Edith Cowan University
Research Online

Theses: Doctorates and Masters | Theses

2003

The effect of eccentric exercise velocity on selected measures of muscle function and soreness of the Human elbow flexors in untrained males and females

Dale W. Chapman
Edith Cowan University

Follow this and additional works at: https://ro.ecu.edu.au/theses

 Part of the Sports Sciences Commons

Recommended Citation

Chapman, D. W. (2003). *The effect of eccentric exercise velocity on selected measures of muscle function and soreness of the Human elbow flexors in untrained males and females.* Edith Cowan University. Retrieved from https://ro.ecu.edu.au/theses/1485

This Thesis is posted at Research Online.
https://ro.ecu.edu.au/theses/1485

Edith Cowan University

Copyright Warning

You may print or download ONE copy of this document for the purpose of your own research or study.

The University does not authorize you to copy, communicate or otherwise make available electronically to any other person any copyright material contained on this site.

You are reminded of the following:

- Copyright owners are entitled to take legal action against persons who infringe their copyright.

- A reproduction of material that is protected by copyright may be a copyright infringement. Where the reproduction of such material is done without attribution of authorship, with false attribution of authorship or the authorship is treated in a derogatory manner, this may be a breach of the author's moral rights contained in Part IX of the Copyright Act 1968 (Cth).

- Courts have the power to impose a wide range of civil and criminal sanctions for infringement of copyright, infringement of moral rights and other offences under the Copyright Act 1968 (Cth). Higher penalties may apply, and higher damages may be awarded, for offences and infringements involving the conversion of material into digital or electronic form.

USE OF THESIS

The Use of Thesis statement is not included in this version of the thesis.

The Effect of Eccentric Exercise Velocity on Selected Measures of Muscle Function and Soreness of the Human Elbow Flexors in Untrained Males and Females

By

Dale Chapman B.Sc. (Sports Science)

This thesis is presented for the award of Masters of Science (Sports Science) from the School of Biomedical and Sport Science, Faculty of Computing, Health and Science, Edith Cowan University, Perth, Western Australia.

Date of Submission

3rd of November 2003

ABSTRACT

Eccentric contractions, where a muscle is repeatedly lengthened while generating torque, result in decreased muscle function and muscle soreness. This study was designed to determine whether there was a difference in muscle response of the elbow flexors from untrained subjects (n = 12) between a bout of high intensity eccentric exercise at $30°\cdot s^{-1}$ (LVE) compared to the equivalent at $210°\cdot s^{-1}$ (HVE). Subjects performed 120 seconds of eccentric exercise of the elbow flexors using a Cybex 6000 Isokinetic Dynamometer. At $30°\cdot s^{-1}$, a total of 30 repetitions were required whilst at $210°\cdot s^{-1}$, 210 contractions were performed (at a 1:7 work/rest ratio).

Both exercise bouts resulted in significant decrements in isometric and dynamic strength measures ($p<0.01$) with HVE resulting in significantly greater reductions and a longer recovery compared to LVE. HVE also showed larger ($p<0.05$) increases in serum CK than LVE and the time taken to return to baseline levels was longer. LVE had significantly ($p<0.05$) smaller changes in the circumference (CIR) of the upper arm as compared to HVE, mean peak increase in CIR after LVE was 0·4 cm (± 0·1) and following HVE it was 0·8 cm (± 0·1) (SEM). Significant ($p<0.05$) levels of palpated, flexed and extended muscle soreness were experienced following both exercise conditions and the recovery time was extended for HVE. The two exercise conditions resulted in significant ($p<0.05$) reductions in subjects' ROM (LVE = 12° ± 4 and HVE = 23° ± 8) and relaxed arm angle (RANG) (LVE = 4° ± 1 and HVE = 11° ± 2) (mean ± SEM). Significant ($p<0.05$) differences were observed between groups and normal function had returned 168 hours following exercise for ROM and RANG.

The most likely explanation for the findings is that a greater mechano-chemical strain is placed on fewer fibres in HVE as compared to LVE, despite similar peak torques. The site for where this increased strain occurs is not easily definable, but may possibly be the contractile protein titin or desmin or contractile structures such as costameres or sarcomeres.

DECLARATION

I certify that this thesis does not, to the best of my knowledge and belief:

(i) incorporate without acknowledgement any material previously submitted for a degree or diploma in any institution of higher education;

(ii) contain any material previously published or written by another person except due reference is made in the text; or.

(iii) contain any defamatory material.

Signature

Date 30/1/04

ACKNOWLEDGEMENTS

I would like to express my utmost gratitude firstly to Dr Paul Sacco and Mike Newton for unwittingly providing me with the inspiration and determination, to not only complete this research but also hopefully take my studies to the next level. You both gave your time freely and were available whenever I needed to ask a question or bounce an idea off you. My thesis submission would not have been possible within the time constraints, if either of you had not been as prompt with my work as you were.

To my fellow post-graduate students over the last two years, thank you very much for your support and ensuring that sanity was maintained within the room 19.127, although others did past through from time to time. Special mention to Carmel Nottle and Nadia Vrdojak for the light-hearted moments during nights spent in front of the AMLAB computer or for thoughtfully providing me with afternoon snacks and a staple diet of m & m's.

The research could not have been completed without the subjects and to this select group of people, thankyou all so very, very much. I know it was not pleasant (I have been a subject in other damage studies) and so your pain and discomfort (although I was not exactly compassionate at the time) was really appreciated.

Also I would like to thank the School of Biomedical and Sport Sciences in general for all of the support and assistance provided especially the financial assistance in attending an international conference and the hours of tutoring in the undergraduate program.

Finally and by no means in the least to my family and friends, this would not have been possible without you all. To my parents, thank you for all of the support that you have given me, both during my under-graduate studies and now for the start of my post-graduate. To my friends, thankyou for reminding me that no matter how many pieces of paper you have it is your friends and a casual beer that keeps us all on the ground.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE

1 INTRODUCTION

1.1 Background

Movement of a limb occurs when there is an imbalance in the torque acting across it. The imbalance may result from internal sources such as that developed by muscles crossing a joint e.g., the elbow flexors and extensors or externally by loads generated by objects and surfaces around the joint e.g., a hand holding a weight. The terminology of respective muscle actions that instigate movement is based on the length of the muscle tendon unit. If the resulting internal torque overcomes any counter or opposing torques then the joint will move with the muscle shortening, described as a concentric contraction. Conversely, if the internal torque applied by the active muscle is less than the opposing torques then controlled movement still occurs, but the muscle will lengthen, identified as an eccentric contraction. When internal torque and external torques balance, no change in joint position occurs and the muscle essentially maintains a constant length, termed an isometric contraction.

Eccentric contractions are critical to the movements of all land-based animals and generally occur under two circumstances. Firstly, where the active muscle undergoes a pre-stretch followed immediately by a concentric contraction. This is commonly referred to as the stretch-shortening cycle (e.g., when the quadriceps vastus actively lengthens immediately prior to shortening in a counter movement vertical jump). Condition two occurs when the activated muscle is lengthened under tension to allow controlled movement around the joint. This action is found when lowering weight, be it an animal's mass during locomotion or a load used in resistance training. These movements, when repeated numerous times or with great force, and when the exercise is novel or unaccustomed can lead to impaired muscle function, muscle

soreness and tenderness. This phenomenon has been termed exercise-induced muscle damage (EIMD) (Clarkson, 1992). Previous investigations into EIMD using human muscles have quantified the extent of the muscle injury elicited by eccentric exercise protocols using indirect measures of muscle function and delayed onset muscle soreness (DOMS) (Nosaka & Newton, 2002b; Nosaka, Sakamoto, Newton, & Sacco, 2001).

The relationship between eccentric torque/force generation and contraction velocity has led to the proposal that the velocity of contraction is a determinant of the extent of EIMD. McCully and Faulkner (1986) were the first to investigate this idea when they investigated injury to skeletal muscle resulting from eccentric contractions in situ using three contraction velocities. They demonstrated that force decrease post intervention was related to increased stretch velocity in conjunction with the duration of the applied stimulation. Warren, Hayes, Lowe, and Armstrong (1993) described similar findings, namely that decrements in peak isometric force post exercise were closely related to the peak forces developed during the eccentric exercise protocol. In other investigations into the effect of contraction velocity, Lynch and Faulkner (1998) and Brooks and Faulkner (2001), using single stretches of mouse muscle, found no significant relationship between contraction velocity and injury. Based on the available evidence, the effect of velocity of stretch on contraction-induced force deficits seems to favour a higher decline in performance with higher velocities of stretch. To date, the effect of velocity of stretch has not been tested on the intact human muscle tendon unit.

1.2 Purpose of the Study

Therefore, the aim of the present study was to compare the effects of two different movement velocities on EIMD resulting from eccentric contractions of the human elbow flexors in untrained subjects. A randomised crossover design was used to determine whether subjects differed in their responses between a bout of eccentric elbow flexor exercise at a velocity of $30°\cdot s^{-1}$ in one arm compared to $210°\cdot s^{-1}$ in the other arm when time under tension was comparable. The dependent variables (criterion measures) consisted of muscle strength, joint range of movement, arm circumference, plasma creatine kinase, surface electromyography and soreness.

1.3 Significance of the Study

Isolated muscle investigations have not conclusively resolved the effect of contraction velocity on the extent of the induced muscle injury. Protocols employed to elicit muscle damage in intact humans have used only a single angular velocity. When compared to human studies, animal models facilitate control of factors such as totality of muscle contraction (voluntary vs stimulated) and fatigue. These models allow for easy investigation of muscle architecture post-exercise but may not be representative of real life events where maximal voluntary contractions (MVC's) are performed. Difficulties arise in extrapolating the results of animal muscle studies due to the use of stretch lengths and velocities, which fall outside human physiological ranges and relative force productions above that which can be achieved voluntarily. The exercise protocol employed for this study has been able to mimic to a minor degree the stretch shortening cycle and may assist in programming considerations such as when and where to place high angular loading exercises in an exercise program. The use of a human elbow flexor model, together with adequate recovery periods, allows for the facilitation of voluntary vs stimulated and fatigue while still employing a movement reflective of what occurs in 'real life' situations. If there were differences in the extent of EIMD incurred and/or a difference in the time course of recovery from low velocity exercise compared to high velocity exercise, the outcomes would be valuable in optimising resistance training programs, which minimise soft tissue injury.

1.4 Research Question

Would there be a difference in criterion measures caused by high velocity eccentric muscle contractions compared to those performed at a lower velocity when time under tension is kept constant?

1.5 Definition of Terms and Abbreviations

ANOVA	Analysis of variance
ATP	Adenosine triphosphate
CIR	Arm circumference

CK	Creatine Kinase – an intra-muscular enzyme released into the blood due to cell wall breakdown. Units are expressed as international units (IU)
Concentric contraction	Actions where an activated muscle produces torque while shortening
Eccentric contraction	Actions where an activated muscle produces torque while lengthening
EMG	Electromyography
EIMD	Exercise-induced muscle damage
FANG	Flexed arm angle
Isometric contraction	Torque production of a muscle associated with no overall change in its gross length
Isokinetic dynamometer	A device used to measure the torque output of isokinetic muscle contractions at any number of velocities
MVC	Maximal voluntary contraction, subject attempts to maximally activate the muscle
RANG	Relaxed arm angle
ROM	Range of motion of a joint
Sarcomere	Smallest functional unit of a muscle fibre
SR	Sarcoplasmic reticulum - An arrangement of membranous vesicles and tubules, located in the cytoplasm of striated muscle
Torque	A measure of angular force. Units are expressed in Newton meters (N·m)
Z line	Defining line of a Sarcomere and the attachment point for the thin filaments

CHAPTER TWO

2 LITERATURE REVIEW

2.1 Introduction

Muscles are able to generate force in three different manners: static, shortening and lengthening of the muscle tendon unit. Static force production is termed an isometric contraction, where the muscle acts against an immoveable resistance causing negligible change in length of the muscle-tendon unit. Shortening of the muscle tendon unit, which can be isotonic (fixed load, changing angular velocity) or isokinetic (fixed angular velocity changing load), is known as a concentric contraction. Finally, muscles may provide an active resistance against an opposing load during a stretching of the muscle tendon unit to a lengthened position. This type of action, known as an eccentric contraction may also be isotonic or isokinetic in nature (Kannus, 1994).

2.2 Torque-Velocity Relationship

The relationship between force generation and the velocity of contraction was first described by Fick in 1882 and was refined by Hill in 1938 (Lindstedt, LaStayo, & Reich, 2001). The theoretical construct presented by Hill was derived from calculations of energy expenditure and proposed that the relationship between force and speed is hyperbolic when the muscle tendon unit is shortening. This relationship was further explored by Katz (1939) and extended to include the effect of increasing force above isometric values. These associations have become known as the force-velocity relationship and generally take the form illustrated in figure 1.

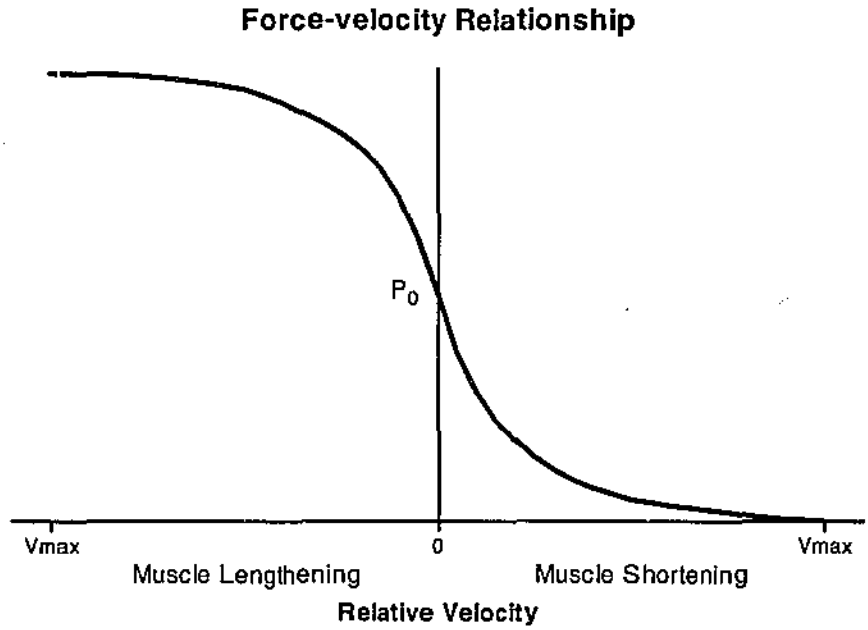


Figure 1 A representation of the theoretical force-velocity relationship in eccentric (lengthening) and concentric (shortening) contractions depicted to the relative maximum velocity of muscle lengthening and shortening (Vmax). Adapted from Allen (2001).

The theoretical relationship described by figure 1 has been validated in isolated muscle studies, in which fibres are stimulated maximally in vitro (Allaf, Goubel, & Marini, 2002; Brooks & Faulkner, 1994). The term force-velocity is applicable to muscle contractions occurring with linear velocity, in contrast when muscle contraction occurs in a limb, about an axis of rotation, which has an angular velocity component it is termed a torque-velocity relationship. Investigations into the torque-velocity relationship in human limbs during different contraction velocities have yielded less clear results. In whole limbs the level of torque generated is dependent on a number of factors: the type of action performed, the velocity of contraction and whether the contraction is voluntary or stimulated (Westing, Seger, & Thorstensson, 1990). The concentric torque-velocity relationship of human limbs using a range of muscle groups, follows the force-velocity relationship demonstrated in vitro using animal models (Caldwell, Adams, & Whetstone, 1993; Gregor, Edgerton, Perrine, Campion, & DeBus, 1979). Debate exists regarding whether the eccentric torque-velocity relationship in human limbs during high velocity maximal voluntary contractions, corresponds to the force-velocity relationship of in-vitro isolated fibres

(i.e. significant increases), plateaus, or decreases (Griffin, 1987; Gulch, 1994; de Ruiter & de Haan, 2001). Imposing an electrical stimulation upon a muscle group already performing a supposedly maximal voluntary eccentric contraction demonstrates that eccentric torque output can sometimes be increased further and can be more representative of the theoretical torque-velocity relationship (Westing et al., 1990). Dudley, Harris, Duvoisin et al. (1990) and Westing et al. (1990), suggested that peak eccentric torque was effected by the ability of the central nervous system to fully activate the muscle and that this inhibition was acting as some form of protective mechanism against extreme muscle tension. Webber and Kriellaars (1997) predicted the maximum eccentric torque following a graded test in which a percentage of the maximal voluntary contraction was reached prior to a stretch being applied. Extrapolating this data it was predicted that the peak maximal eccentric torque for the knee extensors was 151% of maximal voluntary isometric torque, which is consistent with the in-vitro force-velocity relationship. These authors postulated that the predicted increase in eccentric torque production being significantly greater than the actual torque values suggests that a neural regulatory mechanism restricts the recruitment and/or discharge of motor units during this type of contraction.

Force production in skeletal muscle involves both the mechanical and biochemical processes referred to as cross bridge cycling. An active cross bridge is the combination of the S2 myosin neck and the S1 myosin head. An attached cross bridge is formed by the interaction of myosin S1 heads on the thick filament and actin molecules of the thin filament (Gordon, Regnier, & Homsher, 2001). Cross bridge cycling was presented as a two stage process by Huxley in 1957 and relies upon the concept of sliding filaments (Pollack, 1983). It was proposed that cross bridges cycle between two functional conditions: a force producing condition strongly attached with actin and a non-force producing condition where cross bridges are detached from actin (Sieck & Regnier, 2001). There are two main theories for the process of force development, both of which assume that the myosin S1 head has a degree of elasticity. Figure 2 indicates that the myosin S1 head stretches and attaches to the actin site (cross bridge formed). After attachment S1 recoils to a starting length, detaches and repeats the steps (causing the filaments to slide past one another). The alternative explanation (figure 3) still allows for a level of elasticity in

the myosin head but differs in that S1 does not stretch prior to attachment. Instead, at the point of attachment, a change in the angle between the head and filament axis (rotation) drives filament sliding and force production (Irving & Piazzesi, 1997). In both models the force exerted by the myosin head on the actin site causes the filaments to slide past each other, the sarcomere to shorten, and the muscle to develop force.

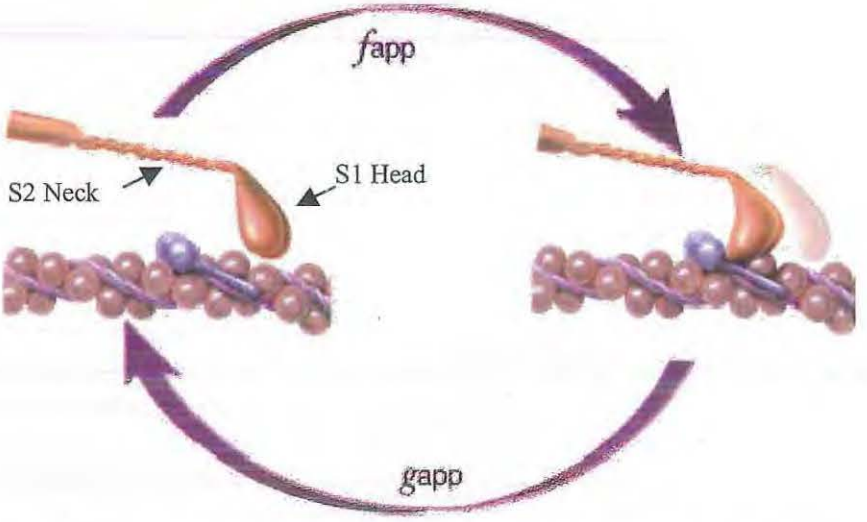


Figure 2 Force generation and contraction results from cross bridges cycling functional states: 1) strongly bound and 2) unbound. f_{app}, rate constant for cross bridge attachment; g_{app}, rate constant for cross bridge detachment. Adapted from Sieck and Regnier (2001).

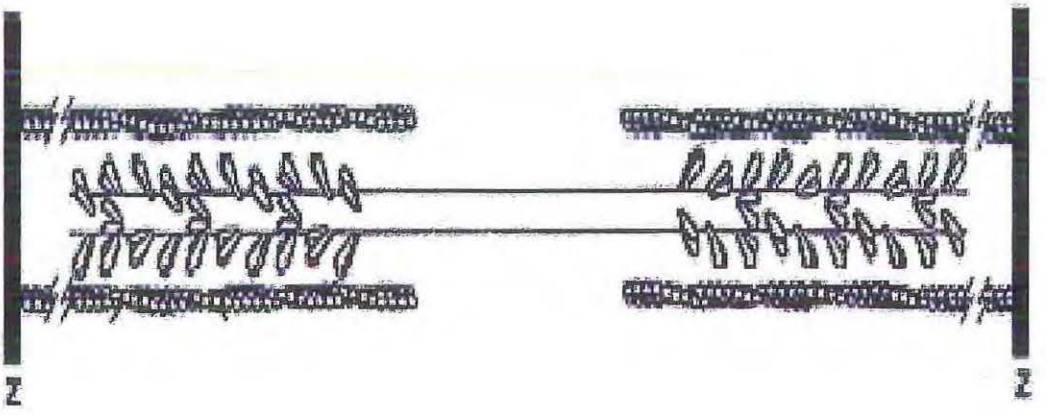


Figure 3 Sarcomere showing the overlap of thin and thick filaments. A subtle rotation of the thick filament in the opposite direction as the helix rotation of the thin filament causes the filaments to slide past one another. Reproduced from Gordon, Regnier and Homsher (2001)

The interaction of the myofilaments to produce force is regulated by calcium (Ca^{2+}) concentration and the availability of ATP hydrolysis at the site of binding between actin and myosin (Irving & Piazzesi, 1997). It is likely that force production via Ca^{2+} release from the sarcoplasmic reticulum (SR) to the sarcoplasm is the dominant regulator and controls steady-state force generation in shortening of the muscle tendon unit (Gordon et al., 2001). Ca^{2+} release from the SR is triggered by an action potential passing down through the T tubule structure. After myosin binding and either S1 recoil or actin rotation, an ATP molecule is attached to the myosin head at the active site after which detachment from actin occurs. For the muscle fibre to relax, the ATP dependent calcium pump removes Ca^{2+} from the intracellular space and returns it to the SR.

The processes outlined above describe the sequence of force production in concentric and isometric contractions, however eccentric contractions may involve different methods to generate tension. The exact mechanism(s) responsible for the greater tension developed during eccentric contraction as compared to isometric, is/are yet to be defined. However, it is generally accepted that cross bridge detachment during stretching occurs through a mechanical process as indicated by the fact that a fall in metabolic heat production occurs, which is a measure of the ATP turnover. The increased tension developed during muscle stretch can be explained using the Huxley model, where by during the stretch the compliant portions of the cross bridges are stretched further than is normally the case in isometric contractions. The sliding filament model predicts the tendency towards a plateau of force at higher velocities, whereby with the increase in stretch velocity fewer cross bridges will be attached but of those that continue to be attached they will sustain greater force. Herzog and Leonard (2002) postulated that the force enhancement observed in actively stretched muscle could be due to a passive structural element. A hypothesised site for such a series elastic element is the large protein titin (Lindstedt et al., 2001). The role which may be to assist in resisting the external load by maintaining sarcomere alignment, and may have an influence on the initiation of cellular signalling to enhance cross bridge recruitment while decreasing ATP metabolic cost (Lindstedt et al., 2001).

2.3 Effect of Contraction Velocity on EIMD

Investigations of the role of contraction velocity on EIMD have employed isolated muscle models. Several studies have examined the effect of velocity of stretch on the loss of muscle function and have used single stretches of muscle fibres, repeated stretches of whole muscle. The conclusions drawn by researchers on the importance of contraction velocity on skeletal muscle injury have varied. McCully and Faulkner (1986) studied injury to skeletal muscle resulting from a maximal stimulation followed by an eccentric contraction at one of three velocities (0·2, 0·5 and 1·0 lengths of fibre per second [$Lf \cdot s^{-1}$]) in situ. Significant decreases in isometric force 3 days post exercise were associated with increases in stretch velocity, which the authors explained by the increased peak forces at higher velocities. Warren, Hayes, Lowe and Armstrong (1993) observed similar responses using slightly higher stretch velocities (0·5, 1·0 and 1·5 $Lf \cdot s^{-1}$) in rat soleus muscle in vitro, (i.e. that decrements in peak isometric force post exercise were closely related to the peak forces developed during the eccentric exercise protocol). However, the researchers noted that greater initial declines in measures of muscle function were observed at higher velocities of lengthening independent of peak force produced.

Using two stretch velocities (3·0 and 4·0 $Lf \cdot s^{-1}$) Talbot and Morgan (1998) found a weak correlation between velocity and changes in muscle function in toad sartorius muscle studied in vitro. Stronger correlations were found between the initial length, the number of contractions and the amplitude of stretch with the measured changes in muscle function as a result of this research. There have been two later studies employing single stretches of extensor digitorum longus muscles but using different muscle preparations. The first used permeabilized fibre segments and five stretch velocities (0·5, 1·0, 2·0, 3·0 and 4·0 $Lf \cdot s^{-1}$) (Lynch & Faulkner, 1998), and the second investigated five stretch velocities (1·0, 2·0, 4·0, 8·0 and 16·0 $Lf \cdot s^{-1}$) in situ (Brooks & Faulkner, 2001). Lynch and Faulkner (1998) demonstrated no relationship between velocity and the severity of the injury to the fibres, however Brooks and Faulkner (2001) showed single stretches of whole skeletal muscle had a weak velocity effect.

Performance of rat plantar flexors in situ was investigated with a slow and fast repeated stretch ($50°\cdot s^{-1}$ and $600°\cdot s^{-1}$ respectively) imposed on a maximally active muscle (Willems & Stauber, 2000). Torque decrements were similar post intervention for both investigated velocities with the authors concluding that contraction velocity was not a critical factor in performance decrement in stimulated muscle in situ. In later work, a comparable result was reported following an investigation using rat plantar flexors in situ where torque decrements resulted from constant or increasing velocity stretches during 20Hz or 80Hz electrical stimulation of the motor nerve (Willems & Stauber, 2002). Investigators have yet to determine whether the results generated during contraction velocity work in animal muscle research can be replicated in human muscle.

2.4 Muscle Fibre type, Recruitment and Activation

Morphological studies have revealed that maximal eccentric contractions cause preferential damage to type II fibres. This has been quantified by measuring disruption to myofibre structure (Lieber & Fridén, 2002; Vijayan, Thompson, Norenberg, Fitts, & Riley, 2001), differences in fibre isometric force (Lieber, Woodburn, & Fridén, 1991; Macpherson, Schork, & Faulkner, 1996) and isokinetic torque loss (Friden, Sjostrom, & Ekblom, 1983). The mechanism underlying preferential damage to specific fibre types remains to be elucidated. Lieber, Woodburn and Fridén (1991) hypothesised that preferential fibre damage was related to fatigue and that type II fibres would be the first to succumb. The oxidative capacity of muscle fibres (metabolic cost of muscle contraction) which is reflective of the rate of cross bridge cycling was identified as central to this hypothesis. If a fibre was to suffer from fatigue the first to do so would be the type II fibres resulting in the fibre being unable to regenerate ATP, leading to a state of rigour or high resting tension. Any subsequent stretch of these stiff fibres would result in a higher mechanical stress being placed on them with the probable resultant disruption to the cytoskeleton and myofibrillar structures. Later work by Patel, Cuizon et al. (1998) demonstrated that the oxidative capacity of the fibre was not a significant factor in fibre injury when rabbit dorsiflexors were trained through isometric stimulation for a three week period. A shift in the optimum length for active tension has been used as an indicator of damage resulting from eccentric exercise (Wood, Morgan, & Proske,

1993). Brockett, Morgan, Gregory and Proske (2002) further researched this finding by investigating slow and fast motor units. It was found that various motor units had different optimum lengths for maximum tension. This research suggested that a motor unit's optimum length relative to the whole muscle might be a better indicator of a unit's vulnerability to EIMD.

The pattern of recruitment in torque production across a joint can be evaluated both macroscopically and microscopically. Macro studies focus on the order and amount that whole muscles are recruited during the production of force. Microscopically, this order of recruitment is dependent upon the fibre type and size of the motor unit involved. The size principle is the most commonly accepted theory on the order of motor unit and fibre recruitment (Cope & Pinter, 1995). It has been proposed the normal order of recruitment in eccentric contractions is reversed (Enoka, 1996), however many researchers remain opposed to such a suggestion. This hypothesis arose out of a demonstrated increase in torque generation but reduction in muscle activation, measured using surface electromyography (EMG), in eccentric contractions when compared to concentric contractions at the same velocity. Further evidence of altered recruitment has been collected following electrical stimulation during a maximal voluntary eccentric contraction in which increased torque output was recorded (Seger & Thorstensson, 2000). Alterations to the recruitment order of motor units during sub-maximal eccentric exercise has been demonstrated together with a greater resistance to fatigue during repeated eccentric contractions (Enoka, 1996).

Kasprisin and Grabiner (2000) reported that activation of the biceps brachii was joint angle dependent only during concentric isokinetic and isometric contractions, in contrast the brachioradialis is affected by elbow joint angle during eccentric contractions as measured by surface EMG signal intensity. The range of motion investigated during this study was constrictive, being only through a range of 5° to 90° of elbow flexion. Kulig, Power, Shellock and Terk (2001) examined the eccentric movement pattern of the elbow flexors during submaximal exercise. In this study an eccentric action of two seconds duration was compared to one of ten seconds and it was revealed that the biceps brachii was preferentially recruited in the fast action while the brachialis was recruited at the slower action velocity.

