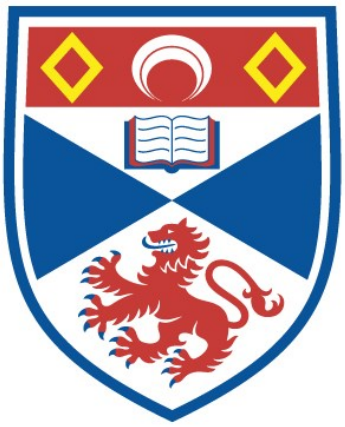
MUSCLE STIFFNESS AND SORENESS FOLLOWING EXERCISE

Fraser Gillies McGlynn

A Thesis Submitted for the Degree of PhD
at the
University of St Andrews



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MUSCLE STIFFNESS AND SORENESS FOLLOWING EXERCISE

BY

FRASER McGLYNN M.A. M.Sc.

A thesis submitted for the degree of

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ABSTRACT

It is in the best interests of Sportsmen and Sportswomen to try to avoid muscle stiffness and soreness. Apart from the discomfort experienced, muscle stiffness and soreness can cause unnecessary interruptions to training, may lead to injury and will reduce performance.

Changes in muscle tone were quantified in terms of the Resonant Frequency (squared) (RF^2) and the Amplitude of Movement (AM) in response to an applied torque. Muscle soreness was measured at twelve sites on the arm.

Study One investigated the effects of a single bout of eccentric exercise on muscle stiffness and muscle soreness. RF^2 increased and AM decreased following exercise and reached a maximum and minimum, respectively, 24-48 hours post exercise ($p<0.01$). Muscle soreness also reached a peak 24-48 hours post-exercise ($p<0.01$). Greatest soreness was in the biceps brachii and in the proximal ends of the brachioradialis and the flexor carpi radialis ($p<0.01$). Voluntary extension was more painful than voluntary flexion following eccentric exercise. Study Two investigated the effect of performing two subsequent exercise bouts (EX1 and EX2), each separated by six days and an adaptation was observed. Each of the variables measured (RF^2, AM, Soreness, Creatine Kinase, Limb Girth) showed a reduced response following EX2 when compared to the results of EX1 ($p<0.01$). The resting angle of elbow flexion appeared to decrease following exercise. Study Three investigated the effect of muscle soreness on motor performance. The ability to perform a simple perception test was not affected while suffering from muscle soreness.

The eccentric exercise is thought to cause damage to the connective tissue and muscle cell membrane leading to a build up of fluid around the joint. This increased edema may explain the increase in muscle stiffness observed. Further research is required to determine whether changes in muscle tone are also observed following isometric and concentric exercise.

DECLARATIONS

(i) I, Fraser Gillies McGlynn, hereby certify that this thesis, which is approximately 70,000 words in length, has been written by me, that it is the record of work carried out by me and that it has not been submitted in any previous application for a higher degree.

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CHAPTER 1

INTRODUCTION

1.1 INTRODUCTION

There is evidence which indicates that muscle damage has occurred following unaccustomed strenuous exercise. Muscle soreness, muscle stiffness, the level of muscle proteins in the blood and urine and strength are all parameters which investigators have monitored following a bout of exercise designed to induce muscular damage.

In the period following unaccustomed strenuous exercise the muscles that have been exercised often feel tight and discomfort is felt when an attempt is made to change the resting length of the muscle. This tightness or resistance to stretch in the resting muscle is taken to be the stiffness of the muscle. Muscles in their "normal" state are stiff to a greater or lesser extent during contraction and relaxation, respectively. However, there is a perception of increased stiffness following unaccustommed exercise which is accompanied by a gradual development of soreness in the muscle groups that were exercised. This increase in stiffness and soreness reaches a peak 24-48 hours later and is often referred to as Delayed Onset Muscle Soreness (DOMS).

Groups most likely to experience DOMS are those individuals who perform muscular work having followed no regular exercise programme prior to the particular activity which has caused the soreness. Similarly, individuals increasing their training load or individuals who perform a different type of exercise, although perceiving themselves as fit, can expect to experience DOMS to a certain degree. Although performing exercise during DOMS is uncomfortable at first, there is temporary relief from the pain and stiffness if the exercise is continued. As the exercise progresses the symptoms disappear, but return soon after the termination of the exercise to resume their normal time course. DOMS can last up to seven days.

In an attempt to investigate the phenomenon of DOMS, investigators have firstly had to induce muscle soreness in their subjects. The extent of the damage and subsequent soreness depends on the particular exercise being carried out. In an exercise like downhill running, several muscle groups are affected, and a longer time period is required to elicit a reasonable level of damage (Donnelly et al. 1988). However, greater damage can be created in a specific muscle group which can be exclusively isolated by a particular exercise. For example, the lowering of weights by the forearm flexors isolates the brachialis and biceps brachii muscles and severe strain can be put on the muscle group containing them (Armstrong 1984, Clarkson et al. 1987). From the wide variety of exercise regimens that have been used, investigators now believe that

exercise involving eccentric muscular contractions creates greatest damage. Asmussen (1956) was the first of many researchers to show that muscle stiffness and soreness developed in muscles that had performed exercise using eccentric contractions and not in muscles that had performed concentric contractions. The site of damage is open to considerable debate; so too is the mechanism that eventually results in stiffness and soreness, however recent research suggests that the injury occurs to the muscle connective tissue (Howell et al 1985, Jones et al. 1987, Fleckenstein et al.1989, Hamill et al.1989, Clarkson & Tremblay 1988).

The delay between performing the exercise and the development of stiffness and soreness suggests that an inflammatory response has taken place, and several investigators have attempted to treat the symptoms with anti-inflammatory drugs. Damage has been reduced using anti-inflammatory drugs in animal studies (Wagner & Critz 1968, Salminen & Kuhlstrom 1985). Pain has been slightly reduced in some studies using aspirin (Bansil et al. 1985, Francis & Hoobler 1987), but other investigators have been unsuccessful in reducing the pain (Kuipers et al. 1985, Headley et al. 1986, Donnelly et al. 1988).

Following an exercise bout that has induced DOMS, certain adaptations are seen to take place. Repetition of the same amount of exercise (work) several weeks after the first bout leads to a decrease in the response of parameters that indicate that damage has occurred (Byrnes et al.1985, Newham et al. 1987, Clarkson & Tremblay 1988, Seaman & Ianuzzo 1988). The first exercise bout seems to have the effect of protecting the exercised muscles from future insult. In exercise involving isometric contractions this protective effect lasts up to four weeks (Clarkson, 1985). In exercise involving eccentric exercise the effect lasts up to six weeks (Byrnes et al. 1985). An exercise bout involving a third of the work of a future more strenuous bout (24 maximal contractions compared to 70 maximal contractions) has been shown to protect individuals from serious discomfort following the second more strenuous bout (Clarkson & Tremblay 1988). This has important implications for coaches and athletes when introducing new elements to a training schedule. By introducing new elements gradually muscle stiffness and soreness can be avoided and interruptions to training can be minimised. Jones et al.(1987) examined pain and stiffness following exercise of the biceps muscle and found that stiffness and soreness developed and reached a peak simultaneously, forty eight hours post exercise. Stiffness was measured as the force required to forcibly extend the forearm. The technique used by these investigators for measuring stiffness had several limitations. It is difficult to know how much the subject was resisting the extending force since during DOMS greatest pain is experienced at the extremes of joint movement. Also in severe cases of DOMS the arm will not fully extend. In such cases it would be difficult to quantify stiffness on a daily basis. This result has not been repeated to date.

Walsh and co-workers (1980) developed a reliable technique for measuring stiffness using an electrical motor to deliver torques to the supported relaxed wrist. By recording the transient oscillation which resulted, Walsh et al.(1980) found that at a certain torque, movement of the wrist reached a maximum. Analysis of the frequency spectrum revealed the resonant frequency from which inferences could be made about the stiffness. This technique has been used successfully by Lakie et al.(1984) to measure resonance at the wrist and by Lakie & Robson (1988) to measure the stiffness of the forearm following exercise of the finger.

In research into rheumatoid arthritis(RA) Helliwell et al.(1988) measured the stiffness of the metacarpalphalangeal joint in 135 patients with progressive degrees of severity of RA. Helliwell et al.(1988) similarly measured the stiffness of the same joint in 135 "normals". Contrary to the previously held belief that RA patients showed increased joint stiffness, Helliwell et al.(1988) showed conclusively that this was not the case. The group of "normal" subjects did show greater stiffness in the joints than the patients suffering from RA. This may also be the case with individuals suffering from DOMS: the inability to move the arm as efficiently may be due to the fact that the individual knows the pain that will be incurred when a particular movement is carried out and not due to an actual increase in stiffness.

If sportsmen and sportswomen suffer from muscle stiffness and soreness then this can affect the ability to maintain the same level of work during training, or even interrupt training completely. This decrease in work could be as a result of individuals holding back in their efforts to avoid experiencing further discomfort. This in turn affects preparation for competition. Further, some sports involve competition over several days and as a result the athlete is expected to produce higher standards of performance as the competition progresses. If the individual suffers from increased muscle stiffness and soreness over the period of the competition then there could be modifications in movement patterns which would alter the sports technique and may even cause injury. The overall result is a reduction in skill level and hence in the level of the performance.

A greater level of understanding of muscle soreness and stiffness and its effect on performance is therefore necessary for coaches, athletes, physiotherapists and orthopaedic surgeons, as well as individuals beginning exercise for the first time. Using the technique for measuring stiffness developed by Walsh & co-workers this research project will therefore investigate further the relationship between muscle stiffness, muscle soreness and performance.

1.2 GUIDE TO EXPERIMENTS

Experiment 1 (Chapter 4)

This study investigates the effect of unaccustomed exercise on the stiffness of the muscles involved in flexion and extension of the forearm. It is hypothesised that muscle soreness will increase following exercise, and will reach a peak 24 to 48 hours post exercise. Further, it is hypothesised that muscle stiffness will increase post exercise and will reach a peak 24 to 48 hours post exercise

Experiment 2 (Chapter 5)

While adaptation leading to reduced muscle soreness and damage has been observed following eccentric exercise, such an adaptation in muscle stiffness has yet to be investigated. This study investigated whether muscle stiffness increased following eccentric exercise of the forearm flexors and, if so, whether an adaptation occurred leading to reduced muscle stiffness following a second bout of the same exercise six days later. To assess the extent of the damage caused as a result of the eccentric exercise the level of plasma creatine kinase (CK) was measured. The hypothesis is that muscle stiffness, muscle soreness, the level of plasma creatine kinase and limb girth will increase following a bout of eccentric exercise, but such increases will not be seen following a bout of the same exercise repeated 6 days later.

Experiment 3 (Chapter 6)

The study investigated a reduction in the angle of elbow flexion during DOMS, and whether a reduction in the range of motion affected motor performance, that is, the ability to perform a simple perception test. The test examined individuals' measuring ability pre- and post-exercise. The post-exercise test was performed when the symptoms of DOMS were thought to be at their peak. It is hypothesised that the ability to reproduce a "mirror image" is reduced when the state of the muscles on opposite sides of the body is different, that is, when the individual is suffering from DOMS in the forearm flexors of the non-dominant arm, with no simultaneous DOMS in the forearm flexors of the dominant arm

CHAPTER 2

REVIEW OF LITERATURE

2.1 ELECTRICAL, MECHANICAL AND BIOCHEMICAL EVENTS DURING MUSCLE CONTRACTION.

2.1.1 Muscle Contraction

Any study concerning exercise is without meaning unless the reader has a basic knowledge of the structure and function of muscle. Many physiological texts provide a thorough account of muscular contraction (Vander, Sherman & Luciano, 1986, Jones & Round 1990, Wilmore & Costill 1994) and the reader is referred to these should additional information be required. The present section will provide a brief account of the electrical, mechanical and biochemical events that take place when muscle is contracting.

Figure 2.1 The levels of structural organisation in skeletal muscle

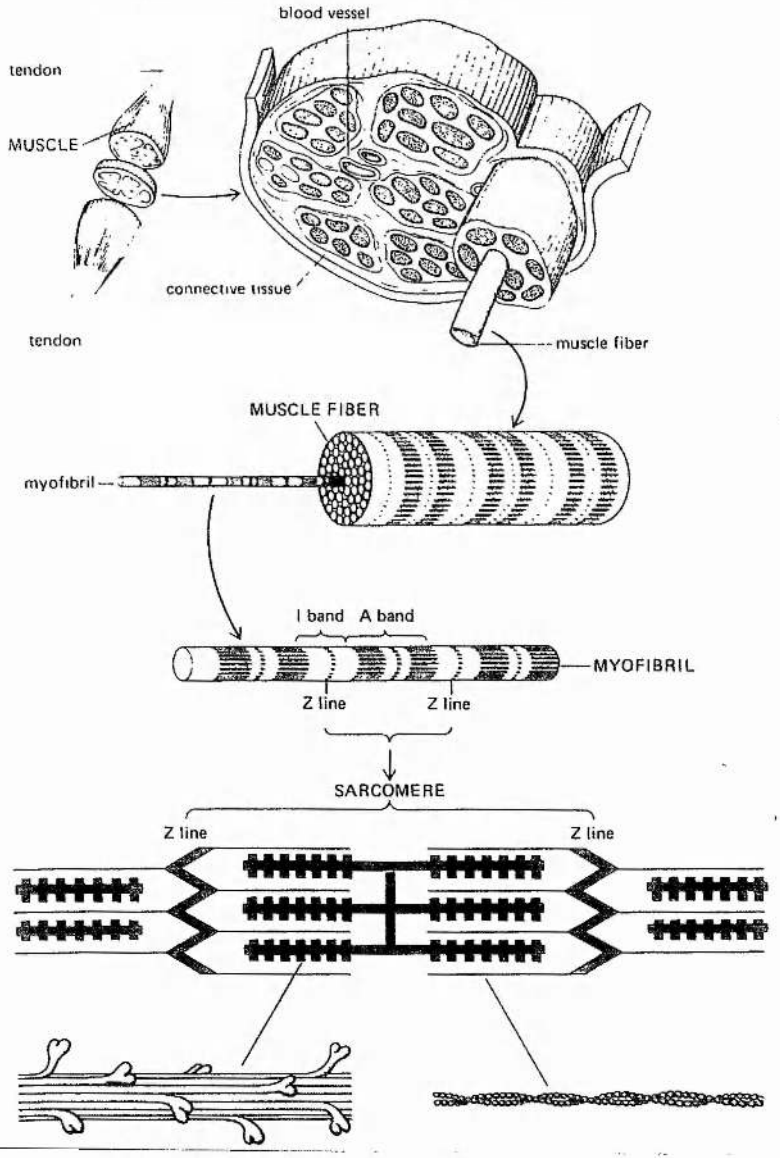
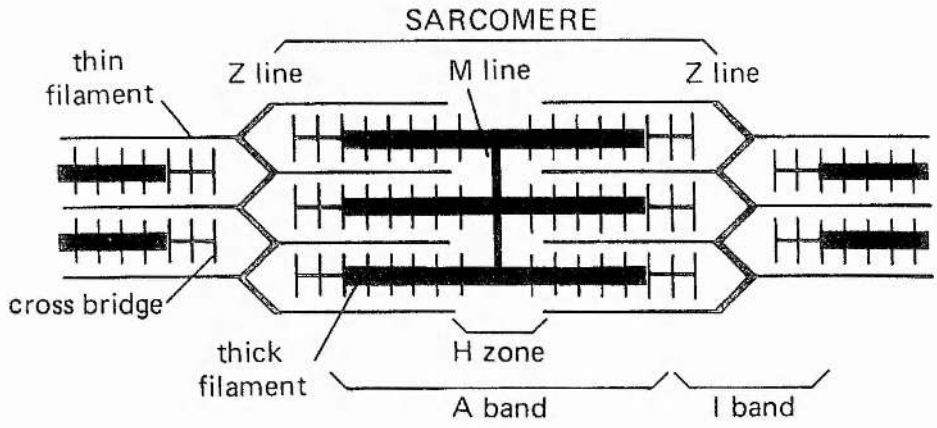


Figure 2.1 shows the levels of structural organisation in skeletal muscle (Vander, Sherman & Luciano, 1986, p257). The muscle is composed of bundles of fibres which are joined together by connective tissue. The muscle fibre, which is a single muscle cell, can have a diameter ranging from 10 m to 100 m and a length of up to 30 cm depending on its anatomical location. The muscle fibre is composed of myofibrils. The number of myofibrils in a fibre can range from hundreds to thousands, depending on the diameter of the fibre. Each myofibril is composed of smaller filaments which form a repeated pattern thoughout its length. One unit of this is pattern is termed a sarcomere. The sarcomeres are connected end to end and run along the length of the myofibril. The smaller filaments within the myofibril are termed actin (thin filament) and myosin (thick filament). It is the interaction between these two types of filament that allow muscle contraction to take place.

Figure 2.2 The filament organisation of myofibrils



From Figure 2.2 (Vander, Sherman & Luciano, 1986, p259) it can be observed that there is an area of overlap of the actin and myosin. Protruding from the myosin are small structures named cross bridges which are portions of myosin molecules. The cross bridges bridge the gap between the actin and myosin. During muscular contraction, the actin and myosin filaments are connected via the cross bridges, and the cross bridges exert force on the actin. During muscular contraction, neither the myosin (thick filament) or actin (thin filament) change their length; the two filaments slide past each other.

Observation of these actions led to Huxley's theory of the sliding - filament mechanism of muscular contraction (Huxley 1957, Huxley & Simmons 1971). It is the action of the cross bridges which results in the movement of the filaments across each other. When connected to the actin, the cross bridge completes an arc of movement parallel to the long axis of the thick

filament, similar to that of a rowing action. This has the effect of moving the actin over the mysosin. Since the resultant displacement caused by one arc of the cross bridge is very small, many similar actions are required. As a result, at the end of the arc, the cross bridge detaches itself, flips back, and performs an action identical to the previous one. The combined result of this continuous cycle of movement from all the cross-bridges is that muscle contraction takes place. The reader is referred back to Figure 2.1 to realise the scale on which this cross bridge cycling operates.

The force exerted by a contracting muscle on an object is known as muscle tension. The force exerted on the muscle by the object is the load. Muscle tension and load are opposing forces. The term contraction refers to the turning on of the tension generating process in muscle. Whether or not contraction produces movement depends on the magnitude of the load and the tension produced by the muscle. To move an object, muscle tension must be greater than the load.

When a muscle develops tension but does not change length, the contraction is said to be isometric. An isometric contraction takes place when a muscle supports a load in a fixed position, or when the load exceeds the muscle tension being exerted. In contrast, an isotonic contraction takes place when the muscle exerts constant tension on a load while changing its length. A muscle performs an isotonic contraction when moving a load. Isotonic contractions can be further classified into concentric and eccentric contraction. In a concentric contraction the muscle shortens while exerting tension, for example the biceps brachii performs a concentric contraction when flexing the foream (bending the arm). In an eccentric contraction the muscle lengthens while exerting tension. For example, the biceps brachii performs an eccentric contraction when extending the forearm (straightening the arm).

The maximum number of cross bridges that can be formed will be set by the degree of overlap between actin and myosin, and by the number of acting binding sites exposed by the binding of calcium to troponin. However, not all the cross bridges will be attached at any one time, since the rates of attachment and detachment are different. The number of cross bridges, n, attached at any one time is given by

$$n = f/(f + g)$$

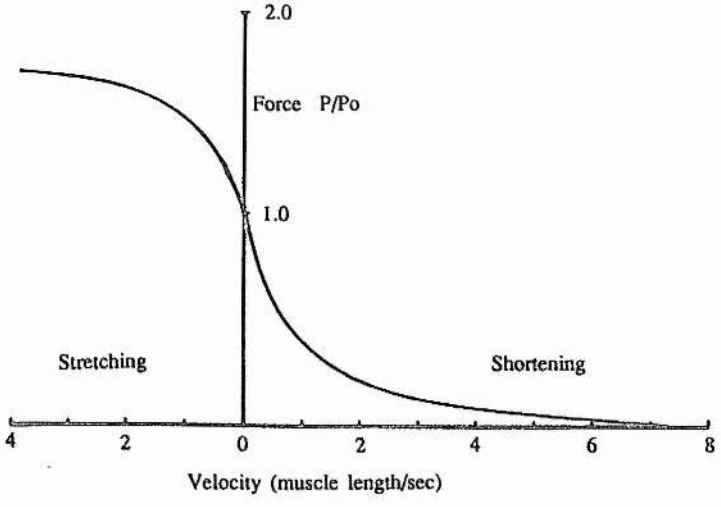
where f = rate constant for attachment and g = rate constant for detachment.

During an isometric contraction it is thought that 80% of cross bridges will be attached, which suggests that f will be around twice g. In concentric contractions, when the muscle is shortening, the force sustained is less than isometric force since the force exerted by each cross

bridge is less than in the isometric state, and since there are fewer cross bridges attached. Jones and Round (1990) state "If the muscle is shortening and the actin binding sites are moving past the myosin cross bridges, then there will only be a limited time during which attachment can take place. The faster the velocity of shortening, the shorter the time available and the lower the proportion of cross bridges that will manage to attach in the region over which the cross bridge can exert a useful force. The number of attached cross bridges, and therefore the force generated though shortening, will be less than during an isometric contraction."

In contrast, during a contraction in which the muscle lengthens the force exerted is greater than the isometric force. The force exerted varies with velocity and with increasing velocity of stretch the force exerted begins to plateau at around 1.8 times the isometric force. This increase in force can be explained through Huxley's model, that the compliant portions S2 of the individual cross bridges are stretched further during an eccentric contraction than during an isometric contraction. This occurs since the rate constant for detachment of cross bridges in an isometric contraction is lower than constant for detachment of cross bridges in an eccentric contraction. With increasing speed of stretch in an eccentric contraction, the number of cross bridges attached will decrease, but those attached will sustain more force. Thus the force maintained by a muscle during stretch tends towards a plateau at higher velocities. This relationship can be illustrated in the characteristic force-velocity curve (Figure 2.3, Jones & Round 1990, p33).

Figure 2.3 Force of mouse soleus muscle during stretch and shortening



2.1.2 Electrical Aspect of Muscle Contraction

An electromyogram (EMG) is the electrical signal association with the contraction of a muscle. Voluntary muscular activity results in an EMG that increases in magnitude with tension. Variables that can influence the signal are velocity of shortening or lengthening of the muscle, rate of tension build up, fatigue and reflex activity (Winter 1977).
Muscle action potentials (m.a.p.) are the electrical signals generated in the muscle fibres, similar to the way axons transmit action potentials. Electrodes placed on the surface or inside the muscle record the sum of all muscle action potentials being transmitted along the muscle fibres at that point in time. The further away the muscle action potentials are from the electrode site, the weaker the signal (Winter 1977).

Each motor unit (a motor neurone plus the fibres it activates) is controlled by a motor neurone through a synaptic junction called a motor end plate. An action potential transported down the motor neurone arrives at the motor end plate and triggers a sequence of electrochemical events. In summary, acetylcholine is released and diffuses from the axon terminals to the motor end plate on the muscle fibre. Acetylcholine binds to receptor sites on the motor end plate membrane, opening ion channels that are permeable to sodium and potassium. Movement of the ions across the motor end plate membrane depolarizes the membrane, producing the end-plate potential (EPP). If the EPP is large enough it will reach a critical level and an action potential is initiated in the adjacent muscle fibre membrane.

This muscle action potential triggers the release of calcium ions from the sarcoplasmic reticulum which then diffuse to bind to troponin. This action results in tropomyosin moving away from its blocking position to expose the cross bridge binding sites on the thin filament. (Troponin and tropomyosin are two regulatory proteins located on actin). The energised cross bridges on myosin bind to the actin (A + M* ADP·Pi ---> A·M*·ADP·Pi)which triggers the release of energy stored in myosin (A·M*·ADP·Pi ---> A·M + ADP·Pi) and produces an angular movement of each cross bridge. ATP then binds to myosin and breaks the actin and myosin link thus allowing the cross bridge to dissociate itself from actin (A·M + ATP ---> A + M·ATP). The ATP bound to myosin is split, transferring energy to the myosin cross bridge, producing an energised cross bridge (M·ATP --->M*ADP·Pi). This process is repeated and produces movement of the thin filaments across the thick filaments. The cross bridge cycling will continue as long as calcium remains bound to troponin. When calcium is transported into the sarcoplasmic reticulum by Ca ATPase the concentration of calcium in the cytosol decreases. The removal of calcium ions from troponin restores the blocking action of tropomyosin, causing the cross bridge cycle to cease. The muscle then assumes a relaxed state.

The electrical and chemical events occurring in muscle fibres are the same in both isometric and isotonic contractions, that is, the cross bridges are activated and exert force on the thin filaments. In an isotonic contraction, the thin filaments move past the thick filaments, causing the muscle to shorten or lengthen. In an isometric contraction, although force is still being exerted by the cross bridges on the the thin filaments, the thick and thin filaments do not slide past each other.

In examining the electromyographic signals from isometric, concentric and eccentric contractions, investigators (Bigland & Lippold 1954) have shown that when a voluntary isometric contraction of a muscle is made, the electical activity bears a linear relationship to the force that is being exerted. These investigators also showed that at a constant velocity of shortening or lengthening, the electrical activity is directly proportional to the force. However, the slope (gradient) of the correlation falls off during lengthening. This means that the degree of muscle excitation required to produce a given force of contraction is smaller when the muscle is forcibly stretched than it is when the muscle shortens at the same velocity. At constant force, the electrical activity increases linearly with velocity of shortening, but it decreases when the muscle is being lengthened (Bigland & Lippold 1954). The integrated EMG activity is the same in maximal contraction of different speeds, indicating the same degree of muscle recruitment in maximal efforts; it does not matter whether the contraction is concentric, isometric or eccentric (Komi 1973, Rodgers & Berger 1974).

2.1.3 Mechanical Aspect of Muscle Contraction

The mechanical response of a muscle fibre to a single action potential is referred to as a "twitch". In an isometric twitch the latent period (the interval of time before the tension begins to increase) lasts a few milliseconds. It is during this time that the process which results in cross bridge activity is taking place. In an isotonic twitch the latent period is longer since the change in length does not begin until muscle tension starts to exceed the load. The relationship between the two variables is such that the heavier the load then the longer the latent period.

Since a single action potential in a muscle fibre lasts for 1 - 2 ms, in contrast to the mechanical response which may last for 100 ms, it is possible for a second action potential to be initiated during the period of mechanical activity. The reaction to a second action potential is that the mechanical response is increased, and the result is known as "summation". The contraction of a muscle fibre can be sustained through repeated stimulation of the fibre at a rate which does not allow the fibre to relax completely between each stimulation. Maintaining a contraction in response to a repetitive stimuli is termed "tetanus". At a low frequency of stimulation the tension generated fluctuates creating an oscillating effect termed as "unfused tetanus". At a high frequency of stimulation no fluctuation takes place an the effect is termed "fused tetanus".

An increase in the frequency of action potentials results in an increase in tension to a point where there is maximum tetanic tension.

The mechanism responsible for the increase in the mechanical response of the muscle fibre to repetitive stimulation involves the elastic properties of muscle fibres, which involves a dissociation between internal tension (the capacity to generate tension by cross bridge activity) and external tension (the tension applied to the load) (Vander Sherman & Luciano, 1986). The maximal tension that can be generated by cross bridge activity at any one time is termed the active state. The calcium produced as a result of one action potential is almost sufficient to ensure that the cross bridges are entirely active and able to exert maximal active state tension. The active state tension is very different from that of the external tension that is exerted on a load during an isometric twitch. The internal tension is transferred through the Z-discs, and the tendons to the load. These structures are collectively known as the series elastic component (SEC) as they all have elastic properties to some extent. The SEC resembles a spring between the force generating cross bridges and the load. The force produced by the cross bridges (the contractile component (CC)) stretches this "spring", which in turn transmits its tension to the external load. The tension in the SEC is therefore the external tension, and is related to the extent to which it has been stretched as a result of cross bridge activity. It takes time to stretch the series elastic component. In a single twitch the tension in the SEC is still rising at a time when the cross bridge tension is declining from its peak value as calcium is removed. Therefore the peak tension developed by the SEC (the external tension) never exceeds the maximal tension in the CC during a single twitch.

Therefore, when a muscle is continually stimulated, then the cross bridges remain active for a longer period, since the level of calcium within the muscle cell is being maintained. Because the cross bridges are active for a longer period of time, the SEC is stretched further and the tension transmitted to the load is greater and summation occurs. In summary, in an isometric contraction the force is generated through the action of the contractile component on the SEC, which results in the SEC being stretched. A concentric contraction when the load is attached to the end of the muscle is always preceded by an isometric contraction with re-arrangments of the lengths of the CC and SEC. The final movement begins when the pulling force of the CC on the SEC equals or slightly exceeds that of the load. In an eccentric contraction some force, for example gravity, and the antagonist muscles force the muscle to lengthen.

The length - tension relationship in muscle refers to the capacity for force generation thtoughout the length range of a muscle. The length - tension relationship can be explained in terms of the sliding filament mechanism (Huxley 1954). Stretching a muscle results in a change in the overlap between the thick and thin filaments. At one extreme, when the muscle is stretched, to

the extent that there is no overlap between the filaments, there is therefore no capacity to generate force. At the other extreme, when the thin filaments from the two halves of a sarcomere overlap this interferes with cross bridge binding and decreases the number of active cross bridges. Also, in this extreme state of overlap, the thick filaments become compressed against the two Z-lines, opposing tension development. In general, the resting length of the muscle in its relaxed state is thought to be the optimal length at which maximum cross bridge binding can take place (Vander et al, 1986).

2.1.4 ATP and Muscular Contraction

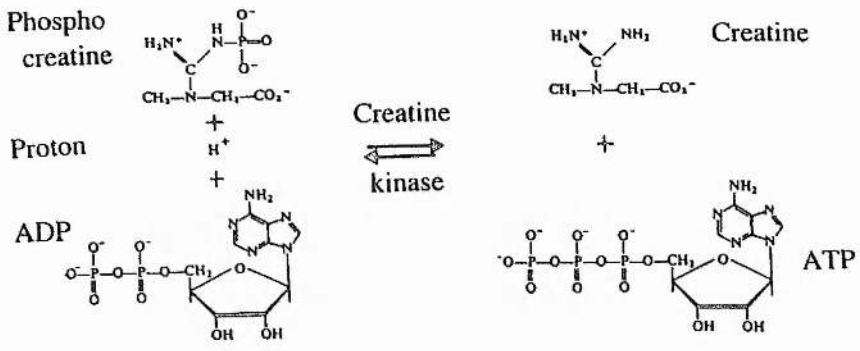
In relation to muscular contraction and relaxation, ATP performs three major functions. Firstly, the splitting of ATP is linked to the movement of the cross bridges. Secondly, it is necessary that ATP binds to myosin in order to break the link between actin and the cross bridges. Thirdly, the energy released from ATP-splitting is used by the calcium pump to pump calcium ions back into the sarcoplasmic reticulum so that relaxation wil take place.

As the store of ATP that exists at the start of muscular contraction is limited, the contraction process is dependent upon various systems to provide ATP. A muscle fibre can form ATP in three ways. The first is by phosphorylation of ADP by creatine phosphate. The second is by oxidative phosphorylisation of ADP in the mitochondria, and the third by substrate phosphorylation of ADP, mainly by the glycolytic pathway. Each of these distinct systems produces discrete levels of ATP at a rate unique to its own system. For example, phosphorylation of ATP by creatine phosphate produces ATP rapidly, but the amount of ATP that can be formed is limited by the initial concentration of creatine phosphate. The reader is referred to any physiological text for a more detailed account of the production of ATP (Vander Sherman & Luciano 1986, Astrand & Rodahl 1986, Wilmore & Costill, 1994).

Of particular interest in the formation of ATP is the enzyme creatine kinase (Figure 2.4, Jones & Round 1990, p37), the levels of which have been monitored by many researchers as a means to ascertain the extent of muscle damage following exercise. Creatine kinase is found exclusively within the muscle cell, and its presence in the bloodstream is an indication that damage has occurred to the cell.

There is a continuing interest in the mechanism which results in the transformation of energy from the hydrolysis of ATP into mechanical work. Jones and Round (1990) state that "there is general agreement that the interaction of actin myosin and ATP proceeds in a stepwise fashion, and that the internediate stages can, or eventually will, be identified with different mechanical steps in the cross bridge cycle."

Figure 2.4 The action of creatine kinase



In examining the metabolic cost of muscular contractions, investigators state that at a given work rate, the oxygen uptake when performing eccentric exercise is significantly less than when performing concentric exercise (Asmussen 1953, Bigland & Lippold 1954). Huxley's model suggests that one ATP molecule is split for every cross bridge detachment, and that as the velocity of shortening and lengthening increases then so too will the ATP splitting. However, Jones and Round (1990) suggest that since ATP splitting has been shown to be very low during stretching (Curtin & Davies 1973), the model needs modification, and does not explain why eccentric contractions have a lower metabolic cost than concentric contractions. Jones and Round (1990) suggest that a further state should be included in the model , "where cross bridge detachment does not involve ATP splitting - the back reaction of the f (the rate constant for attchment)". They suggest further that detachment during shortening would primarily involve the splitting of ATP, however, during stretch, the detachment would primarily be a mechanical process.

2.2 MUSCLE DAMAGE AND REPAIR

There are two possible explanations for the damage that is caused as a result of exercise. These explanations are linked to two separate mechanisms. The first explanation is that the damage is caused by metabolic mechanisms. The pain associated with metabolic mechanisms occurs during exercise and characteristically builds up until the activity is terminated. Thereafter the pain subsides. The second explanation is that the damage is caused by mechanical mechanisms. The pain associated with mechanical damage occurs 12 to 24 hours after the exercise and can be prolonged for up to a week.

2.2.1 Metabolic Mechanisms

Traditionally, athletes and coaches have associated muscle stiffness and soreness with the accumulation of metabolites, in particular lactic acid, that occurs during exercise. This theory was proposed by investigators who believed that ischaemic and hypoxic conditions in the muscle led to degeneration of the tissue (DeVries 1966, Armstrong 1984). Ischaemia is defined as a reduced blood supply to the tissue and hypoxia as a deficiency (but not the complete absence) of oxygen at the tissue level. DeVries (1966) proposed that exercise began a "vicious cycle" that led to muscle spasm brought about by ischaemia. The spasm was evidenced by an increase in resting electromiography (EMG), and was thought to lead to further ischaemia through compression of blood vessels. This in turn would reduce the oxygen supply to the exercising muscle and during these hypoxic conditions glycogen reserves would be utilised to produce muscular contraction. The reduction in blood flow through exercising muscles would slow down the removal of waste products from the muscles and the accumulation which would ensue would result in fatigue. However, no other investigators have been able to support DeVries' view that there was an increase in the resting EMG (Howell et al. 1985, Jones et al. 1987).

Evidence exists to suggest that injury caused as a result of ischaemia resembles the damage observed following exhaustive endurance exercise, for example long distance running (Sjostrom et al. 1987). Ischaemia can occur during marathon running possibly as a result of the delayed clearance of lactic acid that has accumulated during the exercise, and it is suggested that this may cause the damage (Hoppeler 1986). However, Armstrong (1986) suggested that the damage to the gastrocnemius muscle in marathon runners was as a result of the continual lengthening (eccentric contractions) of the muscle during the braking action of the running stride. When the foot strikes the ground this has the initial effect of slowing the body down and the gastrocnemius muscle lengthens at this point. As the body rotates forward over the foot, which acts as a pivot, the muscle subsequently shortens and contributes to the propulsive action which accelerates the body forward. There is considerable strain on the muscle considering that there are many foot strikes during a three to four hour run. Although the eccentric contractions were thought by Armstrong(1986) to be the main cause of the damage, the ischaemia may have contributed to the extent of the damage.

As early as 1956 Asmussen showed that muscles which performed only concentric exercise fatigued earlier than muscles that performed only eccentric exercise when he exercised humans on a cycle that had been modified to permit eccentric and concentric exercise. If the accumulation of waste products was an explanation for the manifestation of DOMS then muscles that had exercised concentrically to exhaustion would exhibit greater damage and pain than those exercised eccentrically. This has been shown to be untrue (Armstrong 1983,

Schwane et al. 1983). In two studies, one on rats (Armstrong et al. 1983) and one on humans (Schwane et al. 1983), downhill running induced severe damage and muscle soreness with very little production of lactic acid. Uphill running, however, produced no damage or soreness, but caused fatigue through the accumulation of lactic acid. These studies question whether the lactic acid has a direct effect on the amount of pain or damaged experienced following exercise and add support to the theory that DOMS is as a result of mechanical damage.

2.2.2 Mechanical Mechanisms

Blix in 1895 was the first to discover that under tetanic stimulation the tension produced by a muscle during shortening (concentric) contractions was less than the corresponding tensions during isometric contractions, and that the tension in the muscle during maximum isometric contractions was less than the tension produced during lengthening (eccentric) contractions. Asmussen (1953) confirmed this through examination of the integrated electromyograms of concentric and eccentric contractions. Based on the assumption that the area under the curve covered by the integrated electromyograms was proportional to the number of discharges, it was clear to Asmussen (1953) that more fibres were active during concentric contractions than during eccentric contractions. This offered an explanation for the reduced oxygen cost of eccentric work compared to concentric work, but Asmussen also believed that this was the main reason that muscular soreness developed in muscles that had performed exercise using eccentric contractions, and not in muscles that had performed concentric contractions (Asmussen 1956). Since there were less fibres active during eccentric contractions than concentric contractions the developed force was distributed over a smaller cross-sectional area and as a result there was greater tension per active muscle fibre. Although Asmussen's conclusion is valid it is unclear whether the soreness was related to the muscle fibres being overloaded or to the connective tissue taking the strain. Subsequent research has supported Asmussen's claims and have tried to identify the site of the damage (Friden et al. 1983, Evans et al. 1986).

McCully and Faulkner (1985) tested the hypothesis that eccentric contractions result in greater injury to skeletal muscle fibres than isometric or concentric contractions. In their well controlled study these investigators anaesthetised mice, secured them to a platform and cut the tendon of the extensor digitorum muscle. The tendon was then attached to the lever arm of a position feedback servo motor and a protocol consisting of either isometric, concentric or eccentric contractions was performed. The tendon was then reattached and the mice allowed to recover. Over a period of 1 to 30 days the muscles were removed for histological examination. Compared to isometric and concentric contractions, which showed no difference histologically, and only a reduction of 20% in maximum isometric force, the eccentric

contractions caused a degeneration of 20% of the fibre population and a reduction of 78% in maximum isometric force. McCully and Faulkner (1985) concluded that greater injury to skeletal muscle was caused by eccentric contractions than by concentric or isometric contractions.

The work of these investigators allowed a thorough investigation of the muscle exercised and a clear picture of the extent of the damage was obtained. This extensive examination is not possible in humans since medical codes of practice only allow that a small sample of muscle be removed. The hazards and shortcomings of muscle biopsy techniques are discussed later in section 2.2.3. The study by McCully and Faulkner is an excellent example of how the results from animal studies can be used to hypothesise that there will be similar findings in humans. This study is one of considerable note in research into DOMS and muscle damage. Animal studies are the crystal ball of physiological research, helping investigators to create hypotheses that can be tested in humans.

2.2.3 Site of Damage

The best evidence that can be obtained to support the claims that there is damage to muscle tissue can be seen by observing histological and ultrastructural changes in tissue that has been extracted by biopsy. Muscle biopsy studies have shown that there are disturbances of the characteristic cross-striated band pattern following repetitive eccentric exercise. Friden et al. (1983) found that there was broadening, streaming and total disruption of the myofibrillar Z-band, particularly in type II fibres and suggested that during overloading the Z-discs constitute a weak link in the myofibrillar contractile chain. Damage also occurs to the sarcolemma (cell membrane) and to the A and I bands of the muscle (Friden et al. 1983). Connective tissue is also damaged as a result of eccentric exercise (Abraham 1977, Friden 1983). Since connective tissue runs in parallel from one end of the muscle to the other, it also experiences the high tensions that are associated with eccentric contractions. The repeated tension which overloads the tissue leads to structural damage. Hydroproxoline is an amino acid found in connective tissue and its presence in the urine is indicative that structural damage has occurred. Abraham (1977) detected higher than normal levels of hydroproxoline in the urine of subjects that were suffering from DOMS.

