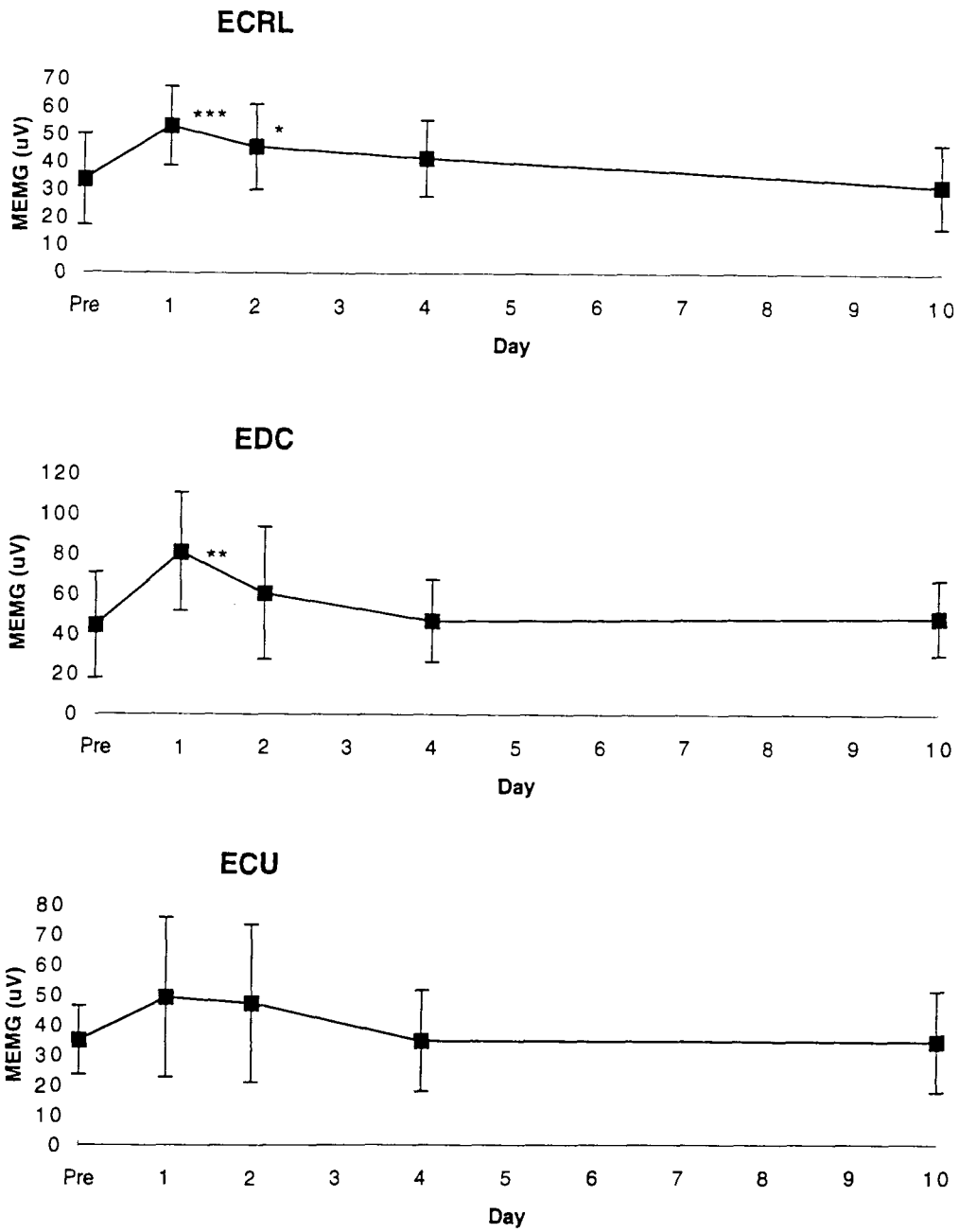

Figure 4.17: Initial MEMG for the wrist extensor muscles for a sustained contraction at 10% of pre-injury wrist extension MVC.

Values are means ± SD.
*** significantly different than pre-injury $p<0.005$.
** significantly different than pre-injury $p<0.01$.
* significantly different than pre-injury $p<0.025$.

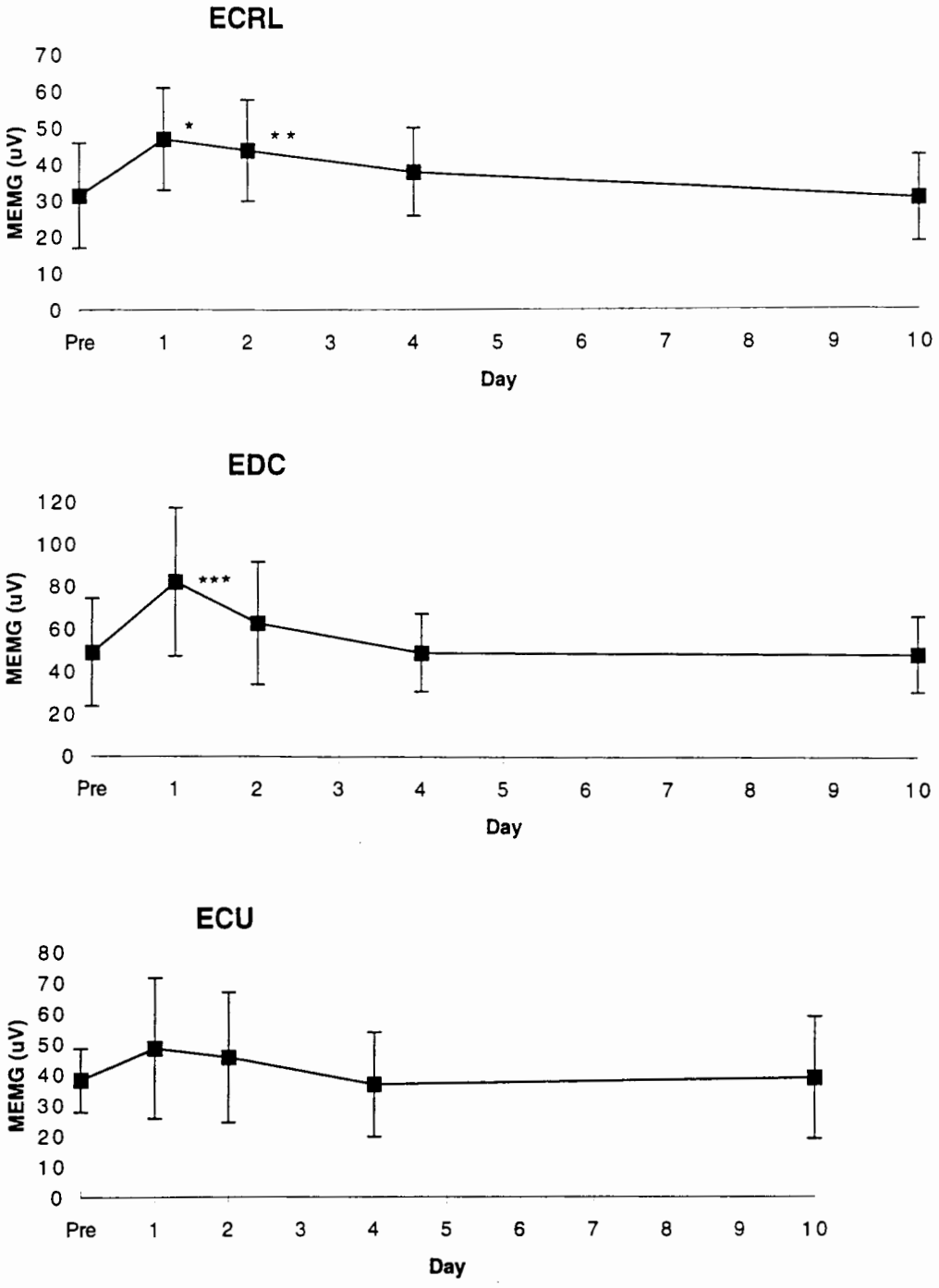

Figure 4.18: Final MEMG for the wrist extensor muscles for a sustained contraction at 10% of pre-injury wrist extension MVC.

Values are means ± SD.
*** significantly different than pre-injury $p<0.005$.
** significantly different than pre-injury $p<0.01$.
* significantly different than pre-injury $p<0.025$.

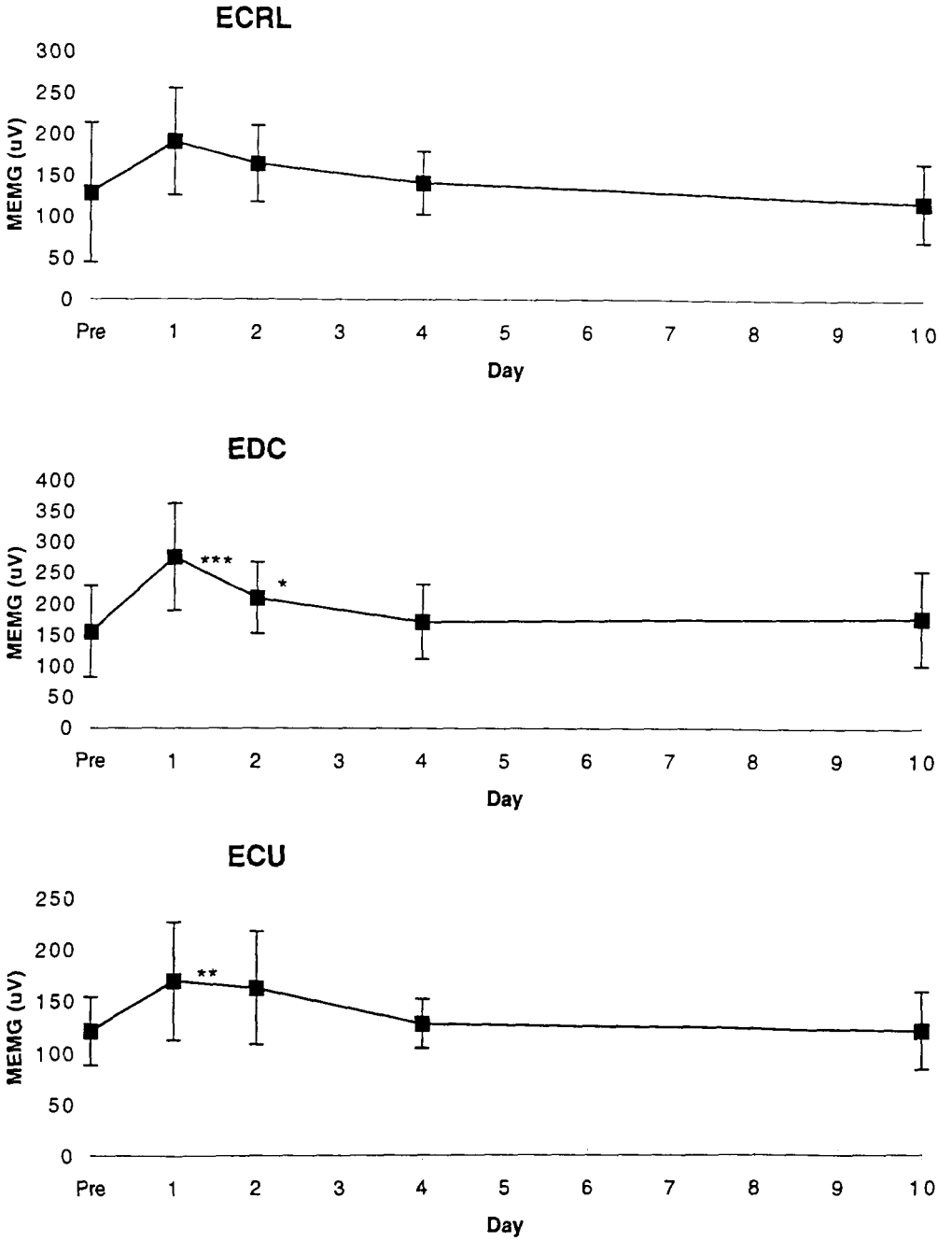

Figure 4.19 : Initial MEMG for the wrist extensor muscles for a sustained contraction at 50% of pre-injury wrist extension MVC.

Values are means ± SD.
*** significantly different than pre-injury $p<0.0001$.
** significantly different than pre-injury $p<0.005$.
* significantly different than pre-injury $p<0.025$.

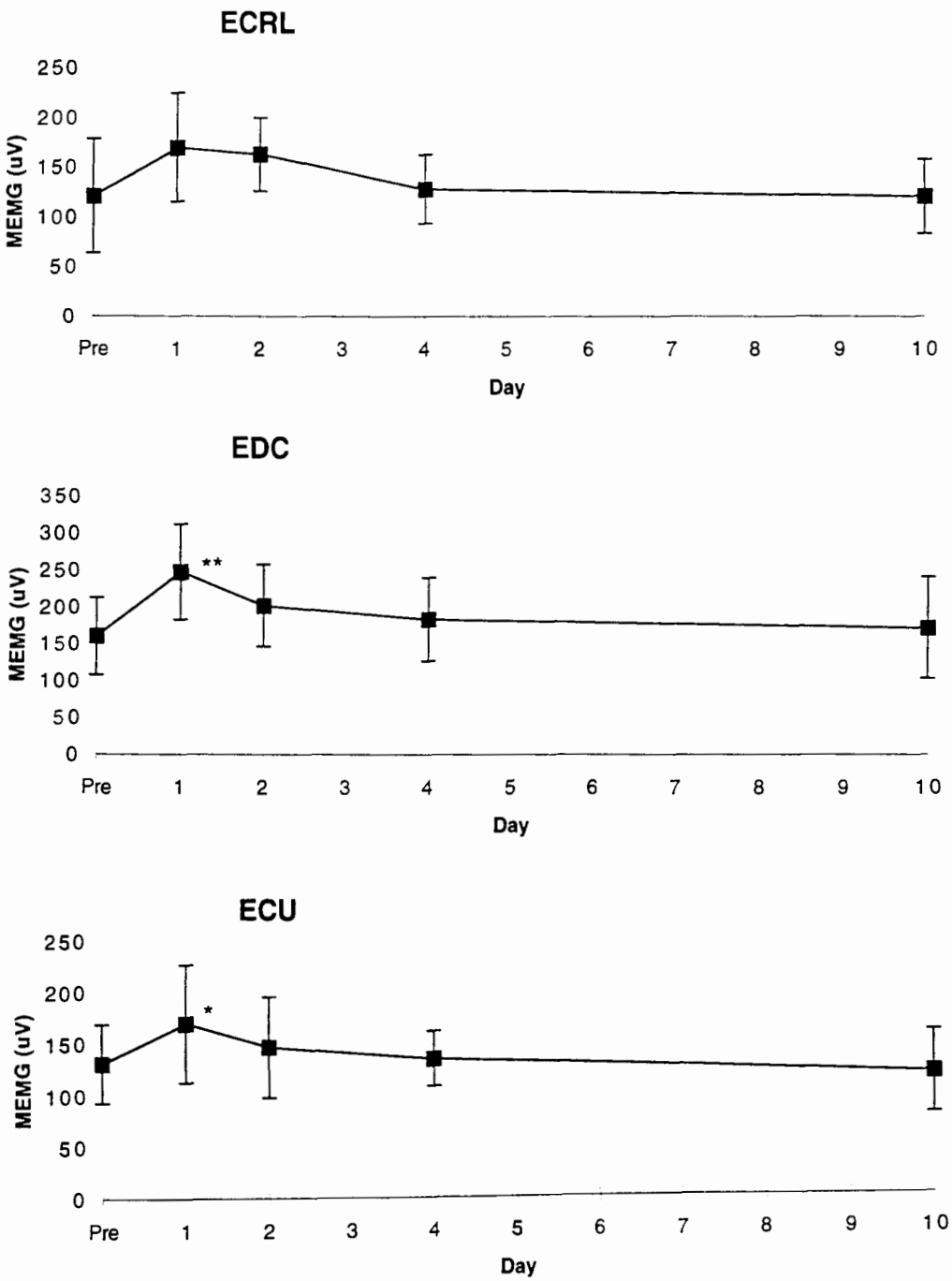

Figure 4.20: Final MEMG for the wrist extensor muscles for a sustained contraction at 50% of pre-injury wrist extension MVC.