2.5 Mechanisms of Exercise-Induced Muscle Damage (EIMD)

EIMD is related to the physiology of force production and muscular contraction. Cross bridge cycling that leads to the shortening of sarcomeres (concentric action) occurs when external forces are smaller than the internal force produced. If the external force is greater than the internal force, then cyclic attachment still occurs but the external force causes the two sets of filaments to be pulled past each other with forced detachment of the myosin head (eccentric action) (Herzog, 2000; Morgan & Allen, 1999). Biochemical steps accompany each of these processes of force production and contraction (Gordon, Homsher, & Regnier, 2000; Herzog, 2000). As such any of these biochemical or mechanical steps are sources for the development of EIMD. Dop Bär, Reijneveld, Wokke, Jacobs and Bootsma (1997) identified two hypotheses which attempt to explain the development of EIMD. The first is a metabolic overload hypothesis, where demand for ATP exceeds its production leading to a cycle of Ca^{2+} overloading and thus a further reduction in ATP production. The second hypothesis is based on the mechanical cost of eccentric exercise, where the mechanical strain per fibre is increased due to there being fewer fibres producing the same relative level of tension. This hypothesis is supported by extensive morphological damage and large effluxes in extracellular enzymes, combined with pain, stiffness and weakness (Ebbeling & Clarkson, 1989).

EIMD has been proposed to progress through four stages of injury known as the 'Initial', 'Autogenetic', 'Phagocytic' and 'Regenerative' Phases (Armstrong, 1990). The initial stage, which includes the instigating event, is considered the trigger and may be mechanical or metabolic in origin. The autogenetic phase follows, commencing upon completion of the trigger event, and lasts for approximately four to six hours. This is the phase that leads to or exacerbates muscle necrosis via activation of several mechanisms (Dop Bär et al., 1997). The phagocytic stage is apparent from 4 – 6 hours after the event through to 2 – 4 days following the exercise and is marked by swelling of the limb and removal of necrotic tissue by activated immune cells, specifically macrophages. A regenerative phase begins approximately 4 – 6 days post exercise and spans about 10 –14 days when the injured muscles once again appear normal (Armstrong, 1990). Pyne (1994) also used this model but described only three stages, eliminating the initial stage and renaming the phagocytic

stage the 'inflammatory phase'. While it is convenient to model muscle damage on a specific timed stage process, it appears that each phase overlaps, and the exact mechanisms responsible and the processes involved are not fully understood (Kendall & Eston, 2002).

Dop Bär et al. (1997) highlighted the importance of Ca^{2+} overload caused by the initial event. The increased intracellular calcium concentration becomes apparent in the autogenetic phase and plays a major role in EIMD. This microscopic event that occurs in damaged muscle has been termed 'loss of intracellular calcium homeostasis' (McArdle & Jackson, 1997). Four mechanisms by which Ca^{2+} damages muscle have been proposed. These are 1) stimulation of calcium-activated proteases, 2) activation of lysosomal processes, 3) mitochondrial overload and 4) activation of lipolytic enzymes (Kendall & Eston, 2002; McArdle & Jackson, 1997). The loss of homeostasis, has been linked to a subsequent Ca^{2+} reduction a phenomenon termed 'excitation-contraction' uncoupling (Dop Bär et al., 1997). In a recent review, Morgan and Allen (1999) suggested that eccentric exercise may play a dual role in excitation-contraction coupling, affecting both Ca^{2+} release and uptake. The disruption of excitation-contraction coupling is linked to a reduction in the force generating capabilities of skeletal muscle (Warren, Ingalls, Lowe, & Armstrong, 2001).

Proske and Morgan (2001) proposed a model that described the cascade of events following eccentric exercise. The sequence follows the eccentric intervention and is characterised by as overstretched and disrupted sarcomeres, membrane damage, local contracture and finally death (necrosis) of the affected fibres. The decrease in muscle tension results from membrane damage, and is proposed to represent excitation-contraction uncoupling dysfunction (Proske & Morgan, 2001; Warren et al., 2001). The model proposes that sarcomere stretching occurs before excitation-contraction uncoupling (Proske & Morgan, 2001). The process by which a sarcomere is damaged is still the subject of speculation. It may involve disruption to the titin filament, anchor point of the myosin filaments to the Z discs, or interference to the structural protein desmin which is the link between adjacent Z discs (Allen, 2001; Proske & Morgan, 2001). It is generally accepted that stretching of the sarcomere leads to streaming of the Z-line (Fridén & Lieber, 1992) and the resulting non-uniformity is

termed sarcomere "popping" (Morgan, 1990). Allen (2001) postulated that the streaming of the "Z" lines is represented by a change in the force-length relationship with a shift to the right when the imposed stretch is on the descending side of the force-length curve. Other authors have postulated that Z-line disruption is associated with a reduction in force production capability (Byrd, 1992; Fridén & Lieber, 2001; Morgan & Allen, 1999).

2.6 Responses to Exercise-Induced Muscle Damage

2.6.1 Strength Measures

Previous investigators have suggested that the best non-invasive measure of the extent of the induced muscle injury appears to be the deficit in maximum isometric force (McCully & Faulkner, 1986). Maximal voluntary force deficit has been shown to occur immediately following eccentric exercise, and last for up to 10 days or longer in severe cases (Bryne, Eston, & Edwards, 2001; Nosaka, Newton, & Sacco, 2002c; Sayers & Clarkson, 2001). The loss of maximal voluntary force generating capacity has been attributed to: 1) excitation-contraction uncoupling, probably due to a functional change of the voltage sensor of the t-tubules (Warren et al., 2001), and 2) alterations to the torque producing and/or transmitting structures (Morgan & Allen, 1999). The resulting loss of force producing capacity following eccentric exercise has the potential to impact greatly on sporting performance.

2.6.2 Serum Creatine Kinase (CK)

Increases in plasma or serum creatine kinase (CK), an enzyme found in high concentration within muscle fibres, has been associated with EIMD, however large individual variations in the level of CK response from similarly exercised individuals have been recorded (Kuipers, 1994; Nosaka & Clarkson, 1996; Sayers, Clarkson, & Lee, 2000b). Plasma CK levels peak approximately 3 –5 days following eccentric exercise (Clarkson, 1997) and are believed to be due to a disruption of the muscle membrane wall that allows the protein to be released (Lee et al., 2002). CK activity in the plasma is reflective of its release from the injured muscle and its removal by the reticuloendothelial system (Clarkson, Nosaka, & Braun, 1992). Plasma CK efflux is the most commonly measured intracellular enzyme used to indicate loss of cellular

homeostasis, however, investigators have also examined the release of L-aspartate aminotransferase, lactate dehydrogenase, CK isoforms, myoglobin, heart fatty acid binding protein, carbonic anhydrase isoenzyme III, contractile and regulatory proteins and troponins (Janssen et al., 1989; Sorichter, Puschendorf, & Mair, 1999).

2.6.3 Soreness (SOR) and Tenderness

The most commonly used marker of injury to skeletal muscle is muscular soreness (Warren, Lowe, & Armstrong, 1999). Some investigators (Rodenburg, Bar, & De Boer, 1993; Stauber, Clarkson, Fritz, & Evans, 1990) have suggested that SOR of an eccentrically exercised muscle is related to the inflammatory response, however others have shown the time course of both to be temporally unrelated (Sayers, Clarkson, & Lee, 2000a). Muscle sensory receptors are polymodal, responding to both mechanical (e.g., swelling) and chemical (e.g., histamines and prostaglandin) stimuli. Histamine is released by mast cell degradation occurring with damage, and neutrophil influx can also result in prostaglandin production (Smith, 1991). The most frequent method for evaluation of SOR is a visual or numerical scale following palpation of the affected muscle (Chen & Hsieh, 2000). In a recent review Warren et al. (1999) reported that SOR has been shown to have a poor correlation to changes in muscle function both in terms of size and the time course of recovery. The discomfort experienced by the subject begins shortly after completion of the exercise and would usually peak 24 or 48 hours post-exercise and recedes by 168 hours post-exercise (Chen & Hsieh, 2001; Clarkson et al., 1992).

2.6.4 Range of Motion (ROM)

Joint ROM, defined as the size of arc that a joint is able to function through, has been shown to be affected by eccentric exercise (Warren et al., 1999). Reductions are commonly measured in the relaxed and flexed limb angle of subjects who have exercised eccentrically. This is regardless of whether the protocol involved voluntary maximal or sub-maximal contractions (endurance exercise) (Nosaka, Newton, & Sacco, 2002b). Changes in relaxed limb angle may be explained by swelling of the muscle, changes in characteristics of contracting filaments, and/or autonomous contracture (Whitehead, Morgan, Gregory, & Proske, 2003). An inability to flex the limb may be due to a change in proprioception and/or over stretched sarcomeres

(Clarkson, 1997). The time course for changes in relaxed and flexed arm angle following eccentric exercise, generally shows a nadir 2 to 4 days post-exercise but may be more protracted before returning to baseline levels (Chen & Hsieh, 2000; Nosaka & Newton, 2002a).

2.6.5 Inflammation

The mechanical disruption to muscle structure is believed to lead to the initiation of the inflammatory response. The primary role of the inflammatory cells is to remove muscle cellular debris and promote repair (Pyne, 1994). The inflammatory reaction is described as a series of six events: 1) tissue injury 2) release of vasoactive substances by the injured tissue, 3) vasodilation, 4) leucocyte adhesion, 5) leucocyte migration from the blood to the injury and 6) tissue repair (Malm, 2001). The inflammatory response following a bout of eccentric exercise has been quantified through the use of circumference measures of the exercised limb (Chen & Hsieh, 2001; Nosaka et al., 2002b) and by the measurement of intra-muscular pressure (Foley, Jayaraman, Prior, Pivarnik, & Meyer, 1999). The swelling of the muscle following exercise was suggested as an explanation for muscle soreness as oedema would be associated with an influx of extracellular proteins, causing stimulation of pain receptor (Pyne, 1994). Early work in EIMD found a correlation between the time courses of resultant inflammation and the soreness and pain experienced after eccentric exercise (Ebbeling & Clarkson, 1989). Inflammation has been linked to a response that muscle uses to adapt to subsequent bouts of eccentric exercise. Pizza and co-workers (2002) found that inflammatory cells resulting from either eccentric and isometric contractions or passive stretches, performed two weeks prior to a bout of eccentric exercise afforded some protection to the extent of EIMD.

2.6.6 Muscle Activation

The activation strategies used to generate torque by muscle during maximal voluntary concentric and eccentric contractions are believed to follow two distinct patterns. Under normal controlled movement there is an ordered recruitment of slow to fast motor units (Thayer, Rice, Pettigrew, Noble, & Taylor, 1993), although in high velocity or eccentric contractions the order may be reversed (Enoka, 1996). Changes in the root mean square EMG signal is a measure of the raw amplitude and

reflects total muscle activation, whereas alterations in the median frequency are related to changes in the type of motor unit recruited (Warren, Hermann, Ingalls, Masselli, & Armstrong, 2000). Alterations in the pattern of voluntary EMG signal during EIMD has shown that the median frequency is reduced for up to 7 days (Linnamo, Bottas, & Komi, 2000). The frequency content of the EMG signals collected from surface electrodes has been used as an indirect measure of motor unit recruitment and a higher median frequency during eccentric contractions may be explained by selective recruitment of fast twitch motor units (McHugh, Connolly, Eston, & Gleim, 2000). EMG signal amplitude has been used to quantify full activation or changes in activation of a desired muscle during and following eccentric contractions. Sbriccoli, Felici et al. (2001) demonstrated that the EMG signal amplitude remained unchanged after an eccentric exercise protocol but torque generated declined indicating a reduced neuromuscular 'efficiency'. The characteristics of the EMG signal power spectrum have also been shown to be affected by the muscle action, velocity of action and muscle length (Komi, Linnamo, Silventoinen, & Sillanpaa, 2000). This research illustrates that muscle activity can be at the same level or lower in eccentric actions as compared to concentric actions and is influenced by joint angle and level of pre-activation.

2.7 Conclusions

To date the majority of available literature has primarily investigated the role of contraction velocity using stimulated animal muscle. The research, while highlighting possible mechanical events leading to EIMD, is not believed to fully represent the factors associated with human voluntary contractions resulting in EIMD. Factors that may be implicated during voluntary contraction are the peak force developed the level of muscle activation, and the use of realistic limb contraction velocities. EIMD has been demonstrated to result from a range of single contraction velocities and in various limbs. The knowledge gained from this research would contribute to the understanding of muscle responses following voluntary contraction.

CHAPTER THREE

3 MATERIALS AND METHODS

3.1 Pilot Study

Differences in peak torque have been identified as a contributing factor in the severity of skeletal muscle injury. As the primary research focused on two isokinetic eccentric velocities it was necessary to conduct a pilot study to establish the eccentric torque-velocity relationship in a population sample possessing physical characteristics similar to those used in the primary research. The pilot study was also used to assess the reliability of the strength measures. The maximal voluntary eccentric torque-velocity relationship was used to determine whether there was a significant difference in torques produced at selected velocities of eccentric exercise.

Fourteen subjects (male n=7 and female n=7), age 26·4yrs (± 6.2); height 1·74m (± 0·07); and weight 69·3kg (± 11·5), volunteered to participate in two testing sessions, consisting of maximal eccentric and isometric contractions of the elbow flexors of their dominant arm. All subjects were physically active but were not participating in resistance training. Subjects were familiarised with the testing protocol and signed an informed consent with the understanding that they could withdraw from the study at any time without prejudice. Measurement variables included peak torque of each contraction, in Newton meters (N·m) and the angle at which the peak torque occurred.

The measurement variables of the elbow flexors were recorded using a Cybex 6000 dynamometer (Ronkonkoma, NY). Subjects were seated with their dominant arm supported at 45° of shoulder flexion on an arm curl bench (figure 4). Selected eccentric exercise velocities were 30°, 90°, 150° and 210°·s^{-1}. Velocities were tested

for all subjects in slow to fast order, with the $30°·s^{-1}$ velocity repeated upon completion of the $210°·s^{-1}$ velocity to account for any learning effects as a result of the order of tested velocities and fatigue. The range of motion for the testing commenced at 60° of elbow flexion and extended through 140° of elbow flexion, with full extension of the arm being 180°. Two maximal eccentric actions were performed at each test velocity with 60 seconds recovery between each trial and 120 seconds between the start of each test velocity. Each test velocity was preceded by two isometric maximal voluntary contractions at 90° of flexion, with 60 seconds rest between each contraction. Following the isometric contractions, subjects warmed up with 2 sub-maximal eccentric actions to gain familiarity with the test velocity.

3.2 Principle Study

Twelve subjects (male n=6 and female n=6) were recruited from volunteers within the Sports Science student population at Edith Cowan University and from associates of the researcher. Values (mean ± SD) for age, weight, and height were 27·5 ± 6·3yrs, 67·9 ± 11·0kg and 1·74 ± 0·08m respectively. All subjects were right-hand dominant and performed both exercise interventions. Recruitment was accomplished through the use of printed leaflets and posters, which were distributed around the Joondalup campus of Edith Cowan University. All subjects were required to sign and complete a combined informed consent (Appendix A) and medical questionnaire (Appendix B). The questionnaire was used to ensure that all subjects were free from any disorder or injury that may contraindicate involvement in the study. Subjects were informed that they were free to withdraw from the study at any time without prejudice. Prior to commencement of data collection ethical approval for the research was granted from the Edith Cowan University Ethics Committee (Appendix C).

3.3 Methodology

A pseudo random counter balanced design was employed ensuring that both arm dominance and velocity intervention were equally balanced across exercise intervention ($30°·s^{-1}$ = low velocity exercise and $210°·s^{-1}$ = high velocity exercise) and order of testing.

3.3.1 Criterion Measure Testing

The criterion strength measures recorded during the study were; isometric torque (90° and 150° of elbow flexion); concentric dynamic torque at $30°\cdot s^{-1}$, $90°\cdot s^{-1}$, $150°\cdot s^{-1}$ and $210°\cdot s^{-1}$ of elbow flexion, and dynamic eccentric torque at $30°\cdot s^{-1}$ and $210°\cdot s^{-1}$ of elbow flexion. Other criterion measures recorded included relaxed (RANG), stretched (SANG) and flexed (FANG) arm angles and ROM of the elbow joint, arm circumference, muscle soreness, plasma CK and the average and peak EMG signal. With the exception of CK, all of the criterion measures were collected on the exercised arm. Strength measures, RANG, SANG and FANG and arm circumference were all recorded during at least 1 familiarisation session, pre exercise, immediately post exercise, 30 minutes post exercise and 1, 2, 3, 4 and 7 days following exercise. Serum CK was determined from a blood sample drawn from the fingertip measured at least from 1 familiarisation session, pre exercise and 1, 2, 3, 4, 7 and 10 days following exercise. Palpated, flexed and extended muscle soreness was considered to be nil before exercise following which it was recorded 1, 2, 3, 4 and 7 days following exercise. Peak and average surface EMG signal of each contraction was recorded from at least 1 familiarisation session, pre exercise, immediately post exercise, 30 minutes post exercise and day 7. Each measure was recorded in the following order: serum CK, RANG, SANG and FANG, muscle soreness, and strength tests. An example of the time line used for familiarisation, baseline, exercise interventions and post-testing sessions are represented in Table 1. The individual collection methods employed for each of the criterion measures are described in detail in 3.3.1.1 – 3.3.1.7 below.

Table 1

Timeline for the recording of criterion measures and exercise sessions.

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Week 1	Familiarisation Session				Baseline measures		
Week 2	Exercise session 1 & pre and post exercise tests	Post exercise test day 1	Post exercise test day 2	Post exercise test day 3	Post exercise test day 4		
Week 3	Post exercise test day 7			Familiarisation Session	Baseline measures		
Week 4	Exercise session 2 & pre and post exercise tests	Post exercise test day 1	Post exercise test day 2	Post exercise test day 3	Post exercise test day 4		
Week 5	Post exercise test day 7						

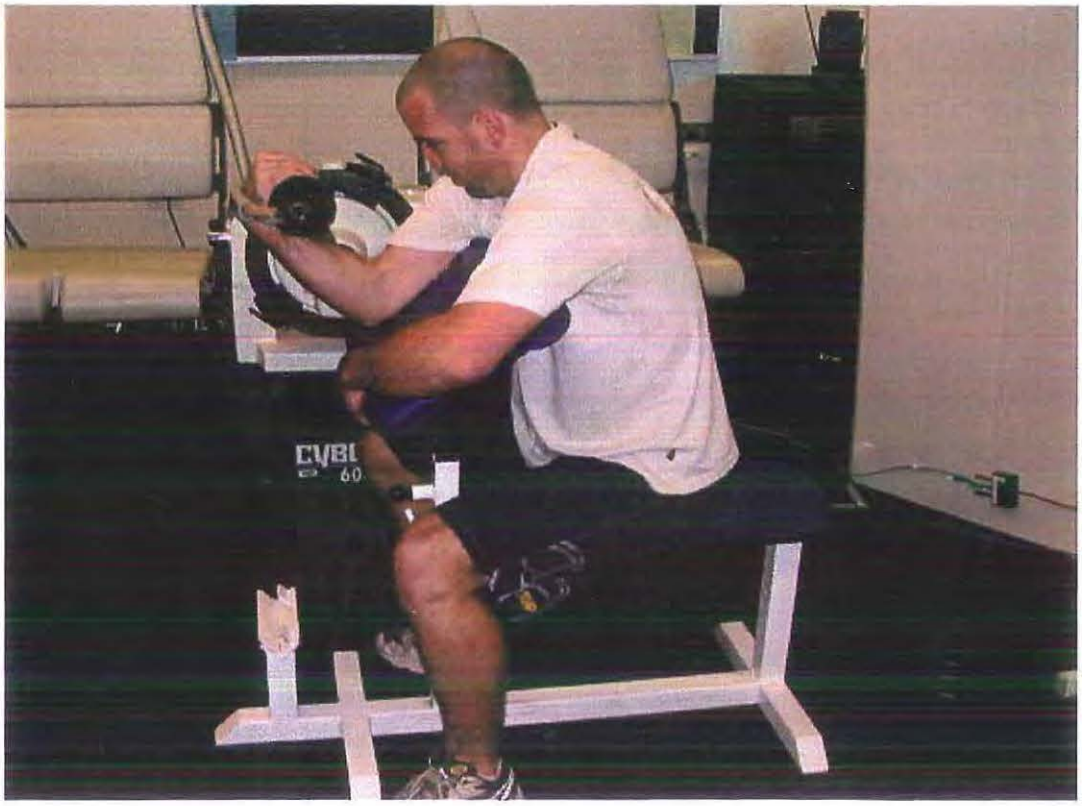


Figure 4 Example of a subject positioned on the preacher curl bench and isokinetic dynamometer with the arm at an isometric contraction angle of 90^0 and the shoulder at 45^0.

3.3.1.1 Isometric Strength

Maximal voluntary isometric torque of the elbow flexors was measured at joint angles of 90° and 150° on a Cybex 6000 isokinetic dynamometer. The subject was positioned with their arm supported at 45° of shoulder flexion on an arm curl bench (figure 4). The elbow of the tested arm was aligned with the centre of rotation of the lever arm of the dynamometer. Subjects were verbally encouraged to perform two maximal contractions, holding each contraction for 4 seconds. The subject was allowed 30 seconds of passive rest between each effort at each specific angle and 60 seconds of rest during the transition between the two joint angles. Torque was recorded and displayed in real time using an IBM desktop computer operating AMLAB II data acquisition software. The torque analogue signal was accessed from the dynamometer and sampled via a 16-bit data acquisition card (Minirack, AMLAB II) on a separate channel to the EMG signal. The signal was processed to provide a recording of the torque output as per the specific schematic (Appendix D). An average peak torque for the two contractions was used during analysis.

3.3.1.2 Dynamic Strength

Maximal voluntary torque production of the elbow flexors at specific concentric and eccentric velocities was measured on the Cybex 6000. Angular testing velocities were 30, 90, 150 and 210°·s^{-1} for the concentric protocol, and 30 and 210°·s^{-1} for the eccentric. The dynamometer set up was the same as that employed in the isometric strength evaluation (figure 4). Subjects performed two maximal voluntary contractions at each velocity in both the concentric and eccentric protocols. The order of contractions was such that all concentric contractions preceded eccentric and test velocities proceeded from slow to fast. A one-minute and a two-minute passive recovery was provided between each successive test velocity and contraction mode, respectively. The range of motion through which the subject's arm moved commenced at 60° of elbow flexion and proceeded through to 160° of elbow flexion (where full extension was considered 180°). Torque was acquired and recorded using the same set-up as that employed for isometric strength (see 3.3.1.1). Torque and angular displacement analogue signals were accessed from the dynamometer and sampled via a 16-bit data acquisition card (Minirack, AMLAB II) on two separate channels to the EMG signal. The signal from one channel was processed to provide velocity and displacement, the second signal was a recording of the torque output and a combination of the two signals as used to determine accumulated work as per the specific schematic (Appendix D). The peak torque of the two contractions at each velocity was averaged and used for analysis.

3.3.1.3 Range of Motion (ROM)

ROM measurements at the elbow were obtained using a plastic goniometer (Baseline Inc.). All measures were determined with the subject in a standing position with the arm initially relaxed by their side. Measurement of the relaxed arm angle (RANG) was taken as the angle at the elbow joint when the subject allows their arm to hang in a relaxed manner by their side. Stretched arm angle (SANG) was recorded as the angle at the elbow joint when the subject attempts to fully extend their arm. Flexed arm angle (FANG) was determined when the subject fully flexed their elbow joint in an attempt to touch their shoulder with the palm. A subject's ROM was determined by deducting FANG from SANG.

Landmarks used to measure the elbow joint angles were the lateral epicondyle of the humerus, the acromion process and the mid-point of the styloid process of the ulna and radius; these sites were marked on the skin with a semi-permanent ink marker to obtain consistent measures. The landmarks were renewed each day.

3.3.1.4 Arm Circumference (CIR)

Circumference of the upper arm was assessed using a constant tension tape (Lafayette Instrument) while the arm was relaxed and hanging by the subject's side. Measurements were taken from sites at 3, 5, 7, 9 and 11cm above the crease line of the elbow of the exercised arm. Each site was marked with a semi-permanent ink marker to obtain consistent measures and an average of the five sites was recorded as the arm circumference and used for analysis.

3.3.1.5 Soreness (SOR) and Tenderness

Muscle soreness was reported using a 100 mm visual analogue scale while the arm was forcibly flexed and extended by the investigator. The subject was instructed to place a mark on a 100 mm line for both the flexion and extension movement, rating the soreness experienced. The subject was instructed that 0 mm indicated no pain at all while 100 mm was an indication of "unbearable" pain. Soreness resulting from palpation of the upper arm and the forearm was considered muscle tenderness and reported using the same analogue scale and reporting method. The arm was palpated in four positions, utilising some of the same markings as those used for arm circumference. Palpation occurred at sites 3 – 5 cm and 7 – 9 cm above the elbow crease, and laterally on both the brachialis and brachioradialis. Nosaka, Newton and Sacco (2002a) have previously described the soreness and tenderness techniques employed. An average the four resulting measures was recorded as the elbow flexor tenderness and used in analysis.

3.3.1.6 Plasma Creatine Kinase (CK) Concentration

Plasma creatine kinase activity was determined from a 30µl sample of whole blood collected from a fingertip puncture made using a spring-loaded lancet. The sample was collected into a capillary tube and immediately pipetted onto a test strip for analysis. Creatine Kinase activity was determined using a Reflotron spectrophotometer (Boehringer-Mannheim) as previously described by McHugh et al. (1999).

3.3.1.7 Electromyography (EMG)

The electromyographic activity of the biceps brachii (BB) of the exercising arm was measured using surface electrodes. Electrode placements were made as per the recommendations reported by Hermens, Freriks, Disselhorst-Klug and Rau (2000). Skin at the placement site was lightly abraded, cleaned with an alcohol wipe and dried. A pair of disposable sliver/sliver chloride pre-gelled surface electrodes (1·5 cm diameter) were applied, with a centre-to-centre distance of 2·5 cm, longitudinally with the muscle fibres approximately halfway from the motor point area to the distal part of the muscle. An earth electrode was placed on the medial epicondyle of the exercising arm. An example of electrode placement is shown in figure 5. Once the electrodes were in place and the subject was positioned on the isokinetic dynamometer, electrode leads were attached and connected to the preamplifier in an IBM desktop computer operating AMLAB II diagnostic software. The analogue signal was sampled via a 16-bit data acquisition card (Minirack, AMLAB II) at 2500 samples per second. Processing of the signal was firstly through a bandpass filter incorporating, a second order quasi-Butterworth high/low pass filter at 1·817 to 95·797 Hz. Full wave rectification was applied to the signal followed by a second low-pass filter at 5·083 Hz outputting a linear envelope. The raw analogue and linear envelope signal was stored using the AMLAB data collection software system (Appendix D). The signal was recorded during the performance of the exercise intervention and testing of isometric and dynamic strength from the familiarisation session, immediately post, 30 minutes post-exercise and day 7. The processing and analysis of the signal after acquisition was accomplished by selection of the data via lever arm displacement positioning and the tension time of each contraction followed by exporting the data in ASCII format. Exported data files were processed in Microsoft Excel. Full wave rectified data was processed and a signal average was calculated for each contraction condition and velocity.

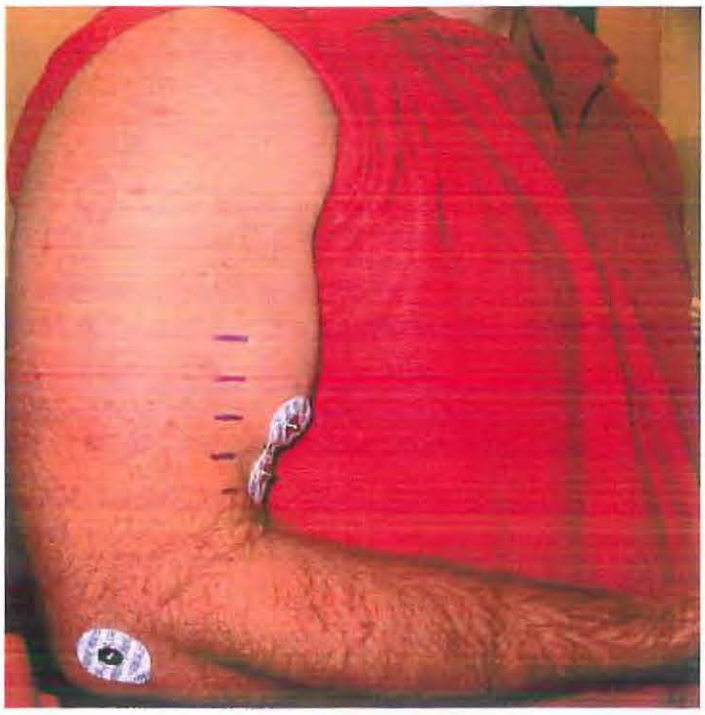


Figure 5 Surface electrode placement sites and circumference measurement sites, with the elbow flexed at 90°. Electrodes positioned on the midline of the biceps brachii and 7cm form the elbow crease with an earth on the medial epicondyle.

3.3.2 Exercise Protocol

The range of motion for the exercise intervention was from 60° of elbow flexion through 180° (full extension). Each subject performed two bouts of eccentric exercise of the elbow flexor muscles (one bout per arm). The two isokinetic exercise protocols, low velocity exercise (LVE = $30°\cdot s^{-1}$) or high velocity exercise (HVE = $210°\cdot s^{-1}$) were designed such that the elbow flexors would spend a total of 120 seconds under eccentric tension (see Table 2). The work:rest ratio for each exercise intervention was controlled and set at 1:7, thus the corresponding velocities for LVE were $30°\cdot s^{-1}$: $10°\cdot s^{-1}$ and for HVE $210°\cdot s^{-1}$: $70°\cdot s^{-1}$ (see Table 2). The 'rest velocity' is the corresponding velocity that the lever arm, and therefore the subject's arm, is returned the starting position. This return phase was a passive movement where the subject was instructed to relax and allow the machine to return their arm to the starting position. A 90-second passive rest period separated each set of 6 repetitions. Subjects were encouraged throughout the lengthening movement of the elbow flexors (a repetition) to apply maximal resistance against the lever arm of the isokinetic dynamometer. The maximal voluntary torque, the work absorbed by the muscle, and surface EMG signal during each repetition was recorded using the same

set up as that employed for isometric strength (see 3.3.1.1) and electromyography (see 3.3.1.7) with the exception that a different schematic was designed (Appendix D). Each subject was provided with visual and verbal feedback of his or her torque output during each repetition and was encouraged to provide a maximal effort.

