1995

# Effect of exercise induced muscle soreness on the motor control properties of the biceps brachii

Alan J. Pearce
*Edith Cowan University*

# Edith Cowan University

# Copyright Warning

# EFFECT OF EXERCISE INDUCED MUSCLE SORENESS ON THE MOTOR CONTROL PROPERTIES OF THE BICEPS BRACHII

BY

Alan J. Pearce

Bachelor of Science (Sports Science)

A thesis submitted in partial fulfilment of the requirements for the award of

Bachelor of Science (Sport Science) with Honours

Date of Submission :

15 November 1995

# USE OF THESIS

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

# DECLARATION

"I certify that this thesis does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any institution of higher education; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text".

ACKNOWLDEGEMENTS

There are many people I need to thank for helping complete this project.

First and foremost I would like to thank my supervisor Dr Paul Sacco for his time and effort throughout the year, and for his support and encouragement when times got tough. Thankyou Paul.

I would also like to thank Dr Michelle Thompson for her assistance, advice and encouragement during the year. Thanks, Mich.

Thankyou to Dr Gary Thickbroom for his support and advice on technical matters during the project, without whom I may still be trying to work out how to complete my first map.

I would also like to thank Dr Amanda Blackmore for her advice and assistance in statistical analysis, and Associate Professor Barry Gibson with enrollment for Honours and for recommendations during the writing up of the thesis.

Thankyou to my friends and work colleges who unselfishly donated their time for being subjects in this project and to my badminton squad members for putting up with my mood swings at training when times got tough.

Personal thanks go to Mrs Janice Redmond for her support and advice in getting me where I am today, Ms Joan Kennington for guiding me through the last few years and helping proofread this work and my closest friends (you know who you are!) for understanding when I couldn't socialise and supporting when I needed friends.

Thankyou,

Alan Pearce
1995.

## ABSTRACT

The objective of this study was to note the time course changes for up to 28 days on the motor control properties of biceps brachii muscle following a bout of eccentric exercise.

Eight subjects (5 male, 25-40 years of age) performed 35 maximal voluntary eccentric contractions with the non-preferred arm of the elbow flexors through 130° of extension of $90°s^{-1}$. Voluntary electromyographic (EMG) activity and motor evoked potentials (MEPs) elicted by transcranial magnetic stimulation (TMS) were recorded via surface electrodes placed over the belly of the biceps brachii muscle. Maximal isometric strength was measured at 90° elbow flexion. A simple elbow flexion/extension tracking task was used to assist visuomotor co-ordination.

Subjects displayed greatest strength loss at 1 day (of control measures) which recovered by 21 days post-exercise. Impairment in the skilled tracking task was noticeable within hours following the exercise, and was greatest 1 day post exercise, but returned to control levels by 3 days. There were no changes in the threshold level of MEP responses to TMS but maximal MEP amplitudes increased on average (although responses were variable). No changes were observed in the EMG activity following exercise.

The changes in the motor performance and corticomotor excitability occur following eccentric exercise which may be related to alterations in the pattern of afferent feedback from weakened and/or painful muscles. The implications from this suggest that coaches need to be sympathetic to the needs of the athlete when balancing physical training with skill training/development.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

# PUBLICATIONS

The following publications arose from the work presented in this thesis (see Appendix G and H).

## ABSTRACTS

Sacco, P., Pearce, A. J., Thompson. M. L., Thickbroom, G. W. and Mastaglia, F. L. (1996). Effects of maximal eccentric exercise on motor control properties of the biceps brachii muscle. *Medicine and Science in Sports and Exercise*, (In Press)

Pearce, A. J., Sacco, P., Thompson. M. L., Thickbroom, G. W. and Mastaglia, F. L. (1996). Effects of maximal eccentric exercise on neuromuscular control of the biceps brachii muscle. *Proceedings of the Australian Neurological Society*, (in Press)

# GLOSSARY OF ABBREVIATED TERMINOLOGY

## UNITS

Système International units were used throughout this thesis.

## ABBREVIATIONS

| | |
|---|---|
| CK | Creatine Kinase |
| COG | The location of the centre of gravity for a map |
| EMG | Electromyogram |
| DOMS | Delayed onset muscle soreness |
| GTO | Golgi Tendon Organ |
| MAX | The maximum value of the map |
| MEP | Motor evoke potential |
| MVC | Maximal voluntary contraction |
| RMS | Root mean square |
| TMS | Transcranial magnetic stimulation |

DEDICATION

In loving memory of my parents John Pearce and Audrey Oh

who supported my decision in returning to study.

# CHAPTER ONE

# INTRODUCTION

## 1.1 Background to the Study

Delayed onset muscle soreness (DOMS), stiffness, and muscle fibre damage, are commonly experienced by individuals after performing unaccustomed exercise or following an increase in training intensity. Although the exact mechanisms which bring about exercise-induced muscle damage are unclear, the specific effects which occur have been well documented. These include prolonged weakness, loss in range of motion and contractile force (Armstong, Warren & Warren, 1991), a leakage of muscle enzymes into the blood stream (Shumate, Brooke, Carroll, & Davis, 1979), muscle swelling, and muscle fibre necrosis. It has also been shown that activities which invlove lengthening of the active muscle (eccentric contractions) produce the greatest soreness and muscle damage (Armstrong, Ogilvie & Schwane, 1983), explaining why some forms of exercise (eg. downhill running) which have a large eccentric exercise component can result in considerable soreness where as others (eg. cycling), which incorporate fewer eccentric contractions, cause little or no damage.

Although many studies have examined the responses to exercise-induced muscle damage, there is very little information on the effect of DOMS on performance. The functional consequences of exercise-induced damage are loss of muscle strength and DOMS, both of which may affect the ability of a subject to perform a task. This is of particular relevance for both coaches and athletes in their planning regarding periodisd training programmes to prevent or lessen the extent of DOMS, tapering for

events which require skilled movements, and training sessions to optimise skill development and practice whilst maintaining fitness levels.

## 1.2 Purpose of the Study

The objective of the study is to identify and characterise the time-course of any change in motor control properties of biceps brachii after a single bout of exercise-induced muscle soreness and damage. Research variables, as outlined in point 1.3, will be followed in subjects before, and at various times after, exercise-induced muscle soreness and damage.

## 1.3 Hypotheses

A period of muscle soreness and weakness following exercise-induced damage will result in changes in the motor control properties of biceps brachii reflected in:

1. muscle strength;
2. plasma creatine kinase (CK) levels
3. skilled performance using a motor tracking task;
4. corticomotor excitability of the biceps brachii; and
5. cortical representation of the biceps brachii.

## 1.4 Organisation of the Thesis

Chapter One provides a discussion on the background, significance and purpose of the study, with a list of the major hypotheses being tested. Chapter Two reviews the related literature pertaining to the study; Chapter Three describes the theoretical framework of the study; and Chapter Four describes the design and methodology, including instruments of testing and measurement, and procedures of data collection and analysis. Results and data analysis are presented in Chapter Five, and the thesis concludes in Chapter Six by discussing the findings of the study, their relation to the literature, and their implications for further research.

# CHAPTER TWO

# REVIEW OF THE LITERATURE

## 2.1 Delayed Onset Muscle Soreness (DOMS) and Muscle Damage

Muscle soreness can arise from intense or unaccustomed strenuous activity (Clarkson, Byrnes, McCormick, Turcotte, & White, 1986). In this context unaccustomed activity can be defined as an activity or exercise that has not been performed previously, or for a period of time longer than 4-6 weeks. Muscle soreness regularly occurs after exercise in individuals who perform physical exercise after a period of inactivity, however, it can also occur in regular competitive and elite athletes, demonstrating that high levels of fitness are no protection against muscle damage; although the better state of training, the more exercise can be tolerated without the symptoms of overuse (Kuipers, Drukker, Fredrick, Geurten & Kraneburg, 1983). Muscle soreness generally occurs following activities which involve the generation of high muscle forces (Armstrong, Olgivie, & Schwane, 1983). A typical feature of exercise-induced muscle soreness is its delayed onset (ie soreness tends to be most severe one to several days after exercise), hence the term 'delayed onset muscle soreness' or DOMS (Kuipers, 1994). DOMS differs from other commonly experienced muscle pains such as cramps, trauma (ie 1st or 2nd degree muscle tears), or ischaemic pain, where the resulting pain is almost immediate. Acute pain from a cramp or trauma is commonly described as 'sharp, intense pain', whereas after eccentric exercise (lengthening of muscle during contraction), it is described as 'dull and aching' (Clarkson & Newham, 1994). Although exercise-induced muscle soreness is common, and its practical consequences are known, there is far less certainty

regarding causative factors or the cellular mechanisms involved (Armstrong et al, 1991).

Armstrong (1984) discusses three mechanisms that have been proposed to account for the presence of muscle soreness and damage following exercise. These are:

1. structural damage in the contractile and/or elastic tissues due to high tension development in the muscle;
2. cell membrane damage leading to a disruption of calcium homeostasis in the injured fibres that produces a cellular necrosis; and
3. stimulation of free nerve endings of Group IV sensory neurones due to accumulation in the interstitium of intracellular contents and products of macrophage activity.

Two basic theories have been put forward to explain how exercise initiates damage. One mechanism describes a disturbance in metabolic function, whereas the other addresses a physical disruption of the cell.

### *2.1.1 Metabolic Paradigm*

During prolonged submaximal exercise, metabolic events, such as ischaemia or hypoxia, ATP depletion and accumulation of muscle metabolites have been proposed to initiate muscle damage (Francis, 1983; Armstrong, 1984; Ebbling & Clarkson, 1989).

Devries (cited in Ebbing & Clarkson, 1989) suggested that exercise may initiate a positive feedback cycle in which local ischaemia leads to muscle spasm that in turn

causes compression of blood vessels and increased ischaemia leading to a reduction in oxygen availability to the muscle.

However, Ebbling & Clarkson (1989) question the basis of the metabolic cause of exercise-induced muscle soreness and damage:

> If metabolic waste products were primarily responsible for exercise-induced muscle damage, then muscles which contract concentrically and fatigue more quickly would show more damage than muscles that develop active tension eccentrically (p.209).

Schwane, Watrous, Johnson & Armstrong (1983) compared skeletal muscle damage following concentric and eccentric contractions in humans and animals. Eccentric contractions had a lower metabolic cost, and produced less lactate ($La^+$) than concentric contractions. However, eccentric contractions caused greater structural damage than concentric exercise, and DOMS was only evident after eccentric work in humans. Similarly, Kuipers, et al (1983) noticed muscle damage in rat hindlimb muscles (soleus, rectus femoris and vastus lateralis) following eccentric low intensity treadmill exercise, but found no elevation in $La^+$ levels. Thus $La^+$ does not seem to be a primary agent in the production of muscle damage.

### *2.1.2 Physical Paradigm*

Many researchers dispute the metabolic hypothesis of muscle soreness, on the finding of greater muscle damage and subsequent delayed soreness following eccentric muscle actions versus concentric actions (Francis, 1983; Armstong et al, 1983; Knutten, 1986; Ebbling & Clarkson, 1989). When muscles develop active tension eccentrically, they require less energy (due to the recruitment of fewer motor units)

than for concentric work, yet experience greater injury than muscles that contract concentrically (Clarkson & Newham, 1994; Kuipers, 1994). As eccentric contractions recruit fewer motor units, this places a greater stress on each individual motor unit. Data from the studies of McCully and Faulkner (1986) suggest that physical muscle fibre damage results from high tensile stresses occurring during eccentric contractions. If the tensile stress exceeds muscular strength, then microscopic damage can occur to contractile elements and connective tissue. Muscle injury is indicated by morphological changes (Armstong et al, 1983) such as cell necrosis, phagocytosis, and inflammatory responses. In addition, performance changes (Mair, et al, 1992), delayed-onset soreness, and increases in muscle proteins in the blood stream (Evans, 1987) can also be observed as a result of muscle damage.

## 2.2 DOMS and Functional Muscle Strength

Exercise-induced damage has been assessed using changes in motor performance, especially functional muscle strength (Ebbling & Clarkson, 1989). It has been demonstrated that maximum voluntary strength declines after eccentric exercise (Jones, Newham, & Clarkson, 1987; McCully & Faulkner, 1986; Newham, Jones and Clarkson, 1987). This is followed by a slow recovery in which strength may remain depressed for a week or longer (Ebbling & Clarkson, 1989; Newham et al, 1987).

The exact mechanisms by which eccentric exercise results in loss of strength have not been clearly identified. One possiblity is that, since subjects experience pain during contraction when muscle damage has occurred, it may be that the discomfort associated with maximal voluntary efforts may inhibit full muscle activation. At present

there is increasing evidence to support the explanation that there is a lowered inherent capacity of the muscle to produce force rather than an inability of subjects to fully activate muscle (Davies & White; 1981; Jones et al, 1987; Newham et al, 1987). Thus, Newham et al (1987) superimposed electrical stimulation on voluntary isometric actions of muscles with DOMS (following eccentric exercise) of the forearm flexors. They found that additional force was generated by electrical stimulation only if voluntary force generation by the subject was submaximal. Results indicated that maximal force was generated throughout the testing period (Ebbling and Clarkson, 1989). Furthermore, experiments in animals using electrical stimulation to assess muscle force following eccentric exercise show similar decrements to those seen in humans (Sacco, Dick, Jones & Vrbora, 1993).

In addition to the reduction in maximal force generation, eccentric exercise also affects contractile properties. This is demonstrated by a change in the force-frequency relationship so that relatively lower forces are generated at low (ie $\leq$ 20Hz) frequencies (Clarkson & Newham, 1994). This is termed low frequency fatigue (LFF) and has been suggested to be the consequence of decreased calcium release by each action potential or changes in the stretch reflex (SR) (Clarkson & Newham, 1994; Jones & Round, 1990). The functional significance of LFF is unknown, but force generation is impaired in the physiological firing range of isometric and eccentric activity.

## 2.3 Serum Levels of Muscle Proteins

Evans (1987) notes that exercise-related increases in the plasma levels of intra-muscular proteins such as creatine kinase (CK), lactate dehydrogenase and myoglobin are the result of exertional rhabdomyolisis (muscle fibre breakdown or necrosis) and further proposes that the exercise-induced increase in plasma CK are directly related to the intensity of the exercise. Support for this concept comes from the studies of Tidus & Ianuzzo (1983) where individuals who exercised at a high intensity and short duration showed greater enzyme activity and soreness as compared to individuals who exercised at lower intensities and for longer durations. More recently Saxton et al (1994) reported an increase in CK activity in eccentrically exercised arm flexors.

However, other researchers disagree. Kuipers, Janssen, Keizer and Verstappen's (1985) found a poor correlation between serum CK and the percentage volume of rat muscle fibres damaged. Likewise, Van der Meulen, Kuipers and Drukker (1991) reported no differences in the amount of histological muscle damage in either male or female rats, although there were notable differences in the amount of serum CK released between sexes. Thus, the actual volume of histological damage was significantly less than would be expected on the basis of enzyme release.

The above findings of Kuipers et al (1985) and Van der Meulen et al (1991) are supported in human studies by the earlier findings of Newham, Jones and Edwards (1983) who, could not explain why some subjects in their study of stepping exercise released a greater efflux of CK whilst others did not. Similarly, Nosaka and Clarkson (1993) compared CK efflux after subjects eccentrically exercised one or both elbow flexors. They found that doubling the amount of damaged muscle (i.e both arms as

opposed to one arm) did not show any significant rise in plasma CK, concluding that CK is a poor correlate of the extent of muscle damage.

Although conflicting evidence exists regarding the amount of CK released following exercise, to this point in time, CK is an useful directive of DOMS as the release of CK indicates that intramuscular damage has occurred.

## 2.4 Electromyographic Studies of Muscle and Muscle Damage

Electromyograph (EMG) studies have been used to record electrical activity of the muscle during contraction by using surface or intramuscular electrodes (Astrand & Rohdahl, 1986, p. 43; Jones & Round, 1990, p. 66).

EMG studies have shown that muscle damage resulting from eccentric exercise does not significantly affect EMG activity during maximal contractions. This supports the argument that restriction of motion and pain do not account for any changes in neuromuscular activity which must therefore arise from other pathophysiological processes (Newham et al, 1987; Howell et al, 1985)

## 2.5 Neuromuscular Function, Motor Control and Muscle Damage

To date, most of the literature in the discussion of neuromuscular function and muscle damage is drawn from animal studies. Much emphasis is being placed on trying to confirm and expand, in conscious humans and primates, conclusions previously reached on the basis of animal experiments (Bigland-Ritchie, 1990, p. 378).

Of the limited research available, the literature suggests that neuromuscular function can be affected by DOMS. This has been illustrated by Miles, Ives, and

Vincent (1993) who showed onset of agonist and antagonist muscle bursts were impeded, and the time from agonist EMG onset to initiation of movement, had also slowed. More recently, Saxton et al (1994) investigated the effects of exercise-induced muscle damage on muscle tremor and motor control/proprioception. After eccentrically exercising the bicep brachii muscle, amplitude and frequency of bicep tremor, perception of voluntary force, joint position and force proprioception were monitored to assess changes in the components of the neuromuscular system. Muscle tremor amplitude increased until 48 hours post exercise, perception of joint position and perception of force were both impaired indicating a loss of motor control and proprioception and maximum strength had not fully been restored by the fifth day post exercise.

A possible mechanism for this impairment of neuromuscular function has been linked to the affect of muscle damage within the muscle-tendon complex. Afferent sensory receptors (located in the muscle tendon complex) provide the means by which an individual is consciously aware of the positions of various parts of the body and whether a particular joint or limb is moving or stationery (Marieb, 1994, p.486).

Few studies have examined the effects of exercise on proprioceptive function. Saxton et al (1994) showed a reduction in the ability to accurately perceive voluntary force in the biceps following eccentric exercise suggesting an alteration in proprioceptive feedback from the muscle with damage. This is supported by the findings of Miles et al (1993) who demonstrated motor reaction time had slowed subsequent to eccentric exercise.

These results, suggest impairment of neuromuscular function preceding full extent of delayed onset muscle damage. For further discussion of proproception and motor control, please refer to section 2.6.

## 2.6 Evaluation of Motor Skill and Coordination

In the last three decades instrumented upper extremity tracking tests have been developed to measure human neurological performance more objectively (Behbehani, Kondraske, Tinter, Tindall, & Imrhan, 1990). Although these tests vary in procedure and configuration, the prime objective is to measure subjects' ability in tracking a moving target using the upper extremity. These tests have been used to study the performance of healthy subjects (Cassell, 1973), and those with Parkinson disease (Hoehn & Yahr, 1967), brain damage (Jones & Donaldson, 1981) and other neurological disorders (Jones, 1980).

Typically, tests involving the evaluation of quantitative measurement of the upper extremity involve the subject using a type of electromechanical device, such as a joystick, interfaced with the display unit via an electric potentiometer. In two recent studies (Neilson, O'Dwyer, & Neilson, 1988; Behbehani et al, 1990), testing of tracking performance involved a microcomputer, a monitor, and a joystick in the evaluation of adaptibility of learning in healthy subjects while the study of Behbehani et al, (1990) focused on the response time with accuracy, in healthy patients versus three patient populations (Parkinson's disease, multiple sclerosis and myasthenia gravis) using oscillitory (left to right) movements. They found that Parkinson's patients had a slower reation time and lower amplitude gain (similar to Hufschmidt and

Lucking (1995) in their study of Parkinson's patients and tracking). Multiple sclerosis patients had similarly slowed reaction times and displayed overshooting oscillations, while myasthenia gravis patients, although faster than the other two populations, were still relatively slower in reaction time compared to healthy subjects.

### *2.6.1 Learning Effect of Motor Skill Tracking Tasks*

Neilson, O'Dwyer and Neilson (1988, p.114) note that "When the target in pursuit tracking is driven by a simple deterministic stimulus signal (such as a sine wave or square wave), subjects can anticipate the future position of the target and thereby compensate for response time delay." For very regular target signals such as sine waves, subjects generate a signal of frequency approximately equal to that of the target and attempt to synchronize the two (Krendel and McRuer, 1960). This pattern generating mode has become known as "precognitive tracking" following Krendel and McRuer (1960), who likened it to the ultimate level of skilled behaviour in their "successive organisation of perception" model of motor skill development.

## 2.7 Evidence of Cortical Reorganisation

Research (Cohen, Bandelli, Findaly, & Hallet, 1991, Topka et al, 1991, Wilson, Thickbroom, & Mastaglia, 1993) has demonstrated that reorganisation of the motor cortex can occur with permanently altered physiology (i.e amputations and spinal lesions). Permanent changes such as higher excitability of the motor cortex and a shift in the area of control when stimulated by TMS (see section 2.8) have been reported in forearm amputees (Cohen et al 1991). The findings are supported in experiments

analysing the motor cortex of human subjects following lower limb (lower leg) amputations (Fuhr et al, 1992). Similar studies examining subjects with spinal cord lesions have also been reported (Levy, et al, 1990; Topka, et al, 1991). These studies identified a pattern of motor system reorganisation that results in enlarged muscle representation areas and muscles immediately proximal to the lesion eliciting an elevated excitable response. In addition, these findings are supported in animal models where motor outputs are reorganised after peripheral nerve lesions (Merzenich, et al, 1983; Kalask & Pomeranz, 1979), removal of body parts (Kelahan, Ray, Carson, Massey & Doetsch, 1981; Merzenich, et al, 1984; Pons et al, 1991) and reversible limb deafferentation by local anaesthesia (Metzler & Marks, 1979). Such capability of the motor cortex to alter outflow to specific muscle groups suggests the possibility that these mechanisms may play a role in short-term or temporary altered physiology (i.e DOMS).

Brasil-Nato et al (1992) have suggested that human motor outputs can experience both short and long term changes. The short term changes are referred to as 'modulation' and the long term changes as 'reorganisation'. Modulation and reorganisation of muscular representation on the motor cortex has been demonstrated in the learning and acquiring of motor skill tasks (Pascual-Leone, Gramna, Hallet, 1994), however, to date, modulation has not been demonstrated under conditions of temporarily altered physiology and motor performance.

## 2.8 Transcranial Magnetic Stimulation of the Motor Cortex

The cerebral cortex is involved in mental activities such as conscious thinking, reasoning, learning, memory, intelligence and sense of responsibility. It is also concerned with perception of the senses and the initiation and control of voluntary muscle contraction (Newton & Joyce, 1990, p.259).

Motor cortex areas are defined as regions in which electrical stimulation produces and controls muscular movement of a part of the body (Newton & Joyce, p.260). Until recently, stimulation of the motor cortex in humans has been possible only by maintaining direct contact between stimulating electrodes and the cortex, either intraoperatively or through subdurally implanted electrodes (Wilson et al, 1993).

Although these studies have provided fundamental insights into the organisation of the motor cortex, their usefulness has been limited by their invasive nature and by ethical considerations since studies have been confined to patients undergoing surgery (Wilson et al, 1993).

Transcranial Magnetic Stimulation (TMS) is a recently developed non-invasive technique for the stimulation of the human motor cortex. With TMS the cortex is painlessly stimulated as a consequence of the rapid discharge of current through a magnetic coil held over the scalp (Barker, Jalinous, & Freeston, 1985). The technique uses a large pulse of magnetic field to induce currents below the stimulus point. The current flow induced in the underlying cortex by the pulse from the magnetic coil is sufficient to activate pyramidal tract neurones trans-synaptically (Day, et al,, 1989) and, under some circumstances, directly (Beradelli, Inghilleri, Cruccu, Manfredi, 1990; Wilson et al, 1993). When the resulting membrane excitability of the α–motorneuron

reaches threshold, a measurable response of the motor evoked potential (MEP) will be recorded by surface electromyogram (EMG). The size of the MEP is directly related to the number of motorneurones activated, hence the excitability of the motor pathway is dependent on the stimulus intensity (Valls-Sole, Tolosa, Pujol, 1992).

### *2.8.1 Mapping of the Motor Cortex*

Transcranial Magnetic Stimulation (TMS) is a non-invasive, painless technique which has been developed for the stimulation and mapping of the human cortex. This technique has been used to explore the functional anatomy of the motor cortex by measuring the motor evoked potential following stimulation at multiple scalp sites (Wilson et al, 1993).

Following the development of increasingly focal stimulation techniques, TMS has been applied in exploratory studies of the organisation of the human corticomotor representation, particularly under conditions of altered physiology (Wilson et al, 1993).

### *2.8.2 Reliability of TMS for Mapping*

Mortifee, Stewart, Schulzer & Eisen (1993) have demonstrated TMS to be reproducible and reliable in muscle representation of the motor cortex. The study mapped the abductor pollicis brevis (APB) and abductor digiti minimi (ADM) motor cortices of six normal subjects, each studied on 2 separate occasions separated by several weeks. Their results showed that the coefficients of variation, which should be low (Fleiss, 1986) ranged from 14% to 37% and coefficients of reliability, which

should be high (Fliess, 1986), ranged from 63% to 94%, indicating that the described technique for motor mapping is reproducible.

## 2.9 Summary

Delayed onset muscle soreness (DOMS), is a common experience of individuals after performing unnaccustomed exercise or following an increase in training intensity. Although the exact mechanisms that bring about exercise-induced damage are unclear, it is well understood that eccentric contractions have been shown to result in the greatest injury to skeletal muscle fibres.

In response to exercise, activity and behaviour; specific molecular, biochemical, electrophysiological and structural changes take place in central nervous systems neurones and neural networks (Cotman & Nieto-Sanpedro, 1982). These plastic changes are part of the structural and physiological processes for recovery of function after injury - either permanent or temporary (Marshall, 1984; Kaas, 1991). Reorganisation in the human motor system has been studied using transcranial magnetic stimulation of individuals following amputations (Cohen et al, 1991; Furh, et al, 1992) and spinal cord lesions (Topka et al, 1991); and a pattern of motor system reorganisation that results in enlarged muscle representation areas of motor cortex and larger motor evoked potentials for muscles immediately proximal to the lesion has been demonstrated. Such capability of the motor cortex to modulate outflow to specific muscle groups introduces the possibility that these mechanisms may play a role in temporary changes to the performance of motor skill tasks after exercise-induced muscle damage.

# CHAPTER THREE

# THEORETICAL FRAMEWORK

## 3.1 Theoretical Framework

DOMS is the result of unnaccustomed or high intensity exercise. Eccentric muscle contractions have been found to induce DOMS with the consequences being:

- muscle pain;
- tenderness and stiffness;
- muscle weakness; and
- muscle fibre damage (as indicated by CK efflux from muscles).