Values are means ± SD.
** significantly different than pre-injury $p<0.0005$.
* significantly different than pre-injury $p<0.005$.

50% MVC (Table 4.8). Activity of the FDS and FCU muscles was also higher on Day 1 and Day 2 compared to pre-injury, but the differences were not significant.

Maximal wrist extension velocity did not change significantly throughout the test period, although it was lower on Day 1 and Day 2 compared to pre-injury (Figure 4.21). Table 4.9 lists the maximal velocity, latency from movement onset to maximal velocity, time required to stabilize within the target window, time of onset and time of peak EMG with respect to movement onset and IEMG of bursts in the wrist extensor and wrist flexor muscles. There were no significant changes in any of these parameters during the 10 days of testing. A trial for one subject pre-injury and Day 1 is shown in Figure 4.22.

The ability to stabilize an unstable load decreased from pre-injury to Day 1 and was still significantly lower than pre-injury on Day 2 (Figure 4.23). Although the maximum wrist stiffness that could be achieved by cocontraction dropped, the MEMG for the wrist extensors and flexors did not change following injury (Table 4.10).

Wrist Flexion Experiments

There were no significant effects of injury to the extensors muscles on any of the parameters measured during wrist flexion experiments. Wrist Flexion MVC did not change (Figure 4.24), nor did the associated MEMG of the wrist flexors or extensors (Table 4.11). Maximal wrist flexion velocity did not change significantly (Figure 4.25), nor did any of the EMG parameters associated with movement (Table 4.12). A trial for one subject for maximum velocity of wrist flexion pre-injury and Day 1 are shown in Figure 4.26.

TABLE 4.8: MEMG of the wrist flexor muscles for a sustained contraction at 50% of pre-injury wrist extension MVC.

	Pre	Day 1	Day 2	Day 4	Day 10
FCR					
50% Extension MVC					
MEMG Initial (μV)	1.60(.45)	3.39(1.4)*	2.99(1.4)*	2.13(1.1)	1.87(.38)
MEMG Final	1.84(.63)	3.60(1.1)*	3.24(1.5)*	2.41(.92)	1.95(.54)
FDS					
50% Extension MVC					
MEMG Initial	13.69(13.3)	18.01(15.2)	14.21(10.3)	11.10(8.1)	13.17(6.2)
MEMG Final	14.47(13.8)	17.32(13.8)	13.61(9.0)	11.21(7.9)	12.41(5.2)
FCU					
50% Extension MVC					
MEMG Initial	9.48(4.20	11.79(2.7)	12.74(5.4)	8.43(1.5)	8.57(1.9)
MEMG Final	12.97(6.5)	12.37(2.6)	13.53(5.6)	9.81(2.7)	9.61(3.2)

Means with Standard Deviations in (); Initial - first second, Final - fifth second

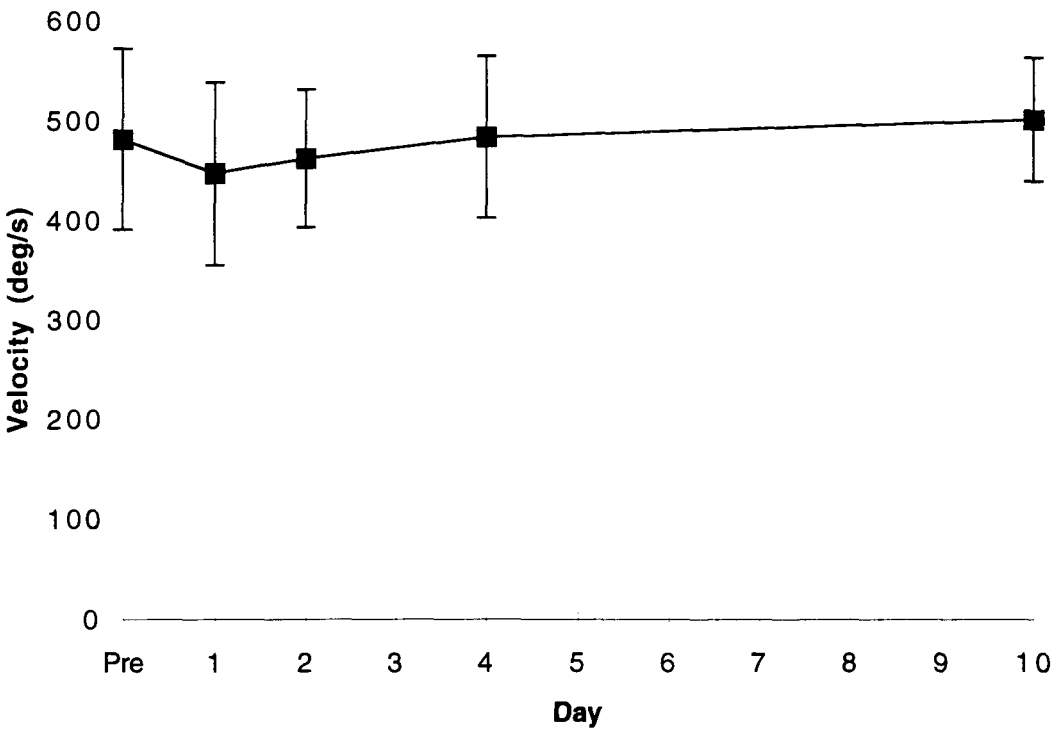

Figure 4.21: Maximum wrist extension velocity.

Values are means ± SD.

TABLE 4.9: Means and standard deviations of the parameters measured during maximum velocity of wrist extension.

	Pre	Day 1	Day 2	Day 4	Day 10
Velocity (deg/s)	481.6(91.1)	447.1(92.1)	462.8(69.1)	484.4(81.6)	502.6(62.3)
Time (ms)	99.8(15.2)	101.5(13.8)	97.7(8.7)	103.2(28.1)	90.0(8.2)
Time of Onset (ms)					
ECRL	-23.95(17.4)	-28.84(9.4)	-26.32(14.3)	-21.19(37.0)	-31.64(10.4)
EDC	-26.11(16.7)	-32.78(12.8)	-32.74(16.4)	-21.91(34.1)	-30.52(8.5)
ECU	-24.37(14.5)	-31.41(10.9)	-31.50(15.2)	-21.82(27.1)	-31.15(11.8)
FCR	76.72(21.2)	55.20(21.7)	67.09(24.7)	66.33(33.8)	51.77(19.4)
FDS	72.57(37.3)	69.00(23.1)	64.55(21.9)	59.05(38.0)	56.73(21.6)
FCU	76.49(22.0)	73.07(30.4)	73.33(15.5)	69.80(22.0)	65.20(20.8)
Time to Peak (ms)					
ECRL	38.53(34.1)	36.78(19.0)	34.88(24.3)	44.21(35.8)	18.93(15.0)
EDC	43.00(32.1)	50.16(29.8)	49.17(36.5)	52.75(37.9)	33.52(24.9)
ECU	32.44(20.2)	37.00(18.2)	36.20(25.1)	38.49(30.55)	27.29(17.9)
FCR	111.4(20.6)	98.25(19.8)	99.37(27.8)	109.4(31.8)	84.95(13.8)
FDS	111.8(33.9)	100.8(25.4)	106.6(15.2)	93.38(40.8)	93.71(18.3)
FCU	106.7(17.2)	103.1(43.1)	103.8(10.6)	110.9(29.3)	94.72(14.3)
IEMG (μV^*s)					
ECRL	13.89(10.5)	13.71(8.2)	10.98(3.0)	12.78(6.7)	10.99(7.1)
EDC	15.32(7.8)	19.98(8.2)	17.10(6.9)	14.50(6.5)	14.41(7.3)
ECU	14.86(6.6)	16.61(7.7)	16.08(5.9)	14.37(6.2)	14.08(7.2)
FCR	1.11(1.1)	.824(.89)	.891(.58)	.883(.68)	.877(.57)
FDS	2.45(4.1)	3.04(4.8)	3.41(4.9)	3.03(4.49)	3.18(2.5)
FCU	3.48(2.2)	2.58(1.4)	3.02(1.9)	3.21(2.2)	3.30(2.7)

Means with Standard Deviations in ()

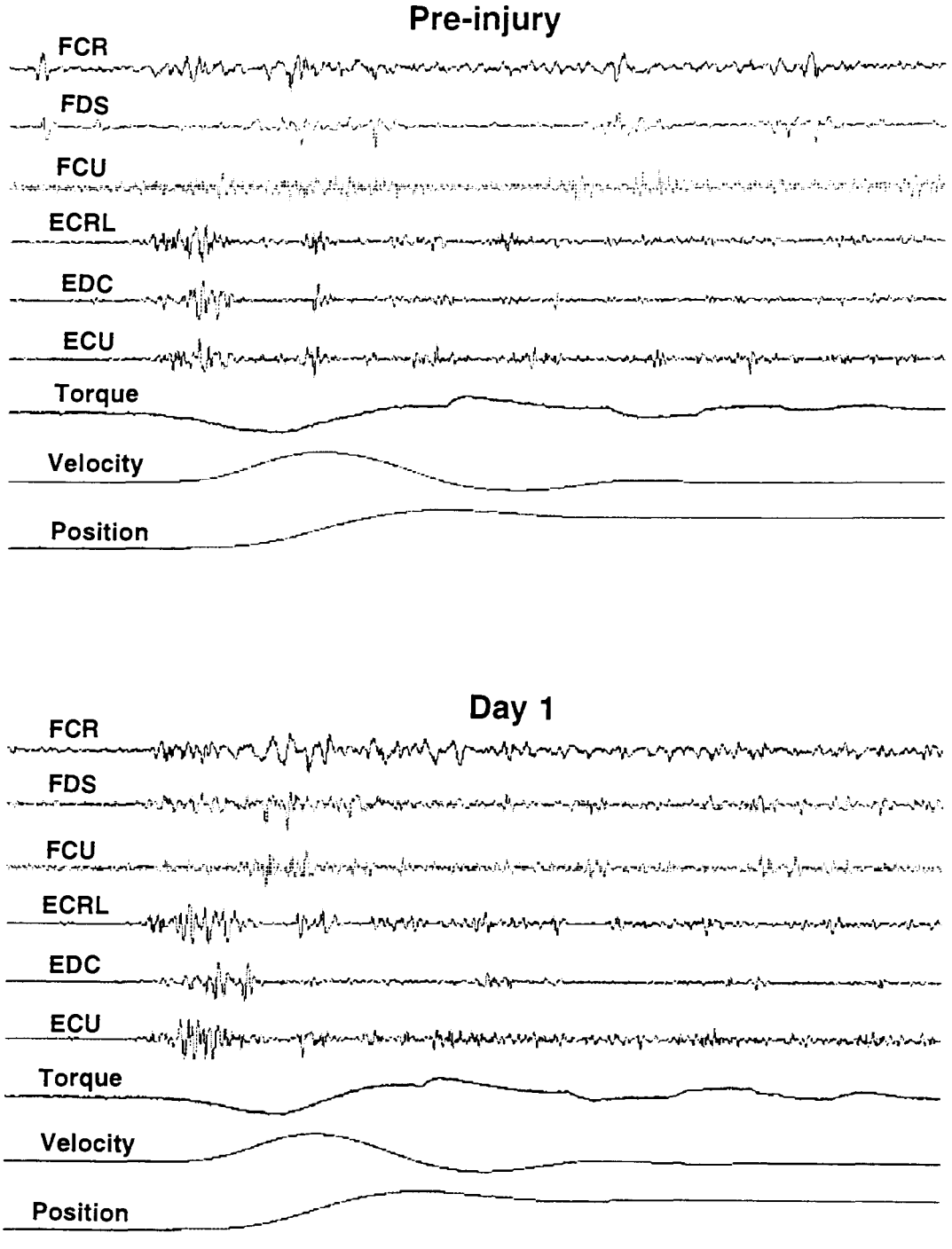

Figure 4.22: Trials for one subject for maximum velocity of wrist extension pre-injury and Day 1.

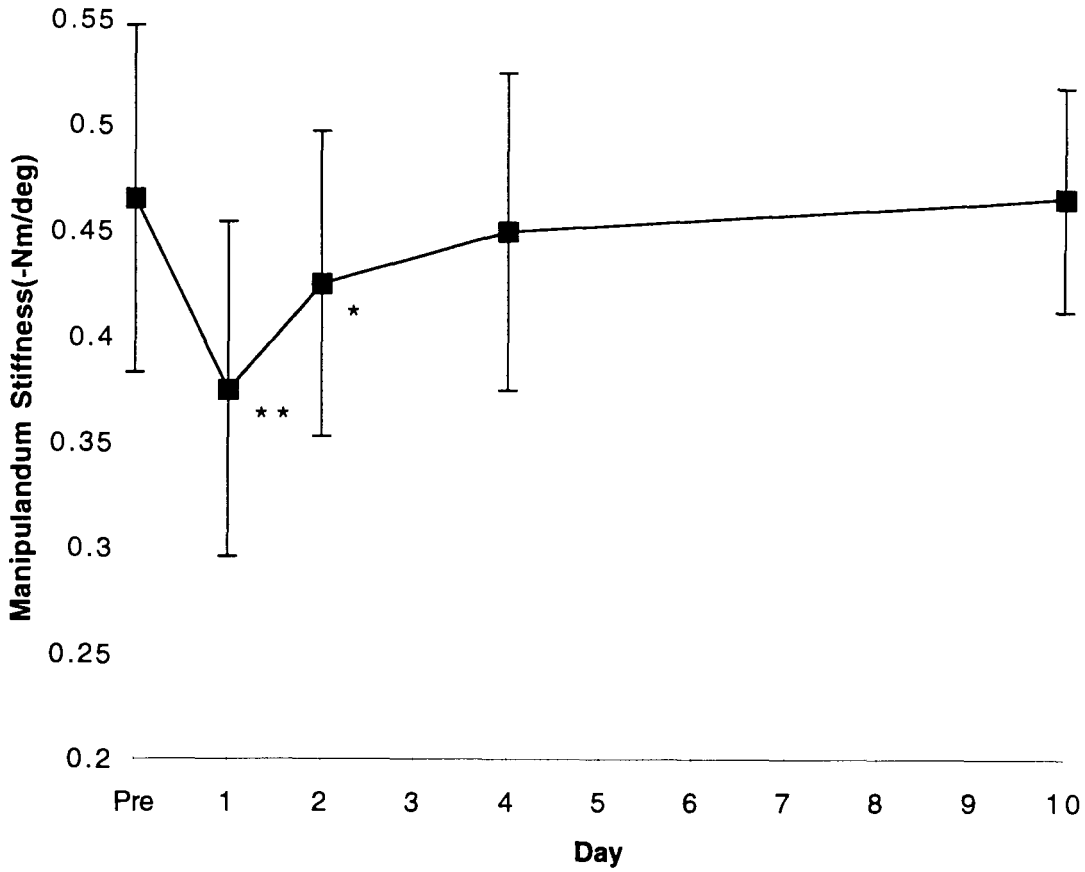

Figure 4.23: Negative load stiffness from the experiment to determine the ability to stabilize and unstable load.

Values are means ± SD.
** significantly different than pre-injury $p<0.001$.
* significantly different than pre-injury $p<0.025$.

TABLE 4.10: Negative load stiffness and MEMG for the experiment to determine the ability to stabilize an unstable load.