Some confusion exists over the time taken for the muscle to repair itself following damage. Friden et al. (1981) observed biopsies that had been taken from the soleus muscle two and seven days following continual stair descents (eccentric exercise) that induced DOMS. In the sample taken seven days post exercise there was less Z-disc disruption than was observed in the sample taken two days after the exercise. This suggested to the investigators that the repair process had begun by the seventh day. In subsequent research involving eccentric exercise on

a cycle ergometer, Friden et al (1983) noted that the repair process had begun by three days after the exercise and by six days post exercise the fibres appeared normal. In research carried out in rats Armstrong et al. (1983) observed monocytes, macrophages and fibroblasts in the damaged tissue on the day following the exercise, suggesting that the repair process had begun by this time. McCully & Faulkner (1985) also reported that in mice damage to the tissues was followed by fibre degeneration and infiltration of macrophages. Myoblasts and myotubes were evident four days after the exercise.

Jones et al. (1986) examined biopsies from the calf and biceps muscles of humans that had performed eccentric exercise to induce muscle soreness. These investigators report that there was little or no change in the first seven days following the exercise but thereafter degenerating fibres were observed, and by twenty days post exercise there were signs of regeneration. In one subject that exhibited extreme damage, type II fibres seemed to be preferentially damaged.

The discrepancies in these studies could be explained by genuine differences, but alternatively by sampling errors. The particular piece of muscle removed can be very small, perhaps 5mm long with a diameter of 2mm in humans, with smaller samples taken from animal muscle. The damage caused by DOMS can be localised and finding the true extent of the damage has been described by Armstrong (1986) as similar to searching for a needle in a haystack. For example, Armstrong et al. (1983) examined the complete cross sections of all the ankle extensor muscles of rats that had performed treadmill exercise and noted that most of the damage occurred in the soleus muscle which only made up 6% of the total muscle mass. If a biopsy was taken in the study cited, that had not come from the soleus, then it may have been presumed that no structural damage had occurred. The removed sample would not have been representative of the damage that had occurred. Evidence from biopsy studies should be viewed with caution bearing in mind the errors that can occur if the sample is not representative of the true extent of damage.

2.3 MUSCLE SORENESS

2.3.1 Exercise to Induce Muscle Soreness

A variety of exercise regimens have been used to induce muscle soreness experimentally. Asmussen (1953, 1956) conducted his preliminary research on a modified bicycle designed to produce concentric and eccentric exercise, whilst Knuttgen et al. (1982) and Evans et al. (1986) used a modified cycle ergometer to induce muscle soreness. Some investigators have used stepping exercise to induce soreness (Newham et al. 1983, Newham et al. 1986, High et al. 1989, Buroker & Schwane 1989), while others have used repeated lifting and lowering of the body by plantar- and dorsiflexing the ankle of one leg (Bobbert et al. 1986).

Equipment designed originally for the purposes of weight training has been modified by some investigators so that muscle soreness can be induced in specific muscle groups. Investigation using such a method has been carried out on the leg extensors (Seaman & Ianuzzo 1988, Clarkson et al. 1987), the leg flexors (Clarkson et al.1987), the elbow flexors (Jones et al.1987, Francis & Hoobler 1987, Kroon & Naeije 1988, Clarkson &Tremblay 1988, Ebbeling & Clarkson 1990), and the lower leg anterior compartments (Friden et al.1988). More recently, downhill running on a motorised treadmill has been used to induce muscle damage (Schwane et al.1987, Donnelly et al. 1988, Hamill et al.1989).

The quality of any investigation into DOMS is dependent on the extent of the muscle stiffness and soreness experienced post exercise. This in turn is as a result of the method used to induce stiffness and soreness. In studies in which several muscle groups perform the exercise, for example downhill running, modified cycling or stepping exercise, there is a wide variety of pain responses. This is due in part to differing levels of pain threshold in the individuals taking part in the study, but also as a result of the time spent exercising. Individuals performing running, cycling or stepping must exercise longer to produce pain similar to that experienced following exercise that isolates a single muscle group, normally carried out using modified weight training equipment. Fatigue sometimes prevents subjects performing running, cycling or stepping for longer periods and the required damage can not be inflicted upon the muscle to produce a high pain response. Isolating a muscle group and performing exercise using weight training equipment seems to guarantee that there will be pain post exercise and allows researchers to explore the theme of their research. If there is a low pain response or no pain response, it is difficult for the study to be carried out, and any conclusions drawn must be viewed carefully. Therefore a compromise has to be made when inducing stiffness and soreness for experimental research. If the results of any research findings are to be applied to sport it is most desirable that the stiffness and soreness are induced by a method which mirrors a specific sporting action or actions. This is not always possible, for the reasons discussed above, and the "pure" research is hypothesised to be representative of the "applied" situation.

Hough (1902) was the first investigator to describe the pain associated with strenuous exercise that characteristically develops in the days following the exercise. The symptoms that Hough described, along with the hypothesised aetiology and mechanisms involved, have stood the test of time and many investigators have since proved his contentions to be true.

DOMS encapsulates several symptoms that suggest that damage has occurred in the exercised muscles. Muscle Soreness, Muscle Stiffness, Limb Volume and plasma Creatine Kinase concentrations, are all factors that are affected during the stages of DOMS.

2.3.2 Assessment of Muscle Soreness

The most widely used method to assess muscle soreness is the use of a questionnaire (Talag 1973, Byrnes et al. 1985, Donnelly et al. 1988, Ebbeling & Clarkson 1990). Following exercise, subjects are requested to complete the questionnaire which normally has sections relating to different muscle groups. Subjects rate the soreness at different sites on each muscle on a graded scale. This usually involves gentle palpation of the selected sites and subsequent assessment of the pain. The pain rating scale can be limited, for example from 1 to 10 where 1 is the normal state and 10 signifies the greatest possible pain. Some questionnaires have written comments alongside the scale, for example, 1 (normal) , 3 (uncomfortable), 7 (sore) etc., to help subjects assess their soreness. Some scales are unlimited and subjects indicate the soreness at some point along the continuum. As the pain increases, subjects indicate a point further along the continuum which they feel to be representative of the pain being experienced. Over a particular time period the soreness can be monitored both for individual muscle groups and overall soreness. The lower the overall score, the less pain the individual has experienced.

The advantages of using a questionnaire to assess soreness are that it is a convenient and simple method that does not require expensive or sophisticated equipment. However, there are disadvantages. The force with which each subject presses the skin is not measured and as the sites become sorer then subjects may palpate with decreasing force and this may affect the pain rating. Also, the force with which individuals palpate the skin could vary from subject to subject and this could influence the pain rating.

A second method of measuring soreness is by using a metal probe containing a strain gauge similar to that described by Edwards et al. (1981). The probe is normally about 2cm in diameter. By pressing the probe on the surface of the skin at various sites on the muscle, a reading can be obtained of the force value at which tenderness is first elicited. In this case the magnitude of the punctate soreness is inversely related to the degree of pain. Some investigators limit the amount of force that is applied to the muscle, believing that if pain is not elicited by a certain force then it does not fall into the category of severe muscle soreness (Buroker & Schwane 1989). This method of assessing soreness has been used by several investigators (Newham et al. 1983, Buroker & Schwane 1989, Jones et al. 1987). The measurement of pain was carried out with the assistance of the tester which standardised the procedure between subjects.

This second technique has some advantages over using a questionnaire. With this method of pain assessment subjects have no means for comparison in the assessment of soreness when taking part in studies using repeated measures. With a questionnaire it is possible to remember pain ratings from the previous day and a comparative rating can be given instead of an absolute

rating. This becomes a problem if subjects have a knowledge of the research area and have some idea of the pattern that the results should follow. By withholding from the subject the value of the force at which they feel tenderness is created, no feedback is given, no comparison can be made and a more controlled assessment can being made.

A third method, incorporating the techniques of the methods previously described, involves delivering a force of known value to sites on the muscle and subsequently determining the discomfort (Seaman & Ianuzzo 1988, Donnelly et al. 1988). Seaman & Ianuzzo (1988), in assessing muscle soreness in the quadriceps, elected nine sites on the muscle and applied 262kPa pressure to each of these sites. In response, subjects assessed the discomfort that this caused on a ten point scale which ranged from "slightly sore" to "extremely sore". The authors give no reasons for applying this amount of pressure. It is likely that any pressure beyond this would have created discomfort even in the "normal" state. Donnelly et al.(1988) delivered a force of 19.6 Newtons with a probe of 2cm diameter to various sites on the abdomen, shoulders and arms and assessed the discomfort that this caused on a scale ranging from "no pain" to "extreme pain".

The advantage of this third technique is a standardised experimental procedure. A constant force was applied to each site tested on every subject. The rating scale used to assess the soreness had written comments attached alongside the scale which gave subjects a further source of reference other than the numerical scale.

The assessment of muscle soreness is subjective and although it is possible to assess and monitor "within subject" variation muscle soreness, it is difficult to refer back to the "whole population" due to "between subject" variation in the perception of pain. Further research is therefore required to develop a more objective method to measure pain.

Despite the limitations in the techniques employed to assess pain, investigators have presented results indicating that during DOMS there is a gradual development of pain and a peak 24-48 hours after the exercise, with a subsequent return to the normal state up to seven days later (Talag 1973, Byrnes et al. 1985, Seaman & Ianuzzo 1988, Donnelly et al. 1988).

2.3.3. Site of Greatest Soreness

Investigators have been able to distinguish sites of greatest and least soreness. To fully understand the location of these sites it is necessary to outline the physiology of muscle and connective tissue.

Skeletal muscle consists of bundles of elongated cylindrical cells called muscle fibres 50 to 200 m in diameter and often several centimetres long. Bundles of muscle fibres each called a fasiculus are surrounded by a connective tissue covering the endomysium. A muscle contains a number of fasciculi encased in a thick outer layer of connective tissue, the perimysium. The connective tissue is much more resistant to stretching than the muscle fibres and as a result the amount of movement in a muscle is limited by its connective tissue. At both ends of the muscle the connective tissue melds into a tendon which attaches to the bony skeleton. There are several kinds of arrangements of muscle fibres in relation to the tendons. In some muscles the muscle fibres run the whole length of the muscle between the tendons, which form at the opposite ends (fusiform). In most muscles (pennate) one of the tendons penetrates through the centre of the muscle; muscle fibres run at an angle to the axis of the whole muscle from the central tendon to the perimysium. If the muscle fibres slant in towards the centre to converge on a single tendon, they are called unipennate. If the fibres converge on both tendons they are called bipennate. Compound muscles with several bellies of converging fibres are said to be multipennate. Because of the angular direction of the muscle fibres, pennate muscles have a mechanical advantage over fusiform muscles, generating more tension per unit of whole muscle weight, although the maximum tension per unit of muscle fibre cross section is the same. However, the fraction of rest length that pennate muscles can shorten is less than for fusiform muscles. Thus degree of shortening is traded for increased maximum tension in pennate muscles.

Greatest pain is often experienced around the joint in the myotendinous junctions of the exercised muscles (Asmussen 1956, Howell et al. 1985, Jones et al. 1987, Clarkson & Tremblay 1988). It seems likely that both muscle and connective tissue are damaged (Asmussen 1956, Abraham 1977) but since connective tissue is less elastic than muscle tissue it would seem to be more open to injury. Also, since the fibres proximal to the myotendinous junctions lie obliquely they are more vulnerable to the high tension that develops during eccentric exercise. (Friden et al. 1986). Asmussen (1956) stated that the possibility existed that the soreness was caused by swelling in or of the connective tissue, since intramuscular haematomas, or infusions of fluid will cause pain by distending the tissue. An increased formation of extracellular fluid in the connective tissues may secondarily influence the blood flow by blocking capillaries and thus augment the edema and the muscle soreness (Asmussen 1956).

Fleckenstein et al. (1989) assessed the magnetic resonance appearance of muscles in six sedentary subjects before and after calf plantar - flexion exercise. The subjects were not all examined at the same time intervals post exercise, therefore the results are described and not analysed statistically. Five of the subjects were examined between 2 and 5 days post exercise:

two subjects were examined at 24, 36, 48, 72 hours and at 13, 23 and 48 days post exercise. The investigators give no explanation why the examination times were different. The potential for using magnetic resonance imaging in research into muscle stiffness and soreness has been wasted with a haphazard approach to the experimental design. Although the results of the research are interesting, and provide a more reliable assessment of the extent of damage than a biopsy sample the investigators only state that there were "abnormalities" in the signal obtained post exercise. This suggests to the reader that there was some disruption of muscle or connective tissue (or both), but this is only referred to as an "abnormality".

Fleckenstein et al. (1989) in the same research paper report that they assessed the magnetic resonance appearance of the lower leg muscles of 8 elite male marathon runners at the end of a race. The investigators noted that signal abnormalities in marathoners were not seen in whole muscles (as observed in the sedentary group already described) but most frequently around the insertions and origins of the muscles. The inference is that there is an alteration in the structure of the tissue in and surrounding the musculotendinous junctions.

Though the research carried out by Fleckenstein et al. (1989) lacked structure it nevertheless gave a non-invasive insight into the potential damage that can be caused following repeated eccentric contractions in a sporting situation. The research also gives some support to the argument that the damage caused is localised in the regions of the musculotendinous junctions.

2.3.4 Pain Mechanisms

Investigators have shown that myelinated group III (or A delta) and unmyelinated group IV (or C) afferent fibres are responsible for transmitting painful sensations in skeletal muscle. The unencapsulated free endings of these fibres are found throughout skeletal muscle but are particularly dense in the tendons i.e. connective tissue (Stacey 1969). The receptors of Group III and IV afferent fibres have been shown to be activated by a variety of noxious stimuli (Iggo 1961) and Armstrong (1984) has suggested that activation of Group IV fibres elicits a response similar to the dull aching pain that is associated with DOMS.

Muscle pain receptors are one of two different types. Chemonociceptors are responsive to chemical stimuli and Mechanononciceptors are responsive to mechanical stimuli. They are a heterogeneous group, in that some may respond to a single chemical substance while others will respond to a variety of chemical, mechanical and thermal stimuli (Mense & Schmidt 1977). It is hypothesised that a chemical is released from the damaged tissue and depolarises the nearby nerve endings, initiating action potentials in the afferent nerve fibres. The chemical has not be identified for certain but bradykinin, serotonin, histamine, potassium and hydrogen ions are likely possibilities (Mense 1982). All of these substances are present after severe exercise.

Armstrong (1984) suggests that the delay in the accumulation of these substances, indicative that there is cell necrosis, could explain the time lapse between the exercise and pain response observed in DOMS.

2.3.5 Treatment of Soreness

Investigators have attempted to find a means by which muscle soreness can be treated. Some investigators have recommended stretching or massage, and others have attempted to use anti-inflammatory drugs to treat the symptoms. These treatments have not met with much success.

Stretching

De Vries in 1961 investigated the effect of stretching on muscle soreness in the forearm flexors, and found that the muscles that were stretched showed a reduction in pain when compared to the muscles that were not stretched. The subjects were required to lock the joints around the sore muscles to ensure that the muscles were at their maximum length. This stretch was held for two minutes, and following a one minute rest the stretch was repeated for a further two minutes. DeVries (1966) confirmed this further when investigating the short term effects of stretching. The same same protocol was used for stretching. The scale used by DeVries to rate the pain only had three points on it. A rating of "1" described "soreness which was so slight as to be felt only on palpation". A rating of "2" described "moderate soreness in which muscle pain was experienced during movement", in this case of the elbow joint. A rating of "3 " described "soreness which limited the range of motion and interfered significantly with normal use". The scale used was not particularly sensitive to change, and this must be borne in mind when examining the results. DeVries gives no indication of the length of time for which there was pain relief as a result of the prescribed stretching, whether it was seconds, minutes or hours.

Abraham (1977), in investigating the short term effects of stretching on DOMS, found that soreness was relieved for only about one to two minutes. It has been suggested that stretching results in the movement of fluid out of the muscle experiencing discomfort that results in the relief of pain (DeVries 1966 Abraham 1977) with a build up of fluid returning soon thereafter. Some individuals have recommended static stretching of the muscles before and after exercise to obtain relief from the pain experienced in muscles following unaccustomed exercise (DeVries 1966, Abraham 1977, Fox et al. 1988).

Since DeVries' findings no other investigator has found that stretching relieves muscle soreness in the hours following exercise (Buroker & Schwane 1989, High 1989, McGlynn et al. 1979). McGlynn et al.(1979) found that there was no relief from the pain of DOMS when

subjects followed a regimen of static stretching at 6, 25, 30, 49 and 54 hours post exercise. The stretching protocol was identical to that used by De Vries (1966) as described above and was repeated four times. Similarly, Buroker & Schwane (1989) carried out a programme of static stretching on subjects suffering DOMS in the knee extensors following a step test. Subjects performed a set of 10 identical static stretches of the knee extensors. Each stretch was for 30 seconds duration and was followed with a 10 second rest period. The investigators observed neither a temporary relief of pain immediately after stretching, nor a general reduction in pain during the three day post exercise period.

Since the subjects tested by DeVries(1961, 1966) did not all experience exercise induced muscle soreness, but accidentally induced muscle pain, it is difficult to compare the studies of DeVries to studies that have used experimentally induced muscle soreness. It may have been that the subjects of DeVries (1961,1966) experienced only moderate pain and therefore the movement caused by the stretching was sufficient to cause a reduction in pain. The results of DeVries (1966) and Abraham (1977) could have been affected by subjects who had read that stretching was supposed to relieve soreness, and therefore displayed the Hawthorn effect. Because the measurement of pain was subjective in the studies by DeVries (1966) and Abraham(1977), the subjects had complete control of the results of these investigations. This is necessarily the case with any study involving stretching to relieve pain from DOMS. Slight changes in pain may pass undetected, and although not "significant " changes in terms of size, they may be "statistically significant" changes, and therefore of some importance.

The contradictory evidence of the effectiveness of static stretching in relieving DOMS could be due to several factors. No quantification of the extent of the stretching procedures was made in any of the published studies. The length of time for which subjects performed each individual stretch, the total length of time that subjects stretched, and the method for inducing soreness varied in each of the studies considered. Also, the extent of the stretch carried out would be dependent on the ability of the subject to withstand pain. From the research it does not seem concrete advice to recommend static stretching as a means of reducing DOMS.

Present day coaching theory advocates that there is a preparatory period before and after training and competition, the "warm up" and "cool down" respectively, in which stretching is performed to reduce the risk of injury, prepare the body for optimal performance and to reduce the risk of muscle stiffness and muscle soreness following exercise. Anecdotal evidence suggests that there is some wisdom in performing stretching exercises after exercise to reduce muscle stiffness. Stretching may only be of benefit to slightly damaged rather than grossly damaged muscles.

Anti-inflammatory drugs

As there is a delay between the end of exercise and the onset of pain, several investigators have suggested that an inflammatory-type response has occurred as a result of the exercise (Zweifach 1973, Armstrong et al. 1980 Armstrong 1984). Evidence of such a response has been found in mice with exercise induced muscle damage (Salminen 1985). Infiltration of macrophages into the damaged tissue was observed and peaked 24-72 hours after the exercise, and followed a similar time course as the increase in lyosomal activity (Salminen 1985). A similar response has been found in runners by Hikida et al.(1983), where the peak infiltration of macrophages showed a similar pattern to that of the study by Salminen(1985). In a study by Jones et al.(1986) where muscle soreness was induced by eccentric exercise of the biceps, peak infiltration of macrophages occurred twelve days after the exercise.

Several investigators, as a result of the belief that the DOMS is an inflammatory condition, have attempted to reduce DOMS by using anti-inflammatory drugs. Wagner & Critz (1968) showed that by administering prednisolone (a synthetic hydrocortisone analogue) to dogs that had undergone running on a treadmill, the efflux of creatine kinase (CK), an indicator of muscle damage, was reduced. Headley et al.(1986) administered prednisolone to subjects who had undergone a stepping exercise, and found no reduction in soreness and no reduction in CK efflux. Salminen and Kulstrom (1987) showed that, by using the non-steroidal anti-inflammatory drug endomethacin, inflammation and histological changes in the muscles of mice were reduced. This treatment, however, has not been repeated on humans.

Bansil et al.(1985) administered aspirin to subjects and were successful in reducing soreness. They showed further that aspirin prevented changes in prostaglandin E and F_2 alpha levels in the control group.

Francis and Hoobler (1987) investigated the effect of aspirin on DOMS. The subjects in this study took "10 grains" of aspirin 4 times a day on the morning, approximately 4 hours prior to the exercise session and continued to do so throughout the 48 hours post exercise period. At twenty four hours following the exercise there was no statistically significant difference in pain between the 10 subjects in the aspirin group and the 10 subjects in the control group. However 48 hours post exercise the aspirin group experienced less soreness than the control group ($p<0.05$). The investigators stated that soreness was reduced by 25%. This is better explained by examining the mean soreness ratings of the experimental group (3.3 ± 0.2) and the control group (4.4 ± 0.4). These investigators gave no indication of the time interval between the doses of aspirin or of the exact dosage. Also, no indication was given of the training status of

the individuals taking part in the study. However, these investigators did show that there was a reduction in pain when subjects took aspirin.

In conclusion, it is apparent that some anti-inflammatory drugs have the effect of reducing the damage caused as a result of eccentric exercise in animals but not in humans, and that aspirin will reduce the discomfort experienced following eccentric exercise.

Massage

Sports massage is recommended by many coaches and practitioners as an aid to rapid recovery from strenuous exercise (Kuprian 1981, Buryovykv et al.1989, Cinque 1989), although the extent of research to support these recommendations is limited. Several studies investigating the effect of massage on DOMS have found that there is no long term relief from the discomfort of DOMS (Molea et al. 1987, Drews et al. 1990, Wenos et al. 1990). The massage was carried out immediately after exercise or 24 and 48 hours after the exercise in these studies. Massage in the first few hours following exercise has been recommended by Soviet physiotherapists as they believe that the timing of the massage is the most important factor in reducing the effects of eccentric exercise (Paikov 1985, Buryovykv et al.1989). No scientific reason is given why the massage should be carried out at this time, but the authors suggest that massage may interrupt a stage in the damage repair process.

In the first few hours following the exercise that has created damage white blood cells (neutrophils) arrive at the site of injury. The pattern is such that there is an increase in the number of neutrophils in circulation before an accumulation of neutrophils takes place at the site of injury. The subsequent accumulation of macrophages and inflammatory response is dependent on this initial build up of white blood cells. If DOMS is caused as a result of this inflammatory response it is thought that by carrying out massage at the time when the neutrophils would normally be accumulating then the inflammation and subsequent degradation of muscle tissue would be avoided. Further research is required to assess whether this is a possibility.

2.4 CHANGES IN LIMB AND MUSCLE VOLUME

There are contrasting views concerning the role that increased limb volume plays in DOMS. A sustained increase in limb volume in the days following the damaging exercise has been observed by some investigators (Talag 1973, Howell et al. 1985, Bobbert et al. 1986, Clarkson & Tremblay 1988) but not by others (Buroker & Schwane 1989). Talag(1973) noted a large increase in limb volume following eccentric exercise which only returned to the

pre-exercise level 24 hours after the end of exercise, however there was a nonsignificant relationship between muscle soreness and limb volume. Bobbert et al.(1986) monitored the volume of the lower leg using a plethysmograph after repeated lifting and lowering of the body (repeated plantar- and dorsiflexion of the ankle of one leg) and noted an increase in limb volume immediately after exercise. The volume of the exercised leg was significantly different ($p<0.05$) from the control leg up to 72 hours post-exercise. These investigators suggested that the increased limb volume was due to a rise in edema and that the edema was responsible for the increased soreness that was also noted post exercise.

Friden (1983, 1986) has suggested that the build up of fluid may be caused by an increase in osmotic pressure as a result of the formation of degraded protein compounds and the release of protein bound ions following the fibre disruption that has occurred as a result of the eccentric exercise. It is suggested that the increase in fluid pressure may activate free nerve endings and create the sensation of soreness.

Howell et al. 1985, in carrying out eccentric exercise of the biceps on seventeen subjects, noted that there was swelling of the arm at the level of the belly of the biceps and just above the humeral epicondyles 48 hours after the exercise bout. The investigation was well controlled and involved extensive EMG measurement before and during the stages of DOMS, as the study was primarily concerned with the relationship between EMG and the restriction of motion during DOMS. These investigators suggested that the swelling caused the restriction in motion that was apparent following the exercise and believed that edema within the perimuscular connective tissue may alter the elastic behaviour of the muscles and cause restriction of motion. Jones et al. 1987 also noted when investigating post exercise muscle stiffness and soreness that a decrease in the resting angle of elbow flexion was apparent following exercise, and that passive movements increased the angle, possibly by squeezing fluid out of the muscle. This may also partly explain why massage has the effect of reducing pain since the massage technique would also squeeze fluid out of the affected area. Similarly, anecdotal evidence suggests that when sportsmen exercise while they are suffering from DOMS there appears to be a temporary relief from pain while they are being active. However, the pain returns following the exercise. Jones et al. (1987) reported that the reduced stiffness following movement was temporary, and that soon after the cessation of movement the stiffness had returned to its previous level.

This is also supported by Lakie & Robson(1988) who investigated thixotropic changes in the stiffness of the relaxed finger muscles. One of the techniques used to measure stiffness used an electrical motor to deliver a pulse to the finger supported by a thin metal cradle connected to the motor, with subsequent analysis of the frequency spectrum of the transient oscillation

which resulted. They found that subsequent movement was always seen to decrease the stiffness and never produced increased stiffness.

2.5 CREATINE KINASE

By measuring the concentration of muscle enzymes in the blood it is possible to obtain further evidence that muscle has been damaged by repetitive strenuous exercise. The exertion involved in muscular exercise is thought to cause damage to muscle cells such that the cell membrane becomes "leaky" and allows muscle proteins to pass into the extracellular space. The greater the concentration of these enzymes in the bloodstream then the greater the damage that has been inflicted (Armstrong 1986).

Myoglobin has been measured frequently as it is a haem-containing protein involved in oxygen transfer and storage within muscle fibres; its increased concentration in the bloodstream is further indication that damage has occurred. Some of the myoglobin is secreted in the urine (Hansen et al. 1982), and its appearance here is one of the first clinical indications that dissolution of muscle, or "rhabdomyolysis", is taking place. Exertional rhabdomyolysis occurs when subjects undergo intense prolonged exercise (Armstrong 1986). Plasma Lactate Dehydrogenase (LDH) and 3-methylhistidine have also been measured as indicators of muscle damage (Dohm et al. 1982, Kanter et al. 1986). Creatine kinase (CK) has been measured most often as this enzyme is found exclusively in muscle tissue (Apple et al. 1983).

In normal subjects the resting plasma CK level is around 100 IU/L (Jones et al. 1986). Following strenuous exercise the level of CK can be as high as 40,000 IU/L (Newham et al. 1983). It should be noted at this point that when the level of CK is measured in the blood, the concentration is representative of the release and subsequent clearance of the enzyme and therefore any comparison of peak changes should be carried out with caution.

The levels of muscle proteins in the blood vary according to the volume, intensity and type of activity that has been carried out and the current level of training (Armstrong 1986). Marathon runners show particularly high levels of plasma CK following a race (Apple et al.1985), to the extent that plasma CK-MB (creatine kinase in its form from cardiac muscle) concentration after a marathon can be as high as it is when someone has suffered a myocardial infarction (Seigel et al. 1981). Eccentric exercise has been shown to result in greater levels of plasma CK (Abraham 1977, Davies & White 1981, Friden et al. 1983), particularly when the intense exercise is isolated on a particular muscle group (Jones et al. 1987, Clarkson & Tremblay 1988). Downhill running only increases CK by 5000 IU/L (Schwane et al.1987, Donnelly et

al. 1988, Hamill et al.1989), whereas prolonged maximal exercise of the biceps (70 maximal contractions) can induce an increase in CK by 20000 IU/L (Clarkson & Tremblay 1988). After twenty minutes of stepping, Newham et al.(1983) reported a rise in CK of 3000-10000 IU/L in subjects after 2-3 days. After stepping of 50 minutes to 2 hours a value of 34,500 IU/L was reported in one subject (Newham et al. 1983).

After exercise which causes only slight elevation of the level of CK (short duration or non weight bearing, for example swimming or rowing), the increase is thought to be caused by increased membrane permeability rather than membrane damage (Haramblie 1973, Noakes 1987). Armstrong 1986 has suggested that ischaemic and hypoxic conditions leading to a reduction in energy supply may change membrane permeability. Increased intracellular calcium levels during hypoxic conditions may cause disruption to muscle (Jones et al.1984), as can the depletion of high energy phosphates (Hunter & Critz, 1971).

When intense eccentric exercise of a specific muscle group is carried out the delay can be up to 48 hours with a peak several days later (Newham et al. 1983, Clarkson & Tremblay 1988). In less intense exercise, for example downhill running the delay is shorter with a release 4-6 hours after the exercise and a peak 18-24 hours post exercise (Byrnes et al. 1985). Jones et al. (1986) suggest that the reason for the delay may be that in the early stages of the eccentric exercise slight damage is caused to the muscle which in turn triggers an immunological response. The damaged tissue then undergoes degradation and repair. During this process the integrity of the muscle cells is lost allowing the CK to leak out into the bloodstream. It has been suggested by Clarkson & Tremblay (1988) that the delayed presence of elevated levels of CK may represent the final stage of the exercise damage process where a loss in sarcolemmal integrity occurs. The model these investigators present proposes that the damage caused by the exercise leads to a build up of calcium which in turn causes the production of bradykinin and histamine which are noxious stimuli and cause muscle soreness. The calcium is also thought to cause a restriction in motion and to inhibit the functioning capacity of the mitochondria and the sarcoplasmic reticulum. The activation of sarcoplasmic proteases then lead to loss of sarcolemmal integrity.

To summarise, mechanical damage seems the most likely candidate for the rise in serum enzyme levels following prolonged weight bearing exercise. The smaller changes in serum enzyme levels are likely to be as a result of changes in membrane permeability, due to muscle glycogen depletion, accumulation of intracellular acyl-CoA, or of lipid peroxidation damage to cell membranes. The larger changes are probably due to initial damage caused by the exercise with subsequent breakdown of this tissue as the repair process begins.

Even although the measurement of levels of CK can be used to determine muscle necrosis, the main drawback of this technique is that the measurement of CK obtained is for the level of CK in the bloodstream. No direct evidence is given about the site from which the CK has appeared (Noakes 1987, Armstrong 1986).

2.6 ADAPTATION

Substantive evidence exists to suggest that following an exercise bout that has induced DOMS, certain adaptations are seen to take place that have the effect of protecting the exercised muscles from future insult (Clarkson & Tremblay 1988, Seaman & Ianuzzo 1988, Newham et al. 1987b, Byrnes et al.1985). The adaptation is such that muscle soreness and the high CK response noted following the first exercise bout is greatly reduced and in some cases eliminated. There is evidence to suggest that the adaptation is confined to the muscle groups that experienced the DOMS (Clarkson et al. 1987) and that the adaptation and protective effect will last for a limited time(Jones & Newham 1985, Byrnes et al. 1985). Evidence also exists to suggest that a gradual increase in the volume of exercise is the best measure to avoid DOMS (Clarkson & Tremblay 1988).

2.6.1 Training Effect

Regular training has been shown to reduce muscle soreness, morphological changes, the CK response and decrements in performance that are observed following a bout of unaccustomed exercise (Hunter & Critz 1971, Ross et al. 1983, Byrnes et al. 1985, Newham et al. 1987, Clarkson et al. 1987b, Clarkson & Tremblay 1988, Ebbelling & Clarkson 1990). Ross et al.(1983) studied serum CK and its isoensyme CK-MB, AST, ALT, LDH and Mb levels in army recruits undergoing an intensive 24 day training course. The serum activities of all these enzymes were higher in the earlier rather than the later part of the training course, despite continued heavy training and without a period of rest. Newham et al.(1987b) examined the effects of three exercise bouts of maximal contractions on the biceps muscle each separated by two weeks. Muscle soreness was greater after the first bout and decreased thereafter. Although the first bout generated very high levels of CK (1500-11000 IU/l), the second and third bouts had no statistically significant effect on the level of plasma CK. The recovery of strength and of force-frequency characteristics was faster after the second and third bouts than after the first. (Strength was reduced to 50% after the initial exercise and increased to 80% of the pre-exercise level after two weeks)

Seaman & Ianuzzo(1988) engaged seven untrained men in 30 minutes of leg extension exercises to induce muscle soreness, using a resistance of 90% of the weight at which they

could do a maximum of ten repetitions. Following a week of rest these subjects began a training programme for five consecutive days carrying out the same amount of exercise each day as had been performed in the initial bout. The exercise was concluded two days following the five day training programme with a further bout of exercise using the same protocol as the initial but with greater resistance. The results showed that during the five training period the soreness and CK response were greatly reduced when compared to the responses following the initial exercise bout. Similarly, even when subjects performed a greater amount of work during the the third exercise phase of the study the soreness and CK responses were lower than those following the first bout. Therefore, the results of this study support the theory that regular exercise will help to reduce the extent of muscle soreness.

Friden et al.(1983) studied muscle soreness and muscle fibre morphology over an eight week period of eccentric training with increasing workloads. The exercise was carried out on a cycle ergometer. Following the initial exercise sessions, subjects reported marked soreness and muscle fibre disruption was also noted (Friden et al. 1983). During the training period that followed, despite the increasing workload, the soreness perceived was much less. An adaptation to the exercise had therefore taken place. Friden (1983) suggested that myofibrillar adaptation takes place to limit the extent of the damage caused by eccentric exercise. He proposed that there may be an increase in sarcomere length, there may be an increase in the number of longitudinal sarcomeres or there may be increased synthesis of Z-band proteins or intermediate filaments to strengthen myofibrils.

2.6.2 Time Period of Training Effect

Jones & Newham(1985-76P) reported a reduction in pain following training and after three weeks the pain disappeared completely as did the CK response. They report further that the pain and high CK response were restored when there was a twelve week interval between the end of training and a further bout of exercise. Byrnes et al.(1985) examined perceived soreness ratings, serum CK and myoglobin (Mb) levels following repeated bouts of downhill running. The protocol was such that each of the three experimental groups repeated a 30 minute downhill run (-10° gradient) 3, 6 or 9 weeks after the original run. Measurements of experimental parameters were taken before exercise and 6, 18 and 42 hours after exercise. In the groups that completed their second run 3 and 6 weeks after the first run, soreness ratings were significantly less ($p<.001$) and smaller increases in CK and Mb were found. In group three, the group which completed the run 9 weeks later, there was no statistically significant difference in soreness ratings, CK or Mb levels. Byrnes et al.(1985) concluded that the performance of a single exercise bout had a prophylactic effect on the generation of muscle soreness and serum protein responses that lasted up to six weeks.

2.6.3 Local or Central Adaptation

Graves et al.(1984) attempted to discover whether the adaptation seen following DOMS was confined to the muscles that had been exercised or whether the adaptation also occurred in other muscles that had not undergone the initial bout of exercise. That is, they wished to determine whether the adaptation was of a local or central nature. In this study the subjects exercised the leg extensor muscles of one leg on one occasion and the muscles of the other leg on a subsequent occasion. Graves et al.(1984) found that the CK response following the second bout was lower than the response following the first bout even when different muscles were exercised on each occasion. These investigators concluded that the adaptation following DOMS was of a central nature and was as a result of faster clearance of CK on the second bout.

In questioning the result obtained by Graves et al. (1984) Clarkson et al.(1987b) assessed muscle soreness and CK activity after two bouts of eccentric exercise separated by seven days. The research protocol was such that on the first occasion both groups performed exercise with only one leg. On the second occasion one group exercised the same leg, the other group exercised the contralateral limb. Clarkson et al.(1987b) found that in the group that exercised the same limb on each occasion, a lower CK and soreness response was found after the second bout. With the group that exercised the contralateral limb on the second occasion no statistically significant difference was found in either the CK or soreness response that was observed following the first exercise bout. Clarkson et al.(1987b) concluded that the adaptation was of a local nature and questioned whether Graves et al. (1984) had taken sufficient action to ensure that the muscles that were supposed to be inactive remained so during the first exercise bout.

Clarkson et al. (1987b) suggest that in the study by Graves et al. (1984) while performing the leg extension exercise with one leg, subjects may have been contracting the leg muscles of the opposite leg, that were supposed to be "inactive" in order to maintain postural stability. Clarkson et al. 1987 believed that the contribution from the supposedly "inactive" muscles during the first bout of exercise was sufficient to ensure that an adaptation took place when these muscles were exercised subsequently as part of the study protocol. Clarkson was a member of the group of researchers that carried out the study by Graves et al.(1984) and had therefore some insight into the experimental procedures. Believing that there was some weaknesses in the procedures used to control the activity of the muscles that were supposed to be inactive Clarkson et al.(1987) used electromyography to ensure that no contribution was made from the muscles that were to remain inactive. On the strength of the quality of research carried out by Clarkson over many years it can be concluded that the adaptation observed

following subsequent bout of exercise is of a local nature.

2.6.4 Exercise as a Preventative Measure

The effect of carrying out a reduced amount of exercise in an attempt to reduce soreness and CK activity in a subsequent more strenuous bout of exercise was investigated by Clarkson & Tremblay (1988). In this study, one arm performed 70 maximal eccentric contractions of the biceps (70MAX) and the other performed 24 maximal contractions (24MAX). After 70MAX statistically significant differences were found in CK activity, muscle soreness, strength and muscle shortening, and the effects were such that recovery had not been reached after five days, the length of time post-exercise that subjects were monitored. The 24MAX condition showed only small changes in the variables being measured. Two weeks later the 24MAX arm performed 70 maximal contractions (70MAX2). After 70MAX2 changes in the experimental variables were significantly smaller than those that occurred after 70MAX, even though more work was performed during 70MAX2. There was no increase in CK activity after 70MAX2 and maximum strength returned one day after exercise. By carrying out a fraction of the work of a future intended heavier exercise bout, the subjects were able to initiate a response which protected them from muscle damage that was observed when the heavier exercise was performed before no prior insult (Clarkson & Tremblay 1988).

2.6.5 Adaptation Before Complete Recovery

Ebbeling and Clarkson (1990) further investigated adaptation in muscle using two groups of subjects. They examining the effects of performing a second eccentric exercise bout prior to recovery (Group 1) and after recovery (Group 2) from a first bout. The exercise was carried out on the biceps muscle. In both groups of subjects the first bout of 70 maximal contractions (70MAX1) produced statistically significant changes in muscle soreness, resting elbow angle, isometric strength (60% of the pre-exercise value) and serum CK. Group 1 repeated the exercise (70MAX2) three days after the first bout when these changes were still apparent ($p<0.01$) but the recovery/adaptation was not inhibited. Indeed muscle soreness disappeared and within three days of 70MAX2, the range of motion and initial strength was restored. Group 2 repeated the 70 maximal contractions fourteen days after the first exercise bout when the variables being measured had returned to baseline values. There was no change in the variables being measured when they were compared to the response that was noted following 70MAX1.

Following unaccustomed exercise the peak in serum CK does not occur until CK efflux from damaged tissue has ended (Clarkson et al.1987a). CK following unaccustomed exercise is released in its isoenzyme form CKMM1 to be transformed to CKMM2 in the bloodstream and later to CKMM3 (Clarkson et al. 1987a). The presence of MM1 indicates that CK has been

newly released. Donnelly et al.(1990) reported that this peak in MM1 occurred day 3 after exercise. By day 5 Donnelly et al.(1990) noted that total serum CK was elevated but that the MM1:MM3 ratio had returned to normal. Based on this evidence, Ebbeling & Clarkson (1990) suggest that the repair of the tissue and the strengthening of the membrane had begun by day 3 following the exercise, before the second exercise bout two days later (at which time strength was still very much reduced). Ebbeling & Clarkson (1990) conclude by saying that complete recovery of the muscle is not necessary before adaptation is apparent.

2.6.6 Mechanism for Adaptation

Various theories have been suggested by investigators to explain the adaptation process when the results of two subsequent bouts of exercise are compared, as discussed above. Armstrong et al. (1983) and Byrnes et al.(1985) suggested that a pool of fragile or stress susceptible fibres were severely damaged by the first exercise bout. Their theory was that following the first exercise bout these damaged fibres were removed by the damage and repair process. The release of CK and the production of noxious stimuli and the chain of events that followed leading to pain and tenderness was as a result of injury to these fragile or stress susceptible fibres being degraded. The noxious stimuli, perhaps products of the inflammatory response, were thought to excite free nerve endings in the muscle. In the second bout of exercise there would be fewer stress susceptible fibres and therefore this was thought to be the reason for the reduced CK and soreness response (Armstrong et al. 1983).