It is proposed that the above consequences of eccentric exercise may alter some aspect of motor control, resulting in measureable changes in :

- skilled tracking task;
- corticomotor excitability; and
- cortical representation of the affected muscle.

With these changes in mind, the functional consequences of exercise induced muscle damage will be explored; that is, what effect does exercise induced muscle damage have on motor function, and how does the time course of any changes interact with the consequences of damage (e.g pain, stiffness and weakness).

A diagrammatical representation of the theoretical framework of the research is shown in Figure 3.1.

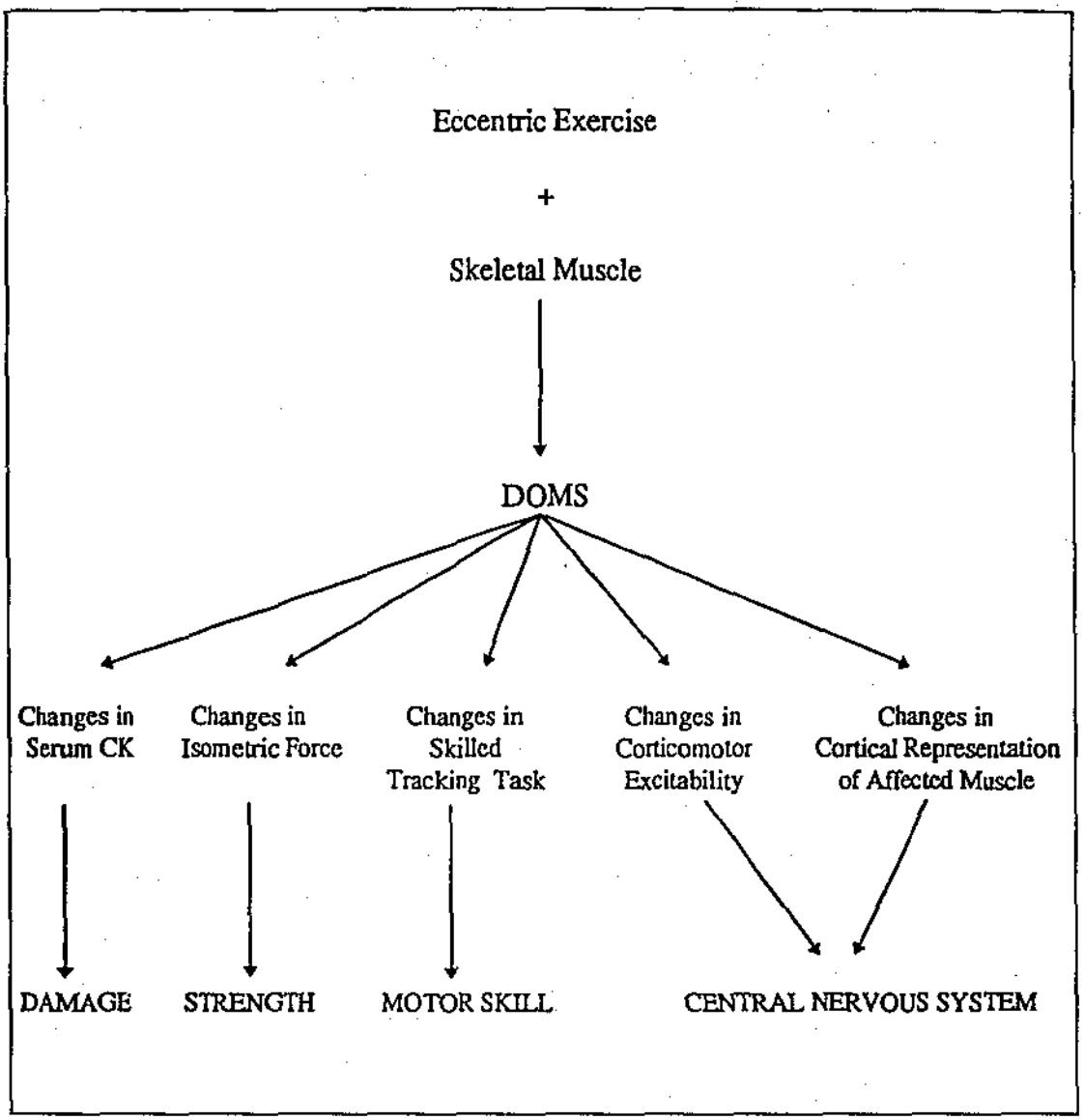



Figure 3.1. Theoretical framework of the research.

# CHAPTER FOUR

# METHODOLOGY

## 4.1 Design

A 'within-subjects, pre-test/post-test design' (Burns, 1995, p.118) was used to investigate the dependent variables of DOMS, against the independent variables. A diagrammatical representation is shown in Table 4.1.

| DEPENDENT VARIABLES | INDEPENDENT VARIABLES |
|---|---|
| Isometric Force | Time (after exercise bout) |
| Creatine Kinase | Exercised Arm |
| Accuracy of Skilled Tracking | Non-Exercised (control) Arm |
| MEP Amplitude | |
| EMG Activity | |

Table 4.1 - Design variables.

In order to quantify any improvement in accuracy of the tracking task over the course of testing, a control group consisting of six age matched healthy subjects (three male), performed the same tracking test over the same time course as the exercise group.

## 4.2 Subjects

Nine healthy subjects (five male, four female), mean age 32 years (range 25-45yrs), not currently undertaking any specific upper body physical training (eg weight training) other than normal daily activities were in the initial group. Two subjects failed to complete the data collection leaving seven subjects to provide data for the study. The protocol for the study was approved by the Ethics Committee of Edith Cowan University and use of transcranial magnetic stimulation by the Sir Charles Gairdner Hospital Human Rights Committee. Subjects were told of the nature and risks of the procedures to be used, and written informed consent was obtained (Appendix A). Subjects were asked to refrain from making any changes to exercise habits over the course of the study.

## 4.3 Instruments

### *4.3.1 Exercise Protocol and Strength Measurements*

Kin-Com Isokinetic Dynamometer (Chattex Corp., U.S.A)

Preacher Bench (45 °)

IBM Microprocessor

SUN Microprocessor

### *4.3.2 Motor Skill Tracking Assessment*

Joystick-Lever Arm

Electronic Potentiometer

IBM Microprocessor

SUN Microprocessor

### *4.3.3 Creatine Kinase Analysis*

Reflotron spectrophotometer (Boehringer Manheim, Australia)

Heparinised 30 µl capillary tubes

Spectrophotometer Strips

### *4.3.4 Corticomotor Properties*

Magstim 200 (Magstim Co., U.K)

Surface EMG electrodes (4 mm diameter, Grass)

Modified Preacher Bench (45°)

IBM Microprocessor

SUN Microprocessor

translucent rubber cap, adhesive tapes, restraining velcro straps, electrode gel

### *4.3.5 General*

Analog to Digital Converter

Goniometer

Data test sheets (Appendix B)

## 4.4 Reliability/Reproducibility

Calibration of all equipment was undertaken weekly (Appendix J). All measurement systems were computer software controlled, where the technical error of measurement was <5%. Testing reliability was ensured through tester training sessions undertaken prior to the commencement of the study. The arm positioning for strength testing, skilled tracking task and TMS recording was carefully standardised for each subject to minimise errors due to alterations in arm position. Subjects were familiarised with the testing equipment prior to the collection of data to minimise the effect of learning on test results. Visual feedback was provided for strength tests and subjects were exhorted to perform maximally throughout all testing sessions.

## 4.5 Data Collection

The data collection took place in two phases. A preliminary study was undertaken to ascertain reliability of measurements of corticomotor excitability and cortical representation (mapping) of the biceps brachii. Once reliability was established, the main data collection phase was staggered over a four week period (Table 4.2)

### *4.5.1 Preliminary Study*

A preliminary study was completed prior to the main study to ascertain the reproducibility of corticomotor properties of mapping the biceps brachii muscle. Wilson et al (1993) have demonstrated reproducibility in distal muscle groups of the

hand (adductor policis brevis and adductor digiti minimi), however, to date, there is no information on these parameters for the proximal muscle groups (ie biceps brachii).

The preliminary study involved seven subjects. The methodology followed Wilson et al (1993) - which is described in detail in section 4.7.3, with subjects attending two recording sessions seven days apart.

## 4.6 Eccentric Exercise Protocol

Once reliability and reproducibility of pre-exercise measurements were established, subjects performed the exercise protocol. This consisted of 7 sets of 5 repetitions maximal voluntary isokinetic eccentric contractions (see Fig 4.1). The limb was moved through 130° of extension at $90°s^{-1}$ and returned at a velocity of $15°s^{-1}$ flexion, giving a work-rest ratio of 1:4. Rest periods of two minutes between sets were provided to minimise the effect of fatigue on force production over the course of the eccentric exercise protocol. Subjects were encouraged to perform maximally throughout the exercise protocol.

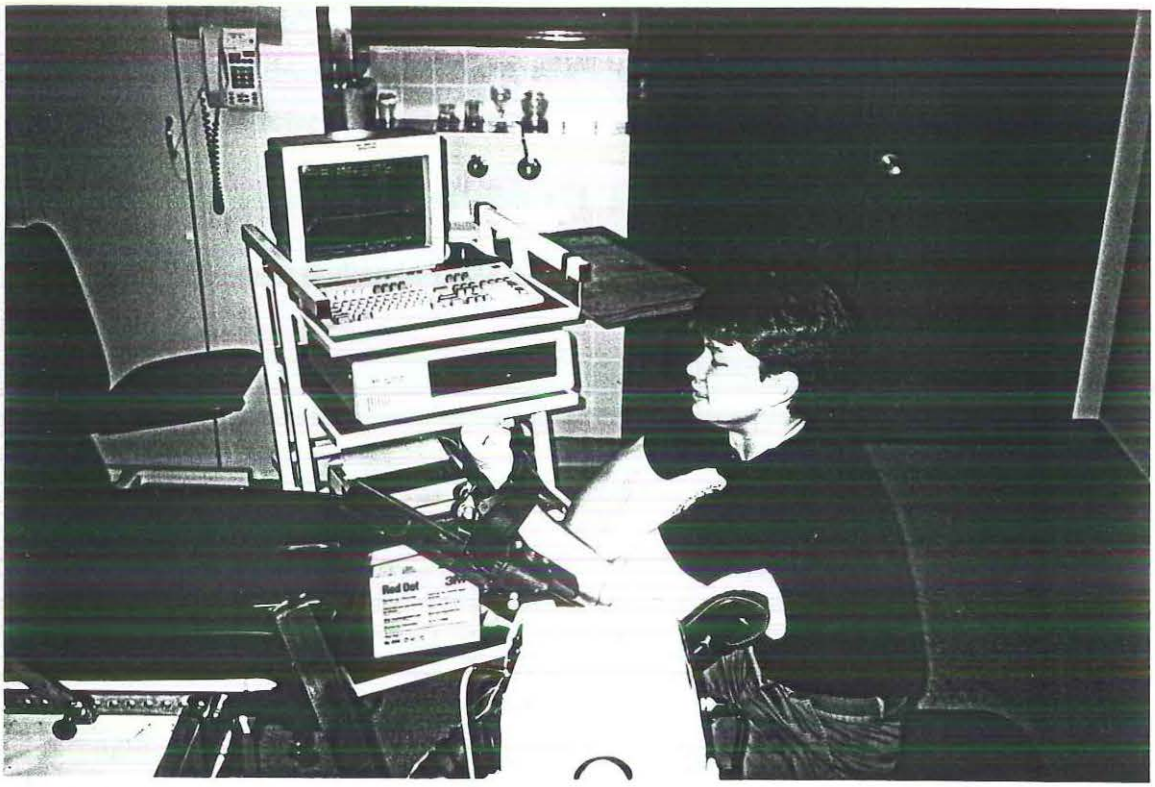



Fig 4.1 Subject completing eccentric exercise protocol on isokinetic dynamometer.

## 4.7 Testing Protocols

The main study consisted of ten separate visits incorporating:

- Two pre-exercise testing sessions - for baseline measurements of variables involved;
- Eccentric exercise protocol; and
- Eight post-exercise testing sessions at 1, 3, 7, 14, 21 and 28 days incorporating the testing variables outline in Table 4.2.

| | Pre-Test | | Post-Test | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | +1 | +3 | +5 | +7 | +14 | +21 | +28 |
| Isometric Strength | * | * | * | * | * | * | * | * | * |
| CK | * | * | * | * | * | * | * | * | * |
| Skill Tracking | * | * | * | * | * | | * | * | * |
| Cortico-motor Properties (non-ex bicep) | * | * | | | | | | | * |
| Cortico-motor Properties (ex bicep) | * | * | * | * | * | * | * | * | * |

Table 4.2 Testing schedule matrix

### *4.7.1 Strength*

Isometric maximal voluntary contraction (MVC) force was assessed at 90° elbow flexion using an isokinetic dynamometer (Kin Com, Chattex Inc, USA) on at least two occasions prior to the eccentric exercise protocol and at the beginning of every testing session following the exercise protocol. Subjects were encouraged to perform maximally for three seconds during the effort. Two MVC's were performed to ensure the attainment of peak torque recording.

Force data was digitised and displayed using a custom made software programme at a rate of 50Hz and stored for subsequent analysis. Maximal strength was taken as the peak force attained above pre-contraction baseline (see Fig 5.2 for typical force trace).

### *4.7.2 Creatine Kinase*

In order to verify that delayed onset muscle soreness was reflected in muscle fibre damage, changes in serum creatine kinase (CK) levels were measured using a spectrophotometer (Reflotron, Boehringer Manheim, Australia). 30µl peripheral capillary blood samples were taken following lancet finger prick from the index finger of the non-dominant hand. The samples were dispensed onto the reagent carrier by slowly depressing the pipette plunger to the red separation zone ensuring the pipette did not touch the surface of the red pad. The reagent strip was then placed into the spectrophotometer for analysis.

### *4.7.3 Transcranial Magnetic Stimulation*

A Magstim 200 magnetic stimulator with a 50mm diameter figure '8' coil (Fig 4.2) was used. The stimulator coil was held in position against the scalp, with the centre of the figure '8' coil over the measured site to be stimulated (Fig 4.3). To maintain consistency of responses, the coil was held in the same position for all scalp sites stimulated (ie tangential to the skull with the handle posterior).

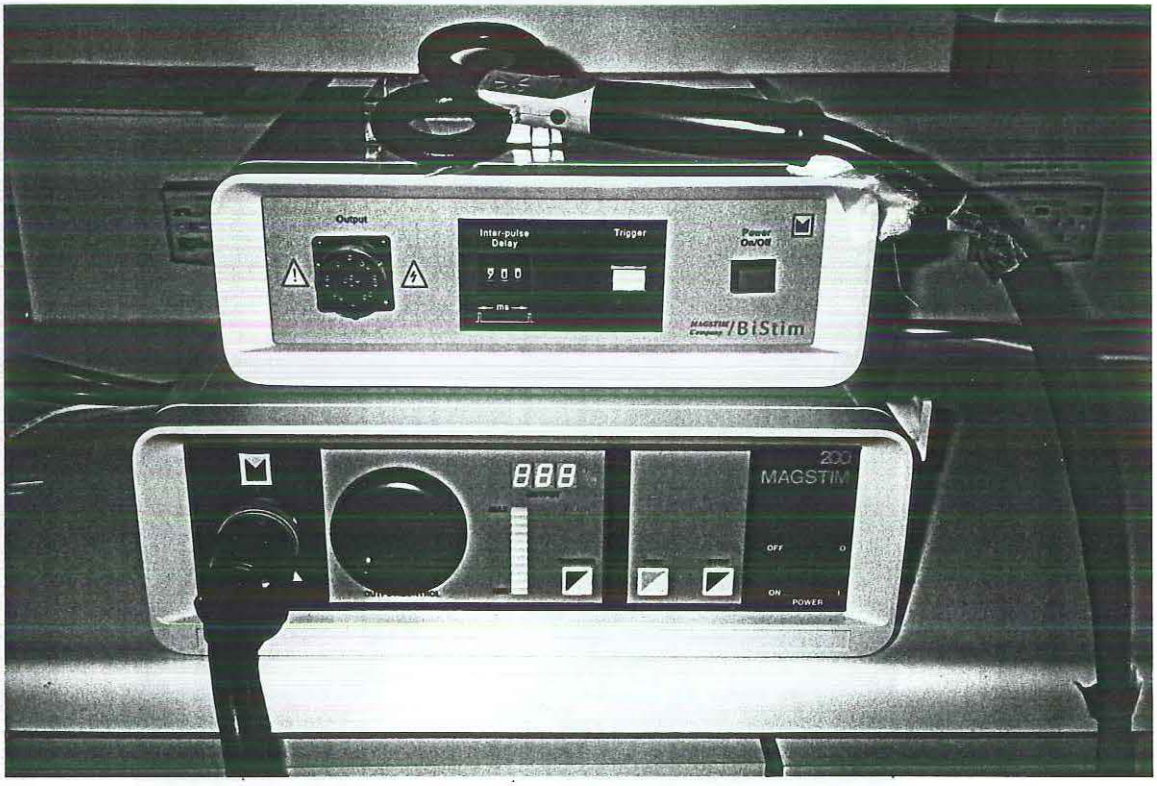



Fig 4.2 Magstim 200 magnetic stimulator with 50mm diameter figure '8'coil.

Fig 4.3 Positioning of figure '8'coil over measured stimulus site.

*4.7.3.1 Recording of Muscular Response*

Surface electromyograph (EMG) (Grass Gold, 4mm diameter) electrodes were placed over the biceps brachii muscle with the active electrode over the motor point of the muscle, and inactive electrode 2 cm distal (Fig 4.4). To ensure reliability of the muscular response, and to maintain accuracy of the electrode placement, the surface of the biceps muscle was 'mapped' (by the use of plastic wrap) using anatomical 'landmarks'. The earth electrodes were placed over the lateral epicondyle of the humerus in both arms. The amplified signal was high pass filtered at 10 Hz and low pass at 2 kHz, and the digitised data was collected at a rate of 200 Hz in 500 ms epochs which were triggered consecutively with the onset of the TMS pulse.

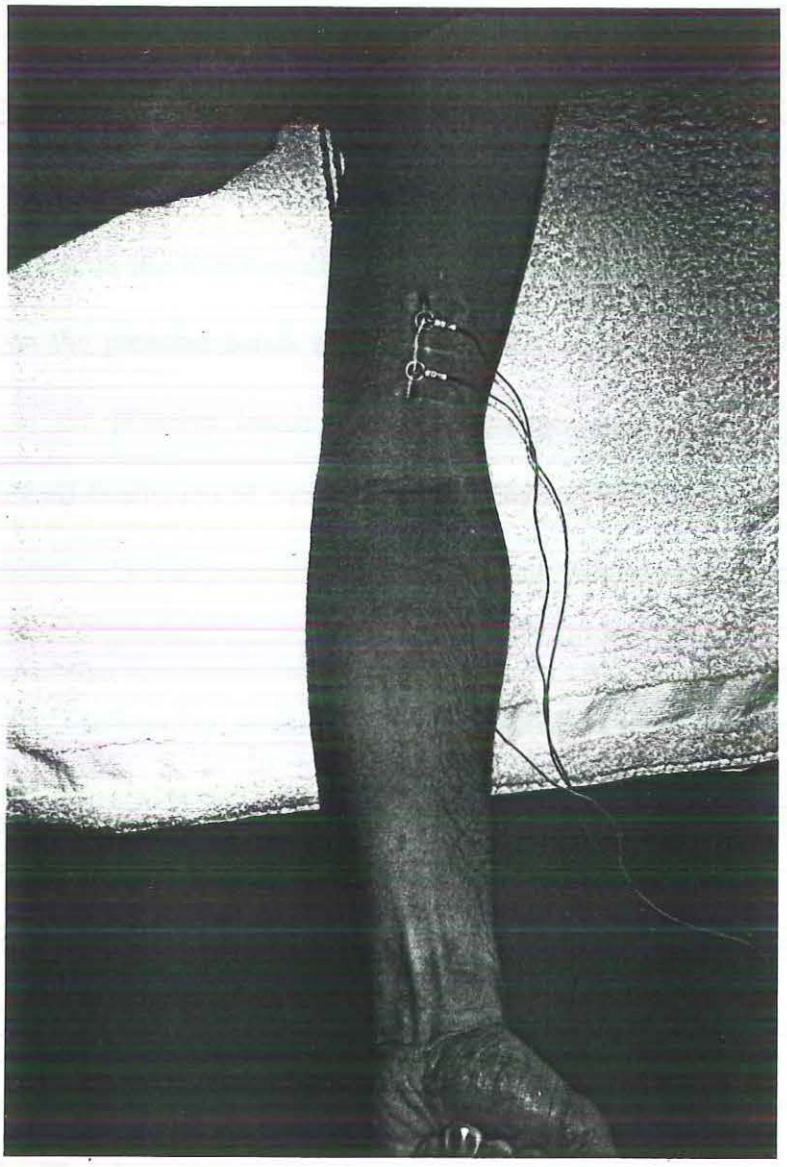


Fig 4.4 Placements of EMG electrodes on surface of biceps.

*4.7.3.2 Facilitation*

Research has shown that a muscle in a slightly contracted state will be activated by TMS at a lower stimulus intensity than a relaxed muscle (Mazzocchio et al, 1994). By using a lower stimulus intensity and slightly contracting the target muscle, neighbouring muscles are less likely to be activated during the experiment. It was important that surrounding muscles were activated as little as possible, as interference may occur in the EMG signal. Biceps brachii facilitation was isolated by resting the arm on the preacher bench with the wrist restrained by velcro straps to the modified part of the preacher bench (Fig 4.5). This kept the elbow at an angle of 90°, minimised facilitation of surrounding muscles and maintained isometric contraction of the biceps. To keep consistency for dominant/non-dominant muscles and between subjects, it was necessary to quantify the contraction level for facilitation of biceps brachii. Each subject performed an isometric MVC (in the above facilitation position) against a manual restraint for three seconds. The root mean square (rms) of the EMG interference pattern during the three second contraction was used as a measure of the maximal voluntary EMG activity. During stimulation, subjects were required to maintain facilitation of the biceps muscles at 10% of MVC for that muscle. The computer displayed the level of contraction as feedback to the subject to maintain that level. The display showed a bar graph illustrating the current level of contraction, which was updated approximately every 500ms. The computer allowed triggering of TMS stimulation to occur only if the subject maintained the contraction within the target range of 10% ± 3% for 1.5 seconds.

Fig 4.5 Facilitation of biceps on modified preacher bench.

*4.7.3.2 MEP*

MEP responses were quantified by measuring the peak to peak response of the biphasic waveform (Fig 4.6). The responses were measured in mV.

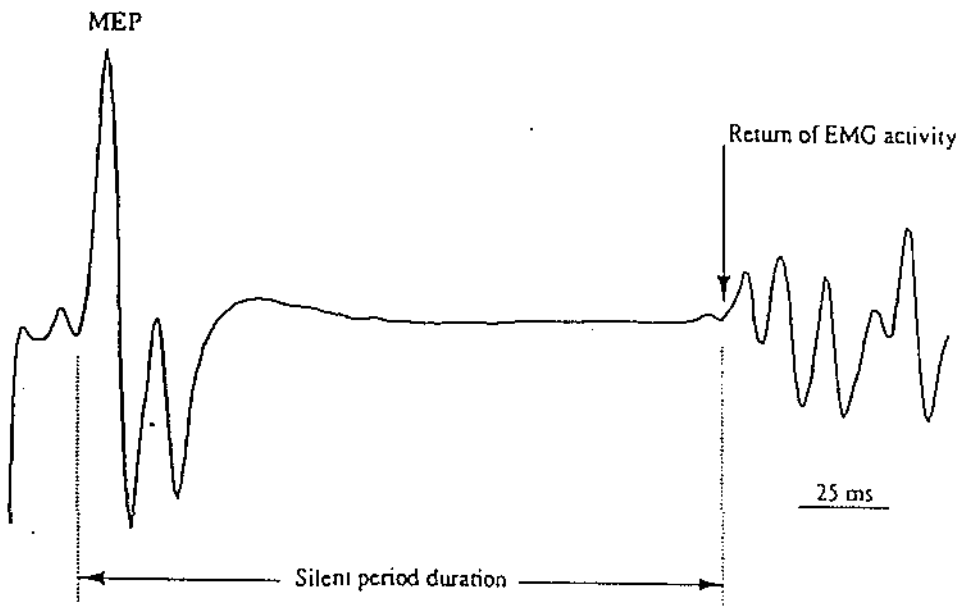



Fig 4.6 Typical MEP responses in the biceps brachii to TMS

*4.7.3.3 Thresholds*

The site of stimulation was in the region expected to control biceps (as determined in the preliminary study) at a longitude of 0cm and a latitude of 4cm from the vertex (Fig 4.7). To locate the subjects optimal site for determination of threshold responses, four stimuli were recorded, at the separate sites moving laterally along the inter-aural line, to determine the site with the largest response, thus being the closest site to the centre of the area controlling the biceps.

To determine the threshold level of stimulation required to induce MEP responses, the optimal site was stimulated, starting at 30% intensity (output range of Magstim 200 being 0% to 100%). At each intensity, four stimuli were given five seconds apart. This was repeated at increments of 5% until an intensity was reached where individual waveforms had ceased to become larger, or 100% intensity stimulation had been achieved. Threshold for the mapping procedure was defined as the intensity at which at least two out of four stimuli evoked a MEP discernible above background EMG (Wilson et al, 1993). Fig 4.8 illustrates recorded MEP responses during the threshold procedure.