	Pre	Day 1	Day 2	Day 4	Day 10
Load Stiffness (-Nm/deg)	.465(.082)	.375(.079)*	.425(.072)*	.450(.075)	.465(.053)
MEMG (μV)					
FCR	8.57(4.02)	5.89(4.12)	6.65(5.26)	9.57(6.99)	9.71(7.72)
FDS	44.89(55.2)	38.34(33.4)	42.60(35.3)	54.15(43.0)	74.21(32.3)
FCU	60.04(29.4)	44.23(23.1)	61.08(37.5)	62.23(30.9)	57.7(37.9)
ECRL	84.83(37.2)	103.95(50.8)	76.49(28.2)	86.46(31.9)	73.86(24.2)
EDC	130.4(52.3)	153.1(48.0)	138.2(55.8)	130.5(68.2)	111.68(47.5)
ECU	93.79(23.4)	99.72(29.1)	101.6(39.4)	100.6(40.5)	77.16(35.8)

Means with Standard Deviations in ()

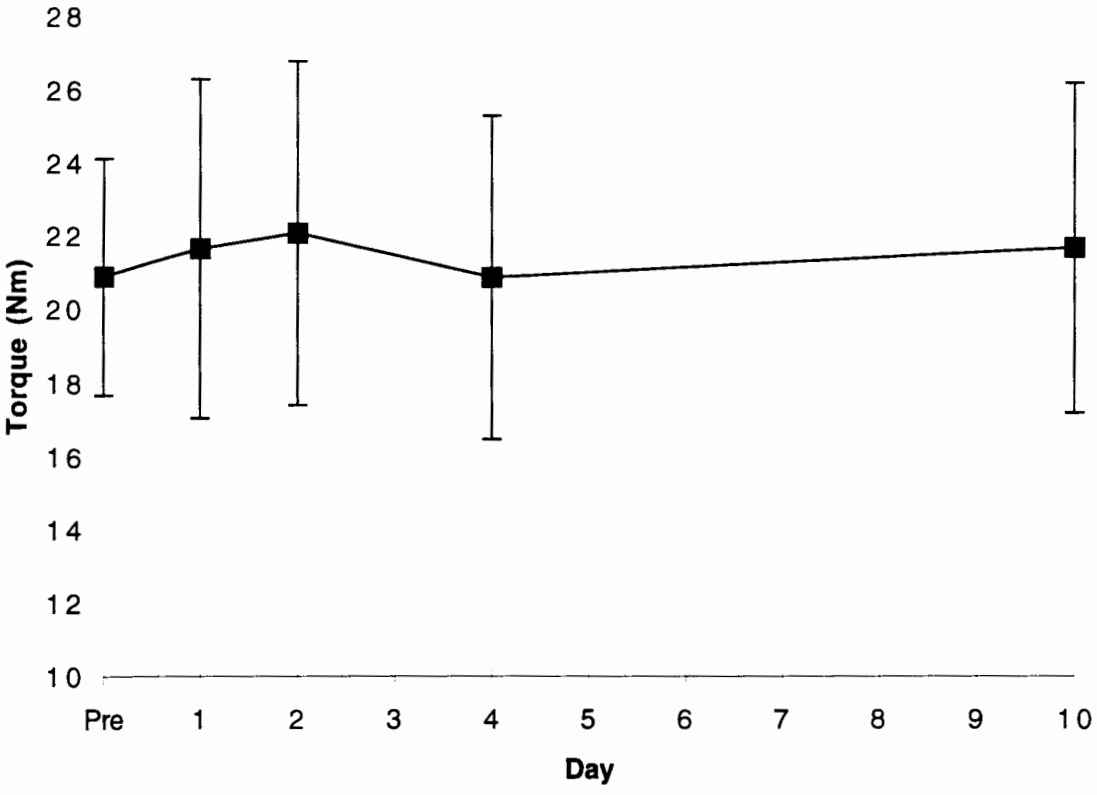

Figure 4.24: Wrist flexion MVC.

Values are means ± SD.

TABLE 4.11: Torque and MEMG for wrist flexion MVC.

	Pre	Day 1	Day 2	Day 4	Day 10
Torque (Nm)	20.9(3.2)	21.7(4.6)	22.1(4.7)	20.98(4.4)	21.7(4.5)
MEMG (μV)					
FCR	95.99(48.7)	79.90(32.7)	89.18(43.7)	75.47(43.1)	110.9(69.7)
FDS	254.2(184.7)	282.1(192.4)	261.6(179.4)	267.6(188.5)	290.1(187.1)
FCU	386.5(105.5)	347.1(116.1)	347.4(77.8)	352.9(133.9)	354.1(87.7)
ECRL	42.34(25.6)	35.91(17.7)	36.91(18.4)	40.66(28.8)	43.23(18.4)
EDC	67.60(36.3)	80.49(45.4)	60.06(31.2)	62.87(48.1)	61.87(31.1)
ECU	64.22(20.8)	71.34(33.7)	52.97(29.6)	49.36(31.6)	64.74(31.8)

Means with Standard Deviations in ()

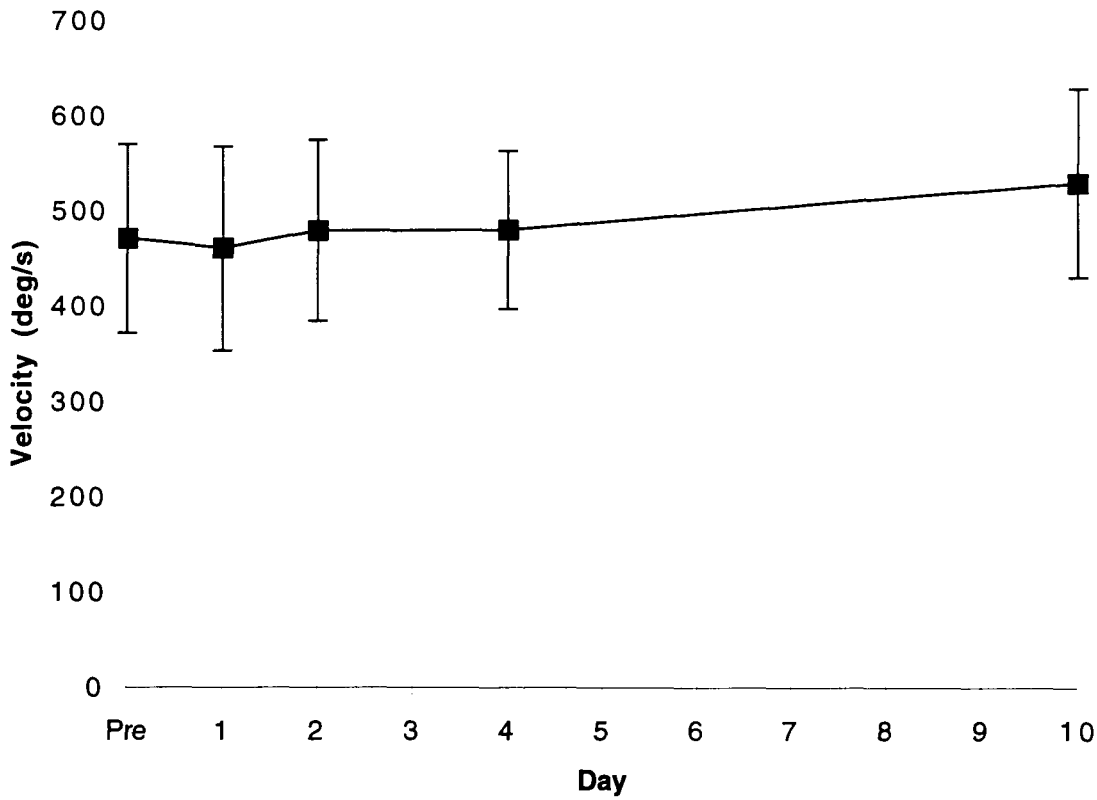

Figure 4.25: Maximum wrist flexion velocity.

Values are means ± SD.

TABLE 4.12: Means and standard deviations of the parameters measured during maximum velocity of wrist flexion.

	Pre	Day 1	Day 2	Day 4	Day 10
Velocity (deg/s)	470.8(99.0)	460.8(107.2)	480.1(94.9)	481.3(83.4)	530.9(99.3)
Time (ms)	100.2(18.6)	93.07(13.7)	61.63(13.8)	94.19(14.5)	97.25(14.8)
Time of Onset (ms)					
FCR	-19.57(16.7)	-23.81(10.3)	-24.47(4.9)	-24.07(10.2)	-17.00(17.1)
FDS	-16.57(14.1)	-19.99(10.2)	-24.97(6.7)	-18.91(9.6)	-17.46(19.7)
FCU	-10.33(15.4)	-16.57(6.2)	-20.80(9.7)	-20.34(9.2)	-15.26(15.1)
ECRL	66.84(23.6)	59.31(28.1)	58.98(27.9)	59.37(18.7)	60.42(24.3)
EDC	54.46(31.9)	47.91(32.3)	40.80(28.6)	36.79(18.9)	51.58(35.2)
ECU	37.10(30.3)	31.96(23.7)	34.37(23.6)	32.48(18.9)	32.74(27.6)
IEMG (μV*s)					
FCR	2.37(1.3)	1.89(.89)	1.76(1.3)	1.76(1.9)	2.19(1.9)
FDS	8.27(9.2)	8.84(7.6)	9.05(8.4)	7.88(6.9)	11.56(5.3)
FCU	13.08(8.5)	11.05(7.6)	11.87(7.9)	11.07(6.7)	11.38(5.6)
ECRL	6.47(4.7)	6.26(5.3)	4.73(3.5)	6.11(3.9)	6.18(5.6)
EDC	5.72(3.8)	7.34(5.3)	7.00(4.8)	6.29(4.8)	6.17(3.6)
ECU	4.77(2.0)	5.79(3.2)	7.05(4.6)	6.43(3.1)	7.61(5.1)

Means with Standard Deviations in ()

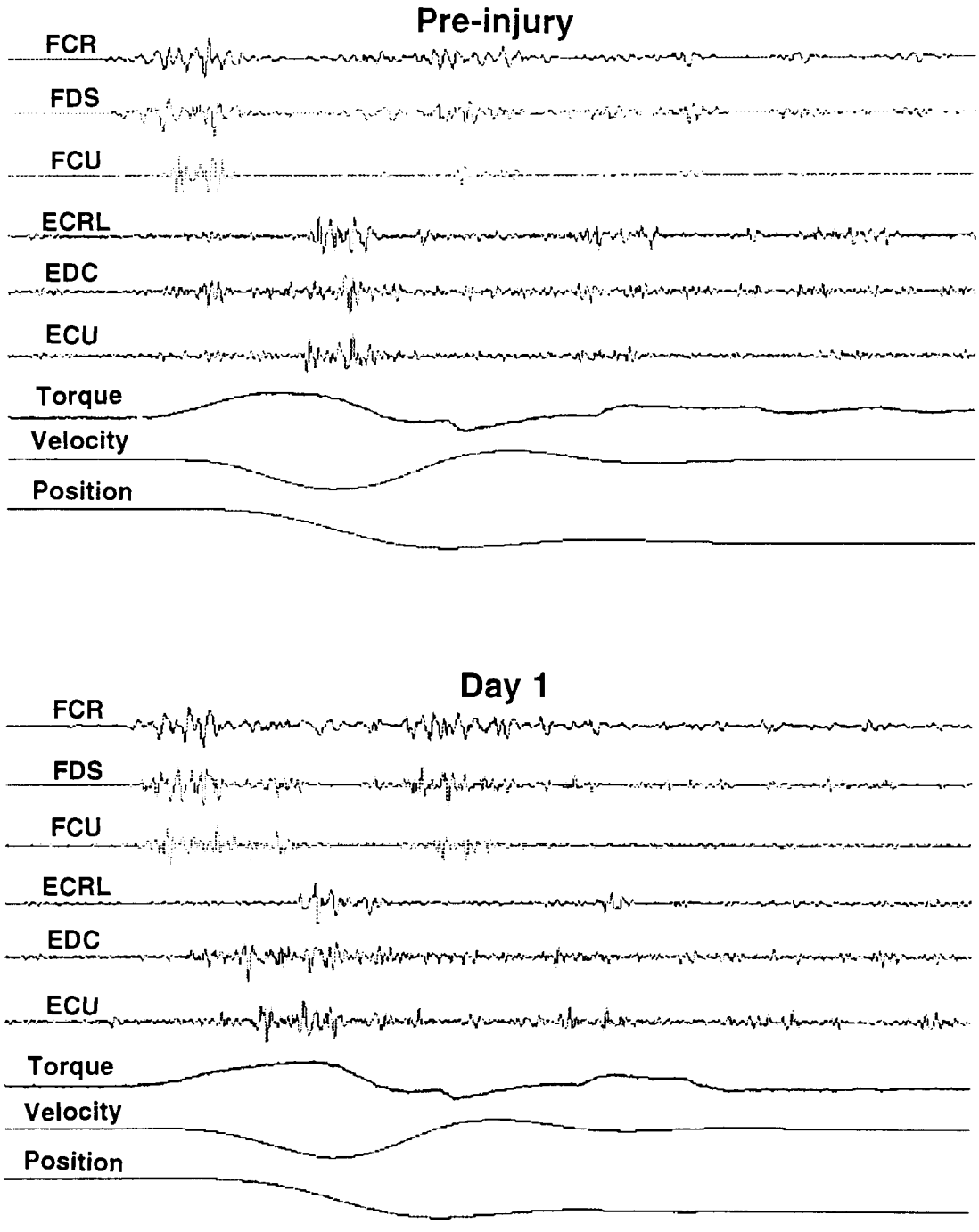

Figure 4.26: Trials for one subject for maximum velocity of wrist flexion pre-injury and Day 1.

The ability to track flexor torque, increasing at a constant rate, did not change significantly (Figure 4.27), nor did the MEMG of the wrist extensors or flexors (Table 4.13). There was no significant change in the torque variance for sustained wrist flexion torque at any of the tested levels (Table 4.14), nor was there any significant change in the MEMG of the wrist flexors or extensors even at 50% MVC (Table 4.15).

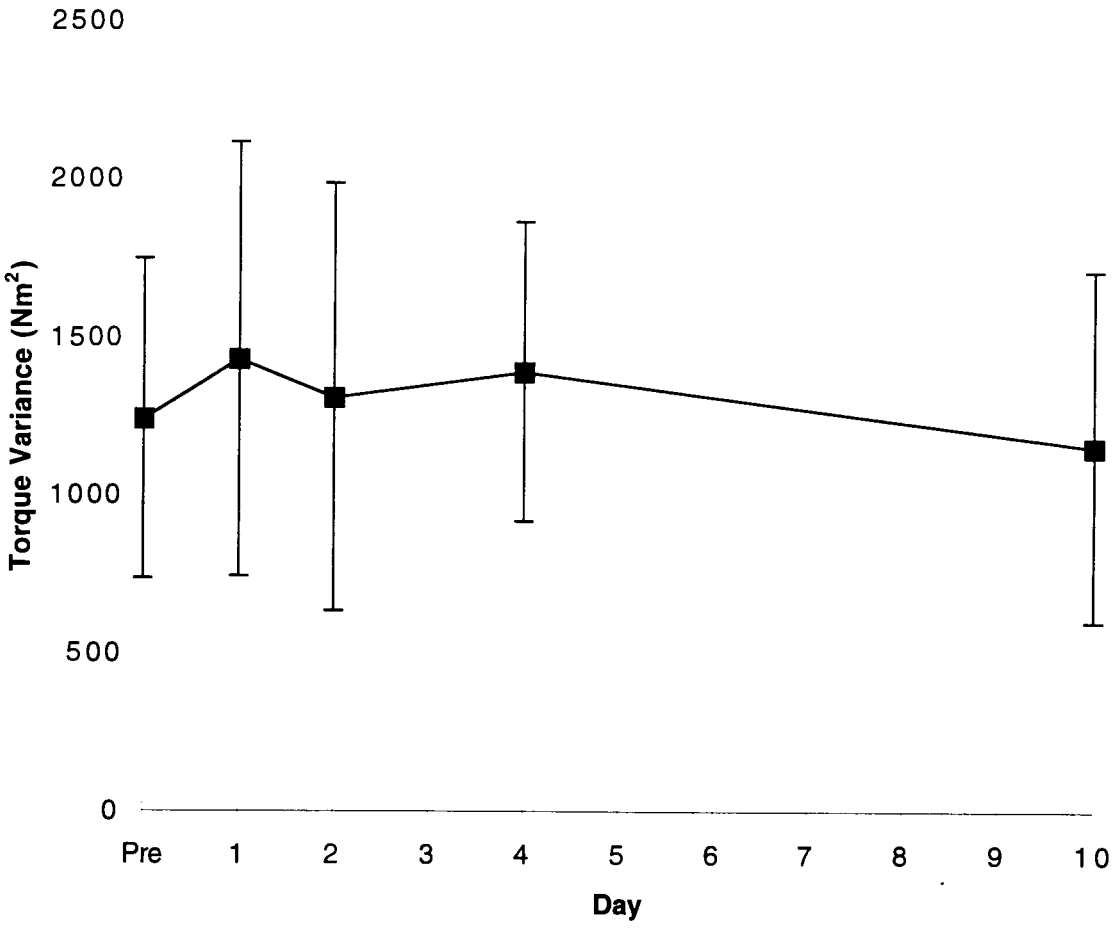

Figure 4.27: Torque variance while tracking flexor torque changing at constant rate.