Jones & Newham (1985) suggested that when sub-maximal contractions were performed, changes in the recruitment pattern of muscle fibres could occur following training, that is, in the time period between subsequent exercise bouts, and could be one possible reason to explain the adaptation process. Alternatively, these investigators offer a second explanation for the adaptation and suggest that more motor units could be recruited in a second exercise bout thereby reducing the force per fibre, and hence the chance of injury. However, findings from subsequent research using maximal contractions contradicted these hypotheses (Newham et al. 1987). Newham et al. (1987) used maximal eccentric contractions in subsequent bouts of exercise to induce soreness in their study. When performing maximal contractions all motor units would be activated. There could be no change in the order of muscle fibre recruitment or in the number of muscle fibres that would be active in subsequent exercise bouts. In the study by Newham et al. 1987 the adaptation process took place. The results of the study by Newham et al. (1987) suggested that a change in the order or extent of muscle fibre recruitment did not offer an explanation for the adaptation.

Investigators have suggested that the pain and stiffness experienced following unaccustomed exercise are as a result of the shortening of connective tissue, and that a training effect initiates some adaptation in the tissue (Howell et al. 1985, Jones et al. 1987, Newham et al. 1987) Clarkson & Tremblay (1988) suggested that some strengthening of the muscle cell membrane and the surrounding connective tissue must take place following exercise that has resulted in DOMS. A reduction in the efflux of CK following a subsequent bout of exercise would suggest that there is less leakage from the cells and therefore that the cell membrane must be stronger. The natural response of the body would therefore seem to be one in which the site of injury is strengthened and protected to prevent it from further insult.

Newham et al. 1987 offered further evidence to suggest that the adaptation takes place in the connective tissue. Following their study in which subjects exercised on three occasions, each separated by two weeks, the muscles did not appear to be stronger or less fatiguable, however the soreness and large increase in CK noted following the first bout had disappeared following the subsequent bouts of exercise. These investigators suggest that the damage caused as a result of the first exercise bout may have acted as a stimulus for collagen synthesis and subsequent strengthening of connective tissue.

In the study carried out by Clarkson & Tremblay (1988) the exercise performed on the first bout was only one third of the exercise performed on the second bout, but was enough to prevent severe muscle damage when the second, more strenuous exercise bout was performed. The results of this study suggest that Armstrong 's (1983) hypothesis (that the adaptation is seen because fibres that were injured during the first bout of exercise become unavailable on the second bout) must be questioned. The stress caused by 24MAX in the study by Clarkson & Tremblay was sufficient to produce changes in variables indicative that muscle damage had occurred. Clarkson & Tremblay (1988) suggest that the stress caused by the first bout must have provided a stimulus for strengthening the membrane and/or surrounding connective tissue against further insult. In such a way, the authors suggest that a final stage in the damage process is prevented; either necrosis or loss of sarcolemmal integrity.

From the evidence presented it seems that the adaptation takes place both in the muscle tissue and in the surrounding connective tissue. The mechanism of the repeated bout effect is still unclear.

Many aspects of the adaptation process are of relevance to individuals taking part in sport. It is comforting for sporadic exercisers to know that following exercise that has resulted in DOMS the symptoms are not fatal, and that recovery will take place. Regular exercisers would be interested to know that if they chose an alternative form of exercise which used different

muscle groups from their previous form of exercise it would be necessary to build up the amount of exercise gradually, even although they perceived themselves to be "fit", since the adaptation is specific to individual groups and types of muscular contraction. This is equally true for competing sportsmen and sportswomen and coaches who set training programmes. The training programme should contain all the movements and exercise which are to be found in the sport. Also the the results from adaptation studies suggest that consideration should be given to the volume, intensity and duration of the training sessions so that the training is appropriate for the nature of the competition in the chosen sport, since DOMS can be debilitating and could affect the preparation for competition. The following section discusses the effects that DOMS can have on performance

2.7 DOMS AND PERFORMANCE

2.7.1 Strength

Several investigators have shown that muscles experiencing exercise induced muscle soreness have a reduced capacity to produce force. (Hough 1902, Komi and Buskirk 1972, Talag 1973, Komi and Rusko 1974, Friden et al 1983, Newham et al. 1983, Clarkson and Tremblay 1988). Talag (1973) observed a decrease in muscular strength following eccentric exercise, and no decrease after concentric exercise. Kroon and Naeije (1988), in investigating the recovery of the maximum voluntary contraction force (MVC) following concentric and eccentric contraction of the biceps, found that up to 25 hours after the exercise, the MVC forces were "significantly different" from pre-exercise values. The period of the study did not extend beyond 25 hours, therefore a return to the pre-exercise level was not observed. Buroker and Schwane (1989) measured the effect of DOMS on maximal isometric torque developed at a knee joint angle of 90° using a Cybex II dynamometer at 24, 48 and 72 hours post-exercise. They noted a decrease in strength, at 24, 48 and 72 hours post-exercise, of the "eccentric" knee extensor muscles following the step test.

It has been suggested that because muscles were sore, there would have a psychological effect on individuals and therefore maximum effort would not be employed in carrying out a subsequent Maximum Voluntary Contraction (MVC). Using electrical stimulation, investigators have refuted these claims and further supported the view that muscles are fully recruited during MVCs, and that MVC force is reduced in muscles suffering from DOMS (Davies and White 1982, Newham et al. 1983, Newham et al. 1987).

In their study, Davies and White (1982) found that 1-2 hours of level running and uphill walking resulted in a reduction of 16ms (-12.6%) in the supra-maximal time to peak tension, a

reduction in the twitch of 11 Newtons (8.9%) and reduction in the tetanic tensions at 10 and 20 Hz of 163 (-17.5%) and 230 (-18.1%) respectively. The reduction in tetanic tensions was associated with a decrease in MVC. (Davies and White 1982).

2.7.2 Range of Movement

Howell at al (1985) in carrying out eccentric exercise of the biceps which induced DOMS, noted a reduction in the elbow resting angle, and a decrease in the range of both flexion and extension of the elbow. They suggested that the shortening was due to swelling in the muscle. Jones et al (1987) observed increased flexion of the elbow after eccentric exercise of the biceps which showed a change from 160°-170° to 130° in the most severely damaged subjects. Full extension of the elbow became painful on account of the muscle shortening caused by the heavy exercise. Jones et al (1987) suggested that the shortening of the muscle was due to shortening of connective tissue, since in examining EMG in the exercised arm, when increased flexion was observed, no increase in EMG was apparent.

Francis and Hoobler (1987) in measuring the effects of aspirin on reducing soreness following eccentric exercise, measured elbow extension and maximum elbow flexor force. Elbow extension was measured using a full scale circle plastic goniometer. Maximum elbow flexor force was measured using the Cybex machine. The investigators found a "significant decrease" in extension at both 24 and 48 hours; a reduction of 29.8° by 48 hours in the control group and a reduction of 14.2° in the aspirin group. Both groups showed a reduction in maximum elbow flexor force 24 and 48 hours after exercise, however "between" groups, differences were not statistically significant.

2.7.3 Reaction Time

Although several studies have shown that following exhaustive exercise there is a decrease in strength and a reduction in subjects' ability to react and move quickly (Klimovitch 1977, Stull & Kearney 1978), there has been limited investigation into the effects of DOMS on motor performance.

Dedrick & Clarkson (1990) investigated the effect of DOMS on motor performance in elderly and young individuals. Both groups performed motor tasks to measure the reaction time and movement time using an apparatus consisting of two micro-switches that activated timers. The subject sat with the forearm and elbow resting on a table at shoulder level. The investigator activated a light stimulus that started the first time. When the subject moved the hand off the micro-switch, the timer was stopped providing a measure of reaction time. The subject then moved the forearm in a horizontal arc across the table to hit the second switch located at a right angle to the first switch. When the second switch was hit, it stopped the second clock,

providing a measure of movement time. Although the older subjects demonstrated a slower strength recovery following the eccentric exercise that had induced damage,there was no change in the reaction time and movement time either following the exercise or between the two groups.

2.7.4 Technique

Given that there are changes in many facets of performance as discussed above it is proposed that there may be changes in individuals' technique during the stages of DOMS. On account of the pain experienced during DOMS, movement patterns could be altered in an attempt to avoid further discomfort. Modifications in movement patterns could result in a decrement in the standard of the technique, further injury and in extreme cases permanent damage.

Hamill et al. 1991 investigated the effect of muscle soreness on the mechanics of running in 10 female runners. Following a maximal oxygen uptake (VO_2max) test on the treadmill on the first day of the study the runners completed a downhill run on the treadmill two days later to induce muscle soreness. They exercised for 30 minutes on a downhill gradient of -15 degrees at a speed which corresponded to 73.5% of the maximum heart rate. On days 2,4 and 5 of the study the runners completed a 15 minute treadmill run, on the level, which corresponded to 80% of VO_2max. During the runs on days 2, 4, and 5 the subjects were filmed so that subsequent biomechanical analysis of the running stride could be carried out. Reflective markers were fixed to appropriate landmarks on the legs (the acromion process, iliac crest, greater trochanter, knee joint, lateral malleolus, the heel of the shoe and the head of the fifth metatarsal joint) and following the run the frames were digitised and the required kinematic data obtained. Five complete running strides were digitised for each subject for each run performed on days 2, 4 and 5. The angles of the thigh, calf and foot were determined.

The downhill run was successful in causing muscle soreness (assessed by questionnaire on each day) and in elevating the level of CK. Muscle soreness affected the support phase of the running action at the ankle, knee and hip, with reductions post-exercise in the angles of knee and hip flexion. This in effect meant that the running style was being altered to avoid discomfort when the foot struck the ground. Hamill et al. 1991 state that although muscle soreness was induced as a result of the downhill run the response was lower than they had expected. They suggest that in future studies a more vigorous form of exercise should be used to create greater damage than was caused by the downhill run.

The results of the study by Hamill et al. 1991 suggest that sporting technique may be altered as a result of the damage and discomfort caused by eccentric exercise.

2.8 MUSCLE STIFFNESS

Accompanying the muscle soreness described above is a perception of stiffness. The exercised muscles often feel tight and there is discomfort when an attempt is made to change their resting length. This resistance to stretch in the resting muscle is taken to be the stiffness of the muscle.

It is still debatable whether the feeling of increased stiffness during DOMS is matched by a real increase in stiffness. In research into rheumatoid arthritis(RA), Helliwell et al.(1988) measured the stiffness of the metacarpalphalangeal joint in 135 patients with progressive degrees of severity of RA. Helliwell et al.(1988) similarly measured the stiffness of the same joint in 135 "normals". Contrary to the previously held belief that RA patients showed increased joint stiffness, Helliwell et al.(1988) showed conclusively that this was not the case. The group of "normal" subjects did show greater stiffness in the joints than the patients suffering from RA. This may also be the case with individuals suffering from DOMS: the inability to move the arm as efficiently may be due to the fact that the individual knows pain will be incurred when a particular movement is carried out. The feeling of stiffness may not be matched by a quantifiable change in muscle tone.

2.8.1 Contributions to Stiffness

Consideration has to be given to the contribution that muscle and joint tissues make to overall stiffness. The skin, muscle, tendons, ligaments and joint capsule all contribute to overall stiffness, however difficulty arises when trying to determine the exact contribution made by each of these components to stiffness (Johns & Wright 1962). Further consideration will now be given to the effect on stiffness of changes in inertia, elasticity, viscosity and thixotropy.

Inertial Forces

The inertia of a limb is not unalterable. Consider the arm, for example, the pivot of which occurs at the shoulder joint. When the hand position is altered, or if the arm is slightly flexed then the radius of gyration alters and the moment of inertia of each point varies with the square of the radius (the distance from all points on the arm to the centre of the shoulder joint). Equally, it follows that the inertia of different individuals' arms may be expected to vary widely. The distance that the limb extends from the central axis influences the inertia, with larger, longer limbs having greater inertia.

Elastic Forces

Muscles are composed of muscle fibres which consist of myofibrils. The myofibrils are composed of protein filaments. Electron micrographs of myofibrils reveal the structure of the interacting actin and myosin filaments, and the basic unit of the sarcomere. The structure of the

muscle is such that many filaments are parallel and many sarcomere elements are in series to make up a single contractile element. The active contractile elements are contained within another fibrous structure of connective tissue called fascia. This tissue encloses the muscles, separating them into layers and groups and ultimately connecting them to the tendon at either end. The mechanical characteristics of connective tissue are important in the overall biomechanics of the muscle. Some of the connective tissue is in series with the contractile element while some is in parallel. The effect of this connective tissue has been modelled as springs and viscous dampers.

The connective tissue that surrounds the contractile element is called the parallel elastic component and it has properties similar to an elastic band. When the muscle is at resting length or less the parallel elastic component is in a slack state with no tension. As the muscle lengthens the parallel elastic component is no longer loose so tension begins to build up slowly at first then more rapidly.

All connective tissue in series with the contractile component, including the tendon is called the series elastic element. During isometric contractions the muscle length is kept constant and therefore any stretching of the series elastic element can only occur if there is an equal shortening of the contractile element itself. This is called internal shortening. The amount of internal shortening from rest to maximum tension is only a few per cent of the resting length. Under isometric contractions the series elastic component does not have any influence on the force-length characteristics. However during dynamic situations the series elastic element does influence the time course of the muscle tension. Researchers have determined the stiffness of the series elastic component from length-tension curves obtained by using "quick release" (Bahler 1967, Jewell & Wilkie 1958) or "controlled release" (Goubel 1978, Bressler & Clinch 1974) methods, and results from such studies have shown that the series elastic component exhibits increased stiffness with force.

Viscous Forces

Treacle will flow less easily than water, because there is more friction between the molecules of treacle as they slide past one another. This property of fluids is termed "viscosity"; the greater the friction then the greater the viscosity. The internal characteristics of a human joint are such that viscosity exists as a result of the presence of synovial fluid within the joint capsule. The synovial fluid acts to lubricate the joints and allow free and easy movement as the surfaces of the bones entering the joint pass across each other. When this lubrication system fails movement becomes restricted.

There is also a level of viscous friction within the connective tissue. This viscous friction is

also referred to as "damping" and has the effect of cushioning the movement. This is thought to occur as a result of tendons slipping in their sheaths, or as a result of motion at fascial planes between muscles (Lakie et al. 1984). Lakie et al.(1984) state further that viscous damping is unlikely to occur in relaxed muscle. When resonance occurs at a joint, as occurs with Walsh's method for measuring stiffness (Walsh 1972, 1975a, 1980, Lakie et al. 1984),the inertial and elastic forces are in balance and it is the damping, or viscous friction, which then determines the extent of the range of movement of the joint. When a given peak torque is applied to a relaxed joint there is a corresponding amplitude of movement. If the same peak torque is applied on subsequent days, any changes in the amplitude of movement can be interpreted as being as a result of changes in the damping or viscous friction of the system. That is, if Lakie's hypothesis is correct (that the viscous friction is due to tendons slipping in their sheaths (Lakie et al. 1984)), these changes could be attributed to alterations in the relationship between the tendons and their sheaths.

Thixotropy

While investigating the stiffness of the tissues surrounding the wrist, Lakie et al. 1979a discovered that there was a progressive increase in stiffness following cessation of movement. This refuted the claims of previous researchers who believed that the resting state of the muscle remained unchanged until it was required to contract. Further, Lakie et al. 1979b noticed that the stiffness could be reduced by movement and believed that this phenomenon was thixotropic in nature. Further research confirmed that this was the case and proposed that during periods of inactivity bonds were formed between actin and myosin to maintain postural stability (Lakie et al. 1984, Lakie & Robson 1988). During movement these bonds are subsequently broken but will re-form again when the movement has ceased.

2.8.2 Muscle Stiffness Following Exercise

Although much has been written on muscle soreness there has been very little published research on muscle stiffness following exercise. Jones et al.(1987) and Lakie & Robson (1988) are the only investigators to examine the effects of exercise on muscle stiffness.

Jones et al.(1987) have carried out the most thorough investigation into the effects of eccentric exercise on muscle stiffness. In their study they examined muscle stiffness and soreness in the elbow flexors following eccentric exercise. To measure stiffness the upper arm rested on a table at shoulder height with the arm in a pronated position. Stiffness was measured by these investigators as the force required to fully extend the arm, since during DOMS there is an involuntary decrease in the elbow flexion angle. The investigators took a resting elbow angle of 180° to be the control value such that if there was no stiffness then the whole arm would rest on the table. Increased stiffness resulted in the wrist not being in contact with the table. The

greater the force required to extend the arm to bring the wrist in contact with the table, then the greater the stiffness.

Immediately after exercise increased involuntary flexion of the elbow was observed in the resting condition in six out of the seven subjects (6-20° difference from the pre-exercise value). Increased stiffness was also apparent immediately after exercise but at this time there were no painful sensations in the muscle. Jones et al.(1987) report that flexion (in the relaxed state) and stiffness peaked at 24-48 hours after the exercise in six out of seven subjects. Further, greatest pain coincided with greatest flexion (in the relaxed state) in all subjects.

Although the technique used by Jones et al. (1987) was successful in quantifying changes in stiffness its reliability must be questioned. In principle these investigators were measuring the resistance to stretch, and the measurement of stiffness was the force required to extend the arm until it rested entirely on the table. During the stages of DOMS in many cases the arm will not fully extend as the pain experienced in carrying out full extension becomes unbearable. Jones et al.(1987) state that in two subjects it was impossible to extend the arm because of the pain caused and stiffness was recorded as a force greater than 30N. Although it was possible to state that there was greater stiffness compared to the previous occasion that measurement was made, it would have been difficult to make discrete objective measurements in subjects where there was severe pain during extension. There were only seven subjects tested in the study and although it was shown that there was an increase in muscle stiffness following exercise, testing a higher number of subjects would have added strength to their case.

An important factor in this study was that, despite the increased flexion of the elbow joint apparent after the exercise, there was no increase in EMG activity in the affected muscles at rest. Even at an involuntary elbow angle of 130° two days after the exercise no electrical activity was observed. Voluntary flexion beyond this point led to sustained electrical activity. The investigators do not mention whether there was electrical activity when the arm was being forcibly extended. That is, if the subject was voluntarily resisting the stretch being imposed when pushing the wrist down onto the table. This would affect the reading obtained as the value for stiffness. However, the results of this study showed that the muscle shortening and increased stiffness were not due to electrical activity in the muscle. The investigators, on this evidence, proposed that the muscle shortening and increased stiffness were not due to damage to the contractile component of the muscle but were caused by shortening of the muscle connective tissue.

The only other study to investigate changes in muscle stiffness following exercise was carried out by Lakie & Robson (1988). In a study that primarily investigated thixotropic changes in

the stiffness of the relaxed finger muscles, Lakie & Robson also investigated the effect of fatiguing exercise on muscle stiffness. They used a motor to deliver rectangular torque impulses (taps) to the relaxed index finger which was supported in a light alloy crank. The taps were adjusted to produce a displacement of approximately 0.1 radian for a time period of 50ms. The transient oscillation which resulted was recorded on disk and subsequently analysed to reveal the resonant frequency. The investigators then exercised the index finger. The hand was placed palm down on a surface with the fingers projecting over the end of a table. The index finger was then forced into flexion while the subject resisted the action with maximum effort. This had the effect of exercising the extensor muscles of the index finger eccentrically. Each effort lasted 5 seconds and was carried out every 10 seconds until the subject could barely resist the force or re-extend the finger. The resonant frequency of the finger was then measured and immediately following the exercise the resonant frequency decreased suggesting that the stiffness had therefore decreased. A slight long-term stiffening began at 24-30 hours after exercise. The investigators state that since movement (exercise) had the effect of decreasing the stiffness, there is logic in advising sportsmen to warm up before exercise. They state further that the effectiveness of some physiotherapy treatments, to reduce muscle stiffness, can be explained through a greater understanding of thixotropy.

At rest, there are bonds which form between actin and myosin to give postural stability and give the muscle its stiffness in its relaxed state, Movement breaks these bonds, and makes the muscle less stiff. However, on cessation of movement the bonds will re-form, giving increased stiffness over time. This can be compared to the the effect that shaking has on tomato ketchup that will not pour form a bottle. The shaking breaks the bonds which hold the consistency together, to make it less rigid and to make pouring possible. When left to stand the ketchup will return to its original state.

The technique used by these investigators to measure the changes in muscle stiffness was developed by Walsh in the 1970s (Walsh 1972, 1975a, 1975b). The technique for measuring stiffness used an electrical motor to deliver torques to the supported relaxed wrist. The subject sat in an experimental chair and rested the arm on a support. The hand was coupled to a light handle which was driven by an adjustable crank attached to the output shaft of the motor. Oscillations of the printed motor therefore moved the wrist in flexion and extension. By recording the oscillation which resulted, Walsh et al.(1980) found that at a certain frequency, movement of the wrist reached a maximum. This maximum was the point at which the resonant frequency had been achieved, from which inferences could be made about the stiffness, since resonant frequency (RF) and stiffness (K) are related by the equation for a Torsional pendulum,

$$RF = \frac{1}{2\pi}\frac{K}{J} \quad (J = \text{Moment of Inertia})$$

By ensuring that the inertia of the limb remained constant then any changes in resonant frequency reflected changes in the stiffness as RF^2 K. The technique could be modified to assess the stiffness of tissues around various joints.

Lakie et al.(1984) found that the resonant frequency of the wrist increased with stiffness, and that the larger the amplitude of the oscillation imposed on the wrist (and hence greater movement) then the lower the value obtained for stiffness. For small torques the system was non linear, with the resonant frequency falling as the torque rose, becoming constant at about 0.22Nm. Further increases in torque were not seen to change the resonant frequencies, however with very small torques the resonant frequency was seen to rise showing that the joint had stiffened. Lakie et al.(1984) stated that the increased stiffening for small movements was not due to pre-stressing of the muscle or to reflex activity. They stated further that the wrist exhibited a thixotropic effect (as described above) with a "memory time" of 1-2 seconds. Active or passive movement of the wrist led to a reduction in its stiffness. Lakie and Robson (1988) suggest that this thixotropic effect is most likely to occur in the muscle. Evidence from animal studies has shown that thixotropy does occur in and around muscles. (Weigner 1987).

Walsh and Wright (1988) investigated the biodynamics of the human hip in an attempt to determine whether the changes in resonant frequency observed when different torques were supplied to the wrist were also observed in the hip joint. By suspending the leg via a cord attached to the ceiling the leg was allowed to swing freely in the horizontal plane. A printed motor fitted with a potentiometer to measure displacement was positioned over the hip joint and this drove the leg by means of a lever with a yoke which clasped the knee. In supplying various strengths of sinusoidal torques the investigators noted that the system was non-linear. For small torques the resonant frequency increased indicating increased stiffness. The use of a biasing force to move the resting position of the leg showed that the stiffening was due to the small movement and not to the usual resting position of the leg. The investigators believed the site of increased stiffness to be in the muscle and suggested that when the muscle rests, as it does following exercise, bonding of cross-bridges begins. The stiffening could be relieved by movement either active or passive suggesting a thixotropic effect.

Hill as early as 1952 suggested that the resultant stiffness during small movements was caused by the presence of bonds between actin and myosin in the relaxed muscle. Grillner(1972) has suggested that this short range stiffness is of value in allowing muscles to resist externally applied loads before the nervous system has had time to react. Active movements break these

bonds and hence decrease stiffness.

A further technique developed by Walsh (1975) was that of "positive velocity feedback". The advantage of this technique is that the measurement of the resonant frequency can take place over a set period of time, and an average taken. In the previous method one discrete measurement is made, and if the subject is not relaxed then this will affect the value obtained for the resonant frequency. Using positive velocity feedback a preparatory period can be used to ensure that the subject is relaxed.

The apparatus was arranged so that the motor was connected to a potentiometer and therefore provided a signal corresponding to angular position. A velocity signal was derived using an operational amplifier as a pseudo-differentiator. The highly amplified velocity signal was passed to the bi-directional power amplifier supplying the motor so that any movements were assisted by the system (motor). That is, if the relaxed limb was displaced initially by the tester in a particular direction, this change in velocity would be detected and would result in a single burst of torque (preset by the tester) being supplied in the same direction by the motor. This torque displaced the limb to a certain extent. Immediately after the limb came to rest, due to the elastic nature of skin, muscle and connective tissue, there was a slight rebound in the opposite direction. This change in direction of the velocity was again detected and the motor supplied a torque in the new (opposite) direction. The limb again came to rest once it had travelled its course and again there was a rebound. The whole process was repeated so that there was a continual oscillation. When the velocity and torque were in phase, as was the case, then resonance took place. In such circumstances the motor carries out positive work on the arm and the muscles carry out negative work on the motor. Once the oscillation had begun then it continued indefinitely.

2.8.3 Current Research

The research of this thesis examines whether there is an increase in muscle stiffness in the forearm flexors following eccentric exercise using "positive velocity feedback", one of the techniques developed by Walsh in the 1970s. Using this technique stiffness can be quantified in terms of the resonant frequency (the rate of oscillation) and in terms of the range of movement that a limb will travel through (the amplitude of movement) in response to a given torque. By measuring the resonant frequency any changes in elasticity can be monitored. By measuring the amplitude of movement any changes in viscous friction (or damping) can be monitored.

It is hypothesised that following eccentric exercise there will be an increase in muscle stiffness and muscle soreness that will peak 24 - 72 hours post exercise. It is further hypothesised that the increase in stiffness will be as a result of changes in the connective tissue. Such changes will alter the amplitude of movement of the oscillation that is imposed by the electrical motor.

CHAPTER 3

PILOT WORK

The main purpose of these preliminary investigations was to establish a reliable and suitable protocol for determining muscle stiffness and muscle soreness. The arm was chosen for measurement since its movement was easier to control between subsequent measurements than the movement of the legs. This was an important consideration as movement is thought to alter stiffness. In particular, the use of the non-dominant arm could be limited to a greater extent than the use of the legs during the period of any study, since contraction of the muscles in the leg are necessary for locomotion.

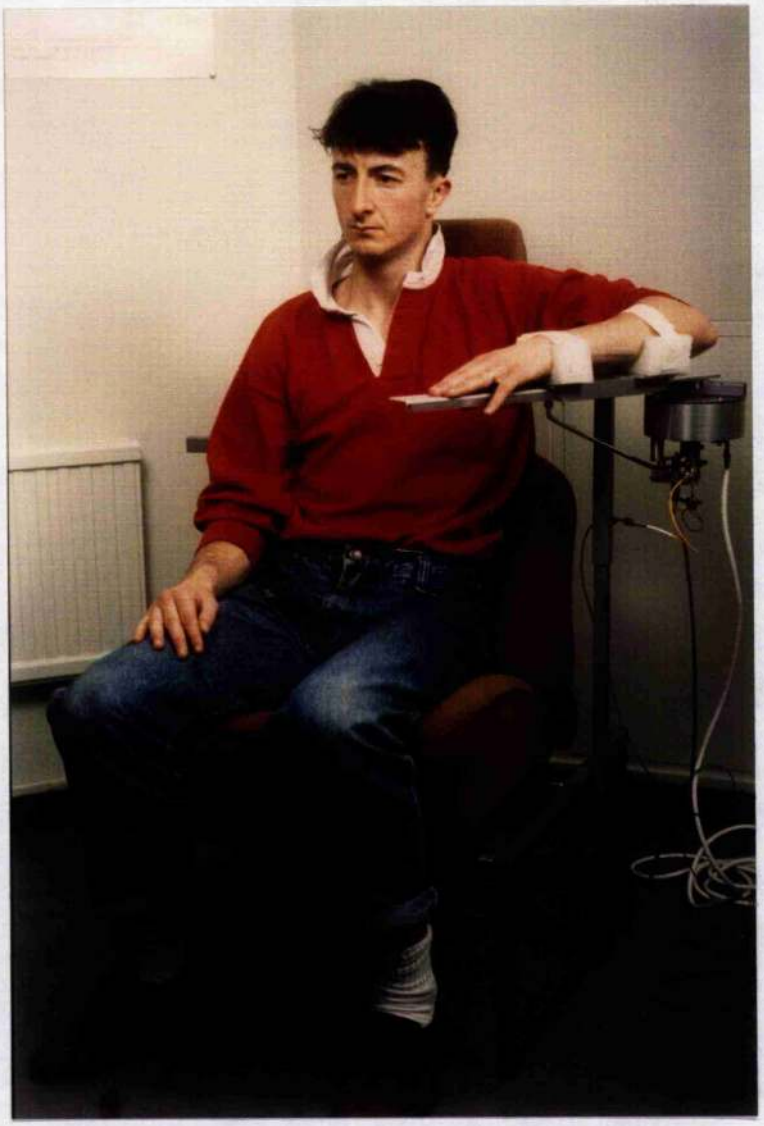
3.1 MEASUREMENT OF MUSCLE STIFFNESS

Muscle stiffness was measured using the "positive velocity feedback" technique developed by Walsh and co-workers in the 1970s and is as described in the previous chapter (Section 2.7.2).

3.1.1 Experimental Set Up

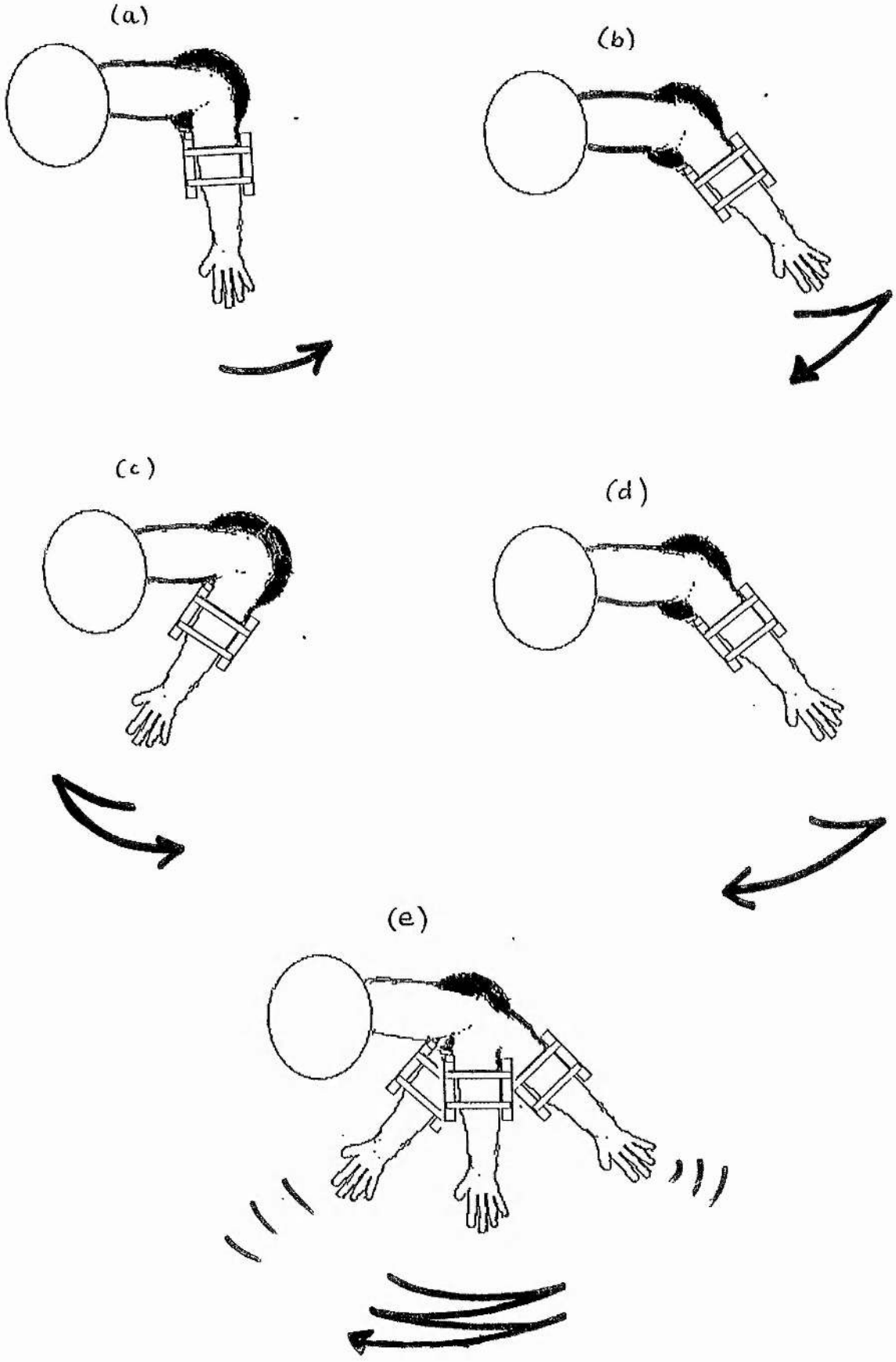
To measure muscle stiffness the subject was seated in a chair which had a support frame built around it. The chair used was a car seat which supported the head, back and legs in a comfortable position and its design restricted any lateral movement of the body. The support frame was designed to house a printed armature electrical motor (Printed motors Ltd. G9M4) and was identical on both sides of the chair, so that the motor could be transferred to either side of the chair to accommodate handedness. The design of the frame was such that the position of the motor could be altered vertically and in both horizontal planes. Scales were drawn on the vertical, horizontal and lateral aspects of the frame so that the motor could be brought to the same position for further measurements if necessary. A metal support arm was attached to the central axis of the motor, upon which the subject's arm rested in a cradle that slid onto the metal support. The arm was secured in the cradle using velcro straps. The distance that the subject's hand rested from the end of the support arm was noted. The elbow joint was concentric with the central axis of the electrical motor. The motor was raised such that the upper arm was in abduction forming a 90 degree angle with the body. The experimental set up is as shown in Figure 3.1.

Figure 3.1 A subject seated in the Experimental Chair



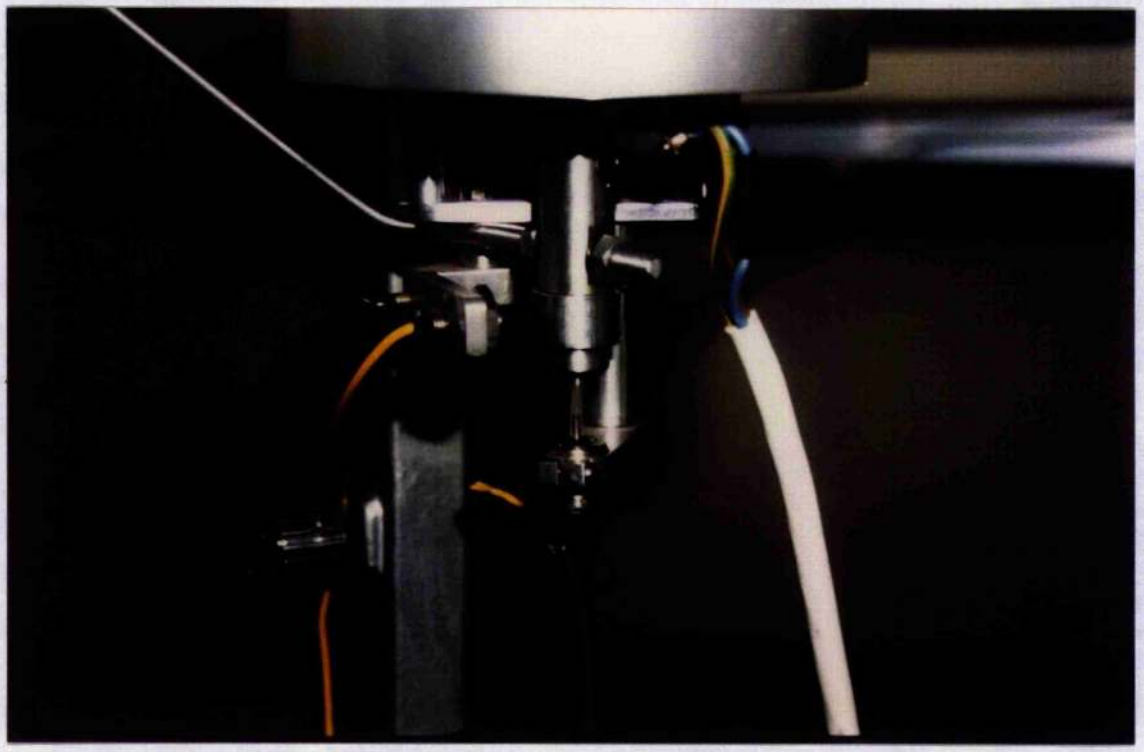
When a current was passed through the electrical motor, the metal support, and hence the subject's arm was moved passively. This movement in effect changed the length of the biceps muscle and the extent of the movement was related to the torque supplied by the electrical motor. The apparatus was arranged so that the motor was connected to a potentiometer and therefore provided a signal corresponding to angular position. A velocity signal was derived using an operational amplifier as a pseudo-differentiator. The highly amplified velocity signal was passed to the bi-directional power amplifier supplying the motor so as to give positive velocity feedback. In effect, any movements were assisted by the system (motor). In such circumstances the motor carried out positive work on the arm and the muscles carried out negative work on the motor. When the velocity and torque were in phase, as was the case here, then resonance took place. Once the oscillation had begun then it continued indefinitely. Figure 3.2 explains the process.

Figure 3.2 The continual oscillation imposed by "positive velocity feedback"



In 3.2a the arm is set in motion. The change in velocity is picked up by the potentiometer (Figure 3.3), and is amplified and passed to the motor which supplies torque in the same direction to assist the motion. The level of torque will determine the extent of the movement. When the arm eventually comes to rest there is a slight rebound in the opposite direction (Figure 3.2b). This change in direction of velocity is again detected and through the process already described the arm is driven back towards the body. When it finally comes to rest, again there is a rebound (Figure 3.2c) and the arm is driven away from the body once more. This process is repeated (Figure 3.2d) and so the oscillation continues (Figure 3.2e) until the motion is resisted or if the torque is removed.

Figure 3.3 The potentiometer attached to the electrical motor



The displacement of the arm was observed on a chart recorder and was used to determine the levels of peak torque for each individual. The trace on the chart recorder was also used to determine whether the subject was relaxing during the measurement of muscle stiffness. Complete relaxation revealed that the peaks of the oscillation occurred at the same level.

Data were recorded using a BBC Microcomputer (Acorn Computers Ltd.) and a Signal Average Programme(Bagust & Pelling 1984), and stored on floppy disc for subsequent evaluation. Using a Fast Fourier Transformation (Spectrum Analysis Programme, Structured Software 1985) it was possible to identify the Resonant Frequency (squared) (RF^2) and the Amplitude of Movement (AM) of the arm.

3.1.2 Experimental Objectives

In attempting to establish the protocol and for comparisons to be made it was necessary to establish the range of values for RF^2 in normal healthy subjects, and how, if at all, the RF^2 and AM changed on a daily basis. Since individuals differ in limb size it was thought that the values obtained for RF^2 would also be different. Before experimentation could begin, the full range of motion through which the elbow joint could be taken using the apparatus had to be established. This in turn was related to the peak torque that could be applied to move the arm to its greatest extent, which also had to be investigated. The least torque and least movement possible were investigated and whether resonance at smaller peak torques produced a higher value for RF^2. The reproducibility of the values established for muscle stiffness were also investigated and whether RF^2 and AM would be altered if there was either a voluntary contraction of the muscles of the arm or a change in the hand position.

It was important to establish whether subjects were relaxing during the measurement of muscle stiffness, and which procedure for using AM to determine muscle stiffness was more suitable and reliable. A suitable time interval for the capture of data was also investigated.

Statistical procedures were not employed during pilot investigations, although ultimately, the design of subsequent studies as a result of pilot work took into account the statistical procedures that would suit the experimental design.

3.1.3 Results and Discussion

The success of the technique used to measure muscle stiffness was dependent on the ability of the subject to relax. "Positive velocity feedback" was a strange sensation for subjects when it was first experienced as it induced an involuntarily oscillation of the forearm, and it was difficult for subjects to relax to allow the system to carry out the effort required to move the arm. Some subjects found it easier than others to relax and this was noticeable from the trace on the chart recorder. Figure 3.4 shows the trace of a subject who had difficulty relaxing whereas Figure 3.5 shows the trace of a subject who exhibited ease in relaxing The difference in relaxation ability can be observed from the level reached by the peaks of each oscillation. If the subject was not relaxing then there was either an attempt to resist the motion that the motor was imposing on the arm or an attempt to assist the motion.