Table 2

Exercise protocol parameters

	Exercise Protocol	
	$30°\cdot s^{-1}$	$210°\cdot s^{-1}$
Total time of eccentric exercise (secs)	120	120
Time of each contraction (secs)	4	0·57
Time of passive component (secs)	12	1·71
Total number of repetitions	30	210
Total number of sets	5	6
Number of repetitions per set	6	6

3.4 Data Analysis

Values in the result section are presented as mean ± standard error of mean unless otherwise stated. All relevant raw data pertaining to the described results can be found in Appendices E (pilot study) and F (principle investigation).

3.4.1 Data Analysis of Pilot Study Results

Results of the pilot study were analysed using a two-way repeated measure ANOVA for the average peak torque achieved under each dynamic test condition. Isometric average peak torque results were analysed using an independent t-test. Method error and coefficient of variation were calculated for torque generated isometrically and dynamically at velocities of $30°\cdot s^{-1}$, $90°\cdot s^{-1}$, $150°\cdot s^{-1}$ and $210°\cdot s^{-1}$ and used to determine reliability.

3.4.2 Data and Statistical Analysis of Principle Research

Statistical analysis was performed separately on each of the criterion measures, using a 1(velocity) x 7(time) one-way Repeated Measures ANOVA. Statistical significance

was set at $p<0.05$ for these analyses. Any significant main effects were assessed through the application of paired t-tests with associated Bonferroni correction. Planned comparison paired t-tests were conducted at relevant time points where a significant difference was considered to exist to identify interactions between interventions with an associated Bonferroni correction.

For clarity the measures recorded during the exercise interventions were analysed using a one-way Repeated Measures ANOVA to test for differences to baseline at exercise time points of 24, 48, 72, 96 and 120 seconds. Planned comparison paired t-tests were conducted at 24, 48, 72, 96 and 120 seconds of muscle tension to identify significant differences between interventions. Statistical significance was set at $p<0.05$ for these analyses.

3.5 Limitations

3.5.1 Subject Delimitations

The investigator imposed the subject delimitations of excluding subjects who had been involved in resistance training in the six months prior to undertaking the research. Subjects were also excluded if they fell outside of the range of 18 – 45 yrs in order to comply with the upper age range set by the ACSM or asymptomatic males.

3.5.2 Subject Limitations

Due to subjects being sought through word of mouth and poster advertising it was difficult to be certain whether this subgroup is representative of the normal untrained population. The rating of soreness method is a subjective measure, which is dependent upon the subject's pain threshold, other psychological factors, and honesty at the time of testing. A subject's ability to maintain a maximal effort for the duration of the exercise intervention and for the term of each eccentric contraction of the elbow flexors is also subjective.

CHAPTER FOUR

4 RESULTS

4.1 Pilot Study

The coefficients of variation for isometric and dynamic eccentric contractions at velocities of 30°, 90°, 150° and 210°·s^{-1} between trial one and two were 2·8%, 8·9%, 7·0%, 8·6% and 9·5% respectively. Two-way ANOVA with repeated measures, indicated that the main effect of the trial condition (test 1 and test 2) was not significantly different ($p<0·05$) (figure 6). The second main effect of velocity was shown to be significant ($p<0·05$). The one-way ANOVA revealed that there was a significant difference between isometric contractions (velocity 0°·s^{-1}) and all contraction velocities (figure 6) but showed no significant differences between any of the velocities. A significant ($p<0·05$) difference was found between the initial test at 30°·s^{-1} and the repeat. On average, torque increased 16·4% (± 2·7)(SEM) above isometric at 30°·s^{-1} compared to 14·4% (± 2·3) above isometric at 210°·s^{-1}. Mean maximal voluntary isometric torque generated at 90° of elbow flexion in an isometric contraction from the five trials that preceded each test velocity (figure 7) was not significantly different between contractions or test sessions but did decline approximately 8% between test 1 and 2.

On examination of the data the joint angle at which peak torque was reached for the four-isokinetic test velocities was similar for all velocities and both trials. The mean joint angle ranged between 76·4° - 72·6° (±4·1 - 2·5) for trial 1 and between 76·1° - 75·7° (± 2·5 - 2· 0) for trial 2.

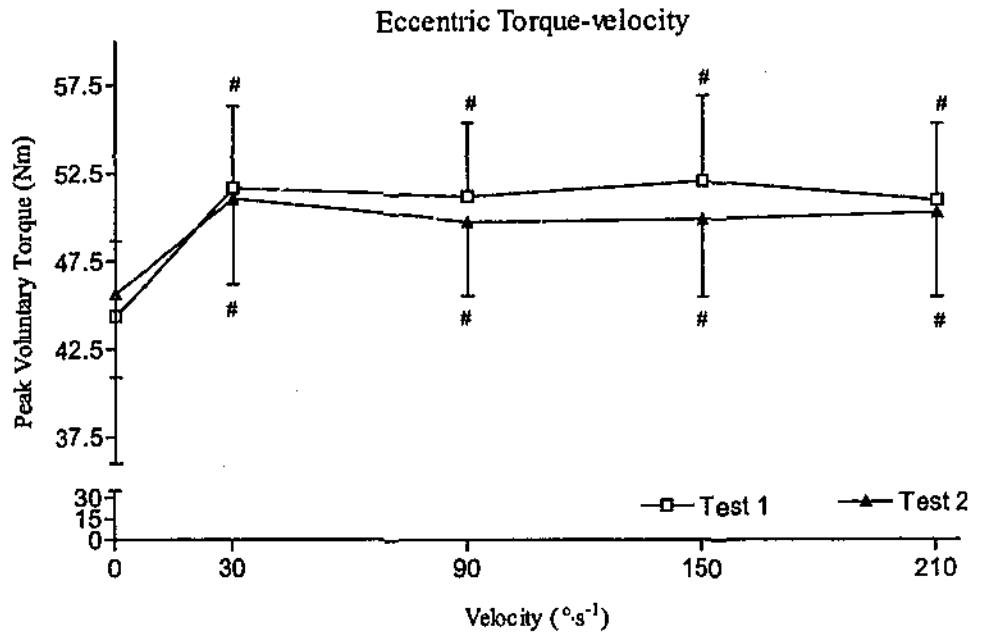


Figure 6 Eccentric torque-velocity relationship (mean ± SEM) of test 1 and test 2 (n = 14). Average torque of two contractions at each contraction velocity. # represents a significant ($p<0.05$) difference to isometric.

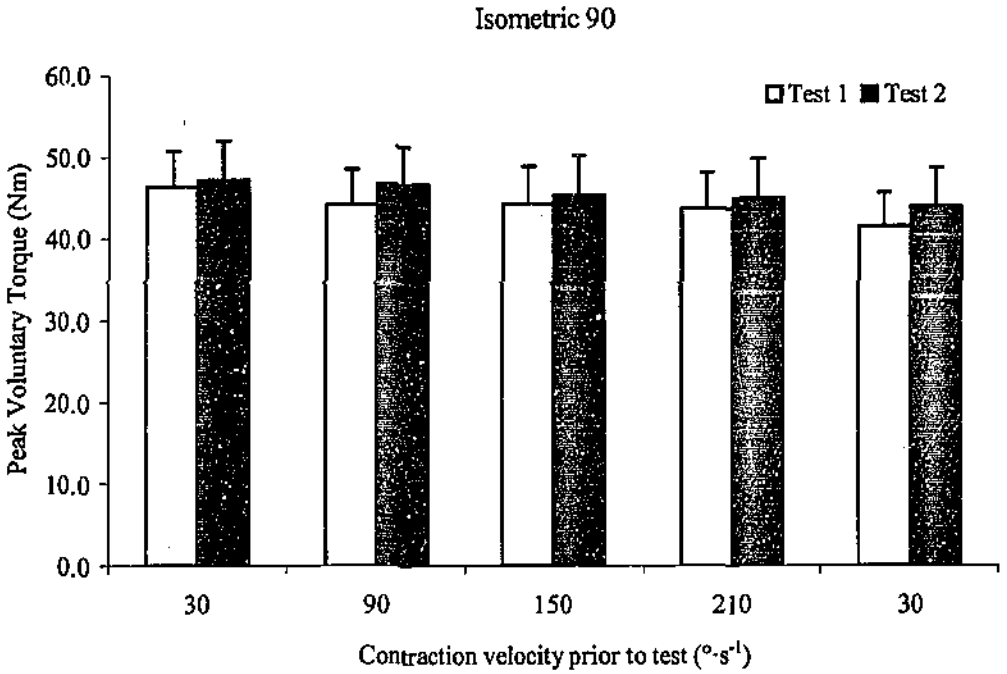


Figure 7 Isometric peak torque at 90° elbow flexion (mean ± SEM) from two contractions prior to each eccentric action velocity.

4.2 Exercise Intervention

4.2.1 Peak Torque

Peak maximal voluntary eccentric torque values were similar for LVE and HVE for the first 4 seconds of muscle tension, ranging from 21·3 - 80·4 Nm (43·0 ± 6·2 Nm, mean ± SEM), and 25·9 to 79·6 Nm (45·7 ± 4·8 Nm) respectively. Figure 8 is a representation of the mean peak torque values produced during 120 seconds of muscle tension for the two contraction velocities, normalised to the first contraction. LVE mean peak torque declined significantly ($p<0·05$) after 24 and 120 seconds of time under tension and significantly ($p<0·01$) after 48, 72 and 96 seconds. In contrast, HVE mean peak torque declined significantly ($p<0·01$) at all compared time points. Differences between exercise velocities were significantly different ($p<0·01$) at all compared time points.

There were distinct differences in the pattern of torque loss between LVE and HVE (figure 8). Torque production during LVE recovered after each successive 24 seconds of muscle tension, although recovery was incomplete each time with the exception of the initial 24 seconds of tension. In comparison HVE declined sharply after 52 seconds of muscle tension to 58% (± 2·8%) of the initial value. For the remaining 68 seconds of muscle tension the peak voluntary torque declined only 4% further.

4.2.2 Work Absorbed

The mean values for total work absorbed between LVE and HVE were markedly different for the first 4 seconds of muscle tension, ranging from 28 – 118 J (58·6 ± 10·2 J, mean ± SEM) for LVE, and 201 - 765 J (373·2 ± 64·4 J) for HVE (figure 9). Figure 10 is a representation of the mean work absorbed during 120 seconds of muscle tension from LVE and HVE normalised to the first 4 seconds of muscle tension. LVE mean normalised work absorbed declined significantly ($p<0·05$) after 24 seconds of muscle tension and significantly ($p<0·01$) following 48, 72, 96 and 120 seconds of muscle tension. HVE mean normalised work absorbed declined

significantly ($p<0.05$) after 24 seconds and significantly ($p<0.01$) following 48 – 120 seconds of muscle tension. Differences between exercise velocities were significant ($p<0.05$) at 48, 72 and 96 seconds and significant ($p<0.01$) at 120 seconds.

4.2.3 Average EMG Signal

Mean full wave rectified EMG signal for the 120 seconds of muscle tension from the biceps brachii was similar between exercise conditions (figure 11), but HVE was consistently higher. The signal average for LVE after 4 seconds of muscle tension was 0·75 mV (± 0·10) and HVE 0·96 mV (± 0·12) (mean ± SEM). In comparison the signal average did not change greatly after 120 seconds of muscle tension where for LVE the signal was 0·88 mV (± 0·09) and HVE was 1·19 mV (± 0·10).

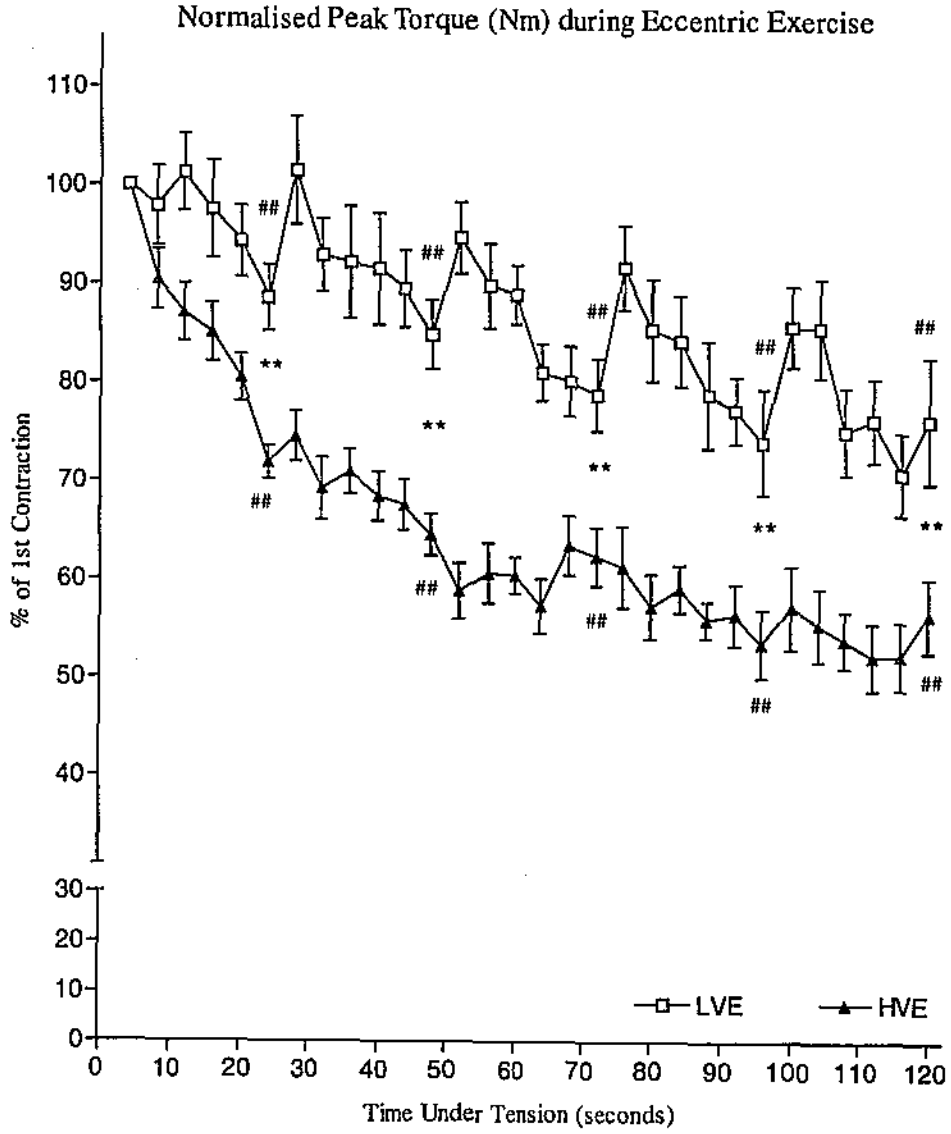


Figure 8 Peak torque generated during LVE (n = 12) and HVE (n = 12), shown as a percentage of the first four seconds of muscle tension (mean ± SEM).
represents a significant (p<0·01) difference to baseline. ** represents a significant (p<0·01) difference between conditions.

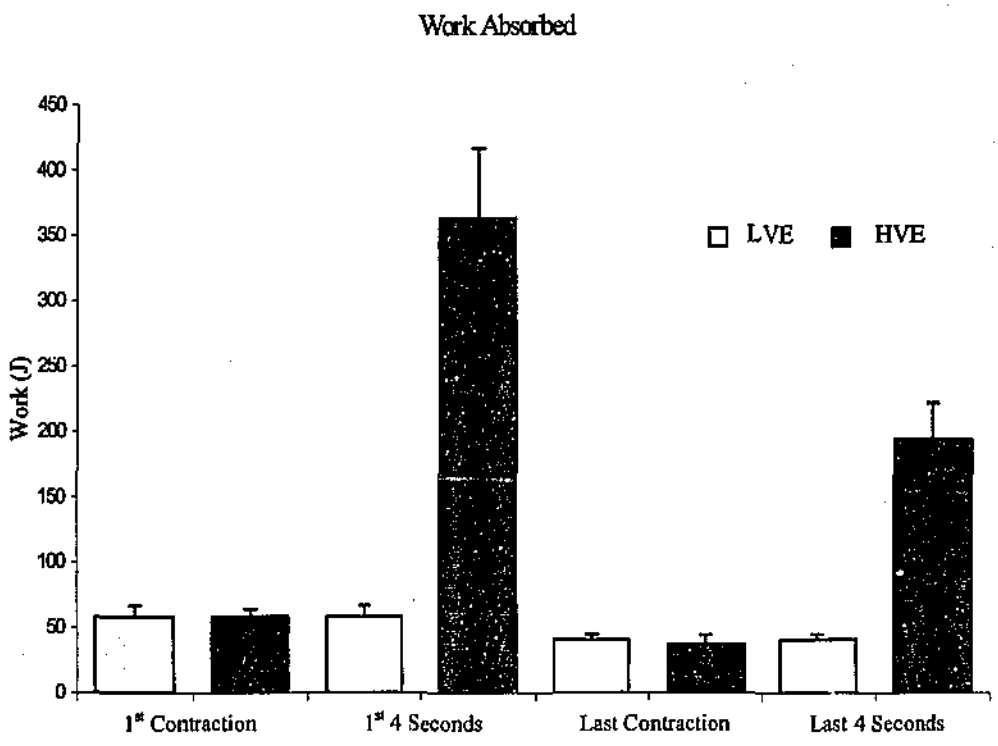


Figure 9 Work absorbed during the first and last eccentric contraction during LVE (n = 12) and HVE (n = 12) (mean ± SEM). Work absorbed during the first and last 4 seconds of muscle tension for exercise interventions LVE (n = 12) and HVE (n = 12) (mean ± SEM).

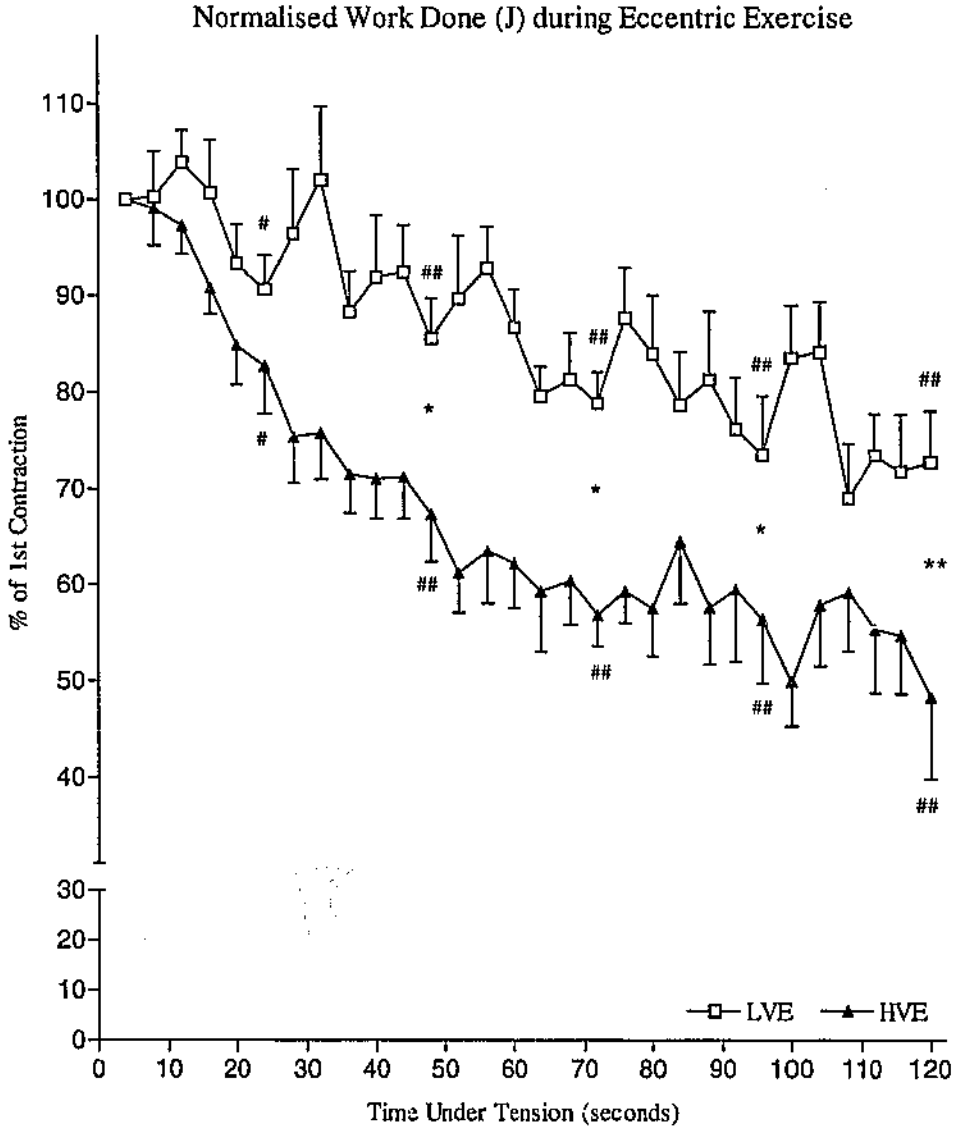


Figure 10 Work absorbed by the exercised limb during LVE (n = 12) and HVE (n = 12), shown as a percentage of the first four seconds of muscle tension (mean ± SEM).
represents a significant ($p<0{\cdot}05$) difference to baseline, ## represents a significant ($p<0{\cdot}01$) difference to baseline. * represents a significant ($p<0{\cdot}05$) difference between conditions, ** represents a significant ($p<0{\cdot}01$) difference between conditions.

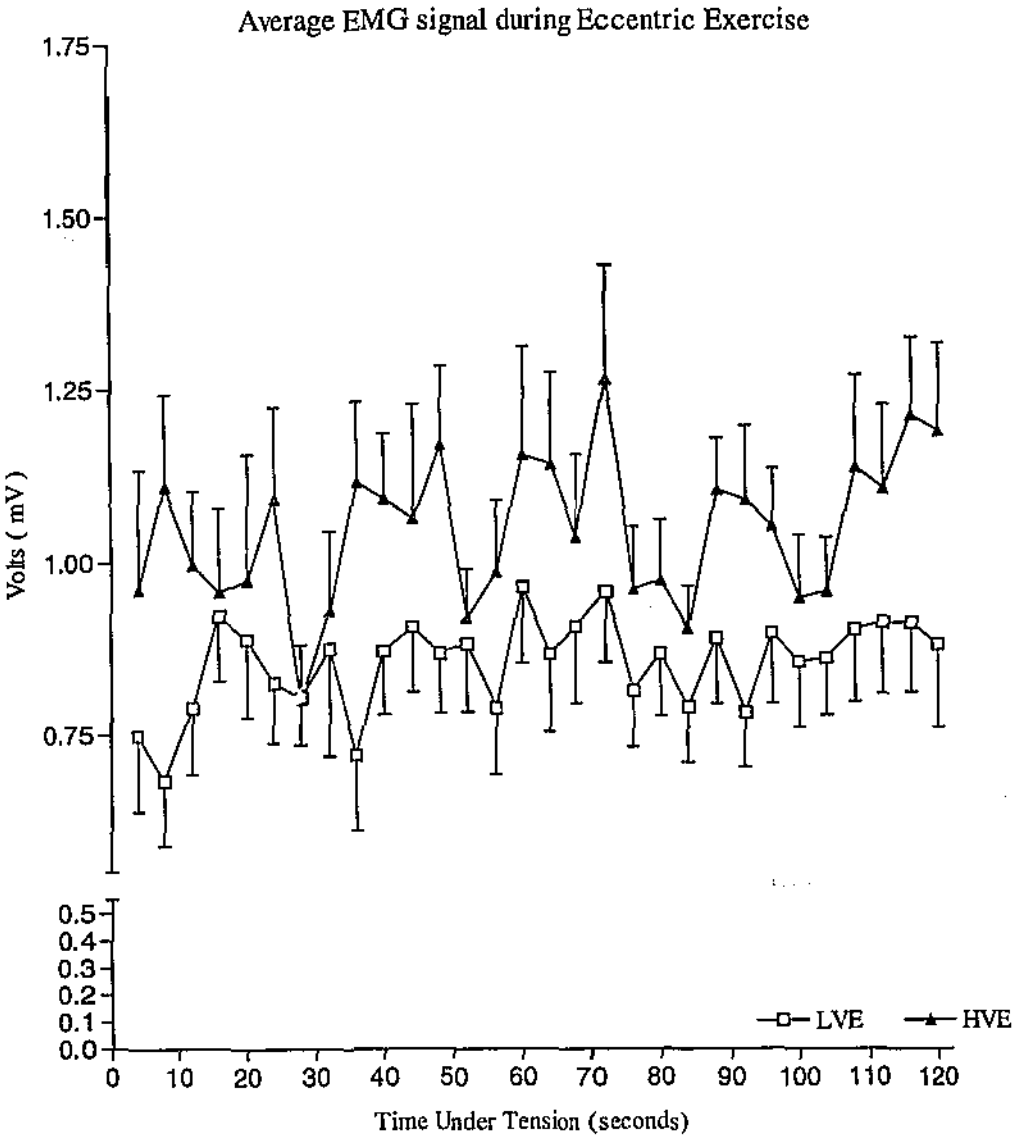


Figure 11 Average rectified EMG signal generated during LVE (n=12) and HVE (n=12), shown in absolute values for 120 seconds of muscle tension (mean ± SEM).

4.3 Isometric Strength

Peak maximal voluntary isometric torques at 90° of elbow flexion were similar for both groups prior to LVE or HVE, ranging from 25·2 - 82·2Nm (47·4 ± 5·32Nm, mean ± SEM) (LVE), and 26·9 to 79·0Nm (45·8 ± 5·1Nm) (HVE). Similarly, there was no significant difference between arms at 150° flexion (LVE = 32·7 ± 4·4Nm, HVE = 29·7 ± 4·5Nm).

Immediately following LVE, mean peak isometric strength declined to 72% of pre-exercise values and continued to decrease to 69% of pre-exercise 30 minutes post-exercise (figure 12). In comparison, following HVE subjects' had a greater torque decrement producing only 32% of baseline immediately post-exercise and 33% at 30 minutes later (figure 12). Maximal voluntary peak torque remained significantly reduced ($p<0·01$) up to 48 hours post exercise and continued to be depressed ($p<0·05$) until 96 hours post following LVE. Following HVE subjects' experienced protracted torque loss that was significantly ($p<0·01$) below baseline 168 hours post-exercise being only 50% of pre-exercise levels. Differences between peak isometric torques at 90° following exercise at the two contraction velocities were significant ($p<0·01$) at all time points.

Peak isometric strength at 150° of elbow extension responded in a similar manner to that at 90° (Appendix F). Isometric strength was significantly ($p<0·01$) decreased for both LVE and HVE up to 48 hours post-exercise when compared to pre-exercise. Responses following HVE continued to be significantly ($p<0·01$) below baseline across all recording time points. In contrast, responses following LVE were significant only to 96 hours post-exercise compared to pre-exercise. Time courses for recovery were similar following both bouts of exercise, though differences between conditions were still significant. Differences between velocities were significant ($p<0·01$) immediately post until 24 hours post exercise. Significant ($p<0·05$) differences were present 48 hours and 168 hours post exercise.

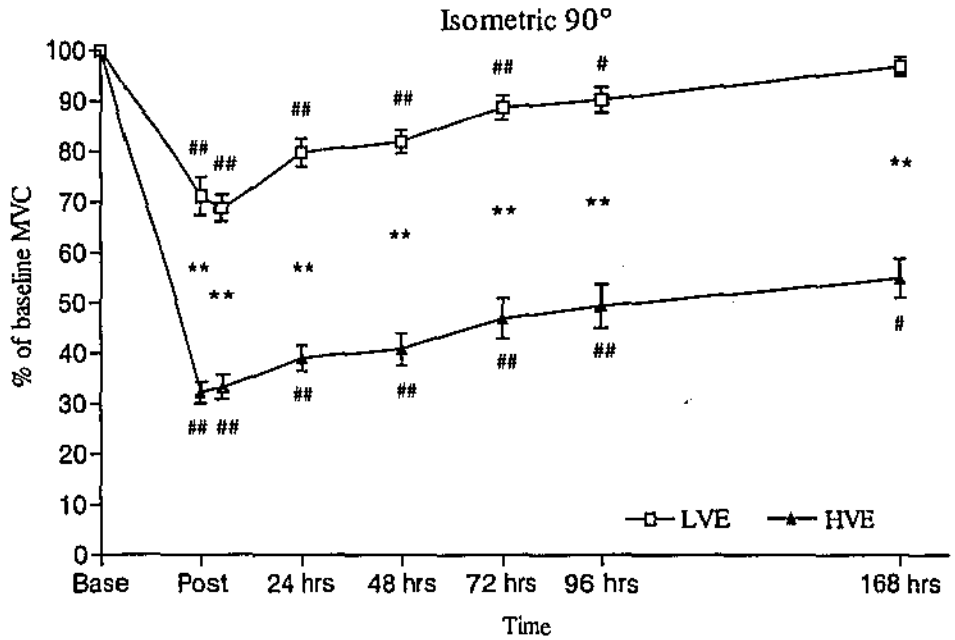


Figure 12 Normalised maximal voluntary isometric torque at 90° (mean ± SEM) of elbow flexion. Expressed as a percentage of pre-exercise, for LVE (n = 12) and HVE (n = 12).
represents a significant ($p<0.05$) difference to baseline, ## represents a highly significant ($p<0.01$) difference to baseline. ** represents a highly significant ($p<0.01$) difference between conditions.