Values are means ± SD.

TABLE 4.13: Torque variance and MEMG while tracking flexor torque changing at constant rate.

	Pre	Day 1	Day 2	Day 4	Day 10
Variance (Nm^2)	1240.6(506.6)	1429.8(686.5)	1309.9(676.6)	1392.5(474.0)	1154.0(555.5)
FCR					
MEMG Initial (μV)	4.46(3.29)	5.07(3.97)	5.59(4.23)	4.46(3.63)	4.63(3.28)
MEMG Final	24.50(17.7)	26.24(17.8)	32.02(21.6)	22.05(16.4)	23.68(17.5)
FDS					
MEMG Initial	32.92(23.4)	38.14(30.3)	40.21(33.9)	36.21(31.0)	43.43(35.5)
MEMG Final	133.1(95.9)	162.4(137.5)	158.6(125.8)	155.8(135.9)	145.5(132.2)
FCU					
MEMG Initial	50.21(16.7)	41.58(26.7)	32.08(13.4)	33.88(14.9)	34.08(14.5)
MEMG Final	176.8(52.7)	163.5(68.7)	151.3(41.7)	156.7(34.6)	148.4(39.6)
ECRL					
MEMG Initial	2.57(.806)	2.58(.530)	2.57(.751)	2.50(.478)	2.52(.582)
MEMG Final	8.96(5.05)	9.09(4.20)	9.60(6.43)	8.21(3.70)	9.74(6.79)
EDC					
MEMG Initial	5.47(2.52)	5.78(2.32)	4.16(1.13)	4.56(1.14)	4.94(1.83)
MEMG Final	18.08(7.62)	17.03(6.71)	13.57(5.47)	14.72(5.93)	15.89(8.80)
ECU					
MEMG Initial	4.29(1.35)	4.69(1.10)	3.92(.821)	4.69(1.49)	4.11(.697)
MEMG Final	15.61(7.76)	17.58(7.32)	14.83(7.07)	13.67(5.62)	16.27(6.91)

Means with Standard Deviations in ()

TABLE 4.14: The torque variances for sustained contractions at 10%, 30% and 50% of pre-injury wrist flexion MVC.

	Pre	Day 1	Day 2	Day 4	Day 10
Torque Variance (Nm^2)					
10% Flexion MVC	636.6(374.3)	656.5(435.8)	508.7(263.5)	363.1(354.8)	624.4(542.7)
30% Flexion MVC	5848.9(4138)	8552.2(7062)	5960.0(4718)	6459.5(3786)	7708.5(9998)
50% Flexion MVC	7516.8(4603)	8265.7(5175)	9213.9(6032)	9880.2(7368)	11134(11416)

Means with Standard Deviations in ()

TABLE 4.15: The MEMG of the wrist flexor muscles for a sustained contraction at 50% of pre-injury wrist flexion MVC.

	Pre	Day 1	Day 2	Day 4	Day 10
50% Flexion MVC					
MEMG Final (μV)					
FCR	32.83(19.2)	35.16(24.1)	37.27(20.3)	27.47(19.1)	39.10(28.6)
FDS	127.4(109.6)	134.7(103.9)	132.7(107.0)	134.6(118.6)	125.2(100.0)
FCU	150.0(40.4)	142.5(47.8)	150.9(40.0)	144.2(44.1)	157.3(52.6)
ECRL	10.31(4.40)	10.22(2.97)	10.57(4.61)	10.82(4.55)	9.93(3.11)
EDC	16.22(6.16)	16.17(2.88)	16.48(3.72)	16.24(6.26)	15.36(3.42)
ECU	14.51(4.57)	15.25(7.23)	15.91(5.91)	15.83(5.47)	16.07(4.24)

Discussion

The purpose of this study was to compare muscle mechanics and neural control of wrist muscles previous to, and for several days during recovery from a bout of maximal eccentric exercise involving wrist extensors. Evidence of injury to these muscles included soreness, a reduced range of passive wrist movement, and reduced maximal isometric wrist extension torque. These symptoms are similar to those which have been reported in other studies of exercise-induced muscle injury where damage to muscle fibers has been confirmed by histological and ultrastructural analysis (Newham et al. 1987; Stauber et al. 1990). The time course of recovery for parameters evaluated in the present study was also similar to that reported after injury to other muscle groups (Jones et al. 1986; Stauber et al. 1990; Howell et al. 1993; Crenshaw et al. 1994; MacIntyre 1994). Therefore, there is compelling evidence of injury to muscle fibers, resulting from the exercise protocol used in the present study.

Clarkson et al. (1992) reported findings from 109 subjects in a review of the changes that occur in the elbow flexor muscles following an exercise regime of two sets of 35 maximal eccentric contractions. Their subjects showed a dramatic loss of isometric strength of up to 50% one day after the exercise bout, which was gradually restored such that by 10 days after exercise only a small deficit remained. Although the loss of isometric strength in the wrist extensors in the present study was only 24.5% one day after the exercise bout, it recovered in a similar manner such that by day 10 no deficit remained. Similarly, the soreness of wrist extensors reported by subjects in the present study was comparable to

the soreness ratings reported by Clarkson et al. (1992). There was an increase in soreness the first day after the exercise, which reached a maximum on Day 2 and then gradually decreased until Day 10.

The reduced passive range of wrist motion (ROM) is analogous to the change in relaxed joint angle reported by Clarkson et al. (1992) and others (Howell et al. 1985; Jones et al. 1987a; Stauber et al. 1990). These measures illustrate the effects of injury at the extremes of the ROM. Overall, these findings are consistent with the expected effects of an exercise-induced muscle injury to the wrist extensors, resulting from damage to muscle tissue.

Mechanical Adaptation

The reduced ROM of the wrist is one indication of injury to the wrist extensor muscles. ROM was determined from the angle at which 0.5 Nm of resistance was first encountered in both flexion and extension. The reduced ROM could indicate increased resistance to stretch of connective tissue as has been suggested in other studies which have reported no increase in muscle electrical activity during passive joint movements (Bobbert et al. 1986; Jones et al 1987a). Stauber et al. (1990) and Howell et al. (1985) have suggested that there is a decrease in resting length of muscle caused by swollen tissue pushing against the fascia and causing muscle to shorten passively. Bobbert et al. (1986) have reported swelling and increased circumference of the lower leg after eccentric exercise of the gastrocnemius muscle with a time course similar to the decreased ROM reported in the present study. Although swelling was not measured, there is reason to believe from these other studies that it occurred

and contributed to reduced ROM in either direction, due to increased internal pressure.

In support of hypothesis 1, quantification of passive joint stiffness by means of resistance to a quick stretch at the neutral position of the wrist showed no change after muscle injury. This finding would suggest that there was no change in the mechanical properties of the non-contractile tissue of the muscle in the mid-range position. Although increased stiffness and difficulty moving a joint have been reported following exercise-induced muscle injury (Howell et al. 1985; Jones et al. 1987a; Stauber et al. 1990), the findings of the present study indicate that there is increased resistance to movement only at the ends of joint ROM. Since mechanical properties of non-contractile tissue appear to be unaffected by eccentric exercise, this resistance is more likely due to swelling within the tissue.

No previous studies have quantified active wrist joint stiffness following muscle injury. Wrist joint stiffness at 10% of pre-injury MVC in the present study did not change after the muscle injury. However, the joint stiffness at 50% of pre-injury MVC was lower one day after the exercise bout. Although this decline was not significantly different from the pre-injury joint stiffness ($p=0.05$), given the level of significance set in this study ($p<0.025$), the time course of changes in joint stiffness was very similar to the time course of changes in wrist extension MVC. Following injury there was an increase in the extensor muscle background MEMG at 50% of MVC which was most evident on Day 1. This elevated extensor muscle activity was accompanied by greater activity in the wrist flexor muscles on Day 1. Such an increase in cocontraction would normally result in greater joint

stiffness. However, this was not the case. The wrist extensor and flexor muscles are comprised of approximately 50% slow oxidative and 50% fast glycolitic muscle fiber types (Johnson et al. 1973). It is expected that by 50% of MVC the large fast-twitch motor units would have begun to be recruited. The decline in wrist joint stiffness despite the increased muscle activity supports hypothesis 2 and suggests that there were changes in the mechanical properties of large motor units, which are required for large torques which also affected the muscle stiffness. Had there been less cocontraction of the wrist flexor muscles, the joint stiffness would probably have decreased following injury as was the case with the FDI muscle.

Neural Adaptation

The response of the wrist extensors to a rapid displacement while active at 50% MVC revealed changes in neural control. There was a smaller short latency reflex response for all three wrist extensors on Day 1 (although significant for the EDC muscle only) compared to pre-injury. However, the intermediate and long-latency reflex responses were unchanged. This finding suggests there was inhibition of the monosynaptic response to rapid displacement when the muscles were highly activated. A similar decline in the short-latency reflex response has been reported after fatiguing contractions (Balestra et al. 1992; Duchateau and Hainaut 1993). It was suggested that the changes in the short latency reflexes were related to peripheral mechanisms such as the inhibitory effects from group III and IV afferents and/or from pre-synaptic inhibition. Also consistent with the present study, was the finding that the long-latency reflex response did not

decrease during voluntary fatiguing contractions suggesting that only the peripheral reflex loop was involved. Bulbulian and Bowles (1992) reported a reduced H-wave, expressed as a ratio of maximal M-wave, immediately after eccentric exercise of the lower extremity, however, it returned to baseline by 24 hours. They suggested the decline was a result of presynaptic modulation of afferent sensitivity to proprioceptive feedback of group I and group II afferents. However, no direct evidence of this was provided.

The EMG amplitude increased for submaximal contraction. This finding supports hypothesis 4 and is in agreement with the findings of Kroon and Naeije (1991) and others (Komi and Viitasalo 1977; Berry et al. 1990) and may reflect greater activation of the smaller motor units to compensate for the damage to larger muscle fibers. Based upon the fiber type composition of the wrist muscles, it is probable that at submaximal levels of contraction large fast-twitch motor units would begin to be recruited. The increased EMG may also be related to the increased cocontraction of wrist flexor muscles. A greater amount of wrist extensor activity would have been required achieve a desired submaximal extensor torque.

Some researchers have suggested that that shifts in the MF of the power spectrum to the low end of the power spectrum reflect a decline in the contribution of large fibers and reliance on the slow fibers (Lindström et al. 1970; Bigland-Ritchie et al. 1981, 1986b; Hagberg and Ericson 1982; Duchateau et al. 1991; De Luca 1985). Based upon the analysis of the wrist extensors MF there was no evidence to support greater contribution of small motor units to

compensate for injury to large muscle fibers. In the present study, the MF did not change during the performance of any task, indicating that motor units of similar activation characteristics were utilized before and following muscle injury. Berry et al. (1990) reported a similar finding in their study of the quadriceps muscle. However, the combined contribution of several muscles to knee extension in their study, and to wrist extension in the present study, may have masked any shifts in motor unit recruitment.

Function and Control of Wrist Extensor Muscles

One aspect of the present study was the investigation of the effects of exercise-induced muscle injury of the wrist extensors on motor control. It was hypothesized that performance of tasks requiring recruitment of large muscle fibers would be impaired. To test this hypothesis the ability of the subjects to track a constantly increasing torque was investigated. Performance of the torque tracking task was most impaired on Day 1 after the muscle injury. This was evaluated by computing the variance of the torque about a line representing a constant rate of increase of the target torque. The greatest variability about the target torque on Day 1 occurred as the target approached the highest torque levels. There was also an increase in the MEMG of the wrist extensors during performance of this test compared to pre-injury. The decline in ability to smoothly increase torque followed the same time course as the decline and recovery of wrist extension MVC. The similarities in time course together with the fact that subjects appeared to have greater impairment at higher torques supports

hypothesis 3 and suggests the lack of control is due to an inability to smoothly recruit larger motor units due to injury to them.

During sustained torque, variation increased at all levels of torque on Day 1 compared to pre-injury. There appeared to be a greater impairment of the ability to maintain a steady torque at higher torques, during which motor units with larger fibers would have been recruited. The torque traces on Day 1 appeared to fluctuate about the target torque more than pre-injury, with the greatest variation at 50% MVC. There was also an increase in the MEMG of the wrist extensors during performance of most tests compared to pre-injury. The decline in the ability to control torque in this study followed a similar pattern to the recovery of pre-injury MVC over the ten days of testing. The decline in ability to maintain constant torque is similar to impairment in control that has been reported for elderly subjects with fewer large motor units (Galganski et al. 1993). These findings support hypothesis 3.

It was hypothesized that damage to the wrist extensors following eccentric exercise, if specific to the large motor units, would affect the first agonist burst during rapid movement, such that there would be a decline in maximum velocity. Despite evidence of injury to the wrist extensors there were no differences in the timing, duration or magnitude of the agonist bursts, or in the maximal velocity. Furthermore, the ability to stabilize or control the movement at the end target position was also unchanged. These findings do not support hypothesis 3 and suggest that the muscle injury had no effect on the strategy or performance of a target orientated ballistic movement. Subjects exhibited a typical three burst

pattern of EMG activity and performed the task in similar ways previous to and following the muscle injury. It is important to note that the nature of the task in this study may have influenced the performance outcomes. Subjects were required to stabilize within a 3^{o} target window at the end of a 30^{o} movement. A trial was only considered successful if the subject stopped within the target window without further movement within 375 ms of movement onset. On each day of testing subjects attempted a number of movements of which 5 were saved. The number of movements that were attempted before 5 acceptable movements had been performed appeared to be greater after the injury. However, no specific count was kept. It is possible that the task could only have been successfully completed in a certain manner and that, therefore, changes in performance due to the muscle injury were not observed.

Subjects' ability to stabilize a mechanically unstable manipulandum was impaired on Day 1 and Day 2 compared to pre-injury. The time course of this deficit was similar to the decline in wrist extension MVC and wrist stiffness at 50% MVC. However, the MEMG for the wrist extensors and flexors was not reduced. The ability to stiffen the wrist while producing zero net torque depends upon the balanced activity of the wrist flexor and extensor muscles. Milner et al (1995) and others (Tyler and Hutton 1986; Jongen et al. 1989; Kearney and Hunter 1990) have reported that muscle activation is constrained to a submaximal level during cocontraction. The MEMG for the wrist extensors in the present study was less than the MEMG recorded during 50% wrist extension MVC. Therefore, the difficulty performing this task does not seem to be due to an

inability to activate and balance the activity between the extensor and flexors muscles, but rather due to a deficit in joint stiffness.

Muscle Synergy

In each of the tests performed in the present study the three wrist extensors were analyzed separately. During maximal voluntary contraction there was no change found in the MEMG for the three wrist extensors despite a decline in the torque produced on Day 1 and Day 2. This would suggest that all three wrist extensors were fully activated during each of the days of testing. From these findings it is not possible to conclude, however, that all three of the muscles were equally injured. As this was a maximal effort it is possible that although one muscle was injured more, it was still activated as the others, but was unable to produce as much force as pre-injury. The MEMG recorded on Day 1 after the muscle injury, for tests requiring submaximal levels of activation, also illustrates that changes in the muscles have occurred. In tests requiring levels of activity ranging from 10% to 50% of pre-injury wrist extension MVC there was an increase in the amount of MEMG suggesting that greater activation was required to compensate for the injured fibers. The MEMG of all three wrist extensors increased and followed a similar pattern over the 10 days of testing. The time course was similar for changes in initial and final MEMG of the wrist extensors during sustained contractions of 10% and 50% pre-injury MVC. The MEMG was generally significantly greater on Day 1 and then declined until pre-injury levels were reached at Day 10. Although the MEMG was not always significantly different from pre-injury, the pattern of change, together with the fact that there

was no extensor muscle which had consistently lower activity, shows that the three muscles responded similarly. The similar pattern of change for the three wrist extensors was not felt to be due to cross-talk, as the electrodes used were relatively immune to cross-talk when recording from trim subjects, as was the case in the present study (de la Berrera and Milner 1994). These findings support hypothesis 5 and are similar to the findings of Newham et al. (1983) who reported no difference in the pattern of recruitment of the rectus femoris, medial and lateral vasti muscles following eccentric exercise. In the present study, there was no suggestion of inhibition or compensation among the injured muscles. It was possible that one muscle was affected more than the others but continued to be recruited as the other muscles, although it contributed less torque after injury. Nonetheless, the effect of the exercise bout on the wrist extensors was to increase the activity of all of the muscles during tasks which required submaximal torque levels. During all tasks which required extensor torque it was evident that all three wrist extensors were equally active. There were similar changes in each of the muscles after the bout of eccentric exercise, indicating that the signal from the CNS may have been a common one to the three muscles.