Figure 3.4 The chart recorder trace of a subject who had difficulty relaxing

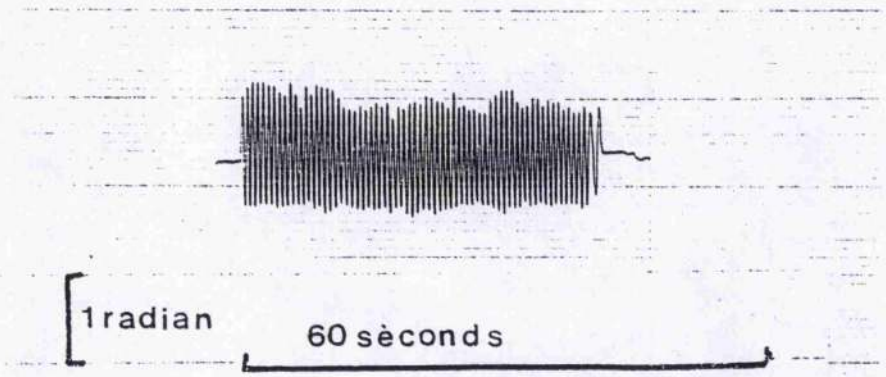
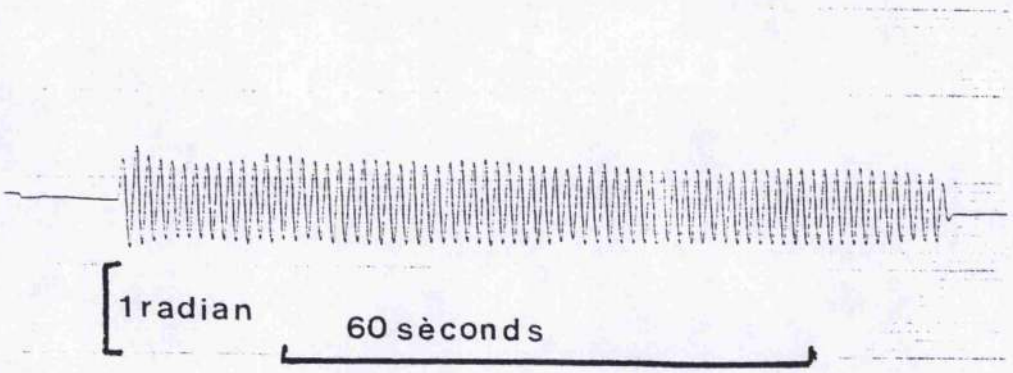


Figure 3.5 The chart recorder trace of a subject who found it easy to relax



On average it took three 10 to 15 minute sessions for subjects to become familiar with the technique being used to measure muscle stiffness and to relax while the measurement was taking place. Some subjects could not relax and were deemed unsuitable for the experimental procedures.

The range of movement through which the forearm could be moved was dictated by the structure of the apparatus. Too great a peak torque forced the arm into full extension which resulted in strain on the elbow joint. At the other extreme, the forearm flexed naturally to 30-35 degrees. Any torque which forced the arm to flex to this extent caused the arm to bounce back and resulted in added velocity to the arm in the opposite direction and forced the arm into hyperextension. The range through which the the elbow joint could be oscillated was

approximately 2 radians (120 degrees) without any discomfort to the subject and without the support arm colliding with the sides of the chair.

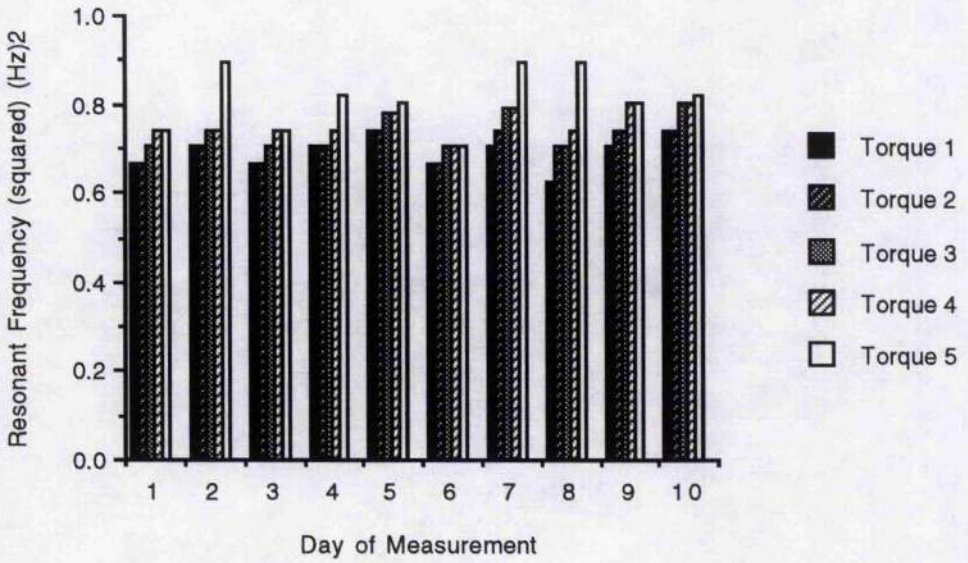
There were two means by which AM was used to monitor the change in stiffness. The first was to alter the level of peak torque to maintain the same amplitude of oscillation. The alternative method was to maintain the peak torque at a constant level and examine the changes in the amplitude of the oscillation. It was discovered that the first technique required a considerable amount of trial and error, and was more time consuming than the alternative technique. The process of matching the torque to the oscillation had to be carried out for every level of peak torque and the reproducibility of this method was questionable. However, using the second method, by maintaining the same level of peak torque, it was possible to carry out measurements a lot quicker. It was easier to monitor the level of peak torque than to try and judge whether an oscillation was the same as one that had been carried out on the previous day.

Once the oscillation had begun, and following familiarisation sessions, it took between 10 to 15 seconds for the subjects to relax and for the peaks of the oscillation to level out. The oscillation could then be recorded on computer disc. When the peaks had levelled out the time interval for data capture thereafter, no matter how small, gave an adequate record for the AM. However, a better average was obtained if the length of time for which the oscillation was recorded was increased. For practical reasons, in order to decrease the time commitment required from subjects, a minute was set as the time for data capture.

Larger arms (in length and composition) required larger peak torques to displace them. Males required larger peak torques than females for the same AM. To displace a light female arm through approximately 2 radians, 0.6 Nm of Torque was required. To displace a well muscled male arm through the equivalent range, 0.8 Nm of Torque was required. At lower values of peak torque it became more difficult to establish a consistent oscillation as subjects wanted to assist the motion. This however was not true of all subjects.

The values obtained for RF^2 at the highest peak torques ranged from 0.34 Hz to 0.89 Hz. RF^2 was a measure of the number of oscillations per second. Larger arms tended to have smaller values for RF^2 and smaller arms larger values for RF^2. As the level of peak torque was reduced there appeared to be an increase in the RF^2 for some subjects. An example of this can be seen for one subject in Figure 3.6.

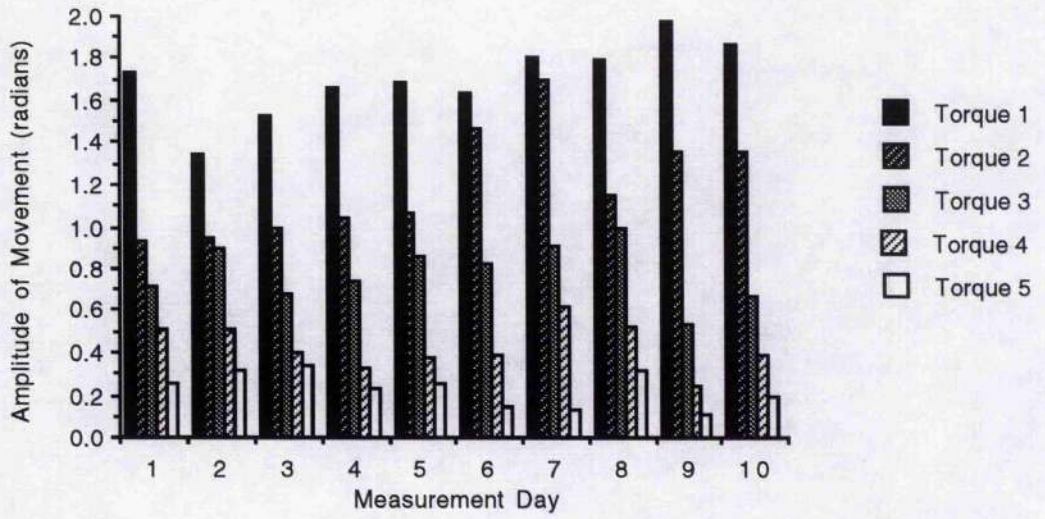
Figure 3.6 **RF^2 values at five discrete peak torques for one subject over ten consecutive days**



This effect has been noted by other investigators (Lakie & Robson 1988) and is thought to be "thixotropic" in nature. The effect can be explained using tomato ketchup as an example. It is easier to pour ketchup if the consistency is disrupted by shaking. This, in effect, breaks the bonds that have formed to make the substance more rigid when the ketchup has been stationary. These bonds will re-form if the ketchup is left to stand and it will be necessary to shake the ketchup if the pouring process is to be made easier on a subsequent occasion. In the present example, as the movement imposed by the level of peak torque is reduced then it is thought that more bonds are allowed to re-form making the system more rigid and hence stiffer. The exact nature of this thixotropic effect is a matter for further investigation.

Although statistical procedures were not employed to analyse these results there appeared to be slight variations in stiffness on a day to day basis. The measurements for ten consecutive days of a typical subject are shown in Figure 3.6 and Figure 3.7. Measurements were taken at five different levels of peak torque which produced displacements throughout the full range of movement. The time interval for data capture was one minute.

Figure 3.7 AM values at five discrete peak torques for one subject over ten consecutive days



Since there was a suggestion that there may be increased stiffness for smaller movements, and that at rest the arm was stiffer, it was thought that it would be necessary to standardise the state of the arm before any measurement took place. Some subjects therefore carried out a warming up procedure which involved a continual oscillation of the arm at the peak torque which produced the largest displacement. Other subjects had fifteen seconds of this oscillation before the measurement took place. The reproducibility of the results appeared to be more consistent when subjects warmed up for a period of approximately two minutes or more. It was thought important that in future there should be a warm up before any measurements of muscle stiffness were made. It was also thought important that in future the amount of physical activity of subjects should be controlled during the period that measurements were taking place, since activity appeared to alter the stiffness.

Since the comparisons that were to be made depended upon keeping the Moment of Inertia of the arm constant, it was necessary to ensure that the position of the arm was identical for each separate measurement. Any change of position of the cradle on the support arm, or the position of the hand, or the distance along the support arm that the subject's arm reached would change the Moment of Inertia and would make it impossible for inferences to be made about the stiffness. Accordingly, the support arm was marked at intervals of 0.5cm in indelible ink and labelled. The position of the cradle on the support arm, the hand position and the distance of

the hand from the end of the support arm could then be noted. Similarly, the support frame was also marked at intervals of 0.5cm to ensure that the position of the motor could be recorded for each subject.

Trial 4 in Figures 3.8a and 3.8b illustrates the effect on RF^2 and AM, respectively, when a subject created voluntary tension in the forearm by gripping a pencil lightly in the hand. Trial 6 in Figures 3.8a and 3.8b illustrates the effect on RF^2 and AM, respectively, when one subject clenched the fist as tightly as possible. This illustrates how tension in the hand can alter the readings and that it is necessary for subjects to relax during the testing procedure.

Figure 3.8a RF2 values during voluntary stiffening of the arm at one peak torque for one subject

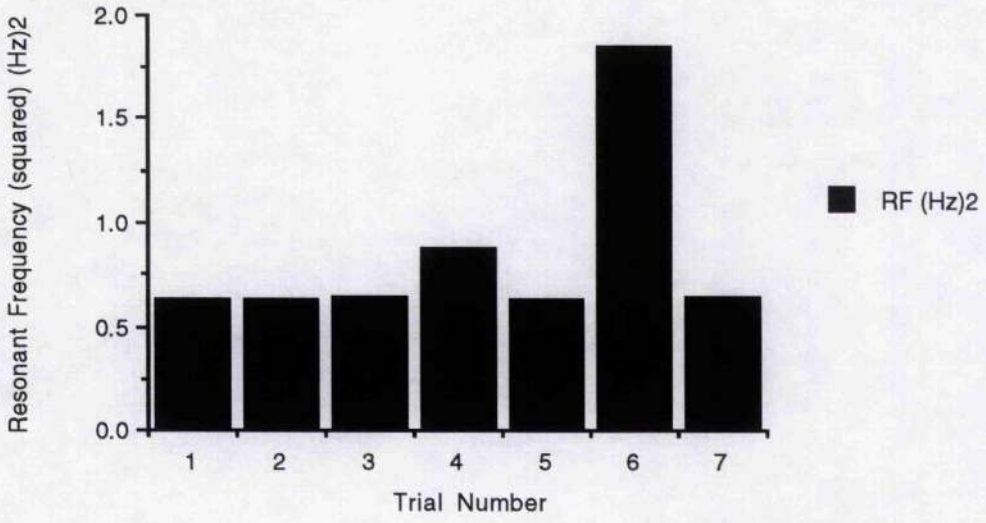
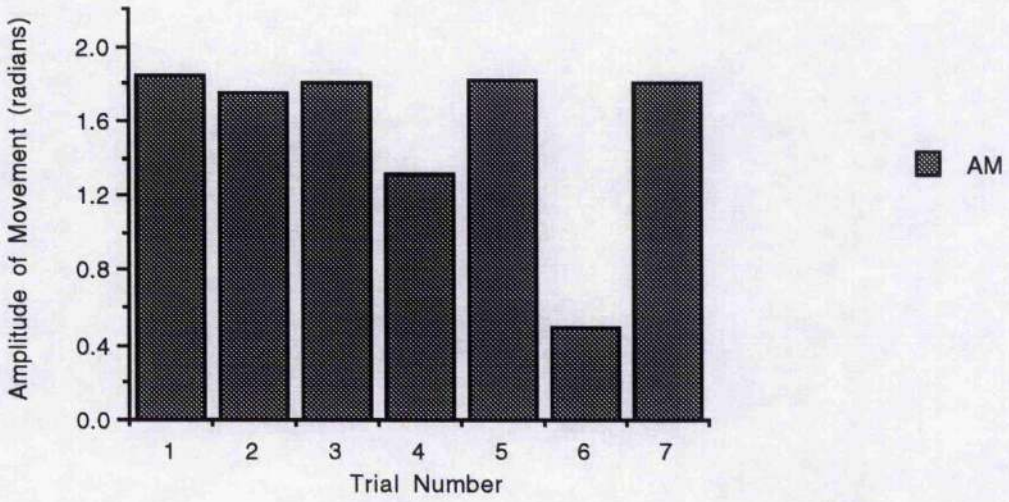


Figure 3.8b AM values during voluntary stiffnening of the arm at one peak torque



The effect of eccentric exercise on muscle stiffness was also investigated in preliminary work and the results for one subject can be seen in Figures 3.9 and 3.10. This response was common amongst all subjects. Figure 3.9 shows RF^2, Figure 3.10 shows AM. There appeared to be an increase in muscle stiffness, as evidenced by a reduction in AM. There appeared to be a slight increase in RF^2, although further investigation will reveal whether this is in fact the case. In some subjects, where small peak torques were used to assess muscle stiffness, subsequently it was found that the arm would not move during DOMS for the same torque. It is recommended that higher peak torques be used in subsequent research to eliminate this possibility.

The muscle stiffness of both arms was measured, as it was possible to locate the motor on either side of the experimental chair. This was carried out with several subjects and there appeared to be no differences. Since the equipment was sensitive and not suited to continual movement, and since calibration of the equipment was time consuming only one arm was tested. It was decided that subjects would act as their own control.

Figure 3.9 **RF2 values pre- and post-exercise at five distinct peak torques for one subject**

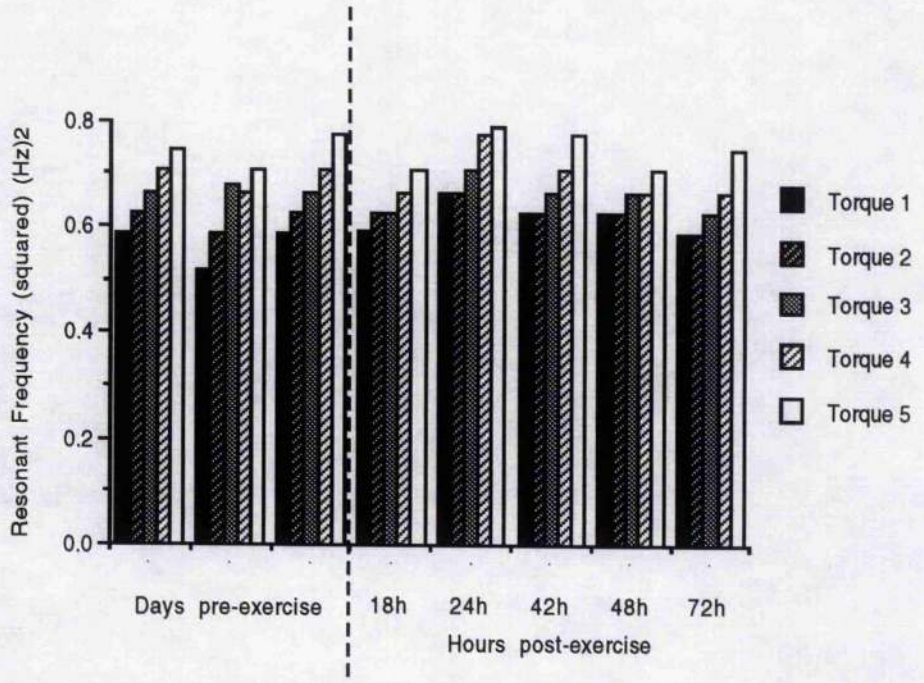
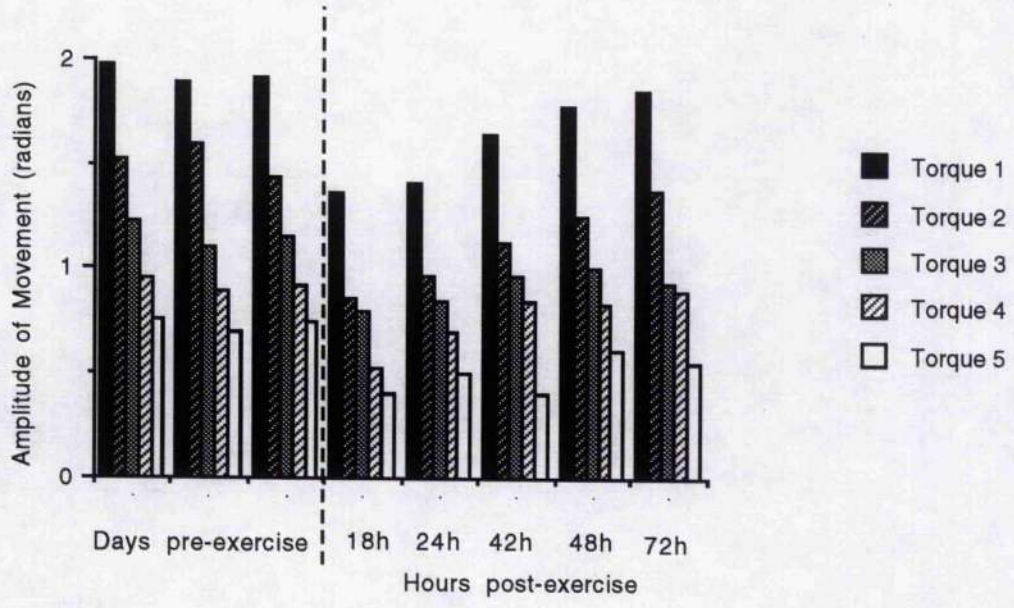


Figure 3.10 **AM values pre- and post-exercise at five distinct peak torques for one subject**



3.2 MEASUREMENT OF MUSCLE SORENESS

Eight healthy male subjects aged 23.7 + 3.7 years (mean + S.D.) volunteered to take part in this pilot study. All were physically active but none had taken part in a weight training programme in the previous three months.

Muscle soreness was measured at twelve sites on the arm, at three sites along the lengths of the biceps, brachioradialis, flexor carpi radialis and triceps. These muscles were chosen as they lie on the surface of the arm and are also the largest muscles involved in flexion and extension of the forearm. Each site was marked with indelible ink. Muscle soreness was assessed immediately before the exercise and at 24, 48, 72, 96, and 120 hours post exercise. Three methods for assessing muscle soreness were examined during these preliminary investigations.

3.2.1 Experimental Methods for Measuring Pain

Method 1

The first method used to measure pain involved the use of a questionnaire in which subjects assessed the soreness by gently palpating the arm at different sites and rated the soreness on a scale of 1 to 10, where 1 indicated a rating of no pain and 10 indicated a rating of very sore.

Method 2

A force of 25N was applied to each of the sites using a spring-loaded centre-punch on the end of which was attached a plastic disc of 2cm diameter. (Any force greater than this resulted in increased pain for subjects in the "normal" state). Subjects were then asked to rate the perceived pain on a scale ranging from 1 to 10, where 1 represented no pain and 10 represented a rating of "very sore".

Method 3

The final method for measuring soreness used a modified Newton Balance with a plastic disc of 2cm diameter attached on the end. Increasing force up to a maximum of 25 Newtons was applied to each of the twelve sites. Subjects were instructed to indicate the point at which the sensation created by the increasing force became "uncomfortable". A reading of the force being applied at this point was then noted.

Subjects were further asked to rate the soreness experienced during voluntary passive flexion and extension of the arm through the full range. This was carried out on the same scale of 1 (no pain) to 10 (very sore).

3.2.2 Exercise to Induce Muscle Soreness

In order to assess the methods that could be used to measure muscle soreness in the arm it was necessary to induce muscle soreness experimentally. Since Clarkson & co-workers had induced muscle soreness successfully using 70 maximal eccentric contractions, this protocol was adopted for the present investigation.

Each subject lowered a weight with the non-dominant arm, using a pulley system, from a position of full flexion to full extension seventy times. The arm was raised in front of the body and rested upon a shelf at shoulder level such that the arm was at ninety degrees to the body as shown in Figure 3.11. The maximum weight that the muscle could lower in a "controlled" manner (lowering to a count of five seconds) was determined prior to the exercise session. If the individual became unable to lower the weight in a controlled manner, some weight was removed so that the exercise could continue. This procedure was repeated until 70 contractions had been completed. The exercise rate was four contractions per minute - a five second contraction followed by a ten second rest period during which time the tester returned the weight to the starting position so that the subject being tested had the arm in a position of full flexion ready for the next contraction. The range of weights lowered was from 25 kilograms to 5 kilograms.

Figure 3.11 The method and equipment used to induce muscle stiffness and soreness

3.2.3 Results

The seventy maximal eccentric contractions induced muscle soreness successfully. All subjects experienced soreness, some to a greater extent than others. The results for Method 1, Method 2 and Method 3 are shown in Figures 3.12, 3.13 and 3.14 respectively. Only five of the twelve sites showed an increase in pain following the exercise bout. Those sites were the upper flexor carpi radialis, the upper brachioradialis and the three sites on the biceps. The site of greatest pain was in the lower biceps. The three methods used to assess muscle soreness suggested that there was an increase in muscle soreness which reached a peak 48 hours after the exercise. The pain decreased thereafter but was still above the pre-exercise rating 5 days post exercise. The pain ratings indicated that greatest pain was experienced around the joint.

Method 1 in which subjects lightly palpated the skin produced similar pain responses to those obtained for Method 2. Method 3 also provided a record of pain similar to Methods 1 and 2, however subjects indicated that they found it difficult to decide when the pain became "uncomfortable".

Some subjects indicated that there was pain experienced during voluntary flexion and extension of the forearm. Some subjects also stated that the arm felt weaker while they were suffering from DOMS.

Figure 3.12 **Mean Soreness Ratings at five sites on the arm for all subjects (n=8) pre- and post-exercise using Method 1**

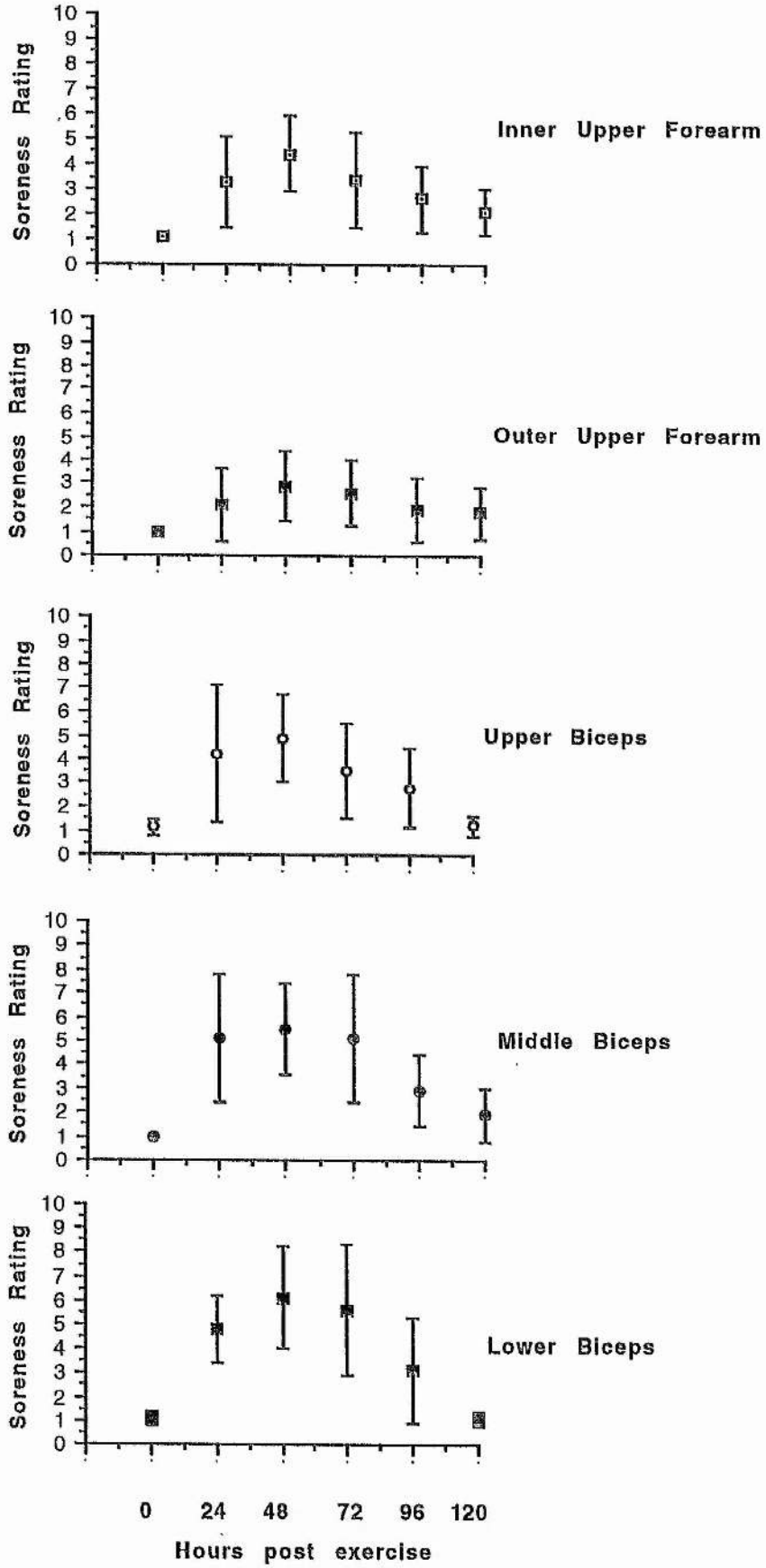


Figure 3.13 **Mean Soreness Ratings at five sites on the arm for all subjects (n=8) pre- and post-exercise using Method 2**

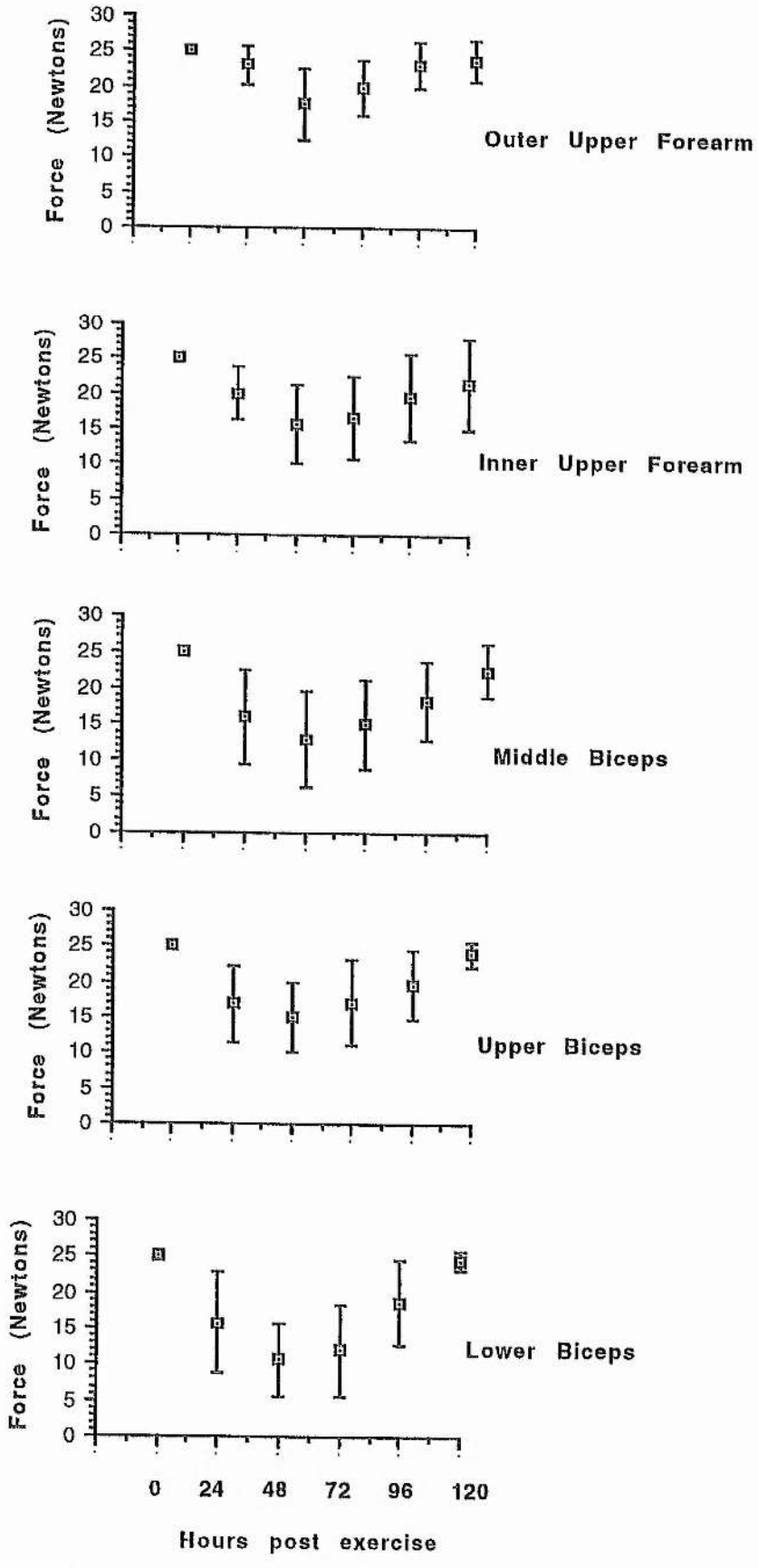
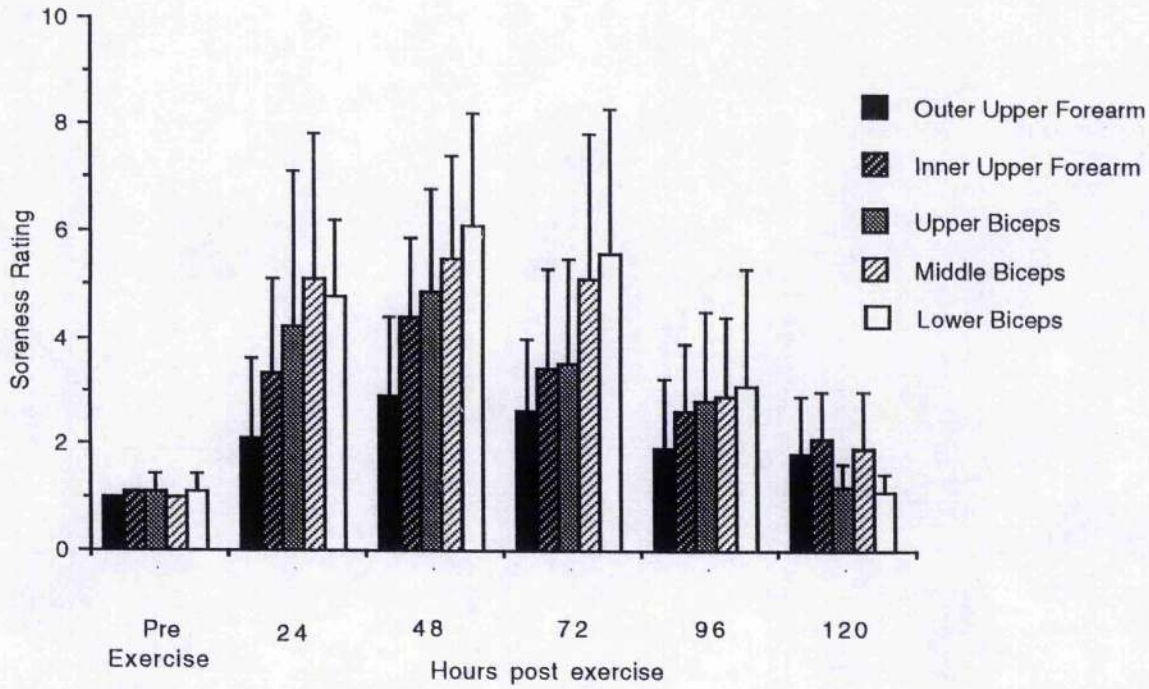


Figure 3.14 Mean Soreness Ratings at five sites on the arm for all subjects (n=8) pre- and post-exercise using Method 3



3.2.4 Discussion

Method 1 did not involve direct intervention by the investigator in the measurement of muscle soreness. Subjects palpated the sites that had been marked themselves and rated the pain accordingly. The force with which each subject pressed the skin was not measured. It was anticipated that as the sites became sorer then subjects would palpate with decreasing force and that this may affect the pain rating. Also, the force with which individuals palpated the skin could vary from subject to subject and again could influence the pain rating. Whether this was the case or not the changes in pain ratings observed using Method 1 were similar to those observed when Methods 2 and 3 were used.

When subjects were assessing pain using Method 1 there was not only palpation on the designated site, but also in the area surrounding the site. This appeared to be a natural response by subjects who wanted to locate the sites of greatest pain. Since subjects palpated in the area surrounding the site, greater pain could be experienced on an area that was not designated as a testing site. Psychologically, this increased pain would be the response that the subject would remember and subsequently record. If the same area was not palpated on subsequent days an inconsistent pain reading would be obtained. In effect, each day the sorest point could be in a different location.

The measurement of pain in Method 2 was carried out with the assistance of the tester. A constant force was applied to each site tested on every subject. This ensured that the pain response was confined to a particular site and that subjects were responding to the same stimulus. As with Method 1 the rating scale used to assess the soreness had written comments attached alongside the scale. Subjects reported that the written comments were a useful guide in assessing the soreness as they gave a further source of reference other than the numerical scale. Some subjects indicated that the scale of 1 to 10 (where 10 represented a rating of "very sore") did not offer the opportunity to rate the pain higher than 10 if on the first day this was the response and on subsequent days there was greater pain. These subjects thought that there should be a continuation of the scale beyond 10 to offer this opportunity.

Method 3 gave subjects no feedback or means for comparison in the assessment of soreness. The force at which there was an "uncomfortable" sensation as the tester pressed on the site was unknown to the subject therefore an unbiased assessment was being made. The results obtained using Method 3 revealed the same patterns in the build up and reduction of pain that were observed using the other two techniques. Subjects indicated that it was easier to assess the soreness using Methods 1 and 2 than using Method 3, as using Method 3 there was only one point of reference, that of the term "uncomfortable". This was perhaps a weakness in the technique. However, human nature is such that individuals wish to know how they are

performing, and since there was no feedback using Method 3 there may have been some insecurity created. Method 3 highlighted the questions that subjects raised concerning the variations that occurred in pain thresholds. A force that is "uncomfortable" for one individual can be "very sore" for another. For example, when increasing force was applied to sites on the arm in its normal state (as in Method 3) before any exercise took place, some subjects reported that at a force of 27 Newtons there was an "uncomfortable" sensation. Other subjects did not feel an uncomfortable sensation in the "normal" arm until a force of 32 Newtons was applied. A force of 25 Newtons was set for Methods 2 and 3 to take into account variations in individual pain thresholds.

3.2.5 Conclusions

There were advantages and disadvantages of the three methods used to assess pain in the present study. Method 1 relied heavily on the subject to carry out the assessment, in which there was ample scope for comparison and feedback, but there were inconsistencies in the assessment procedure. Method 2 involved the subject and the tester and offered greater consistency in the assessment procedure as well as offering the opportunity for comparison and feedback. Method 3 involved both subject and tester and was a consistent assessment procedure, but gave no feedback and little scope for comparison.

As a result of carrying out the procedures of the study and from the comments made by subjects Method 2 was selected as the most suitable means for assessing soreness, but with some modifications. The pain scale would be extended to give subjects the opportunity to indicate a rating greater then "very sore" if they wished to do so. Assessment would also be made of the soreness experienced when passively extending and flexing the arm. The location of the sites would continue to be determined prior to the commencement of the study, even although the sites chosen were not necessarily the exact points at which there was greatest pain. However, the results of the present study suggested that the area surrounding the elbow joint would show the greatest increase in pain, and therefore attention would be given to selecting the exact sites to be tested for soreness around this area.

3.3 OUTCOMES : RECOMMENDATIONS FOR FURTHER EXPERIMENTS

As a result of these pilot investigations the following procedures for creating and measuring muscle stiffness and muscle soreness have been set.

3.3.1 Muscle stiffness

Testing Procedure

Muscle stiffness was measured using the "positive velocity feedback" technique developed by Walsh and co-workers in the 1970s and is as described in Section 2.7.2. The technique was designed to create a continual oscillation at resonance.

Subjects visited the laboratory on several occasions to familiarise themselves with the technique being used to assess muscle stiffness. During these occasions subjects practised relaxing to allow the oscillation of the arm to take place. If subjects did not display the ability to relax after five familiarisation sessions they were asked to withdraw from the study. These occasions were also used to set the levels of peak torque that would be used to measure muscle stiffness. Five discrete peak torques were used for each subject and were applied from highest to lowest on every occasion. Displacement of the arm was observed on a chart recorder and was used to determine the levels of peak torque for each individual. The peak torques were selected to produce displacement of approximately 2.09, 1.75, 1.40, 1.05 and 0.52 radians. Displacement greater than 2.09 radians led to the support arm banging against the metal frame housing the electrical motor. Pilot experiments showed that if a displacement smaller than 0.52 radians was used, then following the exercise bout, the arm would often not move for the same corresponding peak torque. Once controlled measurements were obtained the level of the peak torques applied throughout the period of the study remained unchanged.