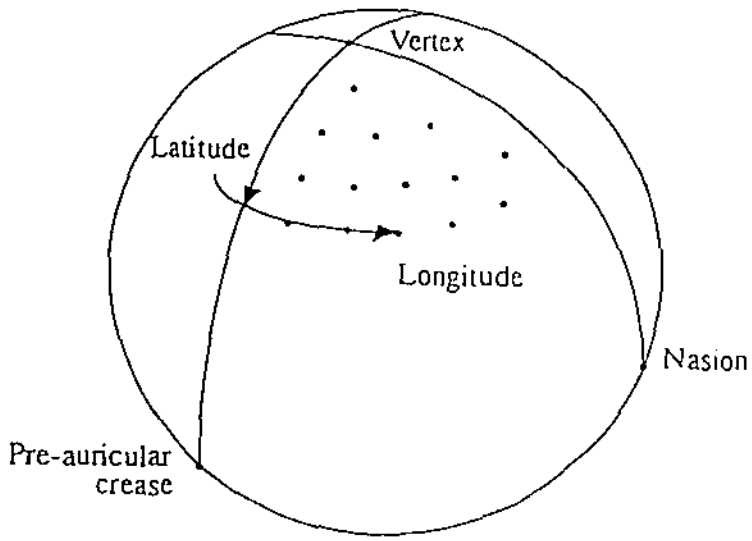



Fig 4.7 Location of stimulus sites on the cap placed over the subject's head. Latitude is defined as cm from the vertex (arc length) and longitude as distance form the interaural line in cm.

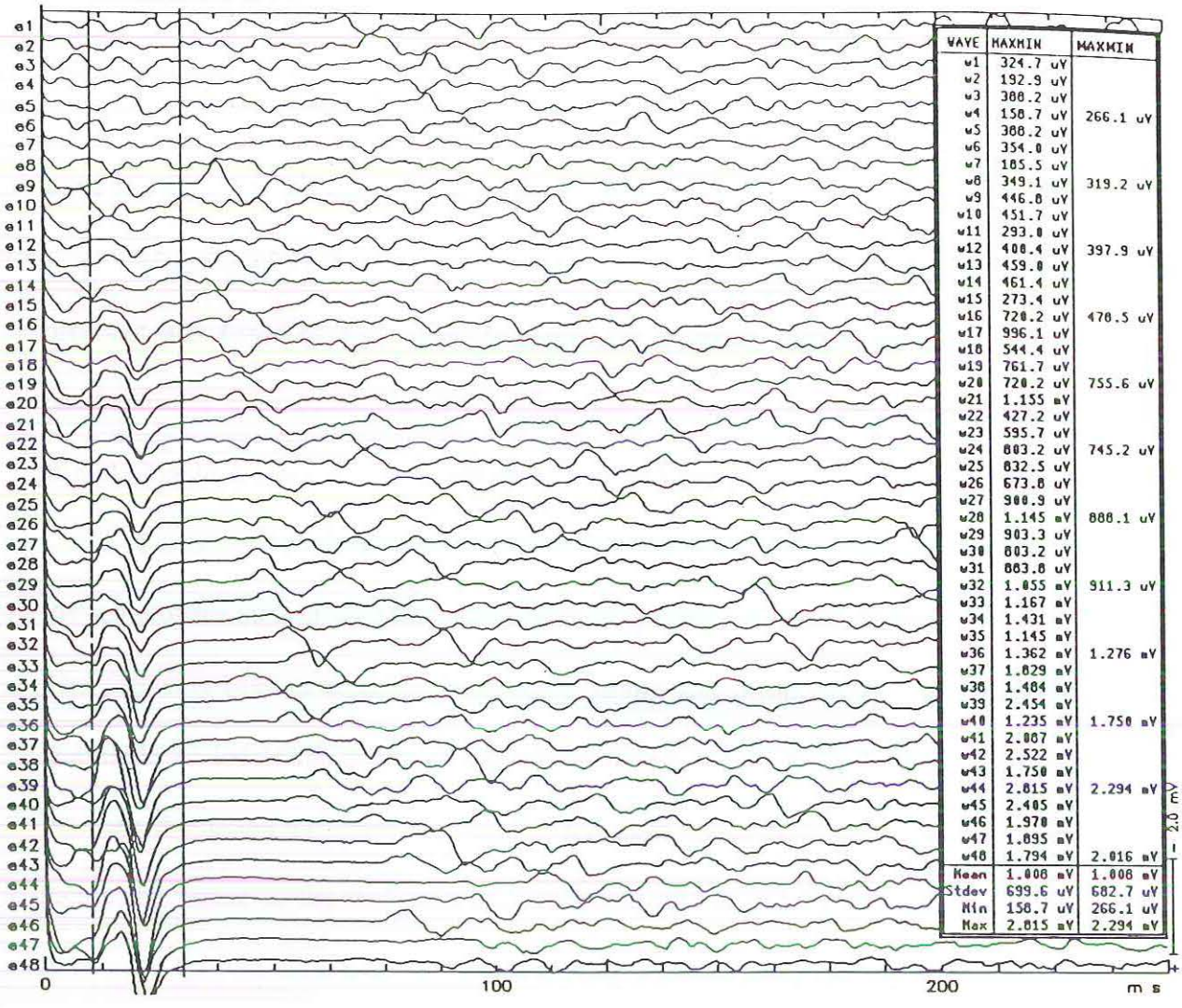



Fig 4.8 Recorded M.E.P responses during threshold protocol.

The left hand axis numbers each 250msec EMG trace and the base axis gives and indication of the time scale for which each event occurs. With each increase in intensity the MEP becomes larger and always begins from 10-20msec following a stimulus. The peak amplitude of the MEPs was averaged (four stimuli representing a 5% increment in intensity) and recorded for each intensity. Threshold data for MEP amplitude at each stimulus intensity was plotted to produce threshold curves. The calculated threshold was used for interhemispheric, pre-exercise and post-exercise threshold comparisons.

*4.7.4 Corticomotor Mapping of the Biceps Brachii*

Mapping of the motor cortex has been previously described in Chapter 2. The protocol was based on techniques developed for mapping cortical representation of hand muscles by Wilson et al (1993). The study followed the same procedure for stimulation the motor cortex, compiling the map and interpreting the data.

To locate the stimulus sites, a flexible, translucent, rubber cap was fitted over the scalp of the subject with pre-marked sites at spacings of one centimetre (Fig. 4.8). The cap was held in place by two velcro straps and positioned using anatomical landmarks to locate the centre of the cap on the vertex of the scalp. Measurements between the nasion and inion, and the left and right preauricular crease were used to locate the vertex at the mid-point and intersection of the nasion-inion line, and the inter-aural line. Stimulus sites were located using a latitude/longitude based coordinate system. Latitude was defined as the distance over the scalp from the nasion-inion line, and longitude as the distance from the inter-aural line (Wilson et al, 1993)

Fig 4.9 Translucent cap with pre-marked sites for stimulation by TMS.

During the mapping process, the stimulator intensity was set at 20% above threshold level to maintain a consistent uniformity above threshold for each hemisphere. The difference in mapping intensities between hemispheres (pre-damage) was within 5% in all seven subjects. The first site stimulated for mapping was the same site used to examine thresholds, being close to the estimated centre of the motor area for the biceps brachii. Mapping the biceps brachii on the motor cortex required the stimulation of all sites around the estimated centre of the map. At each stimulus site (1cm equidistant in latitude, 2cm in longitude) moving away from the estimated centre, the MEP response became smaller, until no measurable response was recorded after stimulation and this signified the border of the representation for the target muscle. This required the stimulation from 20-32 sites. Four muscle responses to stimulation of each scalp position were recorded and the average of the four responses was assigned to represent the scalp position stimulated.

*4.7.4.1 Map Compilation and Interpretation*

Four MEP waveforms from each site were reviewed off line by the experimenter and those not containing artefact (ie noise) were averaged. The peak-to-peak amplitude of the averaged MEP waveforms at each scalp site were assigned to that site as an index of the contribution of the underlying cortex to the control of the biceps brachii. The latitude and longitude of stimulus sites over the scalp (in centimetres) was converted to positions on an 'idealised' sphere of half circumference given by the subjects inter-aural distance (Fig 4.9). From the MEP amplitude measured

at discrete sites over the hemisphere, the expected MEP amplitude for intermediate sites on the hemisphere was estimated. The results are presented in map form, where a square matrix is used to represent the scalp viewed from the superior aspect above the vertex. The map shows a two dimensional representation of the biceps brachii on the motor cortex in contralateral cerebral hemispheres. The map indicates the 'optimal centre of gravity'stimulus site (in cm latitude and longitude) and contours according to the muscle EMG response, decreasing towards the edge of the map until no response in measure (Fig 4.10) The shaded contours are scaled in the key at the base of the figure, and represent from zero (clear) to one hundred percent (black) of the maximum amplitude that is measured or estimated for that representation. The optimal site of each representation which occurs at the calculated point of maximum amplitude is marked on the map with a white cross. The area of the map is calculated (in $cm^2$) from and above the 50% and 75% contours and this study used the 50% area in all subjects.

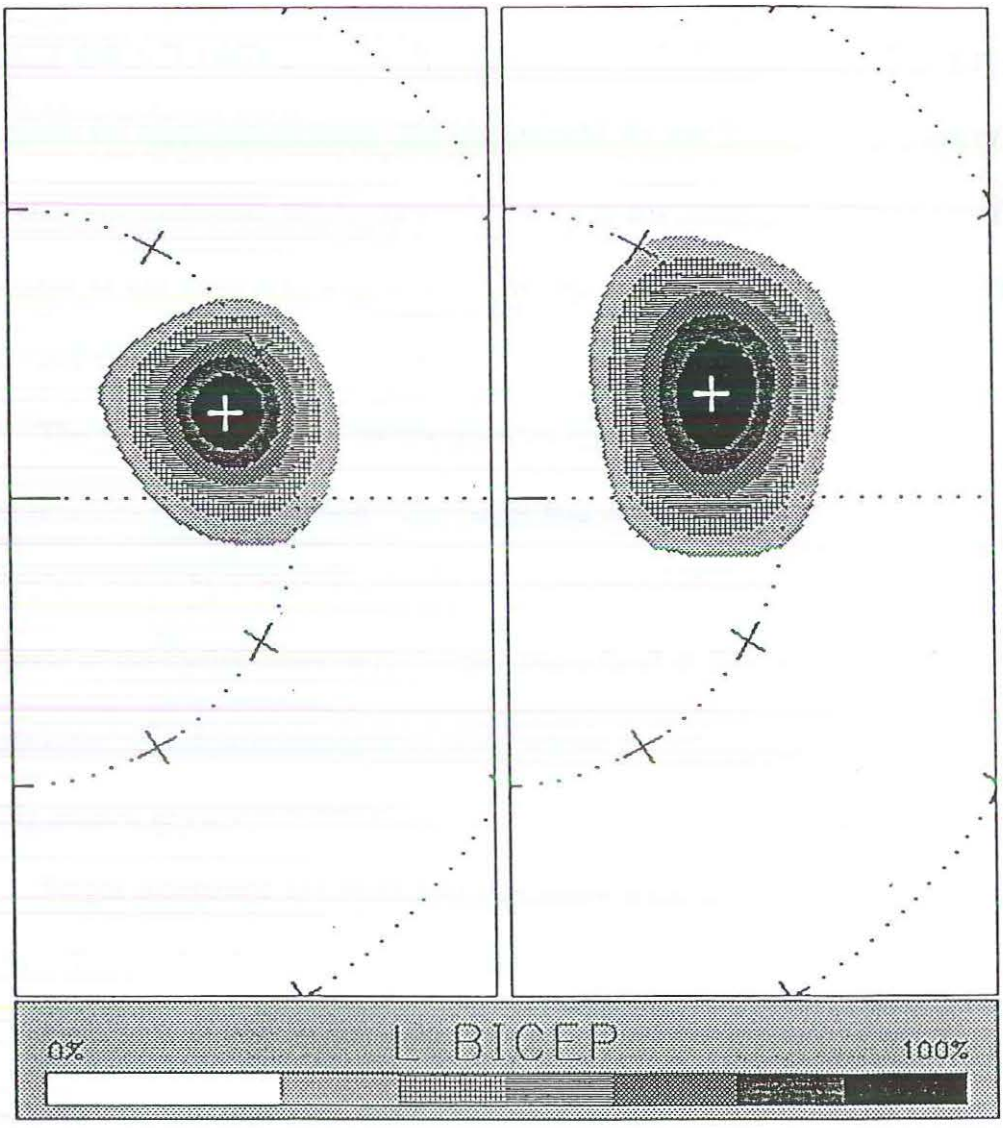



Fig 4.10 Cortico motor representation of the biceps.

### *4.7.5 Skill Tracking*

The motor skill tracking task involved the subject sitting and resting the non-dominant arm on a tabletop so that the upper arm was 90° to the torso (Fig. 4.11) to ensure that the bicep was isolated. The subject held the arm handle by the palm of the hand and was instructed not to move the wrist in any flexion or extension. Thus movement of the lever arm was to be controlled as much as possible by the elbow flexors and extensors.

The tracking task were programmed to run in a IBM colour microprocessor linked to a Sun computer station. The target was a white cursor driven vertically on the display screen by a random generated programme using sine wave cycles. The amplitude of the cycles varied with the frequency fixed at five cycles in ten seconds. The indicator of the arm consisted of a red cursor on the display screen which was directly parallel to the white cursor .

Target movement and lever arm movement were amplified and smoothed in low-pass filters before being displayed on the monitor.

Subjects were instructed to attempt to keep the response marker aligned with the continuously moving target. Five attempts of ten second duration were recorded at a rate of 10Hz and stored in computer files for subsequent analysis.

Fig 4.11 Assesment of motor skill tracking.

Subject moves lever in elbow extension and flexion to correspond with cursor appearing on monitor (not shown).

## 4.8 Data Analysis

Data analysis was conducted using Excel 5.0 (Microsoft Corp.). Interhemispheric differences in MEP map area, threshold MEP's, maximum amplitude for MEP's, centre of gravity optimal stimulus location (latitude and longitude), and MVC EMG amplitude, were tested using the Wilcoxin Signed Rank Test. The level of significance for all tests was set at $p< 0.05$. For accuracy of motor tracking skill, data was analysed using the Wilcoxin Mann Whitney Test as the experimental group were compared to a control group.

Throughout the time course of the data collection subjects were unable to attend testing sessions, thus some data points were missed. Subsequent procedures of using prior knowledge or mean values for the treatment of missing data points were adopted (Tabachnick & Fidell, 1989). Results are described in text quoting p values only, full statistical findings are shown in Appendix D.

## 4.9 Limitations

1. Selection of subjects restricted participants to people who were not currently undertaking rigorous physical activity, and so involved some degree of subjective evaluation on the part of both the researchers and the subject.

2. Volunteer subjects may not be representative of the population as a whole.

3. Central fatigue is a confounding factor, but of minimal importance (James, Sacco, & Jones, 1995), although uncontrollable. However, all the subjects were urged earnestly to perform maximally.

## 4.10 Assumptions

1. Subjects will perform to the best of their ability during the testing sessions.

2. Subjects will not make lifestyle changes likely to confound the results of the investigation, ie major dietary or training adjustments.

# CHAPTER FIVE

# RESULTS

Full results are tabulated in Appendix C, typical MEP maps, and threshold curves, are presented in Appendix E. All group means are given in the text ± standard error of the mean (sem).

## 5.1 Reproducilibity

Test/retest method error for reproducibility was calculated following the protocol described by Thorstensson (1976) for the parameters measured.. Table 5.1 summarises test/retest for all variables. Full results are illustrated in Appendix F.

| | ME | CV% |
|---|---|---|
| Strength (N) | 5.49 | 2.43 |
| CK (lu/l) | 6.613 | 6.31 |
| Voluntary EMG (mV) | 90.97 | 19.08 |
| MEP Amplitude (mV) | 1.19 | 19.1 |
| Threshold Responses (%) | 0 | 0 |
| MEP map location (cm from vertex) | 0.299 | 6.23 |
| MEP map area ($cm^2$) | 3.71 | 34.61 |

Table 5.1 Method error for reproducibility of two tests. ME=Method Error; CV%=Coefficient of Variation.

## 5.2 Strength

As a result of eccentric exercise all subjects showed a dramatic loss in strength immediately following exercise, which was greatest at one day post-exercise (strength loss of 36 ± 11% of pre-exercise values. Fig 5.1). Fig 5.2 illustrates maximal voluntary contraction force traces pre and one day post-exercise in a typical subject. Although strength loss was dramatic, it was noticable that subjects did not report any muscle pain whilst performing isometric contractions, even when they were experiencing DOMS.

Strength recovered gradually over the time course of the study (Fig 5.1) and only returned to normal range 21 days post-exercise. Even by 28 days mean strength had not reached control values (mean = 94 ± 2% for the seven subjects at 28 days).

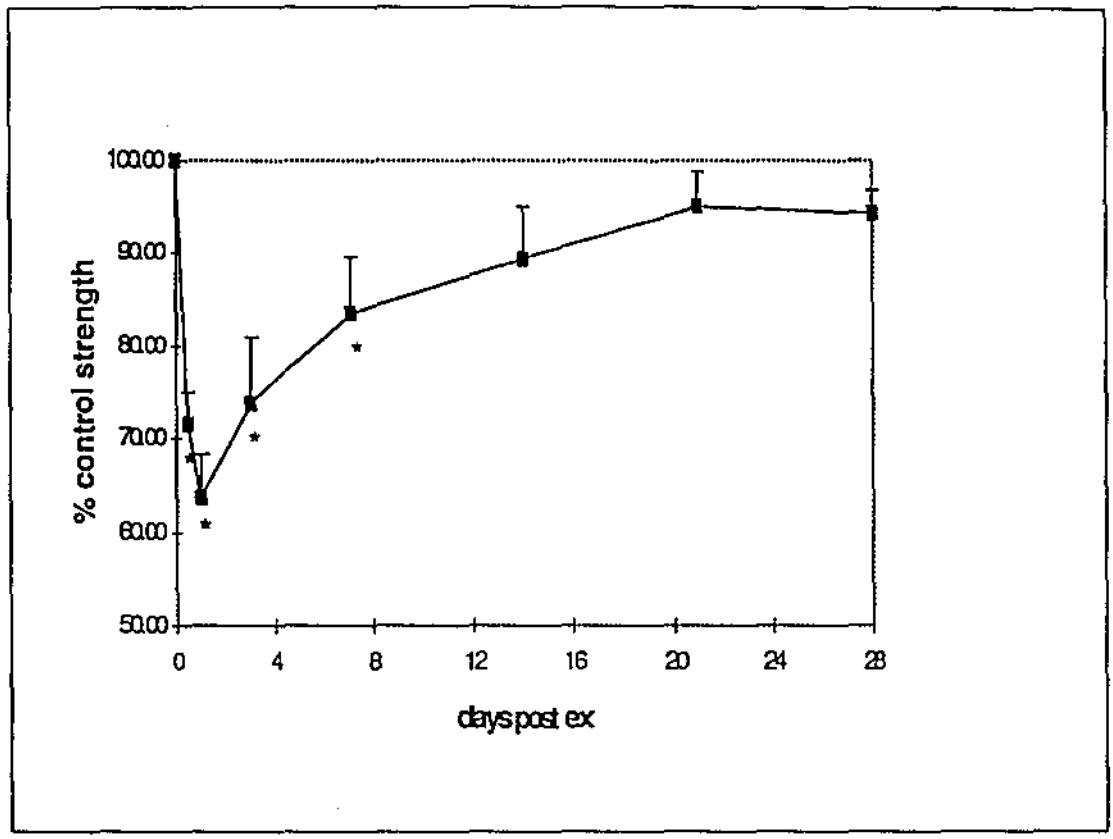



Figure 5.1 Changes in mean elbow flexor strength in seven subjects following exercise protocol. Data expressed as a percentage of pre-exercise strength for each subject (*$p<0.05$)

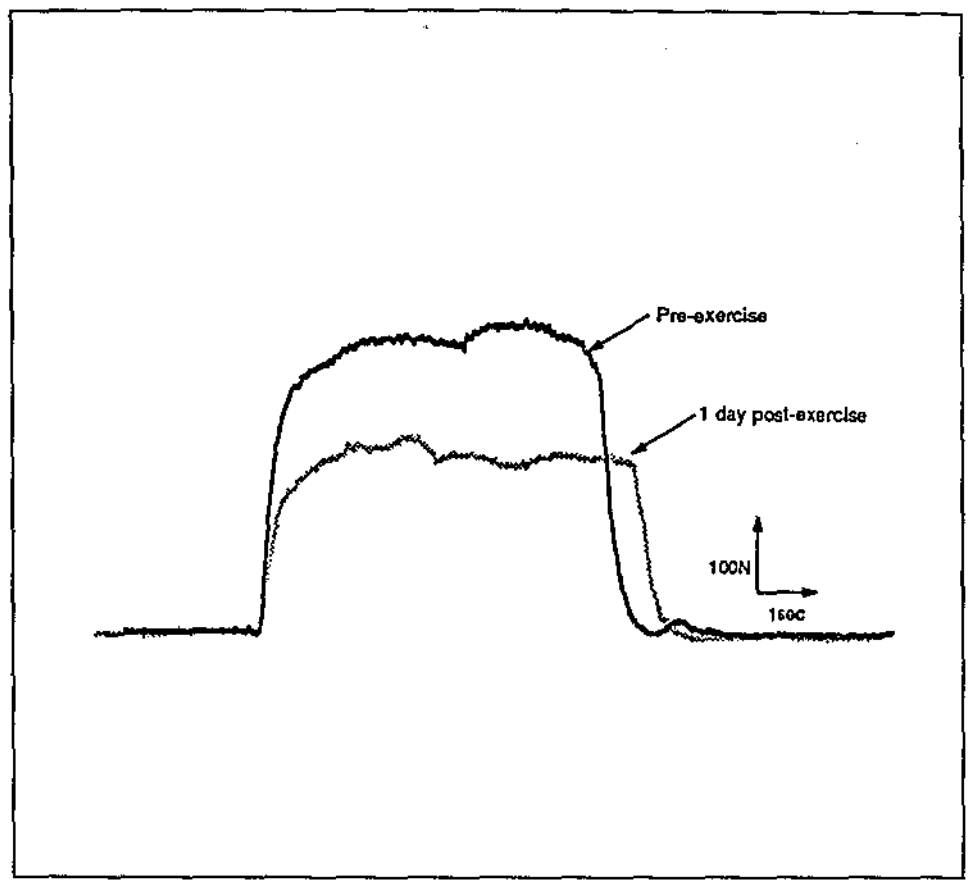



Fig 5.2 MVC force traces pre and one day post-exercise of a typical subject.

## 5.3 Creatine Kinase

Fig. 5.3 illustrates that plasma CK at day seven had significantly increased from pre-test values. However, the responses were quite variable between individuals ranging from peak ck values of 120% to 1864%. By 14 days post-exercise, plasma CK had almost returned to baseline measures.

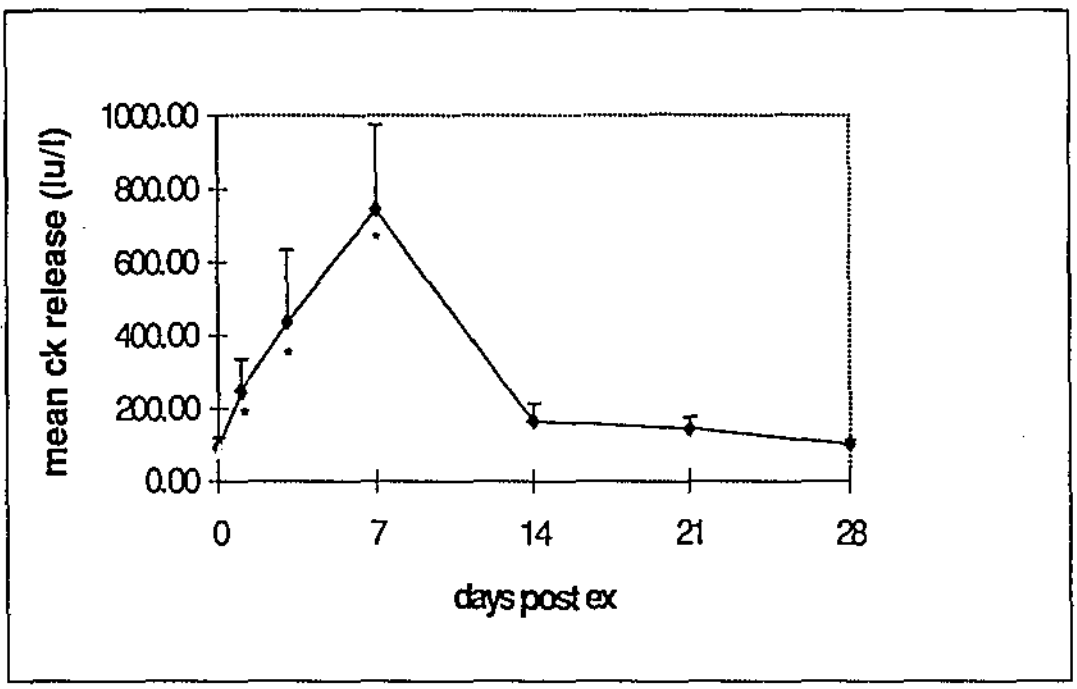



Fig 5.3 Changes in plasma CK following eccentric exercise in seven subjects.(*P<0.05)

## 5.4 Motor Skill Tracking

Accuracy in motor skill tracking was determined by a percentage error from the target cursor as described in section 4.7.5. Figure 5.4 illustrates a typical tracking trace pre and post-exercise.

Significant decreases in skill were noted in the experimental group following the exercise protocol. Accuracy of the experimental group gradually improved over the time course of the study with normal values returning at 14 days. However, control subjects were consistently superior. Figure 5.5 shows normalised mean tracking results for experimental and control groups.