Function and Control of the Antagonist Wrist Flexor Muscles

Several tests were performed for wrist flexion. These were done in order to investigate the influence of injury to the wrist extensors on the control of wrist flexors. In no wrist flexion tests was there a significant change in performance or muscle activation of any wrist flexor muscle. Thus, there was no apparent effect

on the wrist flexors resulting from injury to the wrist extensors, when the wrist flexors acted as the agonists.

There were no changes in the three burst pattern of EMG activity or in the ability of the subjects to rapidly move and control the wrist during a ballistic flexion task. These findings differ from those of Wierzbicka and Wiegner (1992) who found a decline in peak velocity in spinal cord injured patients with weak antagonist muscles. The difference may be because of the comparatively small decline in strength or because of the nature of the task as noted above.

The activity of the wrist flexors was also monitored during the tasks in which the wrist extensors acted as the agonists. An increase in wrist flexor MEMG occurred in the tests of active joint stiffness at 50% of wrist extensor MVC, during the extensor torque tracking task and during the sustained contraction at 50% of MVC. The pattern of MEMG change for the wrist flexors, when acting as antagonists, over the 10 days of testing, was similar to that for the wrist extensors which supports hypothesis 6 . Coactivation of agonist and antagonist muscles is a common occurrence. It has been documented during the acquisition of a new skill (Solomonow et al. 1988) during maximal efforts of agonist muscles (Carolan and Cafarelli 1992; Dimitrijevic et al. 1992; Milner et al 1995) and in cases in where there is joint instability or load instability (Akazawa et al. 1983; Kalund et al. 1990; De Serres and Milner 1991). Coactivation of antagonists allows joint stiffness to be varied independently of joint torque. While it is clear that this enhancement of joint stability is important for joint protection

and movement control this is the first report of alterations in coactivation after exercise-induced muscle injury.

The increases in coactivation seen following the muscle injury were not excessive and occurred during tasks requiring control of torque which suggests that the increased coactivation of wrist flexors was due an increase in common drive to agonist and antagonist muscles (De Luca and Mambrito 1987) following muscle injury or due to reduced reciprocal inhibition of the antagonists whenever the injured muscles are activated.

Summary

Exercise-induced muscle injury by repeated maximal wrist extensions had a profound effect on control of posture and movement of the wrist. The reduced passive ROM, without change in passive or active wrist joint stiffness at the neutral position, suggested that there were changes to the mechanical properties of non-contractile tissue or that swelling occurred which only influenced resistance to movement at the extremes of the ROM. The reduced active wrist joint stiffness at 50% MVC and the impairment of control are consistent with the previous reports that larger muscle fibers are more affected than small muscle fibers (Fridén et al. 1983a; Ebbeling and Clarkson 1990; Fridén and Lieber 1992).

There was an impairment in the ability of the wrist extensors to track and sustain torque, as well as an impairment in the ability to stabilize a mechanically unstable load. The response of all of the wrist extensors following the exercise bout was similar, suggesting that they operated in a synergistic manner. Tests

performed with the wrist flexors as agonists showed no effects of the injury to the wrist extensors.

Many of the effects of eccentric exercise on wrist extensors were similar to those which have been reported for other muscle groups. The greatest deficit in maximal voluntary force occurred on Day 1 after the exercise bout and recovered by Day 10. Muscle soreness peaked on Day 2. The impaired ability to maintain steady torque and to smoothly track a target torque followed the time course of decline and recovery of MVC. This would suggest that the performance deficits were more closely linked to impaired muscle function than to sensation of soreness and supports hypothesis 7. The recovery of all parameters to pre-injury values by Day 10 would suggest that there were no long term adaptations to exercise-induced muscle injury.

CHAPTER FIVE - CONCLUSIONS

The purpose of this thesis was to examine several concepts with regard to exercise-induced muscle injury. These included: 1) changes in mechanical parameters that could affect functional performance; 2) changes in neurophysiological parameters that could affect functional performance; 3) functional performance in a variety of tests requiring various levels of joint torque and recruitment strategies; 4) the influence of muscle injury on the use of synergist and antagonist muscles; and 5) the time course of these changes. In order to meet this objective, several general hypotheses were tested in two studies. These studies have led to a greater understanding of motor control following a common musculoskeletal injury. Furthermore, these studies can serve as a model for other musculoskeletal disorders.

It was shown that under passive conditions there were no changes in joint stiffness at mid-range joint positions. General Hypothesis 1 stated that passive joint stiffness measured in mid-range of joint motion would increase as a result of exercise-induced muscle injury, but this was not supported by either study. The studies in this thesis were the first to quantify joint stiffness at a single mid-range joint position, where the joint is most frequently positioned during normal activities. The findings from both studies suggests that there were no changes in the mechanical properties of non-contractile tissue at the mid-range of joint motion and that the commonly reported sensations of stiffness had no effect on the ability to function or move in neutral joint positions. The decline in

passive joint ROM in the wrist study suggests that the resistance to movement found at the ends of ROM might have been due to swelling. However, this was not specifically examined in this thesis.

Experiments performed to investigate mechanical parameters related to functional performance supported General Hypotheses 2 and 3. Quantification of active joint stiffness revealed no changes in joint stiffness while the injured muscle(s) were producing low torque in either study. Decomposition of MP joint stiffness into its constituents did not reveal any changes either at low torque. This suggests that the mechanical properties of the small slow-twitch motor units, which would have been active during these tests, were unaffected by the eccentric exercise. On the other hand, there were significant changes in joint stiffness when the injured muscle(s) were producing high torque. It is at these levels of torque that the large fast-twitch motor units would be assumed to be active. The decline in active joint stiffness at high torque thus suggests that the mechanical properties of the large motor units had changed as a result of eccentric exercise. Further evidence of the changes in the mechanical properties of large muscle fibers was provided by the increase in stimulated intrinsic stiffness 24 hours after injury to the FDI muscle. The combination of these findings supports previously reported specificity of injury to the large muscle fibers.

Changes in reflex excitation were also evident in the experiments which evaluated active joint stiffness. In both studies, the short latency reflex EMG in response to a rapid stretch decreased in situations where activation of large

motor units was required. The finding that only short latency responses were affected suggests that the changes associated with the decline in reflex excitation occurred at the spinal cord level. A decline in the short latency reflex response has been reported after fatiguing contractions. It was suggested that this decline was mediated by group III and IV receptors which are sensitive to metabolic and mechanical changes associated with muscle fatigue. It is possible that these receptors could have been activated by injury to the large muscle fibers during eccentric exercise leading to presynaptic inhibition of the stretch reflex. It was beyond the scope of this thesis to investigate the specific mechanisms of this inhibition. However, it is likely that the inhibition affected joint stiffness and perhaps the amount of voluntary activation required to perform tasks.

Neurophysiological changes which accompanied the muscle injury also included a shift of the EMG power spectrum to a lower median frequency. The shift to a lower MF in the FDI study suggests that there was increased activation of motor units with slow conduction velocities. This may have been needed to offset a reduced contribution from large motor units because of injury to large muscle fibers. Increased EMG amplitude at sub-maximal torque levels was evident, suggesting that injury to large muscle fibers may have resulted in increased activity (firing rates) of small motor units to compensate. These findings supported General Hypothesis 5. The results of the wrist experiment, however, did not support this hypothesis. Although there was an increase in EMG for all three wrist extensor muscles at sub-maximal torque levels, there

was no accompanying decline in the MF. The failure to observe a decrease in the MF may have been due to the torque levels that were used, which may not have required the recruitment of large muscle fibers, or due to distribution of the load among the three wrist extensors. Berry at al. (1990) also reported no change in the MF of the quadriceps muscles during a sustained contraction following muscle injury. However, they studied four muscles as a single group. The FDI study was the first to show changes in the MF following exercise-induced muscle injury. This parameter may be useful for evaluation of the damage to muscle, and of subsequent changes in the pattern of activation of muscle fibers accompanying musculoskeletal disorders.

Changes in functional performance were clearly evident in both studies. For the most part, the decline in performance during tasks requiring sub-maximal activation supported General Hypothesis 4. Subjects' ability to perform tasks requiring control and sustained levels of torque was impaired after muscle injury. The decline in ability to perform these tasks was most evident at high torque. The increased difficulty may have been a result of the need to increase the number of motor units needed to reach a given torque level following muscle injury. The recruitment of motor units to compensate for the loss of force producing ability in some muscle fibers may have resulted in impaired fine control. The greater impairment at higher torques may reflect preferential damage to large muscle fibers by eccentric exercise. As the large motor units were recruited, some fibers may not have been able to generate force because they were damaged or because of inhibition by peripheral reflex mechanisms.

This may have, in turn, signaled the CNS to recruit additional motor units to compensate. This was suggested by the increased EMG for given submaximal torque levels in both studies. An impairment of the ability to stabilize a mechanically unstable load was also evident in both studies. This coupled, with the reduced active joint stiffness of the injured muscle(s) at high activation levels is further evidence of impairment in motor control following muscle injury.

It was anticipated that injury to large fibers would result in a decline of maximum velocity of movement, but in neither study was this the case. Miles et al. (1993) had previously reported a decline in maximum velocity of movement of the elbow following eccentric exercise by the elbow flexors. In the present studies, the tests may not have been true indicators of maximum velocity: in the case of the FDI because of the unfamiliarity of the task and in the case of the wrist extensors because there was an accuracy constraint. The limits of ability to rapidly recruit motor units may not have been tested. Further study is needed to clarify the effect of muscle injury on this capacity.

During the FDI study, the influence of an injured muscle on an uninjured synergist muscle was investigated. The findings supported General Hypothesis 7 and illustrated the effect that injury to one muscle could have on a synergist muscle. Inhibition of the uninjured FDS muscle, perhaps through heteronymous reflex actions, during maximal isometric MP flexion was evident. Inhibition of a close synergist by group III and IV afferents in a fatiguing muscle has previously been reported. Thus, the inhibition of the FDS in the FDI study may have been mediated by group III and IV afferents which responded to the injury in the FDI

muscle. The increased contribution of the FDS to produce submaximal MP flexion torque provided further evidence that muscle injury affects the control of uninjured synergist muscles.

The wrist study investigated the effect of muscle injury on uninjured antagonist muscles. It was hypothesized that activity of the wrist flexors would increase whenever the wrist extensors were activated sufficiently to recruit the injured motor units. The findings from this study supported this hypothesis. It is possible that, once again, this change was mediated by reflex pathways, which reduced reciprocal inhibition. However, the evidence is only indirect. Although the amount of coactivation produced was not high and had no apparent effect on performance, this finding demonstrates that muscle injury has widespread effects on surrounding musculature.

The impairment of performance and the measured mechanical and neurophysiological parameters recovered with the same time course as the recovery of maximum isometric torque. The ratings of sensations of soreness, however, followed a different time course, indicating that soreness was not the principal cause of impaired performance. The complete recovery of all measured parameters by Day 10 suggests that no long term adaptations occurred.

The findings of these studies are important for clinicians who treat patients with exercise-induced muscle injury. It is clear that the mechanical and neurophysiological changes that accompany muscle injury influence the ability to perform functionally. Patients should be made aware of these functional

deficits so as not to attempt tasks that may result in further injury. Clinicians should encourage rehabilitation of muscle force producing capability, rather than focus on sensations of soreness, in order to facilitate recovery and improve performance. Finally, clinicians should be aware of the effects of the injured muscle(s) on synergist and antagonist muscles, in order to assess fully the adaptations to exercise-induced muscle injury and the compensations which may occur during performance of tasks. The goal of treatment should focus on proper recruitment, rather than stressing the muscle during recovery with tasks that require high levels of activation.

The changes which occur after eccentric exercise are of interest for a number of reasons. There are practical considerations which have been mentioned above, but there is also the potential for studies such as this to provide insight into the mechanisms of underlying impairment or loss of function that could occur in other musculoskeletal disorders. Exercise-induced injury could serve as a model for other musculoskeletal disorders which result in injury to muscle tissue, soreness and declines in force producing capabilities. It is probable that given more severe and extensive muscle damage, the effects noted in this study would be magnified and would result in more long term adaptations. These may include selective inhibition of the injured muscles and increased activation of other muscles to compensate. Reinforcement of this pattern of recruitment over a period of time could result in the continued neuromuscular control problems and muscle imbalances common to many disorders. Improper muscle recruitment, including coordination and timing,

could lead to dominance or substitution where inappropriate muscles take over. This is likely to result in overuse of certain muscles and underuse of others resulting in muscle imbalance.

Normal sensory input and appropriate muscle activity amongst agonist, antagonist and synergists are prerequisites for normal function of the motor system. Performance deficits can occur for a number of reasons including muscle weakness, altered afferent input, and improper muscle recruitment. The results of this thesis have contributed to the understanding of the role of these mechanisms in a common musculoskeletal disorder, as well as the study of muscle injury in general. Future investigations which continue to focus on these mechanisms are warranted. The results of these studies would improve the understanding of the muscle injury recovery and make possible the development of tools for objective evaluation and strategies for treatment intervention.

Future studies that could be extended form this work include an investigation of the susceptibility of muscles with different motor unit compositions to exercise-induced muscle injury, as well as further studies of uninjured synergist muscles following muscle injury. This may include evaluation of the H-reflex and other tests of spinal reflex function which may prove to be useful in understanding the mechanisms of muscle imbalances. In addition, future studies could apply the methods in this thesis to investigate the underlying changes in mechanical and neurophysiological parameters which may accompany other musculoskeletal disorders and affect functional

performance, such as may occur in rheumatoid arthritis. The influence of common treatment techniques such as splinting and joint implants on these parameters could also be evaluated.