On arrival for testing subjects reported the exercise history of the past twenty four hours. As subjects were asked to refrain from exercise during the period of the study, this report was necessary to emphasise the need for abstention from exercise and to check that no exercise had been performed. The subject then sat in the experimental chair and the motor position was adjusted so that it was correct for that particular subject. The motor position had to be altered to take account of individual variation in size and arm length, so that the elbow joint was concentric with the central axis of the motor. The motor was raised such that the upper arm was in abduction forming a 90 degree angle with the body, and the elbow was flexed at 90°. The hand position on the support arm and the position of the cradle were noted to ensure that

they were in the same location as on previous occasions. The non-dominant arm was placed in the support and strapped into the cradle. (This arm was used for practical purposes as following eccentric exercise subjects would experience stiffness and pain in the exercised arm. To avoid unnecessary discomfort, it was thought that the non-dominant arm would be used less often in everyday tasks than the dominant arm.)

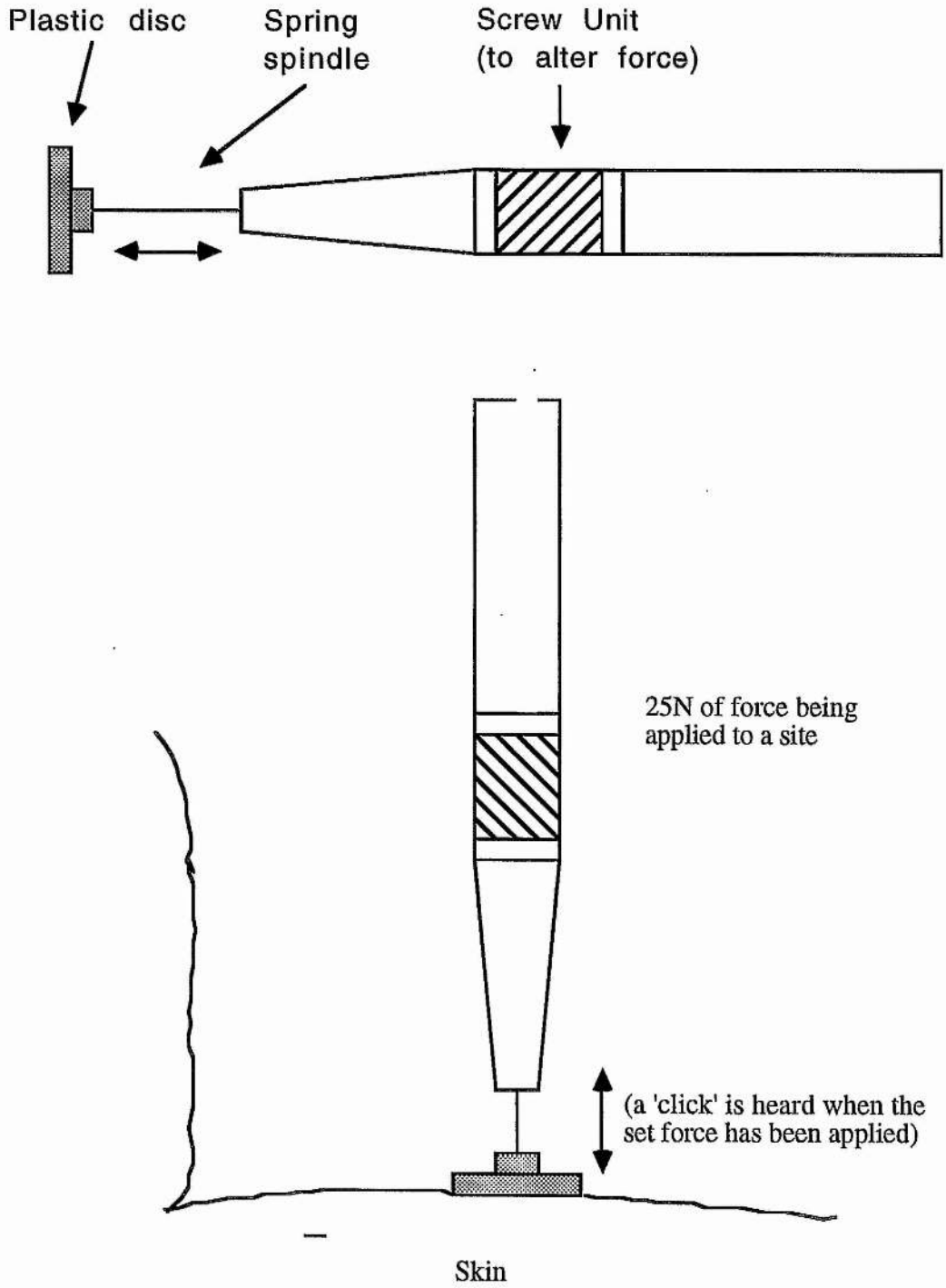
The subject was reminded to relax. The first (and highest) peak torque was then applied to the arm, and the arm was left to oscillate for three minutes. This had the effect of accustomising the subject and helped standardise the state of the arm before the initial measurement. Following these three minutes of "stirring" the first measurement took place at this torque. On completion of the 1 minute capture period the oscillation was recorded on disc. This took approximately fifteen seconds. The next peak torque was then applied and the arm was left to oscillate for fifteen seconds before data were recorded. This procedure was repeated until all five peak torques had been applied to the arm. The three minutes of stirring took place only before the measurement at the first (and highest) peak torque.

Data were recorded using a BBC Microcomputer (Acorn Computers Ltd.) and a Signal Average Programme(Bagust & Pelling 1984), and stored on floppy disc for subsequent evaluation. Using a Fast Fourier Transformation (Spectrum Analysis Programme, Structured Software 1985) it was possible to identify the resonant frequency and the amplitude of movement of the arm.

3.3.2 Muscle soreness

Muscle soreness was measured at twelve sites on the arm: at three sites along the lengths of the biceps, brachioradialis, flexor carpi radialis and triceps. These muscles were chosen as they lie on the surface of the arm and are also the largest muscles involved in flexion and extension of the forearm. Each site was marked with indelible ink. A force of 25N was applied to each of the sites using a spring-loaded centre-punch on the end of which was attached a plastic disc of 2cm diameter (Figure 3.15). Subjects were then asked to rate the perceived pain on a scale ranging from 1 to 16, where 1 represented no pain and 10 represented a rating of "very, very sore". The scale was designed so that if subjects had indicated a pain rating of very, very sore, but felt greater pain in subsequent measurements there would still be the opportunity to indicate such an increase. Subjects were further asked to rate on the same scale the soreness experienced during voluntary passive flexion and extension of the arm through the full range.

Figure 3.15 The spring-loaded centre-punch



3.3.3 Exercise to Induce DOMS

To induce muscle soreness each subject lowered a weight with the non-dominant arm, using a pulley system, from a position of full flexion to full extension seventy times. The arm was raised in front of the body and rested upon a shelf at shoulder level such that the arm was at ninety degrees to the body as shown in Figure 3.11. The maximum weight that the muscle could lower in a "controlled" manner (lowering to a count of five seconds) was determined prior to the exercise session. If the individual became unable to lower the weight in a controlled manner, some weight was removed so that the exercise could continue. This procedure was repeated until 70 contractions had been completed. The range of weights lowered was from 25 kilograms to 5 kilograms. The exercise rate was four contractions per minute - a five second contraction followed by a ten second rest period during which time the tester returned the weight to the starting position so that the subject being tested had the arm in a position of full flexion ready for the next contraction.

CHAPTER 4

MUSCLE STIFFNESS AND SORENESS FOLLOWING ECCENTRIC EXERCISE

4.1 INTRODUCTION

This study investigates the effect of unaccustomed exercise on the stiffness and soreness of the muscles involved in flexion and extension of the forearm. It is hypothesised that muscle soreness will increase following exercise, and will reach a peak 24 to 48 hours post exercise. Further, it is hypothesised that muscle stiffness will increase post exercise and will reach a peak 24 to 48 hours post exercise.

4.2 METHODS

Subjects

Sixteen male subjects were tested, mean age(25.6 ± 2.7). All were physically active but none had engaged in weight training involving the biceps brachii muscle in the previous two months. Each subject visited the laboratory on at least three occasions prior to the start of controlled measurements to familiarise themselves with the technique being used to measure muscle stiffness. Each subject acted as their own control.

Measurement Techniques and Experimental Protocol

The techniques used to assess muscle stiffness and soreness and the exercise used to create muscle stiffness and soreness are as described in Section 3.3. Measurements of muscle stiffness and muscle soreness were taken for ten days prior to the exercise at the same time of day and then at 18, 24, 42, 48, 66, 72, 90, 96 and 120 hours following the exercise bout since previous research has shown that muscle soreness reaches a peak within this time scale.

Exercise to create damage

The protocol used to induce muscle stiffness and muscle soreness is as described in the previous chapter (Section 3.3).

Capture and Storage of Data

Data were recorded as before using a BBC Microcomputer (Acorn Computers Ltd.) and a Signal Average Programme(Bagust & Pelling 1984), and stored on floppy disc for subsequent evaluation. Using a Fast Fourier Transformation (Spectrum Analysis Programme, Structured

Software 1985) it was possible to identify the RF^2 and the AM of the arm.

Treatment of Results

A two-way (Torque x Time) Analysis of Variance design with repeated measures was performed on RF^2 and AM for pre- and post-exercise measurements. A two-way (Site x Time) analysis of variance was performed on Soreness(S). Tukey tests were employed to determine significant differences between means.

4.3 RESULTS

In the ten days of pre-exercise measurements no significant differences were found in RF^2 or AM at the five levels of peak torque.

The mean RF^2 at five distinct peak torques is shown in Figure 4.1 for all subjects before and after exercise. At all peak torques RF^2 increased immediately following exercise and remained elevated for 72 hours following the exercise bout. This effect was more pronounced for higher peak torques, where RF^2 remained above the pre-exercise level until the end of the study. This immediate post-exercise increase was statistically significant ($p<0.01$) and remained so for values up to 72 hours post exercise. In some subjects RF^2 was higher for smaller torques than for larger torques.

The mean AM at five distinct peak torques is shown in Figure 4.2 for all subjects before and after exercise. The level of peak torque was decreased in value from peak torque 1 (highest level) down to peak torque 5 (lowest level). The range of values for AM pre-exercise was 1.56 ± 0.05 radians (mean $\pm$ S.E.) at the highest peak torque to 0.55 ± 0.04 radians (mean $\pm$ S.E.) at the lowest peak torque. At all peak torques AM decreased following the exercise bout and reached a minimum 42 hours post exercise in peak toruqes 1-4 ($p<0.01$). By 90 hours post exercise AM had returned to its pre-exercise level. AM decreased following exercise in 13 of the 16 subjects. At each peak torque, from highest to lowest, respectively, mean AM ($\pm$ S.D.)was reduced to 1.23 ($\pm$ 0.34), 1.02 ($\pm$ 0.30), 0.80 ($\pm$ 0.31), 0.64 ($\pm$ 0.33), 0.49 ($\pm$ 0.3), 42 hours post-exercise.

Statistically significant increases in soreness were observed 18 hours post exercise in five of the sites tested: at three sites on the biceps, at the proximal ends of the flexor carpi radialis and brachioradialis and remained elevated up to 72 hours post exercise ($p<0.01$). The mean soreness ratings at the five sites where pain increased are shown in Figure 4.3. All subjects showed increased soreness following the exercise bout.

Figure 4.1 **Mean RF2 values at five distinct peak torques for all subjects (n=16) pre- and post-exercise**

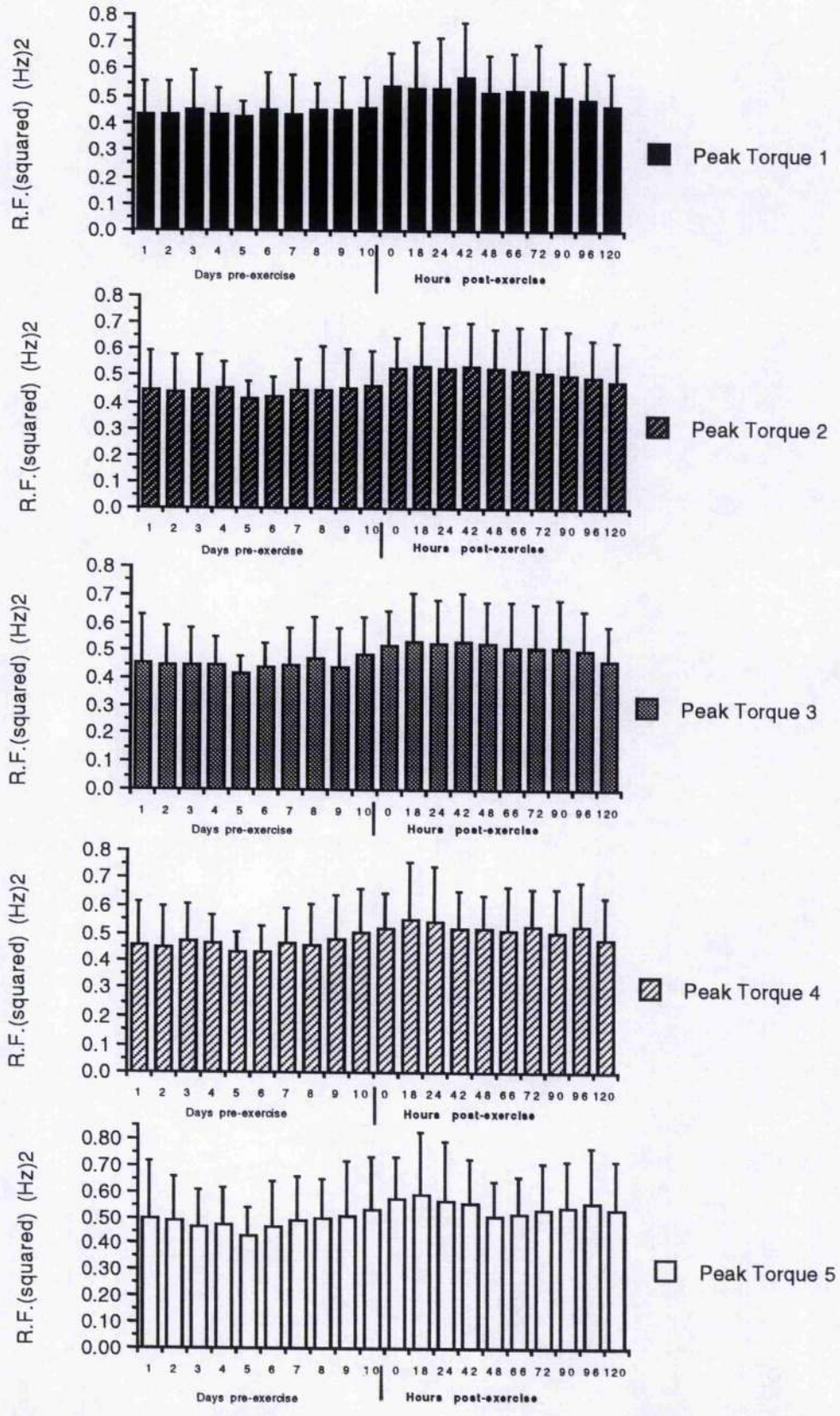


Figure 4.2 **Mean AM values at five distinct peak torques for all subjects (n=16) pre- and post-exercise**

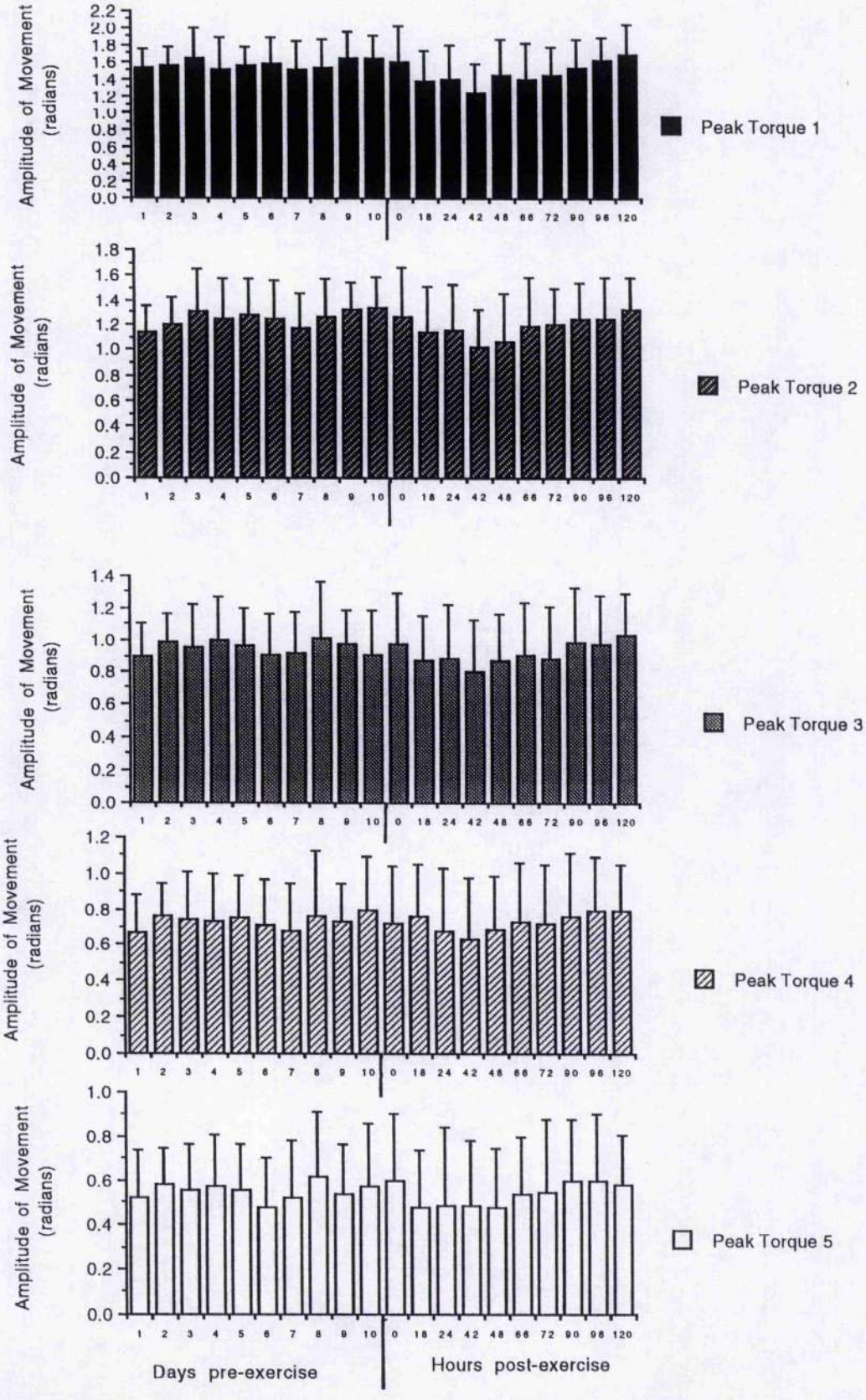
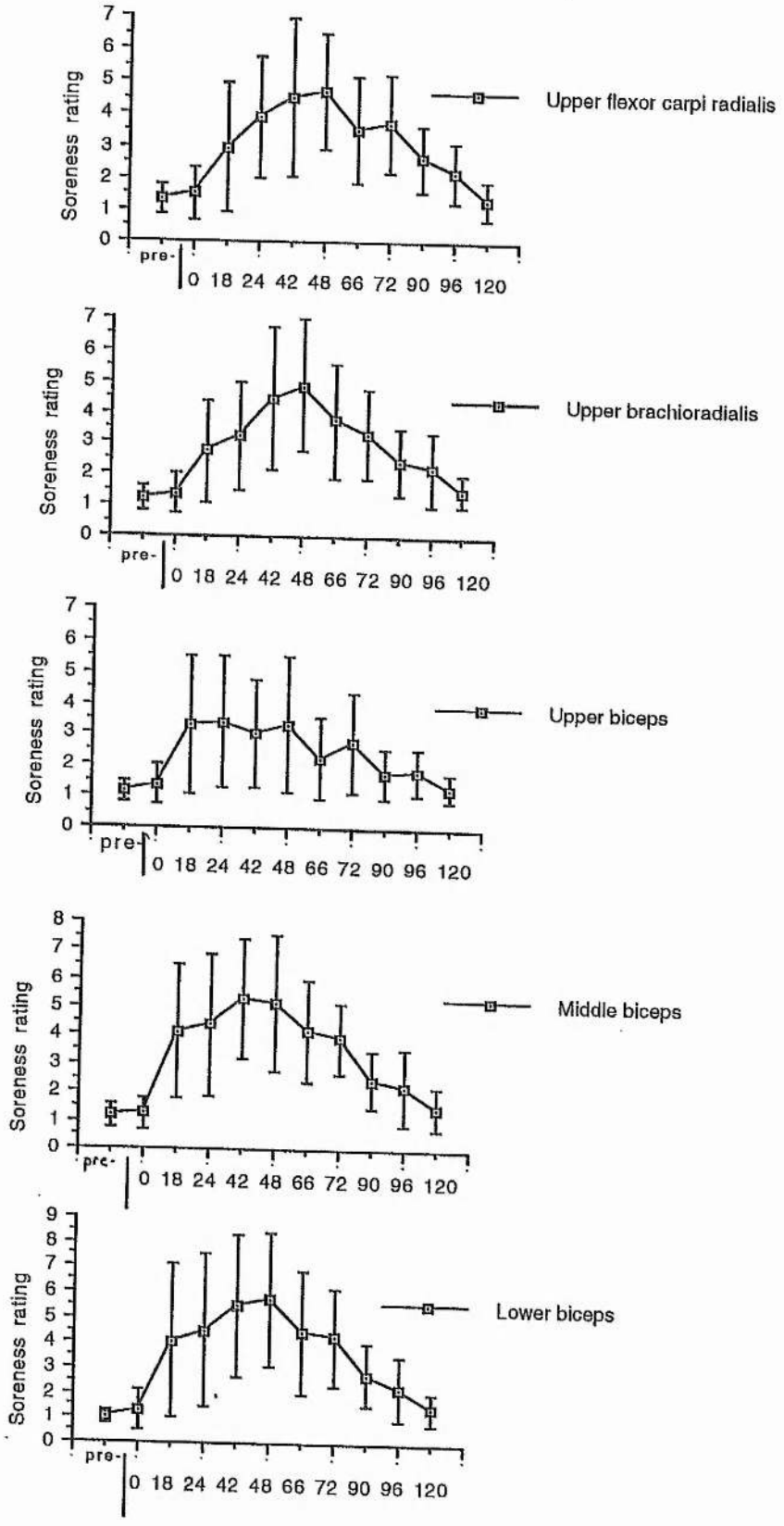


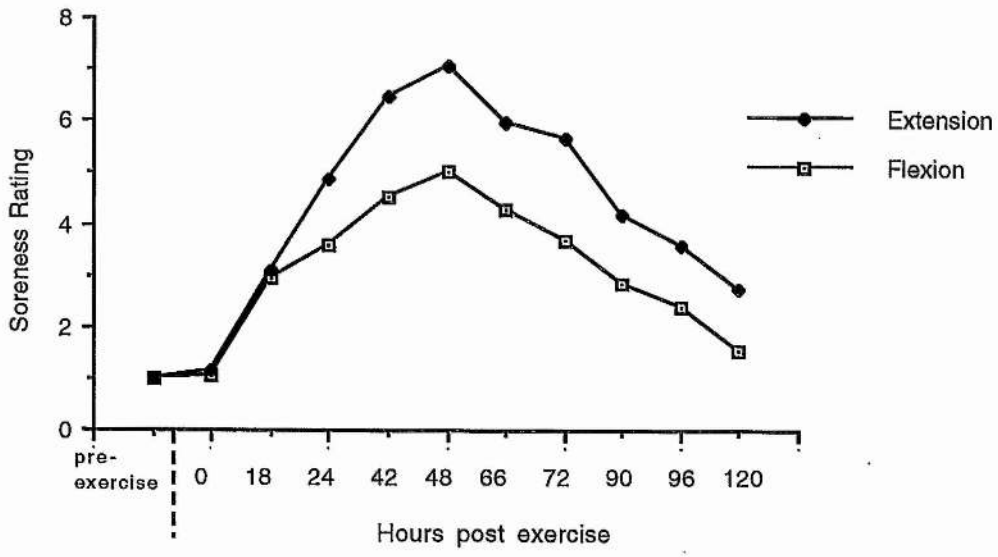
Figure 4.3 **Mean Soreness Ratings at five sites on the arm for all subjects (n=16) pre- and post-exercise**



Soreness increased following exercise and reached a peak 48 hours post exercise. Soreness then decreased until by 5 days post-exercise pain had virtually disappeared. The extent of soreness and individual pain thresholds varied from subject to subject as shown by the relatively large standard deviations. Greatest pain experienced was around the elbow joint in the flexor carpi radialis, in the brachioradialis and in the biceps brachii muscle.

The pain perceived during voluntary flexion and extension of the arm is shown for all subjects in Figure 4.4. At all times post-exercise, subjects experienced more pain during forearm extension than during forearm flexion. Post-exercise swelling was observed in the biceps and around the elbow joint in twelve of the sixteen subjects.

Figure 4.4 Mean Soreness Ratings during voluntary flexion and extension of the arm for all subjects (n=16) pre- and post-exercise



4.4 DISCUSSION

Changes in muscle tone have been quantified in two ways in this study. By examining either RF^2 (the rate of oscillation) or AM (the extent of the oscillation) of the arm it was possible to examine the effect on muscle tone of a bout of eccentric exercise designed to create muscle damage. Any changes in RF^2 reflect changes in elasticity, whereas changes in AM indicate that the viscosity of the system has been altered. Muscle has long been known to have visco elastic properties (Hill 1952).

All processes are subject to energy losses due to friction in one form or another. In vibratory processes the amplitude of the vibration gradually decreases as energy is dissipated. The extent of the frictional forces are related to the "damping" of that process. A vibratory system which is heavily damped will cease vibration very quickly. If the damping of the elbow joint was altered then a change in AM of the arm would be observed. If the arm became physically more rigid, that is stiffer, then this would be reflected in an increase in RF^2.

In thirteen out of the sixteen subjects tested in the present study there was a reduction in AM following exercise. Any reductions in AM imply that changes have occurred to viscous friction or damping of the system. That is, something happens which creates a similar effect to immersing the limb in treacle whereby the movement is restricted. The effect of increasing the damping of an oscillating system is that the resonant frequency of that system is decreased, but only slightly unless the damping is very heavy (Resnick & Halliday, 1960). This can be observed when the amplitude of a driven harmonic oscillator is plotted against the ratio of the driving frequency to the undamped natural frequency (Resnick & Halliday, 1960). In the present study the forearm assumes the role of a driven harmonic oscillator. If the damping of the elbow joint was increased then in theory RF^2 should decrease slightly. This was not observed in the present study. The decrease in AM (increase in damping) occurred when RF^2 was increased.

In the majority of subjects tested in this study RF^2 increased following exercise. Lakie and Robson (1988) also observed a slight long-term stiffening of the finger 24-30 hours after eccentric exercise. This implies that some change occurred to cause increased rigidity in the tissues surrounding the elbow joint. Such an increase may imply that there is greater bonding of actin and myosin which results in a prolonged increase in stiffness.

Increased RF^2 can be brought about when subjects voluntarily stiffen the arm by, for example, clenching the fist, or contracting the muscles of the forearm. In such circumstances, a large torque is required to displace the arm, and RF^2 can be increased fourfold, as evidenced in pilot

work. The increased stiffness observed in the present study may have been as a result of the subject resisting the motion to avoid further discomfort.

In all subjects, pain increased following the exercise bout, and followed a similar response, rising gradually to reach a peak 48-72 hours following exercise and subsiding, until by five days following exercise only slight pain was apparent. Greatest pain was experienced in the area surrounding the elbow joint in the distal end of the biceps brachii and in the proximal ends of the flexor carpi radialis and the brachioradialis and gives an indication that mechanical damage may have occurred as a result of the eccentric exercise. This is supported by studies in which the level of creatine kinase (CK) in the bloodstream has been monitored before and after a bout of eccentric exercise (Friden et al. 1983, Jones et al. 1987, Donnelly et al. 1988, Clarkson & Tremblay 1988, Hamill et al. 1989). It is possible that as pain increased in the present study, so too did the discomfort caused as a result of the technique used to measure stifness. Subjects may have been resisting the motion imposed by the printed motor, and as a result may have led to an increase in stiffness. However, it has been documented that it is at the extremes of joint motion that most pain is experienced (Jones et al. 1987, Clarkson & Tremblay), and in the measurement procedure of the present study there were limited occasions when the torque imposed created an oscillation in this range.

The success of the technique used to quantify changes in muscle stiffness is reliant upon the ability of the subject to relax. Any voluntary contribution made by the subject in assisting or resisting the torque applied by the electrical motor will affect the displacement of the arm and the resonant frequency of the joint. It was possible to determine to a certain extent whether the subject was relaxing by examining the range of movement for each torque. If the subject was relaxing then as the torque was reduced then the range of movement decreased. For five distinct torques there would be five distinct displacements. This failed to happen in a few occasions. Electromyography will be used in future investigations to confirm that no contribution to the displacement of the arm is made by the subject

An immediate increase in RF^2 can also be observed when the level of torque is reduced such that only a small oscillation of the arm takes place (Figure 15 -4.1). Such an effect is referred to as thixotropic stiffening and has been observed previously (Lakie et al, 1979, 1984, Hagbarth et al. 1985, Lakie & Robson, 1988). Actin and myosin bonds which have formed during the period of inactivity to maintain postural stability are broken when large movements take place such as the oscillation at the highest peak torque. As the movement becomes smaller more and more of these bonds reform making the arm more rigid. Lakie and Robson(1988) examined the stiffness of the finger following eccentric exercise and found that the thixotropic stiffening observed before exercise was greatly reduced following the exercise.

In the present study three subjects showed reduced stiffness following exercise and no long term stiffening.

In the present study, stiffness, measured in terms of RF^2, reached a peak at 42 hours post-exercise in three of the five peak torques. Jones et al. (1987) noted that stiffness and soreness developed following exercise and reached a peak simultaneously, 48 hours post-exercise. Stiffness was measured by these investigators as the force required to fully extend the arm, since during DOMS there is an involuntary increase in resting elbow flexion, in effect creating a voluntary shortening of the muscle. The arm rested on a shelf and as a result of the increased flexion, the wrist was not in contact with the shelf. The arm was then extended by the investigators. The greater the force required to extend the arm then the greater the stiffness. These investigators attributed the increase in stiffness to changes in the elasticity of the connective tissue. However, the reliability of this technique is questionable as during the stages of DOMS in many cases the arm will not fully extend. The pain experienced in carrying out full extension becomes unbearable.

Other studies have noted this involuntary muscle shortening following exercise , and that it (the shortening) has not be accompanied by an increase in electrical activity (Howell et al. 1985, Jones et al. 1987). Clarkson & Tremblay (1988) hypothesised that the muscle shortening noted after eccentric exercise may be due to an accumulation of calcium in damaged fibres.

Calcium plays an important role in the sequence of events that results in muscle contraction. Not only does the release of calcium initiate contraction, but the removal of calcium from the troponin binding sites initiates relaxation. In theory, if the calcium is not removed from troponin then the contraction should continue. Brody (1969) discussed the results of a case study in which a patient experienced exercise-induced contractures in muscles that were electrically silent. Following biopsy analysis the investigator reported that there was a decrease in calcium uptake and suggested that this was due to a defect in the ability of the sarcoplasmic reticulum to sequester calcium ions. Some investigators have hypothesised that when actin and myosin are pulled apart during eccentric exercise, the surface membrane of the sarcoplasmic reticulum is damaged, allowing entry of extracellular calcium (Armstrong 1984, Newham et al. 1983). This also impairs the ability of the carrier mediated active transport system (which is located in the membrane of the sarcoplasmic reticulum) to pump the calcium back into the sarcoplasmic reticulum.

Alternatively, the inability of the calcium pump to sequester calcium ions could be as a result of insufficient ATP, the fuel required to provide energy for the process. In the most extreme of cases, following death, when there is no ATP to relax the natural contractility of muscles, the

body assumes a very stiff state, known as "rigor mortis". It has been suggested by some investigators that the increased stiffness following endurance exercise (with an eccentric component) may be as a result of ATP depletion which in turn would affect the muscle's ability to relax (Noakes 1987). However, since the eccentric exercise protocols used by investigators, to induce DOMS, do not deplete ATP stores completely, this seems an unlikely explanation for the post-exercise stiffness observed in the present study (O'Reilly et al. 1987).

Some investigators have suggested that it is the connective tissue more than muscle that is damaged during DOMS (Howell et al. 1985, Jones et al. 1987, Hamill et al. 1989). An increase in the level of hydryproxoline gives an indication that damage has occurred to the connective tissue. There is a higher concentration of connective tissue at the ends of muscles and in the present study this is where most pain was experienced. However, more pain neurones are located in connective tissue than muscle therefore these areas would be more susceptible to pain changes. Jones & Round (1990) suggest that inflammation in the connective tissue may sensitise mechanoreceptors which are probably situated in the connective tissue sheath. It may be for this reason that greater pain was felt around the elbow joint especially when the muscle was lengthened.

The symptoms associated with DOMS are very similar to those associated with acute inflammation: pain; swelling; loss of function; heat and redness; histological changes, biochemical markers and cellular infiltrates (Talag 1973, Abraham 1977, Schwane et al. 1981, Newham et al. 1983, Jones et al. 1987, Clarkson & Tremblay 1988, Donnelly et al., 1988). A detailed account of the similarities between the two sets of symptoms is given by Smith (1991). With particular reference to muscle stiffness, swelling and loss of function are of particular relevance.

In the present study, during DOMS there was visible swelling around the elbow joint, in the upper forearm and in the biceps. This swelling is as a result of the increased permeability of small blood vessels which allow exudate, a protein rich fluid, to leak into the tissue of the damaged area (Friden et al. 1983, Jones et al. 1987, Donnelly et al. 1988, Clarkson & Tremblay 1988). This efflux could have the effect of damping the system, evidenced by a reduction in the AM.

There are contrasting views concerning the role that increased limb volume plays in DOMS. Some investigators have noted short term limb volume changes following eccentric exercise (Hough 1902 , Felman 1963, DeVries 1966, Talag 1973, Abraham 1977, Armstrong 1984,) whereas others have noted a sustained increase in limb volume in the days following the damaging exercise (Clarkson & Tremblay, 1988). Brendstrup (1962) examined the triceps

surae in rabbits killed between 6 and 144 hours after exercise and found increases in the water and chloride contents of the muscle, but no difference between the muscles that had been exercised concentrically and the muscles that had been exercised eccentrically. He believed the edema to be the result of irritation of the intramuscular tissue. DeVries (1966) believed the increase in limb volume to be responsible for the stimulation of pain neurones. This view was later supported by Armstrong(1984). Contradictory evidence therefore exists concerning the role of limb volume after exercise and how, if at all, it affects muscle soreness.

Some investigators have suggested that swelling may contribute to the development of DOMS as a result of an increase in local tissue pressure (Howell et al. 1985, Bobbert et al. 1988). However, although significant increases in intramuscular pressure have been observed in a tight compartment like the anterior compartment of the lower leg (Bobbert et al. 1988), no significant increases in intra-muscular pressure were observed in a less restricted compartment like the biceps brachii (Newham et al. 1987). Newham (1988) suggests that the conflicting results may be due to the compliance of the different compartments.

Talag (1973) found a non-significant relationship between muscle soreness and limb volume as peak limb volume was observed at 72 hours post exercise, and peak pain observed at 48 hours post exercise. This late increase in edema could be explained by Ryan & Majno's (1977) proposal that the swelling seen in the first few hours and up to 48 hours post-exercise was brought about by the accumulation of fluid in the tissues. They suggest that this may be followed by a later increase in limb volume around 72 hours when new connective tissue is being produced. There is evidence to suggest that connective tissue regeneration begins around 72 hours post-exercise (friden et al. 1983, Jones et al. 1986).

The build up and movement of fluid within the muscle may play a more important role in muscle stiffness, more specifically in changing the viscous friction of the affected muscles. Howell et al. (1985) stated that "the edema within the perimuscular tissue may alter the elastic behaviour of the muscles and cause restriction of motion". The swelling observed in the present study may have resulted in the noted increases in stiffness.

The loss of function associated with DOMS is evidenced by a reduction in the level of force that can be produced by the affected muscles. This reduction is apparent immediately following the exercise bout that has been performed to induce DOMS, and can take up to 14 days to return to its pre-exercise level. There is a poor relationship between decrements in strength and the increase in pain (Talag 1973, Newham et al. 1983, Clarkson & Tremblay 1988). The effort sustained to produce a full voluntary contraction while suffering from DOMS is not influenced by pain (Newham et al. 1987). It is hypothesised that the pain associated

with DOMS occurs at a critical time in the healing process, when perhaps new and fragile capillaries are being formed (Peacock 1984) or connective tissue re-synthesis is taking place (Howell et al. 1985). The purpose of the pain is to discourage the sufferer from engaging in further activity which may interrupt this process.

In summary, eccentric exercise has been shown to cause muscle soreness, increased RF^2 and a reduction in AM of the arm for the same given torque. Similar changes in muscle tone may also be observed following isometric or concentric exercise. This has yet to be investigated. The reduction in AM could be as a result of the damage caused by eccentric exercise or as a result of the subject voluntarily resisting the torque applied by the electrical motor in an attempt to avoid further pain being experienced.

CHAPTER 5

MUSCLE STIFFNESS, SORENESS AND ADAPTATION

5.1 INTRODUCTION

Investigators have noted (Abraham 1977, Byrnes et al. 1985, Seaman & Ianuzzo 1988, Clarkson & Tremblay 1988, Ebbeling & Clarkson 1990) that following a bout of exercise leading to DOMS, adaptations are seen to take place within the muscle and connective tissue which protect it from further DOMS and damage when a subsequent bout of the same exercise is performed.

While adaptation leading to reduced muscle soreness and damage has been observed following eccentric exercise, such an adaptation in muscle stiffness has yet to be investigated. This study investigated whether muscle stiffness increased following eccentric exercise and, if so, whether an adaptation occurred leading to reduced muscle stiffness following a second bout of the same exercise six days later. In research of this kind it has been observed that the majority of subjects who have suffered muscle soreness will have recovered by six days following the exercise that resulted in DOMS (Armstrong 1984, Schwane et al. 1987). To assess the extent of the damage caused as a result of the eccentric exercise the level of plasma creatine kinase (CK) was measured. The presence of CK in the blood gives supporting evidence that damage has occurred to muscle tissue when unaccustomed exercise is performed (Friden et al. 1983). The hypothesis is that muscle stiffness, muscle soreness, the level of plasma creatine kinase and limb girth will increase following a bout of eccentric exercise, but such increases will not be seen following a bout of the same exercise repeated 6 days later.

5.2 METHODS

Subjects

Sixteen consenting volunteers aged 22.3 $\pm$ 1.7 years (mean $\pm$ S.D.) acted as subjects in this twelve day study. All were physically active but none had taken part in any weight training programme involving the arms in the eight weeks prior to the commencement of the study.

Parameters Assessed and Experimental Protocol

The parameters assessed were muscle stiffness (RF^2 and AM) muscle soreness, plasma creatine kinase (CK), limb girth and electromyography.

Muscle Stiffness

Muscle stiffness was measured using the "positive velocity feedback" technique developed by Walsh and co-workers in the 1970s and is as described in the previous experiment.

Muscle Soreness

Muscle soreness was measured immediately following the measurement of muscle stiffness at twelve sites on the arm as described previously

Limb Girth

Limb girth was measured following the assessment of muscle soreness. An elastic band was positioned at a right angle to the long axis of the arm around the belly of the biceps brachii at the greatest circumference. A ring was then drawn around the arm with indelible ink using the elastic band as a guide. The band was then removed. The circumference was measured around the ink trace using a metal measuring tape while the subject stood with the arm hanging loosely at the side of the body in a relaxed state.

Creatine Kinase

Before measurement of muscle stiffness, muscle soreness or limb girth took place, blood samples (2ml) were taken by venous puncture from an ante-cubital vein, transferred to a test tube and allowed to clot at room temperature. The samples were then centrifuged for ten minutes and serum was removed using a pipette and stored at -20°C until analysed. A Boehringer-Mann test kit was used to assess the level of creatine kinase. The CK reagent was prepared and 1ml transferred to a cuvet and brought to the incubation temperature of 30°C. 0.02ml of the sample was added to the reagent and mixed by inversion. The cuvet was then placed in a constant cuvet compartment and incubated for three minutes. The absorbance reading through a 1cm light path was taken at the start and the end of the 3 minute period using a spectrophotometer. The change in amplitude was recorded as a trace on a chart recorder and the rate of change per minute was then calculated. CK activity was determined using the equation, CK(U/l) = Change in absorbance/minute x 8200 Samples were analysed in duplicate.