4.4 Dynamic Strength

4.4.1 Concentric Torque

Peak maximal voluntary concentric torques were similar for both groups prior to LVE and HVE (table 3). Both LVE and HVE resulted in significant torque decrements ($p<0.01$) immediately post and 30 minutes following exercise for all tested dynamic concentric velocities. Recovery trends were similar to those displayed for isometric strength (figure 13 and 14).

Immediately following LVE, mean peak concentric torque at $30°\cdot s^{-1}$ declined to 73% of pre-exercise values and continued to decrease to 70% of pre-exercise 30 minutes later (figure 13). In comparison, following HVE subjects' experienced a greater torque decrement, producing only 44% immediately post and no change 30 minutes following exercise (figure 13). Torque remained reduced up to 72 hours post-exercise ($p<0.01$) and continued until 96 hours following LVE ($p<0.05$). Following HVE subjects' experienced a protracted torque loss at $30°\cdot s^{-1}$ that was significantly ($p<0.01$) below baseline 168 hours post-exercise. Subjects had recovered to 96% of baseline 168 hours post-exercise following LVE, but at the equivalent time following HVE they had recovered to 80% of pre-intervention values (figure 13). Differences between responses for concentric torque at $30°\cdot s^{-1}$ following LVE and HVE were significant ($p<0.01$) at all recorded time points up to 72 hours following exercise and were still significant ($p<0.01$) 168 hours post-exercise.

Following LVE, mean peak concentric torques at $210°\cdot s^{-1}$ declined to 72% of pre-exercise values immediately following exercise and continued to decline to 67% of pre-exercise 30 minutes later (figure 14). In comparison, following HVE subjects' experienced a greater torque decrement producing only 45% of baseline immediately post-exercise and decreased to 41% 30 minutes later (figure 14). Torque was significantly ($p<0.05$) below pre-intervention levels at all time points post-exercise for both conditions. Subjects had recovered to 91% of baseline 168 hours post-exercise following LVE compared to only 75% recovery following HVE at the

same time point. Differences between interventions for peak concentric torque at $210°\cdot s^{-1}$ were significant ($p<0{\cdot}05$) at all recorded time points post-exercise with the exception of 96 hours.

Table 3

Baseline maximal voluntary concentric torque (Nm)

	Exercise Velocities					
	LVE			HVE		
	Mean (SEM)	Min	Max	Mean (SEM)	Min	Max
Concentric action						
$30°\cdot s^{-1}$	35·6 (4·59)	17·4	67·7	33·9 (4·25)	16·9	61·0
$90°\cdot s^{-1}$	33·3 (3·9)	19·1	55·5	32·3 (3·8)	16·4	54·4
$150°\cdot s^{-1}$	30·7 (3·5)	16·7	50·4	29·0 (3·4)	14·7	47·0
$210°\cdot s^{-1}$	28·4 (3·42)	14·3	44·4	25·9 (3·20)	13·2	42·0

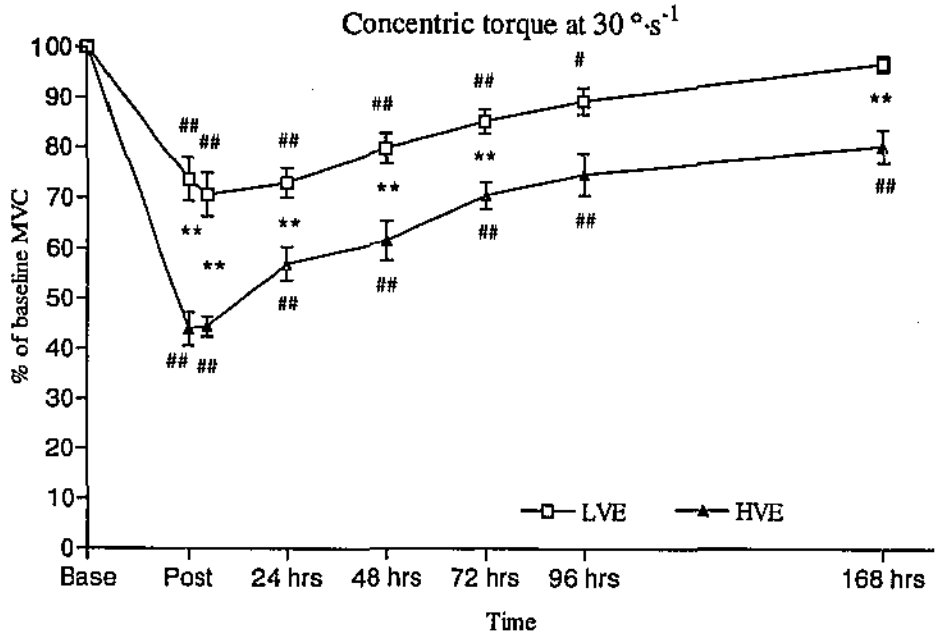


Figure 13 Normalised maximal voluntary concentric torque at 30°·s^{-1} (mean ± SEM) of elbow flexion. Expressed as a percentage of pre-exercise, for LVE (n = 12) and HVE (n = 12).
represents a significant (p<0·05) difference to baseline, ## represents a significant (p<0·01) difference to baseline. ** represents a significant (p<0·01) difference between conditions.

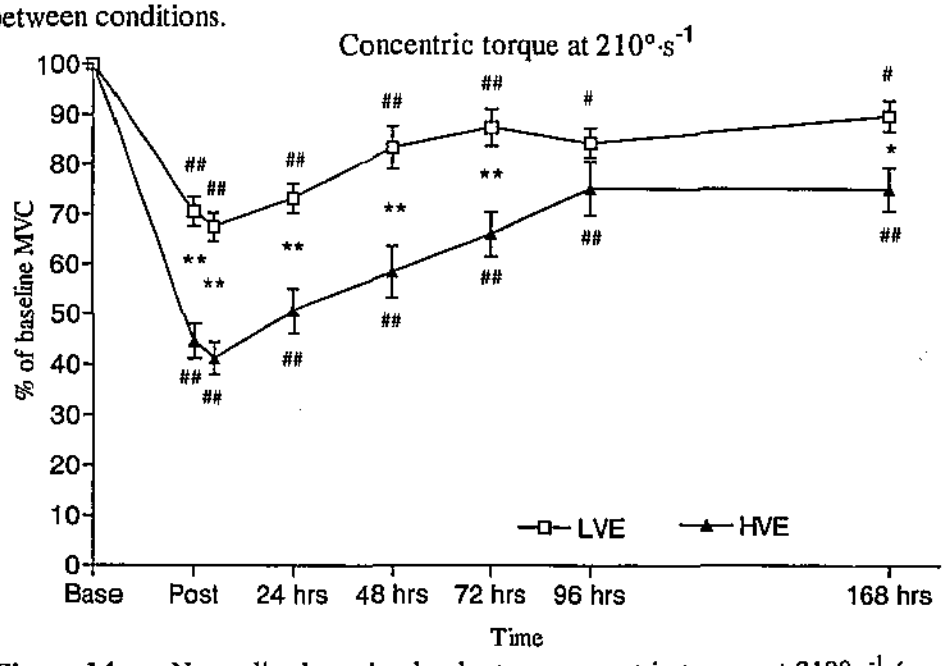


Figure 14 Normalised maximal voluntary concentric torque at 210°·s^{-1} (mean ± SEM) of elbow flexion. Expressed as a percentage of pre-exercise, for LVE (n = 12) and HVE (n = 12).
represents a significant (p<0·05) difference to baseline, ## represents a significant (p<0·01) difference to baseline. * represents a significant (p<0·05) difference between conditions, ** represents a significant (p<0·01) difference between conditions.

4.4.2 Eccentric Torque

Peak maximal voluntary eccentric torques at $30°\cdot s^{-1}$ were similar for both arms prior to LVE and HVE, ranging from 28·6 - 91·6Nm (51·1 ± 5·83Nm, mean ± SEM), and 28·9 to 80·9Nm (47·0 ± 5·13Nm), respectively. Similarly, no significant difference was apparent for mean peak maximal voluntary eccentric torques at $210°\cdot s^{-1}$ for both arms prior to LVE and HVE, with values ranging from 30·5 - 83·3Nm (49·6 ± 4·75Nm, mean ± SEM), and 29·0 to 81·0Nm (48·4 ± 4·85Nm), respectively.

Differences were significant ($p<0·01$) between conditions (LVE and HVE) at an eccentric contraction velocity of $30°\cdot s^{-1}$ for up to 72 hours post-exercise (figure 15). Recovery of peak torque at this action velocity was similar in both conditions, yet torque generation remained significantly below baseline 168 hours post-exercise for HVE. Similar recovery trends shown in other strength measures were also apparent at an eccentric test velocity of $30°\cdot s^{-1}$.

Differences between conditions (LVE and HVE) at an eccentric contraction velocity of $210°\cdot s^{-1}$ were also significant ($p<0·01$) up to 72 hours post exercise (figure 16). The recovery trend of torque generated at this action velocity was similar to that developed at a contraction velocity of $30°\cdot s^{-1}$. Interestingly, eccentric torque production at $210°\cdot s^{-1}$ following the HVE bout was similar to that described for the same velocity concentric torque production after HVE, in as far as there was a similar levelling 96 hours post-exercise and remained significantly below baseline through 168 hours after exercise.

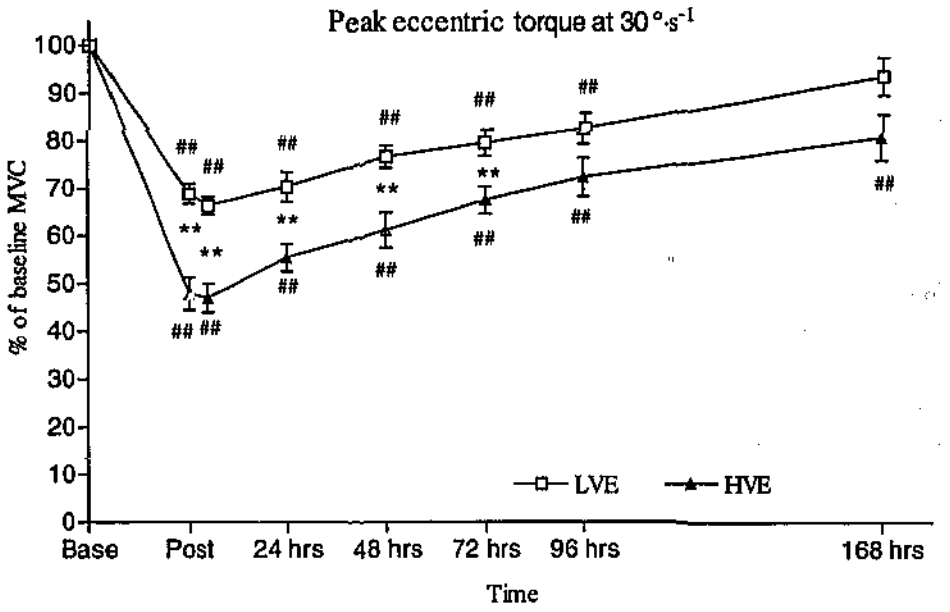


Figure 15 Normalised maximal voluntary eccentric torque at 30°·s^{-1} (mean ± SEM) of elbow flexion. Expressed as a percentage of pre-exercise, for LVE (n = 12) and HVE (n = 12).
represents a significant (p<0·01) difference to baseline. ** represents a significant (p<0·01) difference between conditions.

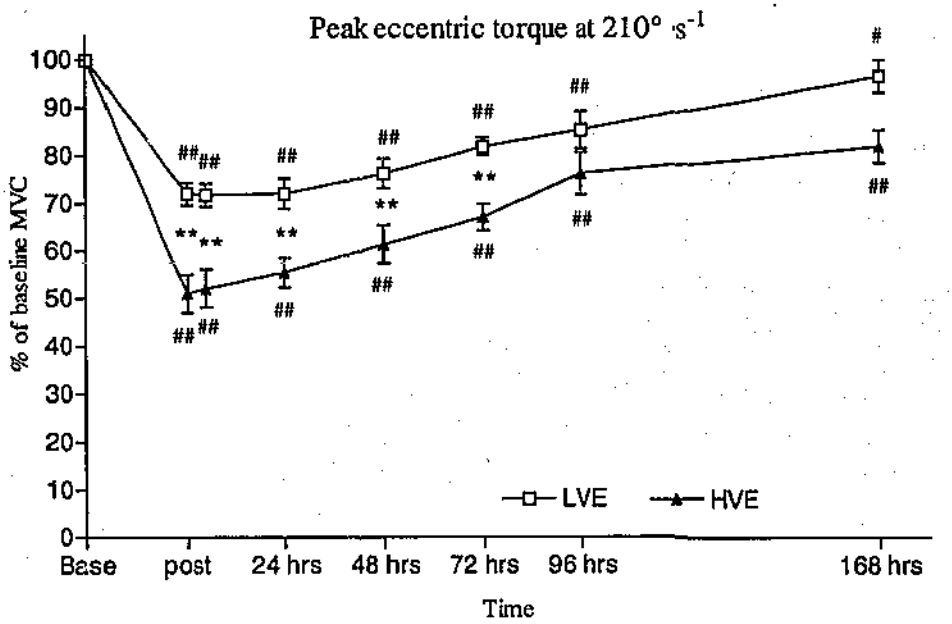


Figure 16 Normalised maximal voluntary eccentric torque at 210°·s^{-1} (mean ± SEM) of elbow flexion. Expressed as a percentage of pre-exercise, for LVE (n = 12) and HVE (n = 12).
represents a significant (p<0·05) difference to baseline, ## represents a significant (p<0·01) difference to baseline. ** represents a significant (p<0·01) difference between conditions.

4.5 Range of Motion (ROM)

ROM values were similar for both groups prior to LVE and HVE, ranging from 123·5° - 163·0° (135·4° ± 3·0, mean ± SEM) (LVE), and 124·0° to 159·0° (136·8° ± 2·4) (HVE). Both exercise interventions resulted in significant (LVE, $p<0·01$ and HVE, $p<0·05$) reductions in ROM immediately post-exercise (LVE = -10·23° ± 2·2, HVE = -23·9° ± 7·7) compared to that measured before the interventions (figure 17). ROM continued to decrease following LVE with the peak occurring 30 minutes post (-12·2° ± 4) while ROM had began to recover for subjects' following HVE (-20·4° ± 5·8). The ROM was significantly reduced (LVE, $p<0·05$ and HVE, $p<0·01$) compared to baseline up to 96 hours post-exercise (figure 17). Differences in the change in ROM between exercise bouts were significant ($p<0·01$) 24 to 72 hours post-exercise, and 96 hours later ($p<0·05$). ROM 168 hours following exercise had recovered to 133·9° ± 2·7 and 130·4° ± 2·9 for LVE and HVE respectively.

4.5.1 Relaxed Elbow Angle (RANG)

RANG was similar for both groups prior to LVE or HVE, ranging from 148° - 156° (152·3° ± 0·7, mean ± SEM), and 146° - 165° (154·8° ± 1·7) respectively. A reduced RANG indicates that, when the subject allows the arm to hang by their side, it is in a more flexed position. LVE resulted in a significantly reduced RANG immediately post-exercise and at 24, 48 ($p<0·05$) and 96 hours post-exercise ($p<0·01$) returning to baseline levels by 168 hours (figure 18). In response to LVE, RANG declined -3·5° ± 1·4 immediately post-exercise and recovered transiently 30 minutes later to -2·8° ± 1·9 before achieving a peak reduction of -4° ± 1·4 at 48 hours following exercise. In HVE RANG declined -6·2° ± 1·5 immediately following exercise with no recovery 30 minutes later (-6·2° ± 1·8) and continued to decrease reaching a nadir of -10·9° ± 2·1 at 72 hours post-exercise. The changes in RANG in response to HVE were significant immediately after exercise, and at all other time points post-exercise ($p<0·05$). In contrast to LVE a return to baseline was not achieved by 168 hours (-4·4° ± 1·8). The changes in RANG between conditions were significant following exercise ($p<0·01$) 72 hours post-exercise and significant at 48, 96 and 168 hours post-exercise ($p<0·05$).

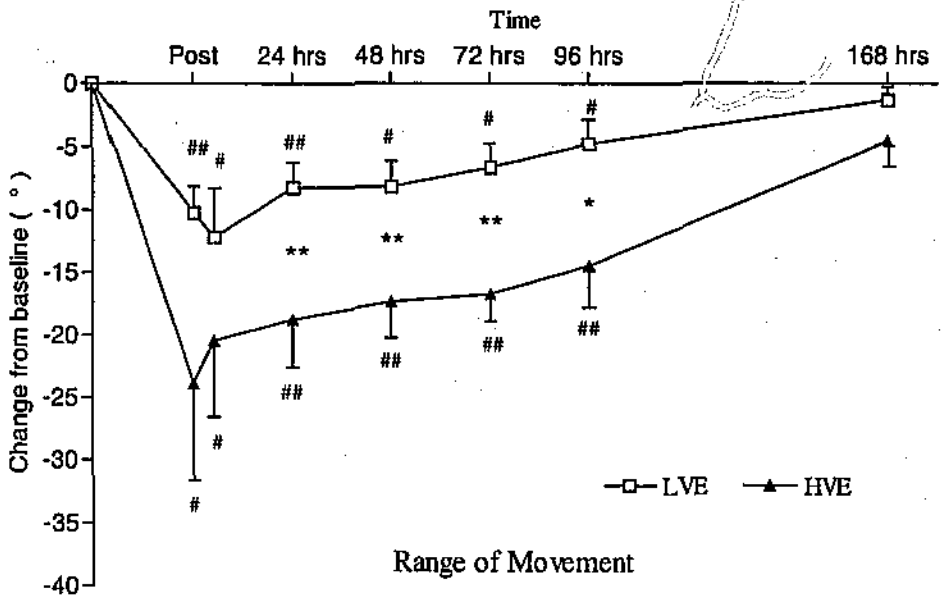


Figure 17 Change in ROM (mean ± SEM), expressed as degrees from pre-exercise, for LVE (n = 12) and HVE (n = 12).
represents a significant (p<0·05) difference to baseline, ## represents a significant (p<0·01) difference to baseline. * represents a significant (p<0·05) difference between conditions, ** represents a significant (p<0·01) difference between conditions.

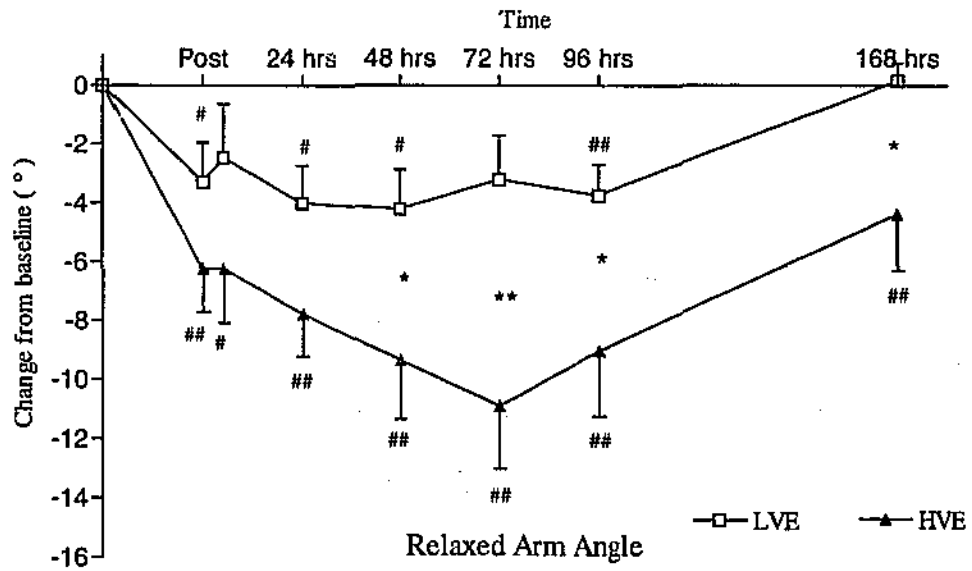


Figure 18 Change in RANG (mean ± SEM), expressed as degrees from pre-exercise, for LVE (n = 12) and HVE (n = 12).
represents a significant (p<0·05) difference to baseline, ## represents a highly significant (p<0·01) difference to baseline. * represents a significant (p<0·05) difference between conditions, ** represents a highly significant (p<0·01) difference between conditions.

4.6 Creatine Kinase (CK)

Baseline plasma CK values were within the 'normal' range prior to LVE and HVE, 174·1 IU ± 31·6, (mean ± SEM), and 150·9 IU ± 27·8) respectively. The HVE elicited a significant ($p<0·05$) increase in CK from baseline 48 to 168 hours post-exercise (figure 19). In contrast, LVE did not differ significantly from baseline. Peak concentrations for HVE occurred 96 hours after exercise (1298·2 IU ± 427·7) and remained elevated 240 hours post-exercise (467·2 IU ± 146·1). In contrast LVE elicited a non-significant average peak of 279·9 IU ± 89·2 at 72 hours post-exercise, significant differences were observed between conditions at 48, 72, 96 and 168 hours following exercise.

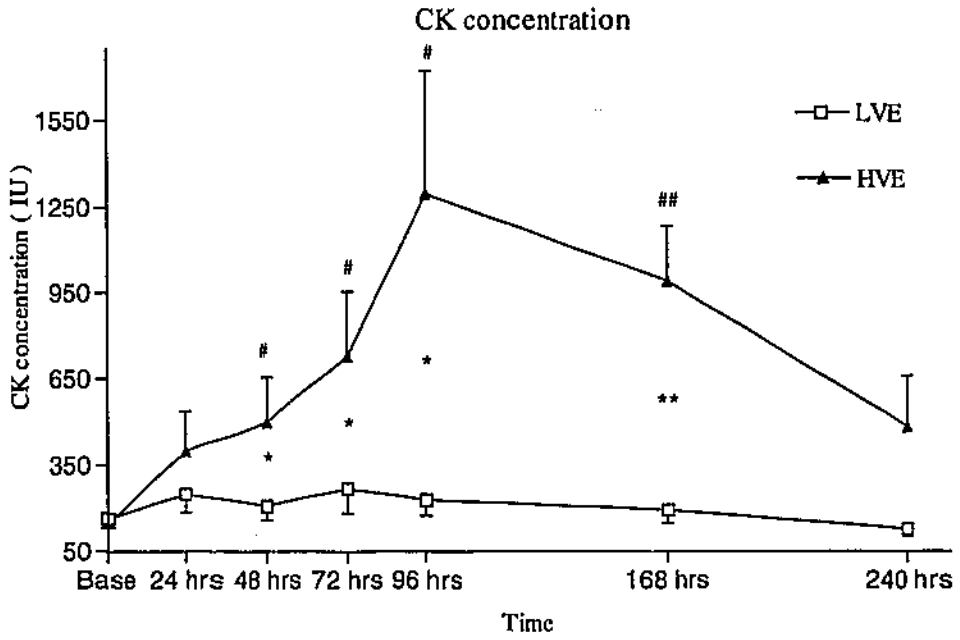


Figure 19 Change in serum CK concentration (mean ± SEM), expressed in absolute values in IU, for LVE (n = 12) and HVE (n = 12).
represents a significant ($p<0·05$) difference to baseline, ## represents a highly significant ($p<0·01$) difference to baseline. * represents a significant ($p<0·05$) difference between conditions, ** represents a highly significant ($p<0·01$) difference between conditions.

4.7 Soreness (SOR) and Tenderness

4.7.1 Tenderness

Subjects reported significant levels of muscle tenderness (figure 20) ($p<0.01$) up to 96 hours post-exercise HVE intervention. In contrast responses to LVE were only significant until 72 hours following exercise ($p<0.05$). Significant differences between responses to each condition (LVE and HVE) were evident 72 and 96 hours post-exercise ($p<0.05$). Palpated soreness peaked 24 hours earlier for LVE (19mm ± 6) compared to the HVE condition at (36mm ± 8). Muscle tenderness had dissipated by 168 hours post-exercise following both LVE and HVE.

4.7.2 Extension Soreness

Reported ratings of extension soreness (figure 21) following HVE were significantly above baseline from 24 to 96 hours following exercise ($p<0.01$), with forced extension still eliciting soreness (non-significant) 168 hours post-exercise intervention. Extension soreness resulting from LVE was significantly above baseline from 24 through 72 hours following exercise ($p<0.05$). Ratings of extension soreness peaked for both interventions 48 hours post-exercise (LVE = 24.9mm ± 7.1, HVE = 56.6 ± 7.7). Differences in extension soreness between conditions were significant from 24 through 96 hours following exercise ($p<0.05$).

4.7.3 Flexion Soreness

Flexion soreness was similar to extension for LVE with a peak in soreness occurring 48 hours post-exercise (20.6mm ± 7) and values significantly ($p<0.05$) above baseline evident at 24 through 72 hours following the intervention (figure 22). HVE flexion soreness increased significantly ($p<0.01$) from baseline at each time point up to 96 hours following the exercise intervention (peak of 42mm ± 8.4) before returning to baseline at 168 hours post-exercise. Passive flexion soreness was statistically different between groups ($p<0.05$) at only 72 and 96 hours after exercise.

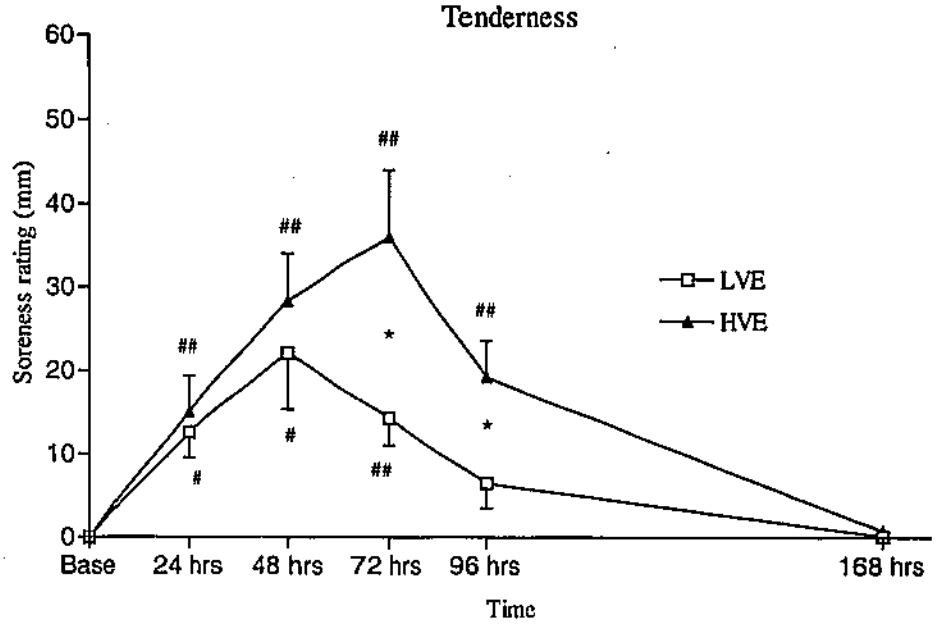


Figure 20 Soreness upon palpation of the elbow flexors (mean ± SEM) for LVE (n = 12) and HVE (n = 12).
represents a significant (p<0·05) difference to baseline, ## represents a significant (p<0·01) difference to baseline. * represents a significant (p<0·05) difference between conditions.

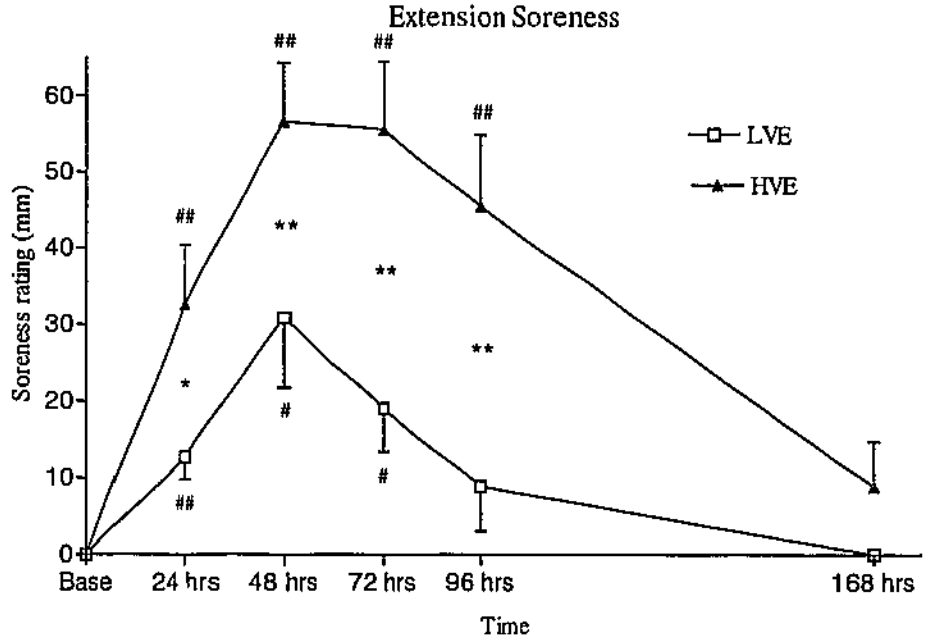


Figure 21 Soreness upon extension of the elbow (mean ± SEM) for LVE (n = 12) and HVE (n = 12).
represents a significant (p<0·05) difference to baseline, ## represents a significant (p<0·01) difference to baseline. * represents a significant (p<0·05) difference between conditions, ** represents a significant (p<0·01) difference between conditions.