REFERENCES

Abbott, B. C., Bigland, B. and Ritchie, J. M. The physiological cost of negative work. *J. Physiol.* 117: 380-390, 1952

Akazawa, A., Milner, T. E. and Stein, R. B. Modulation of reflex EMG and stiffness in response to stretch of human finger muscles. *J. Neurophysiol.* : 16-27, 1983

Armstrong, R. B. Mechanisms of exercise-induced delayed onset muscle soreness: a brief review. *Med. Sci. Sports Exerc.* 16: 529-538, 1984

Armstrong, R. B. Initial events in exercise-induced muscular injury. *Med. Sci. Sports Exerc.* 22: 429-435, 1990

Armstrong, R. B., Ogilvie, R. W. and Schwane, J. A. Eccentric exercise-induced injury in rat skeletal muscle. *J. Appl. Physiol.* 54: 80-93, 1983

Asmussen, E. Positive and negative muscular work. *Acta Physiol. Scand.* 28: 365-382, 1953

Aura, O. and Komi, P. V. Mechanical efficiency of pure positive and pure negative work with special reference to the work intensity. *Int. J. Sports Med.* 7: 44-49, 1986

Balestra, C., Duchateau, J. and Hainaut, K. Effects of fatigue on the stretch reflex in a human muscle. *Electroenceph. clin. Neurophysiol.* 85: 46-52, 1992

Balnave, C. D. and Thompson, M. W. Effect of training on eccentric exercie-induced muscle damage. *J. Appl. Physiol.* 75(4): 1545-1551, 1993

Basmajian, J. R. *Muscles Alive: Their Function Revealed by Electromyography*, 2nd edition, p. 221. Williams and Wilkins, Baltimore, 1967

Bellemare, F. and Garnzaniti, N. Failure of neuromuscular propagation during maximum voluntary contraction. *J. Appl. Physiol.* 64:1084-1093, 1988

Bennett, D. J., Hollerbach, J. M., Xu, Y. and Hunter, I. W. Time-varying stiffness of human elbow joint during cyclic voluntary movement. *Exp. Brain Res.* 88: 433-442, 1992

Bennett, J. G and Stauber, W. T. Evaluation and treatment of anterior knee pain using eccentric exercise. *Med. Sci. Sport Exerc.* 18: 526-530, 1986

Bernstein, N. The problem of interrelation of co-ordination and localization. In: *The Co-ordination and Regulation of Movements*. New York: Pergamon, 15-19, 1967

Berry, C. B., Moritani, T. and Tolson, H. Electrical activity and soreness in muscles after exercise. *Am. J. Phys. Med. Rehabil.* 69:60-66, 1990

Bigland-Ritchie, B., Cafarelli, E. and Vollestad, N. K. Fatigue of submaximal static contractions. *Acta Physiol. Scand.* 128 (suppl 556): 137-148, 1986b

Bigland-Ritchie, B., Dawson, N. J., Johansson, R. S. and Lippold, O. C. J. Reflex origin for the slowing of motoneurone firing rates in fatigue of human voluntary contractions. *J. Physiol.* 379:451-459, 1986a

Bigland-Ritchie, B., Donovan, E. F. and Roussos, C. S. Conduction velocity and EMG power spectrum changes in fatigue of sustained maximal efforts. *J. Appl. Physiol.* 51: 1300-1305, 1981

Bigland-Ritchie, B., Kukulka, C. G., Lippold, O. C. J. and Woods, J. J. Excitation frequency and muscle fatigue: electrical responses during human voluntary and stimulated contractions. *J. Physiol. (Lond)* 330: 265-278, 1982

Bigland, B. and Lippold, O. C. J. The relation between force, velocity, and integrated electrical activity in human muscles. *J. Physiol.* 123: 214-224, 1954

Bigland-Ritchie, B. and Woods, J. J. Integrated EMG and O_2 uptake during positive and negative work. *J. Physiol. (Lond)* 260: 267-277, 1976

Bilodeau, M., Cincera, M., Gervais, S., Arsenault, A. B., Gravel, D., Lepage, Y. and McKinley, P. Changes in the electromyographic spectrum power distribution caused by a progressive increase in the force level. *Eur. J. Appl. Physiol.* 71:113-123, 1995

Bobbert, M. F., Hollander, A. P. and Huijing, P. A. Factors in delayed onset muscular soreness of man. *Med Sci. Sports Exerc.* 18: 75-81, 1986

Brown, L. M. and Hill, L. Some observations on variations in filament overlap in tetanized muscle fibers and fibers stretched during tetanus, detected in the electron microscope after rapid fixation. *J. Muscle Res. Cell Motil.* 12:171-182, 1991

Brown, I. E. and Loeb, G. E. A " damaged" state in cat caudofemoralis muscle induced by high forces. *Soc. Neurosci. Abs.* 25: 85, 1994

Buchanan, T.S., Rovai, G. P. and Rymer, W. Z. Strategies for muscle activation during isometric torque generation at the human elbow. *J. Neurophysiol.* 1201-1212, 1989

Bulbulian, R. and Bowles, D. K. Effect of downhill running on motoneuron pool excitability. *J. Appl. Physiol.* 73(3): 968-973, 1992

Byrd, S. K. Alterations in the sarcoplasmic reticulum: a possible link to exercise-induced muscle damage. *Med. Sci. Sports Exerc.* 24: 531-536, 1992

Byrnes, W. , Clarkson, P. M., White, W., Hsieh, S. S., Frykman, P. N. and Maughan, R. J. Delayed onset muscle soreness following repeated bouts of downhill running. *J. Appl. Physiol.* 59(3): 710-715, 1985

Canon, S. C. and Zahalak, G. I. The mechanical behavior of active human skeletal muscle in small oscillations. *J. Biomech.* 15, 111-121, 1982

Carlson, B. and Faulkner, J. The regeneration of skeletal muscle fibers following injury: a review. *Med. Sci. Sports Exerc.* 15 (3): 187-198, 1983

Carolan, B. and Cafarelli, E. Adaptations in coactivation after isometric training. *J. Appl. Physiol.* 73 (3): 911-917, 1992

Carter, R. R., Crago, P. E. and Keith, M. W. Stiffness regulation by reflex action in the normal human hand. *J. Neurophysiol.* 64: 105-118, 1990

Clarkson, P. Exercise-induced muscle damage - animal and human models. *Med. Sci. Sports Exerc.* 24: 510-511, 1992

Clarkson, P. M., Byrnes, W. C., Gillisson, E. and Harper, E. Adaptation to exercised-induced muscle damage. *Clin. Sci.* 73: 383-386, 1987

Clarkson, P., Byrnes, W., McCormick, K. M., Turcotte, L. P. and White, J. S. Muscle soreness and serum creatine kinase activity following isometric, eccentric and concentric exercise. *Int. J. Sports Med.* 7: 152-155, 1986

Clarkson, P. M., Nosaka, K. and Braun, B. Muscle function after exercise-induced muscle damage and rapid adaptation. *Med. Sci. Sports Exerc.* 24: 512-520, 1992

Clarkson, P. and Tremblay, I. Exercise-induced muscle damage, repair, and adaptation in humans. *J. Appl. Physiol.* 65(1): 1-6, 1988

Crenshaw, A. G., Thornell, L. E. and Fridén, J. Intramuscular pressure, torque and swelling for the exercise-induced sore vastus lateralis muscle. *Acta Physiol. Scand.* 152: 265-277, 1994

Cupido, C. M., Galea, V. and McComas, A. J. Potentiation and depression of the M wave in the human biceps brachii. *J. Physiol.* 491.2: 541-550, 1996

Curtin, N. and Davies, R. E. Chemical and Mechanical changes during stretching of activated frog skeletal muscle. *Cold Spring Harbour Symposia on Quantitative Biology.* 37: 619-626, 1973

Curwin, S. and Stanish, W. D. (Ed.) *Tendonitis: its etiology and treatment.* Lexington, Massachusetts: D. C. Heath and Company, 1984

Daanen, H. A. M., Mazure, M., Holewijn, M. and Van der Velde, E. A. Reproducibility of the mean power frequency of the surface electromyogram. *Eur. J. Appl. Physiol.* 61: 274-277, 1990

Davies, C. T. M. and Barnes, C. Negative (eccentric) work. I Effects of repeated exercise. *Ergonomics* 15: 3-14, 1972

Davies, C. T. M. and White, M. J. Muscle weakness following eccentric work in man. *Pflügers Arch.*392: 168-171, 1981

de la Barrera, E. J. and Milner, T. E. The effects of skinfold thickness on the selectivity of surface EMG. *Electroenceph. Clin. Neurophysiol.* 93: 91-99, 1994

De Luca, C. Myoelectric manifestation of localized muscular fatigue in humans. *CRC Crit. Rev. Biomed. Eng.* 11: 251-279, 1985

De Luca, C., LeFever, R., McCue, M. and Xenakis, A. Behavior of human motor units in different muscles during linearly varying contraction. *J. Physiol. (Lond)* 329: 113-128, 1982

De Serres, S. J. and Milner, T. E. Wrist muscle activation patterns and stiffness associated with stable and unstable mechanical loads. *Exp. Brain Res.* 86: 451-458, 1991

Desmedt, J. E. and Gadaux, E. Spinal motoneuron recruitment in man: deordering with direction, but not with speed of voluntary movement. *Science* 214: 933-936, 1981

Dick, R. W. and Cavanagh, P. R. An explanation of the upward drift in oxygen uptake during prolonged sub-maximal downhill running. *Med. Sci. Sports Exerc.* 19: 310-317, 1987

Dimitrijevic, M. R., McKay, W. B., Sarjanovic, I., Sherwood, A. M., Svirtlih, L. and Vrbovà, G. Co-activation of ipsi- and contralateral muscle groups during contraction of ankle dorsiflexors. *J. Neurol. Sci.* 109: 49-55, 1992

Doemges, F. and Rack, P. M. H. Changes in the stretch reflex of the human first dorsal interosseous muscle during different tasks. *J. Physiol.* 447: 563-573, 1992

Donnelly, A. E., Clarkson, P. M. and Maughan, R. J. Exercise-induced muscle damage: effects of light exercise on damaged muscle. *Eur. J. Appl. Physiol.* 64: 350-353, 1992

Duchateau, J., Guissard, N. and Hainaut, K. EMG power spectrum of disused muscle during fatigue. In Anderon, P. A., Hobart, D. J. and Danoff, J. V. (Eds.) *Electromyographical Kinesiology* (pp 311-314). Elsevier Science Publishers B.V., 1991

Duchateau, J. and Hainaut, K. Electrical and mechanical failures during sustained and intermittent contractions in humans. J. Appl. Physiol. 58: 942-947, 1985

Duchateau, J. and Hainaut, K. Behaviour of short and long latency reflexes in fatigued human muscles. *J. Physiol.* 471: 787-799, 1993

Ebbeling, C. and Clarkson, P. Exercise-induced muscle damage and adaptation. *Sports Med.* 7: 207-234, 1989

Edman, K. A. P. The velocity of unloaded shortening and its relation to sarcomere length and isometric force in vertebrate muscle fibres. *J. Physiol. (Lond)* 291: 143-159, 1979

Edman, K. A. P., Elzinga, G. and Noble, M. I. M. Enhancement of mechanical performance by stretch during tetanic contractions of vertebrate skeletal muscle fibers. *J. Physiol.* 281: 139-155, 1978

Edwards, R. H. T. Greater low frequency fatigue produced by eccentric than concentric muscle contractions. *J. Physiol. (Lond)* 317: 17p, 1981

Enoka, R. (Ed.), *Neuromechanical Basis of Kinesiology*, Champaign, IL: Human Kinetics. 277-280, 1994

Enoka, R. M., Robinson, G. A. and Kossev, A. R. Task and fatigue effects on low-threshold motor units in human hand muscle. J. *Neurophysiol.* 62: 1344-1359, 1989

Evans, W. J. and Cannon, J. G. The Metabolic Effects of Exercise-induced muscle damage, In J.O Holoszy (Ed.),*Exercise and Sport Science Reviews*, Baltimore: Williams and Wilkins. 19: 99-125, 1991

Fatt, P. and Katz, B. The electrical properties of crustacean muscle fibers. *J. Physiol.* 120: 171-204, 1953

Faulkner, J. A., Brooks, S. V. and Opiteck, J. A. Injury to skeletal muscle fibers during contractions: conditions of occurrence and prevention. *Phys. Ther.* 73: 911-921, 1993

Fenton, J., Garner, S. and McComas, A. J. Abnormal M-wave responses during exercise in myotonic muscular dystrophy: a Na^+-K^+ pump defect. *Muscle Nerve* 14: 79-84, 1991

Feinstein, B., Lindegård, B., Nyman, E. and Wohlfart, G. Morphological studies of motor units in normal human muscle. *ACTA Anat.* 23: 127-142, 1955

Fitch, S. and McComas, A. J. Influence of human muscle length on fatigue. *J. Physiol.* 362: 205-213, 1985

Flament, D., Goldsmith, P., Buckley, C. J. and Lemon, R.N. Task dependence of response in the first dorsal interosseous muscle to magnetic brain stimulation in man. *J. Physiol. (Lond)* 464: 361-378, 1993

Fridén, J. and Lieber, R. L. Structural and mechanical basis of exercise-induced muscle injury. *Med. Sci. Sports Exerc.* 24: 521-530, 1992

Fridén, J., Lieber, R.L. and Thornell, L. E. Subtle indications of muscle damage following eccentric contractions. *Acta Physiol. Scand.* 142: 523-524, 1991

Fridén, J., Seger, J. and Ekblom, B. Sublethal muscle fiber injuries after high-tension anaerobic exercise. *Eur. J. Appl. Physiol.* 57: 360-368, 1988

Fridén, J., Seger, J., Sjöström, M. and Ekblom, B. Adaptive response in human skeletal muscle subjected to prolonged eccentric training. *Int. J. Sports Med.* 4: 177-183, 1983b

Fridén, J., Sfakianos, P. N. and Hargens, A. R. Delayed muscle soreness and intramuscular fluid pressure: comparison between eccentric and concentric load. *J. Appl. Physiol.* 61:2175 -2179, 1986

Fridén, J., Sjöström, M. and Ekblom, B. Myofibrillar damage following intense eccentric exercise in man. *Int. J. Sports Med.* 4: 170-176, 1983a

Galea, V. and McComas, A. J. Effects of ischemia on M-wave potentiation in the human biceps brachii. *J. Physiol. (Lond)* 438: 212P, 1991

Galganski, M. E., Fuglevand, A. J. and Enoka, R. M. Reduced control of motor unit output in a human hand muscle of elderly subjects during submaximal contractions. *J. Neurophysiol.* 69: 2108-2115, 1993

Gamet, D. and Maton, B. The fatigability of two agonistic muscles in human isometric voluntary submaximal contraction: an EMG study. I. Assessment of muscular fatigue by means of surface EMG. *Eur. J. Appl. Physiol.* 58:361-368, 1989

Gerdle, B., Henriksson-Larsén, K., Lorentzon, R. and Wretling, M. L. Dependence of the mean power frequency of the electromyogram on muscle force and fiber type. *Acta Physiol. Scand.* 142: 457-465, 1991

Ghez, C. and Gordon, J. Trajectory control in targeted force impulses. I. Role of opposing muscles. *Exp. Brain Res.* 67: 225-240, 1987

Ghez, C. and Martin, J. H. The control of rapid limb movements in the cat. III. Agonist - antagonist coupling. *Exp. Brain Res.* 45: 115-125, 1982

Gordon, A. M., Huxley, A. F. and Julian, F. J. The variation in isometric tension with sarcomere length in vertebrate muscle fibers. *J. Physiol.* 184: 170-192, 1966

Hagbarth, K. E. and Young, R. R. Participation of the stretch reflex in human physiological tremor. *Brain.* 102: 509-526, 1979

Hagberg, M. and Ericson, B. E. Myoelectric power spectrum dependence on muscular contraction level of elbow flexors. *Eur. J. Appl. Physiol.* 48: 146-156, 1982

Hagiwara, S., Chichibu, S. and Naka, K. The effects of various ions on resting and spike potentials of barnacle muscle fibers. *J. Gen. Physiol.* 48: 163-179, 1964

Hamill, J., Freedson, P. S., Clarkson, P. M. and Braun, B. Effects of delayed onset muscle soreness on lower extremity function during running. *Med. Sci. Sport Exerc.* 22: S5, 1990

Harada, Y. and Koubu, Y. The inhibition of muscle activity from the central nervous system in rheumatoid arthritis with special reference to the pathogenesis of muscle atrophy. *Electromyographical Kinesiology, Elsevier Science Publishers B.V. (Biomedical Division),* 417-420, 1991

Harris, C., Wilcox, A., Smith, G., Quinn, C. and Lawson, L. The effect of delayed onset muscle soreness (DOMS) on running kinematics. *Med. Sci. Sport Exerc.* 22 (2): S200, 1990

Harry, J. D., Ward, A. W., Heglund, N. C., Morgan, D. L. and McMahon, T. A. Crossbridge cycling theories cannot explain high-speed lengthening behavior in frog muscle. *Biophys. J.* 57: 201-208, 1990

Hasson, S. M., Daniels, J. C., Divine, J. G., Niebuhr, B. R., Richmond, S., Stein, P. G. and Williams, J. H. Effect of ibuprofen use on muscle soreness, damage, and performance: a preliminary investigation. *Med. Sci. Sports Exerc.* 25(1): 9-17, 1993

Hayward, L., Breitbach, D. and Rymer, W. Z. Increased inhibitory effects on close synergists during muscle fatigue in the decerebrate cat. *Brain Res.* 440: 199-203, 1988