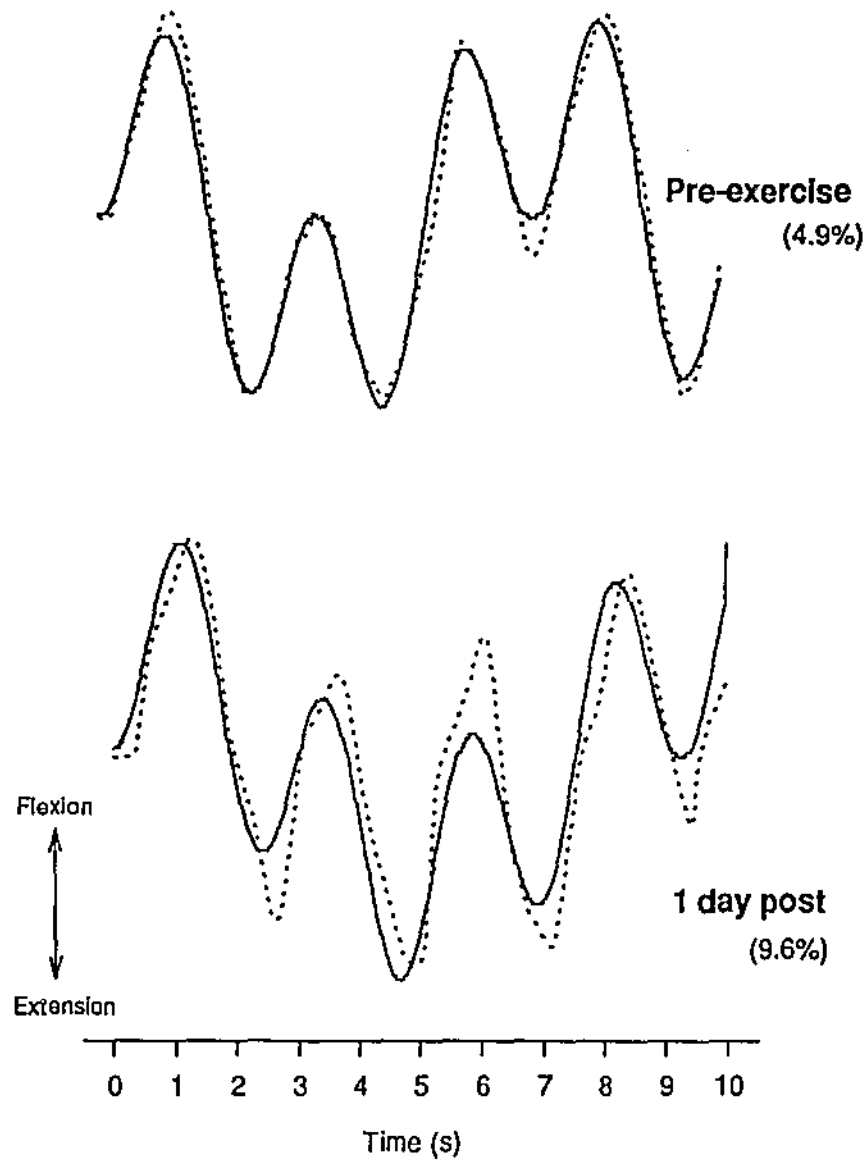



Figure 5.4 Typical tracking trace of elbow flexors and extensors pre and one day post-exercise (% deviation from the target shown in brackets).

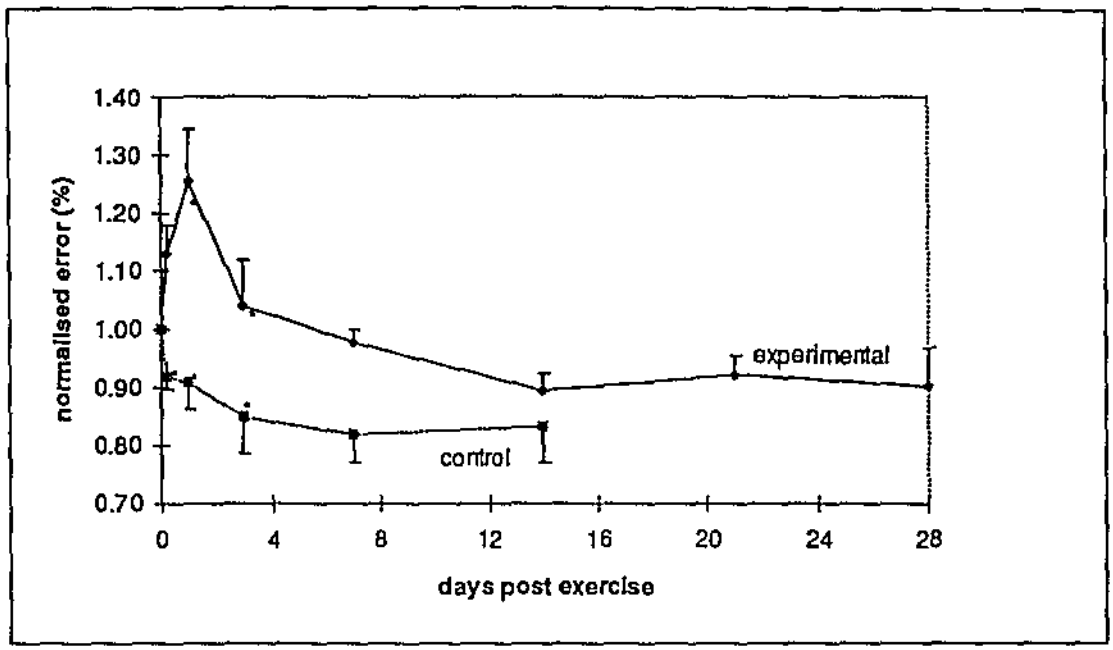



Fig 5.5 Mean change in tracking error of experimental and control subjects normalised to inital values.(*P<0.05)

## 5.5 Voluntary EMG Responses

The average EMG activity recorded during a maximal voluntary contraction (Fig 5.6) showed no consistent change following eccentric exercise. Of the six subjects measured at one and three days post-exercise, two showed a consistent decrease in maximal EMG, two showed increased activity, and two showed no change (see Appendix F for full results). At one day post-exercise (when strength had markedly decreased by 26%) the mean maximal EMG activity had declined by only 12%.

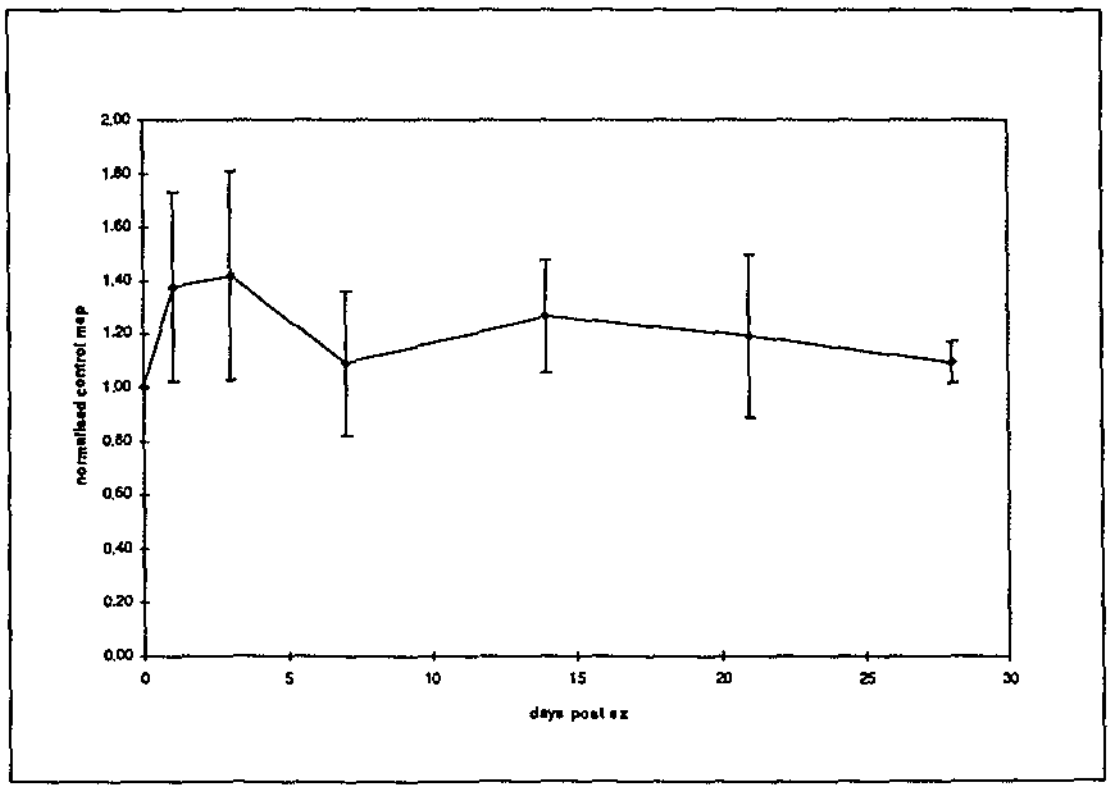



Fig 5.6 Mean EMG changes during time course of study in all subjects.

## 5.6 MEP Responses

The amplitude of the MEP reponse to TMS showed a mean increase at one and three days after eccentric exercise (Fig 5.8). However, not all subjects showed this response (accounting for the large error bars at one, three and seven days).

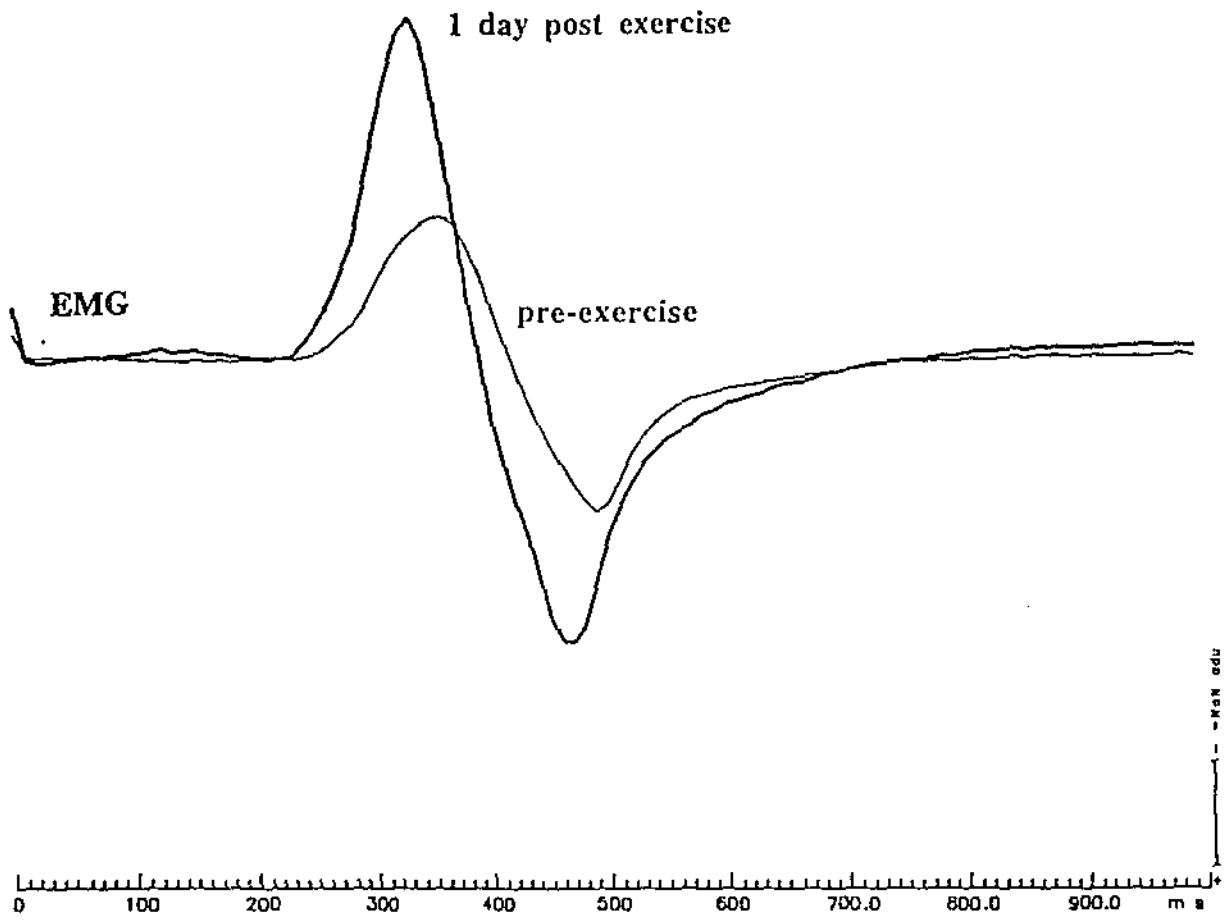



Fig 5.7 Size changes in MEP pre and post exercise (each MEP is average of four responses).

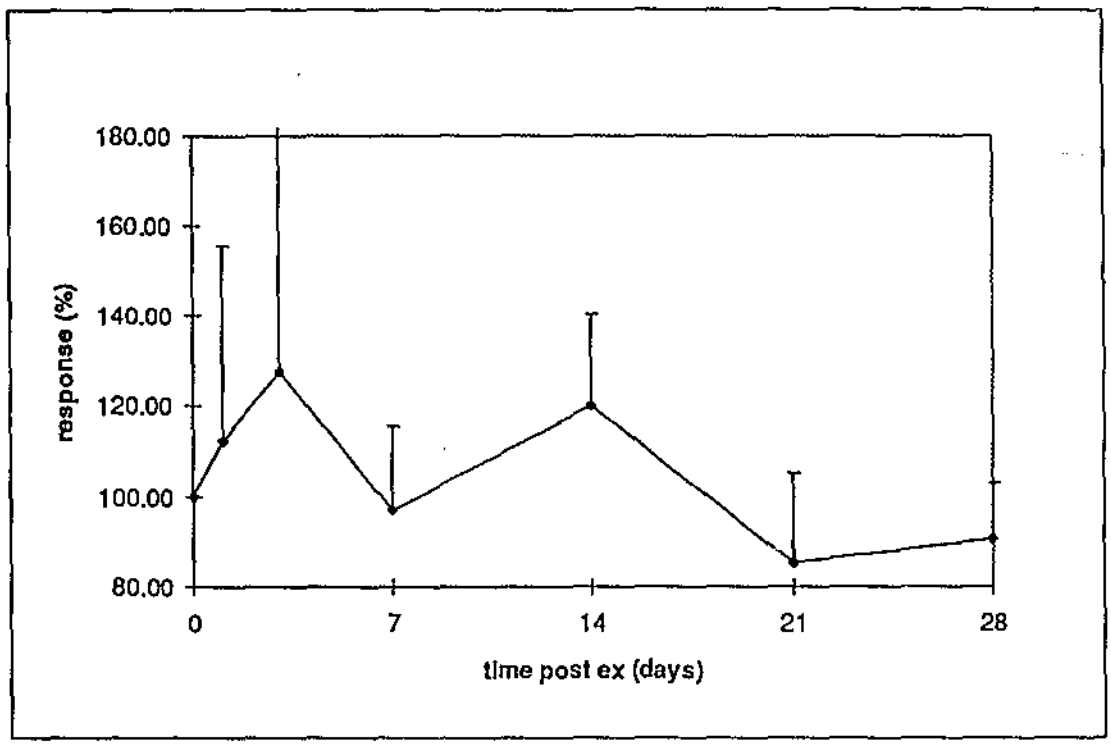



Fig. 5.8 Mean maximal MEP amplitude ± SEM of seven subjects during time course of study.

## 5.7 Threshold Responses

Threshold MEP responses in the seven subjects to TMS did not change during the time course of the study. Slight increases (2.5%) were observed at seven days, however, the increase was not significant. Little variablity was also noted between subjects (as indicated on error bars in Fig 5.9) during the period of the study. Subject threshold responses ranged from 40% - 75% of the maximum stimulator output.

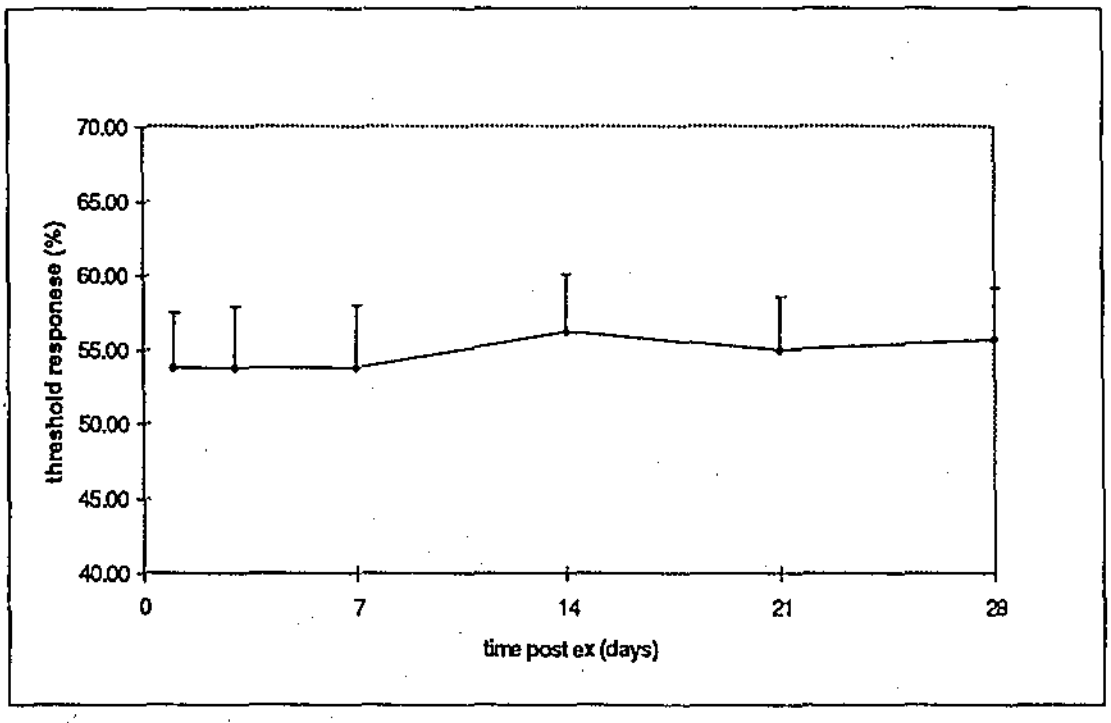



Fig 5.9 Mean threshold responses to TMS for seven subjects pre and post-exercise.

## 5.8 Corticomotor Representation

Figure 5.10 shows a trend towards a medial shift in the centre of gravity location of the area that 'controls' the biceps brachii in the seven days following exercise. However, not all subjects showed a response towards a medial shift; two subjects exhibited a lateral shift (Fig 5.11) which influenced the mean shift at 21 and 28 days laterally.

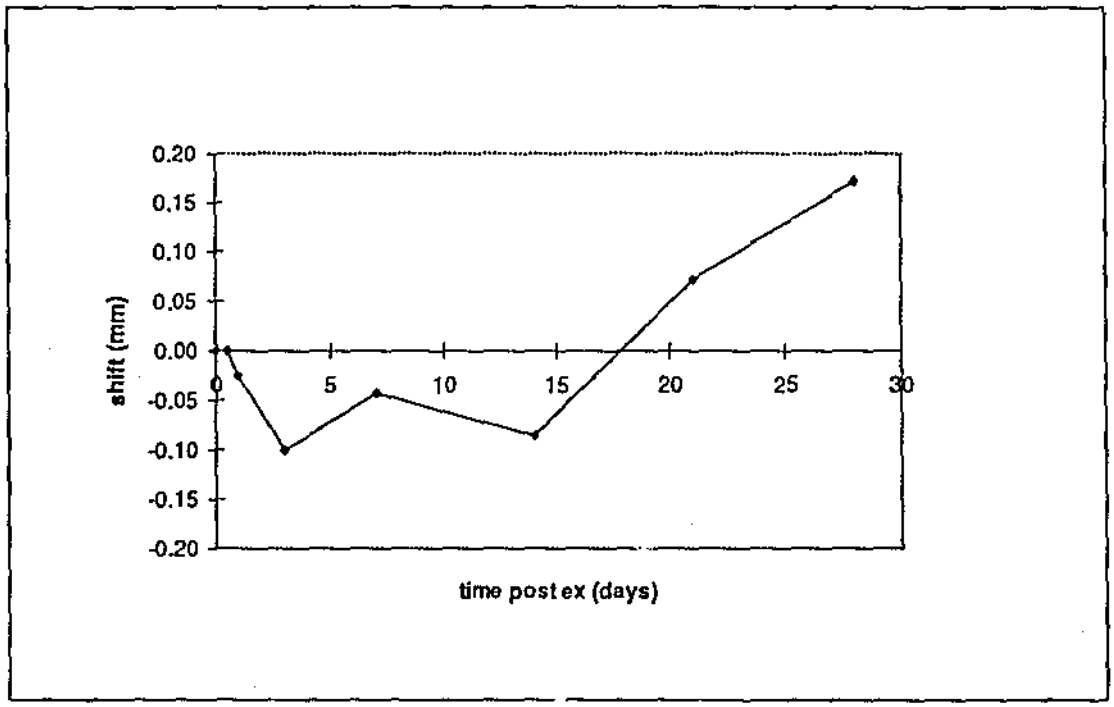
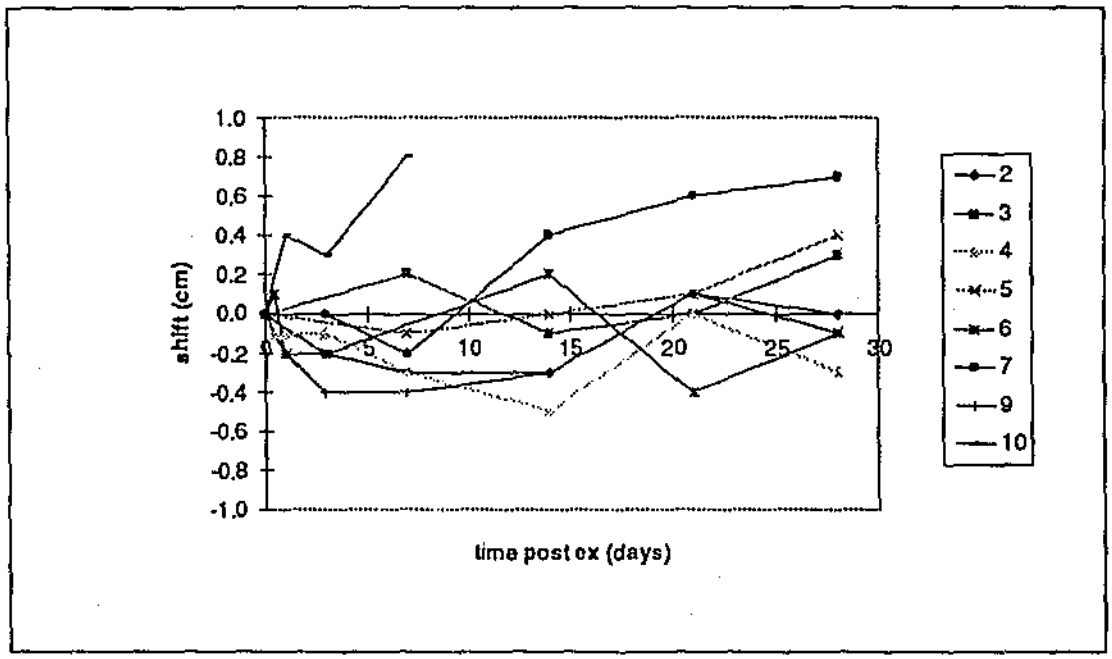



Fig 5.10 Mean shift in Centre of Gravity location following excerise protocol. Negative numbers represent shift medial towards the centre of the head, positive numbers represent lateral shift away from centre.




5.11 Optimal centre of gravity location in seven subjects.

Negative numbers represent shift medial to centre of head, positive numbers represent lateral shift away from centre (number legend represents subjects).

## 5.9 Map Areas

Figure 5.10 illustrates increases in area size of corticomotor representation in a typical subject for the biceps. Corticomotor map areas showed a marked mean increase 14 days after the exercise protocol (table 5.2). However, not all subjects increased in area, two subjects displayed a decrease in map area, although this decrease was not significant.

| | Mean Corticomotor Map Area ($cm^2 \pm$ SEM) | | | | | | |
|---|---|---|---|---|---|---|---|
| | Post exercise (days) | | | | | | |
| *Pre Exercise* | *Immed* | *1* | *3* | *7* | *14* | *21* | *28* |
| mean 11.75 | 11.9 | 10.46 | 11.57 | 12.68 | 13.1 | 12.45 | 11.27 |
| s.e.m ± 2.35 | ± 0.21 | ± 0.724 | ± 1.05 | ± 2.44 | ± 1.78 | ± 1.26 | ± 1.33 |

Table 5.2 Mean corticomotor map areas (in $cm^2$) during time course of study.

# CHAPTER SIX

# DISCUSSION

The objective of this study was to investigate the corticomotor properties of the biceps brachii muscle after a single bout of eccentric exercise resulting in muscle soreness and damage.

The first hypothesis was that functional muscle strength would change after an eccentric exercise insult. All subjects in the study experienced a decline in force immediately following the exercise protocol with strength depressed for up to three weeks (fig 5.1) preceding the exercise bout. This supports the findings of Newham et al (1987) and Ebbling and Clarkson (1989) who eccentrically exercised healthy volunteers and reported force decrements of up to 50% with recovery taking 14 days. Although the degree of force is well characterised the mechanism for weakness is unclear. One possibility by which eccentric exercise results in such a dramatic loss of strength may be a lowered inherent capacity of the muscle to produce force rather than an inability of subjects to fully activate muscle (Jones et al, 1987; Newham et al, 1987). Studies have supported this proposal with superimposed electrical stimulation over damaged muscle, demonstrating full voluntary activation during isometric contractions of painful human muscle (Rutherford et al, 1986; Newham et al, 1987). Furthermore, the changes in human muscle are compatible with those of studies on electrically stimulated animal muscle (Warren et al, 1993; Faulkner et al, 1989). Another reason, as proposed by Friden, Seger, Sjostrom, & Ekblom (1983), is possibly due to greater damage in type II fibres. Type II fibres are preferentially recruited when strength demands increase. Due to damage to these fibres, when a larger recruitment is needed

in maximal contractions, the fibres are not able to produce force, thus strength is impaired.

The second question posed was that plasma CK values would increase as a result of the eccentric exercise insult. The increase in plasma CK (Fig 5.3) was significant, with CK concentration rates differing in subjects ranging from 121% to 1864% increase from pre-exercise values and peaking at day seven post-exercise . This is consistent of the findings of Jones et al. (1986) and Clarkson et al (1986), confirming that muscle damage had indeed taken place in the subjects studied.

Though this investigation is cheifly concerned with motor control properties following eccentric exercise, it is necessary to ascertain that damage has been induced to assess its effect on motor control and corticomotor representation. CK has been shown to be a good indicator that skeletal damage has occurred (Newham et al., 1983), but it is inconsistent in determining the degree of damage sustained, or the muscle mass involved in the damaging exercise (Nosaka, Clarkson, & Apple, 1992). The degree and time course of CK efflux for damaged groups in the study was comparable to that reported previously (Newham et al, 1983; Ebbling & Clarkson 1989), as were the intersubject variability (Nosaka et al, 1992) as seen in Appendix C. Although the exact mechanisms of enzyme efflux following eccentric exercise is unknown, it is generally assumed to reflect some form of membrane damage (Newham et al, 1987; Jones, & Clarkson, 1987).