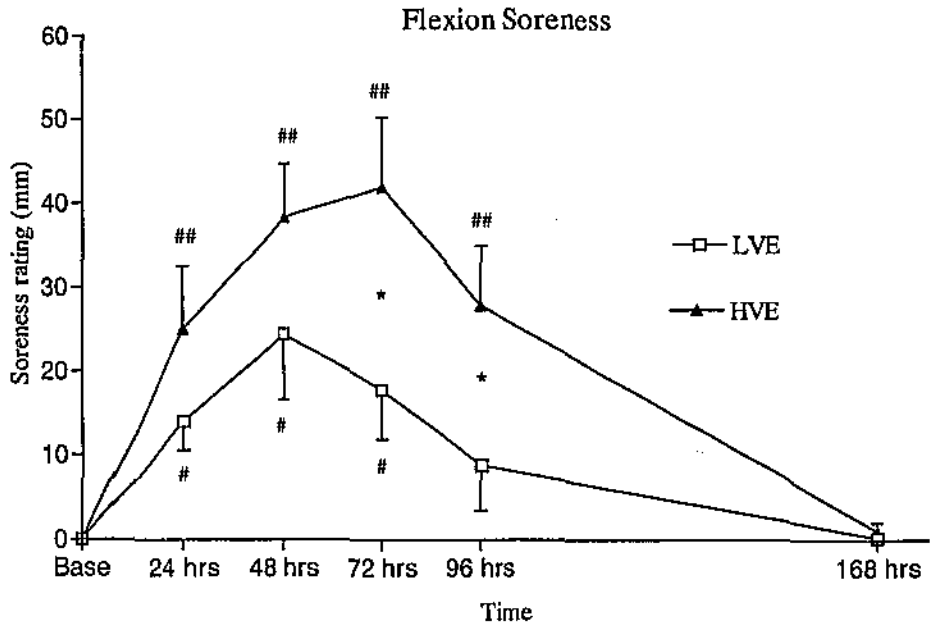


Figure 22 Soreness upon flexion of the elbow (mean ± SEM) for LVE (n = 12) and HVE (n = 12).
represents a significant ($p<0.05$) difference to baseline, ## represents a significant ($p<0.01$) difference to baseline. * represents a significant ($p<0.05$) difference between conditions.

4.8 Arm Circumference (CIR)

Upper arm CIR values were similar for both arms prior to LVE and HVE, ranging from 22·1 - 30·0cm (26·2cm ± 0·7, mean ± SEM), and 22·5 - 29·9cm (26·4cm ± 0·6) respectively. Figure 23 represents the change in CIR following exercise for each condition. In contrast to LVE which elicited no significant CIR increase over time, immediately following HVE subject's CIR had significantly increased ($p<0·01$). CIR 30 minutes following HVE was elevated significantly ($p<0·05$) and remained so at all subsequent time points. Peak CIR following HVE occurred 72 hours post-exercise with a mean increase of 0·8 cm ± 0·1. Significant ($p<0·05$) differences between conditions (LVE and HVE) were recorded at 24 through 168 hours post-exercise.

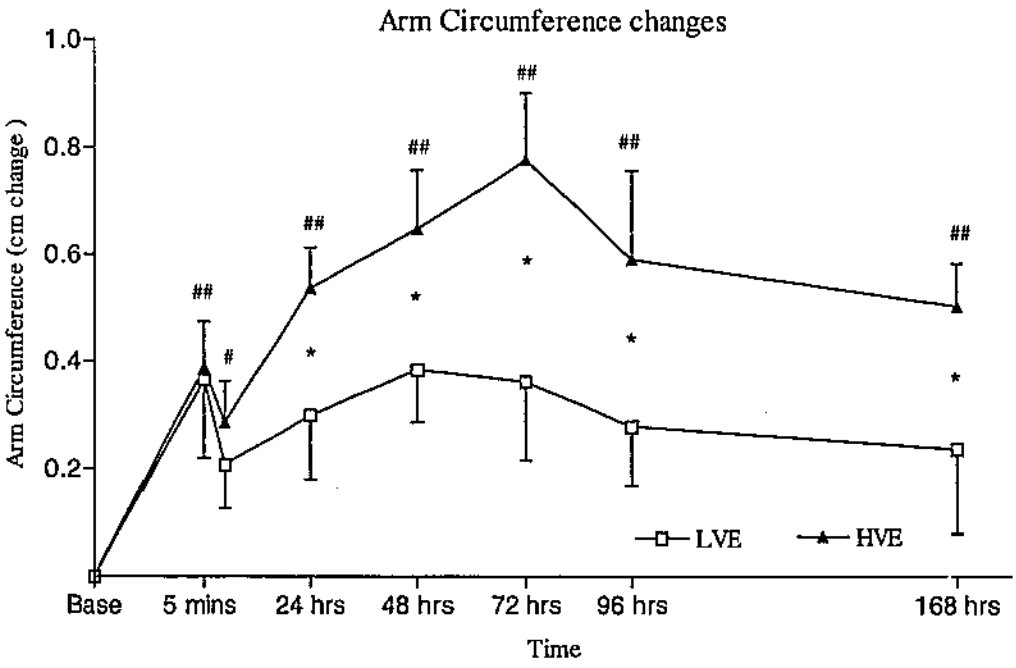


Figure 23 Change in arm circumference (mean ± SEM), expressed as centimetres change from pre-exercise, for LVE (n = 12) and HVE (n = 12). # represents a significant ($p<0·05$) difference to baseline, ## represents a highly significant ($p<0·01$) difference to baseline. * represents a significant ($p<0·05$) difference between conditions.

4.9 Electromyography (EMG)

The observed trends seen in the size of the mean average full wave rectified EMG signal for the entire ROM was similar for all contraction modalities (table 4). To ensure the observations were accurate and not from changes occurring due to skin impedance, familiarisation and pre-test responses were compared using a t-test for contraction modalities. The t-test showed no significantly different variation between testing occasions for either exercise condition (LVE and HVE). No individual contraction modality elicited a significantly different response between either exercise condition (LVE and HVE). The signals recorded during isometric contractions at 90° elbow flexion were similar for both contraction modalities, with a slight increase in the average signal 30 minutes post-exercise and returning to baseline levels after 7 days, at all times the signal recorded during HVE was slightly higher. Average rectified EMG signal during isometric contractions at 150° elbow flexion were very similar following LVE and HVE, with the only observable difference occurring 7 days post-exercise. At this time the signal recorded following LVE had returned to baseline. In contrast the signal 7 days after HVE was at its lowest average value. The EMG signal recorded during maximal voluntary concentric contractions at $30°\cdot s^{-1}$ decreased slightly following both exercise interventions. The EMG signal during maximal voluntary concentric contractions at $210°\cdot s^{-1}$ followed similar trends to those recorded during concentric contractions at $30°\cdot s^{-1}$.

Table 4

Average full wave rectified surface EMG signal

Contraction Mode		Time			
		Base	Post	30 min Post	7 Days Post
Isometric 90°	LVE	0.856 (0·11)	0.937 (0·11)	0.975 (0·10)	0.855 (0·07)
	HVE	0.964 (0·49)	1.034 (0·49)	1.088 (0·51)	1.096 (0·51)
Isometric 150°	LVE	1.236 (0·11)	1.091 (0·10)	1.156 (0·09)	1.356 (0·13)
	HVE	1.264 (0·45)	1.152 (0·49)	1.223 (0·55)	1.157 (0·48)
Concentric 30°·s^{-1}	LVE	1.008 (0·10)	0.946 (0·11)	0.952 (0·08)	1.114 (0·15)
	HVE	1.162 (0·49)	1.075 (0·49)	1.083 (0·58)	1.097 (0·56)
Concentric 210°·s^{-1}	LVE	1.178 (0·12)	1.007 (0·08)	0.960 (0·09)	1.218 (0·13)
	HVE	1.169 (0·46)	1.046 (0·34)	1.064 (0·42)	1.072 (0·38)
Eccentric 30°·s^{-1}	LVE	0.873 (0·07)	0.886 (0·07)	1.01 (0·08)	0.817 (0·09)
	HVE	0.92 (0·47)	0.852 (0·35)	0.942 (0·43)	0.718 (0·28)
Eccentric 210°·s^{-1}	LVE	0.741 (0·08)	0.900 (0·07)	0.856 (0·07)	0.769 (0·07)
	HVE	0.821 (0·45)	0.791 (0·41)	0.852 (0·47)	0.645 (0·28)

Values expressed in mV (mean ± SEM) LVE (n = 12) and HVE (n = 12).

CHAPTER FIVE

5 DISCUSSION

Limited research has been conducted into the role of stretch velocity in exercise-induced muscle damage (EIMD); mostly this has involved animal models or isolated muscle fibres. In either instance, muscle activation was externally induced and the stretch lengths used may have been outside of the anatomical range of motion. The research completed for the purposes of this thesis used maximal voluntary contractions of elbow flexors in situ, with the time that muscles were under tension of 120 seconds and comparisons made of two stretch velocities ($30°·s^{-1}$ and $210°·s^{-1}$).

5.1 Preliminary Torque-Velocity Relationship

The testing protocol used in the pilot study to investigate the eccentric torque-velocity relationship was found to have a small but not statistically significant effect on isometric MVC torque (figure 7). Brooks and Faulkner (2001) showed that EIMD can occur as a result of a single eccentric contraction. Muscle damage is also known to be dependent on the length at which contraction commences, with the severity being greater at longer muscle lengths (Child, Saxton, & Donnelly, 1998). For this reason the range of movement tested was restricted to a mid-range of 80° of the subjects ROM, eliminating angles at which the muscle was most extended (stretched) to reduce the potential for EIMD. Contraction was initiated before the lever arm commenced the lengthening action so that full voluntary activation was achieved and that the peak torque generated would occur within the normal optimal range of 70°-110° of elbow flexion.

The pilot study findings (figure 6) of a 14 – 16% increase in peak voluntary torque above isometric values is consistent with the findings of Griffin (1987) using female subjects and those of Hortobagyi and Katch (1990) in subjects with low strength. A further finding of no significant difference between the tested velocities despite a decreasing trend, supports findings by Cramer et al. (2002). However, further studies by Griffin et al. (1993) using men and women and Hortobagyi and Katch (1990) in strength trained subjects presented contrary data. Previous research has indicated that the eccentric torque-velocity relationship of the elbow flexors at high velocities is inconsistent, with four patterns described. Slowly increasing with increasing velocity (Hortobagyi & Katch, 1990), a slowly decreasing with increasing velocity (Komi et al., 2000), or a plateau (Griffin, 1987; Griffin et al., 1993). Other researchers using various muscle groups have shown there to be a steady trend for strength to increase with velocity of eccentric contraction (de Ruiter & de Haan, 2001; Westing, Seger, Karlson, & Ekblom, 1988).

Methodological differences could account for the inconsistencies observed in the maximal voluntary eccentric torque-velocity findings between researchers but the differences between human and isolated animal muscle remain unclear. The hypothesis that some form of neural inhibition leading to a reduction in the number of active units used for torque production (Aagaard et al., 2000) is supported by research using external electrical stimulation of the muscle, since stimulation downstream from the spinal junction identifies a lack of full activation. In the leg extensors, superimposing electrical stimulation on a maximal voluntary eccentric contraction, evokes an eccentric torque increase of 10 – 40% (Dudley et al., 1990; Seger & Thorstensson, 2000; Westing et al., 1990) depending upon the training status of the subjects. It has been proposed that this phenomenon may originate from the Golgi tendon organs (Hortobagyi & Katch, 1990; Westing et al., 1990) but this has not been conclusively demonstrated. It is also known that increased feedback from cutaneous pain and/or joint receptors is involved in torque regulation (Houk & Henneman, 1967).

The test-retest reliability of the test protocol showed a coefficient of variation of 2·8 - 9·5% across each of the tested velocities. Griffin (1987) in a similar study, reported intraclass correlation coefficients of 0·72 - 0·80, proposing that the low reliability

may be due to the order of test contractions (concentric-eccentric), fatigue, lack of test familiarisation and incomplete stabilisation (subjects were supine). The influences of these factors were minimised in the protocol used for this study. Only eccentric test contractions were used, subject fatigue, if it occurred, was identified through the use of isometric contractions interspersed between eccentric velocities. This was only considered to be a substantial factor if output declined by greater than 10%. Test familiarisation was improved through the use of two trial arm extensions at each test velocity prior to measurement. Only the effect of incomplete stabilisation was not be controlled fully. The study used the preacher curl bench, where the position of the lever arm and the movement involved, encouraged the subject's shoulder and torso to be pulled down, and into the bench thus reducing body movement and only allowing movement about the elbow (figure 4). de Morton and Keating (2002) found that the use of a 65% pre-load of each subject's maximal voluntary isometric contraction significantly increased the reliability compared to one of a 5% pre-load. Although, in this investigation the elbow flexors were contracted prior to initiation of the eccentric movement, the isometric torque level was not controlled.

The length tension relationship predicts that the elbow angle for peak torque production should range between 90° to 110°, which has been demonstrated by Singh and Karpovich (1966). This investigation found an elbow angle where peak torque was developed to range between 75° to 77° (full extension was 180°). Specific isometric pre-loading prior to lengthening during testing may assist in increasing the angle at which peak torque is developed bringing it closer to that which would be expected from the length tension relationship.

The coefficient of variation for isometric MVC at 90° flexion between trials was 2·8% and did not change significantly over time (figure 7). On questioning, subjects reported little soreness following the testing, which, if experienced, lasted 2 –3 days post exercise. The isometric contractions were used to assess whether or not fatigue or damage occurred as a result of the test protocol. Mean isometric strength loss for both tests was approximately 8%, suggesting that, on average, fatigue and damage associated with the eccentric testing protocol were not significant factors.

5.2 Effect of Contraction Velocity on EIMD

The present investigation used an arm to arm comparison in the same subjects. This model has been demonstrated to be a powerful tool for the investigation of EIMD in various muscle groups (Child et al., 1998; Farr, Nottle, Nosaka, & Sacco, 2002; Nosaka & Newton, 2002b), since it minimises the effects of variation. The fact that all parameters were similar between the two groups at baseline prior to LVE or HVE excludes the contribution of inter-arm differences in the responses observed. Although all subjects were right hand dominant, potential effects of dominance were controlled through the use of a pseudo-random counter-balanced design. This ensured that arm dominance and order of testing velocities were equally balanced across exercise interventions.

Both LVE and HVE resulted in significant declines in all criterion measures except average surface EMG signal following exercise with the changes following HVE significantly greater than LVE in all strength measures. Moreover the time necessary for strength recovery was significantly longer following HVE. It should be noted the decrease in strength immediately after exercise was approximately 30% larger for HVE than LVE but minimal strength recovery occurred for either test velocity 30 minutes later. The reported deficits in strength and the time courses of recovery found after LVE and HVE are similar to those reported previously using this model of EIMD (Nosaka & Newton, 2002a, 2002d). The minimal recovery of strength parameters in the short-term indicates torque decrements resulted from muscle damage and not fatigue. This supports the findings of Ryschon, Fowler, Wysong, Anthony and Balaban (1997) who demonstrated that eccentric exercise had a lower metabolic cost than comparable concentric exercise.

The protracted decline in strength following HVE has significant implications for athletes participating in sports incorporating such high angular loadings. The decrement in isometric and dynamic strength which was still evident 7 days following the exercise bout highlights the importance of correct athlete tapering when high velocity loading is included in training programs. The effects of eccentric training may only be limited to strength decrements immediately following exercise as repeated bouts of eccentric loading imposed on previous EIMD does not seem to

inhibit strength recovery nor increase muscle soreness or blood borne muscle proteins (Chen & Hsieh, 2001). This is supported by the work of Paddon-Jones, Leveritt, Lonergan, and Abernethy (2001) who reported positive benefits of exclusive high velocity eccentric training following a ten week training program. The periodisation of training and the adaptive process of super-compensation have been well documented (Fry, Morton, & Keast, 1992; Kraemer et al., 2002). However, the effect on performance of the interaction between moderate to high velocity resistance training with other training stimuli is less clear and provides exciting potential for further research.

HVE likely involves greater contribution for torque generation from those motor units capable of developing attached cross bridges in the time available, vis a vis fast-twitch units. Thus, it seems reasonable to hypothesise that the greater decrements in measured strength variables were attributable to the fast twitch motor units being preferentially damaged. The HVE may have selectively damaged these fibres, but this can not be verified from the present data. Using rat, single muscle fibre segments, Macpherson, Schork et al. (1996) demonstrated that under comparable conditions, the size of the induced injury was greater to fast fibres compared to slow fibres. Presently invasive techniques (muscle biopsies) have been used to quantify fibre damage following eccentric exercise in human muscle. Quantification of disrupted fibres following eccentric exercise of the elbow flexors has been conducted by Stauber, Clarkson, Fritz and Evans (1990) and Gibala, MacDougall, Tarnopolsky, Stauber and Elorriaga (1995), but neither of these studies differentiated between fibre types. Alternatively examining the release of myosin heavy chains can be used to differentiate between disrupted fibres in a non-invasive manner (Sorichter et al., 2001). To identify whether the protocol in this research preferentially afflicted fast-twitch motor units, replication of the protocol with additional parameters of muscle biopsies using staining techniques (Vijayan et al., 2001) and/or through closer examination of myosin heavy chain isoforms which differ depending on the fibre classification.

The number of contractions necessary to reach 120 seconds of eccentric muscle tension may also have influenced the increased strength loss associated with HVE. An increased metabolic cost could be related to the number of contractions (see table

2), whereby susceptible fibres reach a state of fatigue or rigour and become more likely to be stretched past digitation and unable to re-digitate. A fatigued fibre state due to a higher metabolic cost is not supported by the present research as total rest to total time in contraction was higher in HVE protocol compared to LVE protocol (90 seconds between sets, LVE = 6 sets and HVE = 35 sets). In a study investigating the effect of contraction number on EIMD in the knee extensors utilising maximal voluntary contractions, Brown, Child, Day and Donnelly (1997) demonstrated no significant difference between the number of contractions when velocity was constant. To fully dismiss the effects of contraction number, it would be necessary to repeat the protocol and control the number of contractions performed under each condition.

Damage to the sarcotubular system, cytoskeleton, and contractile proteins is well known to result from eccentric exercise. Ingalls, Warren et al. (1998) suggested that the decline in strength following eccentric exercise is caused largely by E-C uncoupling for the first few days, and that longer term decrements result from disruption to the contractile elements. It is possible that the strength loss observed in this study following LVE and HVE could result from E-C coupling disruption, the extent of damage, as measured by torque decrement and protracted recovery, was smaller following LVE compared to HVE. This suggests that structural damage to contractile elements was less following LVE, and in the latter case, any fibre damage, did not lead to the affected fibres becoming necrotic (Armstrong, 1984).

The contrast in the degree of damage is well illustrated by the differences in plasma CK efflux following exercise, with HVE showing, on average, a 450% greater peak response (see figure 4.19) than LVE. Plasma CK did not significantly change following LVE which is in contrast to the literature where changes in plasma CK levels are reported to occur following novel and/or strenuous exercise with a peak 3 – 5 days post-exercise (Nosaka, Clarkson, & Apple, 1992) then a return to baseline values. Although similar to previous investigations (Clarkson & Ebbeling, 1988; Nosaka & Clarkson, 1996) exercise encompassing predominantly eccentric exercise results in an influx of CK and a large inter-individual variability, which was found following both HVE and LVE. The release of CK has been used to quatify the extent to which the exercised muscle has lost cell homeostasis. The change in plasma CK

after LVE showed evidence of a slight bi-modal response but at no stage was the response significantly elevated from the normal range. This would suggest that LVE resulted in no major loss of muscle cell homeostasis, which is in contrast with the significant and protracted loss of muscle strength observed. In contrast plasma CK efflux after HVE peaked 4 days post and continued to be inflated 7 days after exercise. This time course of elevation supports severe disruption to contractile elements as demonstrated by the large torque deficits present also at this time. The distinctly different plasma CK responses between LVE and HVE suggest that the number of fibres damaged after HVE is higher or, that of the fibres damaged, more have become degenerative.

The changes in muscle stiffness after LVE or HVE are distinct, with all subjects experiencing a considerable loss of ROM immediately following both exercise interventions. Changes in ROM are representative of an altered FANG and SANG, and both may contribute to different extents. Changes in FANG have been shown to correspond to torque loss and recovery (Clarkson & Tremblay, 1988). It has been postulated that a decreased FANG is related to connective tissue changes (Clarkson et al., 1992). Clarkson et al. (1992) hypothesised that alterations to FANG are attributed to over stretched sarcomeres no longer being able to form their maximal number of cross bridges and thus reducing the ability to shorten the muscle tendon unit. It was further postulated that changes in FANG may result from a Ca^{2+} deficiency in the SR where by inhibiting the cross bridge cycling process (Clarkson et al., 1992).

Spontaneous muscle shortening identified by changes in RANG was markedly different following LVE as compared to HVE. Although changes in RANG after LVE occurred, there was no easily identifiable peak and full recovery had occurred after 7 days. In comparison, immediately after HVE the angle became acute and continued to be exaggerated until the greatest change 3 days after exercise, RANG continued to be reduced 7 days later. Ebbeling and Clarkson (1989) proposed that changes to RANG occurred because of alterations to the connective tissue occurring due to an influx of Ca^{2+} which resulted from a loss of sarcolemmal integrity or SR dysfunction. This upset the balance between actin and myosin, causing involuntary contraction. Michaut, Pousson et al. (2001), however, found that eccentric exercise

did not modify the intrinsic properties of the series elastic component, indicating that factors other than structural dysfunction must contribute to the changes in RANG.

The larger changes in ROM and RANG following HVE suggests that either an increased number of fibres are altered or, that of the fibres altered, more are significantly degraded. With the evidence presented earlier this would support the notion that HVE results in a preferential damage to certain motor units. The changes associated with the ROM and RANG can not be conclusively identified from the present data. In order to quantify what initiates these changes it would be necessary to repeat this protocol and include examination of collagen breakdown (Brown, Day, & Donnelly, 1999). Using techniques such as changes to magnetic resonance image T_2 signal intensity for closer examination of alterations to contractile and/or connective tissue elements (Sorichter et al., 2001).

Another factor which may have contributed to an altered RANG are changes in limb circumference. Howell, Chila, Ford, David and Gates (1985) proposed that oedema resulting from eccentric exercise leads to changes in the perimuscular connective tissue, altering the elastic behaviour of the muscles and causing a reduction in motion. This mechanism is supported by the present study since changes in CIR were related to those in RANG. CIR was significantly elevated immediately after HVE and peak circumference occurred 72 hours post-exercise, which also followed the same time course as soreness and tenderness. The non-significant increases in CIR following LVE is in accord with the minimal changes observed in RANG. The smaller changes in CIR after HVE (averaging 8mm) are in contrast with previously reported increases in the elbow flexors of greater than 18mm (Murrayama, Nosaka, Yoneda, & Minamitani, 2000; Nosaka & Newton, 2002c).

All measures of soreness and tenderness were greater for HVE than LVE and had returned to normal by 7 days after exercise, but the pattern of recovery differed between exercise. Recovery occurred rapidly following LVE but the trend was shifted to the right (ie slowed) for HVE (figure 20 - 22). The peak in soreness and tenderness following LVE 2 days after exercise is a more commonly described response (McHugh et al., 1999; Rinard, Clarkson, Smith, & Grossman, 2000). Others have reported that following eccentric endurance exercise of the elbow

flexors soreness peaks 1 day after exercise (Nosaka et al., 2002b). The soreness findings in this study correlated well with other indicators of EIMD (e.g RANG). DOMS has been associated with the muscle inflammatory response measured by CIR (Murrayama et al., 2000). The data presented supports this relationship, as response time courses between CIR and measures of soreness and tenderness were similar in time to peak recordings.

5.3 Effect of Contraction Velocity during Eccentric Exercise

The pilot study conducted prior to the primary research demonstrated that elbow flexor MVC's at eccentric angular velocities of 30°, 90°, 150° and 210°·s^{-1} resulted in similar torque outputs (figure 6). Therefore it could be assumed that the torque generated by the elbow flexors would not be a mitigating factor in the resulting muscle damage incurred post exercise when the time under tension was constant.

The peak torque decrements for HVE period were similar to those observed utilising stimulated contractions (Nosaka et al., 2002c), where a sharp initial decline occurred followed by a period of little further reduction in peak torque. The peak mean torque production after 4 seconds of respective eccentric loading (ie 1-contraction verses 7-repeated contractions) for LVE and HVE respectively was similar for the two groups. However, over the duration of exercise, HVE resulted in a much greater loss in torque compared to LVE for the same relative time under tension. This could be explained by a reduced muscle activation but the EMG signal findings argue against this explanation, since there was no significant change in the size of the average rectified EMG signal recorded during the exercise period for either condition. Alternatively the size of the decrements experienced during HVE could be the result of groups of fibres selectively suffering from fatigue and thus no longer being able to generate torque as previously described by (Lieber et al., 1991). This would be consistent with the EMG signal that did not significantly decline from start to finish. However, seems unlikely, given that there was no significant recovery of strength 30 minutes following either exercise protocol. A mechano-chemical explanation would provide an argument that is consistent with the EMG signal data and strength deficits.

Although initial torque generation did not differ between velocities, the relative properties of motor units responsible for torque generation may differ. During LVE the subjects were required to maintain a maximal voluntary contraction for 4 seconds where as in HVE the maximal voluntary contraction lasted 0·57 seconds. LVE would allow for a larger number of units and thus cross bridges to share the production of the same relative amount of torque as that generated in a single contraction during HVE. If this were to be the case then it would be possible to assume that during HVE fewer fibres share the workload and thus, these fibres have a greater amount of strain placed upon them. This condition that has been identified as one that may lead to contractile structure damage (Lieber & Fridén., 1993). The site for this strain injury to contractile structures could be the thin and thick filament attachment position (Z line) and/or other structures necessary for force transfer and/or production.

Z-line streaming has previously been shown to result from novel eccentric exercise as has disruption to the sarcolemma and the myofibrils (Hortobagyi et al., 1998). A further potential further site for the investigation of EIMD is a costamere, the structure proposed to be necessary for lateral force transmission in skeletal muscle (Bloch & Gonzalez-Serratos, 2003). The costameres are proposed to act as mechanical attachments used to distribute contractile forces laterally through the sarcolemma (Danowski, Imanaka-Yoshida, Sanger, & Sanger, 1992), thereby facilitating uniform sarcomere length between fibres of active and non-active motor units (Rybakova, Patel, & Ervasti, 2000). As has been previously reported, the non-uniformity of sarcomeres is associated with a reduction in the force generation capacity of the muscle (Byrd, 1992). Investigations into whether costamere disruption occurs and how this interacts with other measures of stretched induced contractile injury may provide further evidence as to how force generation is increased in contractions involving stretches and whether this is a site for rapid adaptation. The costamere interaction with other measures of stretched induced injury could be investigated with the use of histological studies via muscle biopsies of the biceps brachii. Investigations into the costamere may also need to consider the interaction of desmin protein, which is critical for sarcomere integrity (Rybakova et al., 2000).

Brockett et al. (2002) postulated that torque decrement occurs due to a selective number of fibres being recruited first and that these fibres have a shorter optimum length. They reported that fast glycolytic fibres have a shorter optimum tension length as compared to fast oxidative glycolytic and slow oxidative fibres. The shorter length necessary for maximum tension of these fibres means that when stretched, their sarcomeres are the first to be stretched past digitation and are unable to re-digitate, they are "popped" and unable to generate torque (Morgan, 1990). Allen (2001) proposed that following stretching of the sarcomere a change in the force-length relationship occurs, shifting to the right when the imposed stretch is on the descending side of the force-length curve. Sarcomere stretching may involve disruption to the titin filament, anchor point of the myosin filaments to the Z discs, a necessary protein for force generation and transfer between sarcomeres (Lindstedt et al., 2001; Proske & Morgan, 2001). Closer examination of the titin protein during eccentric contractions may yield new information on the processes by which eccentric contractions are able to generate such high forces. Through the use of genetically modified rodents whereby specific markers are placed on the titin protein or the protein itself is modified, its involvement in eccentric contractions and the adaptations resulting from EIMD could be quantified. Lindstedt et al. (2001) has suggested that the role of titin in lengthening contractions may include cellular signal initiation for cross bridge enhancement and that differing titin isoforms may play a role in maintaining the amount of work a muscle is able to perform.

The size of strain placed on a fibre has been implicated in the level of induced muscle injury (Brooks & Faulkner, 2001; Lieber & Fridén., 1993; Lieber et al., 1991; Lynch & Faulkner, 1998). The strain imposed on a fibre may be related to the work absorbed by a muscle in eccentric contraction, but it is difficult to quantify from the data collected for this investigation. The two exercise interventions began with similar amounts of work absorbed in a single contraction as would be expected from the similar observed mean peak torques (figure 9). The total amount of work absorbed between the two interventions were vastly different with HVE resulting in approximately 7 times as much work absorbed which is a reflection of the number of contractions performed (ie the average torque times the distance moved). The HVE resulted in a greater relative loss in work absorbed by the contracting muscle upon completion of 120 seconds of muscle tension (figure 10). This could reflect the

increased level of strain placed on fewer cross bridges and/or the increased number of times contraction occurred. The number of times that the muscle contracted can be discounted as a factor in the greater decrement in relative work absorbed, because the amount of mean work absorbed was similar for the last contraction of each exercise intervention, an indication fatigue was not a factor in the observed differences.

The time for each contraction relative to the intervention is possibly very important. As fewer fibres would have the ability to reach full tension during HVE or fewer fibres would have the capacity to form strongly attached cross bridges during the contraction time even though muscle activation was comparable between LVE and HVE (table 2). Only faster acting cross bridges would be able to attach during the HVE and as such may be reflective of the duty cycle of the motor units. It has previously been hypothesised that in fatiguing conditions, the neural motor drive is organised in such a way that torque production is optimised as generation capabilities change (Michaut et al., 2001). Tesch, Dudley, Duvoisin et al. (1990) suggested that a de-recruitment of fatigued or damaged motor units alternating with the recruitment of fresher units occurs. The alternating of active motor units may be possible but cannot be verified from the data collected. Thus the larger work absorbed decrement and EIMD following HVE may result from fewer fibres having attached cross bridges and thus these cross bridges (fibres) are required to absorbed a greater proportion of the strain. Repeating the experiment and matching the work absorbed (number of repetitions) between investigated velocities instead of the time of muscle tension, is necessary to resolve some of these issues.