Hayward, L., Wesselmann, U. and Rymer, W. Z. Effects of muscle fatigue on mechanically sensitive afferents of slow conduction velocity in the cat triceps surae. *J. Neurophysiol.* 65: 360-370, 1991

Hicks, A., Cupido, C. M., Martin, J. and Dent, J. Muscle excitation in elderly adults:the effects of training. *Muscle Nerve* 15:87-93, 1992

Hicks, A., Fenton, J., Garner, S. and McComas, A. J. M wave potentiation during and after muscle activity. *J. Appl. Physiol.* 66(6): 2606-2610, 1989

Hicks, A. and McComas, A. J. Increased sodium pump activity following repetitive stimulation of rat soleus muscles. *J. Physiol.* 414: 337-349, 1989

Hoffer, J. A. and Andreasson, S. Regulation of soleus muscle stiffness in premammillary cats: intrinsic and reflex components. *J. Neurophysiol.* 45; 267-285, 1981

Hoffman, D. S. and Strick, P. L. Step-tracking movements of the wrist in humans. II. EMG analysis. *J. Neurosci.* 10 (1): 142-152, 1990

Hogan, N. J. Adaptive control of mechanical impedance by coactivation of antagonist muscles. *IEEE Trans. Automat. Contr.* 29: 681-690, 1984

Hone, L., Siler, W. and Schwane, J. Does delayed onset muscle soreness affect oxygen consumption and selected gait parameters during running? *Med. Sci. Sport Exerc.* 22 (2): S202, 1990

Howell, J. N., Chelboun, G. and Conatser, R. Muscle stiffness, strength loss, swelling, and soreness following exercise-induced injury in humans. *J. Physiol.* 464: 183-196, 1993

Howell, J. N., Chila, A. G., Ford, G., David, D. and Gates, T. An electromyographic study of elbow motion during postexercise muscle soreness. *J. Appl. Physiol.* 58(5): 1713-1718, 1985

Hultborn, H. Convergence on interneurons in the reciprocal Ia inhibitory pathway to motoneurons. *Acta Physiol. Scand. Suppl.* 375:1-42, 1972

Huxley, A. F. Muscle structure and theories of contraction. *Pro. Biophys. Biophys. Chem.* 7: 255-318, 1957

Huxley, A. F. and Peachy, L. D. The maximum length for contraction in vertebrate striated muscle. *J. Physiol.* 156: 150-165, 1961

Huxley, H. E. The mechanism of muscular contraction. *Science* 164: 1356-1366, 1969

Jaeger, R. J., Gottlieb, G. L. and Agarwal, G. C. Myoelectric responses at flexors and extensors of human wrist to step torque perturbations. *J. Neurophysiol.* 48:388-402, 1982

Johnson, M. A., Polgar, J., Weightman, D. And Appleton, D. Data on the distribution of fiber types in thirty-six human muscles: an autopsy study. *J. Neurol. Sci.* 18:111-129, 1973

Jones, D. A., Newham, D. J. and Clarkson, P. M. Skeletal muscle stiffness and pain following eccentric exercise of the elbow flexors. *Pain.* 30: 233-242, 1987a

Jones, D. A., Newham, D. J., Round, J. M. and Tolfree, S. E. J. Experimental human muscle damage: morphological changes in relation to other indices of damage. *J. Physiol.* 375: 435-448, 1986

Jones, D. A., Newham, D. J. and Torgan, C. Mechanical influences on long-lasting human muscle fatigue and delayed-onset muscle pain. *J. Physiol.* 412: 415-427, 1989

Jones, D. A. and Rutherford, O. M. Human muscle strength training: the effects of three different regimes and the nature of the resultant changes. *J. Physiol. (Lond)* 391: 1-11, 1987b

Jones, K. E., Lyons, M., Bawa, P. and Lemon, R. N. Recruitment order of motoneurons during functional tasks. *Exp. Brain Res.* 100: 503-508, 1994

Jongen, H. A. H., Denier van der Gon, J. J. and Gielen, C. C. A. M. Inhomogeneous activation of motoneurone pools as revealed by co-contraction of antagonistic human arm muscles. *Exp Brain Res.* 75: 555-562, 1989

Johnson, M. A., Polgar, J. Weightman, D. and Appleton, D. Data on the distribution of fiber types in thirty-six human muscles. *J. Neurol. Sci.* 18: 111-129, 1973

Jubrias, S. and Klug, G. A. Are eccentric contractions required to induce the skeletal muscle fiber disruption that occurs following unaccustomed activity? *Muscle Nerve*, Dec: 1422-1423, 1993

Kalund, S., Sinkjaer, T., Arendt-Nielsen, L. and Simonsen, O. Altered timing of hamstring muscle action in anterior cruciate ligament deficient patients. *Am. J. Sports Med.* 18: 245-248, 1990

Katz, B. The relation between force and speed in muscular contraction. *J. Physiol.* 96: 45-64, 1939

Kearney, R. E. and Hunter, I. W. System identification of human joint dynamics. *Crit. Rev. Biomed. Eng.*, 18: 55-86, 1990

Ketchum, L. D., Thompson, D., Pocock, G. and Wallingford, D. A clinical study of forces generated by the intrinsic muscles of the index finger and the extrinsic flexor and extensor muscles of the hand. *J. Hand Surg.* 3: 571-578, 1978

Klausen, K. and Knuttgen, H. G. Effects of training on oxygen consumption in negative muscular work. *Acta Physiol. Scand.* 83: 319-323, 1971

Knuttgen, H. G. Human performance in high-intensity exercise with concentric and eccentric muscle contractions. *Int. J. Sport Med.* (*suppl*) 7: 6-9, 1986

Knuttgen, H. G., Bonde-Peterson, F. and Klausen, K. Oxygen uptake and heart rate response to exercise performed with concentric and eccentric muscle contractions. *Med. Sci. Sports* 3: 1-5, 1971

Koester, J. Passive membrane properties of the neuron. In Kandel, E. R., Schwartz, J. H. and Jessell, T. M. (Eds.) *Principles of neural science* (3rd. ed., pg 100). New ypork: Elsevier, 1991

Komi, P. V. Relationship between muscle tension, EMG, and velocity of contraction under concentric and eccentric work. In Desmedt J.E. (ed): *New Developments in Electromyography and Clinical Neurophysiology.* Karger, Basel, vol 1, 596-606, 1973

Komi, P. V. and Tesch, P. EMG frequency spectrum, muscle structure and fatigue during dynamic contractions in man. *Eur. J. Appl. Physiol. Occup. Physiol.* 42: 41-50, 1970

Komi, P. V. and Viitasalo, J. T. Changes in motor unit activity and metabolism in human skeletal muscle during and after repeated eccentric and concentric contractions. *Acta physiol. scand.* 100: 246-254, 1977

Korr, I. M. Proprioceptors and somatic dysfunction. *J. Am. Osteopath. Assoc.* 74: 638-650, 1976

Kroon, G. W. and Naeije, M. Recovery of the biceps electromyogram after heavy eccentric, concentric or isometric exercise. *Eur. J. Appl. Physiol.* 63: 444-448, 1991

Kuipers, H. Exercise-induced muscle damage. *Int. J. Sports Med.* 15: 132-135, 1993

Kuipers, H., Keizer, H. A., Verstappen, F. T. J. and Costill, D. L. Influence of a prostaglandin-inhibiting drug on muscle soreness after eccentric work. *Int. J. Sports Med.* 6: 336-339, 1985

Kupa, E. J., Roy, S. H., Kandarian, S. C. and De Luca, C. J. Effects of muscle fiber type and size on EMG median frequency and conduction velocity. *J. Appl. Physiol.* 79(1): 23-32, 1995

Landsmeer, J. M. F. and Long, C. The mechanism of finger control, based on electromyograms and location analysis. *Acta Anat.* 60: 330-347, 1965

Lieber, R. L. and Baskin, R. J. Intersarcomere dynamics of single skeletal muscle fibers during fixed-end tetani. *J. Gen. Physiol.* 82: 347-364, 1983

Lieber, R. L. and Fridén, J. Selective damage of fast glycolitic muscle fibers with eccentric contraction of the rabbit tibialis anterior. *Acta Physiol. Scand.* 133: 587-588, 1988

Lieber, R. L. and Fridén, J. Muscle damage is not a function of muscle force but active muscle strain. *J. Appl. Physiol.* 74: 520-526, 1993

Lieber, R. L., Woodburn, T. M. and Fridén, J. Muscle damage induced by eccentric contractions of 25% strain. *J. Appl. Physiol.* 70(6): 2498-2507, 1991

Lindström, L., Magnusson, R. and Petersén, I. Muscular fatigue and action potential conduction velocity changes studied with frequency analysis of EMG signals. *Electromyography* 4:341-356, 1970

Lombardi, V. and Piazzesi, G. The contractile response during steady lengthening of stimulated frog muscle fibers. *J. Physiol. (Lond)* 431: 141-171, 1990

MacIntyre, D. L. Delayed muscle soreness, muscle function, and evidence of leukocytes in human skeletal muscle following eccentric exercise, Ph. D. dissertation, University of British Columbia, 1994

MacIntyre, D. L., Reid, W. D., Lyster, D. M., Szasz, I J. and McKenzie, D. M. Presence of WBC, decreased strength, and delayed soreness in muscle after eccentric exercise. *J. Appl. Physiol.* (80)3: 1006-1013, 1996

Macpherson, J. M. Strategies that simplify the control of quadrupedal stance. II. Electromyographic activity. *J. Neurophysiol.* 60: 218-231, 1988

Marsden, C. D., Obeso, J. A. and Rothwell, J. C. The function of the antagonist muscle during fast limb movements in man. *J. Physiol.* 335: 1-13,1983

Maton, B. and Gamet, D. The fatigability of two agonistic muscles in human isometric voluntary submaximal contraction: an EMG study. II. Motor unit firing rate and recruitment. *Eur. J. Appl. Physiol.* 58: 369-374, 1989

McArdle, A., Edwards, R. H. T. and Jackson, M. J. Release of Creatine kinase and prostaglandin E_2 from regenerating skeletal muscle fibers. *J. Appl. Physiol.* 76(3): 1274-1278, 1994

McComas, A. J. Human neuromuscular adaptations that accompany changes in activity. *Med. Sci. Sports Exerc.* 26: 1498 -1509, 1994

McComas, A. J., Galea, V. and Einhorn, R. W. Pseudofacilitation: a misleading term. *Muscle Nerve* 17: 599-607, 1994

McCully, K. and Faulkner, J. Characteristics of lengthening contractions associated with injury to skeletal muscle fibers. *J. Appl. Physiol.* 61(1): 293-299, 1986

Meech, R. W. and Strumwasser, F. Intracellular calcium injection activates potassium conductance in Aplysia nerve cells. *Fed. Proc.* 29: 834, 1970

Miles, M. P., Clarkson, P. M., Bean, M., Ambach, K., Mulroy, J. and Vincent, K. Muscle function at the wrist following 9 d of immobilization and suspension. *Med. Sci. Sports Exerc.* 26: 615-623, 1994

Miles, M. P., Ives, J. and Vincent, K. R. Neuromuscular control following maximal eccentric exercise. *Med. Sci. Sports Exerc.* 25: S176, 1993

Milner, T. E. and Cloutier, C. Compensation for mechanically unstable loading in wrist movements. *Exp. Brain Res.*, 94: 522-532, 1993

Milner, T. E., Cloutier, C., Leger, A. B. and Franklin, D. W. Inability to maximally activate muscles during co-contraction and the effects on joint stiffness. *Exp. Brain Res.* 107: 293- 305, 1995

Milner-Brown, H. S. and Miller R. G. Muscle membrane excitation and impulse propagation velocity are reduced during muscle fatigue. *Muscle Nerve* 9: 367-374, 1986

Milner-Brown, H. S., Stein, R. B. and Yem, R. The orderly recruitment of human motor units during linearly changing voluntary contractions. *J. Physiol. (Lond)* 230: 359-370, 1973

Morgan, D. L. New insights into the behavior of muscle during active lengthening. *Biophys. J.* 57: 209-221, 1990

Moritani, T., Muramatsu, S. and Muro, M. Activity of motor units during concentric and eccentric contractions. *Am. J. Phys. Med.* 66: 338-349, 1988

Moritani, T., Nagata, A. and Muro, M. Electromyographic manifestations of muscular fatigue. *Med. Sci. Sports Exerc.* 14: 198-202, 1982

Nagata, A. and Christianson, J. C. M-Wave modulation at relative levels of maximal levels of maximal voluntary contraction. *Eur. J. Appl. Physiol.* 71: 77-86, 1995

Nakazawa, K., Kawakami, Y., Fukunaga, T., Yano, H. and Miyashita, M. Differences in activation patterns in elbow flexor muscles during isometric, concentric and eccentric contractions. *Eur. J. Appl. Physiol.* 66:214-220, 1993

Nardone, A. and Schieppati, M. Shift of activity to fast muscle during lengthening contractions of the triceps surae muscles in humans. *J. Physiol.* 395: 363-381, 1988

Nashner, L. M. Analysis of stance posture in humans. In: *Handbook of Behavioural Neurobiology*, edited by A. L. Towe and E. S. Luscher. New York: Plenum, 527-565, 1981

Newham, D. J. The consequences of eccentric contractions and their relationship to delayed onset muscle soreness. *Eur. J. Appl. Physiol.* 58: 353-359, 1988

Newham, D. J. and Jones, D. A. Intra-muscular pressure in the painful human biceps. *Clin Sci.* 69:27P, 1985

Newham, D. J., Jones, D. A. and Clarkson, P. M. Repeated high-force eccentric exercise: effects on muscle pain and damage. *J. Appl. Physiol.* 63, 1381-1386, 1987

Newham, D. J., Jones, D. A., Ghost, G. and Aurora, P. Muscle fatigue and pain after eccentric contractions at long and short length. *Clin. Sci.* 74: 553-557, 1988

Newham, D. J., Mills, K. R., Quigley, B. M. and Edwards, H. T. Pain and fatigue after concentric and eccentric muscle contractions. *Clin. Sci.* 64: 55-62, 1983

Nichols, T. R. and Houk, J. C. Improvement in linearity and regulation of stiffness that results from from actions of the stretch reflex. *J. Neurophysiol.* 39: 119-142, 1976

Pearson, D. R. and Costill, D. C. The effects of constant external resistance exercise and isokinetic exercise training on work-induced hypertrophy. *J. Appl. Sport Sci. Res.* 2: 39-41, 1988

Petit, J., Filippi, G. M., Emonet-Dénand, F., Hunt, C. C. and Laporte, Y. Changes in muscle stiffness produced by motor units of different types in peroneus longus muscle of cat. *J. Neurophysiol.* 63: 190-197, 1990

Pousson, M., Van Hoeke, J. and Goubel, F. Changes in elastic characteristics of human muscle induced by eccentric exercise. *J. Biomech.* 23: 343-348, 1990

Rodenburg, J. B., Bär, P. R. and De Boer, R. W. Relations between muscle soreness and biochemical and functional outcomes of eccentric exercise. *J. Appl. Physiol.* 74: 2976-2983, 1993

Roy, S. Combined use of surface electromyography and ^{31}P-NMR spectroscopy for the study of muscle disorders. *Phys. Ther.* 73: 892-901, 1993

Sargeant, A. J. and Dolan, P. Human muscle function following prolonged eccentric exercise. *Eur. J. Appl. Physiol.* 56: 704-711, 1987

Saxton, J. M., Clarkson, P. M., James, R., Miles, M., Westerfer, M., Clark, S. and Donnelly, A.E. Neuromuscular function following maximum voluntary eccentric exercise. *Med. Sci. Sports Exerc.* 26: S115, 1994a

Saxton, J. M., Clarkson, P. M., James, R., Miles, M., Westerfer, M., Clark, S. and Donnelly, A.E. Neuromuscular dysfunction following eccentric exercise. *Med. Sci. Sports Exerc.* 27: 1185-1193, 1995

Saxton, J. M. and Donnelly, A. E. Characteristics of impaired voluntary torque generating capacity after maximal voluntary eccentric muscular work in man. *J. Physiol.* 477.P: 57P-58P, 1994b