In Section 1.0, it was hypothesised that myogenic weakness would result in changes in skilled performance using a motor tracking task. Results show that significant decrements in performance were evident, immediately following the exercise, and lasted up to three days. Since there is a learning effect when performing

motor skill tasks, two groups (the experimental and age-matched controls) undertook the skilled performance motor tracking task. Results support this hypothesis with the experimental group showing significantly decreased performance following exercise (Fig 5.5). To further quantify this decrease in performance the control group significantly improved over the first seven days and overall performed better than the experimental group. This is consistent with results reported in Behvehani et al (1990) and Hufschmidt & Lucking (1995) using patients with neuromuscular disorders (myesthenia gravis and multiple sclerosis).

The motor skill tracking control group displayed behaviours of "pre-cognitive tracking" as described by Neilson et al (1988) (section 2.5.1). Similarly, the experimental group also illustrated a learning effect, however, the onset of eccentric exercise damage, disrupted the "pattern generating" mode (Neilson et al, 1988), if only for one day. Learning did resume in the experimental group (Fig 5.5), but it was clearly noticable that the experimental group lagged behind the control group in terms of accuracy.

Significant decreases in skill were found following eccentric exercise. Immediately following the exercise protocol, skill (as measured by the tracking task) declined by 13%, and at one day 27% (appendix C). Improvements in the tracking task returned at day three and continued to improve for the rest of the investigation.

The decline in motor control may be attributed to damage affecting afferent signals as proposed by Saxton et al (1994) and Miles et al (1994). As DOMS has been reported to generally being localised in the distal portion of the muscle in the region of the muscle-tendon junction (Armstrong, 1984), this may also include damage of the Golgi Tendon Organ (GTO). As the GTO acts as a receptor of

the amount of force the muscle is generating (Sage, 1984, p. 168), during time of regeneration from damage, a damaged GTO may not be fully receptive to the forces generated to properly control movment (especially in sudden changes of direction from the muscle). Afferent feedback from the muscle appears to be affected as a result of damage to structures in the muscle-tendon junction as demonstrated by the dramatic decrease in visuomotor tracking skill of the experimental group. This loss of motor performance following exercise is all the more impressive given the significant learning improvement in the control group (Fig 5.5).

Examination of tracking task traces following the exercise protocol revealed that the majority of the tracking error resulted from overshooting of the limb during the turning phases of the task (Fig 5.4) This is in agreement with the findings of Miles et al (1994) that when subjects moved their arm to a pre-determined, stationary target, overshooting occurred following eccentric exercise. The decrements in skill tracking cannot be explained by myogenic weakness. Marked strength loss lasted for up to three weeks following the exercise protocol, however, accuracy loss in skill tracking only lasted three days following the exercise insult. Furthermore, at no point did any of the subjects complain of discomfort during the tracking activity, discounting the possibility that pain may have impaired tracking performance. A more likely explaination is that damage to GTO receptors may have resulted in inappropriate afferent feedback during sudden changes in direction.

The tracking task was performed at lower velocities (5 cycles in 10 seconds) for several reasons. By having the stimulus move at a slower velocity, subjects will learn specific features of the stimulus signal quicker (Neilson et al, 1988). Similarly, slowing of the response movments assists in improving accuracy (viz the speed-

accuracy trade-off described by Fitts' law [Neilson et al, 1988]) thus obtaining reliable baseline measurements sooner than at higher velocities (as a result of quicker learning). By using a lower velocity, the muscle fibre recruitment needed to perform the task at the slower rate (the rate used in this study) would automatically invlovle fibres not damaged and preferentially tend towards type I fibres, leaving out the possibly more affected type II fibres.

The results in this investigation are consistent with the findings of Saxton et al (1994) and Miles et al (1994) regarding impairment of nueromuscular control. However, this is the first known investigation to demonstrate a decline in motor control of a dynamic tracking task following exercise-induced muscle weakness (see appendices G and H).

Changes in EMG activity in the current investigation compliment the research of Newham, Mills, Quigley and Edwards (1983) who reported no change in EMG activity in the days following eccentric damage. Similarly, this study found that following eccentric exercise mean EMG activity (measured under maximal isometric contraction) did not consistently change (Fig 5.6). Of six subjects measured, two showed decreases in EMG, two elicited no change and two produced increased activity. With these findings it can be assumed that DOMS does not affect the electrical activity in the muscle, and that the pain and soreness associated with DOMS does not come from any affected signal. EMG activity is further discussed in relation to cortical excitability, later in this section.

Following myogenic weakness the question was posed that there would be a change in cortical representation of the biceps. Findings are suggestive of changes in cortical representation (Fig 5.10). These changes have included a shift in the optimal

site that "controls" the biceps brachii following eccentric exercise and the area size representing the biceps brachii on the motor cortex. Although it is thought this has not been demonstrated before in regards to short term muscle disease or injury, the findings seem consistent with Cohen et al (1990); and Topka et al (1991) who reported plasticity of the motor cortex with cortical reorganisation occuring in long-term changes in physiology such as stroke, spinal cord lesions and limb amputations. Brasil-Neto et al (1992) have suggested that short term changes are possible and are referred to as "modulation". Modulation of muscular representation on the motor cortex has been reported (Pascual-Leone, Grafman & Hallet, 1994) and it may be postulated in this study, due to the plasticity of the motor cortex, that temporary physiological changes (such as swelling, non-use, and injury brought on by eccentric damage) may elicit a "modulation" response in the motor cortex until functional use of the biceps is regained.

Evidence from this study supports the hypothesis that corticomotor excitability in the biceps would change. Corticomotor excitabiltiy changed in all subjects (Fig 5.8) following the exercise protocol, with a mean increase over the first seven days of 40%. These findings compliment the findings in other studies where short term physiological changes (Brasil-Nato et al, 1992) and changes in physiology long term (Cohen et al, 1991; Topka et al, 1992; Wilson et al, 1993) result in increases in MEP excitability. In relation to this hypothesis, the question was posed as to the reason for the change in MEP size when physiological changes occur. There are two possible explanations to account for the changes in MEP amplitude observed following eccentric exercise. The first being that when muscles are damaged through eccentric exercise, continually stimulated afferent signals from the nerves (due to pain caused by

damage) cause an "overload" effect in the central nervous system (CNS), thus when stimulated (for example by TMS), the excitability of the motor cortex is increased. Alternatively, excitability from the motor cortex arises in the brain itself through a feedback mechanism from the muscle. Receptors in the muscle detect change in function which is interpreted by the sensory cortex. The motor cortex then counters this inhibition of the muscle by "driving" the muscle harder in order to achieve the same output as when the muscle was healthy. This increased neural drive is reflected in an increased excitability of the motor cortex.

However, the second proposal cannot be supported in this study as an increase in neural drive would be reflected in a change in EMG activity. No consistent change in EMG was observed, thus it can be assumed that the changes observed in MEP excitability were due to an altered pattern of afferent signals from the sensory nerves.

To quantify and validate the changes observed in this study, test/retest measurements were obtained prior to the investigation (see section 4.5; table 5.1; and appendix C) using the protocol as described by Thortensson (1976). Test/Retest coefficent of variation (CV) for thresholds and strength were 0% and 2.43% respectively, indicating high reliability. CK had a CV of 6.31% suggesting that reliability was not quite as significant, however, with increases in CK of up to 1864% from pre-test values, test-retest variations are satisfactory. Cortico-motor representations had varying degrees of variability with the optimal centre of gravity position, representational area of the biceps, and motor cortex MEP excitability being 6.23%, 34.61%, and 19.1% respectively. These results are consistent with the findings of Fliess (1986) who demonstrated coefficients of variation from 14% to 37%. This variability may explain the lack of significant alterations observed in these parameters

following exercise in that the changes may have been too subtle to be picked up by the techniques used. This suggests that the protocols employed in this study for measuring cortical representation need to be further controlled in order to increase the sensitivity of measurement. Alternatively, it may be that muscles required in gross movements (such as the biceps brachii) do not possess precisely defined areas of motor cortex as do muscles which are used in fine movments and precision skills such as the adductor pollicis brevis or flexor digiti minimi which have demonstrated reliability of cortciomotor representation in other studies using test/retest protocols (Wilson et al, 1993; Thompson et al, 1995).

This investigation has demonstrated that delayed onset muscle soreness and damage following eccentric exercise was detrimental to neuromuscular function. In particular the characteristic pattern of errors observed in the motor skill tracking task suggests that the central nervous system had difficulty integrating the proprioceptive information arising from the exercised muscle. The most likely explanation for this is that damage to golgi tendon organs or other mechanoreceptors within the muscle-tendon complex had occurred with exercise. Furthermore, the changes in corticomotor excitabilty (as judged by MEP amplitude), although variable, imply that adaptations in the central nervous system had occurred so as to increase the motor drive to the exercised muscle, either in response to a reduction in the perceived force because of muscle weakness, or due to alterations in the pattern of feedback from other afferents (eg. pain receptors).

Functionally, the results and assumptions put forward in this study, can be applied by coaches and athletes in all settings from the beginning sportsperson through to elite athletes in many sports that involve high tensile forces and eccentric

contractions (such as the racquet sports or sports involving lunging and quick changes in direction). With the importance of periodisation of training in recent years, coaches need to understand balancing physical training with skill sessions, especially with athletes who are in the cognitive learning phase or developing skills in the associative phase (Sage, 1984).

The importance of motor skill and muscle damage is unquestionable. From the findings of this investigation, additional research is necessary to further understand the relationship between neuromuscular control and muscle damage.

## REFERENCES


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., Ogilvie, R.W., & Schwane, J.A. (1983). Eccentric exercise-induced injury to rat skeletal muscle. *Journal of Applied Physiology, 54,* 80-93

Armstrong, R.B., Warren, G.l., & Warren, J.A. (1991). Mechanisms of exercise-induced muscle fibre injury. *Sports Medicine, 12* (3), 184-207.

Astrand, P., & Rodahl, K. (1986) *Textbook of work physiology.* NY:McGraw-Hill

Barker, A., Jalinous, R., Freeston, I. (1985). Non-invasive magnetic stimulation of human motor cortex. *Lancet, 1,* 1106-1107.

Behbehani, K., Kondraske, G. V, Tintner, R., Tindall, A.S., Imrhan, S. N. (1990). Evaluation of quantitative measures of upper extremity speed and co-ordination in healthy persons and in three patient populations. *Arch. of Physical and Med Rehab, 71,* 106-111.

Beradelli, A., Inghilleri, M., Cruccu, G., Manfredi, M. (1990). Descending volley after electrical and magnetic transcranial stimulation in man. *Neurosci Lett., 112,* 54-58.

Bigland-Ritchie, B. (1990). Discussion : Nervous system and sensory adaptation. In Bouchard et al. (Eds). *Exercise, fitness, and health.* Champaign : Human Kinetics.

Brasil-Neto, J.P., Cohen, L.G., Pascual-Leone, A., Jabir, F.K., Wall, R.T., Hallet, M. (1992) Rapid reversible modulation of human motor outputs after transient deafferentation of the forearm: A study with transcranial magnetic stimulation. *Neurolog, 42,* 1302-1306.

Byrnes, W. C., Clarkson, P. M., & Katch, F. I. (1985). Muscle soreness following resistance exercise with and without eccentric contractions. *Research Quarterly for Exercise and Sport, 56,* 283-285

Cassell, K. J. (1973). The usefullness of a temporal correlation technique in the assessment of human motor performance on a tracking device. *Medical and Biological Engineering and Computing, 11,* 755-761.

Clarkson, P.M., Byrnes, W.C., McCormick, K. M., Turcotte, L. P., & White, J.S. (1986). Muscle soreness and serum creatine kinase activity following ismetric, eccentric, and concentric exercise. *International Journal of Sports Medicine, 7,* 152-155.

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

Clarkson, P.M., & Newham, D.J. (1994). Associations between muscle sorness, damage, and fatigue. *In Miami Project to Cure Paralysis Symposium:Neural and Neuromuscular Aspects of Muscle Fatigue* (In Press)

Cohen, L., Bandinelli, S., Findley, T., Hallet, M. (1991). Motor reorganisation after upper limb amputation in man:A study with focal magnetic stimulation. *Brain, 114,* 615-627.

Cotman, C.W., Nieto-Sanpedro, M. (1982) Brain function, synapse renewal, and plasticity. *Annual Review of Psychology, 33,* 371-401.

Day, B., Dressler, D., Maertens de Noordhout, A., Marsden, C., Nakshima, K., Rothwell, J., & Thompson, P. (1989). Electic and magnetic stimulation of human

motor cortex : Surface EMG and single motor unit responses. *Journal of Physiology, 412*, 449-473.

Davies, C. T. M., & White, M. J. (1981). Muscle weakness following eccentric work in man. *Pfluger's Arch. ges. Physiology, 392*, 168-171.

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

Edwards, D. J. (1994). *Is there a difference in cortical representation between dominant and non-dominant arm muscles of elite badminton players?* Unpublished honours dissertation, Edith Cowan University, Perth, WA

Evans, J. W. (1987). Exercise-induced skeletal muscle damage. *The Physician and Sportsmedicine 15, 1*, 89-100.

Faulkner, J. A., Jones, D. A., & Round, J. M. (1989). Injury to skeletal muscles of mice by forced lengthening during contractions. *Quarterly Journal of Experimental Physiology, 74*, 661-670.

Fleiss, J. L. (1986). *The design and anaylsis of clinical experiments.* NY: Willey

Francis, K. T. (1983). Delayed muscle soreness : A review. *Journal of Orthapaedic and Sports Physical Therapy, 5, 1*, 10 - 13.

Friden, J., Seger, J., Sjostrom, M., & Ekblom, B. (1983). Adaptive responses in human skeletal muscle subjected to prolonged eccentric training. *International Journal of Sports Medicine, 4*, 177-183.

Furh, P., Cohen, L.G., Dang, N., Findley, T., Haghighi, S., Oro, J. et al. (1992) Physiological analysis of motor reorganisation following lower limb amputation. *Electroencephalography and Clinical Neurophysiology, 85*, 53-60

Gollnick, P.D. (1987). Skeletal muscle morphology : Adaptation to altered demands, *Future Directions in Exercise and Sports Science Research*, (n.d) 275-292.

Guyton, A.C. (1981). *Textbook of medical physiology* (6th ed). Philadelphia : W B Saunders Company.

Hough, T. (1902). Ergographic studies in musclular soreness. *American Journal of Physiology, 7*, 76-92.

Hoehn, M. N., & Yahr, M.D. (1967). Parkinism : Onset, progression, and mortality. *Neurology, 17*, 427-42.

Howell, J. N., Chila, A. G., Ford, G., Douglas, D., & Gates, T. (1985) An elctromyographic study of elbow motion during post-exercise muscle soreness. *Journal of applied physiology 58, 5*, 1713-1718.

Huerta, M.F., Wall, J.T. Kaas J.H. (1986) Changes in topography of somatosensory cortex after nerve loss in adult and neonatal marmoset monkeys (abstract). *Soc. Neurosci. Abstr.* ,12, 954.

Hufschmidt, A., & Lucking, C. H. (1995). Abnormalities of tracking behaviour in Parkinson's disease. *Movement Disorders 10, 3*, 267-276.

James, C., Sacco, P., and Jones, D. A. (1995). Loss of power during fatigue of human leg muscles. *Journal of Physiology 484, 1*, 237-246.

Jones, R.D., & Donaldson, I. M (1986). Measurement of sensory-motor intergrated function in neurological disorders:Three computerised tracking tasks. *Medical and Biological Engineering and Computing, 24*, 536-540.

Jones, D. A., & Round, J. M. (1990). Skeletal muscle in health and disease : A textbook of muscle physiology. Manchester:Manchester University Press.

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

Jones, D.A, Newham, J.M, Round, J.M, Tolfree, E.J. (1986). Experimental human muscle damage : Morphological changes in relation to other indices of damage. *Journal of Physiology, 375,* 435-448.

Kaas,J.H. (1991) Plasticity of sensory and motor maps in adult mammals. *Annual Review of Neuroscience, 14,* 137-167.

Kalaska, J., Pomeranz, B. (1979) Chronic paw denervation causes an age-dependent appearance of novel responses from forearm in "paw cortex" of kittens and adult cats. *Journal of Neurophysiology, 42,* 618-633.

Kelahan, A.M., Ray, R.H., Carson, L.V., Massey, C.E., Doetsch, G.S. (1981) Functional reorganisation of adult raccoon somatosensory cerebral cortex following neonatal digit amputation. *Brain Res., 223,* 151-159.

Knutten, H. G. (1986). Human performance of high-intensity exercise with concentric and eccentric muscle sontractions. *International Journal of Sports Medicine, 7* (Supplement 1), 6-9.

Krendal, E. S., & McRuer, D. T (1960). A servo-system apprach to skill development. *Journal of the Franklin Institute, 269,* 24-42.

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

Kuipers, H., Drukker, J., Frederik, P., Geurten, P., & Kranenburg, G. (1983). Muscle degeneration after exercise in rats. *International Journal of Sports Medicine, 4,* 45-51.

Kuipers, H., Janssen, E., Keizer, H., Verstappen, F. (1985). Serum CPK and amount of muscle damage in rats. *Medicine and Science in Sport and Exercise, 17,* 195.

Levy, W.J., Amassian, V.E., Traad, M., Cadwell, J. (1990) Focal magnetic coil stimulation reveals motor cortical system reorganized in humans after traumatic quadriplegia. *Brain Research, 510,* 130-134.

Mair, J., Koller, A., Artner-Dworzak, E., Haid, C., Wicke, K. Judmaier, W., & Puschendorf, B. (1992). Effects of exercise on plasma myosin heavy chain fragments and MRI of skeletal muscle. *Journal of Applied Physiology, 72* (2), 656-663.

Marieb, E. N. (1994). *Human anatomy and physiology* (3rd ed.). California : Benjamin/Summings Publishing Co.

Marshall, J.F. (1984) Brain function: neural adaptations and recovery from injury. *Annual Review of Psychology, 35,* 277-308.

Merzenich, M.M., Nelson, R.J., Stryker M.P., Cynder, M.S., Shoppmann, A., Zook, J.M. (1984) Somatosensory cortical map changes following digit amputation in adult monkeys. *J. Comp. Neurol., 224,* 591-605.

McCully, K. K., & Falkner, J.A. (1986). Injury to skeletal muscle fibres of mice following lengthening contractions. *Journal of Applied Physiology, 59,* 119-126.

Metzler, J., Mark, P.S. (1979) Functional changes in cat somatic sensory-motor cortex during short term reversible epidermal blocks. *Brain Research, 177,* 379-383.

Miles, M., Ives, J., & Vincent, K. (1993). Neuromuscular control following maximal eccentric exercise. *Medicine and Science in Sport and Exercise, 25,* S176.

Miles, M.P. & Clarkson, P.M. (1994). Exersice-induced muscle pain, soreness, and cramps. *Journal of Sports Medicine and Physical Fitness, 31,* 25-30.

Mortifee, P. , Stewert, H., Schulzer, M., & Eisen, A. (1993). Reliability of trascrannial magnetic stimulation for mapping the human motor cortex. *Electroencephalography and clinical neruophysiology, 93,* 131-137

Neilson, P.D, O'Dwyer, M. J, & Neilson, M. D. (1988). Stochastic prediction in pursuit tracking:An experimental test of adaptive model theory. *Biological Cybernetics, 58,* 113-122

Newham, D. J., Jones, D. A., & Clarkson, P. M. (1987). Repeated high force eccentric exercise: Effects on muscle pain and damage. *Journal of Applied Physiology, 63,* 1381-1386.

Newham, D. J., Jones, D. A., & Edwards, R.H.T. (1983). Large and delayed plasma creatine kinase changes after stepping exercise. *Muscle and Nerve, 6,* 36-41.

Newham, D. J., Mills, K. R., Quigley, B. M., & Edwards, R. H. T. (1983a). Muscle pain and fatigue after concentric and eccentric muscle contractions. *Clinical Science, 64,* 55-62.

Newton, T. & Joyce, A. (1990). Human perspectives. New York:McGraw-Hill.

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

Nosaka, K., & Clarkson, P. (1993). Plasma creatine kinase response to a subsequent bout of eccentric exercise with the contralateral limb. *Medicine and Science in Sports and Exercise, 25,5,* S33.

Pascual-Leone, A., Grafman, J., Hallet, M. (1994) Modulation of cortical motor output maps during development of implicit and explicit knowledge. *Science, 263,* 1287-1289.

Pons, T.P., Garraghty, P.E., Ommaya, A.K., Kaas, J.H., Taub, E., Mishkin, M. (1991) Massive cortical reorganisation after sensory deafferentation in adult macaques. *Science, 252,* 1857-1860.

Rutherford, O. M., & Jones, D. A. (1986). The role of earning and co-ordination In strength training. *European Journal of Applied Physiology, 55,* 100-105.

Sacco, P., Jones, D.A., Dick, J.R.T., & Vrbova, G. (1992). Contractile properties and suseptibility to exercise induced damage of normal and mdx mouse tibialis anterior muscle. *Clinical Science, 82,* 227-236.

Sage. G. H. Motor learning and control : A neruopsychological approach. (1984). USA: Wm. C. Brown Publishers.

Saxton, J., Carkson, P., James, R., Miles, M., Westerfer, M., Clark, S., Donnelly, A. (1994). Neuromuscular function following maximum voluntary eccentric exercise. *Medicine and Science in Sport and Exercise, 26,* S115.

Schwane, J., Watrous, B., Johnson, S., Armstrong, R. (1983). Is lactic acid related to delayed-onset muscle soreness? *Physician and Sportsmedicine, 11,* 124-131.

Shumate, J., Brooke, M., Carroll, J., & Davis, J. (1979). Increased serum CK after exercise: A sex linked phenomenon. *Neurology (Minneap), 29,* 902-904.

Stauber, W.T. (1989). Eccentric action of muscles : Physiology, injury, and adaptation. *Exercise and Sports Science Reviews, 17,* 157-185

Tabachnick, B. G., & Fidell, L. S. (1989). *Using multivariate statistics.* New York: Harper and Row.

Thickbroom, G., Wilson, S. & Mastaglia, F. (1992). Topographic mapping of the corticomotor representation using transcranial magnetic stimulation : methodology. *Australian, New Zealand Journal of Medicine, 22,* 438.

Thompson, M.L., Thickbroom, G.T., Sacco, P., Wilson, S.A., Stell, R. & Mastaglia, F.L. (1995) Corticomotor representation of intrinsic hand muscles in writer's cramp. *Journal of Neurological Sciences* (In Press).

Thorstensson, A. (1976). Muscle strength, fibre types and enzyme activites in man. *Acta Physiologica Scandanavica, 98 (Supp. 43),* 1-45.

Tidus, P., & Ianuzzo, C. (1983). Effects of intensity and duration of muscualr exercise on delayed soreness and serum enzyme activities. *Medicine and Science in Sport and Exercise, 15,* 461-465.

Topka, H., Cohen, L.G., Cole, R.A., Hallet, M. (1991) Reorganization of corticospinal pathways following spinal cord injury. *Neurology, 41,* 1276-1283.

Valls-Sole, J., Tolosa, E.S., Pujol, M. (1992). Myokimic discharges and enhanced facial nerve reflex responses after recovery from idiopathic facial palsy. *Muscle and Nerve, 15,* 37-42.

Van der Meulen, J., Kuipers, H., & Drukker, J. (1991). Relationahip between exercise-induced muscle damage adn enzyme release in rats. *Journal of Applied Physiology, 71* (3), 999-1004.

Warren, G. L, Hayes, D. A., Lowe, D. A., Prior, B. M., & Armstrong, R. B. (1993). Materials fatigue initiates eccentric contraction-induced injury in rat soleus muscle. *Journal of Physiology, 464*, 477-489.

Wilson, S.A., Thickbroom, G.W., & Mastaglia, F.L. (1993). Transcranial magnetic stimulation mapping of the motor cortex in normal subjects - The representation of two hand muscles. *Journal of the Neurological Sciences, 118* (19993), 134-144.

# APPENDIX A

Informed Consent Sheets

**Australian Neuromuscular Research Institute: Consent Form**

*Eccentric exercise of elbow flexor muscles*

Procedure: The procedure is non-invasive. Electrode discs will be taped onto the both biceps muscles. The activity in the muscles will be recorded via these electrodes and the information will be fed into the computer. Magnetic stimulation will be used. A snugly fitting cap with pre-marked spacings will be placed on the head. The magnetic coil will be positioned on various sites of the cap and that part of the brain will be stimulated. Each stimulation will be very short, much less than 1 second. This is not painful, but some small movements may be noticed in the target muscle. Occasionally, tingling or a tap on the scalp may be felt. During the session you will be asked to contract muscles in the arm maximally for 5 seconds and submaximally also during the stimulation. You will be shown how to perform these contractions and will be given a chance to practice. We will start the session with a few practice runs, and there will be a rest period after each set of trials. There are very few possible discomforts associated with these procedures. On rare occasions magnetic stimulation may cause a headache. If this occurs, or for any other reason you wish to stop the session, we will stop the session.

I understand that I am free to withdraw from the study at any time.

I acknowledge that I have read the above statement which explains the nature and object and the possible risks of the investigation and the statement has been explained to me to my satisfaction. Before signing this document I have been given the opportunity to ask any questions relating to any possible physical or mental harm I might suffer as a result of my participation and I have recieved satisfactory answers. I agree that research data gathered from the result of the study may be published provided my name is not used.

In the light of the forgoing, I hereby release the Australian Neuromuscular Research Institute or any employee, member or representitive thereof, from all or any claim that I may have arising out of my participation on this experiment. I understand that this document in no way limits my rights at law from any damage that might arise from negligence on the part of the investigators.

To the best of my knowledge I am not pregnant. I do not have a cardiac pacemaker and I do not have metal implants in my head.

I,....., age.....years, agree to participate as a subject in a study of the type described above.

Signed.....

Witness..... Date.....