The susceptibility to EIMD of fast glycolytic (FG) fibres or fast oxidative glycolytic (FOG) fibres has previously been reported in studies using animal muscle and where the size of contraction was controlled (Lieber et al., 1991; Macpherson et al., 1996; Vijayan et al., 2001). In contrast findings from human muscle suggest that during voluntary eccentric contractions susceptibility to EIMD is not fibre type specific (Friden et al., 1983; O'Reilly et al., 1987; Sorichter et al., 2001). A biochemical explanation for the preferential damage to FG or FOG fibres was refuted by Patel et al. (1998) indicating that the explanation may be mechano-chemical. The findings from the current research provide anecdotal evidence that during maximal voluntary eccentric contractions, EIMD is fibre specific when the velocity of action is varied

and the time of muscle tension is constant. Testing of this hypothesis needs to be undertaken using muscle biopsies where the sample is immunostained with antifast myosin and antitotal myosin as described by Vijayan et al. (2001). Furthermore the investigated velocities need to be more reflective of movement patterns in the sporting arena, where velocities in excess of $500°\cdot sec^{-1}$ have been recorded (Elliott, Marshall, & Noffal, 1996; Zehr, Sale, & Dowling, 1997).

5.4 Conclusions

This research has demonstrated that faster eccentric contractions had a greater effect on the induced muscle injury than slow ones when time under tension was comparable. But in EIMD models that use maximum voluntary eccentric contractions of the elbow flexor, contraction velocity is a major determinant compared to the initial peak torque and needs to be considered when comparing responses to eccentric exercise.

The findings presented have provided evidence that higher velocity eccentric contractions require longer recovery periods and as such should be considered when structuring training programs. This would be highly recommended in the programming of stretch-shortening exercise (commonly known as plyometrics). When considered for use in the resistance training scenario, it may be feasible to structure workouts using super slow sets closer together, as the time needed for full recovery is less than higher velocity sets.

Further research is needed to fully understand the phenomenon of EIMD and the role of contraction velocity using maximal voluntary eccentric contractions. It should include, but not be limited to, investigations into whether slow contraction velocities provide protection to eccentric exercise at a substantially faster rate. This same experiment could be manipulated to have relevance to endurance athletes by investigating whether a bout of maximal slow velocity contractions protects against EIMD from a comparative bout of sub-maximal high velocity contractions? A comparison study is also needed between muscle groups (ideally a lower limb and an upper limb) performing a similar exercise task (time of muscle tension or work absorbed) and how do these relate to varying stretch velocities?

REFERENCES

Aagaard, P., Simonsen, E. B., Andersen, J. L., Magnusson, S. P., Halkjar-Kristensen, J., & Dyhre-Poulsen, P. (2000). Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. *Journal of Applied Physiology., 89*(6), 2249-2257.

Allaf, O., Goubel, F., & Marini, J. F. (2002). A curve-fitting procedure to explain changes in muscle force-velocity relationship induced by hyperactivity. *Journal of Biomechanics, 35*(6), 797-802.

Allen, D. G. (2001). Eccentric muscle damage: mechanisms of early reduction of force. [Review]. *Acta Physiologica Scandinavica., 171*(3), 311-319.

Armstrong, R. B. (1984). Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. *Medicine and Science in Sports and Exercise, 16*, 529-538.

Armstrong, R. B. (1990). Initial events in exercise-induced muscular injury. *Medicine and Science in Sports and Exercise, 22*, 429-435.

Bloch, R. J., & Gonzalez-Serratos, H. (2003). Lateral force transmission across costameres in skeletal muscle. *Exercise & Sport Sciences Reviews, 31*(2), 73-78.

Brockett, C. L., Morgan, D. L., Gregory, J. E., & Proske, U. (2002). Damage to different motor units from active lengthening of the medial gastrocnemius muscle of the cat. *Journal of Applied Physiology., 92*(3), 1104-1110.

Brooks, S. V., & Faulkner, J. A. (1994). Isometric, shortening, and lengthening contractions of muscle fibre segments from adult and old mice. *American Journal of Physiology, 267*, C507-C513.

Brooks, S. V., & Faulkner, J. A. (2001). Severity of contraction-induced injury is affected by velocity only during stretches of large strain. *Journal of Applied Physiology., 91*(2), 661-666.

Brown, S. J., Child, R. B., Day, S. H., & Donnelly, A. (1997). Exercise-induced skeletal muscle damage and adaptation following repeated bouts of eccentric muscle contractions. *Journal of Sports Sciences, 15*(2), 215-222.

Brown, S. J., Day, S. H., & Donnelly, A. E. (1999). Indirect evidence of human skeletal muscle damage and collagen breakdown after eccentric muscle actions. *Journal of Sports Sciences, 17*, 397-402.

Bryne, C., Eston, R. G., & Edwards, R. H. T. (2001). Characteristics of isometric and dynamic strength loss following eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine and Science in Sports, 11*(3), 134-140.

Byrd, S. K. (1992). Alterations in the sarcoplasmic reticulum: a possible link to exercise-induced muscle damage. *Medicine and Science in Sports and Exercise., 24*(5), 531-536.

Caldwell, G. E., Adams, W. B., III , & Whetstone, M. R. (1993). Torque-velocity properties of human knee muscles: Peak and angle specific estimates. *Canadian Journal of Applied Physiology., 18*(3), 274-290.

Chen, T. C., & Hsieh, S. S. (2000). The effects of repeated maximal voluntary isokinetic eccentric exercise on recovery from muscle damage. *Research Quarterly for Exercise and Sport, 71*(3), 260-266.

Chen, T. C., & Hsieh, S. S. (2001). Effects of a 7-day eccentric training period on muscle damage and inflammation. *Medicine and Science in Sports and Exercise, 33*(10), 1732-1738.

Child, R. B., Saxton, J. M., & Donnelly, A. E. (1998). Comparison of eccentric knee extensor muscle actions at two muscle lengths on indices of damage and angle-specific force production in humans. *Journal of Sports Sciences, 16*(4), 301-308.

Clarkson, P. M. (1992). Exercise-Induced muscle damage - Animal and Human models. *Medicine and Science in Sports and Exercise, 24*(5), 510-511.

Clarkson, P. M. (1997). Eccentric exercise and muscle damage. *International Journal of Sports Medicine., 18*(Supplement 4), S314-S317.

Clarkson, P. M., & Ebbeling, C. (1988). Investigation of serum creatine kinase variability after muscle-damaging exercise. *Clinical Science, 75*(3), 257-261.

Clarkson, P. M., Nosaka, K., & Braun, B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. *Medicine and Science in Sports and Exercise, 24*(5), 512-520.

Clarkson, P. M., & Tremblay, I. (1988). Exercise-induced muscle damage, repair and adaptation in humans. *Journal of Applied Physiology., 65*, 1-6.

Cope, T. C., & Pinter, M. J. (1995). The Size Principle: Still Working After All These Years. *News in Physiolgical Sciences, 10*, 280-286.

Cramer, J. T., Housh, T. J., Evetovich, T. K., Johnson, G. O., Ebersole, K. T., Perry, S. R., & Bull, A. J. (2002). The relationship among peak torque, mean power output, mechanomyography, and electromyography in men and women during maximal, eccentric isokinetic muscle actions. *European Journal of Applied Physiology, 86*, 226-232.

Danowski, B., Imanaka-Yoshida, K., Sanger, J., & Sanger, J. (1992). Costameres are sites of force transmission to the substratum in adult rat cardiomyocytes. *J. Cell Biol., 118*(6), 1411-1420.

Dop Bär, P. R., Reijneveld, J. C., Wokke, J. J. H., Jacobs, S. C. J. M., & Bootsma, A. L. (1997). Muscle damage induced by exercise: nature, prevention and repair. In S. Salmons (Ed.), *Muscle Damage*. Oxford: Oxford Medical Publications.

Dudley, G. A., Harris, R. T., Duvoisin, M. R., Hather, B. M., & Buchanan, P. (1990). Effect of voluntary vs. artificial activation on the relationship of muscle torque to speed. *Journal of Applied Physiology., 69*(6), 2215-2221.

Ebbeling, C. B., & Clarkson, P. M. (1989). Exercise-induced muscle damage and adaptation. *Sports Medicine., 7*, 207-234.

Elliott, B., Marshall, R., & Noffal, G. (1996). The role of upper limb segment rotations in the development of racket-head speed in the squash forehand. *Journal of Sports Sciences, 14*, 159-165.

Enoka, R. M. (1996). Eccentric contractions require unique activation strategies by the nervous system. *Journal of Applied Physiology., 81*(6), 2339-2346.

Farr, T., Nottle, C., Nosaka, K., & Sacco, P. (2002). The effects of therapeutic massage on delayed onset muscle soreness and muscle function following downhill walking. *Journal of Science and Medicine in Sport., 5*(4), 297-306.

Foley, J. M., Jayaraman, R. C., Prior, B. M., Pivarnik, J. M., & Meyer, R. A. (1999). MR measurements of muscle damage and adaptation after eccentric exercise. *Journal of Applied Physiology., 87*, 2311-2318.

Fridén, J., & Lieber, R. L. (1992). Structural and mechanical basis of exercise-induced muscle injury. *Medicine and Science in Sports and Exercise., 24*, 521-530.

Fridén, J., & Lieber, R. L. (2001). Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components. *Acta Physiologica Scandinavica., 171*(3), 321-326.

Friden, J., Sjostrom, M., & Ekblom, B. (1983). Myofibrillar damage following intense eccentric exercise in man. *International Journal of Sports Medicine, 4*(3), 170-176.

Fry, R. W., Morton, A. R., & Keast, D. (1992). Periodisation of Training Stress - A review. *Canadian Journal of Applied Physiology., 17*(3), 234-240.

Gibala, M. J., MacDougall, J. D., Tarnopolsky, M. A., Stauber, W. T., & Elorriaga, A. (1995). Changes in human skeletal muscle ultrastructure and force production after acute resistance exercise. *Journal of Applied Physiology., 78*(2), 702-708.

Gordon, A. M., Homsher, E., & Regnier, M. (2000). Regulation of contraction in striated muscle. *Physiological Reviews, 80*(2), 853-924.

Gordon, A. M., Regnier, M., & Homsher, E. (2001). Skeletal and Cardiac Muscle Contractile Activation: Tropomyosin "Rocks and Rolls". *News in Physiological Sciences, 16*(2), 49-55.

Gregor, R. J., Edgerton, V. R., Perrine, J. J., Campion, D. S., & DeBus, C. (1979). Torque velocity relationships and muscle fibre composition in elite female athletes. *Journal of Applied Physiology., 47*(2), 388-392.

Griffin, J. W. (1987). Differences in elbow flexion torque measured concentrically, eccentrically and isometrically. *Physical Therapy, 67*(8), 1205-1208.

Griffin, J. W., Tooms, R. E., Vander Zwaag, R., Bertorini, T. E., & O'Toole, M. L. (1993). Eccentric muscle performance of elbow and knee muscle groups in untrained men and women. *Medicine and Science in Sports and Exercise, 25*(8), 936-944.

Gulch, R. W. (1994). Force - velocity relations in human skeletal muscle. *International Journal of Sports Medicine, 15*(Supplement 1), S2-S10.

Hermens, H. J., Freriks, B., Disselhorst-Klug, C., & Rau, G. (2000). Development of recommendations for SEMG sensors and sensor placement procedures. *Journal of Electromyography and Kinesiology, 10*(5), 361-374.

Herzog, W. (2000). Considerations on the theoretical modelling of skeletal muscle contraction. In W. Herzog (Ed.), *Skeletal Muscle Mechanics*. West Sussex: John Wiley & Sons, Ltd.

Herzog, W., & Leonard, T. R. (2002). Force enhancement following stretching of skeletal muscle: a new mechanism. *The Journal of Experimental Biology, 205*(9), 1275-1283.

Hortobagyi, T., Houmard, J., Fraser, D., Dudek, R., Lambert, J., & Tracy, J. (1998). Normal forces and myofibrillar disruption after repeated eccentric exercise. *Journal of Applied Physiology., 84*, 492-498.

Hortobagyi, T., & Katch, F. I. (1990). Eccentric and concentric torque velocity relationships during arm flexion and extension. *European Journal of Applied Physiology and Occupational Physiology, 60*(5), 395-401.

Houk, J., & Henneman, E. (1967). Responses of Golgi tendon organs to active contractions of the soleus muscle of the cat. *Journal of Neurophysiology, 30*(3), 466-481.

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

Ingalls, C. P., Warren, G. L., Williams, J. H., Ward, C. W., & Armstrong, R. B. (1998). E-C coupling failure in mouse EDL muscle after in vivo eccentric contractions. *Journal of Applied Physiology., 85*, 58-67.

Irving, M., & Piazzesi, G. (1997). Motions of myosin heads that drive muscle contraction. *News in Physiological Sciences, 12*(6), 249-254.

Janssen, G. M. E., Kuipers, H., Willems, G. M., Does, R. J. M. M., Janssen, M. P. E., & Geurten, P. (1989). Plasma activity of muscle enzymes: Quantification of skeletal muscle damage and relationship with metabolic variables. *International Journal of Sports Medicine, 10*(Supplement 3), S160-S168.

Kannus, P. (1994). Isokinetic evaluation of muscular performance: implications for muscle testing and rehabilitation. *International Journal of Sports Medicine, 15*(Supplement 1), S11-S18.

Kasprisin, J. E., & Grabiner, M. D. (2000). Joint angle-dependence of elbow flexor activation levels during isometric and isokinetic maximum voluntary contractions. *Clinical Biomechanics, 15*(10), 743-749.

Katz, B. (1939). The relation between force and speed in muscular contraction. *Journal of Physiology (London), 96*, 45-64.

Kendall, B., & Eston, R. G. (2002). Exercise-induced muscle damage and the potential protective role of estrogen. *Sports Medicine, 32*(2), 103-123.

Komi, P. V., Linnamo, V., Silventoinen, P., & Sillanpaa, M. (2000). Force and EMG power spectrum during eccentric and concentric actions. *Medicine and Science in Sports and Exercise., 32*(10), 1757-1762.

Kraemer, W. J., Adams, K., Cafarelli, E. D., Dudley, G. A., Dooly, C., Feigenbaum, M. S., Fleck, S. J., Franklin, B., Fry, A. C., Hoffman, J. R., Newton, R. U., Jeffrey Potteiger, J., Stone, M. H., Ratamess, N. A., & Triplett-McBride, T. (2002). American College of Sports Medicine Position Stand: Progression

models in resistance training for healthy adults. *Medicine & Science in Sports & Exercise, 34*(2), 364-380.

Kuipers, H. (1994). Exercise-induced muscle damage. *International Journal of Sports Medicine, 15*(3), 132-135.

Kulig, K., Power, C. M., Shellock, F. G., & Terk, M. (2001). Effects of eccentric velocity on activation of elbow flexors: evaluation by MRI. *Medicine and Science in Sports and Exercise., 33*(2), 196-200.

Lee, J., Goldfarb, A. H., Rescino, M. H., Hegde, S., Patrick, S., & Apperson, K. (2002). Eccentric exercise effect on blood oxidative-stress markers and delayed onset of muscle soreness. *Medicine and Science in Sports and Exercise, 34*(3), 443-448.

Lieber, R. L., & Fridén, J. (2002). Mechanisms of muscle injury gleaned from animal models. *American Journal of Physical Medicine and Rehabilitation, 81*(11), S70-S79.

Lieber, R. L., & Fridén., J. (1993). Muscle damage is not a function of muscle force but active muscle strain. *Journal of Applied Physiology., 74*, 520-526.

Lieber, R. L., Woodburn, T. M., & Fridén, J. (1991). Muscle damage induced by eccentric contractions of 25% strain. *Journal of Applied Physiology., 70*(6), 2498-2507.

Lindstedt, S. L., LaStayo, P. C., & Reich, T. E. (2001). When Active Muscles Lengthen: Properties and Consequences of Eccentric Contractions. *News in Physiological Sciences, 16*(6), 256-261.

Linnamo, V., Bottas, R., & Komi, P. V. (2000). Force and EMG power spectrum during and after eccentric and concentric fatigue. *Journal of Electromyography and Kinesiology, 10*(5), 293-300.

Lynch, G. S., & Faulkner, J. A. (1998). Contraction-induced injury to single muscle fibers: velocity of stretch does not influence the force deficit. *American Journal of Physiology: Cell Physiology, 275*(6), C1548-1554.

Macpherson, P. C., Schork, M. A., & Faulkner, J. A. (1996). Contraction-induced injury to single fiber segments from fast and slow muscles of rats by single stretches. *Am J Physiol Cell Physiol, 271*(5), C1438-1446.

Malm, C. (2001). Exercise-induced muscle damage and inflammation: fact or fiction. *Acta Physiologica Scandinavica., 171*, 233-239.

McArdle, A., & Jackson, M. J. (1997). Intracellular mechanisms involved in skeletal muscle damage. In S. Salmons (Ed.), *Muscle Damage*. Oxford: Oxford Medical Publications.

McCully, K. K., & Faulkner, J. A. (1986). Characteristics of lengthening contractions associated with injury to skeletal muscle fibers. *Journal of Applied Physiology., 61*(1), 293-299.

McHugh, M. P., Connolly, D. A., Eston, R. G., & Gleim, G. W. (2000). Electromyographic analysis of exercise resulting in symptoms of muscle damage. *Journal of Sports Sciences, 18*(3), 163-172.

McHugh, M. P., Connolly, D. A., Eston, R. G., Kremenic, I. J., Nicholas, S. J., & Gleim, G. W. (1999). The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. *American Journal of Sports Medicine, 27*(5), 594-599.

Michaut, A., Pousson, M., Ballay, Y., & Van Hoeche, J. (2001). Short-term changes in the series elastic component after acute eccentric exercise of the elbow flexors. *European Journal of Applied Physiology, 84*(6), 569-574.

Morgan, D. (1990). New insights into the behavior of muscle during active lengthening. *Biophysical Journal, 57*(2), 209-221.

Morgan, D. L., & Allen, D. G. (1999). Early events in stretch-induced muscle damage. *Journal of Applied Physiology., 87*, 2007-2015.

de Morton, N. A., & Keating, J. L. (2002). The effect of preload on variability in dynamometric measurements of knee extension. *European Journal of Applied Physiology, 86*, 355-362.

Murrayama, M., Nosaka, K., Yoneda, T., & Minamitani, K. (2000). Changes in hardness of the human elbow flexor muscles after eccentric exercise. *European Journal of Applied Physiology, 82*(5-6), 361-367.

Nosaka, K., & Clarkson, P. M. (1996). Variability in serum creatine kinase response after eccentric exercise of the elbow flexors. *International Journal Sports Medicine, 17*, 120-127.

Nosaka, K., Clarkson, P. M., & Apple, F. S. (1992). Time course of serum protein changes after strenuous exercise of the forearm flexors. *Journal of Laboratory & Clinical Medicine, 119*(2), 183-188.

Nosaka, K., & Newton, M. (2002a). Concentric or eccentric training effect on eccentric exercise-induced muscle damage. *Medicine and Science in Sports and Exercise, 34*(1), 63-69.

Nosaka, K., & Newton, M. (2002b). Difference in the magnitude of muscle damage between maximal and submaximal eccentric loading. *Journal of Strength and Conditioning Research, 16*(2), 202-208.

Nosaka, K., & Newton, M. (2002c). Is recovery from muscle damage retarded by a subsequent bout of eccentric exercise inducing larger decreases in force? *Journal of Science and Medicine in Sport, 5*(3), 204-218.

Nosaka, K., & Newton, M. (2002d). Repeated eccentric exercise bouts do not exacerbate muscle damage and repair. *Journal of Strength and Conditioning Research, 16*(1), 117-122.

Nosaka, K., Newton, M., & Sacco, P. (2002a). Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine and Science in Sports, 12*, 337-346.

Nosaka, K., Newton, M., & Sacco, P. (2002b). Muscle damage and soreness after endurance exercise of the elbow flexors. *Medicine and Science in Sports and Exercise, 34*(6), 920-927.

Nosaka, K., Newton, M., & Sacco, P. (2002c). Response of human elbow flexor muscles to electrically stimulated force lengthening exercise. *Acta Physiologica Scandinavica., 174*(2), 137-145.

Nosaka, K., Sakamoto, K., Newton, M., & Sacco, P. (2001). How long does the protective effect on eccentric exercise-induced muscle damage last? *Medicine and Science in Sports and Exercise, 33*(9), 1490-1495.

O'Reilly, K. P., Warhol, M. J., Fielding, R. A., Frontera, W. R., Meredith, C. N., & Evans, W. J. (1987). Eccentric exercise-induced muscle damage impairs muscle glycogen replet.. *Journal of Applied Physiology., 63*(1), 252-256.

Paddon-Jones, D., Leveritt, M., Lonergan, A., & Abernethy, P. (2001). Adaptation to chronic eccentric exercise in humans: the influence of contraction velocity. *European Journal of Applied Physiology, 85*(5), 466-471.

Patel, T. J., Cuizon, D., Mathieu-Costello, O., Friden, J., & Lieber, R. L. (1998). Increased oxidative capacity does not protect skeletal muscle fibers from eccentric contraction-induced injury. *Am J Physiol Regul Integr Comp Physiol, 274*(5), R1300-1308.

Pizza, F. X., Koh, T. J., McGregor, S. J., & Brooks, S. V. (2002). Muscle inflammatory cells after passive stretches, isometric contractions, and lengthening contractions. *Journal of Applied Physiology., 92*(5), 1873-1878.

Pollack, G. H. (1983). The cross-bridge theory. *Physiological Reviews, 63*(3), 1049-1113.

Proske, U., & Morgan, D. L. (2001). Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *Journal of Physiology., 537*(2), 333-345.

Pyne, D. B. (1994). Exercise-induced muscle damage and inflammation: A review. *The Australian Journal of Science and Medicine in Sport, 26*(3/4), 49-58.

Rinard, J., Clarkson, P. M., Smith, L. L., & Grossman, M. (2000). Response of males and females to high-force eccentric exercise. *Journal of Sports Sciences, 18*, 229-236.

Rodenburg, J. B., Bar, P. R., & De Boer, R. W. (1993). Relations between muscle soreness and biochemical and functional outcomes of eccentric exercise. *Journal of Applied Physiology., 74*(6), 2976-2983.

de Ruiter, C. J., & de Haan, A. (2001). Similar effects of cooling and fatigue on eccentric and concentric force-velocity relationships in human muscle. *Journal of Applied Physiology., 90*, 2109-2116.

Rybakova, I. N., Patel, J. R., & Ervasti, J. M. (2000). The Dystrophin Complex Forms a Mechanically Strong Link Between the Sarcolemma and Costameric Actin. *Journal of Cell Biology, 150*(5), 1209-1214.

Ryschon, T. W., Fowler, M. D., Wysong, R. E., Anthony, A.-R., & Balaban, R. S. (1997). Efficiency of human skeletal muscle in vivo: comparison of isometric, concentric, and eccentric muscle action. *Journal of Applied Physiology., 83*(3), 867-874.

Sayers, S. P., & Clarkson, P. M. (2001). Force recovery after eccentric exercise in males and females. *European Journal of Applied Physiology, 84*(1-2), 122-126.

Sayers, S. P., Clarkson, P. M., & Lee, J. (2000a). Activity and immobilization after eccentric exercise: 1. Recovery of muscle function. *Medicine and Science in Sports and Exercise, 32*(9), 1587-1592.

Sayers, S. P., Clarkson, P. M., & Lee, J. (2000b). Activity and immobilization after eccentric exercise: 2.Serum CK. *Medicine and Science in Sports and Exercise, 32*(9), 1593-1597.

Sbriccoli, P., Felici, F., Rosponi, A., Aliotta, A., Castellano, V., Mazza, C., Bernardi, M., & Marchetti, M. (2001). Exercise induced muscle damage and recovery assessed by means of linear and non-linear sEMG analysis and ultrasonography. *Journal of Electromyography and Kinesiology, 11*(2), 73-83.

Seger, J. Y., & Thorstensson, A. (2000). Electrically evoked eccentric and concentric torque-velocity relationships in human knee extensor muslces. *Acta Physiologica Scandinavica., 169*, 63-69.

Sieck, G. C., & Regnier, M. (2001). Plasticity in Skeletal, Cardiac, and Smooth Muscle: Invited Review: Plasticity and energetic demands of contraction in skeletal and cardiac muscle. *Journal of Applied Physiology., 90*(3), 1158-1164.

Singh, M., & Karpovich, P. V. (1966). Isotonic and isometric forces of forearm flexors and extensors. *Journal of Applied Physiology., 21*(4), 1435-1437.

Smith, L. L. (1991). Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Medicine and Science in Sports and Exercise, 23*(5), 542-551.

Sorichter, S., Mair, J., Koller, A., Muller, E., Kremser, C., Judmaier, W., Haid, C., Calzolari, C., & Puschendorf, B. (2001). Creatine kinase, myoisn heavy chains and magnetic resonance imaging after eccentric exercise. *Journal of Sports Sciences, 19*, 687-691.

Sorichter, S., Puschendorf, B., & Mair, J. (1999). Skeletal muscle injury induced by eccentric muscle action: Muscle proteins as markers of muscle fiber injury. *Exercise Immunology Review, 5*, 5-21.

Stauber, W. T., Clarkson, P. M., Fritz, V. K., & Evans, W. J. (1990). Extracellular matrix disruption and pain after eccentric muscle action. *Journal of Applied Physiology., 69*(3), 868-874.

Talbot, J. A., & Morgan, D. L. (1998). The effects of stretch parameters on eccentric exercise-induced damage to toad skeletal muscle. *Journal of Muscle Research Cell Motility, 19*, 237-245.

Tesch, P. A., Dudley, G. A., Duvoisin, M. R., Hather, B. M., & Harris, R. T. (1990). Force and EMG signal patterns during repeated bouts of concentric or eccentric muscle actions. *Acta Physiologica Scandinavica., 138*, 263-271.

Thayer, R. E., Rice, C. L., Pettigrew, F. P., Noble, E. G., & Taylor, A. W. (1993). The fibre composition of skeletal muscle. In J. R. Poortmans (Ed.), *Principles of Exercise Biochemistry, 2nd, rev ed.* (2nd ed., Vol. 38, pp. 25-50): Medicine and Sport Science.

Vijayan, K., Thompson, J. L., Norenberg, K. M., Fitts, R. H., & Riley, D. A. (2001). Fiber-type susceptibility to eccentric contraction-induced damage of hindlimb-unloaded rat AL muscles. *Journal of Applied Physiology., 90*(3), 770-776.

Warren, G. L., Hayes, D. A., Lowe, D. A., & Armstrong, R. B. (1993). Mechanical factors in the initiation of eccentric contraction-induced injury in rat soleus muscle. *Journal of Physiology, 464*, 457-475.

Warren, G. L., Hermann, K. M., Ingalls, C. P., Masselli, M. R., & Armstrong, R. B. (2000). Decreased EMG median frequency during a second bout of eccentric contractions. *Medicine and Science in Sports and Exercise, 32*(4), 820-829.

Warren, G. L., Ingalls, C. P., Lowe, D. A., & Armstrong, R. B. (2001). Excitation-contraction uncoupling: major role in contraction-induced muscle injury. *Exercise and Sport Sciences Reviews, 29*(2), 82-87.

Warren, G. L., Lowe, D. A., & Armstrong, R. B. (1999). Measurement tools used in the study of eccentric contraction-induced injury. *Sports Medicine, 27*, 43-59.

Webber, S., & Kriellaars, D. (1997). Neuromuscular factors contributing to in vivo eccentric moment generation. *Journal of Applied Physiology., 83*(1), 40-45.

Westing, S. H., Seger, J. Y., Karlson, E., & Ekblom, B. (1988). Eccentric and concentric torque-velocity characteristics of the quadriceps femoris in man. *European Journal of Applied Physiology, 58*, 100-104.

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

Whitehead, N. P., Morgan, D. L., Gregory, J. E., & Proske, U. (2003). Rises in whole muscle passive tension of mammalian muscle after eccentric contractions at different lengths. *Journal of Applied Physiology., 95*(3), 1224-1234.

Willems, M. E. T., & Stauber, W. T. (2000). Performance of plantar flexor muscles with eccentric and isomeric contractions in intact rats. *Medicine and Science in Sports and Exercise, 32*(7), 1293-1299.

Willems, M. E. T., & Stauber, W. T. (2002). Force deficits by stretch of activated muscles with constant or increasing velocity. *Medicine and Science in Sports and Exercise, 34*(4), 667-672.

Wood, S. A., Morgan, D. L., & Proske, U. (1993). Effects of repeated eccentric contractions on structure and mechanical properties of toad sartorius muscle. *American Journal of Physiology: Cell Physiology, 265*(3), C792-800.

Zehr, E. P., Sale, D., & Dowling, J. J. (1997). Ballistic movement performance in karate athletes. *Medicine and Science in Sports and Exercise, 29*(10), 1366-1373.

APPENDIX A: Informed Consent

Informed Consent Form

For the study

The Effect of Eccentric Exercise Velocity on Selected Measures of Muscle Function and Soreness of the Human Elbow Flexors in Untrained Males and Females

Thankyou for expressing an interest in my research. The reason for providing you with the following information is to fully inform you of the purpose and nature of the study.

Purpose of the study

The objective of this study is to investigate whether movement velocity significantly effects the selected measures of muscle function and soreness in the elbow flexors when exercise is performed eccentrically.