Schwarz, N. H., Podoll, K. and Motamedi, F. Evidence that low-threshold muscle afferents evoke long-latency stretch reflexes in human hand muscles. *J. Neurophysiol.* 65: 1089-1097, 1991

Sherrington, C. Reflex inhibition as a factor in the co-ordination of movements and posture. *Q. J. Exp. Physiol.* 6: 251-310, 1913

Singh, M. and Karpovich, P. V. Isotonic and isometric forces of forearm flexors and extensors. *J. Appl. Physiol.* 21: 1435-1437, 1966

Sinkjaer, T., Toft, E., Andreassen, S. and Hornemann, B. C. Muscle stiffness in human ankle dorsiflexors: intrinsic and reflex components. *J. Neurophysiol.* 60: 1110-1121, 1988

Solomonow, M., Baratta, R., Zhou, B. H. and D'Ambrosia, R. Electromyogram coactivation patterns of the elbow antagonist muscles during slow isokinetic movements. *Exp. Neurol.* 100: 470-477, 1988

Solomonow, M., Baten, C., Smit, J., Baratta, R., Hermens, H., D'Ambrosia, R. and Shoji, H. Electromyogram power spectra frequencies associated with motor unit recruitment strategies. *J. Appl. Physiol.* 68(3): 1177-1185, 1990

Spencer, J. D., Hayes, K. C. and Alexander, I. J. Knee joint effusion and quadriceps reflex inhibition in man. *Arch. Phys. Med. Rehabil.* 65: 171-177, 1984

Smith, L. Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Med. Sci. Sports Exerc.* 23: 542-551, 1991

Stauber, W. Eccentric action of muscles: physiology, injury, an adaptation. In Pandolf K. B. (Ed.),*Exs. Sport Sci Review*, Baltimore: Williams and Wilkins. 17: 157-185, 1989

Stauber, W. T., Clarkson, P. M., Fritz, V. K. and Evans, W. J. Extracellular matrix disruption and pain after eccentric muscle action. *J. Appl. Physiol.* 69: 868-874, 1990

Stauber, W. T., Fritz, V. K., Vogelbach, D. W. and Burkhardt, D. Characterization of muscle injured by forced lengthening. I. Cellular infiltrates. *Med. Sci. Sports Exerc.* 20: 345-353, 1988

Stein, R. B. and Lee, R.G. Tremor and clonus. In: *Motor Control: Handbook of Physiology*, Sect. 1, vol. 2, V. B. Brooks (Ed.). Bethesda, MD: American Physiological Society, 1981, pp. 325-343, 1981

Stephens, J. A. and Taylor, A. Fatigue maintained voluntary muscle contraction in man. *J. Physiol. (Lond)* 220:1-18, 1972

Tax, A. A. M., Danier van der Gon, J. J. and Erkelens, C. J. Differences in coordination of elbow flexor muscles in force tasks and movement tasks. *Exp. Brain Res.* 81: 567-572, 1990

Thilmann, A. F., Schwarz, M., Topper, R., Fellows, S. F. and Noth, J. Different mechanisms underlie the long-latency reflex response of active human muscle at different joints. *J. Physiol.* 444: 631-643, 1991

Thomas, C. K., Ross, B. H. and Calancie, B. Human motor unit recruitment during isometric contractions and repeated dynamic movements. *J. Neurophysiol.* 57: 311-324, 1987

Thomas, C. K., Ross, B. H. and Stein, R. B. Motor-unit recruitment in the human first dorsal interosseous muscle for static contractions in three different directions. *J. Neurophysiol.* 55: 1017-1029, 1986

Tiidus, P. and Ianuzzo, C. D. Effects of intensity and duration of muscular exercise on delayed soreness and serum enzyme activities. *Med. Sci. Sports Exerc.*. 15: 461-465, 1983

Toft, E., Sinkjaer, T., Andreassen, S. and Larsen, K. Mechanical and electromyographic responses to stretch of human ankle extensors. *J. Neurophysiol.* 65: 1402-1410, 1991

Tyler, A. E. and Hutton, R. S. Was Sherrington right about co-contractions? *Brain Res.* 370: 171-175, 1986

Van Atteveldt, H. and Crowe, A. Active tension changes in frog skeletal muscle during and after mechanical extension. *J. Biomech.* 13: 323-331, 1980

Vaughn, V. Effects of upper limb immobilization on isometric muscle strength, movement time, and triphasic electromyographic characteristics. *Phys. Ther.* 69: 119-129, 1989

Wadman, W. J., Denier van der Gon, J. J. and Derksen, R. J. A. Muscle activation patterns for fast goal-directed arm movements. *J. Human Mov. Studies.* 6: 19-37, 1980

Warren, G. L., Hayes, D. A., Lowe, D. A., and Armstrong, R. B. Mechanical factors in the initiation of eccentric contraction-induced injury in rat soleus muscle. *J. Physiol.* 464: 457-475, 1993c

Warren, G. L., Hayes, D. A., Lowe, D. A., Karwoski, C. J., Prior, B. M. and Armstrong, R. B. Excitation failure in eccentric contraction-induced injury of mouse soleus muscle. *J. Physiol.* 468: 487-499, 1993a

Warren, G. L., Hayes, D. A., Lowe, D. A., Prior, B. M. and Armstrong, R. B. Materials fatigue initiates eccentric contraction-induced injury in rat soleus muscle. *J. Physiol.* 464: 477-489, 1993b

Warren, G. L., Hayes, D. A., Lowe, D. A., Williams, J. H. and Armstrong, R. B. Eccentric contraction-induced injury in normal and hindlimb-suspended mouse soleus and EDL muscles. *J. Appl. Physiol.* 77(3): 1421-1430, 1994

Watts, R. L., Wiegner, A. W. and Young, R. R. Elastic properties of muscles measured at the elbow in man: patients with parkinsonian rigidity. *J. Neurol. Neurosurg. Psychiatry.* 49: 1177-1181, 1986

Weiss, P. L., Kearney, R. E. and Hunter, I. W. Position dependence of ankle joint dynamics: passive mechanics. *J. Biomech.*, 19: 727-735, 1986

Westing, S. H., Seger, J. Y. and Thorstensson, A. Effects of electrical stimulation on eccentric and concentric torque-velocity relationships during knee extension in man. *Acta Physiol. Scand.* 140: 17-22, 1990

Wierzbicka, M. M. and Wiegner, A. W. Effects of weak antagonist on fast elbow flexion movements in man. *Exp. Brain Res.* 91: 509-519, 1992

Wilcox, A., Climstein, M., Quinn, C. and Lawson, L. The effects of delayed onset muscular soreness (DOMS) on running economy. *Med. Sci. Sport Exerc.* 21 (2): S535, 1989

Wood, S. A., Morgan, D. L. and Proske, U. Effects of repeated eccentric contractions on structure and mechanical properties of toad sartorius muscle. *Am. J. Physiol.* 265: C792-C800, 1993

Wretling, M. L., Gerdle, B. and Henriksson-Larsén, K. EMG: a non-invasive method for determination of fiber type. *Acta Physiol. Scand.* 131: 627-628, 1987

Zijdewind, I. and Kernell, D. Index finger position and force of the human first dorsal interosseous and its ulnar nerve antagonist. *J. Appl. Physiol.* 77(2): 987-997, 1994a

Zijdewind, I. and Kernell, D. Fatigue associated EMG behavior of the first dorsal interosseus and adductor pollicis muscles in different groups of subjects. *Muscle Nerve* 17:1044-1054, 1994b

APPENDIX A

Ethics Approval and Informed Consent

SIMON FRASER UNIVERSITY

VICE-PRESIDENT, RESEARCH

BURNABY, BRITISH COLUMBIA
CANADA V5A 1S6
Telephone: (604) 291-4152
FAX: (604) 291-4860

June 28, 1995

Mr. Andrew Leger
Graduate Student
Kinesiology
Simon Fraser University

Dear Mr. Leger:

Re: **The Mechanical and Neurophysiological Changes During Recovery from Eccentric Exercise**

I am pleased to inform you that the above referenced Request for Ethical Approval of Research has been approved on behalf of the University Research Ethics Review Committee. This approval is in effect for twenty-four months from the above date. Any changes in the procedures affecting interaction with human subjects should be reported to the University Research Ethics Review Committee. Significant changes will require the submission of a revised Request for Ethical Approval of Research. This approval is in effect only while you are a registered SFU student.

Best wishes for success in this research.

Sincerely,

Bruce P. Clayman, Chair
University Research Ethics Review
Committee

c: T.E. Milner, Supervisor
J. Hoffer, Director

BR/hme

SIMON FRASER UNIVERSITY

INFORMATION SHEET FOR SUBJECTS

The Mechanical and Neurophysiological Changes During Recovery From Eccentric Exercise

Experiment 1

The study that you have agreed to participate in involves the investigation of the changes that occur in muscle after a regime of eccentric exercise. As a participant you will be required to be available for testing on two days within a period of one week. Testing on each of the days will require approximately two hours.

On the first day of testing you will be required to perform an exercise protocol involving maximal effort. The specific exercise will be to contract the abductor muscle of the index finder (first dorsal interosseous) to produce a maximal isometric contraction. Superimposed on this maximal contraction will be a slow stretch of approximately 25° provided by a computer controlled manipulandum. This stretch will occur over a period of 3 seconds, while you continue to maintain a maximal effort. You will be provided with visual feedback of your effort. The total exercise session consists of 100 repeated contractions. These will be performed in two sets of 50 contractions with rest in between. A protocol of this design will cause some feelings of immediate fatigue after the exercise. In addition, you may experience some feelings of discomfort in the exercised muscle over the next few days. This discomfort will be similar to those feelings you may experience after performing a novel exercise for the first time. These feelings should persist for no longer than a week (usually two to three days) and will have no permanent effects.

Prior to, and the day following the exercise session a variety of tests will be performed to study the changes that occur in the exercised finger muscle. These tests will include measures of joint stiffness, range of motion, strength, movement and control of force capabilities on the manipulandum. During these tests you may be asked to relax completely or to produce resistance to an offset torque provided by the manipulandum. Superimposed upon this may be a quick stretch of 5^0 provided by the manipulandum. One test will involve the use of a muscle stimulator and surface electrodes to produce a muscle contraction in the index finger muscle. The intensity of the stimulation will be set to produce a mild contraction equal to less than 20% maximum voluntary contraction. This may involve some irritation of the skin during the brief period of stimulation.

Testing will require application of surface electromyographic (EMG) electrodes and surface stimulating electrodes to the skin of the hand and forearm. To allow repeated placement of these electrodes you will be asked to shave the hair from your forearm in specific locations and indelible ink will be used to mark your skin.

You will be secured to the manipulandum with Thermoplast splinting material. All torques and stretches provided by the manipulandum will be computer controlled. Safety stops will be in place to ensure that joint movement does not go beyond normal range of motion.

SIMON FRASER UNIVERSITY

INFORMATION SHEET FOR SUBJECTS

The Mechanical and Neurophysiological Changes During Recovery From Eccentric Exercise

Experiment 2

The study that you have agreed to participate in involves the investigation of the changes that occur in muscles after a regime of eccentric exercise. As a participant you will be required to be available for testing on five days during a period of one week. Testing on each of the days will require approximately two hours.

On the first day of testing you will be required to perform an exercise protocol involving maximal effort. The specific exercise regime will involve lowering a weight, which is attached to the hand, while activating the wrist extensor muscles. The weight will be equal to 80% of maximal isometric wrist extensor capability. Once lowered the weight will be returned to the starting position by the experimenter. You will be required to continue lowering the weight until you are unable to do so in a controlled manner. This requires typically 50 repetitions. A protocol of this design will cause some feelings of immediate fatigue after the exercise. In addition, you may experience some feelings of discomfort and strength loss in the wrist extensor muscles over the next few days. This discomfort will be similar to those feelings you may experience after performing a novel exercise for the first time. These feelings should persist for no longer than a week (usually three to four days) and will have no permanent effects.

Prior to, and for four subsequent days following the exercise session a variety of tests will be performed to study the wrist extensor muscles and the wrist flexor muscles. These tests will include measures of joint stiffness, range of motion, strength, movement and control of force capabilities on the manipulandum. During these tests you may be asked to relax completely or to produce resistance to an offset torque provided by the manipulandum. Superimposed upon this may be a quick stretch of 3^{o} provided by the manipulandum.

Testing will require application of surface electromyographic (EMG) electrodes and surface stimulating electrodes to the skin of the hand and forearm. To allow repeated placement of these electrodes you will be asked to shave the hair from your forearm in specific locations and indelible ink will be used to mark your skin.

You will be secured to the manipulandum with Thermoplast splinting material. All torques and stretches provided by the manipulandum will be computer controlled. Safety stops will be in place to ensure that joint movement does not go beyond normal range of motion

SIMON FRASER UNIVERSITY

The Mechanical and Neurophysiological Changes During Recovery From Eccentric Exercise

SAFETY PRECAUTIONS

The nature of this research involves repetitive eccentric exercise which can cause feelings of discomfort for days following the exercise. This muscle soreness is temporary and will dissipate within a weeks time. There is no permanent muscle injury that is associated with this type of exercise. Subjects can discontinue participation at any time during the exercise regime or during testing. A qualified physiotherapist will be present during all phases of the study to address any questions or concerns of a musculoskeletal nature.

Subjects will be immobilized and secured to the manipulandum with Thermoplast splinting material. All offset torques and changes in position on the manipulandum will be computer controlled. To ensure that no harm can come from the manipulandum mechanical safety stops will be in place to limit movement to normal range of motion.

A muscle stimulator will be used to cause a mild contraction in the first dorsal interosseous muscle (FDI). Surface electrodes will be applied to the skin over the FDI muscle. A mild contraction will be produced with a battery operated (1.5V) muscle stimulator (Mikrofes). Subjects will be in control of an on/off switch and the stimulation amplitude.

Surface electromyography (EMG) electrodes will be applied to the skin. The preamplifier in these electrodes is powered by a 12 V low current power supply. In the unlikely event of a large power transient to the electrodes the preamplifier would be damaged causing it to open circuit and no further current could pass and thus pose no threat of injury to the subject. If for any reason the circuit drew too much current for an extended period the electrode would heat up. In this case the power supply would immediately be turned off.

All equipment is connected to an isolation transformer to eliminate the effects of a power surge from the line source.

SIMON FRASER UNIVERSITY

INFORMED CONSENT BY SUBJECTS

The Mechanical and Neurophysiological Changes During Recovery From Eccentric Exercise

Having been asked by Andy Leger of the School of Kinesiology of Simon Fraser University to participate in a research project experiment, I have read the procedures specified in the document "Information Sheet for Subjects".

I do not suffer from any cardiovascular disorders, musculoskeletal disorders, not presently being treated by a physician or on any prescription medication and to the best of my knowledge I am fit and healthy.

I understand the procedures to be used in this experiment and the personal risks to me in taking part.

As a subject confidentiality of my identity will be ensured at all times and at no point during the study or presentation of results will my identity be revealed.

I understand that I may withdraw my participation in this experiment at any time.

I also understand that I may register any complaint I might have about the experiment with the chief researcher named above or with Dr. J.A. Hoffer, Director of the School of Kinesiology, Simon Fraser University.

Copies of the results of this study, upon its completion, may be obtained by contacting: Andy Leger or Dr. T. Milner, School of Kinesiology.

I agree to participate by performing a series of tests on the left hand or wrist for five subsequent days if necessary, before and following a protocol requiring repetitive, high intensity, eccentric exercise. This has been described in the document referred to above.

NAME (please print): _____

ADDRESS: _____

_____

SIGNATURE: _____

WITNESS: _____

DATE: _____

APPENDIX B

Muscle Soreness Rating

DESCRIPTION	SCORE
NO SORENESS	0
DULL FEELING OF SORENESS	1
LIGHT CONTINOUS SORENESS	2
MORE THAN LIGHT SORENESS	3
ANNOYING SORENESS	4
SEVERE SORENESS	5
INTOLERABLE SORENESS	6

*score in half points if desired

Procedures:
I. Score while pressing over muscle.

II. Score while stretching muscle.

TOTAL : Combined score out of 12