**Australian Neuromuscular Research Institute: Consent Form**

*Mapping of elbow flexor muscles*

Procedure: The procedure is non-invasive. Electrode discs will be taped onto the both biceps muscles. The activity in the muscles will be recorded via these electrodes and the information will be fed into the computer. You will be asked to perform 7 sets of 5 maximal 'eccentric' contractions with your non-preferred arm. Some localised discomfort may be experienced in the week following the eccentric contractions, however, you possibly may some discomfort during the exercise. If this occurs and you wish to stop, or for any other reason you wish to stop the session, we will stop the session.

I understand that I am free to withdraw from the study at any time.

I acknowledge that I have read the above statement which explains the nature and object and the possible risks of the investigation and the statement has been explained to me to my satisfaction. Before signing this document I have been given the opportunity to ask any questions relating to any possible physical or mental harm I might suffer as a result of my participation and I have recieved satisfactory answers. I agree that research data gathered from the result of the study may be published provided my name is not used.

In the light of the forgoing, I hereby release the Australian Neuromuscular Research Institute or any employee, member or representitive thereof, from all or any claim that I may have arising out of my participation on this experiment. I understand that this document in no way limits my rights at law from any damage that might arise from negligence on the part of the investigators.

To the best of my knowledge I am not pregnant and I do not have a cardiac pacemaker.

I,....., age.....years, agree to participate as a subject in a study of the type described above.

Signed.....

Witness..... Date.....

# APPENDIX B

Data Test Sheets

SKILL TRACKING

NAME - DATE - FILENAME -

ATTEMPT 1-

ATTEMPT 2-

ATTEMPT 3-

ATTEMPT 4-

ATTEMPT 5-

MEAN SCORE -

COMMENTS -

STRENGTH

NAME - DATE - FILE NAME-

MVC 1-

MVC 2-

MVC 3-

CK READING-

COMMENTS-

# APPENDIX C

Test/Retest Data

# TEST.RETEST

| COG | SUBJECT | TEST 1 | TEST 2 | DIFF |
|---|---|---|---|---|
| | 1 | 5.4 | 5.2 | 0.2 |
| | 2 | 5.5 | 5.3 | 0.2 |
| | 3 | 3.5 | 4.4 | -0.9 |
| | 4 | 4.9 | 4.9 | 0 |
| | 5 | | | |
| | 6 | 5 | 4.9 | 0.1 |
| | 7 | 4.4 | 4 | 0.4 |
| | 9 | 4.9 | 4.8 | 0.1 |
| | 10 | | | |
| | 11 | | | |
| | MEAN | 4.8 | 4.785714 | 0.014286 |
| | S.D | 0.678233 | 0.452506 | 0.422013 |
| | S.E.M | 0.242226 | 0.161609 | 0.150719 |
| | M.E | 0.298409 | | |
| | C.V | 6.229 | | |

| STR | SUBJECT | TEST 1 | TEST 2 | DIFF |
|---|---|---|---|---|
| | 1 | | | |
| | 2 | 280 | 271 | 9 |
| | 3 | 166 | 163 | 3 |
| | 4 | 261 | 241 | 20 |
| | 5 | | | |
| | 6 | 158 | 149 | 9 |
| | 7 | 270 | 266 | 4 |
| | 9 | 255 | 234 | 21 |
| | 10 | | | |
| | 11 | | | |
| | MEAN | 231.6667 | 220.6667 | 11 |
| | S.D | 54.67967 | 52.23281 | 7.771744 |
| | S.E.M | 19.52845 | 18.65458 | 2.775623 |
| | M.E | 5.495503 | | |
| | C.V | 2.43 | | |

| CK | SUBJECT | TEST 1 | TEST 2 | DIFF |
|---|---|---|---|---|
| | 1 | | | |
| | 2 | | | |
| | 3 | 120 | 102 | 18 |
| | 4 | 51 | 51 | 0 |
| | 5 | 122 | 116 | 6 |
| | 6 | 36 | 28 | 8 |
| | 7 | | | |
| | 9 | 209 | 217 | -8 |
| | 10 | 120 | 124 | -4 |
| | 11 | | | |
| | MEAN | 109.6667 | 102.8 | 4.8 |
| | S.D | 61.80831 | 66.13522 | 9.352362 |
| | S.E.M | 22.07439 | 23.61972 | 3.340129 |
| | M.E | 6.613181 | | |
| | C.V | 6.31 | | |

| AREA | SUBJECT | TEST 1 | TEST 2 | DIFF |
|---|---|---|---|---|
| | 1 | 11 | 11.2 | -0.20 |
| | 2 | 8.70 | 8.60 | 0.10 |
| | 3 | 7.00 | 13.00 | -6.00 |
| | 4 | 14.80 | 9.90 | 4.90 |
| | 5 | | | |
| | 6 | 8.00 | 13.50 | -5.50 |
| | 7 | 17.10 | 8.70 | 8.40 |
| | 9 | 11.10 | 8.40 | 2.70 |
| | 10 | | | |
| | 11 | | | |
| | MEAN | 11.1 | 10.35 | 0.766667 |
| | S.D | 3.69414 | 2.135193 | 5.250306 |
| | S.E.M | 1.319336 | 0.762569 | 1.875109 |
| | M.E | 3.7125 | | |
| | C.V | 34.61 | | |

| MEP MAX | SUBJECT | TEST 1 | TEST 2 | DIFF |
|---|---|---|---|---|
| | 1 | 4.4 | 4.5 | -0.1 |
| | 2 | 5.04 | 4.51 | 0.53 |
| | 3 | 9 | 9.8 | -0.8 |
| | 4 | 7.13 | 9.69 | -2.56 |
| | 5 | 1.58 | 2.29 | -0.71 |
| | 6 | 2.39 | 2.24 | 0.15 |
| | 7 | 12.88 | 9.88 | 3 |
| | 9 | | | |
| | 10 | | | |
| | 11 | | | |
| | MEAN | 6.06 | 6.401667 | -0.065 |
| | S.D | 3.950447 | 3.544103 | 1.682102 |
| | S.E.M | 1.410874 | 1.265751 | 0.600751 |
| | M.E | 1.19 | | |
| | C.V | 19.1 | | |

| EMG | SUBJECT | TEST 1 | TEST 2 | DIFF |
|---|---|---|---|---|
| | 1 | 343 | 533 | -190 |
| | 2 | 378 | 442 | -64 |
| | 3 | 122 | 166 | -44 |
| | 4 | 944 | 730 | 214 |
| | 5 | 418 | 422 | -4 |
| | 6 | 273 | 333 | -60 |
| | 7 | 771 | 616 | 155 |
| | 9 | 559 | 577 | -18 |
| | 10 | | | |
| | 11 | | | |
| | MEAN | 476 | 477.375 | -1.375 |
| | S.D | 269.9524 | 176.5429 | 128.6623 |
| | S.E.M | 96.41156 | 63.05105 | 45.95081 |
| | M.E | 90.97 | | |
| | C.V | 19.08 | | |

# APPENDIX D

Statistical Data

NORMALISED STRENGTH

| pre | immed | 1d | 3d | 7d | 14d | 21d | 28d |
|---|---|---|---|---|---|---|---|
| 100.00 | 76.43 | 85.00 | 93.57 | 98.21 | 105.00 | 104.64 | 105.36 |
| 100.00 | 69.57 | 71.00 | 74.00 | 84.47 | 86.96 | 93.79 | 92.55 |
| 100.00 | 87.66 | 75.74 | 87.23 | 90.64 | 102.55 | 110.21 | 98.30 |
| 100.00 | 69.70 | 69.00 | 72.00 | 80.00 | 84.85 | 92.12 | 89.70 |
| 100.00 | 75.00 | 71.15 | 83.97 | 82.69 | 98.08 | 101.28 | 92.95 |
| 100.00 | 65.73 | 56.99 | 60.84 | 83.92 | 85.31 | 87.41 | 87.76 |
| 100.00 | 71.37 | 67.06 | 71.37 | 98.82 | 90.00 | 83.92 | 93.73 |
| 100.00 | 57.09 | 47.16 | 46.10 | 47.87 | 62.77 | 86.88 | 96.00 |

Stats analysis for strength

----- Wilcoxon Matched-Pairs Signed-Ranks Test

IMMED
with PRE

Mean Rank Cases

.00 0 - Ranks (PRE LT IMMED)
4.50 8 + Ranks (PRE GT IMMED)
0 Ties (PRE EQ IMMED)
-
8 Total

Z = -2.5205 2-Tailed P = .0117

----- Wilcoxon Matched-Pairs Signed-Ranks Test

ONEDAY
with PRE

Mean Rank Cases

.00 0 - Ranks (PRE LT ONEDAY)
4.50 8 + Ranks (PRE GT ONEDAY)
0 Ties (PRE EQ ONEDAY)
-
8 Total

Z = -2.5205 2-Tailed P = .0117

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with THREEDAY

| Mean Rank | Cases | |
|---|---|---|
| 4.50 | 8 | - Ranks (THREEDAY LT PRE) |
| .00 | 0 | + Ranks (THREEDAY GT PRE) |
| | 0 | Ties (THREEDAY EQ PRE) |
| | - | |
| | 8 | Total |

Z = -2.5205 2-Tailed P = .0117

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with SEVENDAY

| Mean Rank | Cases | |
|---|---|---|
| 4.50 | 8 | - Ranks (SEVENDAY LT PRE) |
| .00 | 0 | + Ranks (SEVENDAY GT PRE) |
| | 0 | Ties (SEVENDAY EQ PRE) |
| | - | |
| | 8 | Total |

Z = -2.5205 2-Tailed P = .0117

----- Wilcoxon Matched-Pairs Signed-Ranks Test

FOURTEEN
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 2.50 | 2 | - Ranks (PRE LT FOURTEEN) |
| 5.17 | 6 | + Ranks (PRE GT FOURTEEN) |
| | 0 | Ties (PRE EQ FOURTEEN) |
| | - | |
| | 8 | Total |

Z = -1.8204 2-Tailed P = .0687

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYONE

| Mean Rank | Cases | |
|---|---|---|
| 5.60 | 5 | - Ranks (TWENTYON LT PRE) |
| 2.67 | 3 | + Ranks (TWENTYON GT PRE) |
| | 0 | Ties (TWENTYON EQ PRE) |
| | - | |
| | 8 | Total |

Z = -1.4003 2-Tailed P = .1614

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYEIGHT

| Mean Rank | Cases | |
|---|---|---|
| 4.71 | 7 | - Ranks (TWENTYEI LT PRE) |
| 3.00 | 1 | + Ranks (TWENTYEI GT PRE) |
| | 0 | Ties (TWENTYEI EQ PRE) |
| | - | |
| | 8 | Total |

Z = -2.1004 2-Tailed P = .0357

CK

| PRE | 1D | 3D | 7D | 14D | 21D | 28D |
|---|---|---|---|---|---|---|
| 24.00 | 150.00 | 202.00 | 248.00 | 126.00 | 191.00 | 109.00 |
| 120.00 | 420.00 | 600.00 | 1270.00 | 170.00 | 105.00 | 92.00 |
| 50.90 | 80.00 | 145.00 | 99.00 | 81.00 | 91.00 | 105.00 |
| 122.00 | 220.00 | 500.00 | 1050.00 | 123.00 | 120.00 | 116.00 |
| 21.90 | 32.00 | 117.00 | 281.00 | 39.00 | 54.00 | 42.00 |
| 74.00 | 434.00 | 1400.00 | 1380.00 | 433.00 | 282.00 | 93.00 |
| 209.00 | 441.00 | 258.00 | 253.00 | 117.00 | 109.00 | 100.00 |
| 120.00 | 200.00 | 490.00 | 1380.00 | 228.00 | 124.00 | 112.00 |

STATS ANALYSIS FOR CK

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

ONEDAY
with PRE

Mean Rank Cases

4.50 8 - Ranks (PRE LT ONEDAY)
.00 0 + Ranks (PRE GT ONEDAY)
0 Ties (PRE EQ ONEDAY)
-
8 Total

Z = -2.5205 2-Tailed P = .0117

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with THREEDAY

Mean Rank Cases

.00 0 - Ranks (THREEDAY LT PRE)
4.50 8 + Ranks (THREEDAY GT PRE)
0 Ties (THREEDAY EQ PRE)
-
8 Total

Z = -2.5205 2-Tailed P = .0117

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with SEVEN

| Mean Rank | Cases | |
|---|---|---|
| .00 | 0 | - Ranks (SEVEN LT PRE) |
| 4.50 | 8 | + Ranks (SEVEN GT PRE) |
| | 0 | Ties (SEVEN EQ PRE) |
| | - | |
| | 8 | Total |

Z = -2.5205 2-Tailed P = .0117

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

FOURTEEN
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 4.43 | 7 | - Ranks (PRE LT FOURTEEN) |
| 5.00 | 1 | + Ranks (PRE GT FOURTEEN) |
| | 0 | Ties (PRE EQ FOURTEEN) |
| | - | |
| | 8 | Total |

Z = -1.8204 2-Tailed P = .0687

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYON

| Mean Rank | Cases | |
|---|---|---|
| 3.33 | 3 | - Ranks (TWENTYON LT PRE) |
| 5.20 | 5 | + Ranks (TWENTYON GT PRE) |
| | 0 | Ties (TWENTYON EQ PRE) |
| | - | |
| | 8 | Total |

Z = -1.1202 2-Tailed P = .2626

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYEI

| Mean Rank | Cases | |
|---|---|---|
| 4.00 | 4 | - Ranks (TWENTYEI LT PRE) |
| 5.00 | 4 | + Ranks (TWENTYEI GT PRE) |
| | 0 | Ties (TWENTYEI EQ PRE) |
| | - | |
| | 8 | Total |

Z = -.2801 2-Tailed P = .7794

COMBINED TRACKING

| group | pre | immed | 1d | 3d | 7d | 14d |
|---|---|---|---|---|---|---|
| 2.00 | 1.00 | .99 | .94 | 1.00 | .97 | 1.07 |
| 2.00 | 1.00 | .98 | 1.01 | .89 | .82 | .75 |
| 2.00 | 1.00 | .87 | .87 | .77 | .75 | .76 |
| 2.00 | 1.00 | .92 | .74 | .58 | .60 | .60 |
| 2.00 | 1.00 | .94 | .92 | .92 | .87 | .87 |
| 2.00 | 1.00 | .87 | 1.09 | 1.05 | .95 | 1.01 |
| 2.00 | 1.00 | .88 | .79 | .73 | .77 | .78 |
| 1.00 | 1.00 | 1.14 | 1.30 | .92 | 1.03 | .89 |
| 1.00 | 1.00 | 1.01 | 1.30 | 1.00 | .96 | .87 |
| 1.00 | 1.00 | 1.29 | 1.43 | 1.14 | .88 | .94 |
| 1.00 | 1.00 | 1.19 | 1.30 | 1.00 | 1.01 | .89 |
| 1.00 | 1.00 | 1.33 | 1.48 | .97 | .92 | .81 |
| 1.00 | 1.00 | .93 | .99 | .96 | .95 | .75 |
| 1.00 | 1.00 | 1.06 | 1.20 | 1.38 | 1.07 | .96 |
| 1.00 | 1.00 | 1.11 | 1.18 | .87 | .99 | 1.03 |

STATISTICAL TEST FOR TRACKING (2 IND GRPS)

----- Mann-Whitney U - Wilcoxon Rank Sum W Test

PRE
by GROUP

Mean Rank Cases

8.00 8 GROUP = 1.00
8.00 7 GROUP = 2.00
--
15 Total

| | | Exact | Corrected for ties | |
|---|---|---|---|---|
| U | W | 2-Tailed P | Z | 2-Tailed P |
| 28.0 | 56.0 | 1.0000 | .0000 | 1.0000 |

----- Mann-Whitney U - Wilcoxon Rank Sum W Test

IMMED
by GROUP

Mean Rank Cases

11.13 8 GROUP = 1.00
4.43 7 GROUP = 2.00
--
15 Total

| | | Exact | Corrected for ties | |
|---|---|---|---|---|
| U | W | 2-Tailed P | Z | 2-Tailed P |
| 3.0 | 31.0 | .0022 | -2.8958 | .0038 |

----- Mann-Whitney U - Wilcoxon Rank Sum W Test

ONEDAY
by GROUP

| Mean Rank | Cases | |
|---|---|---|
| 11.25 | 8 | GROUP = 1.00 |
| 4.29 | 7 | GROUP = 2.00 |
| | -- | |
| | 15 | Total |

| | | Exact | Corrected for ties | |
|---|---|---|---|---|
| U | W | 2-Tailed P | Z | 2-Tailed P |
| 2.0 | 30.0 | .0012 | -3.0197 | .0025 |

----- Mann-Whitney U - Wilcoxon Rank Sum W Test

THREEDAY
by GROUP

| Mean Rank | Cases | |
|---|---|---|
| 9.81 | 8 | GROUP = 1.00 |
| 5.93 | 7 | GROUP = 2.00 |
| | -- | |
| | 15 | Total |

| | | Exact | Corrected for ties | |
|---|---|---|---|---|
| U | W | 2-Tailed P | Z | 2-Tailed P |
| 13.5 | 41.5 | .0939 | -1.6856 | .0919 |

----- Mann-Whitney U - Wilcoxon Rank Sum W Test

SEVEN
by GROUP

| Mean Rank | Cases | |
|---|---|---|
| 10.69 | 8 | GROUP = 1.00 |
| 4.93 | 7 | GROUP = 2.00 |
| | -- | |
| | 15 | Total |

| | | Exact | Corrected for ties | |
|---|---|---|---|---|
| U | W | 2-Tailed P | Z | 2-Tailed P |
| 6.5 | 34.5 | .0093 | -2.4904 | .0128 |

----- Mann-Whitney U - Wilcoxon Rank Sum W Test

FOURTEEN
by GROUP

| Mean Rank | Cases | |
|---|---|---|
| 9.00 | 8 | GROUP = 1.00 |
| 6.86 | 7 | GROUP = 2.00 |
| | -- | |
| | 15 | Total |

| | | Exact | | Corrected for ties |
|---|---|---|---|---|
| U | W | 2-Tailed P | Z | 2-Tailed P |
| 20.0 | 48.0 | .3969 | -.9283 | .3532 |

STATISTICAL TEST FOR EXPERIEMENTAL GROUP (WITHIN GROUP)

----- Wilcoxon Matched-Pairs Signed-Ranks Test

IMMED
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 4.71 | 7 | - Ranks (PRE LT IMMED) |
| 3.00 | 1 | + Ranks (PRE GT IMMED) |
| | 0 | Ties (PRE EQ IMMED) |
| | - | |
| | 8 | Total |

Z = -2.1004 2-Tailed P = .0357

----- Wilcoxon Matched-Pairs Signed-Ranks Test

ONEDAY
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 5.00 | 7 | - Ranks (PRE LT ONEDAY) |
| 1.00 | 1 | + Ranks (PRE GT ONEDAY) |
| | 0 | Ties (PRE EQ ONEDAY) |
| | - | |
| | 8 | Total |

Z = -2.3805 2-Tailed P = .0173

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with THREEDAY

| Mean Rank | Cases | |
|---|---|---|
| 2.50 | 4 | - Ranks (THREEDAY LT PRE) |
| 5.50 | 2 | + Ranks (THREEDAY GT PRE) |
| | 2 | Ties (THREEDAY EQ PRE) |
| | - | |
| | 8 | Total |

Z = -.1048 2-Tailed P = .9165

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with SEVEN

| Mean Rank | Cases | |
|---|---|---|
| 5.10 | 5 | - Ranks (SEVEN LT PRE) |
| 3.50 | 3 | + Ranks (SEVEN GT PRE) |
| | 0 | Ties (SEVEN EQ PRE) |
| | - | |
| | 8 | Total |

Z = -1.0502 2-Tailed P = .2936

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

FOURTEEN
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 1.00 | 1 | - Ranks (PRE LT FOURTEEN) |
| 5.00 | 7 | + Ranks (PRE GT FOURTEEN) |
| | 0 | Ties (PRE EQ FOURTEEN) |
| | - | |
| | 8 | Total |

Z = -2.3805 2-Tailed P = .0173

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYON

| Mean Rank | Cases | |
|---|---|---|
| 4.42 | 6 | - Ranks (TWENTYON LT PRE) |
| 1.50 | 1 | + Ranks (TWENTYON GT PRE) |
| | 1 | Ties (TWENTYON EQ PRE) |
| | - | |
| | 8 | Total |

Z = -2.1129 2-Tailed P = .0346

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYEI

| Mean Rank | Cases | |
|---|---|---|
| 5.60 | 5 | - Ranks (TWENTYEI LT PRE) |
| 2.67 | 3 | + Ranks (TWENTYEI GT PRE) |
| | 0 | Ties (TWENTYEI EQ PRE) |
| | - | |
| | 8 | Total |

Z = -1.4003 2-Tailed P = .1614

STATISTICAL TEST FOR CONTROL GROUP (WITHIN GROUP)

----- Wilcoxon Matched-Pairs Signed-Ranks Test

IMMED
with PRE

| Mean Rank | Cases | |
|---|---|---|
| .00 | 0 | - Ranks (PRE LT IMMED) |
| 4.00 | 7 | + Ranks (PRE GT IMMED) |
| | 0 | Ties (PRE EQ IMMED) |
| | - | |
| | 7 | Total |

Z = -2.3664 2-Tailed P = .0180

----- Wilcoxon Matched-Pairs Signed-Ranks Test

ONEDAY
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 2.50 | 2 | - Ranks (PRE LT ONEDAY) |
| 4.60 | 5 | + Ranks (PRE GT ONEDAY) |
| | 0 | Ties (PRE EQ ONEDAY) |
| | - | |
| | 7 | Total |

Z = -1.5213 2-Tailed P = .1282

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with THREEDAY

| Mean Rank | Cases | |
|---|---|---|
| 4.00 | 5 | - Ranks (THREEDAY LT PRE) |
| 1.00 | 1 | + Ranks (THREEDAY GT PRE) |
| | 1 | Ties (THREEDAY EQ PRE) |
| | - | |
| | 7 | Total |

Z = -1.9917 2-Tailed P = .0464

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with SEVEN

| Mean Rank | Cases | |
|---|---|---|
| 4.00 | 7 | - Ranks (SEVEN LT PRE) |
| .00 | 0 | + Ranks (SEVEN GT PRE) |
| | 0 | Ties (SEVEN EQ PRE) |
| | - | |
| | 7 | Total |

Z = -2.3664 2-Tailed P = .0180

----- Wilcoxon Matched-Pairs Signed-Ranks Test

FOURTEEN
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 1.50 | 2 | - Ranks (PRE LT FOURTEEN) |
| 5.00 | 5 | + Ranks (PRE GT FOURTEEN) |
| | 0 | Ties (PRE EQ FOURTEEN) |
| | 7 | Total |

Z = -1.8593 2-Tailed P = .0630

STATISTICAL TEST FOR EMG (MAX ADU)

----- Wilcoxon Matched-Pairs Signed-Ranks Test

ONEDAY
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 3.67 | 3 | - Ranks (PRE LT ONEDAY) |
| 5.00 | 5 | + Ranks (PRE GT ONEDAY) |
| | 0 | Ties (PRE EQ ONEDAY) |
| | - | |
| | 8 | Total |

Z = -.9802 2-Tailed P = .3270

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with THREEDAY

| Mean Rank | Cases | |
|---|---|---|
| 5.00 | 3 | - Ranks (THREEDAY LT PRE) |
| 4.20 | 5 | + Ranks (THREEDAY GT PRE) |
| | 0 | Ties (THREEDAY EQ PRE) |
| | - | |
| | 8 | Total |

Z = -.4201 2-Tailed P = .6744

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with SEVEN

| Mean Rank | Cases | |
|---|---|---|
| 4.83 | 6 | - Ranks (SEVEN LT PRE) |
| 3.50 | 2 | + Ranks (SEVEN GT PRE) |
| | 0 | Ties (SEVEN EQ PRE) |
| | 8 | Total |

Z = -1.5403 2-Tailed P = .1235

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

FOURTEEN
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 5.75 | 4 | - Ranks (PRE LT FOURTEEN) |
| 3.25 | 4 | + Ranks (PRE GT FOURTEEN) |
| | 0 | Ties (PRE EQ FOURTEEN) |
| | 8 | Total |

Z = -.7001 2-Tailed P = .4838

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYON

| Mean Rank | Cases | |
|---|---|---|
| 4.67 | 3 | - Ranks (TWENTYON LT PRE) |
| 4.40 | 5 | + Ranks (TWENTYON GT PRE) |
| | 0 | Ties (TWENTYON EQ PRE) |
| | 8 | Total |

Z = -.5601 2-Tailed P = .5754

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYEI

| Mean Rank | Cases | |
|---|---|---|
| 4.50 | 4 | - Ranks (TWENTYEI LT PRE) |
| 3.33 | 3 | + Ranks (TWENTYEI GT PRE) |
| | 0 | Ties (TWENTYEI EQ PRE) |
| | - | |
| | 7 | Total |

Z = -.6761 2-Tailed P = .4990

STATISTICAL TEST FOR THRESHOLD MAX MEP

----- Wilcoxon Matched-Pairs Signed-Ranks Test

ONEDAY
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 4.20 | 5 | - Ranks (PRE LT ONEDAY) |
| 5.00 | 3 | + Ranks (PRE GT ONEDAY) |
| | 0 | Ties (PRE EQ ONEDAY) |
| | 8 | Total |

Z = -.4201 2-Tailed P = .6744

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with THREEDAY

| Mean Rank | Cases | |
|---|---|---|
| 3.67 | 3 | - Ranks (THREEDAY LT PRE) |
| 5.00 | 5 | + Ranks (THREEDAY GT PRE) |
| | 0 | Ties (THREEDAY EQ PRE) |
| | 8 | Total |

Z = -.9802 2-Tailed P = .3270

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with SEVEN

| Mean Rank | Cases | |
|---|---|---|
| 3.60 | 5 | - Ranks (SEVEN LT PRE) |
| 6.00 | 3 | + Ranks (SEVEN GT PRE) |
| | 0 | Ties (SEVEN EQ PRE) |
| | 8 | Total |

Z = .0000 2-Tailed P = 1.0000

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

FOURTEEN
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 4.50 | 6 | - Ranks (PRE LT FOURTEEN) |
| 4.50 | 2 | + Ranks (PRE GT FOURTEEN) |
| | 0 | Ties (PRE EQ FOURTEEN) |
| | 8 | Total |