Exercise and Measurements

If you agree to participate in this study you will be asked to attend the laboratory on twelve separate occasions. The first occasion will be two days prior to the first exercise session. This initial session will be used to familiarise you with 1) the testing and exercising equipment, 2) the testing and exercising procedures to be used in the study and thirdly to record some base line data to indicate your individual starting point. The actual exercise and testing will take place over two seven-day blocks, with a three-day break between the two blocks. Several measurements will be taken two days before and immediately prior to the exercise session, 30 minutes after the exercise, and then 1, 2, 3, 4 and 7 days following exercise. On each of these occasions we will also require your approval to take a small sample of blood from your fingertip for analysis of an enzyme called creatine kinase. The day of the exercise session will take approximately two and a half-hours, and approximately 30 minutes for each of the remaining days of the block. The exercise and measurements will take place at a sports science research laboratory located on the Edith Cowan University Joondalup campus, room 19·150.

Exercise: You will be asked to perform your exercise task on a machine known as a Cybex 6000 isokinetic dynamometer. Your upper arm will be resting on the arm support of a preacher curl bench forming a 45° angle with the trunk of your body. Your wrist will be secured to the pad of a lever arm, which will cause your elbow joint to form an angle of 60°, which will be the starting position of the exercise. During the exercise sessions the lever arm will be driven in a downward motion at either a speed of $30°\cdot sec^{-1}$ or $210°\cdot sec^{-1}$ by the motor of the Cybex, forcing the arm angle to extend to a position of 180 degrees. You will be verbally encouraged to maximally resist the motion of the lever arm and thereby produce what is referred to as a "maximal voluntary contraction" of the elbow flexor muscles. The lever arm and therefore your arm will be returned to the starting position by the Cybex during which time you will be requested to "relax and let the machine move your arm back

to the starting position". Exercise will consist of sets of 6 maximal repetitions, with a 90-second rest between sets.

Measurements: The following measurements will be taken from the exercised arm.

Maximal isometric torque: Maximal voluntary isometric torque of the elbow flexors at an elbow angle of 90° and 150° will be measured twice, for 4 seconds each, using the Cybex dynamometer and a preacher curl bench.
Maximal dynamic torque: Maximal voluntary torque of the elbow flexors will be measured concentrically and eccentrically through a set range of motion (90°). The concentric contractions will be at 30°, 90°, 150° and 210°·sec^{-1}. Eccentric contractions will be performed at velocities of 30°·sec^{-1} and 210°·sec^{-1}. Two attempts will be made at each velocity using the Cybex dynamometer and a preacher curl bench.
Electromyography: The electrical activity of one of the elbow flexor muscles (biceps brachii) will be recorded during the above measures of torque on the day of exercise and on day 7. Three removable surface electrodes will be placed onto your skin and connected to a signal acquisition device. To obtain consistent measurements, three marks will be placed on the skin by a semi-permanent ink marker.
Plasma creatine kinase activity: Approximately 30μl of blood will be collected into a capillary tube following the piercing of a selected finger with a spring-loaded lancet. The blood will be immediately assessed by a spectrophotometer for plasma creatine kinase concentration.
Range of motion: your elbow joint angle will be measured by an investigator using a plastic goniometer when you, in a standing position, try to fully flex the elbow by touching your shoulder with the palm, try to straighten the elbow joint, and relax your arm at your side. To ensure the measurements remain consistent, three marks will be placed on the skin by a semi-permanent ink marker.
Upper arm circumference: Circumference will be assessed by a constant tension tape measure at three sites on your upper arm (3, 7 and 11cm from the elbow crease) when you relax and let arm hang by your side. In order to keep the measurements consistent the three sites will be marked on the skin by semi-permanent ink marker.
Muscle soreness: This will be assessed by palpating the selected elbow flexor muscles (primarily the biceps brachii) at a number of sites, and forcibly extending and flexing the elbow joint, during which time you the subject will be asked to mark your level of discomfort using a visual analog scale (VAS) of a 100mm line (0: no pain, 100: very painful).

·Risk and Ethical considerations

You may experience some degree of muscle soreness and decreases in muscle function, such as muscle strength and range of motion, in the days following the exercises. You may also experience swelling of the upper arm and forearm. these symptoms are often seen after unaccustomed exercise containing eccentric muscle actions, and will disappear in a week or so.
You will experience some transient discomfort when a lancet pierces your finger during the process of blood sampling for creatine kinase analysis. Since blood is withdrawn by an experienced researcher in accordance with a safety manual of blood sampling, risk for infections or injury are negligible. Other measurements employed in the study are risk free.

No direct comparisons between different individuals participating in the study will be made at any stage of the testing. Analysis of the data will be made on a group basis with means and variance between groups compared. You are therefore not in competition with any other individuals in the study and will in no way be made to feel as if your results are inadequate or incorrect.
All personal information and test results recorded will remain confidential and will not be used for any other purpose other than the current study. Moreover, no data analysis will include your name or information that may identify you as the specific subject involved.
You will be free to withdraw from this study at any stage and for any reason without prejudice.

Requirements

As the study is aimed at assessing any changes that may occur across a period of time, you will be requested not to perform any unaccustomed exercises or sporting activities, not to take any anabolic steroids, anti-inflammatory drugs or nutritional supplements, and not to alter your diet and lifestyle (sleeping habits etc) that may influence your results during the experimental periods.
Additional, as the study involves an exercise protocol, it is requires that you be healthy at the time of testing. For this reason, you will be asked to complete a medical questionnaire prior to the commencement of testing.

Should you have any questions relating to any of the information provided above, please feel free to contact me for further explanation. If you have any concerns about this research, or would just like to speak to an independent person, you may contact the Head of our school, Assoc Prof. Barry Gibson on telephone (9400 5037).

Thankyou very much for your cooperation and contribution to the study.

Yours Sincerely,

Dale Chapman B.Sci (MSc. candidate)
School of Biomedical and Sports Science, Edith Cowan University
100 Joondalup Drive, Joondalup WA 6027
Phone: 9400 5159 E-mail: d.chapman@ecu.edu.au

APPENDIX B: Medical Questionnaire

Medical Questionnaire

The following questionnaire is designed to establish a background of your medical history, and identify any injury and or illness that may influence your testing and performance.
Please answer all questions as accurately as possible and if you are unsure about any thing please ask for clarification. All information provided is strictly confidential. If you answer yes to any non-exercise related question that may contraindicate you from completing this study a clearance from a qualified medical practitioner will be required prior to commencement of any exercising or testing.

Personal Details
Name: _____ Subject Code: _____

Date of Birth (D/M/Y): _____ Gender: Female Male

Medical History
Have you ever had, or do you currently have any of the following?

			If YES, please provide details
High or abnormal blood pressure	Y	N	_____
High cholesterol	Y	N	_____
Rheumatic fever	Y	N	_____
Heart abnormalities	Y	N	_____
Asthma	Y	N	_____
Diabetes	Y	N	_____
Epilepsy	Y	N	_____
Recurring back pain	Y	N	_____
Recurring neck pain	Y	N	_____
Severe allergies	Y	N	_____
Any infectious diseases	Y	N	_____
Any neurological disorders	Y	N	_____
Any neuromuscular disorders	Y	N	_____

If YES, please provide details

Are you currently on any medications?	Y	N	_____
Have you had the flu in the last two weeks?	Y	N	_____
Have recently injured yourself?	Y	N	_____
Do you have any recurring muscle or joint injuries?	Y	N	_____
Have you had any elbow or shoulder problems in the past?	Y	N	_____
Have you participated in resistance training in the last six months?	Y	N	_____
Is there any other condition not previously mentioned which may affect your upper arm exercise?	Y	N	_____

Lifestyle Habits

Do you exercise regularly? Y N
If YES, what do you do?

_____

How many hours per week?

_____

Do you smoke tobacco? Y N
If YES, how much per day?

_____

Do you consume alcohol? Y N
If YES, how much per week?

_____

Do you consume tea or coffee?
If YES, how many cups per day?

_____

Declaration

I acknowledge that the information provided on this form is to the best of my knowledge, a true and accurate indication of my current state of health.

Participant

Name: _____
Date (DD/MM/YYYY): _____

APPENDIX C: Ethical Clearance

EDITH COWAN UNIVERSITY

MOUNT LAWLEY CAMPUS

2 Bradford Street,
Mount Lawley
Western Australia 6050
Telephone (08) 9370 6111
Facsimile (08) 9370 2910

ABN 54 361 485 361

2002 futures

4th December, 2002

Mr Dale Chapman
[redacted]

Dear Mr Chapman,

It is with pleasure that I write on behalf of the Faculty of Communication, Health & Science, Higher Degrees Committee to advise you that your Master's research proposal has been approved – *The effect of eccentric exercise velocity on selected measures of muscle function and soreness of the human elbow flexors in untrained males and females.*

I am pleased to advise that your proposal complies with the provisions contained in the University's policy for the conduct of ethical research, and your application for ethics clearance has been approved. Your ethics approval number is 02-143 and period of approval is 18th September, 2002 to 31st December, 2003. A copy of the Conditions of Approval is attached. You may now commence your data collection

Approval is given for your supervisory team to consist of:

Principal Supervisor: Dr Paul Sacco

The examination requirements on completion are laid down in *Part 4* of *The University (Admissions, Enrolment and Academic progress) Rules for Courses Requiring the Submission of Theses* (contact office if you require a copy) or check the web at: www.cowan.edu.au/secretariat/unical/rules/aprcont.htm.

Additional information and documentation relating to the examination process can be found at our web site: www.cowan.edu.au/research/gsmain.html.

Please note:

1. The Research Students and Scholarship Committee has resolved to restrict Master theses to a maximum of 70,000 words with a provision that under special circumstances a candidate may seek approval from the Faculty Research and Higher Degrees Committee for an extension to the word length. **[RSSC 99/24]**

Finally, could I take this opportunity to offer you our best wishes for your research and the development of your thesis.

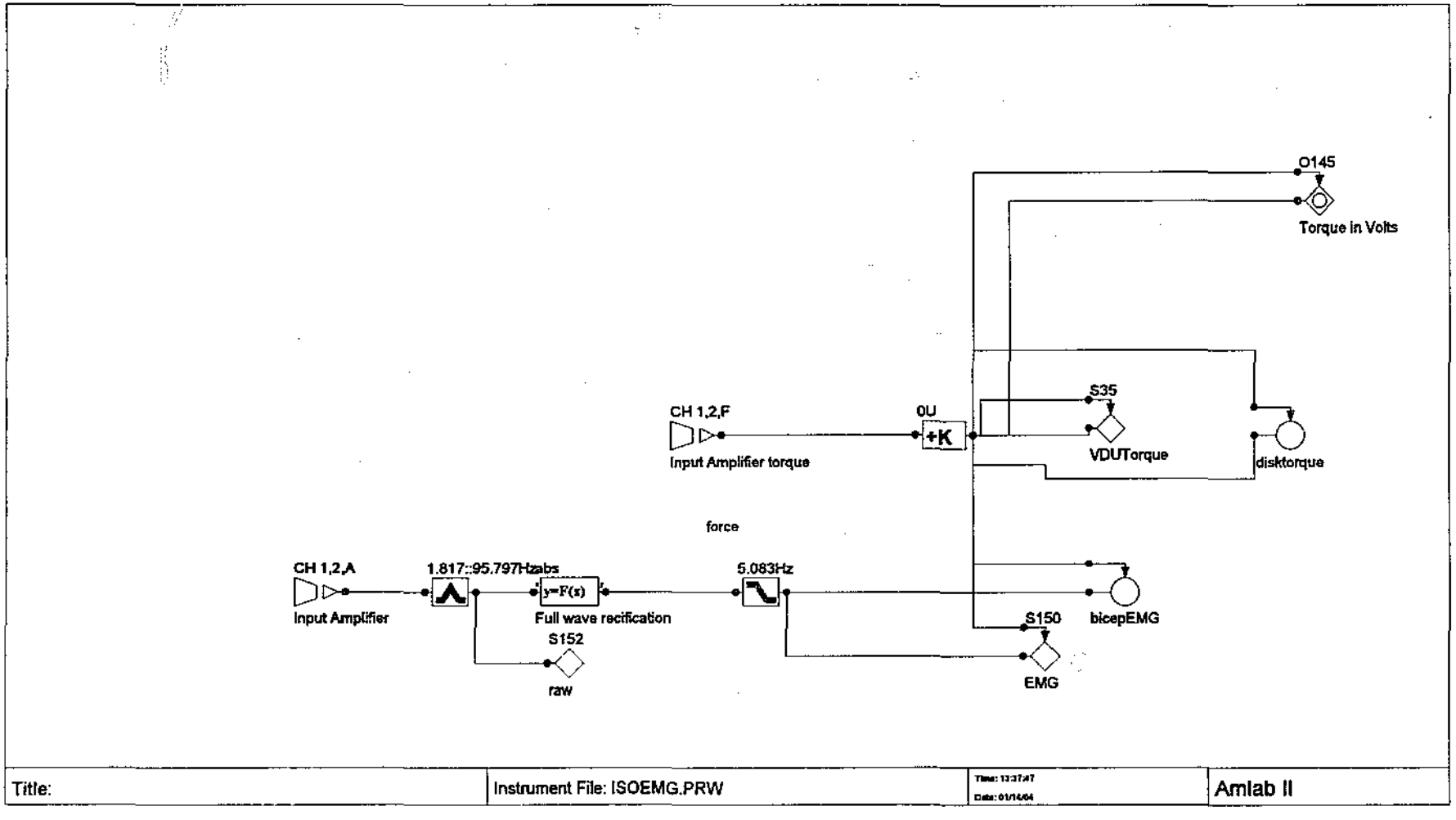
Sincerely

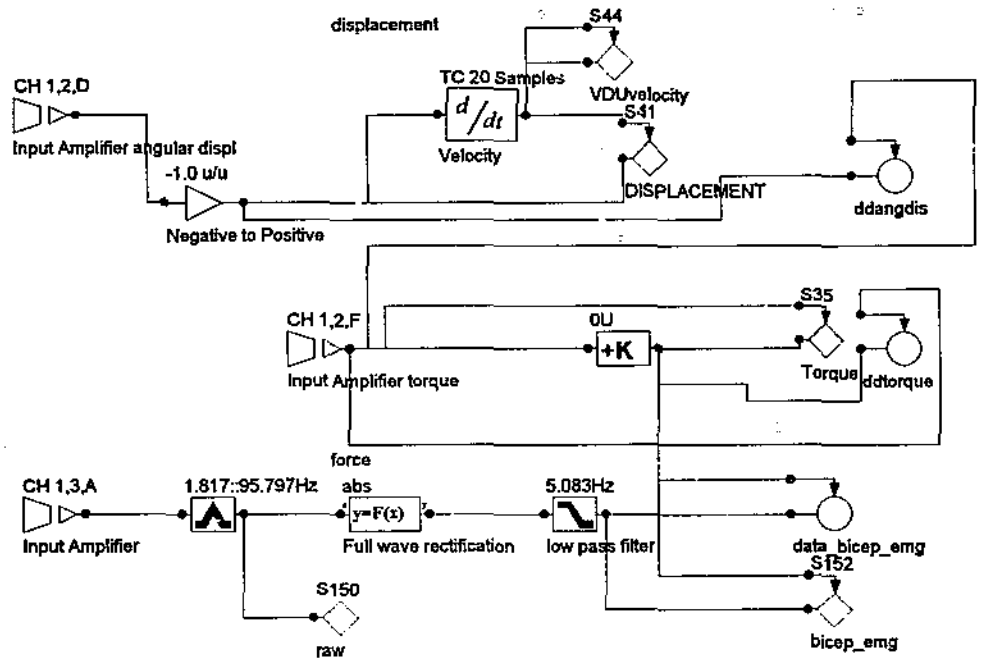
[redacted]

Karen Leckie
Manager
Graduate School

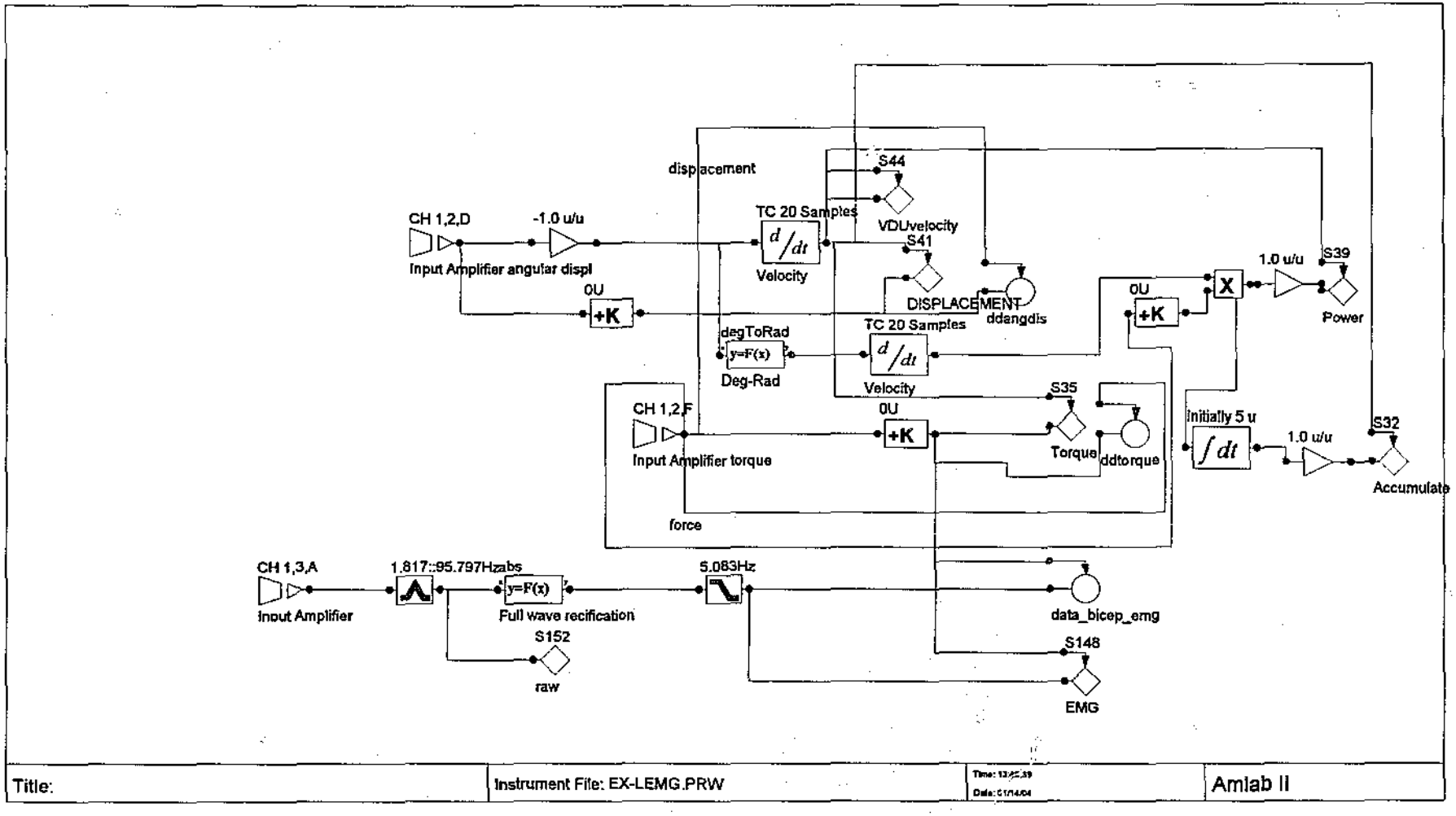
cc: P Sacco
R Treloar-Cook
Graduate School File

APPENDIX D: Amlab Schematics


O145
Torque in Volts
S35
VDUTorque
disktorque
CH 1,2,F
Input Amplifier torque
0U
+K
force
CH 1,2,A
Input Amplifier
1.817::95.797Hzabs
y=F(x)
Full wave recification
5.083Hz
S152
raw
S150
EMG
bicepEMG
Title:
Instrument File: ISOEMG.PRW
Amlab II



Title:	Instrument File: FVR-EMG.PRW	Time: 13:42:23 Date: 01/04/04	Amlab II


displacement
S44
CH 1,2,D
-1.0 u/u
TC 20 Samples
VDUvelocity
S41
Input Amplifier angular displ
Velocity
0U
+K
DISPLACEMENT
ddangdis
1.0 u/u
S39
X
Power
degToRad
y=F(x)
Deg-Rad
TC 20 Samples
Velocity
0U
+K
S35
CH 1,2,F
Input Amplifier torque
Torque
ddtorque
Initially 5 u
1.0 u/u
S32
Accumulate
force
CH 1,3,A
1.817::95.797Hzabs
y=F(x)
5.083Hz
Input Amplifier
Full wave recification
data_bicep_emg
S152
S148
raw
EMG
Title:
Instrument File: EX-LEMG.PRW
Amlab II

APPENDIX E: Pilot Study Data

Pilot Study Table 1

Subject Eccentric Peak Torque, Nm (average of 2 trials)

	Eccentric Velocity							
	30		90		150		210	
Subject	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2
1	49.5	40.0	43.0	41.0	58.0	42.5	57.0	43.0
2	27.5	24.5	27.5	30.5	27.0	28.5	29.0	30.5
3	22.0	20.5	26.5	19.0	19.0	18.5	19.5	17.0
4	35.0	41.0	34.5	36.0	34.5	38.0	35.0	32.0
5	42.0	46.0	38.5	46.0	39.0	46.5	40.0	45.0
6	76.0	71.0	71.0	67.5	72.5	67.0	71.0	67.5
7	81.0	79.0	70.5	73.0	73.5	71.0	70.0	69.5
8	67.0	56.0	69.0	60.5	70.0	62.5	62.5	79.0
9	51.5	45.5	49.5	46.5	48.5	43.5	48.0	44.0
10	66.5	74.0	65.0	62.0	75.0	68.5	71.0	66.5
11	63.0	75.0	65.0	71.0	67.5	72.0	65.0	69.0
12	51.0	52.0	56.5	49.0	50.0	43.0	48.0	43.0
13	44.0	41.5	47.5	43.0	44.5	42.0	43.0	42.0
14	47.5	49.0	53.0	51.5	50.0	55.0	54.5	56.0
Mean	**51.7**	**51.1**	**51.2**	**49.8**	**52.1**	**49.9**	**51.0**	**50.3**
SEM	4.7	4.9	4.2	4.2	4.8	4.4	4.4	4.8

Pilot Study Table 2

Subject Isometric Peak Torque, Nm (average of 2 trials)

	Isometric contraction prior to Eccentric Velocity									
	30		90		150		210		30	
Subject	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2
1	53.0	45.0	46.0	42.0	45.0	41.0	45.0	42.0	41.0	42.0
2	27.0	24.0	24.0	26.0	23.0	24.0	26.0	24.0	23.0	24.0
3	19.0	18.0	18.0	18.0	19.0	14.0	18.0	15.0	16.0	14.0
4	28.0	30.0	27.0	34.0	27.0	31.0	24.0	30.0	26.0	28.0
5	39.0	38.0	35.0	37.0	33.0	37.0	31.0	37.0	30.0	35.0
6	60.0	57.0	62.0	58.0	61.0	58.0	58.0	62.0	57.0	65.0
7	69.0	70.0	69.0	73.0	65.0	66.0	61.0	66.0	61.0	61.0
8	71.0	71.0	62.0	71.0	66.0	69.0	62.0	68.0	61.0	68.0
9	41.0	34.0	38.0	34.0	35.0	30.0	33.0	28.0	35.0	28.0
10	64.0	66.0	62.0	64.0	66.0	62.0	64.0	62.0		64.0
11	57.0	72.0	58.0	68.0	61.0	71.0	65.0	71.0	62.0	65.0
12	37.0	52.0	37.0	52.0	43.0	56.0	50.0	49.0	49.0	46.0
13	39.0	38.0	38.0	34.0	35.0	34.0	34.0	34.0	37.0	34.0
14	45.0	46.0	43.0	43.0	43.0	43.0	42.0	42.0	42.0	42.0
Mean	**46.4**	**47.2**	**44.2**	**46.7**	**44.4**	**45.4**	**43.8**	**45.0**	**41.5**	**44.0**
SEM	4.4	4.8	4.3	4.7	4.5	4.9	4.4	4.9	4.2	4.8

APPENDIX F: Principle Study Data

Table 1
Peak Isometric 90 Torque (LVE)

					Time				
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	25.23	19.77	17.25	20.05	20.89	25.51	26.09	25.07
04	F	28.16	24.81	22.85	26.20	27.04	26.48	24.80	28.72
07	F	31.25	23.18	21.73	28.13	22.31	26.24	24.20	32.41
08	F	31.54	18.53	20.27	23.47	24.78	27.83		29.58
10	F	31.18	22.89	23.47	22.75	25.65	25.95	27.84	31.76
12	F	40.71	27.83	24.64	34.09	36.85	39.61	39.47	42.23
01	M	53.61	29.28	30.96	37.11	39.63	44.66	49.69	55.01
02	M	51.95	47.18	44.10	50.81	46.90	50.53	50.25	51.93
05	M	70.39	55.29	46.06	49.97	55.29	64.52	65.63	63.68
06	M	60.18	33.48	32.64	41.03	42.43	43.26	51.65	52.21
09	M	46.79	25.07	31.80	36.56	39.61	37.72		41.65
11	M	82.27	65.63	62.84	65.91	69.55	75.70	65.63	74.86
Mean									
Total		**46.10**	**32.74**	**31.55**	**36.34**	**37.58**	**40.67**	**42.52**	**44.09**
SEM		5.24	4.37	3.86	3.97	4.25	4.72	4.72	4.51

Table 2
Peak Isometric 90 Torque (HVE)

					Time				
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	26.89	9.08	8.64	7.33	7.91	7.92	8.64	11.34
04	F	29.23	8.09	9.20	9.63	10.62	9.82	12.83	
07	F	30.88	7.63	10.24	10.68	6.89	11.69	11.84	14.75
08	F	29.14	7.48	6.46	10.82	11.26	17.66	16.20	19.26
10	F	28.58	7.09	7.45	10.38	9.37	9.66	6.89	11.55
12	F	44.06	15.77	14.89	17.22	14.89	15.04	27.11	26.52
01	M	52.27	18.37	15.02	22.85	25.92	29.56	31.24	
02	M	54.87	18.12		19.69	26.48	32.64	28.44	29.28
05	M	67.68	23.18	23.69	26.38	32.62	35.64	41.27	37.39
06	M	62.96	14.60	17.73	19.84	22.45	23.33	20.56	26.20
09	M	44.98	20.42	20.71	25.94	26.24	31.62	32.78	33.94
11	M	79.06	33.94	38.65	41.86	44.38	45.78	46.90	53.33
Mean									
Total		**45.88**	**15.31**	**15.70**	**18.55**	**19.92**	**22.53**	**23.72**	**26.36**
SEM		5.10	2.36	2.73	2.86	3.37	3.57	3.75	3.78

Table 3
Peak Isometric 150 Torque (LVE)

				Time					
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	17.53	10.82	13.95	13.06	13.34	14.02	16.35	13.58
04	F	20.75	16.14	20.05	11.38	18.09	16.42	17.54	20.33
07	F	19.31	14.75	14.89	14.46	15.33	17.65	18.67	23.43
08	F	19.55	14.17	13.15	16.64	15.18	18.09		23.18
10	F	18.24	13.44	12.42	13.29	16.35	15.04	17.22	20.42
12	F	26.74	20.71	19.69	24.93	26.24	28.27	28.27	29.44
01	M	42.28	23.13	21.73	27.32	22.29	25.08	28.16	27.60
02	M	42.85	30.68	34.03	37.95	35.43	38.79	40.75	40.74
05	M	45.08	33.20	30.68	35.15	41.02	46.90	43.82	41.59
06	M	38.93	20.89	21.45	27.33	26.78	24.53	27.88	31.24
09	M	22.27	16.49	15.91	20.13	21.44	21.29		23.91
11	M	65.77	42.14	41.03	48.02	50.25	52.49	54.73	58.92
Mean									
Total		**31.61**	**21.38**	**21.58**	**24.14**	**25.14**	**26.55**	**29.34**	**29.53**
SEM		4.39	2.73	2.63	3.34	3.34	3.71	3.80	3.56

Table 4
Peak Isometric 150 Torque (HVE)

				Time					
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	15.76	9.08	8.64	7.33	7.91	7.92	8.64	11.34
04	F	16.74	8.09	9.20	9.63	10.62	9.82	12.83	
07	F	18.35	7.63	10.24	10.68	6.89	11.69	11.84	14.75
08	F	15.40	7.48	6.46	10.82	11.26	17.66	16.20	19.26
10	F	14.32	7.09	7.45	10.38	9.37	9.66	6.89	11.55
12	F	25.65	15.77	14.89	17.22	14.89	15.04	27.11	26.52
01	M	38.79	18.37	15.02	22.85	25.92	29.56	31.24	
02	M	33.47	18.12		19.69	26.48	32.64	28.44	29.28
05	M	50.55	23.18	23.69	26.38	32.62	35.64	41.27	37.39
06	M	38.65	14.60	17.73	19.84	22.45	23.33	20.56	26.20
09	M	24.75	9.37	10.97	12.28	14.75	16.06	13.29	14.02
11	M	63.82	33.94	38.65	41.86	44.38	45.78	46.90	53.33
Mean									
Total		**29.69**	**14.39**	**14.81**	**17.41**	**18.96**	**21.23**	**22.10**	**24.36**
SEM		4.56	2.35	2.72	2.81	3.34	3.51	3.74	3.85

Table 5
Peak Torque: Concentric 30 (LVE)