Electromyography

To determine whether the muscle was active or inactive while the arm was oscillating during the measurement of muscle stiffness electromyography was monitored. Disposable electrodes (Biotabs) were attached to the belly of the biceps. The signal was recorded on a chart recorder (Linearecorder markVII WR3101, Environmental Equipments(Northern)Ltd.) alongside the trace which indicated the change in displacement of the arm.

All parameters were measured immediately before a bout of eccentric exercise designed to induce DOMS and at 24, 48, 72, 96 and 120 hours after exercise. This procedure was repeated six days following the first exercise bout. The subjects were instructed to refrain from any exercise involving the arms during the period of the study.

Exercise to create damage

The protocol used to induce muscle stiffness and soreness is as before and is described in Section 3.4.5. The number of repetitions at each weight was recorded so that the amount of work performed would be the same in both exercise bouts. If the amount of work performed in Exercise Bout 2 (EX2) was less than that of Exercise Bout 1 (EX1) then it could have been argued that the absence of pain following EX2, as was hypothesised, was due to less work being performed in EX2. Performing the same amount of work allowed direct comparison of the effect of each exercise bout.

Capture and Storage of Data

The capture and storage of data is as described previously (section 4.2.3)

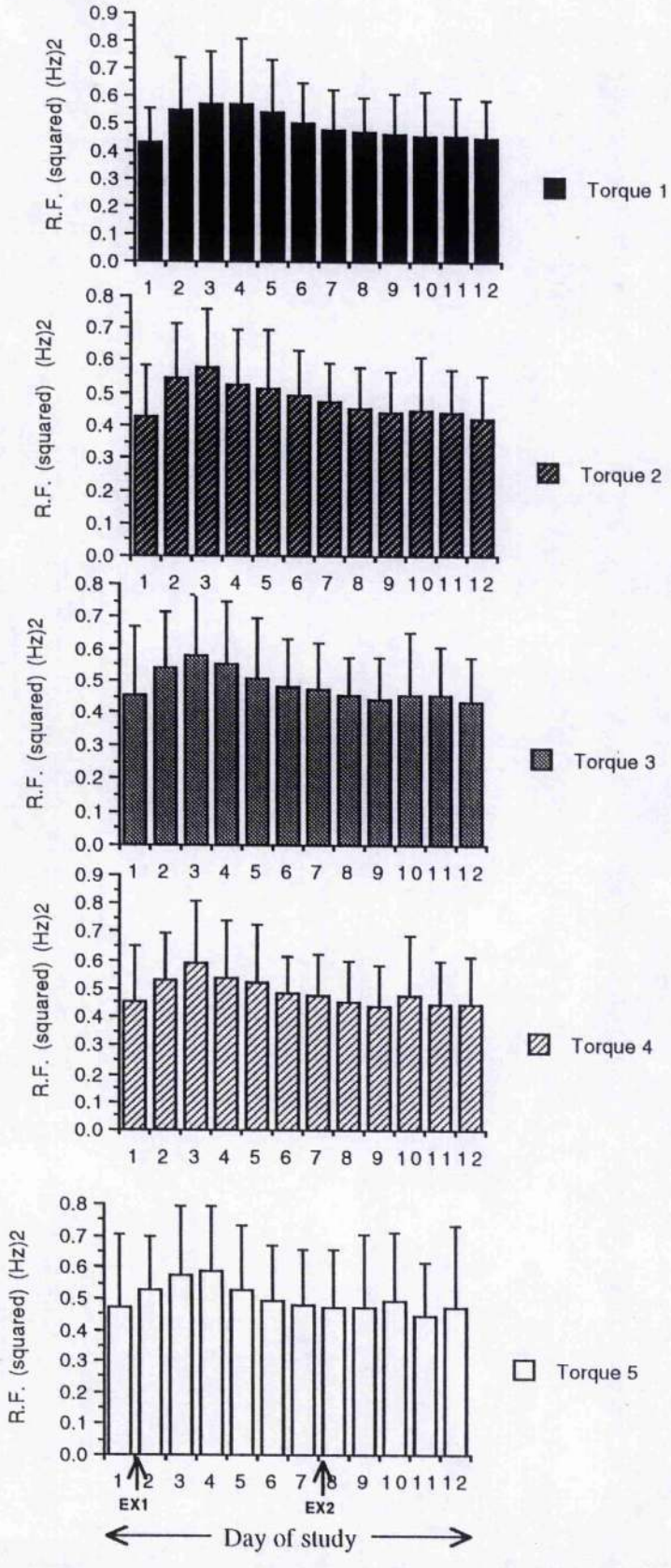
Treatment of Results

A two way (Torque x Time) Analysis of Variance with repeated measures design was performed on RF^2 and AM for pre- and post-exercise measurements. A two way (Site x Time) analysis of variance was performed on soreness. A one way Analysis of Variance was performed on limb girth, and Page's L Trend Test (Cohen & Holladay, 1984, p226) performed on Creatine Kinase data. Tukey tests were employed to determine significant differences within treatments.

5.3 RESULTS

Figure 5.1 shows RF^2 plotted against time for each of the five distinct peak torques. Twenty four hours following the first exercise bout (EX1) RF^2 had increased at each of the five peak torques ($p<0.01$) and remained significantly different from the pre exercise value each day, until three days following EX1. The values obtained for RF^2 at 24, 48 and 72 hours following EX1 were compared with the corresponding values of RF^2 following the second exercise bout (EX2) and statistically significant differences were observed. A peak in RF^2 occurred 48 hours following EX1. Thereafter RF^2 decreased throughout the period of the study. No increase in RF^2 was seen following EX2. No differences were observed in the values obtained for RF^2 at each of the peak torques.

Figure 5.1 **Mean RF² values at five distinct peak torques for all subjects (n=16) pre- and post-exercise bout 1 and exercise bout 2**



AM of the arm at five peak torques is shown in Figure 5.2. AM decreased following exercise and reached a minimum 48 hours after EX1 ($p<0.01$). The reduction in AM (radians $\pm$ S.D.) at each of the peak torques can be seen in . No reduction in AM at any of the peak torques was seen following EX2. As the level of torque decreased the response remained the same.

Figure 5.2 Mean AM values at five distinct peak torques for all subjects (n=16) pre- and post-exercise bout 1 and exercise bout 2

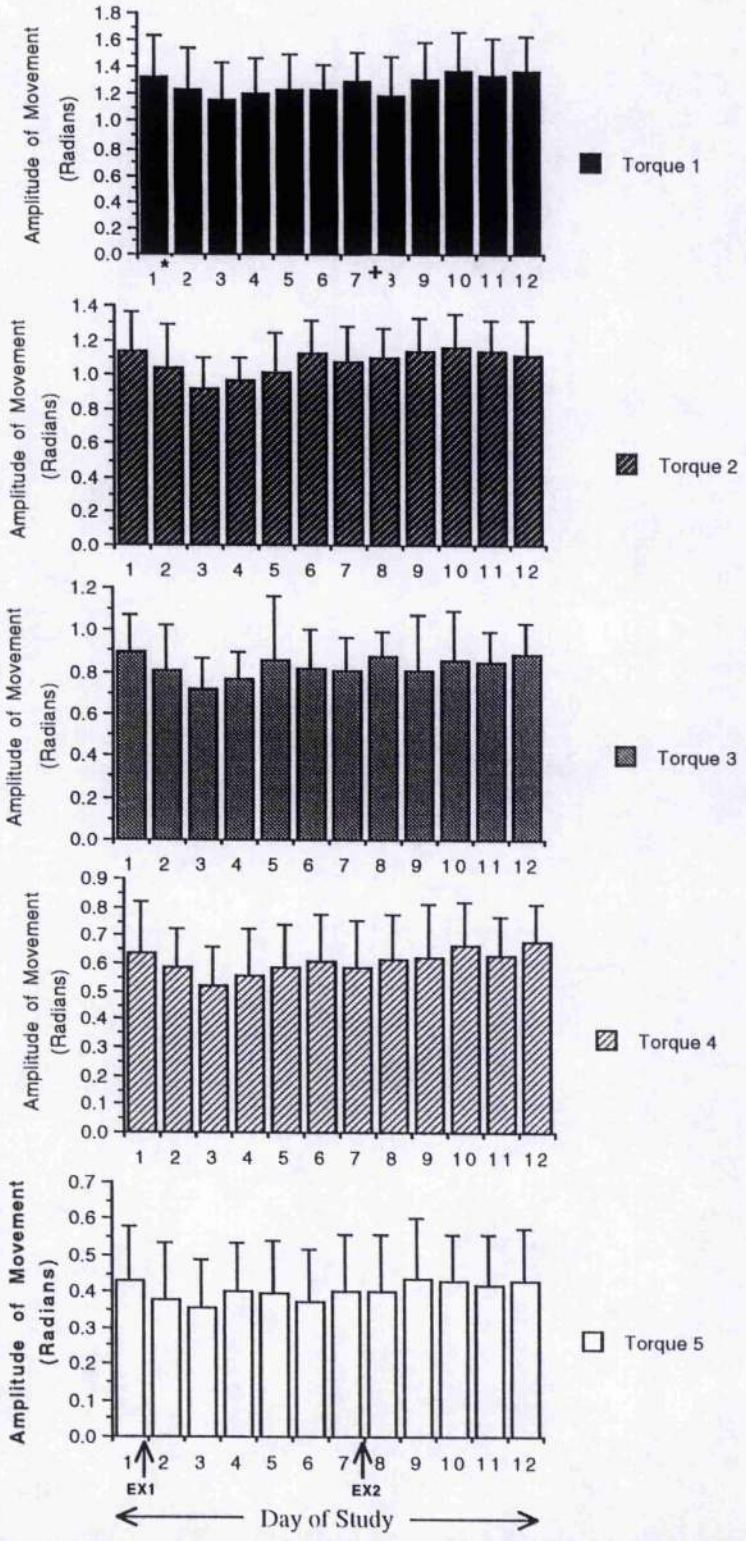


Figure 5.3 Mean reduction in AM at each of the five distinct peak torques for all subjects (n=16) pre- and post-exercise bout 1 and exercise bout 2

TORQUE	PRE EXERCISE		48 HOURS POST EX1	
1	1.32	(± 0.31)	1.15	(± 0.29)
2	1.14	(± 0.22)	0.92	(± 0.18)
3	0.90	(± 0.17)	0.72	(± 0.15)
4	0.66	(± 0.19)	0.52	(± 0.14)
5	0.43	(± 0.15)	0.35	(± 0.13)

Statistically significant increases in muscle soreness ($p < 0.05$) were observed at five of the twelve sites tested (Figure 5.4). Greatest soreness was experienced around the elbow joint, in the upper brachioradialis, the upper flexor carpi radialis and in the three sites tested on the biceps muscle. Soreness peaked at 48 hours following EX1. The large increases in soreness observed following EX1 were not observed following EX2. Subjects were significantly sorer ($p < 0.01$) after EX1 than after EX2 at 24, 48, 72 and 96 hours post exercise. Following EX1 pain was experienced during passive voluntary flexion and extension of the forearm. Pain peaked at 48 hours post exercise. Figure 5.5 illustrates that at all times post exercise greater pain was experienced during voluntary extension than voluntary flexion. Subjects rated the pain experienced during passive extension and flexion on a scale of 1 - 16. The results shown are the mean values for all subjects on each day of the study. Standard deviation bars have been eliminated to avoid cluttering of the figure. The pain response following EX2 was significantly different ($p<0.01$) from that seen following EX2. No real increase was observed in pain following EX2, only a respite in recovery from EX1, as indicated by the change in gradient of the curves for extension and flexion 24 hours following EX2.

The pre-exercise values obtained for serum creatine kinase ranged from 55 to 226 U/l. Serum creatine kinase increased by varying degrees following exercise. Peak values ranged from 78 to 18005 U/l, four or five days following EX1. To display data, subjects have been combined in four groups according to the level of creatine kinase detected. Figure 5.6a displays the two groups of subjects with highest responses (n=4, n=3), and Figure 5.6b displays the two groups of subjects with lower responses (n=4, n=5). Page's Trend Test was used to analyse the data and to test the hypothesis that serum creatine kinase would peak four days following exercise, and that the response would be greatly reduced following EX2 : the hypothesis was correct. The worked example of the test is shown in Appendix E. There was no significant difference between the predicted and actual order of CK response.

Figure 5.4 **Mean Soreness Ratings for all subjects (n=16) at five sites on the arm pre- and post-exercise bout 1 and exercise bout 2**

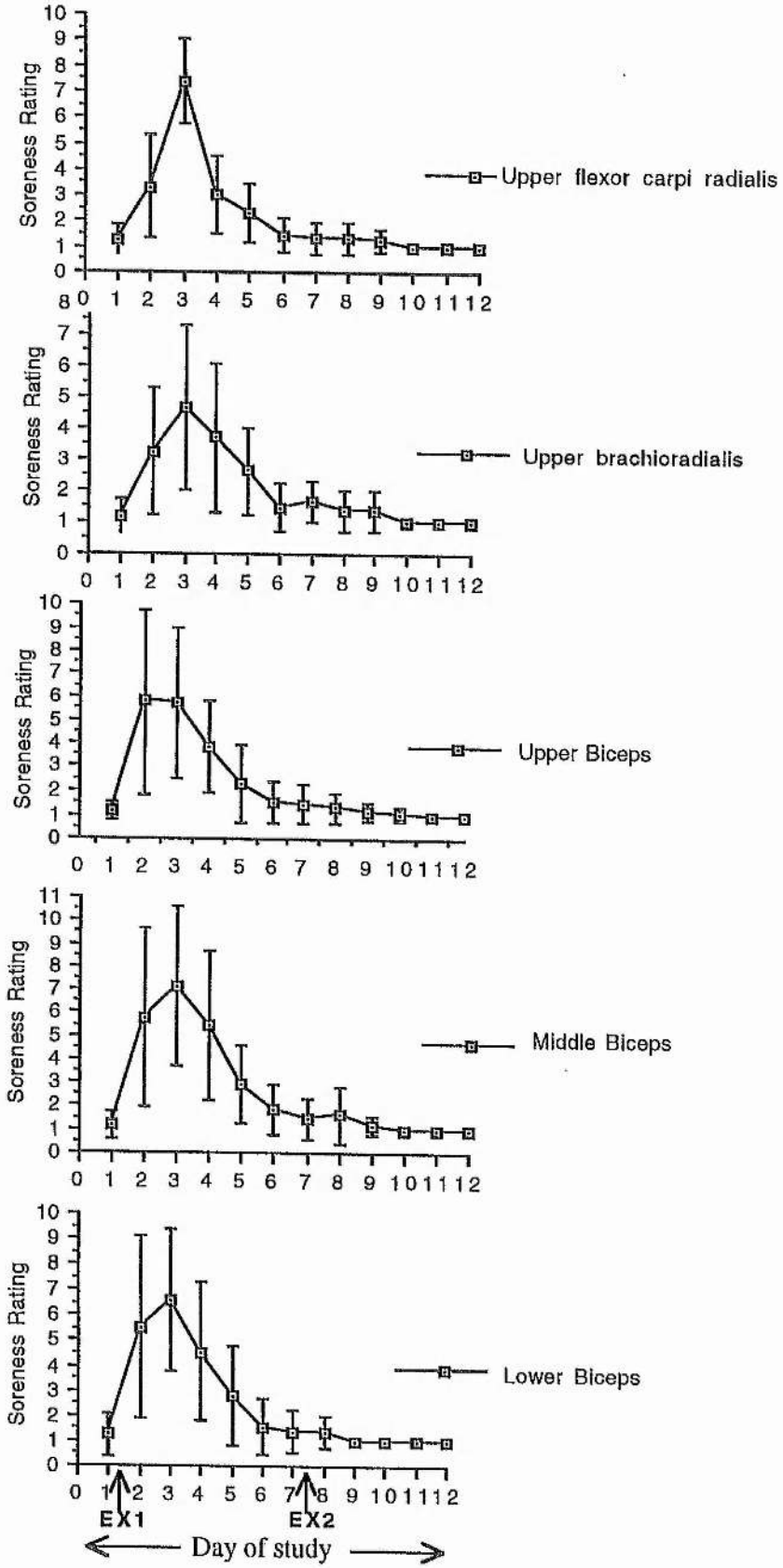


Figure 5.5 **Mean Soreness Ratings during voluntary flexion and extension of the arm for all subjects (n=16) pre- and post-exercise bout 1 and exercise bout 2**

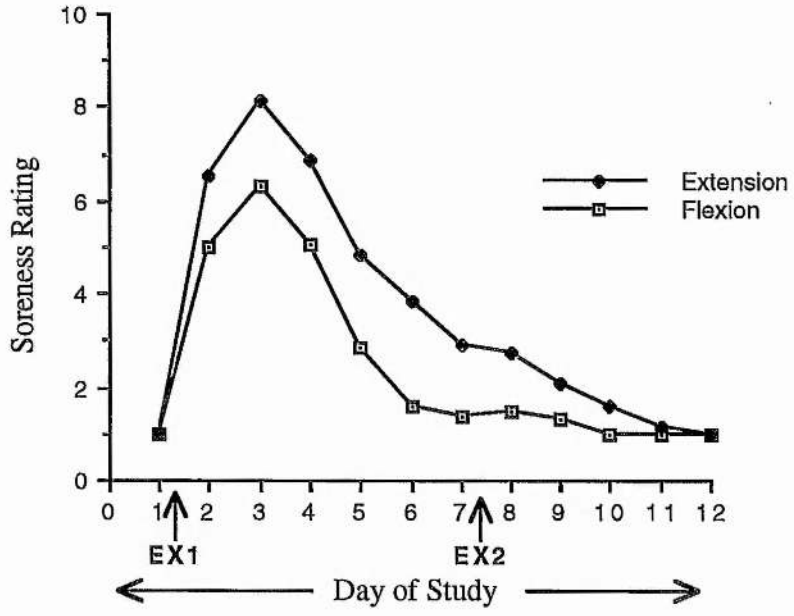


Figure 5.6a **Mean serum Creatine Kinase values in the two higher responding groups pre- and post exercise bout 1 and exercise bout 2**

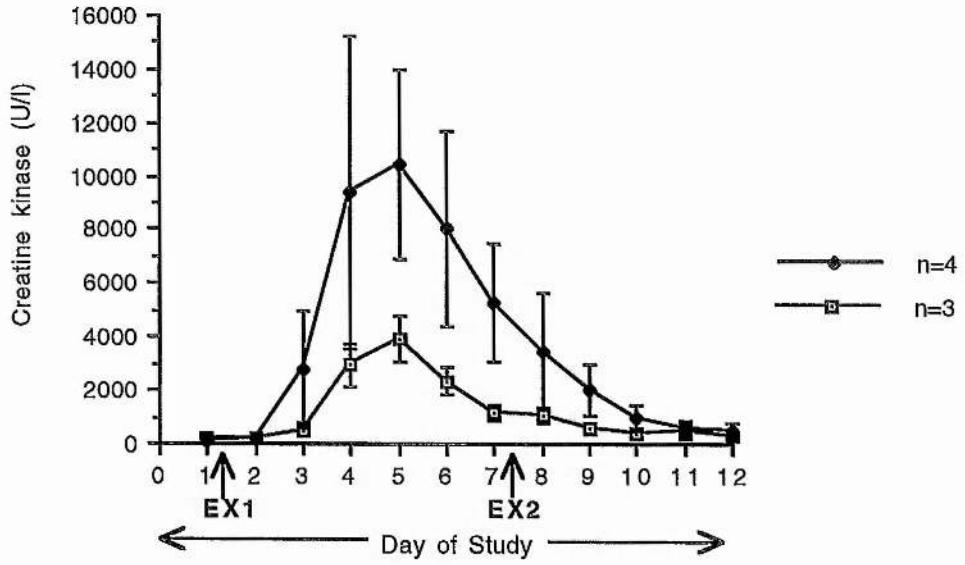


Figure 5.6b Mean serum Creatine Kinase values in the two lower responding groups pre- and post exercise bout 1 and exercise bout 2

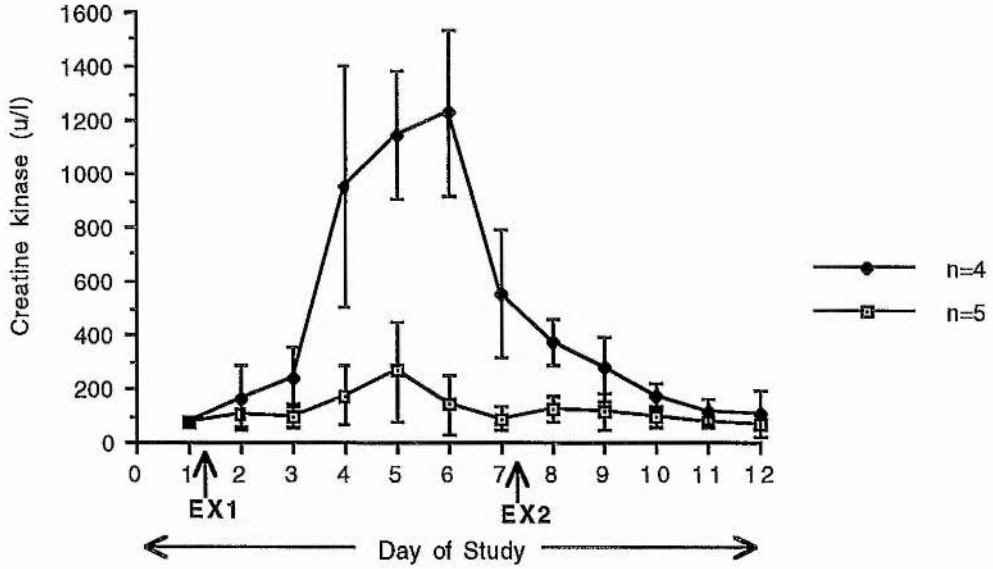
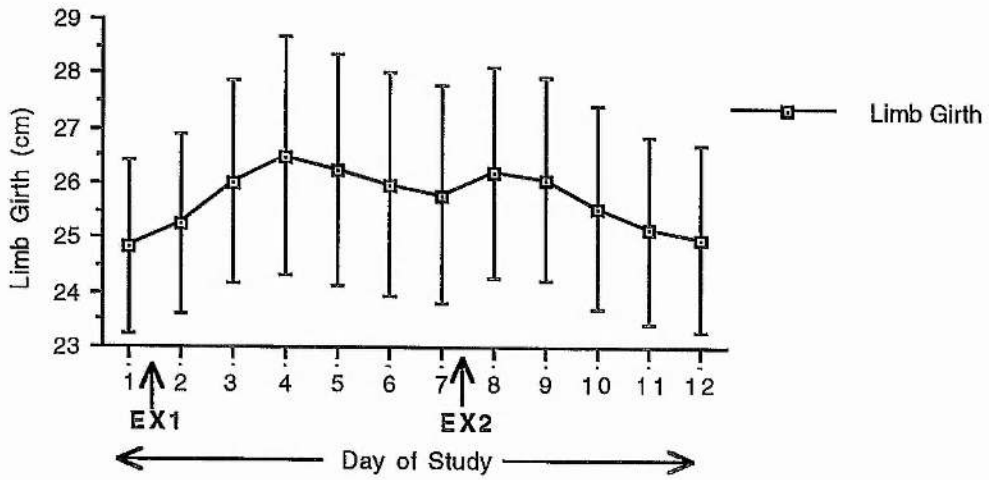


Figure 5.7 illustrates the changes in limb girth over the period of the study. Mean limb girth increased following EX1 from 24.7 cm (± 1.8) to 25.8cm (± 2.1) 48 hours post-exercise ($p<0.01$) and reached a peak of 26.2 cm (± 2.5) 72 hours post exercise ($p < 0.01$). On the seventh day of the study, immediately before EX2, mean limb girth (25.6 cm ± 2.5) had still not returned to its pre EX1 value. Twenty four hours following EX2 limb girth had increased to 25.8 cm (± 2.4). This was significantly different from the pre-EX1 measurement ($p<0.01$) but no different from the measurement taken at 24 hours after EX1. Limb girth then decreased and had returned to its pre-exercise value by the end of the study.

During measurement of muscle stiffness the electrical activity in the biceps muscle was monitored to ensure that subjects did not contribute to the motion of the arm and that the movement was driven entirely by the motor. The muscle remained inactive during the measurement of muscle stiffness in all but a few trials. Any contribution (in helping the motion) from the subject tended to be at the smaller peak torques. In such cases where there was a contribution from the subject the trial was repeated. There was no apparent resistance to movement by subjects who wished to avoid further pain.

Figure 5.7 **Mean limb girth for all subjects (n=16) pre- and post-exercise bout 1 and exercise bout 2**



While subjects were suffering from DOMS there appeared to be an involuntary shortening of the forearm flexors, that is, a decrease in the natural resting flexion angle of the forearm as it hung in a relaxed state by the subject's side. No objective monitoring of this angle was carried out.

5.4 DISCUSSION

In this study, stiffness, quantified in terms of RF^2 and AM, was seen to increase following unaccustomed eccentric exercise. Any changes in RF^2 reflected changes in elasticity, whereas changes in the AM indicated that the viscosity of the system has been altered. Similarly, an increased pain and CK response was observed post-exercise. Limb girth also increased. Following a second bout of the same exercise, six days later, the large increases seen in stiffness, pain, CK and limb girth following EX1 were not observed following EX2 ($p<0.01$). An adaptation is thought to have taken place which led to this reduced response.

By 24 hours post EX1, RF^2 had increased at each of the five peak torques, reached a maximum at 48 hours, and remained elevated for a further day ($p<0.01$). The increase in RF^2 indicated that the arm was more rigid, that is, stiffer. This post exercise increase in RF^2 has been observed in the study in the previous chapter (Section 4.4). In that study the post exercise increase in RF^2 lasted for four days following the exercise bout, with a peak at 42 hours post exercise in three of the five peak torques. In the present study no measurement was taken 42 hours post exercise for practical reasons.

The pattern of change in RF^2 was the same at each of the five peak torques with a rise following the exercise and subsequent decrease, albeit that the peaks occurred at different times post-exercise. It was expected that as the level of peak torque decreased then there would be an increase in RF^2 with the highest value for RF^2 at the lowest peak torque, as observed in other research of this type (Lakie & Robson 1988). The theory behind this is that during periods of inactivity actin and myosin bond together to maintain postural stability. During movement these bonds are broken but as movement decreases then the bonds are allowed to re-form, in effect making the system more rigid, or stiffer as explained in Section 3.1.3. This increase in RF^2 as torque decreases was perhaps not observed because the level of torque supplied was not low enough.

The increased rigidity, as evidenced by an increase in RF^2, can be brought about voluntarily, as when the fist is clenched or when the fingers are straightened. This, in effect, increases the interaction between actin and myosin at any one time. The perception of soreness may have caused the subjects to tense the arm muscles in the hope of reducing discomfort. This may have been the case in the study carried out by Jones et al. 1987 where stiffness was measured as the force required to extend the arm in the horizontal plane while it rested on a shelf. No mention is made by these investigators whether there was voluntary resistance or whether there was increased pain while the arm was being straightened during the measurement of stiffness. These investigators claimed that stiffness did increase following exercise. The changes in RF^2

invoked by voluntary stiffening had been observed in pilot work, hence the need to monitor EMG to ensure the subject's arm was relaxed throughout the test. It is also difficult to stiffen voluntarily in a consistent way, which leads to irregularities in the spectrum. If the subject had voluntarily stiffened the arm to resist the motion which the electrical motor imposed upon it then this would have been picked up in the present study by an increased electrical signal. No such increase in EMG was observed. A change had therefore occurred altering elastic properties of the exercised muscles.

In the present study there was an observable change in the angle of flexion of the forearm in most subjects following exercise. The arm hung in a more flexed position after exercise and this appeared to peak 2-4 days following the exercise bout. Difficulty and pain were experienced when subjects attempted to extend the forearm. Indeed, at times it was impossible to straighten the arm to its full extent. However, in this increased state of involuntary flexion there was no increase in electrical activity in the biceps. Possible hypotheses for this voluntary shortening have been suggested in the previous chapter (Section 4.4). Jones et al. 1987 suggested that the decrease in flexion angle post exercise may be as a result of shortening of the connective tissue and not of shortening of the muscle as previously thought. The evidence in the present study seems to support this suggestion.

AM was reduced following exercise and reached a minimum 48 hours post EX1 at each of the five peak torques. This was further evidence that there had been a change in muscle tone following the exercise bout. Changes in AM reflected changes in the viscosity of the system. The effect is similar to the resistance experienced when trying to stir a spoon in treacle after stirring a spoon in water. For the same effort there is less movement. The increase in viscous friction could be as a result of swelling in the area surrounding the elbow joint . There are many contrasting views concerning the role that increased limb volume plays in muscle soreness but the quality of the research is questionable and the evidence inconclusive. The possible effects of swelling on muscle stiffness and soreness are discussed in greater details in the previous chapter (Section 4.4).

However, of more significance are research studies that suggest that the build up and movement of fluid within the muscle may play a more important role in muscle stiffness. In the present study the limb girth of the biceps brachii increased and reached a peak 72 hours post exercise and supports the hypothesis that the increased edema could lead to increased damping and hence a restriction in movement. This sustained increase in limb volume in the days following the damaging exercise has been observed elsewhere (Talag 1973, Bobbert et al. 1986, Clarkson & Tremblay 1988) and can perhaps be explained by Ryan and Marjo's (1977) suggestion that connective tissue is being re-synthesised at this stage.

Howell et al. (1985), in carrying out eccentric exercise of the biceps on seventeen subjects, noted that there was swelling of the arm at the level of the belly of the biceps and just above the humeral epicondyles 48 hours after the exercise bout. The investigation was well controlled and involved extensive EMG measurement before and during the stages of DOMS, as it was primarily concerned with the relationship between EMG and the restriction of motion during DOMS. These investigators suggested that the swelling caused the restriction in motion that was apparent following the exercise and believed that edema within the perimysium may alter the elastic behaviour of the muscles and cause restriction of motion. Jones et al. 1987 also noted when investigating post-exercise muscle stiffness and soreness that a flexion deformity was apparent following exercise, and that passive movements reduced the deformity, possibly by squeezing fluid out of the muscle. Jones et al. (1987) reported that the reduced stiffness following movement was temporary, and that soon after the cessation of movement the stiffness had returned to its previous level. This is also supported by Lakie & Robson (1988) who investigated thixotropic changes in the stiffness of the relaxed finger muscles. They found that movement was always seen to decrease the stiffness and never produced increased stiffness.

These suggestions are in line with the results of the present study. Forty eight hours after the exercise the elbow flexion angle was visibly reduced, movement of the arm was restricted and there was a marked increase in limb volume at the time when muscle stiffness was at its greatest. However, the increase in muscle stiffness observed in the present study may also be due to increased levels of extracellular and/or intracellular calcium, a reduction in ATP levels or inflammation. The potential effect of each of these factors on muscle stiffness have been discussed in the previous chapter (Section 4.4).

Subjects in the present study experienced greatest pain around the elbow joint. This was also noted in the previous study and this has been discussed in the previous chapter (Section 4.4).

Evidence that damage has been caused to muscle tissue is presented with the increased levels in serum CK following EX1. The extent of the increase varied from subject to subject with peak values ranging from 104 U/L to 18005 U/L. This varied response has been reported elsewhere (Newham et al. 1987, Clarkson et al. 1987, Schwane et al 1987, Maughan et al. 1989). The enzyme release after EX1 was as reported by other investigators for this type of exercise, being large and delayed for several days following the exercise (Newham et al. 1987, Jones et al. 1986, Clarkson & Tremblay 1988, Schwane et al. 1987). Further, despite the variation between subjects in the extent of the release, the levels of CK were higher after EX1 than after EX2 for all subjects. This response has also been noted elsewhere (Clarkson et al. 1987, Clarkson & Tremblay 1988, Newham et al. 1985, Byrnes et al. 1985, Trifletti et al. 1988).

Following downhill running the peak in CK is usually 18 to 24 hours after the exercise (Byrnes et al. 1985, Kirwan et al. 1986, Trifletti et al. 1988). The peak in CK following isolated eccentric exercise occurs up to 5 days following the exercise. In downhill running peak levels of CK range from 177 U/L to 1,136 U/L. In isolated eccentric exercise, peak values range from 1036U/L to 30120 U/L. It is thought that changes in membrane permeability associated with metabolic disturbances (hypoxia and ischaemia) may play a role in CK efflux immediately or several hours after exhaustive exertion or high tension isometric exercise. The peak in CK seen up to 5 days following intensive eccentric exercise has been suggested to occur at the same time as necrosis.

In the present study there was a large variation between subjects in the extent of CK release. Peak values ranged from 78 I.U/l (indicating very little damage) to 18005 I.U/l (indicating severe damage). The peak occurred between four and five days following the exercise bout. The factors that could have influenced this are the training status of the subjects involved, the effort applied during the exercise, or the amount of exercise performed. However, despite the variation between the subjects in the extent of the release, the levels of CK were invariably higher after EX1 than after EX2. Although the release of CK and the pain experienced were significantly reduced following EX2 it seems unlikely that the two are causally related (Newham et al. 1986, Schwane et al. 1987, Maughan et al. 1989). The two are always separated by time with peak pain occurring 24-72 hours post exercise and peak CK occurring 4-5 days post exercise. Individual variation is such that although one individual may have extreme pain there may be little or no evidence of muscle membrane damage (Newham et al. 1986), as measured by CK leakage. In the present study however, the subject that showed the greatest efflux of CK experienced the greatest pain. This relationship was not maintained by the other subjects.

Clarkson & Tremblay (1988) suggest that the time sequence for necrosis to occur may explain the delayed appearance of CK in the blood. They suggested that exercise-induced damage may cause an accumulation of Ca++ resulting in : (1) the production of noxious stimuli such as bradykinin and histamine causing muscle soreness; (2) muscle contractures leading to decreased range of motion; (3) impairment of sarcoplasmic proteases resulting in loss of sarcolemmal integrity and delayed release of CK.

Some studies have shown that the uptake of technetium-99 labelled pyrophosphate is related to the loss of sarcolemmal integrity (Vita & Harris, 1981, Newham et al. 1986). In their study, Newham et al.(1986) found that the uptake of technetium-99 in eccentrically exercised muscle occurred at approximately the same time that CK peaked in the blood. Clarkson & Tremblay (1988) suggest that the delayed appearance of CK in the blood may represent the final stage of

the exercise damage process where an eventual loss in sarcolemma integrity occurs. The exercise is suggested to cause the damage which triggers the run of events that results in the eventual leakage of CK.

Six days following EX1, EX2 took place. The amount of exercise performed was the same in EX1 and EX2, as during EX1 the number of contractions performed at each weight was noted. No increase in RF^2 was observed following EX2. The release of CK and the pain response were also significantly reduced. An adaption had occurred in the space of six days suggesting that EX2 had little, if any, effect on the stiffness and soreness of the muscles that had performed the exercise, and also little effect on CK leakage and limb girth.

Investigators have suggested that the pain and stiffness experienced following unaccustomed exercise are as a result of the shortening of connective tissue, and that a training effect initiates some adaptation in the tissue (Howell et al. 1985, Jones et al. 1987, Newham et al. 1987) Clarkson & Tremblay (1988) suggested that some strengthening of the muscle cell membrane and the surrounding connective tissue must take place following exercise that has resulted in DOMS. A reduction in the efflux of CK following EX2 would suggest that there is less leakage from the cells and therefore that the cell membrane must be stronger. The natural response of the body would therefore seem to be one in which the site of injury is strengthened and protected to prevent it from further insult. Similarly, the increase in limb volume noted after EX1 was not observed following EX2 suggesting that the amount of edema that built up following EX1 was greatly reduced after EX2, again because the cell membrane has strengthened. Therefore it seems that the adaptation takes place both in the muscle tissue and in the surrounding connective tissue.

Grimby & Saltin (1983) have shown that throughout life a small percentage of the total muscle fibre population degenerates, resulting in a decrease in the total number of fibres with increasing age. Armstrong(1984) suggests that a bout of unaccustomed exercise would damage this pool of fragile or stress-susceptible fibres. As a result, the process of degeneration-regeneration would mean that there would be fewer stress-susceptible fibres when the exercise was repeated, and therefore in theory, there would be less damage. This is supported by Friden et al. (1983) who observed disturbances in myofibrillar cross-striated band patterns, particularly in Z-line streaming. These investigators stated that only a small percentage of the total number of muscle fibres showed any signs of damage and that this number of fibres was greatly reduced following subsequent exercise bouts. However, if the number of stress-susceptible fibres decreased with time then the symptoms of DOMS should also subside with time. This is not necessarily the case as from anecdotal evidence sportsmen report that they continue to suffer from DOMS throughout their playing lives. This theory for

the explanation of the adaptation which occurs and lasts up to six weeks (Byrnes et al. 1985) is therefore questionable.

Jones & Newham (1985) also suggested that it was possible that changes in the recruitment pattern of muscle fibres could occur following training when sub-maximal contractions were performed, or alternatively that more motor units could be recruited in a second exercise bout thereby reducing the force per fibre, and hence the chance of injury. However, findings from subsequent research using maximal contractions contradicted these hypotheses (Newham et al. 1987) and are discussed in the previous chapter.

In the present study, therefore, it has been shown that stiffness, quantified in terms of RF2 and AM, does increase following eccentric exercise and reaches a peak 48 hours post exercise. At this time greatest pain is experienced, there is swelling in the exercised muscles and a decrease in the natural angle of flexion of the elbow joint. High levels of CK were also observed. Following a subsequent bout of the same exercise six days later there is no apparent increase in stiffness. An adaptation is seen to take place which not only results in reduced stiffness but in a reduced CK and pain response, indicating that the extent of the damage caused by the exercise is greatly reduced following the second exercise bout. The damage is thought to occur to the connective tissue, resulting in swelling in and around the elbow joint. The site of greatest pain is also in the tendons of the muscles around the elbow joint. The swelling is thought to influence the increase in muscle stiffness observed, but muscle stiffness may also be affected by the increase in extracellular and intracellular calcium levels described elsewhere (Clarkson & Tremblay 1988, Section 4.4).

CHAPTER 6

MUSCLE STIFFNESS, SORENESS AND PROPRIOCEPTION

6.1 INTRODUCTION

It has been noted by investigators that following eccentric exercise of the forearm flexors that has resulted in DOMS the natural angle of flexion of the elbow joint decreases (Jones et al. 1987, Clarkson & Tremblay 1988). This has also been noted in the previous study (Chapter 5). This is thought to occur as a result of shortening of the connective tissue (Howell et al. 1985, Jones et al. 1987, Newham et al. 1987). The range of motion that can be achieved by the elbow joint is reduced, as extension beyond this new angle of elbow flexion results in increased pain (Jones et al. 1987, Clarkson & Tremblay 1988, Chapter 5, present thesis). Performing routine tasks becomes difficult, firstly because of the discomfort, but secondly because there is a restriction in movement. Some investigators have reported that during the symptoms of DOMS in the forearm flexors it is often physically impossible to straighten the arm (Clarkson & Tremblay 1988). This reduction in function could impair sporting performance and in order to investigate this it is intended to compare one limb suffering from DOMS with the other in a rested state.

Normally, the limbs of the body can carry out symmetrical actions so that one side of the body will be a mirror image of the other side. For example, if an individual closes their eyes and is instructed to raise the arms out to the side then the height that each of the arms is raised will be very similar if not identical. Body awareness and memory allow the brain to recall reproducible images. Rowing, squash, volleyball, basketball, and tennis are examples of sports where the forearm flexors have an important role to play in carrying out desired movement patterns. In rowing and volleyball the arms often have to perform identical actions simultaneously. If the efficiency of one side of the body in carrying out a movement is altered then this will ultimately affect the level of skill. Equally, if both sides are impaired skill will deteriorate.