Z = -1.2603 2-Tailed P = .2076

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYON

| Mean Rank | Cases | |
|---|---|---|
| 3.50 | 6 | - Ranks (TWENTYON LT PRE) |
| 7.50 | 2 | + Ranks (TWENTYON GT PRE) |
| | 0 | Ties (TWENTYON EQ PRE) |
| | 8 | Total |

Z = -.4201 2-Tailed P = .6744

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYEI

| Mean Rank | Cases | |
|---|---|---|
| 4.00 | 2 | - Ranks (TWENTYEI LT PRE) |
| 4.00 | 5 | + Ranks (TWENTYEI GT PRE) |
| | 0 | Ties (TWENTYEI EQ PRE) |
| | 7 | Total |

Z = -1.0142 2-Tailed P = .3105

STATISTICAL TEST FOR THRESHOLDS

----- Wilcoxon Matched-Pairs Signed-Ranks Test

ONEDAY
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 1.50 | 1 | - Ranks (PRE LT ONEDAY) |
| 1.50 | 1 | + Ranks (PRE GT ONEDAY) |
| | 6 | Ties (PRE EQ ONEDAY) |
| | - | |
| | 8 | Total |

Z = .0000 2-Tailed P = 1.0000

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with THREEDAY

| Mean Rank | Cases | |
|---|---|---|
| 1.50 | 1 | - Ranks (THREEDAY LT PRE) |
| 1.50 | 1 | + Ranks (THREEDAY GT PRE) |
| | 6 | Ties (THREEDAY EQ PRE) |
| | - | |
| | 8 | Total |

Z = .0000 2-Tailed P = 1.0000

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with SEVEN

| Mean Rank | Cases | |
|---|---|---|
| 5.50 | 1 | - Ranks (SEVEN LT PRE) |
| 3.10 | 5 | + Ranks (SEVEN GT PRE) |
| | 2 | Ties (SEVEN EQ PRE) |
| | - | |
| | 8 | Total |

Z = -1.0483 2-Tailed P = .2945

----- Wilcoxon Matched-Pairs Signed-Ranks Test

FOURTEEN
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 2.50 | 3 | - Ranks (PRE LT FOURTEEN) |
| 2.50 | 1 | + Ranks (PRE GT FOURTEEN) |
| | 4 | Ties (PRE EQ FOURTEEN) |
| | 8 | Total |

Z = -.9129 2-Tailed P = .3613

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYON

| Mean Rank | Cases | |
|---|---|---|
| .00 | 0 | - Ranks (TWENTYON LT PRE) |
| 1.50 | 2 | + Ranks (TWENTYON GT PRE) |
| | 6 | Ties (TWENTYON EQ PRE) |
| | 8 | Total |

Z = -1.3416 2-Tailed P = .1797

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYEI

| Mean Rank | Cases | |
|---|---|---|
| .00 | 0 | - Ranks (TWENTYEI LT PRE) |
| 1.00 | 1 | + Ranks (TWENTYEI GT PRE) |
| | 7 | Ties (TWENTYEI EQ PRE) |
| | 8 | Total |

Z = -1.0000 2-Tailed P = .3173

## STATISTICAL TESTS FOR COG LOCATION

- - - Wilcoxon Matched-Pairs Signed-Ranks Test

ONEDAY
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 4.00 | 1 | - Ranks (PRE LT ONEDAY) |
| 2.75 | 4 | + Ranks (PRE GT ONEDAY) |
| | 0 | Ties (PRE EQ ONEDAY) |
| | 5 | Total |

Z = -.9439 2-Tailed P = .3452

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with THREEDAY

| Mean Rank | Cases | |
|---|---|---|
| 2.50 | 4 | - Ranks (THREEDAY LT PRE) |
| 5.00 | 1 | + Ranks (THREEDAY GT PRE) |
| | 1 | Ties (THREEDAY EQ PRE) |
| | 6 | Total |

Z = -.6742 2-Tailed P = .5002

- - - - - Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with SEVEN

| Mean Rank | Cases | |
|---|---|---|
| 4.60 | 5 | - Ranks (SEVEN LT PRE) |
| 4.33 | 3 | + Ranks (SEVEN GT PRE) |
| | 0 | Ties (SEVEN EQ PRE) |
| | 8 | Total |

Z = -.7001 2-Tailed P = .4838

----- Wilcoxon Matched-Pairs Signed-Ranks Test

FOURTEEN
with PRE

| Mean Rank | Cases | |
|---|---|---|
| 4.67 | 3 | - Ranks (PRE LT FOURTEEN) |
| 3.50 | 4 | + Ranks (PRE GT FOURTEEN) |
| | 1 | Ties (PRE EQ FOURTEEN) |
| | - | |
| | 8 | Total |

Z = .0000 2-Tailed P = 1.0000

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYON

| Mean Rank | Cases | |
|---|---|---|
| 4.00 | 1 | - Ranks (TWENTYON LT PRE) |
| 3.40 | 5 | + Ranks (TWENTYON GT PRE) |
| | 2 | Ties (TWENTYON EQ PRE) |
| | - | |
| | 8 | Total |

Z = -1.3628 2-Tailed P = .1730

----- Wilcoxon Matched-Pairs Signed-Ranks Test

PRE
with TWENTYEI

| Mean Rank | Cases | |
|---|---|---|
| 1.50 | 2 | - Ranks (TWENTYEI LT PRE) |
| 4.00 | 3 | + Ranks (TWENTYEI GT PRE) |
| | 1 | Ties (TWENTYEI EQ PRE) |
| | - | |
| | 6 | Total |

Z = -1.2136 2-Tailed P = .2249

# APPENDIX E

Typical Subject Results

COLLECTION: thresh@6,0@45%

CLASS: All epochs CHANNEL: L BICEP

NAME:

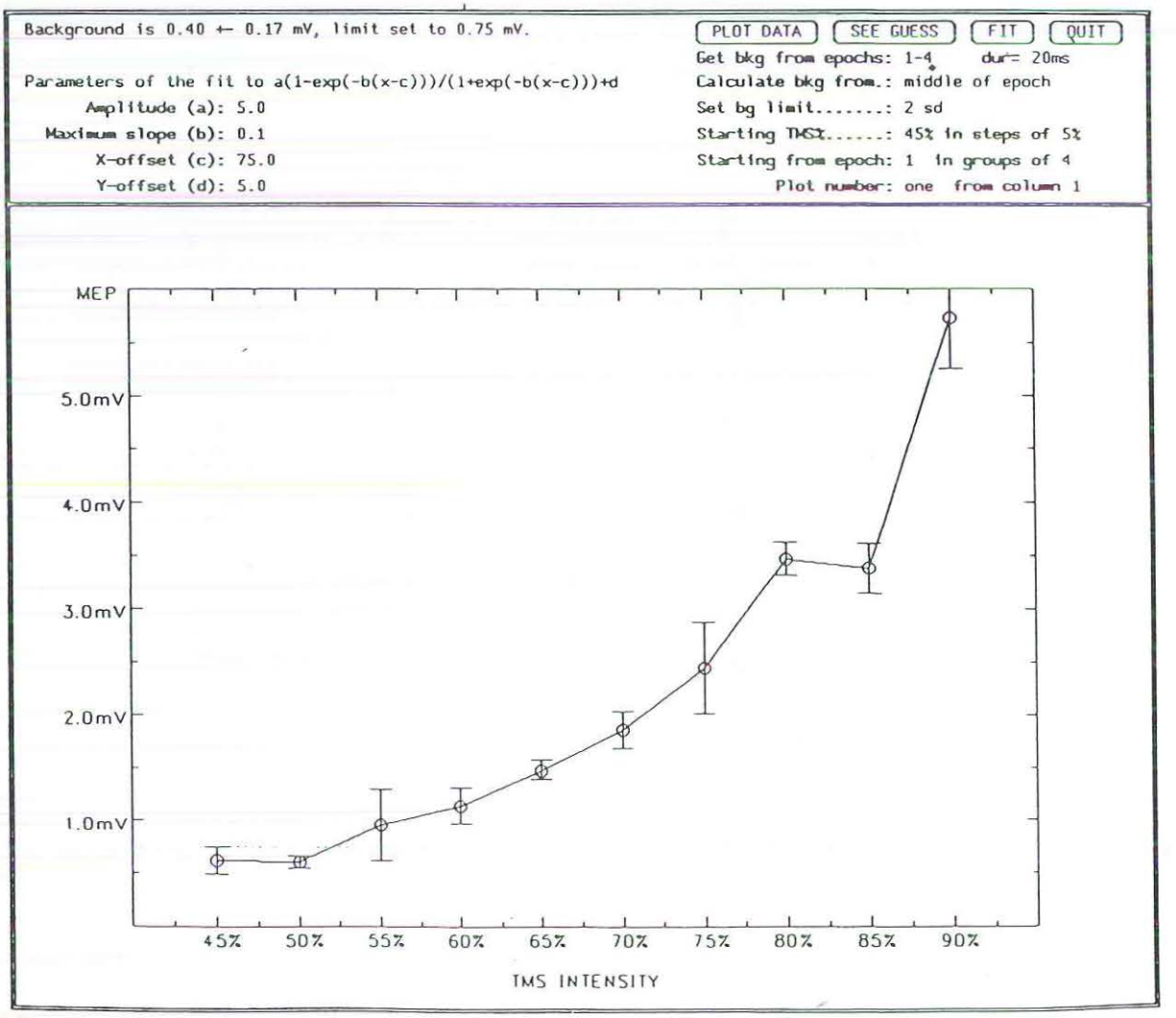
URN: unspecified AGE: unspecified SEX: unspecified

DATE: Thu Jul 06 10:44:41 1995 - printed Thu Jul 6 11:15:53 1995.

FILE: /home/gary/pc/data/mapping/damage/alan/

SERIES: replicate?

COMMENTS:



COLLECTION: L BICEP

NAME:

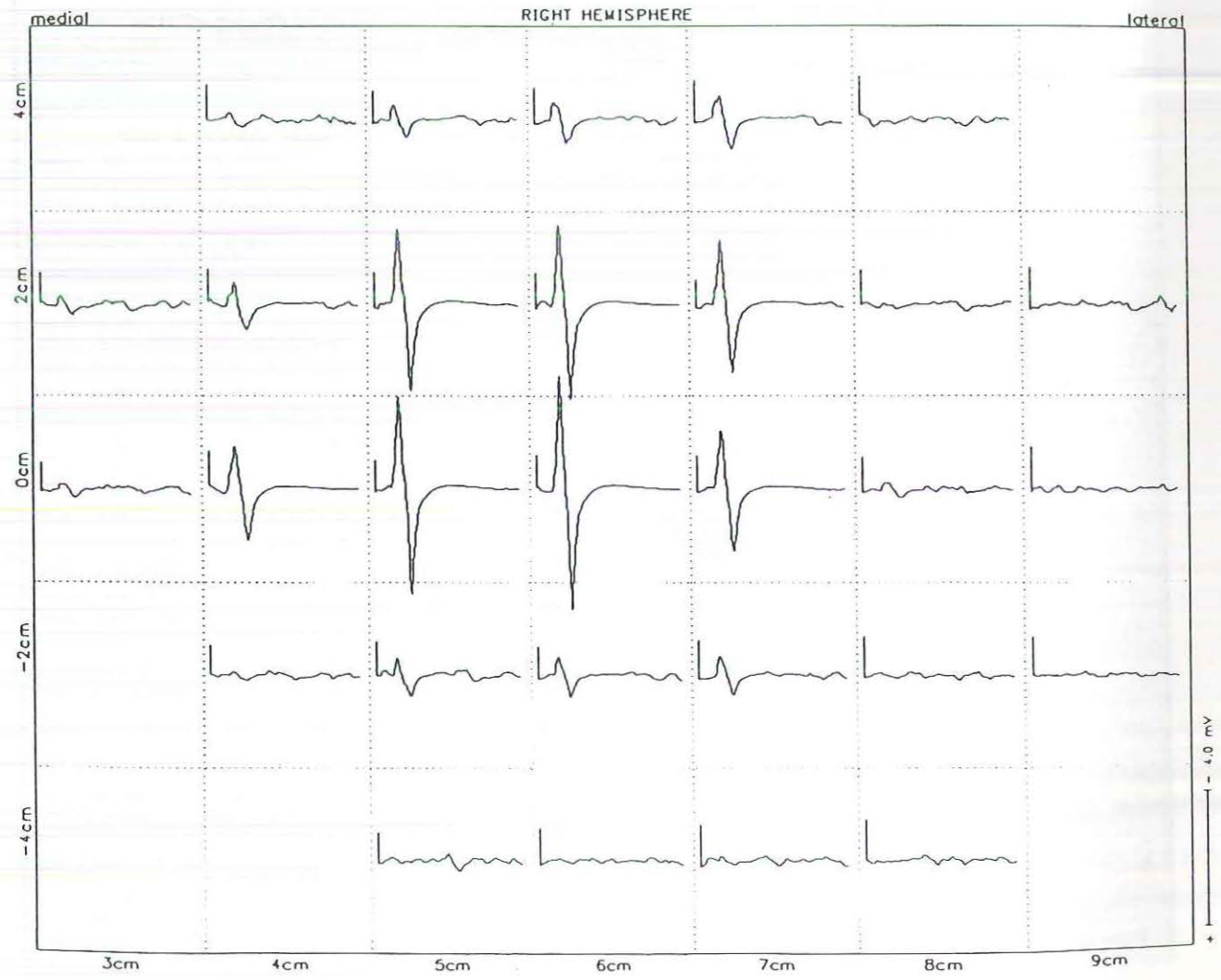
URN: unspecified AGE: unspecified SEX: unspecified

DATE: Thu Jul 06 10:44:41 1995 - printed Fri Jul 7 13:53:27 1995.

FILE: /home/gary/pc/data/mapping/damage/alan/

SERIES: replicate?

COMMENTS:



COLLECTION: L BICEP

NAME:

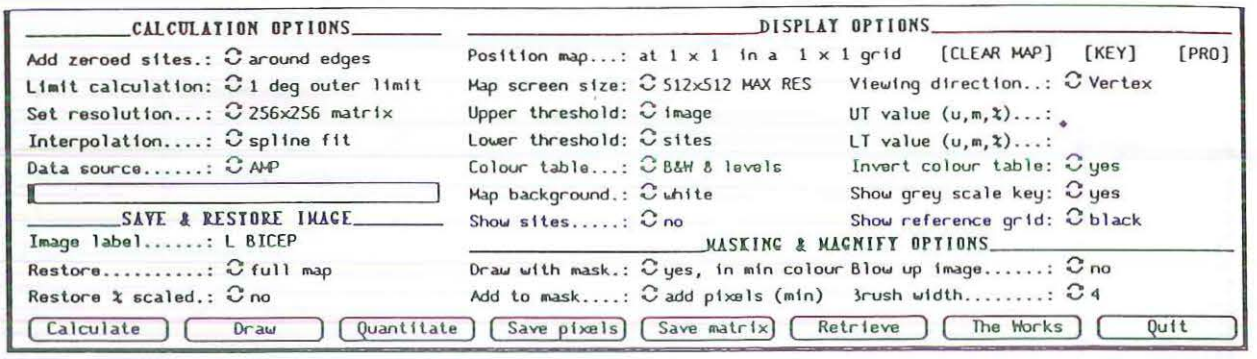
URN: unspecified AGE: unspecified SEX: unspecified

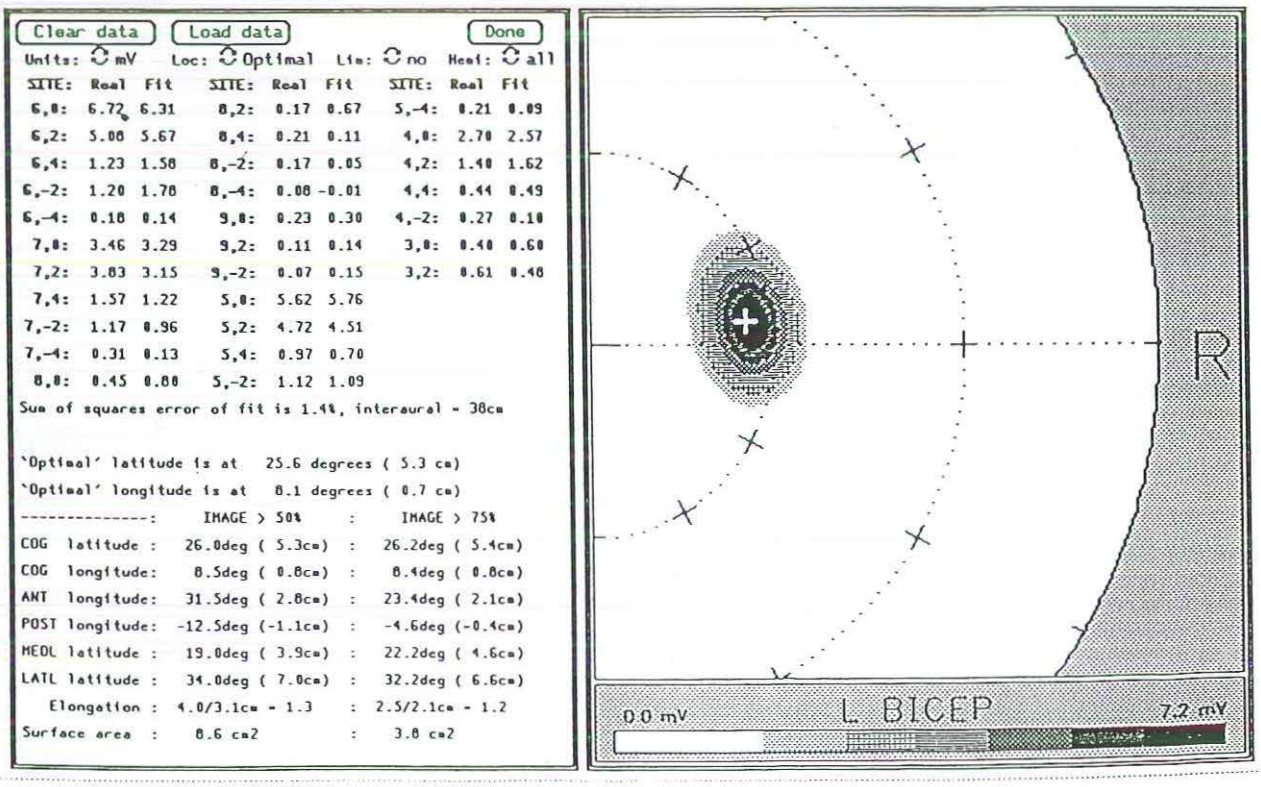
DATE: Thu Jul 06 10:44:41 1995 - printed Fri Jul 7 15:07:04 1995.

FILE: /home/gary/pc/data/mapping/damage/alan/

SERIES: replicate?

COMMENTS:



COLLECTION: L BICEP

NAME:

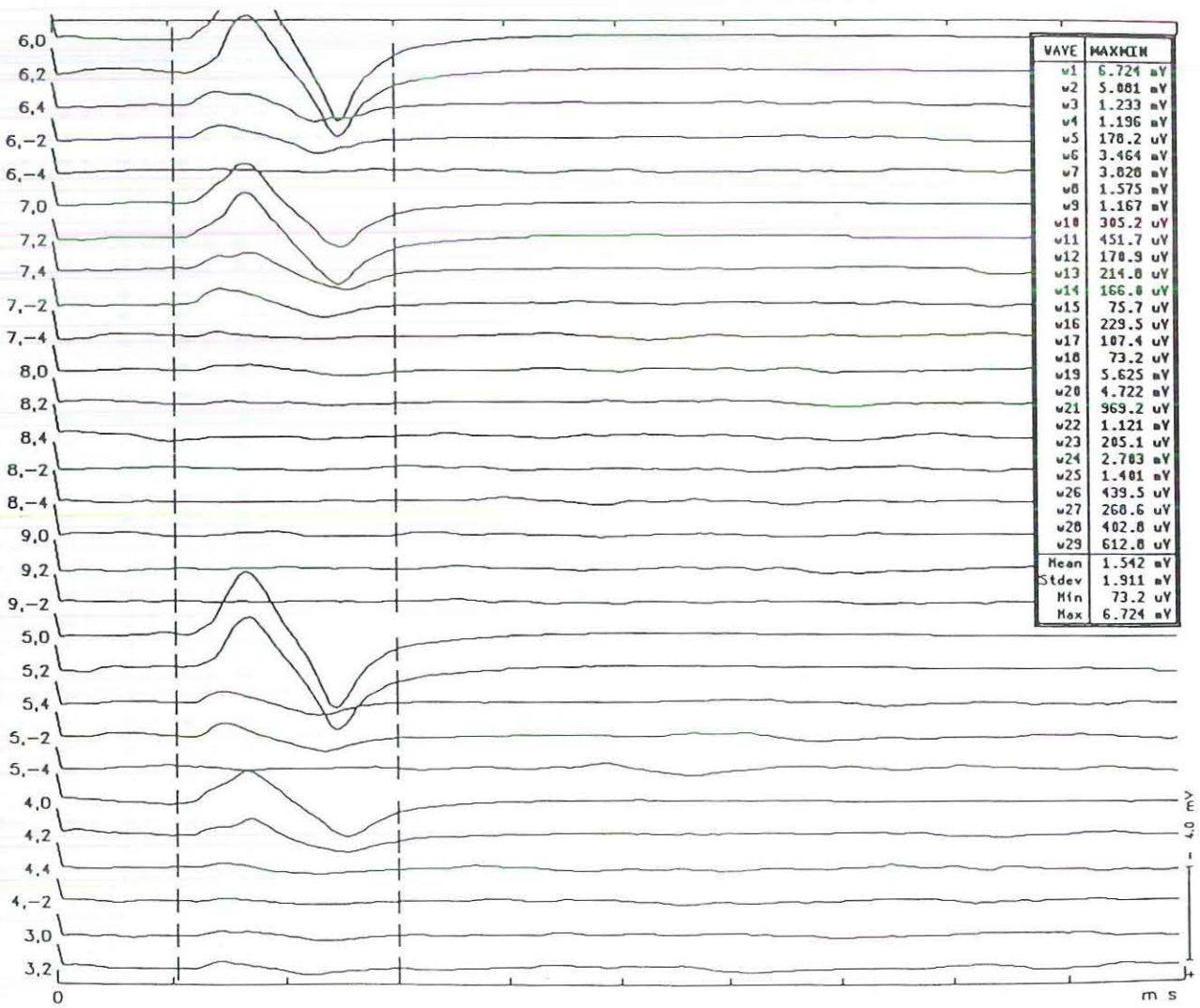
URN: unspecified AGE: unspecified SEX: unspecified

DATE: Thu Jul 06 10:44:41 1995 - printed Fri Jul 7 13:55:38 1995.

FILE: /home/gary/pc/data/mapping/damage/alan/lml3.bic

SERIES: replicate?

COMMENTS:




| WAVE | MAXMIN |
|---|---|
| w1 | 6.724 mV |
| w2 | 5.081 mV |
| w3 | 1.233 mV |
| w4 | 1.196 mV |
| w5 | 178.2 uV |
| w6 | 3.464 mV |
| w7 | 3.828 mV |
| w8 | 1.575 mV |
| w9 | 1.167 mV |
| w10 | 305.2 uV |
| w11 | 451.7 uV |
| w12 | 178.9 uV |
| w13 | 214.8 uV |
| w14 | 166.8 uV |
| w15 | 75.7 uV |
| w16 | 229.5 uV |
| w17 | 107.4 uV |
| w18 | 73.2 uV |
| w19 | 5.625 mV |
| w20 | 4.722 mV |
| w21 | 969.2 uV |
| w22 | 1.121 mV |
| w23 | 205.1 uV |
| w24 | 2.703 mV |
| w25 | 1.401 mV |
| w26 | 439.5 uV |
| w27 | 260.6 uV |
| w28 | 402.8 uV |
| w29 | 612.8 uV |
| Mean | 1.542 mV |
| Stdev | 1.911 mV |
| Min | 73.2 uV |
| Max | 6.724 mV |

COLLECTION: 6,0

NAME:

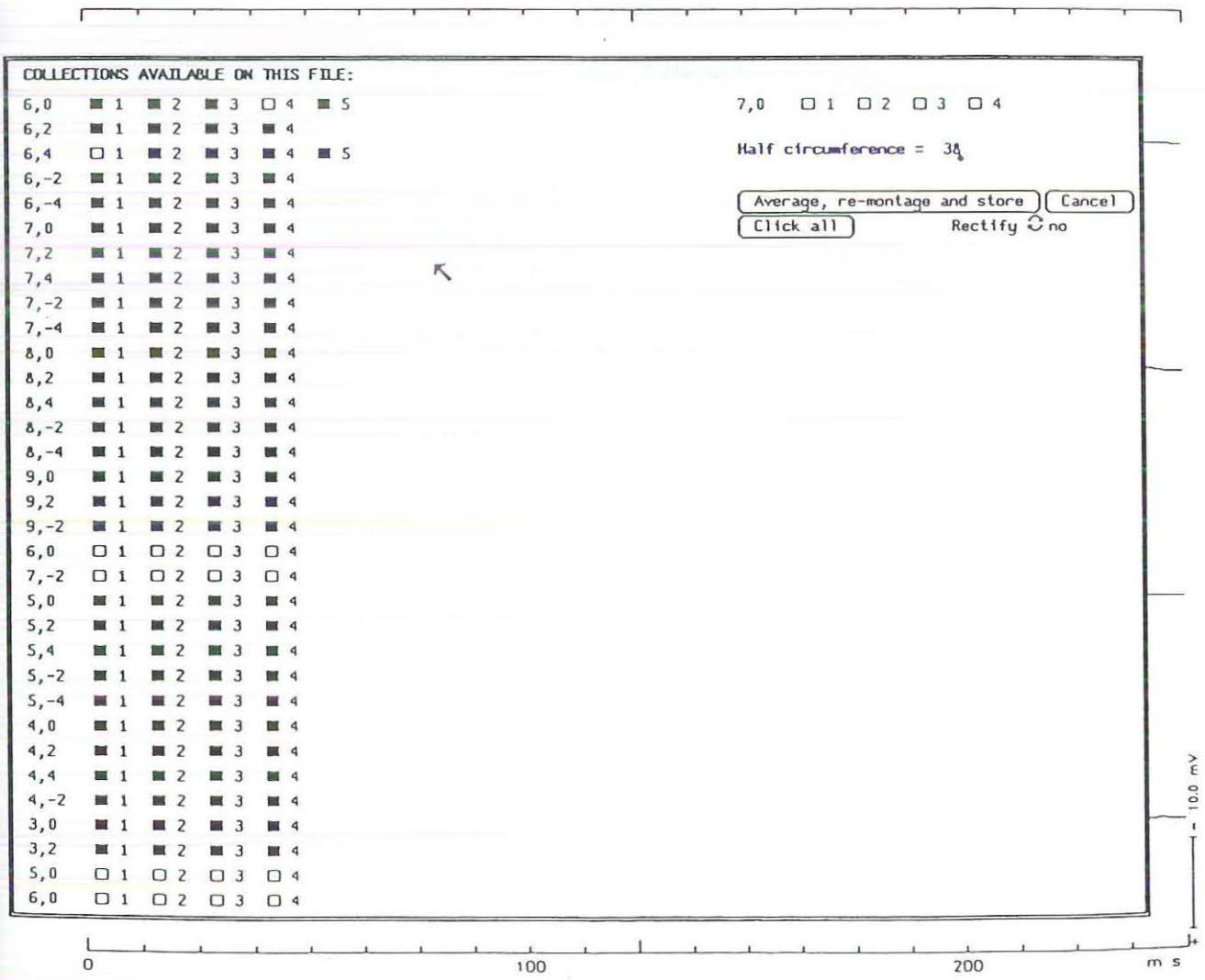
URN: unspecified AGE: unspecified SEX: unspecified

DATE: Thu Jul 06 10:44:41 1995 - printed Fri Jul 7 13:50:31 1995.