					Time				
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	17.45	12.88	13.29	13.16	14.23	16.38	17.85	17.05
04	F	21.68	19.87	16.11	16.65	18.52	18.26	19.33	18.39
07	F	21.54	16.24	14.23	14.50	14.23	15.30	19.46	22.76
08	F	19.41	14.35	14.23	15.48	16.23	18.23		19.60
10	F	18.25	14.50	13.42	13.56	16.51	15.35	15.60	18.23
12	F	31.79	22.23	17.23	26.98	26.85	27.98	29.98	31.35
01	M	40.62	22.43	22.69	22.86	25.72	31.20	36.40	38.40
02	M	44.48	31.20	39.01	41.61	39.27	44.22	44.48	40.60
05	M	58.02	36.91	33.50	36.24	43.08	49.39	51.47	56.52
06	M	41.14	25.10	23.35	26.71	27.12	30.07	32.62	40.07
09	M	32.50	35.34	34.23	22.68	31.28	29.13		33.02
11	M	64.73	39.74	43.09	46.11	50.46	54.17	48.46	59.87
Mean									
Total		**34.30**	**24.23**	**23.70**	**24.71**	**26.96**	**29.14**	**31.56**	**32.99**
SEM		4.58	2.73	3.15	3.26	3.49	3.94	3.87	4.25

Table 6
Peak Torque: Concentric 30 (HVE)

					Time				
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	21.78	10.07	10.87	16.24	18.53	17.85	22.01	18.38
04	F	20.28	7.53	8.18	10.25	10.74	12.21	16.65	17.76
07	F	19.46	5.91	8.98	10.47	9.26	12.88	11.28	12.75
08	F	19.66	11.10	10.23	16.48	16.98	16.73	15.48	18.35
10	F	16.91	5.37	6.84	8.22	8.86	9.13	13.56	16.38
12	F	34.73	12.73	13.98	16.10	22.35	24.35	24.60	27.10
01	M	40.74	20.13	14.77	24.29	27.78	31.95	36.38	
02	M	40.74	26.98		24.43	27.11	31.41	27.92	30.07
05	M	55.46	22.10	25.37	31.13	34.23	39.33	48.73	49.76
06	M	41.38	12.22	13.91	20.92	21.75	27.11	24.97	27.34
09	M	34.89	18.25	18.39	15.71	15.71	26.04	22.15	27.52
11	M	61.01	31.54	31.41	33.29	34.09	37.72	34.36	42.68
Mean									
Total		**33.92**	**15.33**	**14.81**	**18.96**	**20.61**	**23.89**	**24.84**	**26.19**
SEM		4.25	2.44	2.20	2.32	2.57	2.93	3.12	3.31

Table 7
Peak Torque: Concentric 90 (LVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	19.15	15.44	15.71	15.03	15.03	16.78	17.58	18.93
04	F	19.13	17.05	15.17	16.64	17.31	18.59	17.32	19.20
07	F	20.00	13.83	12.48	16.92	17.05	15.43	17.85	21.37
08	F	18.54	13.23	13.73	15.73	14.98	16.48		17.35
10	F	18.79	13.82	14.23	12.35	15.17	14.73	15.23	18.10
12	F	29.73	19.23	15.85	20.60	24.60	25.85	24.48	27.85
01	M	37.54	20.84	22.13	22.60	23.64	30.68	32.24	35.88
02	M	40.70	34.06	34.84	36.66	36.14	38.49	42.39	39.73
05	M	53.96	29.80	30.06	26.18	35.71	43.66	44.78	51.48
06	M	41.54	27.25	25.23	26.57	25.90	27.65	28.32	39.40
09	M	30.21	25.59	20.94	24.96	32.89	26.31		30.07
11	M	55.51	35.71	31.35	29.67	36.38	40.74	43.43	51.14
Mean									
Total		**32.06**	**22.15**	**20.97**	**21.99**	**24.56**	**26.28**	**28.36**	**30.87**
SEM		3.96	2.34	2.24	2.06	2.54	2.99	3.39	3.63

Table 8
Peak Torque: Concentric 90 (HVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	22.12	10.18	10.47	16.11	17.59	17.45	19.33	18.77
04	F	20.44	10.86	6.32	8.83	10.98	11.66	15.29	19.10
07	F	20.20	5.78	7.42	6.98	9.53	10.61	11.95	13.36
08	F	18.04	13.60	12.23	13.98	18.10	15.23	15.48	15.98
10	F	16.44	6.04	6.31	8.99	10.07	8.59	14.90	16.24
12	F	33.66	11.23	11.48	11.60	16.98	18.60	21.23	24.60
01	M	36.72	14.23	16.51	21.21	24.97	28.32	32.69	
02	M	37.51	22.55		25.91	26.85	28.99	25.77	28.45
05	M	51.67	18.01	19.33	23.33	26.18	30.33	41.21	43.11
06	M	46.09	11.08	12.58	15.44	20.67	22.15	27.65	29.74
09	M	30.32	8.32	7.29	13.96	17.05	18.93	18.12	20.98
11	M	54.43	25.64	26.04	27.11	23.76	29.26	24.16	31.14
Mean									
Total		**32.30**	**13.12**	**12.36**	**16.12**	**18.56**	**20.01**	**22.31**	**23.77**
SEM		3.85	1.78	1.78	1.97	1.78	2.25	2.45	2.53

Table 9

Peak Torque: Concentric 150 (LVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	18.59	16.24	13.43	15.98	14.63	15.17	16.92	16.91
04	F	19.26	16.24	15.97	15.57	16.78	16.58	16.78	19.06
07	F	19.06	12.48	11.95	15.70	16.51	15.03	17.45	17.78
08	F	19.91	14.35	12.48	15.23	13.48	15.23		14.60
10	F	16.71	12.21	11.94	11.28	13.69	14.10	14.48	14.35
12	F	23.79	16.98	14.23	22.73	24.60	25.23	23.10	26.10
01	M	33.16	21.64	22.41	23.12	24.42	33.80	30.15	36.40
02	M	39.79	29.89	29.11	33.28	30.67	34.84	36.40	33.35
05	M	50.44	30.07	29.40	24.43	33.82	39.87	40.08	45.11
06	M	41.55	28.99	25.23	32.48	30.47	32.22	29.26	32.69
09	M	29.79	20.65	18.93	24.83	25.50	26.98		26.44
11	M	45.94	23.29	29.00	32.35	36.72	39.74	39.07	44.10
Mean									
Total		**29.83**	**20.25**	**19.50**	**22.25**	**23.44**	**25.73**	**26.37**	**27.24**
SEM		3.47	1.91	2.07	2.20	2.39	2.94	2.86	3.19

Table 10

Peak torque: Concentric 150 (HVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	22.45	9.13	8.05	15.44	15.30	18.79	20.13	18.46
04	F	17.62	8.41	7.28	7.92	10.12	11.78	14.18	13.73
07	F	16.71	4.69	8.44	7.12	9.13	7.79	9.66	11.68
08	F	16.60	13.48	10.48	12.23	15.10	13.98	13.35	14.73
10	F	14.70	6.31	4.96	6.44	7.79	6.85	13.29	14.90
12	F	29.79	9.73	12.23	10.73	13.60	16.85	19.73	20.23
01	M	35.04	17.05	13.42	19.20	23.36	30.61	29.33	
02	M	34.97	17.05		21.88	22.28	25.64	23.62	26.31
05	M	46.97	19.23	16.64	20.29	27.65	33.55	28.66	33.60
06	M	42.59	12.89		16.11	21.34	24.03	22.42	30.64
09	M	26.43	7.27	8.33	10.20	10.74	15.30	14.77	18.39
11	M	45.10	21.88	20.40	22.42	20.00	23.36	23.36	29.66
Mean									
Total		**29.08**	**12.26**	**11.02**	**14.16**	**16.37**	**19.04**	**19.37**	**21.12**
SEM		3.40	1.60	1.36	1.69	1.86	2.47	1.84	2.21

Table 11
Peak Torque: Concentric 210 (LVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	16.38	14.23	12.21	12.21	11.14	15.30	14.49	14.23
04	F	17.54	15.30	14.36	16.24	15.98	16.28	15.44	17.05
07	F	15.48	10.61	11.01	12.89	16.51	14.09	14.77	14.05
08	F	17.85	10.73	11.98	14.23	13.35	13.98		13.60
10	F	14.29	10.74	10.34	10.33	11.01	13.85	12.35	13.48
12	F	22.66	14.48	16.35	16.98	21.60	25.98	21.23	22.73
01	M	39.68	21.64	21.28	23.38	31.45	32.24	28.33	31.19
02	M	36.43	28.07	28.59	28.33	31.97	30.67	32.24	30.35
05	M	44.40	27.78	22.77	25.23	33.42	36.11	35.38	39.40
06	M	42.35	27.52	25.64	26.31	23.09	26.17	27.78	32.02
09	M	24.99	18.32	14.23	18.12	24.43	20.54		23.89
11	M	37.89	27.99	26.65	28.33	35.71	35.04	33.03	42.08
Mean									
Total		**27.49**	**18.95**	**17.95**	**19.38**	**22.47**	**23.35**	**23.50**	**24.50**
SEM		3.38	2.10	1.92	1.90	2.60	2.51	2.55	2.99

Table 12
Peak Torque: Concentric 210 (HVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	20.27	8.86	5.91	15.17	15.84	14.09	17.05	14.23
04	F	13.65	6.31	7.39	7.66	10.43	12.64	13.38	12.65
07	F	14.63	4.01	6.24	5.90	7.78	7.12	9.13	9.26
08	F	14.16	8.98	8.98	10.23	13.23	12.48	14.23	11.60
10	F	13.22	6.44	4.16	5.50	6.31	7.38	13.16	12.75
12	F	24.60	9.73	9.48	10.10	11.60	12.35	16.23	18.48
01	M	33.36	15.44	13.69	18.26	21.75	26.17	24.30	
02	M	30.47	18.39		20.54	18.52	24.23	26.17	25.77
05	M	42.04	16.12	16.51	19.15	27.38	28.46	32.75	36.64
06	M	40.02	12.14	15.40	18.12	16.11	22.82	21.48	25.11
09	M	23.87	7.54	6.59	5.37	9.26	12.35	12.62	12.48
11	M	40.07	23.76	19.33	18.79	14.23	21.48	20.27	24.29
Mean									
Total		**25.86**	**11.47**	**10.33**	**12.90**	**14.37**	**16.80**	**18.39**	**18.48**
SEM		3.20	1.69	1.46	1.73	1.75	2.14	1.97	2.44

Table 13
Peak Torque: Eccentric 30 (LVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	28.59	21.34	19.87	19.87	23.09	23.10	27.92	23.76
04	F	33.42	26.31	25.77	29.66	27.65	28.05	27.35	31.60
07	F	29.26	19.06	18.52	25.10	24.30	29.80	28.99	37.58
08	F	35.29	23.85	22.60	23.10	25.35	26.23		33.60
10	F	34.56	24.70	24.29	20.40	26.84	24.48	28.48	30.98
12	F	49.91	29.35	28.98	38.10	41.10	42.10	37.98	42.85
01	M	56.80	33.90	34.32	34.32	40.31	42.39	46.04	53.33
02	M	59.84	46.30	43.96	40.05	42.39	44.48	45.78	46.48
05	M	76.04	56.91	49.26	40.40	49.93	54.23	60.81	66.58
06	M	59.40	34.63	33.42	40.27	38.52	46.45	51.82	60.21
09	M	42.62	29.80	29.66	34.23	39.06	38.25		45.10
11	M	91.58	63.90	63.56	64.23	70.61	64.23	58.86	75.98
Mean									
Total		**49.77**	**34.17**	**32.85**	**34.14**	**37.43**	**38.65**	**41.40**	**45.67**
SEM		5.71	4.12	3.86	3.53	3.93	3.70	3.78	4.56

Table 14
Peak Torque: Eccentric 30 (HVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	29.00	12.62	12.89	17.99	19.33	19.20	22.01	27.35
04	F	30.81	16.05	15.95	16.52	18.99	21.75	23.10	27.24
07	F	30.81	12.01	14.46	16.64	15.98	16.38	17.58	21.88
08	F	30.80	24.35	22.10	25.85	29.73	28.85	29.73	30.35
10	F	28.45	11.81	11.28	14.63	16.51	18.39	23.22	30.60
12	F	47.79	21.48	19.85	22.10	26.85	30.10	29.23	33.35
01	M	50.31	21.21	19.59	28.72	35.57	37.59	42.76	
02	M	53.86	31.54		29.39	28.99	34.50	41.75	44.43
05	M	68.12	27.01	27.89	36.24	38.53	46.17	58.26	59.33
06	M	66.17	23.94	24.17	31.68	32.88	38.93	34.23	35.30
09	M	46.98	21.61	24.97	24.27	28.06	31.14	26.84	32.08
11	M	80.87	41.48	41.75	40.00	42.95	54.23	51.68	56.51
Mean									
Total		**47.00**	**22.09**	**21.35**	**25.33**	**27.86**	**31.43**	**33.36**	**36.22**
SEM		5.13	2.51	2.48	2.36	2.54	3.36	3.67	3.50

Table 15

Peak Torque: Eccentric 210 (LVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	30.51	23.35	21.48	21.07	20.40	27.60	23.76	25.77
04	F	33.49	27.92	27.78	26.31	25.23	22.82	26.78	33.73
07	F	32.75	26.04	24.56	30.34	27.65		38.12	36.78
08	F	37.54	28.35	26.48	27.10	27.10	30.60		36.73
10	F	39.13	24.96	28.99	27.12	29.53	33.10	32.23	36.48
12	F	49.54	29.10	26.98	38.73	37.48	37.10	38.10	38.35
01	M	48.72	34.18	32.50	33.80	35.10	39.27	45.00	45.52
02	M	55.02	42.92	42.91	41.35	46.56	45.52	49.16	50.35
05	M	67.65	45.64	52.62	40.27	47.11	55.57	52.08	64.57
06	M	62.46	36.81	37.18	39.33	39.19	51.68	48.12	65.57
09	M	43.81	32.35	34.77	37.05	45.51	36.51		53.15
11	M	83.36	64.23	59.54	43.09	59.54	73.96	69.60	73.63
Mean									
Total		**48.66**	**34.65**	**34.65**	**33.79**	**36.70**	**41.25**	**42.29**	**46.72**
SEM		4.64	3.35	3.37	2.08	3.30	4.23	3.91	4.30

Table 16

Peak Torque: Eccentric 210 (HVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	29.00	13.15	16.64	20.54	20.81	19.73	26.04	25.03
04	F	32.60	22.82	27.15	16.15	21.81	25.26	26.00	25.72
07	F	33.36	12.94	15.79	18.25	16.37	16.64	21.34	25.10
08	F	36.55	28.73	23.73	28.23	34.98	29.23	34.23	31.35
10	F	32.95	13.43	15.71	17.32	21.34	24.16	29.39	33.02
12	F	44.35	23.48	22.73	26.98	28.23	32.98	34.60	40.23
01	M	50.00	25.62	17.35	27.92	33.69	38.79	46.45	
02	M	50.62	31.81		30.06	22.95	29.80	41.34	47.52
05	M	69.64	23.12	26.68	34.36	34.76	42.82	56.51	60.14
06	M	65.10	24.05	28.32	23.89	33.29	41.34	37.99	40.67
09	M	55.97	25.37	27.38	27.92	32.75	31.68	29.93	38.66
11	M	81.05	44.97	45.10	38.92	42.28	50.87	41.61	58.78
Mean									
Total		**48.43**	**24.12**	**24.23**	**25.88**	**28.60**	**31.94**	**35.45**	**38.74**
SEM		4.85	2.58	2.46	2.01	2.26	2.90	2.87	3.62

Table 17
Range of Movement (LVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	131.8	114.0	112.0	117.5	119.0	125.5	126.5	129.0
04	F	135.0	131.0	133.0	130.5	135.0	134.5	142.0	138.5
07	F	138.5	125.5	127.5	131.0	127.0	126.0	132.0	137.0
08	F	142.0	132.0	130.5	134.5	132.0	137.5		142.5
10	F	133.8	118.5	117.0	121.0	124.0	121.0	124.5	133.0
12	F	137.0	129.0	128.5	134.5	134.0	135.0	135.0	137.0
01	M	163.0	154.5	155.5	164.0	156.0	157.0	157.5	154.0
02	M	140.0	135.5	132.0	131.5	134.5	138.0	136.5	139.5
05	M	123.5	113.5	117.5	114.0	119.0	117.0	118.5	119.5
06	M	126.5	99.5	75.5	101.5	100.5	105.5	109.5	121.0
09	M	131.5	129.0	126.5	126.0	123.0	122.5		134.0
11	M	128.5	126.5	129.5	126.5	129.0	132.0	128.0	130.5
Mean									
Total		**135.92**	**125.71**	**123.75**	**127.71**	**127.75**	**129.29**	**131.00**	**134.63**
SEM		2.93	3.93	5.39	4.33	3.78	3.73	3.80	2.69

Table 18
Range of Movement (HVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	137.0	123.5	121.0	117.0	118.5	121.5	128.0	142.0
04	F	136.0	126.0	126.0	124.5	125.5	124.5	131.0	135.0
07	F	130.8	63.0	72.0	95.5	102.0	103.0	102.0	114.5
08	F	141.3	132.5	128.0	130.0	135.0	135.0	138.5	141.0
10	F	135.8	122.5	120.0	125.5	126.5	130.5	136.0	134.0
12	F	132.8	122.0	123.5	123.5	125.0	122.5	125.5	128.5
01	M	158.8	135.5	133.0	144.0	140.5	137.5	148.0	
02	M	143.0	131.0		128.0	123.5	125.5	127.5	136.5
05	M	133.0	116.5	119.5	116.5	113.5	118.5	121.0	131.0
06	M	124.3	33.0	65.0	69.5	83.0	96.5	85.0	110.5
09	M	132.5	128.0	134.0	120.5	123.5	110.0	111.0	134.5
11	M	137.5	122.5	132.5	123.0	118.0	117.0	115.5	126.5
Mean									
Total		**136.88**	**113.00**	**115.86**	**118.13**	**119.54**	**120.17**	**122.42**	**130.36**
SEM		2.44	9.08	6.94	5.46	4.36	3.54	4.95	2.89

Table 19
Relaxed arm Angle (LVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	149.0	141.5	142.0	142.5	145.0	145.0	149.0	152.0
04	F	154.0	151.5	141.5	148.5	152.5	155.0	149.0	155.0
07	F	152.5	155.0	155.0	155.0	151.0	150.0	149.0	150.5
08	F	156.5	156.0	157.5	150.5	150.0	154.5		156.5
10	F	150.0	145.0	142.0	141.0	143.0	138.5	143.5	149.0
12	F	152.5	154.0	154.5	154.5	157.0	155.5	154.0	157.0
01	M	152.5	152.5	155.0	150.5	150.0	152.0	149.5	153.5
02	M	153.8	147.0	161.5	150.5	153.5	160.0	150.0	154.0
05	M	156.0	151.0	156.5	151.5	147.5	150.0	154.5	155.5
06	M	154.5	140.5	142.5	142.5	140.5	144.0	144.5	153.0
09	M	152.5	152.0	149.5	153.5	147.5	146.5		152.5
11	M	147.8	146.0	144.5	143.0	143.5	142.0	142.0	145.0
Mean									
Total		**152.63**	**149.33**	**150.17**	**148.63**	**148.42**	**149.42**	**148.50**	**152.79**
SEM		0.76	1.50	2.11	1.46	1.40	1.84	1.19	0.98

Table 20
Relaxed arm Angle (HVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	149.3	152.0	153.5	147.5	155.5	147.0	149.5	149.5
04	F	157.0	157.5	151.0	152.0	152.5	152.0	151.0	156.0
07	F	148.0	135.5	134.5	131.5	133.5	132.5	131.0	135.0
08	F	152.8	147.0	153.0	151.0	141.0	148.5	152.0	150.0
10	F	152.0	148.0	150.0	145.0	146.0	145.5	147.5	150.5
12	F	146.3	138.5	132.5	135.5	136.5	137.5	140.5	142.5
01	M	160.0	159.0	156.0	153.5	150.5	150.0	153.5	
02	M	160.0	151.5		155.0	153.0	151.0	154.5	168.0
05	M	165.0	150.5	153.5	153.0	153.5	157.0	154.0	158.5
06	M	151.0	143.0	137.5	140.5	133.5	132.0	128.0	136.0
09	M	156.3	146.0	151.5	142.0	135.5	128.0	134.0	149.5
11	M	160.8	155.0	156.5	158.5	155.0	146.5	154.5	154.5
Mean									
Total		**154.85**	**148.63**	**148.14**	**147.08**	**145.50**	**143.96**	**145.83**	**150.00**
SEM		1.69	2.07	2.55	2.40	2.57	2.65	2.84	2.78

Table 21
Creatine Kinase (LVE)

		Time						
Subject	Gender	Base	Day 1	Day 2	Day 3	Day 4	Day 7	Day 10
03	F	251.6	381.0	42.7	32.5	98.3	95.7	69.6
04	F	80.0	129.0	169.0	116.0	135.0	205.0	109.0
07	F	72.3	102.0	66.9	66.9	72.6	82.0	98.8
08	F	71.3	69.8	65.5	153.0		91.3	74.1
10	F	86.5	80.6	57.8	67.1	71.3	78.5	61.6
12	F	131.5	135.0	126.0	328.0	210.0	91.5	199.0
01	M	74.6	154.3	102.0	105.0	221.0	70.8	29.0
02	M	99.6	115.0	215.0	161.0	101.0	109.0	
05	M	342.5	578.0	375.0	379.0	354.0	506.0	326.0
06	M	185.0	755.0	530.0	549.0	519.0	341.0	254.0
09	M	220.5	403.0	324.0	195.0		463.0	94.6
11	M	371.5	110.0	438.0	1080.0	526.0	219.0	119.0
Mean								
Total		**165.57**	**251.06**	**209.33**	**269.38**	**230.82**	**196.07**	**130.43**
SEM		31.32	65.20	48.27	85.73	50.91	45.31	26.30

Table 22
Creatine Kinase (HVE)

		Time						
Subject	Gender	Base	Day 1	Day 2	Day 3	Day 4	Day 7	Day 10
03	F	59.8	134.0	674.0	1040.0	1862.0	931.0	158.0
04	F	118.5	139.0	78.7	144.0	229.0	914.0	
07	F	85.3	97.7	75.5	121.0	465.0	1010.0	285.0
08	F	106.3	160.0	134.0	95.7	74.2	85.3	67.6
10	F	91.8	133.0	94.6	80.3	95.4	100.0	96.0
12	F	147.0	126.0	98.8	121.0	529.0	593.0	239.0
01	M	33.5	134.0	60.7	87.5	78.1	64.3	
02	M	174.0	242.0	381.0	436.0	546.0	902.0	315.0
05	M	316.5	1650.0	1330.0	1390.0	1920.0	1130.0	482.0
06	M	247.5	1060.0	1010.0	1120.0	1600.0	2232.0	1600.0
09	M	108.3	461.0	462.0	1760.0	4500.0	1330.0	269.0
11	M	323.0	447.0	1600.0	2360.0	3680.0	1716.0	1160.0
Mean								
Total		**150.95**	**398.64**	**499.94**	**729.63**	**1298.23**	**917.30**	**467.16**
SEM		27.75	138.55	156.06	227.14	427.72	190.37	146.07

Table 23
Soreness Tenderness

		Time LVE					Time HVE				
Subject	Gender	Day 1	Day 2	Day 3	Day 4	Day 7	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	3.5	6.5	2.3	1.0	0.0	8.4	17.0	24.3	9.5	0.0
04	F	28.0	12.3	7.8	0.5	0.0	13.4	22.3	14.3	4.4	0.0
07	F	14.1	55.3	12.3	1.9	0.4	34.8	34.0	96.0	31.8	2.0
08	F		51.3	36.6		0.0	8.8	26.0	30.3	13.8	0.5
10	F	11.0	18.1	26.4	30.0	0.0	11.9	15.0	18.8	21.3	0.0
12	F	4.5	4.1	0.3	0.0	0.0	6.5	46.5	68.9	25.9	1.5
01	M	2.5	3.6	5.8	2.4	1.1	9.5	4.1	7.8	3.8	0.0
02	M	31.3	61.4	19.3	0.5	0.0	54.0	80.4	69.1	57.0	0.0
05	M	4.5	10.1	17.0	9.9	0.0	3.5	13.0	9.5	4.3	0.0
06	M	21.9		28.0	12.4	0.0	5.5	31.1	31.6	22.8	6.0
09	M	12.6	11.0	8.5		0.5	18.1	27.5	23.1	14.6	0.0
11	M	5.0	9.3	7.5	6.5	0.0	6.3	23.0	37.9	21.6	0.0
Mean											
Total		**12.63**	**22.08**	**14.29**	**6.50**	**0.17**	**15.04**	**28.32**	**35.95**	**19.21**	**0.83**
SEM		2.96	6.42	3.26	2.69	0.10	4.28	5.69	7.98	4.33	0.51

Table 24
Soreness Extended

		Time LVE					Time HVE				
Subject	Gender	Day 1	Day 2	Day 3	Day 4	Day 7	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	20	10	3	2	0	22	59	47	44	0
04	F	17	10	4	0	0	23	34	20.5	15	0
07	F	7	82	41	10.5	0	100	100	100	100	70
08	F		90	51		0	31	82	39	1	0
10	F	35	48.5	55	60	0	27	40	64.5	65	0
12	F	9	3.5	2.5	0	0	15.5	64	92	73	0
01	M	0	3	5	2	0	9	9	7	3	0
02	M	10	30.5	25	2	0	54.5	81	83	64	21
05	M	2	10.5	20.5	5	0	13	28.5	28	15	1.5
06	M	9.5		11.5	2	0	1.5	53	55	59.5	13.5
09	M	14	27	8		0	33	45	37.5	27.5	0
11	M	16	24.5	2	5	0	61	83.5	93	78.5	0
Mean											
Total		**12.68**	**30.86**	**19.04**	**8.85**	**0.00**	**32.54**	**56.58**	**55.54**	**45.46**	**8.83**
SEM		2.77	8.80	5.68	5.26	0.00	7.91	7.73	8.94	9.42	5.90

Table 25
Soreness Flexed

		Time LVE					Time HVE				
Subject	Gender	Day 1	Day 2	Day 3	Day 4	Day 7	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	5	10	2	1	0	30	28	37.5	16	0
04	F	8	6	3	2	0	7	12	7	7	0
07	F	15	79	48	10.5	1	100	42	65	8	0
08	F		63	51		0	23	46	38	10	0
10	F	33	40	53	56.5	0	33	45	63.5	63.5	0
12	F	1.5	3	0	0	0	12	65	93	49	12
01	M	10	3	4	1	0	3	7	0	1	0
02	M	32	23	19	6	0	31.5	71.5	58	53	0
05	M	2.5	7.5	10	3	2	7.5	6.5	10.5	2.5	0
06	M	22		6.5	6.5	0	20.5	41	29.5	49	0
09	M	20.5	29	11		0	30	34	27	14	0
11	M	5	6	6	2	0	3	63	75	62	0
Mean											
Total		**14.05**	**24.50**	**17.79**	**8.85**	**0.25**	**25.04**	**38.42**	**42.00**	**27.92**	**1.00**
SEM		3.28	7.54	5.91	4.92	0.18	7.57	6.37	8.41	7.18	1.00

Table 26
Arm Circumference (LVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	23.39	23.30	23.32	23.40	23.42	23.48	23.51	23.61
04	F	25.01	24.81	24.83	24.93	24.94	25.13	25.15	25.23
07	F	26.35	26.21	26.20	26.28	26.29	26.40	26.42	26.47
08	F	22.09	21.90	21.90	22.33	22.32	22.16		22.22
10	F	26.35	26.12	26.15	26.27	26.30	26.41	26.39	26.46
12	F	24.44	24.38	24.42	24.51	24.52	24.56	24.58	24.62
01	M	26.52	26.32	26.30	26.34	26.33	26.45	26.50	26.60
02	M	29.03	28.89	28.93	29.06	29.09	29.19	29.24	29.31
05	M	27.96	27.94	28.01	28.14	28.22	28.27	28.25	28.25
06	M	30.34	30.25	30.35	30.60	30.72	30 94	31.03	31.22
09	M	25.18	25.06	25.05	25.14	25.15	25.34	25.38	25.49
11	M	28.09	28.00	28.04	28.19	28.25	28.41	28.50	28.64
Mean									
Total		**26.23**	**26.10**	**26.12**	**26.27**	**26.30**	**26.40**	**26.81**	**26.51**
SEM		0.69	0.69	0.70	0.69	0.70	0.72	0.64	0.73

Table 27
Arm Circumference (HVE)

		Time							
Subject	Gender	Base	Post	30 Post	Day 1	Day 2	Day 3	Day 4	Day 7
03	F	24.07	24.03	24.06	24.16	24.19	24.30	24.38	24.51
04	F	25.47	25.36	25.48	25.68	25.76	25.88	25.90	25.96
07	F	25.89	25.75	25.77	25.87	25.89	26.02	26.05	26.17
08	F	22.44	22.34	22.33	22.37	22.37	22.44	22.52	22.57
10	F	27.44	27.26	27.27	27.40	27.40	27.53	27.57	27.67
12	F	25.32	25.28	25.32	25.41	25.44	25.52	25.54	25.62
01	M	26.40	26.30	26.34	26.47	26.52	26.65	26.69	26.81
02	M	27.35	27.19	27.19	27.29	27.27	27.37	27.37	27.43
05	M	27.66	27.55	27.64	27.81	27.94	28.13	28.29	28.49
06	M	29.00	28.79	28.85	28.96	28.97	29.12	29.18	29.36
09	M	24.80	24.66	24.64	24.72	24.72	24.85	24.91	24.99
11	M	29.99	29.86	29.84	29.96	29.98	30.08	30.11	30.18
Mean									
Total		**26.32**	**26.20**	**26.23**	**26.34**	**26.37**	**26.49**	**26.54**	**26.65**
SEM		0.61	0.60	0.60	0.61	0.61	0.61	0.61	0.62