It is hypothesised that the ability to reproduce a "mirror image" is reduced when the state of the muscles on opposite sides of the body is different, that is, when the individual is suffering from DOMS in the forearm flexors of the non-dominant arm, with no simultaneous DOMS in the forearm flexors of the dominant arm. The study investigated a reduction in the angle of elbow flexion during DOMS, and whether a reduction in the range of motion, as a result of this, affected the ability to perform a simple perception test. The test examined individuals'

measuring ability pre- and post- exercise. The post-exercise test was performed when the symptoms of DOMS were thought to be at their peak.

6.2 METHODS

Subjects

Sixteen consenting adults (11 males, 5 females) mean age 24.7 + 3.7 years acted as subjects in this study. Subjects signed an informed consent document and visited the laboratory on two occasions, each separated by 48 hours.

Parameters Assessed and Experimental Protocol

Resting Joint Angle

On arrival at the laboratory, the resting flexion angle of the elbow joint of both arms was measured. Subjects either wore loose armless clothing, usually a running vest, or removed the clothing of the upper body, to permit the measurement of the elbow angle to take place. Joint centres of the shoulder, elbow and wrist joints were located, using the method of Dempster (1959) and marked with indelible ink. Subjects stood with the arms hanging loosely by the sides in a relaxed manner. The joint angles were measured using a clear plastic goniometer, the arms of which were long enough to ensure that the joint angle of even the longest human arm could be measured. The arms of the goniometer were marked along their central axis with a thin black line. The centre of the goniometer was held concentric with the centre of the elbow joint, and the goniometer arms were adjusted so that the black lines passed through the joint centres. The joint angle was measured three times on each arm, to the nearest degree and the average taken.

Muscle Soreness

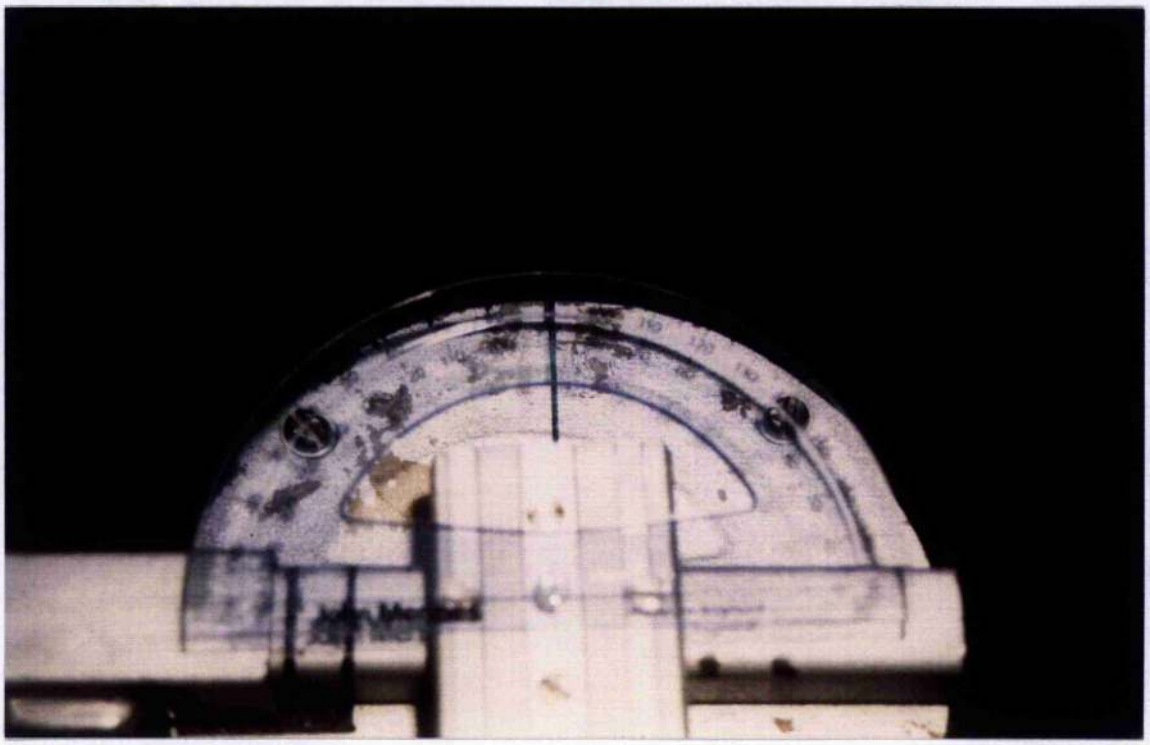
The method of assessing soreness is identical to that described previously in Section 3.3.2 of this thesis. The sites tested for soreness were also identical.

Perception Test

The equipment used to perform the perception test was a modified chair which had a support frame built around it. A metal support arm, upon which the subject's arm rested in a cradle, was attached to each side of the chair on the support frame. The support arm could be rotated about a central axis. The position of the support arm on the support frame could be altered vertically and horizontally in both planes to suit differing arm lengths. The design and fabric of the support frame was identical on both sides of the chair.

A 180° protractor was fixed concentrically with the central axis of each support arm so that the angle through which the support arm was moved could be measured (Figure 6.1).

Figure 6.1 The protractor attached to the support arm to measure the angle of elbow flexion during the perception test



The device was calibrated by drawing a straight line on the support frame through the central axis of each support arm. The protractor was then permanently fixed so that the line passed through 0° and 180° simultaneously. Readings had to be taken from the side, as the arm rested directly above the protractor and obscured the protractor scale. A thin piece of clear plastic was therefore attached to the support frame, vertically, and a thin arrowhead drawn upon it to make it easier to take a reading of the angle.

The positions of the support arm were altered so that they were identical on both sides of the chair. The subject then placed each arm in the cradle of the appropriate support arm. The cradles were moved until the arm positions were identical, with the elbow joints concentric with the central axes of the support arms. The support was raised such that the upper arm was in abduction forming a 90° angle with the body and the elbow was flexed at 90°. The arms were secured in the cradles using velcro straps. The subject was then blindfolded and the perception test began.

Experimental Protocol

The tester displaced the dominant arm so that it was flexed at one of six angles that had been decided prior to the commencement of the test. The angles chosen were representative of the full range of joint motion even when the symptoms of DOMS had manifested themselves. The subject was then instructed to bring the non-dominant arm to the position that he/she thought was the angle of elbow flexion identical to that of the dominant arm. The elbow flexion angle of the non-dominant arm was then noted. This matching procedure was carried out until all six angles of elbow flexion had been attempted. The experimental angles of elbow flexion were 30°, 60°, 80°, 100°, 130°, 150°. The order in which these angles were attempted was randomly assigned.

The purpose of the perception test was to assess whether subjects could copy the movement pattern and position of one arm with the contra-lateral limb and whether the symptoms of DOMS altered this judgement. The test was carried out before exercise and 48 hours later, when subjects were experiencing DOMS. Also, the angle of elbow flexion was measured and muscle soreness assessed on both occasions.

Exercise to Induce DOMS

Subjects performed a bout of eccentric exercise, designed to induce DOMS as described in Section 3.3.3, with the non-dominant arm.

Treatment of Results

A two-way (Site x Time) Analysis of Variance was performed on Soreness. The six discrepancies in the perception test (the difference between the actual angle and the attempted angle) were averaged and a students t - test performed on the pre- and post-exercise measurements. The three measures of resting elbow flexion angle were averaged and a students t - test performed on the pre- and post- exercise measurements.

6.3 RESULTS

Soreness had increased significantly ($p < 0.01$) by 48 hours after the exercise bout in five of the twelve sites tested. The sites exhibiting most pain were the three sites on the biceps and the upper sites of the flexor carpi radialis and the brachioradialis. The increases in pain are shown in Figure 6.2. Most pain was experienced around the elbow joint. The high standard deviation values give an indication of the range of pain thresholds between subjects.

Figure 6.2 Mean Soreness Ratings for all subject (n=16) at twelve sites on the arm pre- and post-exercise

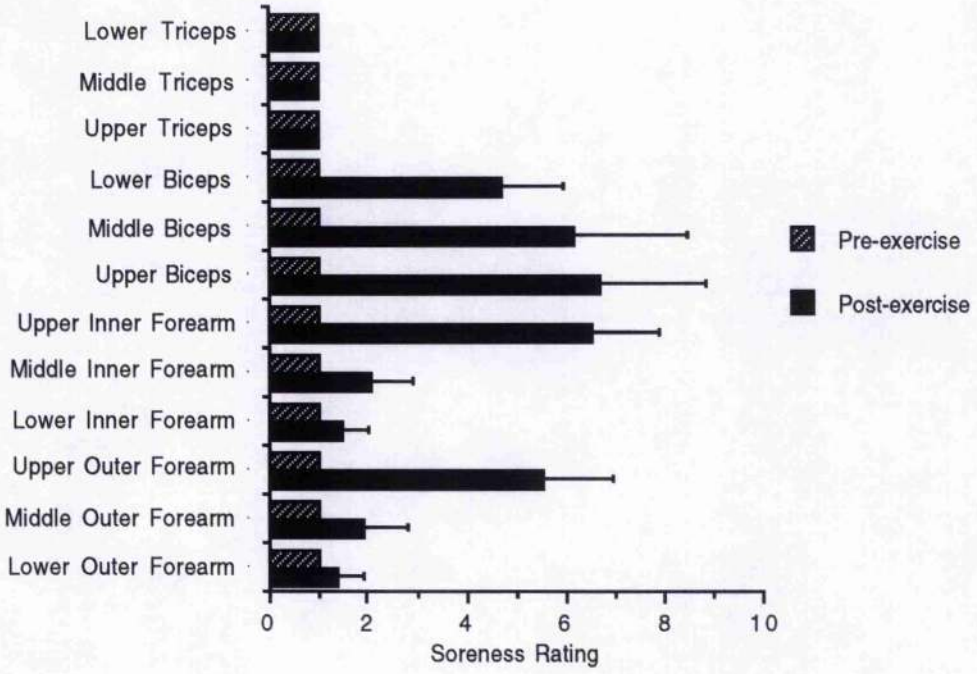


Figure 6.3 illustrates the pain experienced during passive flexion and extension of the forearm pre- exercise and at 48 hours post-exercise for all subjects. All subjects experienced more pain during passive extension than during passive flexion ($p < 0.01$)

The pre- and post-exercise measurements of the resting angle of elbow flexion in the exercised arm were significantly different ($p < 0.01$) and are displayed in Figure 6.4. The pre-exercise resting angle of elbow flexion ranged from 155° to 172°. Post exercise this range became 145° to 165°. The greatest decrease in elbow flexion angle occurred in subject 6, from 172° to 149°. Subjects 4, 12 and 15 showed little or no decrease in the elbow flexion angle. There was visible swelling around the elbow joint in some subjects. In the non-exercised arm there was no difference between the pre- and post-exercise measurement of resting angle of flexion.

The results of the perception test are shown in Figure 6.5. It had been expected that following the bout of exercise when the symptoms of DOMS had manifested themselves the ability to perform the perception test would have deteriorated. This was not the case. The discrepancies post exercise showed no differences from those observed pre-exercise. Some subjects performed the perception test with more success than others, that is, the difference between the

experimental angle and the matched angle was smaller for some subjects than for others. However, these subjects similarly showed no change in the size of the discrepancy between the experimental angle and the matched angle following exercise.

Figure 6.3 Mean Soreness Ratings during voluntary flexion and extension of the arm for all subjects pre- and post exercise

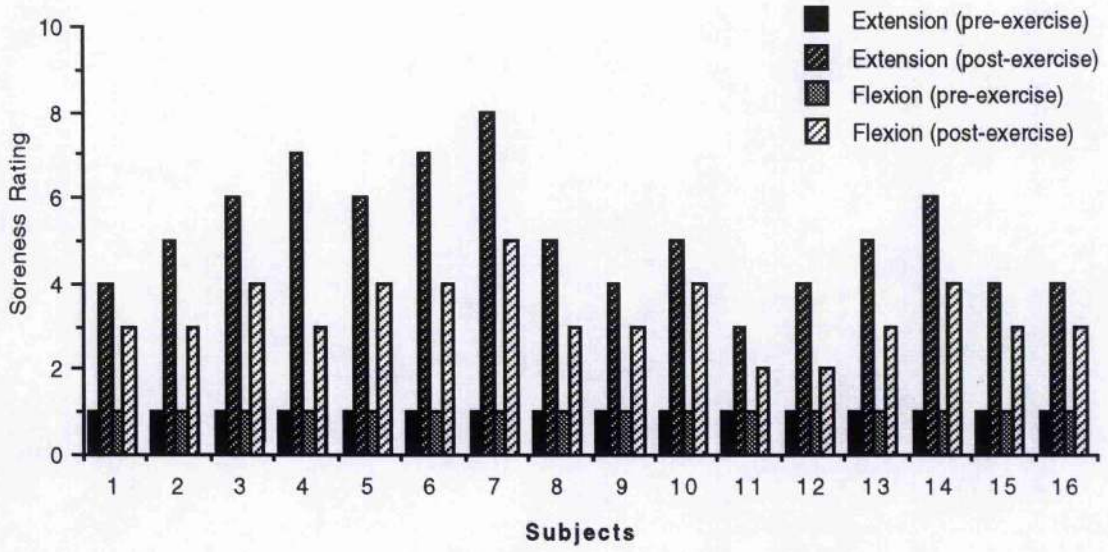


Figure 6.4 Mean resting angle of elbow flexion of the non-dominant arm for all subjects (n=16) pre- and post-exercise

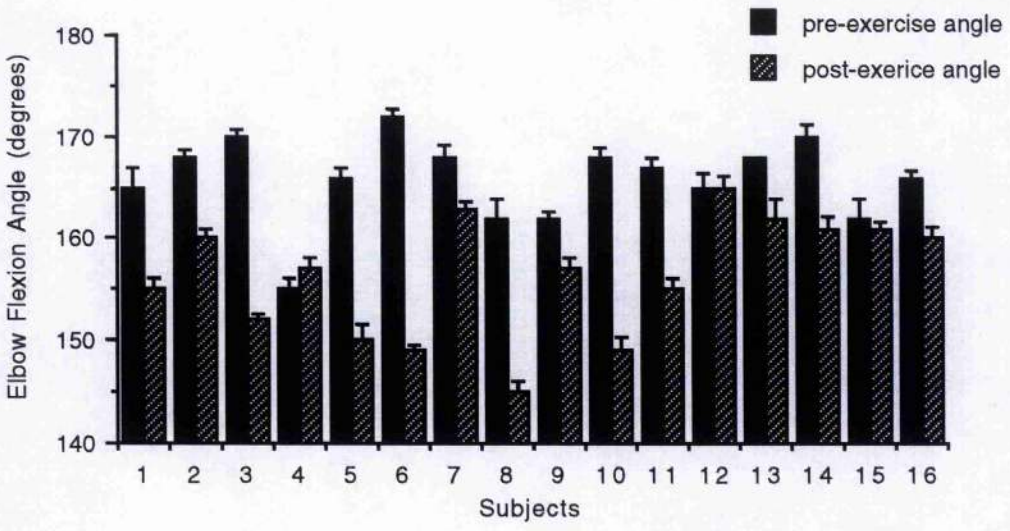
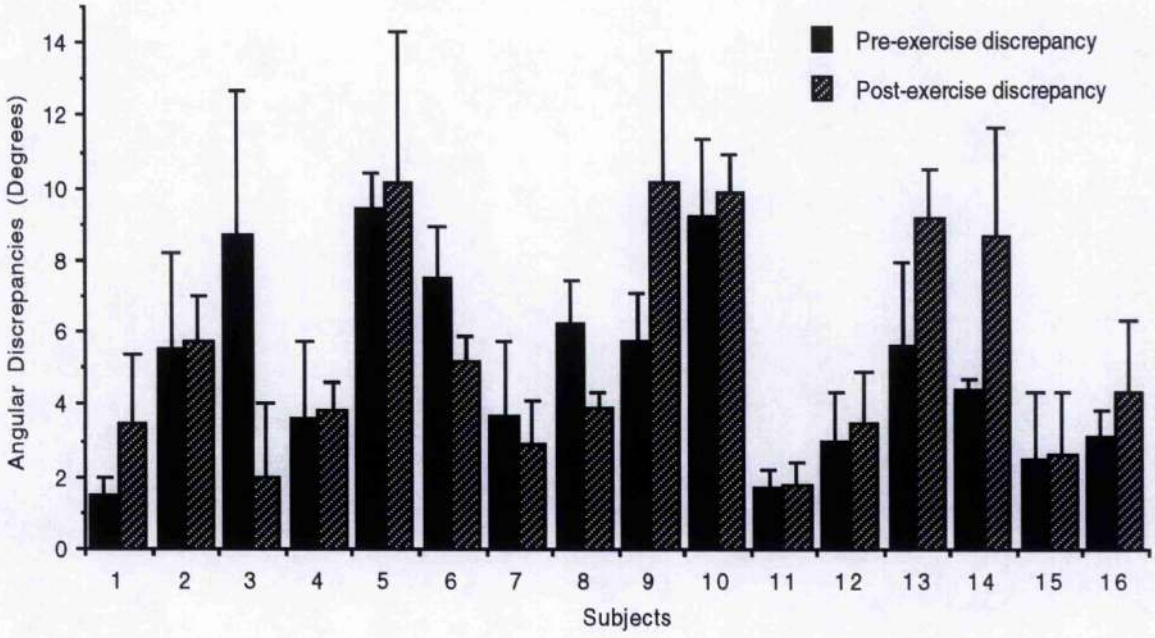


Figure 6.5 The mean discrepancies between the flexion angle of the dominant arm and the matched flexion angle of the non-dominant arm in all subjects (n=16) pre- and post exercise



6.4 DISCUSSION

DOMS was induced successfully using 70 maximal eccentric contractions. This exercise regimen has been utilised for the studies mentioned earlier in this thesis and the results are in keeping with earlier findings.

Previous research has suggested that during the stages of DOMS there may be a shortening of the connective and muscle tissue affected by the symptoms (Howell et al. 1985, Jones et al. 1987, Newham et al. 1987). This appeared to be the case in the present study as a decrease in the natural angle of elbow flexion was observed 48 hours after the exercise bout. The decrease was noticeably different from the subject's unexercised arm when the two were compared. One arm appeared "straight" and the other "bent" by comparison. Measurement of the angle confirmed that in some cases the angle had decreased by up to 23 degrees. Jones et al. 1987 noted a decrease of between 6 and 20 degrees post-exercise. Clarkson & Tremblay (1988) noted a mean decrease of 15 degrees at 48 hours post-exercise and that even by five days post exercise the angle had not returned to its original value.

Previous studies have shown that any attempt to exceed the new, temporary, post-exercise angle of elbow flexion results in greater pain being experienced (Clarkson & Tremblay 1988, Chapter 4 & Chapter 5, present thesis). It it also sometimes physically impossible to straighten the arm if the symptoms of DOMS are particularly severe. Several studies investigated the electrical activity in the muscle before and after DOMS had developed, and in particular examined the electrical activity in the arm when it rested at a new angle of flexion as mentioned above (Howell et al.1985, Jones et al. 1987, Chapter 5). These studies reported that no electrical activity was present at the new involuntary angle of elbow flexion observed post-exercise, even although the angle had decreased by up to 20 degrees in some cases. The investigators report retrospectively that prior to the exercise when the arm was voluntarily flexed from its resting angle of flexion to the same extent there was electrical activity. It would have been difficult in these studies to know the extent to which the exercised arm would flex involuntarily post-exercise, however, presumably the authors voluntarily flexed the unexercised arm through a range which would have included the angle of involuntary flexion that was observed post-exercise.

The suggestion that the connective tissue had shortened could explain the change in the elbow flexion angle (Jones et al. 1987). This hypothesis could also explain why there is greater pain during passive extension than during passive flexion since the connective tissue is relatively inelastic and any attempts to stretch it, as in extension of the forearm, could stimulate pain receptors to a greater extent. However, it is also feasible that the shortening of the muscle could be as a result of an increase in extracellular and/or intracellular calcium. Clarkson & Tremblay (1988) suggest the sequence of events (as discussed in Section 5.4), initiated by an abnormal increase of calcium in the cell, that could result in involuntary contraction. Brody (1969) also describes high levels of cellular calcium in a patient who exhibited muscle stiffness through electrically silent involuntary contractions.

Swelling around the damaged tissue, in particular around the joint, is also thought to affect muscle stiffness, as has been discussed in sections 4.4 and 5.4. Although unlikely, it has also been suggested that ATP depletion could result in increased muscle stiffness (Section 4.4).

The decrease in elbow angle was expected post-exercise. The range of joint motion achievable without an increase in pain was therefore reduced and it was intriguing to investigate whether this reduction in the range of motion would affect individuals in a sporting situation. It has been well documented that during the stages of DOMS there are decrements in strength (Talag 1973, Newham et al. 1987, Clarkson & Tremblay 1988, Dedrick & Clarkson 1990), which will undoubtedly affect performance, particularly in power sports, for example weightlifting, volleyball or sprinting. Dedrick & Clarkson (1990) investigated the effects of DOMS on

reaction time and movement time but found that these remained unaltered when compared to pre-exercise measurements.

An individual's ability to reproduce movement is dependent upon several cues: velocity, direction, terminal location, duration and extent of movement. In investigating individuals' ability to reproduce movement from memory, location (the position in space which the limb occupies) and distance (the extent through which the limb is moved) have received special attention from investigators. Most of this research has been carried out using some form of limb-positioning task. The task normally requires a blindfolded subject to move a device, for example, a movable handle along a trackway to a physical stop which indicates the end of the movement. This initial trial is termed the criterion movement, and following this the individual is asked to recall this movement in repeated trials after the stop is removed. Normally, the same limb is used for the criterion movement and the repeated movement. In the present study both the location and distance were examined and the task involved both limbs.

The symptoms of DOMS appeared to have no effect on the subjects' ability to perform the perception test. Some individuals performed the test better than others but their performance in the retest was unaltered. There was no angle that produced either a best or worst performance. The subjects' perception may have been altered in the range beyond the new resting elbow flexion angle observed post-exercise but the ability of subjects to extend the arm to angles beyond this range was limited by the subjects' ability to tolerate pain.

CHAPTER 7

MUSCLE STIFFNESS, SORENESS AND SPORTING PERFORMANCE

7.1 PRESENT RESEARCH AND IMPLICATIONS FOR PERFORMANCE

The results of the study in Chapter 4 suggested that that there was an increase in muscle stiffness following eccentric exercise. Since the findings of research into muscle stiffness are limited compared with the results obtained for soreness studies, it was unclear if this post-exercise increase in stiffness was "normal". The study seemed to support the previously held perceptions of individuals who claimed to be "stiff" as a result of unaccustomed physical exercise. It is important to note that the distinction was made between stiffness and soreness, as both were measured independently, since the two symptoms are often confused under the heading DOMS.

In the same study no differences were noted in the values obtained for stiffness in the ten pre-exercise controlled measurements. It was decided that in subsequent research, if subjects exhibited the ability to relax during the familiarisation sessions, it would only be necessary to record one controlled measurement for stiffness prior to the exercise bout being performed.

It was assumed that if individuals maintained the same behavioral pattern on a day-to-day basis, as happened when controlled measurements were being taken, then stiffness would not be affected. This could equally be true of sportsmen and sportswomen: if the elements in their training schedule remained unchanged then they should not suffer from muscle stiffness and soreness. However, if progress is to be made then the volume, intensity and sometimes frequency of training sessions has to increase. The extent of the increase in training load will determine the likelihood of the sportsman or sportswoman experiencing DOMS. It seems logical to progress training gradually. The threshold at which DOMS begins to manifest itself is unknown, since the effects of the condition are not known until days after the training has taken place. There is no clear cut method of prescribing exercise, to ensure that muscle stiffness and soreness does not manifest itself, other than to advise gradual progress.

This increased resistance to stretch in the muscle and connective tissue observed in the first study suggested that the physical state of the exercised tissues was altered. Physically, if any structure is altered, whether it is metal, brick, wood or human tissue, questions arise as to whether the structure is as robust as before and whether the structure is more susceptible to damage in its changed state. If muscles that are stiff as a result of unaccustomed exercise do not perform their function to the same extent as before then there will be an effect on the

activities for which this muscle is used. This is true of everyday household and work tasks, as well as sporting activity. However, unlike metal, brick or wood, human tissue has the ability to repair itself. Research suggests that following damage the muscle is repaired and strengthened in an attempt to ensure that further damage does not occur. This is also true when an individual has suffered from muscle soreness. There seems to be a protective effect that lasts for at least six weeks (Byrnes et al. 1985). When individuals repeat the exercise that has previously caused the muscle soreness there is no soreness.

This protective effect had not been noted with stiffness other than that subjects had reported that they did not "feel" stiff following a repeated bout of exercise. This formed the topic of investigation for the second study (Chapter 5). This study noted an increase in stiffness following exercise with a peak at 48 hours post-exercise. When the same amount of exercise was performed 6 days following the first bout of exercise there was no increase in stiffness. The results of the study have important implications for coaches and athletes. Following a bout of exercise that has resulted in DOMS an adaptation takes place which protects the individual from further insult if the same exercise is carried out in the weeks that follow. Exposing the athlete to lower intensities of exercise will also result in this adaptation (Clarkson & Tremblay, 1988). It is therefore important to introduce new elements into the training programme gradually so that DOMS can be minimised or avoided.

It is important to remember that the adaptation that occurs following DOMS is specific to a particular activity or movement. That is, if an individual suffers from DOMS following one type of exercise, this will not necessarily mean that the individual will not suffer from DOMS when another activity is attempted which involves different movements. For example, a highly trained sprinter who has been undergoing strenuous sprint, circuit and weight training without suffering from DOMS, will be prone to DOMS if he/she plays squash or badminton, because the movements involved in these games are different from the movements in sprinting. To avoid DOMS it is therefore useful to involve individuals in cross training which will expose them to different movement patterns and hence different ways in which the muscle is exercised, since, if individuals perform eccentric exercise that is not specific to their sport, for example an athlete playing a game of squash, DOMS will manifest itself. Training should include all of the movements that are likely to be experienced in that sport, e.g. road and cross country runners should train on a variety of uphill and downhill gradients, and not just relatively flat courses or tracks. This can only be of benefit to the performer as the days lost through training while suffering from muscle soreness and muscle stiffness will be greatly reduced.

In the second study while subjects were suffering from muscle stiffness and soreness there was a noticeable decrease in the resting angle of flexion of the arm. Extension of the arm from

this new angle resulted in increased pain. This in turn meant that there was a decrease in the range of pain free movement of the elbow joint. It was anticipated that there was some shortening of muscle or connective tissue, or both, to cause this decrease in the resting angle of flexion. It was thought that this shortening of the tissues may affect the perception of movement, which is an aspect of performance which is particularly important in sports where skill is an important factor. This led to the protocol of the third study (Chapter 6).

In wishing to examine perception, difficulties arose when searching for a test that would isolate the muscles affected by DOMS. It was also difficult to find a test that would mirror a realistic sporting situation involving the muscles being exercised and which would allow easy measurement of muscle stiffness. It was thought that if the perception was altered then this would affect the coordination and performance of an individual who was required to perform fine movements in their chosen sport. However, the results of the study in Chapter 6 may suggest that the brain is able to match the movement required even when the limb is stiff and sore. The brain probably has a motor memory which will recall the movement even when the arm is sore.

One interesting finding in studies of this subject matter is reported by Marshall et al. 1992. In a series of three well controlled experiments with independent subjects, these investigators examined the reliability of subjects in reproducing 10 subjectively defined steps of the same length (experiment 1), 10 subjectively defined linear arm movements of the same length (experiment 2) or 10 successive movements of a single experimenter defined linear arm movement (experiment 3). In each of these studies the repetitions of the movement became longer each time.

This systematic bias in the reproduction of movements is reported elsewhere in the literature concerning short-term motor memory (Adams & Dijkstra, 1966, Marshall 1972), and the reasons for the bias occurring can be justified. However, Marshall et al. 1992 report that this was an unexpected finding in the context of their experiments in that the lengthening was observed in three similar linear positioning tasks. They could not offer an explanation for this. They report that it is not uncommon to find shortening in reproduction attempts when the time between the the criterion movement and the repeated movement is increased for time intervals greater than thirty seconds. Similarly, any movements that are made before the criterion movement can influence the ability to reproduce the criterion movement. However, in their first two studies there was no experimenter defined criterion movement since the subject dictated the extent of movement. Also, the time between successive attempts at the linear positioning task was no more than 10 seconds.

The bias in other studies has been explained by "range effects", which imply that the range through which the subject has to operate can effect the reproducibility of the movement. This range is dictated by the experimenter. In the first two studies by Marshall et al. 1992 there was no experimenter defined movement since the subjects themselves chose the range within which they wished to operate to produce their movements. Therefore, the bias could not be explained by "range effects". Alternatively, the literature on the accuracy of memory between experimenter-defined and subject-defined movement, involving the "preselection paradigm" , can offer an explanation for the bias. In general, subjects are more accurate at reproducing a movement that they have defined themselves than at reproducing a movement that has been defined by the experimenter (Martenuik 1973, Stelmach et al. 1975). However in the series of experiments carried out by Marshall et al. 1992 there was bias (in the form of the increasing length of movements as the trials progressed) following both subject-defined and experimenter-defined movement. It may have been that there was indirect pressure imposed upon the subjects that took part in this study, and that the need to reproduce the movement may have been overstressed. As a result the subjects may have been tense, and being keen to perform well may not have been relaxed when performing the movement.

In the study in Chapter 6 it was impossible to note any bias as only one trial was performed at each of the selected arm positions, however, subjects did not exhibit any pattern of either constantly over- or under-shooting in trials at the six designated positions. The subject's ability to match the position of one arm with that of the other did not change throughout the period of the test nor did the ability to match the position improve during the second testing session. It may have been the case that any learning effect from the first testing session may have been cancelled out by the condition imposed upon the arm as a result of the eccentric exercise. However, it seems most likely that despite the change in the physical condition of the arm the brain still has the ability to override the condition to allow the matching to take place. This is useful in a sporting context as at times sportsmen and sportswomen have to compete when they are experiencing muscle stiffness and soreness. A tournament that lasts several days demands that a high standard of performance is maintained, and when skills are involved it is comforting to know that the movement involved in the skill seems to be recalled from memory.

It may be that the static nature of the test was not representative of a true sporting situation since any movement in sport tends to be of a more dynamic nature. The test itself looked at the ability of the brain to match the position of the dominant arm with the non-dominant arm. A more thorough investigation involving the perception test of the present study may reveal that motor performance is affected during DOMS. For example, subjects could be asked to perform a criterion movement and attempt to match this movement over several trials in a normal state. The performance could be measured in terms of the time to complete the

movement as well as matching the position. This could then be repeated when subjects were suffering from muscle stiffness and soreness to assess whether there is a change in the results.

This study was carried out to attempt to ascertain whether the ability to perform a simple skill was affected when subjects were suffering from DOMS, the reasoning being that if there is a decrement in the level of skill then the level of performance will also fall. However, if there had been a decrement in the performance of the perception test it would have been difficult to investigate whether sporting performance would be affected since many factors can affect performance. The results of the study suggest that performance of a simple perception test is not affected when suffering from muscle stiffness and soreness. It is impossible to state whether the ability to perform sporting skills would be affected as a result of DOMS.

Sportsmen and sportswomen expect to produce a similarly high performance even when they are suffering from DOMS. The remainder of this chapter outlines ways in which sporting performance is affected by muscle stiffness and soreness.

7.2 THE EFFECTS OF MUSCLE STIFFNESS AND SORENESS ON PERFORMANCE

It is perhaps pertinent at this stage to recall the tale of the individual who was completing a test on a cycle ergometer to assess maximal oxygen uptake. Having completed several increasingly difficult workloads on the cycle the subject eventually stopped pedalling. The tester thanked the individual for his time and effort and explained to the individual that the reason that he had stopped pedalling was because he had reached his maximal capacity for exercise. The subject, however, explained to the tester that the real reason for the cessation of the exercise was that the seat of the cycle ergometer was so uncomfortable that he couldn't suffer the pain any longer.

This seems to illustrate that there are many factors that can affect human sporting performance. Intrinsically, these factors can be physiologically, biomechanically or psychologically based. It is therefore extremely difficult to state that one particular factor can be responsible for a particularly poor or excellent performance, when several factors can be acting simultaneously to influence the performance. DOMS is one such factor, and the various ways in which performance could be affected are now discussed.

Researchers have assessed "pure" physiological performance and "applied" physiological performance during DOMS. Pure performance refers to individual physiological parameters outwith a sporting context, for example strength, flexibility, reaction time, all of which affect

performance. Applied physiological performance refers to, for example, oxygen consumption or force velocity characteristics of a muscle during a sporting activity, usually running. In the assessment of pure performance presumption is made on the basis that if these factors are affected in their own right then there must be a subsequent affect on performance where these factors are important. Difficulties arise when assessing applied sporting performance since the measured parameter may not be the only factor affecting performance.

Several investigators have shown that muscles experiencing exercise-induced muscle soreness have a reduced capacity to produce force. These strength decrements as a result of DOMS have been discussed previously in Section 2.6.1. Strength decrements could affect performance in sports in which strength and power are essential. Weightlifting, sprinting, volleyball and judo are examples of sports that fall into this category. Individuals competing in these sports cannot risk being put in a situation where they are suffering from DOMS. In simple terms the weightlifter cannot lift as much, the sprinter cannot accelerate as effectively from the starting blocks, the volleyball player cannot jump as high either to attack or defend at the net, and the judo player cannot throw his/her opponent as effectively when strength is reduced. Often the competitions for sporting events are held over several days and it is necessary to produce increasingly superior performances as the competition progresses. If DOMS manifests itself during a competition of this nature then the consequences as a result of decrements in strength could be severe.

During the stages of DOMS when individuals are suffering from stiffness and soreness it has been noted in the present research (Sections, 5.3 and 6.3) and by other investigators that there was a reduction in the resting angle of elbow flexion and a decrease in the range of both flexion and extension of the elbow. This has been discussed earlier (Section 6.4) and the effects of reduced flexibility on one particular aspect of performance examined (Chapter 6). This reduced flexibility did not seem to affect the subjects' ability to perform the perception test described in Chapter 6.

Reductions in flexibility could affect performance in two ways. The first is the disadvantage caused as a result of a reduced range of movement around a joint. The second is as a result of the increased pain experienced when an attempt is made to exceed the limits that have been imposed upon the range of motion of the joint. Flexibility is important in most sports as execution of the technique required for the particular sport is reliant upon the full range of movement being achieved. In tennis, for example, if there is reduced mobility of the shoulder joint, as a result of DOMS in the muscles of the upper back ,the service will be affected. Similarly if a hurdler is suffering form DOMS in the hamstrings that are tight then the technique crossing the hurdle will be affected. Also, in gymnastics flexibility is an important component.

Decrements in flexibility will affect the execution of these skills.

When flexibility is reduced while individuals are suffering from DOMS there is a greater risk of injury occurring. The pain experienced when performing stretching exercises during the preparatory phase prior to competition (warm up) can be severe and as a result poorly executed. Investigators have shown that stiffness can be reduced temporarily by passive and active movement (Lakie et al. 1984, Jones et al. 1987, Lakie & Robson 1988), but that the stiffness will return following cessation of activity. Anecdotal evidence also exists to suggest that activity leads to a reduction in muscle soreness, but like stiffness the symptom returns to run its normal time course. Although the pain may reduce it is debatable whether the full range of movement will have returned. The muscles could be inadequately prepared and possibly more susceptible to injury. The likelihood of this happening is increased in sports where there are fast limb movements, for example sprinting, jumping, racquet sports, leading to ballistic movements going beyond the normal range of movement.

Given that there are changes in many facets of performance as discussed above it is proposed that there may be changes in an individual's technique during the stages of DOMS. On account of the pain experienced during DOMS movement patterns could be altered in an attempt to avoid further discomfort. Hamill et al. 1991 investigated the biomechanical and physiological factors in runners suffering from DOMS and noted that the running style was altered following a downhill run that induced the soreness. Kinematic analysis revealed that during the support phase of the running action there were reductions in maximum knee and hip flexion suggesting that shock absorption was being compromised. These investigators also reported that to compensate for these reductions, there was an increase in ankle dorsiflexion. Modifications in movement patterns like those highlighted by Hamill et al. 1991 could result in a decrement in the standard of the technique, further injury and in extreme cases permanent damage.

Hamill et al (1989) investigated the effects of DOMS on oxygen consumption by carrying out a series of runs on a treadmill. These researchers hypothesised that the oxygen cost of running at the same speed would be altered when subjects were suffering from DOMS because of a decline in running economy. On day three of this five day study the subjects completed a downhill run to induce muscle soreness. The subjects then completed a run on the next two days. No elevation in oxygen consumption was observed after DOMS, and the investigators concluded that the damage that produced DOMS had not occurred in the contractile elements of the muscles. (If the damage had occurred in the elastic components, then the oxygen consumption would have increased). However, it is questionable whether the downhill run was effective in creating DOMS to the extent that damage was caused to the contractile component. The conclusions of the study are therefore questionable.

Several studies have shown that following exhaustive exercise there is a reduction in subjects' ability to react and move quickly (Klimovitch 1977, Stull & Kearney 1978), however, there has been limited investigation into the effects of DOMS on motor performance. Dedrick & Clarkson (1990) investigated the effect of DOMS on motor performance in elderly and young individuals. Both groups performed motor tasks to measure the reaction time and movement time using an apparatus consisting of two micro-switches that activated timers. The subject sat with the forearm and elbow resting on a table at shoulder level. The investigator activated a light stimulus that started the first timer. When the subject moved the hand off the micro-switch, the timer was stopped providing a measure of reaction time. The subject then moved the forearm in a horizontal arc across the table to hit the second switch located at a right angle to the first switch. When the second switch was hit, it stopped the second clock, providing a measure of movement time. Although the older subjects demonstrated a slower strength recovery following the eccentric exercise that had induced damage,there was no change in the reaction time and movement time either following the exercise or between the two groups.

In today's society success in sport can bring with it financial reward. The number of sports which are now professional is increasing, with rugby union the latest to relinquish its amateur status. An increasing number of sportsmen must perform to their optimum and avoid injury if their livelihood is to be preserved and their financial gain maximised. DOMS can be painful and debilitating and can cause injury that interrupts training. The potential loss of earnings, which could result from injury, possibly as a result of suffering from DOMS, may encourage coaches and sportsmen to progress their training programmes with care, attention and planning. Then the undesirable phenomenon of muscle stiffness and soreness can be avoided.

CHAPTER 8

SUMMARY & CONCLUSIONS

8.1 SUMMARY

This study examined the effect of eccentric exercise of the forearm flexors on muscle stiffness and muscle soreness. Changes in muscle tone were quantified in terms of the rate of oscillation squared (RF^2) at resonance or by determining the amplitude of movement (AM) in response to an applied torque. Muscle soreness was measured at twelve sites on the arm. A force of 25N was applied to each of the sites and subjects rated perceived pain on a scale of 1-16. Subjects also rated the soreness experienced during voluntary flexion and extension of the arm.

8.1.1 Summary: Study 1

RF^2 increased following exercise ($p<0.01$). AM for a specific torque was reduced following exercise and reached a minimum 42 hours post-exercise ($p<0.01$). Muscle soreness increased following exercise and reached a peak 42 hours post-exercise ($p<0.01$). Greatest soreness was in the biceps brachii and in the proximal ends of the brachioradialis and the flexor carpi radialis ($p<0.01$). Voluntary extension was more painful than voluntary flexion following eccentric exercise.

These changes may have been as a result of mechanical events within the muscle or as a result of voluntary stiffening by individuals to avoid further discomfort.

8.1.2 Summary: Study 2

This study monitored muscle stiffness, muscle soreness, limb girth, serum creatine kinase concentration and electromyography following two subsequent exercise bouts (EX1 and EX2), each separated by six days. RF^2 increased and AM decreased following EX1 and reached a maximum and minimum, respectively, 48 hours post exercise ($p<0.01$). Muscle soreness also reached a peak 48 hours post-exercise ($p<0.01$). Greatest pain was experienced around the elbow joint. Serum creatine kinase concentration increased following EX1 and reached a peak 4-5 days post-exercise, and limb girth reached a peak 72 hours post-exercise ($p<0.01$). During the measurement of muscle stiffness the biceps brachii was electrically silent. The resting angle of elbow flexion appeared to decrease following EX1.