FILE: /home/gary/pc/data/mapping/damage/alan/lml3.bic

SERIES: replicate?

COMMENTS:



# APPENDIX F

Raw Data Collected

STRENGTH

| | | | | NORMALISED STRENGTH | | | | |
|---|---|---|---|---|---|---|---|---|
| Subject | Max % | Immed | Post 1d | Post 3d | Post 7d | Post 14d | Post 21d | Post 28d |
| Subject | 0 | 0.5 | 1 | 3 | 7 | 14 | 21 | 28 |
| 2 | 100.00 | 76.43 | | 93.57 | 98.21 | 105.00 | 104.64 | 105.36 |
| 3 | 100.00 | 69.57 | | | 84.47 | 86.96 | 93.79 | 92.55 |
| 4 | 100.00 | 87.66 | 75.74 | 87.23 | 90.64 | 102.55 | 110.21 | 98.30 |
| 5 | 100.00 | 69.70 | | | 80.00 | 84.85 | 92.12 | 89.70 |
| 6 | 100.00 | 75.00 | 71.15 | 83.97 | 82.69 | 98.08 | 101.28 | 92.95 |
| 7 | 100.00 | 65.73 | 56.99 | 60.84 | 83.92 | 85.31 | 87.41 | 87.76 |
| 9 | 100.00 | 71.37 | 67.06 | 71.37 | 98.82 | | 83.92 | 93.73 |
| 10 | 100.00 | 57.09 | 47.16 | 46.10 | 47.87 | 62.77 | 86.88 | |
| | | | | | | | | |
| mean | 100.00 | 71.57 | 63.62 | 73.85 | 83.33 | 89.36 | 95.03 | 94.33 |
| sd | 0.00 | 8.83 | 11.51 | 17.98 | 15.97 | 14.40 | 9.41 | 5.88 |
| sem | 0 | 3.531002 | 4.605814 | 7.191309 | 6.388188 | 5.760539 | 3.764092 | 2.351715 |

CK

| | | | | | CREATINE KINASE | | |
|---|---|---|---|---|---|---|---|
| Subject | Pre 1 | Post 1d | Post 3d | Post 7d | Post 14d | Post 21d | Post 28d |
| | 0 | 1 | 3 | 7 | 14 | 21 | 28 |
| 2 | 24.00 | | 202.00 | 248.00 | 126.00 | 191.00 | 109.00 |
| 3 | 120.00 | | | 1270.00 | 170.00 | | 102.00 |
| 4 | 50.90 | 80.00 | 145.00 | 99.00 | 81.00 | 91.00 | 150.00 |
| 5 | 122.00 | | | 1050.00 | 123.00 | | 116.00 |
| 6 | 21.90 | 32.20 | 117.00 | 281.00 | 39.80 | 54.60 | 42.00 |
| 7 | 74.00 | 434.00 | 1400.00 | 1380.00 | 433.00 | 282.00 | 93.00 |
| 9 | 209.00 | 441.00 | 258.00 | 253.00 | 117.00 | 109.00 | 95.00 |
| 10 | 120.00 | | 490.00 | 1380.00 | 228.00 | 124.00 | 105.00 |
| | | | | | | | |
| | | | | | | | |
| mean | 92.73 | 246.80 | 435.33 | 745.13 | 164.73 | 141.93 | 101.50 |
| SD | 62.91038 | 221.0829 | 490.9361 | 572.8107 | 121.9704 | 82.04113 | 29.96665 |
| SEM | 25.16415 | 88.43314 | 196.3744 | 229.1243 | 48.78816 | 32.81645 | 11.98666 |

POT TRACKING

| | | TRACKING TASK - MEAN OF 5 TRIALS | | | | | | | | TRACKING TASK - MEAN OF 5 TRIALS | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Subject | Pre 1 | Immed | Post 1d | Post 3d | Post 7d | Post 14d | y | Subject | Pre 1 | Immed | Post 1d | Post 3d | Post 5d | Post 7d | Post 14d | Post 21d | Post 28d |
| Subject | 0 | 0.5 | 1 | 3 | 7 | 14 | | Subject | 0 | 0.5 | 1 | 3 | 5 | 7 | 14 | 21 | 28 |
| a | 4.62 | 4.58 | 4.32 | 4.64 | 4.50 | 4.96 | | 2 | 6.56 | 7.46 | | 6.02 | | 6.78 | 5.86 | 6.52 | 6.86 |
| b | 6.56 | 6.46 | 6.64 | 5.84 | 5.36 | 4.94 | | 3 | 4.78 | 4.83 | | | | 4.58 | 4.16 | 4.76 | 4.98 |
| c | 7.52 | 6.52 | 6.52 | 5.76 | 5.66 | 5.68 | | 4 | 6.10 | 7.84 | 8.72 | 6.96 | 5.38 | 4.94 | 5.76 | 4.72 | 4.18 |
| d | 5.98 | | 5.58 | 4.34 | 4.50 | 4.48 | | 5 | 4.08 | 4.84 | | | | 4.12 | 3.62 | 3.96 | 3.56 |
| e | 3.36 | 3.16 | 3.10 | 3.10 | 2.92 | 2.92 | | 6 | 5.72 | 7.60 | 8.44 | 5.56 | 4.98 | 5.28 | 4.64 | 5.10 | 4.43 |
| f | 3.68 | 3.20 | 4.00 | 3.86 | 3.48 | 3.70 | | 7 | 3.60 | 3.62 | 3.85 | 3.76 | | 3.70 | 2.94 | 3.12 | 2.98 |
| g | 7.30 | 6.46 | 5.80 | 5.34 | 5.63 | 5.70 | | 9 | 3.24 | 3.42 | 3.88 | 4.48 | 3.46 | 3.16 | 3.12 | 3.08 | 3.66 |
| | | | | | | | | 10 | 3.88 | 4.32 | 4.56 | 3.36 | | 3.86 | 3.98 | 3.90 | |
| | | | | | | | | | | | | | | | | | |
| X | 5.57 | 5.06 | 5.14 | 4.70 | 4.58 | 4.63 | | X | 4.25 | 4.94 | 5.08 | 4.73 | 4.71 | 4.82 | 5.34 | 6.24 | 7.33 |
| SD | 1.698145 | 1.634107 | 1.348897 | 1.019113 | 1.068931 | 1.021745 | | SD | 1.954643 | 2.398145 | 2.980227 | 1.483182 | 0.850157 | 1.336928 | 3.406265 | 5.63616 | 8.434022 |

| | | | | MAP C.O.G LATITUDE - LEFT BICEP | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Subject | Pre 1 | Pre 2 | Pre 3 | Immed | Post 1d | Post 3d | Post 5d | Post 7d | Post 14d | Post 21d | Post 28d |
| 2 | 5.5 | 5.3 | 5.7 | | | 5.3 | | 5.2 | 5.2 | 5.6 | |
| 3 | 3.5 | 4.4 | | | | | | 4.6 | 4.3 | 4.4 | |
| 4 | 4.9 | 4.9 | | 4.8 | 4.8 | 5.2 | 4.8 | 4.6 | 4.4 | 4.9 | 4.6 |
| 5 | 5.5 | | | | | | | 5.4 | 5.5 | 5.6 | |
| 6 | | 5.0 | 4.9 | 5.1 | 4.8 | 4.8 | 4.9 | 5.1 | 5.2 | 4.6 | 4.9 |
| 7 | 4.4 | 4.0 | | | 3.7 | 4.0 | | 3.8 | 4.4 | 4.6 | 4.7 |
| 9 | 4.9 | 4.8 | | | 4.7 | 4.8 | 4.5 | 4.5 | 4.6 | 5.0 | 4.8 |
| 10 | 4.3 | | | | 4.7 | 4.6 | | 5.1 | 5.2 | | |
| 11 | | | | | | | | | | | |
| | | | | | | | | | | | |
| X | 4.71 | 4.73 | 5.30 | 4.95 | 4.54 | 4.78 | 4.73 | 4.79 | 4.85 | 4.96 | 4.75 |
| SD | 0.712808 | 0.463321 | 0.565685 | 0.212132 | 0.472229 | 0.466548 | 0.208167 | 0.516686 | 0.472077 | 0.482553 | 0.129099 |
| SEM | 0.274157 | 0.178201 | 0.217571 | 0.081589 | 0.181626 | 0.179441 | 0.080064 | 0.198725 | 0.181568 | 0.185597 | 0.049654 |

| | | | | | MAP C.O.G LATITUDE - SHIFTS | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Immed | Post 1d | Post 3d | Post 7d | Post 14d | Post 21d | Post 28d |
| | Pre 1 | Pre 2 | Subject | 0 | 0.5 | 1 | 3 | 7 | 14 | 21 | 28 |
| | 5.5 | 5.3 | 2 | 0.0 | | | -0.2 | -0.3 | -0.3 | 0.1 | 0.0 |
| | 3.5 | 4.4 | 3 | 0.0 | | | | 0.2 | -0.1 | 0.0 | 0.6 |
| | 4.9 | 4.9 | 4 | 0.0 | -0.1 | -0.1 | -0.1 | -0.3 | -0.5 | 0.0 | -0.3 |
| | 5.5 | | 5 | 0.0 | | | | -0.1 | 0.0 | 0.1 | 0.4 |
| | 5.0 | 4.9 | 6 | 0.0 | 0.1 | -0.2 | -0.2 | | 0.2 | -0.4 | -0.1 |
| | 4.4 | 4.0 | 7 | 0.0 | | | 0.0 | -0.2 | 0.4 | 0.6 | 0.7 |
| | 4.9 | 4.8 | 9 | 0.0 | | -0.2 | -0.4 | -0.4 | -0.3 | 0.1 | -0.1 |
| | 4.3 | | 10 | 0.0 | | 0.4 | 0.3 | 0.8 | | | |
| | | | 11 | | | | | | | | |
| | | | | | | | | | | | |
| X | 4.75 | 4.72 | x | 0.00 | 0.00 | -0.03 | -0.10 | -0.04 | -0.09 | 0.07 | 0.17 |
| SD | 0.667618 | 0.453505 | sd | 0 | 0.141421 | 0.287228 | 0.236643 | 0.41975 | 0.313202 | 0.29277 | 0.39036 |
| SEM | 0.256776 | 0.174425 | sem | 0 | 0.054393 | 0.110472 | 0.091017 | 0.161442 | 0.120462 | 0.112604 | 0.150138 |

| | | | MEP MAPS - LEFT BICEP | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre 2 | Post 1d | Post 3d | Post 5d | Post 7d | Post 14d | Post 21d | Post 28d |
| Subject | 0 | 1 | 3 | 5 | 7 | 14 | 21 | 28 |
| 2 | 7.20 | | 4.50 | | 3.80 | 2.60 | 4.40 | |
| 3 | 6.30 | | | | 4.30 | 7.60 | 5.80 | |
| 4 | 12.40 | 3.90 | 5.20 | 4.70 | 9.20 | 11.60 | 4.10 | 6.90 |
| 5 | 1.70 | | | | 2.00 | 3.50 | 3.30 | |
| 6 | 2.40 | 1.50 | 1.70 | 1.90 | 1.90 | 2.70 | 2.10 | 2.30 |
| 7 | 14.00 | 22.00 | 17.70 | | 14.70 | 18.70 | 9.30 | 16.80 |
| 9 | 10.80 | 5.50 | 8.20 | 10.90 | 7.40 | 7.60 | 6.80 | |
| 10 | 0.85 | 2.20 | 3.30 | | 1.80 | 1.60 | | |
| | | | | | | | | |
| | | | | | | | | |
| x | 6.96 | 7.02 | 6.77 | 5.83 | 5.64 | 6.99 | 5.11 | 8.67 |
| sd | 5.073562 | 8.516866 | 5.777081 | 4.605793 | 4.551589 | 5.831304 | 2.406539 | 7.409678 |
| sem | | | | | | | | |
| | | | | | | | | |
| | | | | | | | | |
| | | | | | | | | |
| | | | | | | | | |
| | | | MEP MAPS - LEFT BICEP | | | | | |
| | | | | | | | | |
| | Pre 2 | Post 1d | Post 3d | Post 5d | Post 7d | Post 14d | Post 21d | Post 28d |
| Subject | 0 | 1 | 3 | 5 | 7 | 14 | 21 | 28 |
| 2 | 100.00 | | 62.50 | | 52.78 | 36.11 | 61.11 | |
| 3 | 100.00 | | | | 68.25 | 120.63 | 92.06 | |
| 4 | 100.00 | 31.45 | 41.94 | 37.90 | 74.19 | 93.55 | 33.06 | 55.65 |
| 5 | 100.00 | | | | 117.65 | 205.88 | 194.12 | |
| 6 | 100.00 | 62.50 | 70.83 | 79.17 | 79.17 | 112.50 | 87.50 | 95.83 |
| 7 | 100.00 | 157.14 | 126.43 | | 105.00 | 133.57 | 66.43 | 120.00 |
| 9 | 100.00 | 50.93 | 75.93 | 100.93 | 68.52 | 70.37 | 62.96 | |
| 10 | 100.00 | 258.82 | 388.24 | | 211.76 | 188.24 | | |
| | | | | | | | | |
| | | | | | | | | |
| mean | 100.00 | 112.17 | 127.64 | 72.67 | 97.17 | 120.11 | 85.32 | 90.49 |
| s.d | 0 | 95.21452 | 130.6914 | 32.01041 | 50.83912 | 56.66028 | 51.73745 | 32.50811 |
| sem | 0 | 43.27933 | 54.45475 | 18.50313 | 18.15683 | 20.23582 | 19.89902 | 12.50312 |

THRESH

| | | | MAP THRESHOLD- LEFT BICEP | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | | |
| Subject | | Post 1d | Post 3d | Post 7d | Post 14d | Post 21d | Post 28d |
| Subject | 0 | 1 | 3 | 7 | 14 | 21 | 28 |
| 2 | 55.00 | 55.00 | 50.00 | 60.00 | 60.00 | 55.00 | 55.00 |
| 3 | 40.00 | 40.00 | 40.00 | 50.00 | 40.00 | 40.00 | 40.00 |
| 4 | 50.00 | 50.00 | 50.00 | 50.00 | 55.00 | 55.00 | 55.00 |
| 5 | 60.00 | 55.00 | 60.00 | 50.00 | 55.00 | 60.00 | 60.00 |
| 6 | 70.00 | 75.00 | 75.00 | 75.00 | 70.00 | 70.00 | 70.00 |
| 7 | 45.00 | 45.00 | 45.00 | 50.00 | 50.00 | 55.00 | 45.00 |
| 9 | 45.00 | 45.00 | 45.00 | 45.00 | 45.00 | 45.00 | 45.00 |
| 10 | 65.00 | 65.00 | 65.00 | 70.00 | 65.00 | 65.00 | |
| 11 | | | | | | | |
| | | | | | | | |
| X | 53.75 | 53.75 | 53.75 | 56.25 | 55.00 | 55.63 | 52.86 |
| SD | 10.6066 | 11.57275 | 11.87735 | 10.93814 | 10 | 9.797048 | 10.35098 |
| SEM | 3.761206 | 4.103812 | 4.211826 | 3.878772 | 3.546099 | 3.47413 | 3.670561 |

MAP AREA

| | | | | | MAP AREA - LEFT BICEP | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Subject | Pre 1 | Pre 2 | Immed | Post 1d | Post 3d | Post 5d | Post 7d | Post 14d | Post 21d | Post 28d |
| 1 | | | | | | | | | | |
| 2 | 8.70 | 8.60 | | | 8.80 | | 8.30 | 9.50 | 9.30 | |
| 3 | 7.00 | 13.00 | | | | | 14.30 | 13.50 | 11.10 | |
| 4 | 14.80 | 9.90 | 11.50 | 11.70 | 15.10 | 14.30 | 8.70 | 13.40 | 15.00 | 14.90 |
| 5 | 25.60 | | | | | | 27.60 | 23.20 | 19.40 | |
| 6 | 8.00 | 13.50 | 12.30 | 11.90 | 12.50 | 12.70 | 12.30 | 15.00 | 12.40 | 10.90 |
| 7 | 17.10 | 8.70 | | 11.90 | 10.60 | | 9.90 | 8.50 | 11.40 | 8.00 |
| 9 | 11.10 | 8.40 | | 8.50 | 14.00 | 7.90 | 11.20 | 11.30 | 12.90 | 10.30 |
| 10 | 12.20 | | | 8.30 | 8.40 | | 9.10 | 10.40 | 8.00 | |
| 11 | | | | | | | | | | |
| | | | | | | | | | | |
| X | 13.06 | 10.35 | 11.90 | 10.46 | 11.57 | 11.63 | 12.68 | 13.10 | 12.44 | 11.03 |
| SD | 6.123943 | 2.312358 | 0.565685 | 1.883614 | 2.752938 | 3.330666 | 6.359863 | 4.637117 | 3.53874 | 2.869814 |
| SEM | 2.355363 | 0.889369 | 0.217571 | 0.724467 | 1.058822 | 1.281025 | 2.446101 | 1.783507 | 1.361054 | 1.103775 |

# APPENDIX G

American College of Sports Medicine Abstract

# ABSTRACT OF PROPOSED FREE COMMUNICATION SLIDE OR POSTER

**$25 ABSTRACT SUBMISSION FEE**

1996 American College of Sports Medicine Annual Meeting
DEADLINE: Must be received in the National Center by November 1, 1995
(Abstracts received after this date will be returned.)

MAILING ADDRESS OF FIRST AUTHOR
Please type. Provide full name (no initials).

ACSM I.D. Number: Degree:
Birth Date:
Name: Last First Middle Initial
Address:
City: State: Zip
Country:
Home Telephone: Area Code #
Business Telephone:
Fax:
☐ This address is different than listing in ACSM membership files—please change files to reflect this address

B Each Abstract form submitted MUST BE SIGNED by a member of the American College of Sports Medicine. The undersigned first author is responsible for ensuring that the paper, if accepted, will be presented and that the material will not be published or presented before the Annual Meeting.

First Author's Signature

Is first author a student? ( ) Yes ( ) No

Is first author a member of ACSM? ( ) Yes ( ) No

If not, the signature of an ACSM sponsor is needed.

Print name

ACSM Member's Signature

READ ALL ACCOMPANYING INSTRUCTIONS BEFORE BEGINNING. Remember, your abstract will appear in *Medicine and Science in Sports and Exercise* exactly as you type it.

IMPORTANT: Abstracts will undergo a blind review. Submit the original form completely filled in plus five blind photocopies showing no information about institution, clinic, or address (see accompanying instructions).

SUBMIT FLAT with cardboard backing to:

ABSTRACTS BY MAIL:
American College of Sports Medicine
P.O. Box 1440
Indianapolis, IN 46206-1440 USA

FOR COURIER SERVICE USE STREET ADDRESS:
American College of Sports Medicine
401 West Michigan Street
Indianapolis, IN 46202-3233 USA

For National Center use only
Abstract No.

Blue lines are printer's cut lines; do not type on or outside of these lines. See sample abstract attached.

## EFFECTS OF MAXIMAL ECCENTRIC EXERCISE ON MOTOR CONTROL PROPERTIES OF THE BICEPS BRACHII MUSCLE

P. Sacco, A. J. Pearce*, M. L. Thompson*, G. W. Thickbroom*, F.L. Mastaglia*, University of Western Australia, Perth, WA 6009.

We have studied the time course of changes in voluntary strength/motor skill and corticomotor excitability for up to 28 days following a bout of eccentric exercise. Eight subjects (5 male, 25-40 years of age) performed 35 maximal voluntary eccentric contractions of the elbow flexors through 130° of extension at $90°s^{-1}$. Voluntary electromyographic (EMG) activity and motor evoked potentials (MEP's) elicited by transcranial magnetic stimulation (TMS) were recorded via surface electrodes placed over the belly of the biceps brachii muscle. Maximal isometric strength was measured at 90° elbow flexion. A simple elbow flexion/extension tracking task was used to assess visuomotor co-ordination. Threshold curves were generated of MEP amplitude vs intensity of TMS and maximum MEP amplitude was taken from the plateau of the threshold curve. Strength loss was greatest 1 day after exercise (64±5% (mean ± sem) of pre-exercise value) and recovered to 89±6% by 14 days. Subjects showed an impairment in the skilled tracking task within hours after exercise (13±4% mean increase in tracking error) which was most noticeable 1 day post exercise (25±9%) but returned to control levels by 3 days. There were no changes in the threshold level of MEP responses to magnetic stimulation, but maximal MEP amplitudes increased on average by 38% and 42% of control values at 1 & 3 days post-exercise. No such increases in the root mean square EMG during maximal voluntary efforts were observed. We conclude that changes in motor performance and corticomotor excitability occur following eccentric exercise which may be related to alterations in the pattern of afferent feedback from weakened and/or painful muscles.

Major Category Number: Enter the major category number.

Subcategory Number: When appropriate, enter the subcategory number which represents the INTENDED FOCUS of your abstract.

Presentation Preference will not affect acceptance. (i.e. poster, slide, indifferent) (Check one only:)

☐ Poster session
☐ Slide session
☐ Indifferent

COMPLETE ALL SECTIONS. INCOMPLETE FORMS WILL BE RETURNED.

# APPENDIX H

Australian Neurological Society Abstract

# EFFECTS OF MAXIMAL ECCENTRIC EXERCISE ON NEUROMUSCULAR CONTROL OF THE BICEPS BRACHII MUSCLE

A. J. Pearce*,P. Sacco, , M. L. Thompson, G. W. Thickbroom, F. L. Mastaglia

University of Western Australia, Perth, WA 6009.

* Edith Cowan University, Joondalup, WA 6027

We have studied the time course of changes in voluntary strength, neuromuscular control and corticomotor excitability for up to 28 days following a bout of eccentric exercise. Eight subjects (5 male, 25-40 years of age) performed 35 maximal voluntary eccentric contractions of the elbow flexors through 130° of extension at $90°s^{-1}$. Voluntary electromyographic (EMG) activity and motor evoked potentials (MEP's) elicited by transcranial magnetic stimulation (TMS) were recorded via surface electrodes placed over the belly of the biceps brachii muscle. Maximal isometric strength was measured at 90° elbow flexion. A simple elbow flexion/extension tracking task was used to assess visuomotor co-ordination. Threshold curves were generated of MEP amplitude vs intensity of TMS and maximum MEP amplitude was taken from the plateau of the threshold curve. Strength loss was greatest 1 day after exercise (64±5% (mean ± sem) of pre-exercise value) and recovered to 89±6% by 14 days. Subjects showed an impairment in the skilled tracking task within hours after exercise (13±4% mean increase in tracking error) which was most noticeable 1 day post exercise (25±9%) but returned to control levels by 3 days. There were no changes in the threshold level of MEP responses to magnetic stimulation, but maximal MEP amplitudes increased on average by 38% and 42% of control values at 1 & 3 days post-exercise. No such increases in the root mean square EMG during maximal voluntary efforts were observed. We conclude that changes in neuromuscular control and corticomotor excitability occur following eccentric exercise which may be related to alterations in the pattern of afferent feedback from weakened and/or painful muscles.

# APPENDIX I

Subject Characteristics

| Subject | Gender | Age (yrs) | Height (cm) | Weight (kg) |
|---|---|---|---|---|
| 1 | f | 30 | 173 | 70 |
| 2 | m | 42 | 170 | 63 |
| 3 | f | 37 | 165 | 57 |
| 4 | m | 32 | 176 | 66 |
| 5 | f | 42 | 166 | 60 |
| 6 | f | 30 | 165 | 64 |
| 7 | m | 30 | 170 | 60 |
| 9 | m | 25 | 176 | 70 |
| 10 | m | 25 | 180 | 76 |
| 11 | m | 27 | 182 | 90 |
| | | | | |
| a | f | 22 | 165 | 60 |
| b | f | 30 | 173 | 70 |
| c | m | 42 | 173 | 80 |
| d | m | 41 | 180 | 70 |
| e | f | 43 | 175 | 72 |
| f | m | 45 | 190 | 85 |
| g | m | 25 | 176 | 70 |

# APPENDIX J

Isokinetic Calibration

| weight (kg) | adu | load (adu) | unload (adu) | mean (adu) | adu/N |
|---|---|---|---|---|---|
| 0 | 112 | 0 | 0 | 0 | 0 |
| 1 | 816 | 480 | 480 | 480 | 48.9 |
| 6 | 2480 | 1984 | 1968 | 1976 | 33.6 |
| 11 | 3750 | 3312 | 3344 | 3328 | 30.9 |
| 16 | 5504 | 5120 | 4944 | 5032 | 32.1 |
| 21 | 6800 | 6400 | 6336 | 6368 | 30.9 |
| 26 | 8336 | 8000 | 8000 | 8000 | 31.3 |
| 21 | 6688 | | | | |
| 16 | 5440 | | | | |
| 11 | 3760 | | | | |
| 6 | 2288 | | | | |
| 1 | 688 | | | | |
| 0 | 112 | | | | |

100 physical units = 3100 adu