Following EX2 an adaptation was observed. Each of the variables above showed a reduced response when compared to those following EX1. Statistically significant differences were observed between the values obtained 48 hours following EX1 and the values obtained 48 hours following EX2 ($p<0.01$). The adaptation was thought to take place in both the muscle and connective tissue.

8.1.3 Summary: Study 3

The effect of DOMS on proprioreception was investigated in this study.

Muscle soreness was assessed before exercise and 48 hours post-exercise. The resting angle of elbow flexion was assessed at the same time using a clear plastic goniometer. A perception test was employed to assess whether motor performance was impaired in a limb that exhibited involuntary shortening either of the muscle or connective tissue or both. The test involved positioning the non-dominant arm at an angle of elbow flexion equivalent to that of the dominant arm at six discrete angles.

Muscle soreness increased following the exercise ($p<0.01$). The ability to perform the perception test was not affected as a result of involuntary shortening of the forearm flexor muscles. It was thought that even although the physical state of the arm had changed the brain had pre-learned the task and could match the position of the arm by recalling the required motor performance form memory.

8.2 CONCLUSIONS

The investigations in this thesis succeeded in further enhancing the understanding of muscle stiffness and soreness following exercise. Muscle stiffness was quantified and seen to show a "real" increase following exercise to suggest that muscle stiffness following exercise is not merely a perception. It is important to note that, in the present study, muscle stiffness was not measured following concentric or isometric exercise. The changes in muscle stiffness that have been observed following eccentric muscular contractions may also be observed following concentric or isometric exercise. Further research is therefore required in this area, and the results of this thesis must be viewed in this context.

The time course of muscle stiffness was similar to that of pain with a peak 24-48 hours

post-exercise. Muscle stiffness exhibited the same adaptive qualities observed in previous research concerning muscle soreness. When a bout of exercise that originally created muscle stiffness was repeated six days later there was no increase in muscle stiffness. This was true for both the resonant frequency (squared) of the arm (RF^2) and the range through which the arm would move in response to a set peak torque (AM). Evidence suggests that the adaptation is activity-specific.

The effect of muscle soreness on proprioception was investigated. The ability to copy a series of movements and positions of the dominant arm with the non-dominant arm in its normal state was unchanged when the test was repeated with the non-dominant arm suffering from muscle soreness. The effect that eccentric exercise has on other aspects of performance, for example, flexibility, strength, psychology, technique have been investigated more thoroughly over the years.

The physiology behind the increase in muscle stiffness in the forearm flexors is thought to be as follows. Immediately following the eccentric exercise, the arm is in a weakened state. The eccentric exercise is thought to cause damage to the muscle cell membrane which leads to loss of sarcolemmal integrity allowing the contents to leak from within muscle cells. This leads to a build up of fluid which reaches a peak 48-72 hours post-exercise. This has the effect of altering the viscosity around the joint, and in effect makes it more difficult to perform flexion and extension of the forearm. This build up of fluid partly explains the increase in muscle stiffness.

The damage to the cell membrane is also thought to inhibit the process by which calcium is removed from the calcium-binding sites on troponin and pumped back into the sarcoplasmic reticulum, leading to an increase in intracellular calcium. Eccentric exercise is also thought to disrupt the integrity of the sarcoplasmic reticulum leading to an increase in the level of extracellular calcium. This build up of calcium is thought to cause involuntary contraction of the muscle. When the integrity of the cell membrane is restored, the process for the removal of calcium will function effectively once more. This, in effect, switches off the contraction.

Exercise in the form of passive flexion and extension of the elbow leads to fluid being squeezed out of the area surrounding the joint, and has the effect of decreasing the stiffness. Following cessation of movement the fluid returns to the area surrounding the affected joint and stiffness continues to run its normal time course. The eccentric exercise also creates damage to the connective tissue and the site of greatest damage is thought to be in and around

the musculo-tendinous junction. This relatively inelastic tissue is stretched during the eccentric contractions and as a result micro-tears appear in the tissue. In response to the damage there is thought to be a shortening of the connective tissue, observable by examining the resting angle of elbow flexion post-exercise. This is like a safety mechanism, to protect the arm from extending through its full range, where discomfort is greatest. This shortening of the connective tissue also partly explains the increase in muscle stiffness. The stiffness peaks 48 hours post exercise and in most cases has returned to its normal value six days following the exercise.

Following a bout of exercise that has created muscle stiffness it is thought that some strengthening of both the muscle and connective tissue takes place. This repair and over-compensation process can begin 1-3 days following the initial insult and endeavours to remove the damaged tissue and strengthen the remaining tissue. It is during this period that there is a gradual removal of the edema that has built up around the muscle. As a result, when eccentric exercise is repeated there is little or no damage to the cell membrane, therefore little or no leakage of intracellular proteins (in particular calcium), little or no build up of fluid and hence no increase in muscle stiffness. Similarly, the connective tissue is strengthened sufficiently to be able to cope with the same extent of exercise that has caused the damage in the first place. There is no observable change in the resting angle of elbow flexion to suggest that shortening of the connective tissue has taken place. It is unknown how long the complete adaptation process takes. Byrnes et al. (1985) have shown that the adaptation lasts for six weeks for muscle soreness however it is unknown how long the adaptation will last for muscle stiffness. Subsequent research may answer this question.

Several questions underlying the explanation of events that leads to muscle stiffness still remain unanswered and could be the subject of further research. The effect that muscle stiffness has on performance could be an area of further research if the variables affecting performance could be controlled sufficiently, to allow skill to be scrutinised and quantified. This would necessarily mean that a skill would have to be chosen involving the muscles that could be measured for muscle stiffness using the equipment of the present study. Alternatively, equipment could be built to measure muscle stiffness in other muscle groups.

The area of research is captivating because the symptoms have affected most people at one time. No treatment has managed to cure the symptoms of muscle stiffness and muscle soreness. Exercise seems to be the best measure to prevent these symptoms from manifesting themselves and should involve a gradual increase in the frequency, intensity and volume of

training across the full range of movement likely to be experienced by the sportsman or sportswoman. From the results of the third study (Chapter 6) it would seem that muscle stiffness and muscle soreness do not affect the ability to recall movement from memory.

It is in the best interests of Sportsmen and Sportswomen to try to avoid muscle stiffness and soreness. Apart from the discomfort experienced, muscle stiffness and soreness can cause unnecessary interruptions to training and may lead to injury. Muscle stiffness and soreness will also reduce performance.

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APPENDICES

APPENDIX A

MUSCLE SORENESS QUESTIONNAIRE

UNIVERSITY OF ST ANDREWS

Muscle Soreness Questionnaire

NAME _____ DATE _____

The purpose of this questionnaire is to assess the degree of muscle soreness after exercise

SORENESS SCALE

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Normal		Uncomfortable				Sore			Very Sore						

IN COLUMN X please rate the degree of soreness of different parts of the muscle when a force of 25 newtons is applied using the spring-loaded centre punch.

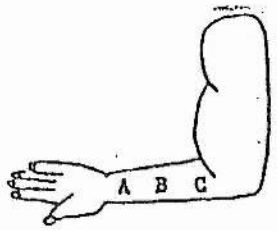
OUTER FOREARM

X

A_____

B_____

C_____



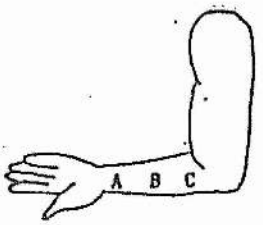
INNER FOREARM

X

A_____

B_____

C_____



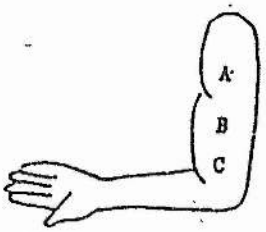
ARM BICEPS

X

A_____

B_____

C_____



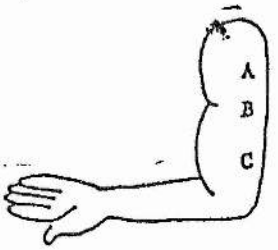
ARM TRICEPS

X

A_____

B_____

C_____



Please rate the general degree of soreness during forearm:

Flexion_____ Extension_____

APPENDIX B

ANOVA TABLES FOR STUDY ONE (CHAPTER 4)

Analysis of Variance for RESONANT FREQUENCY

Source	DF	SS	MS	F	P
TORQUE	4	0.17265	0.04316	1.68	0.166
SUBJ	15	20.60761	1.373849	12.74	0.000
TIME	19	2.53771	0.13356	7.27	0.000
TORQUE*SUBJ	60	1.53901	0.02565	17.04	0.000
SUBJ*TIME	285	5.23508	0.01837	12.20	0.000
TORQUE*TIME	76	0.17665	0.00232	1.54	0.003
Error	1140	1.71590	0.00151		
Total	1599	31.98461			

Analysis of Variance for AMPLITUDE OF MOVEMENT

Source	DF	SS	MS	F	P
TORQUE	4	183.8461	45.9615	274.35	0.000
SUBJ	15	58.7199	3.9147	234.51	0.000
TIME	19	7.1896	0.3784	2.97	0.000
SUBJ*TIME	285	36.2901	0.1273	7.63	0.000
TORQUE*SUBJ	60	10.0518	0.1675	10.04	0.000
TORQUE*TIME	76	2.1823	0.0287	1.72	0.000
Error	1140	19.0298	0.0167		
Total	1599	317.3096			

Analysis of Variance for SORENESS

Source	DF	SS	MS	F	P
SUBJ	15	754.863	50.324	74.44	0.000
DAY	10	1409.597	140.960	27.32	0.000
SITE	13	1983.187	152.553	23.53	0.000
SUBJ*DAY	150	773.936	5.160	7.63	0.000
DAY*SITE	130	946.960	7.284	10.78	0.000
SUBJ*SITE	195	1264.404	6.484	9.59	0.000
Error	1950	1318.235	0.676		
Total	2463	8451.182			

APPENDIX C

DATA FOR STUDY ONE
(CHAPTER 4)

		RESONANT FREQUENCY (SQUARED)					AMPLITUDE OF MOVEMENT					SORENESS					
												Inner forearm	Outer forearm	Biceps	Triceps	Flexion	Extension
	1	0.441	0.494	0.441	0.343	0.291	1.351	1.278	0.935	0.894	0.754						
	2	0.299	0.441	0.441	0.391	0.441	1.369	1.173	0.958	0.855	0.731						
	3	0.441	0.494	0.494	0.494	0.494	1.287	1.127	0.944	0.848	0.728						
	4	0.391	0.494	0.551	0.494	0.494	1.616	1.345	1.102	0.907	0.867						
	5	0.441	0.441	0.494	0.494	0.441	1.186	1.095	0.944	0.846	0.776						
	6	0.551	0.551	0.551	0.551	0.494	1.566	1.095	1.019	0.995	0.738						
	7	0.672	0.610	0.672	0.610	0.551	1.222	0.943	0.960	0.882	0.775						
	8	0.551	0.551	0.551	0.441	0.494	1.269	1.256	0.973	0.925	0.698						
	9	0.551	0.551	0.391	0.494	0.494	1.334	1.260	1.074	0.933	0.736						
pre-exercise	10	0.494	0.610	0.610	0.610	0.673	1.448	1.338	1.297	1.226	0.870	1	1	2	1	1	2	1 2 1	1	1 2	1	1
immed. after	11	0.610	0.673	0.673	0.673	0.610	1.583	1.282	1.236	0.674	0.653	1	1	2	1	1	2	2 1 1	1	1 1	1	1
18h	12	0.673	0.673	0.673	0.739	0.673	1.219	1.088	1.075	0.962	0.328	1	1	1	1	1	1	1 2 1	2	1 1	1	1
24h	13	0.673	0.673	0.610	0.673	0.673	1.569	1.320	0.722	0.697	0.412	1	1	2	1	1	1	2 2 1	2	2 1	1	2
42h	14	0.610	0.610	0.610	0.610	0.673	0.535	0.805	0.631	0.542	0.451	1	2	2	1	1	2	2 3 2	2	2 1	3	4
48h	15	0.610	0.610	0.610	0.610	0.610	1.949	1.224	0.855	0.648	0.492	1	1	2	1	1	2	1 1 2	2	2 1	3	4
66h	16	0.610	0.610	0.610	0.673	0.673	1.593	1.520	1.253	1.026	0.553	1	2	2	1	1	2	2 3 2	1	2 1	3	5
72h	17	0.673	0.673	0.673	0.610	0.610	1.568	1.383	1.158	1.047	0.919	1	2	3	1	1	2	2 4 3	1	2 2	3	6
90h	18	0.551	0.551	0.551	0.551	0.551	1.590	1.526	1.274	1.096	0.859	1	2	3	1	1	2	2 3 2	2	1 1	3	5
96h	19	0.610	0.610	0.610	0.610	0.610	1.653	1.594	1.173	1.019	0.524	1	1	2	1	1	1	2 1 1	1	1 1	2	4
120h	20	0.551	0.551	0.610	0.610	0.610	1.582	1.431	0.913	0.733	0.513	1	1	1	1	1	1	1 1 1	1	1 1	2	3

1	0.391	0.441	0.391	0.391	0.343	1.359	1.102	0.891	0.580	0.434						
2	0.494	0.441	0.441	0.441	0.441	1.378	1.134	0.861	0.733	0.516						
3	0.391	0.391	0.391	0.391	0.391	1.856	1.391	0.996	0.888	0.522						
4	0.343	0.391	0.391	0.391	0.391	1.329	1.268	0.972	0.889	0.618						
5	0.391	0.343	0.343	0.343	0.391	1.481	0.811	0.660	0.550	0.433						
6	0.391	0.391	0.391	0.391	0.391	1.531	1.037	0.873	0.762	0.522						
7	0.299	0.343	0.343	0.343	0.343	1.188	0.901	0.734	0.382	0.286						
8	0.391	0.391	0.391	0.391	0.391	1.350	1.174	0.761	0.642	0.477						
9	0.391	0.391	0.391	0.391	0.441	1.456	1.142	1.067	0.754	0.536						
10	0.391	0.391	0.391	0.391	0.441	1.397	1.159	0.883	0.742	0.506	1 1 1	1 1 1	1 1 1	1 1 1	1	1
11	0.441	0.391	0.441	0.441	0.441	0.971	0.536	0.638	0.478	0.400	1 1 1	1 1 1	1 1 1	1 1 1	1	1
12	0.441	0.441	0.441	0.441	0.441	1.001	0.845	0.653	0.533	0.328	1 2 4	1 2 2	7 8 9	2 3 3	3	3
13	0.441	0.441	0.441	0.441	0.391	1.067	1.002	0.720	0.513	0.326	1 3 5	1 2 5	8 10 9	2 2 2	6	4
14	0.494	0.494	0.441	0.441	0.441	0.961	0.828	0.525	0.350	0.226	2 2 4	1 1 4	3 8 9	2 3 3	5	7
15	0.391	0.391	0.391	0.391	0.343	1.246	1.072	0.756	0.738	0.405	2 3 6	2 2 7	5 8 9	2 3 3	6	8
16	0.441	0.441	0.391	0.391	0.391	1.436	1.045	0.750	0.636	0.389	1 2 4	1 1 6	3 5 7	1 1 1	3	6
17	0.391	0.391	0.391	0.391	0.391	1.190	1.071	0.734	0.646	0.457	1 1 3	1 1 5	6 6 8	2 2 1	3	6
18	0.391	0.391	0.391	0.391	0.391	1.656	1.359	1.250	0.934	0.656	1 1 3	1 1 4	3 3 5	1 1 1	2	4
19	0.391	0.391	0.391	0.391	0.391	1.474	1.299	0.956	0.672	0.438	1 1 2	1 1 4	3 5 4	2 2 2	2	4
20	0.391	0.391	0.391	0.391	0.391	1.418	1.289	1.013	0.685	0.420	1 1 1	1 1 2	2 3 2	1 1 1	1	3

0·494	0·494	0·551	0·441	0·391	1·791	1·227	0·962	0·772	0·623										
0·343	0·391	0·391	0·391	0·441	1·408	1·154	1·118	1·029	0·789										
0·391	0·391	0·441	0·441	0·441	1·987	1·595	1·089	1·047	0·756										
0·441	0·391	0·391	0·391	0·441	1·852	1·787	1·135	0·945	0·710										
0·441	0·441	0·441	0·391	0·391	1·514	1·349	0·936	0·835	0·697										
0·441	0·441	0·441	0·441	0·494	1·733	1·526	1·085	0·911	0·761										
0·494	0·441	0·441	0·494	0·494	1·827	1·133	1·089	0·720	0·679										
0·391	0·441	0·441	0·441	0·494	1·837	1·495	1·157	0·905	0·704										
0·494	0·441	0·441	0·441	0·441	2·032	1·801	1·298	0·957	0·879										
0·441	0·441	0·441	0·441	0·441	1·961	1·570	1·186	0·954	0·729	1	1	1	1	1	1	1	1	1	1
0·610	0·610	0·610	0·610	0·610	1·872	1·431	1·182	0·942	0·868	1	1	1	1	1	1	1	1	1	1
0·551	0·551	0·551	0·551	0·551	2·065	1·958	1·211	0·951	0·695	1	1	2	1	1	2	2	3	2	1	1	1	3	3
0·494	0·494	0·494	0·494	0·494	1·902	1·490	1·245	0·736	0·672	1	1	3	1	1	3	2	4	2	2	1	1	3	9
0·673	0·610	0·610	0·673	0·673	1·402	1·318	1·147	0·852	0·805	1	2	5	1	3	6	2	6	8	1	1	1	8	13
0·610	0·610	0·610	0·610	0·610	1·243	1·074	1·038	0·858	0·593	1	2	5	1	1	6	2	5	7	1	1	1	10	14
0·673	0·610	0·610	0·610	0·610	1·351	1·275	1·091	1·057	0·595	1	2	2	1	1	6	1	7	8	1	1	1	9	13
0·610	0·610	0·610	0·673	0·673	1·762	1·571	1·274	1·201	0·831	1	1	4	1	1	5	2	4	4	1	1	1	8	13
0·441	0·494	0·494	0·494	0·494	1·858	1·596	1·350	1·143	0·862	1	2	3	1	1	3	1	4	3	1	1	1	7	11
0·441	0·441	0·494	0·494	0·494	1·872	1·286	1·161	0·843	0·804	1	2	3	1	1	2	2	3	4	1	1	1	6	9
0·441	0·441	0·441	0·441	0·494	1·982	1·646	1·220	1·024	0·774	1	1	1	1	1	1	1	1	1	1	1	1	3	7

1	0.343	0.343	0.391	0.391	0.391	1.976	1.482	1.287	1.160	0.889								
2	0.441	0.391	0.391	0.391	0.441	1.875	1.539	1.230	0.970	0.828								
3	0.391	0.391	0.391	0.391	0.391	2.173	1.907	1.501	0.956	0.852								
4	0.391	0.441	0.441	0.441	0.441	2.038	1.536	1.299	0.970	0.873								
5	0.441	0.441	0.441	0.391	0.391	2.006	1.929	1.313	1.092	0.866								
6	0.391	0.441	0.391	0.391	0.391	2.122	1.743	1.402	0.825	0.472								
7	0.494	0.494	0.494	0.441	0.494	1.992	1.556	1.256	0974	0.453								
8	0.441	0.441	0.494	0.494	0.551	2.009	1.353	1.181	0.899	0.895								
9	0.441	0.441	0.441	0.441	0.441	2.098	1 613	1 170	0.885	0.654								
10	0.494	0.494	0.551	0.551	0.551	1.997	1.321	1.148	1.040	0.681	1	1	1 1 1	1 1 1	1 1 1	1 1 1	1	1
11	0.494	0.494	0.441	0.441	0.441	1 992	1 656	1.120	1.048	0.828	1	1 1	1 1 1	1 1 1	1 1 1	1 1 1	1	1
12	0.441	0.441	0.441	0.441	0.494	1.458	1 273	1 177	0.802	0 447	3	4 7	2 5 7	4 5 5	3 2 2	5	5	
13	0.494	0.494	0 494	0.494	0.441	1.774	1.532	0.839	0.485	0.233	3	4 8	2 4 5	5 4 4	2 3 2	3	5	
14	0.441	0.391	0.391	0.391	0.391	1 497	1 335	1 049	0.555	0.409	4	6 7	2 2 5	3 3 3	2 2 2	5	7	
15	0.441	0.441	0.441	0.441	0.441	2132	1.936	1.617	1.174	0.683	2	3 8	2 2 7	7 6 5	4 2 2	5	8	
16	0.494	0 441	0.441	0.391	0.391	2.063	1.887	1.099	0.968	0 694	2	3 6	1 2 5	3 4 2	2 1 2	4	5	
17	0.441	0 441	0 391	0 391	0.343	2.070	1 466	1225	0.772	0.771	2	2 6	1 2 4	2 3 3	1 2 1	2	3	
18	0 441	0 441	0 341	0.391	0 391	2.087	1.472	1 336	0 989	0 683	1	2 4	1 2 3	1 2 2	2 1 2	2	2	
19	0.391	0.441	0 391	0 391	0.343	2.142	1 805	1 418	1.256	0.989	1	2 3	1 2 4	1 2 1	2 1 1	2	1	
20	0.391	0 391	0.391	0 391	0.391	2.096	1 729	1.411	1 219	0.931	1	1 2	1 1 2	1 1 1	1 1 1	1	1	

1	0.391	0.391	0.391	0.391	0.391	1.205	1.001	0	0.471	0.418						
2	0.391	0.391	0.391	0.391	0.391	1.400	1.063	0.857	0.592	0.434						
3	0.391	0.343	0.343	0.343	0.343	1.082	0.79	0.550	0.293	0.242						
4	0.441	0.441	0.441	0.441	0.441	0.784	0.634	0.453	0.259	0.138						
5	0.343	0.343	0.343	0.441	0.391	1.347	1.154	0.909	0.648	0.517						
6	0.343	0.343	0.343	0.343	0.391	1.144	1.054	0.603	0.427	0.329						
7	0.343	0.343	0.343	0.343	0.391	1.042	0.884	0.762	0.422	0.330						
8	0.391	0.494	0.391	0.391	0.494	1.059	1.008	0.947	0.445	0.472						
9	0.391	0.391	0.391	0.391	0.391	1.715	1.500	1.061	0.821	0.556						
10	0.391	0.391	0.391	0.391	0.391	1.517	1.480	0.953	0.745	0.409	1 1 1.	1 1 1...	1 1. 1	1 1 1	1	1
11	0.551	0.551	0.610	0.551	0.673	1.117	0.780	0.683	0.445	0.209	1 1 2	1 1 2	3 3 2	1 1 1	1	1
12	0.441	0.441	0.494	0.494	0.551	0.970	0.765	0.464	0.351	0.271	1 1 2	1 1 3	2 2 3	1 1 1	5	5
13	0.441	0.441	0.441	0.441	0.441	0.735	0.613	0.416	0.210	0.104	1 2 4	1 1 2	3 2 4	1 1 1	5	4
14	0.441	0.441	0.441	0.441	0.441	1.202	0.777	0.441	0.339	0.269	1 1 3	1 1 3	2 2 3	1 1 1	4	4
15	0.441	0.461	0.441	0.461	0.441	0.988	0.848	0.482	0.365	0.263	1 1 3	1 1 3	1 2 4	1 1 1	2	3
16	0.441	0.441	0.441	0.441	0.441	1.388	0.968	0.611	0.495	0.256	1 1 2	1 1 2	1 1 2	1 1 1	2	2
17	0.391	0.391	0.391	0.441	0.441	1.245	0.754	0.457	0.304	0.149	1 1 2	1 2 2	1 2 2	1 1 1	2	3
18	0.441	0.441	0.391	0.391	0.441	1.144	0.879	0.545	0.368	0.19	1 1 1	1 1 2	1 1 1	1 1 1	1	2
19	0.391	0.391	0.441	0.441	0.441	1.314	0.973	0.513	0.403	0.281	1 1 1	1 1 2	1 1 1	1 1 1	1	2
20	0.391	0.441	0.391	0.391	0.441	1.085	0.789	0.515	0.427	0.374	1 1 1	1 1 2	1 1 1	1 1 1	1	2

1	0.391	0.343	0.343	0.343	0.343	1.485	1.269	1.121	0.852	0.711						
2	0.391	0.343	0.343	0.299	0.343	1.783	1.262	1.132	0.859	0.706						
3	0.343	0.391	0.343	0.391	0.343	1.709	1.507	0.992	0.861	0.731						
4	0.391	0.391	0.391	0.391	0.343	1.742	1.432	1.189	0.912	0.722						
5	0.391	0.343	0.343	0.391	0.343	1.650	1.227	1.028	0.887	0.676						
6	0.391	0.391	0.391	0.391	0.391	1.485	1.024	0.907	0.752	0.663						
7	0.343	0.391	0.391	0.391	0.343	1.742	1.467	1.131	1.105	0.912						
8	0.391	0.391	0.343	0.391	0.391	2.037	1.868	1.578	1.424	1.123						
9	0.343	0.343	0.391	0.343	0.343	1.825	1.329	1.093	0.820	0.692						
10	0.391	0.391	0.391	0.391	0.343	1.880	1.671	1.401	1.285	1.039	1 1 2	1 1 1	3 1 3	2 1 2	1	1
11	0.551	0.494	0.551	0.494	0.494	2.052	1.562	1.236	1.103	0.932	1 1 1	1 1 1	1 1 1	1 1 2	1	1
12	0.494	0.494	0.441	0.391	0.441	1.915	1.617	1.220	0.976	0.867	1 1 3	1 2 4	1 3 1	2 1 1	3	1
13	0.494	0.551	0.494	0.494	0.494	1.923	1.578	1.258	1.131	1.027	1 1 4	1 1 5	1 5 4	1 1 1	4	5
14	0.494	0.494	0.441	0.494	0.441	1.890	1.487	1.263	1.186	0.965	1 2 6	1 3 7	2 7 6	1 1 1	4	7
15	0.494	0.551	0.494	0.494	0.494	1.901	1.553	1.305	1.156	1.042	1 3 6	1 1 8	1 7 7	1 2 2	7	9
16	0.494	0.494	0.441	0.441	0.494	1.821	1.609	1.116	0.860	0.706	1 3 5	1 1 6	1 5 6	2 1 1	4	6
17	0.441	0.441	0.391	0.391	0.391	1.621	1.302	1.103	0.942	0.762	1 3 5	1 1 5	1 4 5	1 1 1	3	6
18	0.494	0.494	0.494	0.494	0.494	1.589	1.306	1.096	1.058	0.755	1 1 3	1 1 3	1 1 2	1 1 1	2	4
19	0.441	0.441	0.441	0.494	0.441	1.911	1.821	1.426	1.265	1.170	1 1 2	1 1 3	1 1 2	1 1 1	2	4
20	0.494	0.441	0.441	0.441	0.494	2.140	1.553	1.399	1.137	1.054	1 1 1	1 1 2	1 1 1	1 1 1	1	2

1	0·391	0·391	0·441	0·551	0·954	1·672	0·868	0·642	0·452	0·373								
2	0·391	0·391	0·441	0·494	0·61	1·836	0973	0·851	0·667	0·453								
3	0·391	0·391	0·391	0·494	0·610	1·756	1·132	0·811	0·551	0·381								
4	0·441	0·551	0·551	0·610	0·807	1·376	0·868	0·673	0·321	0·291								
5	0·441	0·391	0·494	0·551	0·738	1·716	1·582	0·694	0·309	0·297								
6	0·441	0·441	0·551	0·610	0·954	1·789	1·728	0·772	0·257	0·217								
7	0·391	0·394	0·441	0·551	0·807	1·977	1·101	0·638	0·445	0·300								
8	0·494	0·441	0·610	0·610	0·673	1·628	1·187	0·839	0·344	0·273								
9	0·441	0·494	0·551	0·673	0·954	1·791	1·248	0·607	0·448	0·155								
10	0·610	0·551	0·673	0·807	0·954	1·770	1·188	0·695	0·375	0·100	1	1	1	1	1	1	1	1	1	1
11	0·441	0·494	0·610	0·610	0·879	2·203	1·739	0·733	0·337	0·197	1	1	1	1	1	1	1	1	1	1
12	0·551	0·610	0·673	0·807	0·879	1·256	1·042	0·442	0·283	0·152	1	1	2	1	1	4	6	7	5	1	1	1	3	6
13	0·441	0·494	0·551	0·673	1·031	0·963	0·710	0·376	0·388	0·051	1	1	4	1	1	4	5	6	8	1	1	1	5	7
14	0·551	0·551	0·673	0·673	0·807	0·884	0·621	0·378	0·144	0·071	1	1	7	1	1	8	9	9	10	1	1	1	8	10
15	0·441	0·441	0·551	0·610	0·673	1·078	0·486	0·292	0·172	0·077	1	1	6	1	1	7	8	9	11	1	1	1	8	11
16	0·391	0·441	0·441	0·441	0·494	0·696	0·596	0·249	0·172	0·134	1	1	4	1	1	2	6	6	6	1	1	1	8	11
17	0·391	0·391	0·494	0·551	0·551	1·044	0·925	0·310	0·173	0·167	1	1	4	1	1	2	5	5	6	1	1	1	6	8
18	0·441	0·391	0·441	0·494	0·807	1·216	0·938	0·651	0·451	0·395	1	1	2	1	1	1	2	3	4	1	1	1	3	4
19	0·391	0·441	0·441	0·610	0·954	1·745	0·960	0·668	0·387	0·295	1	1	2	1	1	1	2	3	3	1	1	1	3	4
20	0·391	0·391	0·441	0·494	0·673	1·735	1·145	0·816	0·451	0·372	1	1	7	1	1	1	2	2	3	1	1	1	2	3

7)

1	0.441	0.441	0.494	0.551	0.551	1.408	1.095	0.748	0.521	0.219							
2	0.441	0.441	0.441	0.494	0.551	1.317	1.162	0.757	0.475	0.353							
3	0.441	0.441	0.441	0.441	0.441	1.344	1.215	0.757	0.524	0.465							
4	0.391	0.441	0.441	0.494	0.441	1.063	1.021	1.000	0.665	0.532							
5	0.441	0.441	0.441	0.494	0.494	1.749	1.461	1.053	0.789	0.396							
6	0.441	0.494	0.441	0.441	0.391	1.062	0.708	0.304	0.195	0.169							
7	0.391	0.391	0.391	0.494	0.494	1.262	1.000	0.484	0.292	0.234							
8	0.391	0.391	0.391	0.391	0.494	1.269	0.740	0.356	0.239	0.218							
9	0.391	0.391	0.391	0.441	0.441	1.355	1.158	1.042	0.505	0.364							
10	0.391	0.391	0.441	0.441	0.494	1.482	1.203	0.783	0.541	0.376	122	111	111	222	1	1	
11	0.673	0.673	0.551	0.551	0.551	1.807	1.394	1.018	0.702	0.541	224	223	224	322	2	2	
12	0.494	0.441	0.441	0.494	0.494	1.342	0.987	0.503	0.392	0.337	348	235	358	344	6	6	
13	0.551	0.494	0.494	0.551	0.551	1.062	0.978	0.875	0.729	0.625	238	247	457	344	5	7	
14	0.551	0.494	0.494	0.494	0.551	1.352	0.887	0.637	0.502	0.409	2511	239	378	233	7	6	
15	0.551	0.551	0.610	0.551	0.494	1.658	1.068	1.043	0.725	0.314	236	245	375	233	6	7	
16	0.441	0.441	0.494	0.441	0.494	1.617	1.246	1.145	1.010	0.70	234	233	243	442	6	6	
17	0.494	0.441	0.441	0.494	0.494	1.514	1.317	0.876	0.632	0.415	234	233	243	222	5	5	
18	0.494	0.441	0.441	0.441	0.441	1.754	1.450	0.944	0.715	0.541	234	223	232	223	4	3	
19	0.441	0.441	0.441	0.494	0.494	1.329	1.006	0.913	0.672	0.413	223	122	222	222	3	2	
20	0.441	0.494	0.441	0.441	0.494	1.516	1.213	0.762	0.501	0.397	122	112	122	222	2	2	

(5)

1	0.494	0.494	0.494	0.551	0.673	1.734	0.937	0.719	0.504	0.249						
2	0.441	0.441	0.441	0.441	0.441	1.346	0.947	0.693	0.508	0.320						
3	0.610	0.610	0.551	0.673	0.610	1.526	0.990	0.676	0.396	0.339						
4	0.551	0.494	0.494	0.551	0.551	1.658	1.047	0.741	0.325	0.225						
5	0.494	0.441	0.441	0.494	0.494	1.684	1.066	0.858	0.381	0.250						
6	0.610	0.494	0.551	0.551	0.551	1.637	1.466	0.826	0.387	0.140						
7	0.610	0.610	0.610	0.551	0.738	1.803	1.696	0.904	0.618	0.130						
8	0.610	0.610	0.494	0.494	0.441	1.294	1.156	0.988	0.523	0.320						
9	0.494	0.494	0.391	0.610	0.673	1.978	1.356	0.531	0.244	0.111						
10	0.610	0.673	0.610	0.673	0.738	1.870	1.358	0.662	0.391	0.200	1 1 1	1 1 1	1 1 1	1 1 1	1	1
11	0.673	0.551	0.551	0.551	0.610	2.073	1.552	1.000	0.570	0.442	1 1 1	1 1 1	1 1 1	1 1 1	1	1
12	0.610	0.738	0.673	0.738	0.738	1.277	1.081	0.935	0.865	0.408	1 1 2	1 1 2	3 2 2	1 1 1	1	2
13	0.738	0.673	0.610	0.610	0.738	1.305	1.174	1.049	0.572	0.242	1 1 3	1 1 1	2 3 2	2 1 1	1	2
14	0.807	0.807	0.807	0.738	0.738	1.732	1.073	0.836	0.581	0.341	1 1 4	1 1 1	3 4 3	1 1 1	2	3
15	0.807	0.807	0.738	0.738	0.673	1.415	0.820	0.440	0.531	0.206	1 2 5	1 1 3	3 5 4	1 1 1	3	5
16	0.807	0.673	0.673	0.807	0.673	0.878	0.717	0.462	0.362	0.269	1 1 4	1 1 2	3 4 4	1 1 1	3	5
17	0.807	0.807	0.673	0.738	0.879	1.259	1.092	0.905	0.587	0.240	1 1 4	1 1 2	3 4 4	1 1 1	3	5
18	0.738	0.807	0.879	0.879	0.879	1.555	1.117	0.764	0.395	0.304	1 1 2	1 1 1	2 3 2	1 1 1	2	2
19	0.738	0.738	0.738	0.807	0.879	1.638	1.052	0.916	0.535	0.296	1 1 1	1 1 1	1 2 1	1 1 1	2	3
20	0.551	0.738	0.494	0.610	0.807	2.006	1.329	1.100	0.576	0.326	1 1 1	1 1 1	1 2 1	1 1 1	1	2

(16)

1	0·807	0·879	0·9766	0·879	0·879	1·425	1·209	0·943	0·733	0·610						
2	0·807	0·879	0·879	0·879	0·954	1·767	1·696	1·280	0·675	0·584						
3	0·879	0·807	0·807	0·807	0·807	1·976	1·462	0·827	0·566	0·421						
4	0·673	0·673	0·673	0·673	0·673	1·769	1·482	1·381	0·712	0·464						
5	0·551	0·551	0·494	0·494	0·494	1·576	1·482	1·378	1·216	0·586						
6	0·610	0·494	0·494	0·494	0·610	1·448	0·925	0·567	0·547	0·360						
7	0·673	0·673	0·673	0·738	0·673	1·408	1·363	1·085	0·777	0·736						
8	0·807	0·879	0·879	0·879	0·879	1·886	1·161	0·881	0·740	0·584						
9	0·807	0·879	0·879	0·879	0·879	1·348	1·144	1·051	0·814	0·582						
10	0·673	0·673	0·738	0·738	0·807	1·679	1·089	0·771	0·699	0·530	1 1 1	1 1 1	1 1 1	1 1 1	1	1
11	0·738	0·738	0·879	0·738	0·879	1·089	0·757	0·646	0·418	0·349	1 1 1	1 1 1	1 1 1	1 1 1	1	1
12	1·031	0·954	1·031	1·112	1·196	0·848	0·481	0·357	0·345	0·176	1 1 3	1 1 4	2 3 3	1 1 1	2	3
13	1·031	0·954	0·954	1·112	0·954	1·329	0·753	0·536	0·380	0·258	1 1 3	1 1 3	2 3 3	1 1 1	5	7
14	1·112	0·879	0·807	0·738	0·807	0·793	0·755	0·557	0·466	0·214	1 1 5	1 1 5	2 4 4	1 1 1	3	7
15	0·738	0·807	0·738	0·673	0·673	0·843	0·546	0·422	0·342	0·167	1 2 6	1 1 6	4 5 6	1 1 1	4	7
16	0·738	0·954	0·807	0·807	0·807	0·834	0·727	0·515	0·274	0·298	1 2 5	1 1 5	2 5 6	1 1 1	5	5
17	0·879	0·879	0·807	0·807	0·807	0·780	0·681	0·538	0·315	0·122	1 2 5	1 1 5	2 4 5	1 1 1	4	5
18	0·807	0·879	0·738	0·807	0·807	0·952	0·615	0·484	0·352	0·154	1 1 4	1 1 4	2 3 4	1 1 1	3	5
19	0·807	0·807	0·807	0·879	0·879	1·472	0·819	0·755	0·527	0·433	1 1 4	1 1 3	2 3 3	1 1 1	2	4
20	0·807	0·807	0·807	0·879	0·954	1·381	1·103	0·815	0·732	0·541	1 1 3	1 1 2	2 2 2	1 1 1	2	3

(11)

1	0·391	0·391	0·391	0·494	0·494	1·436	1·149	0·693	0·366	0·253						
2	0·391	0·391	0·391	0·441	0·494	1·482	1·064	0·863	0·584	0·521						
3	0·343	0·343	0·391	0·494	0·391	1·846	1·626	1·780	1·221	0·774						
4	0·343	0·343	0·391	0·391	0·391	1·203	1·096	1·047	0·930	0·686						
5	0·343	0·391	0·441	0·391	0·391	1·456	1·394	1·095	0·987	0·742						
6	0·299	0·391	0·441	0·391	0·391	1·521	1·375	1·278	1·146	0·695						
7	0·441	0·441	0·441	494	0·441	1·563	1·211	1·083	0·762	0·785						
8	0·391	0·391	0·441	0·391	0·441	1·936	1·757	1·573	1·438	1·136						
9	0·391	0·391	0·391	0·391	0·441	1·494	1·294	0·879	0·831	0·604						
10	0·391	0·391	0·441	0·441	0·441	1·837	1·812	1·124	1·039	0·944	1 1 1	1 1 1	2 2 2	1 1 1	1	1
11	0494	0·441	0·391	0·441	0·441	1·834	1·728	1·562	1·413	1·220	1 1 2	1 1 2	222	1 1 1	1	2
12	0·391	0·441	0·441	0·441	0·441	1 589	1·434	1 282	1·158	1·027	1 1 2	1 1 2	789	1 1 1	1	2
13	0·441	0 441	0·441	0·391	0·441	1·948	1·840	1·794	1 550	1·276	2 2 4	1 1 2	3 4 10	1 1 1	1	5
14	0·441	0·441	0·391	0·391	0·441	1·666	1·533	1·343	1·309	1 032	1 1 3	1 1 4	4 6 6	1 1 1	1	4
15	0·391	0·441	0·441	0·441	0·391	1·292	1 090	1 032	0·833	0·792	1 2 4	1 1 4	5 5 7	1 1 1	1	4
16	0·391	0·391	0·391	0·441	0·441	1·741	1·595	1·418	1 224	1 122	1 1 3	1 1 3	2 4 5	1 1 1	1	2
17	0·391	0·391	0·391	0·391	0 391	1·883	1·691	1 100	1·108	0·957	1 1 3	1 1 2	4 5 6	1 1 1	1	2
18	0·391	0 343	0·391	0·391	0 391	1·791	1·587	1 223	1 124	1 030	1 1 2	1 1 2	3 2 3	1 1 1	1	1
19	0 391	0·391	0·391	0·391	0 391	1 829	1 523	1 281	1 130	1·037	1 1 3	1 1 3	3 2 3	1 1 1	1	1
20	0·391	0·391	0·391	0·391	0 391	1·732	1·406	1·119	0·963	0 832	1 1 2	1 1 2	2 1 2	1 1 1	1	1

0.441	0.391	0.343	0.391	0.494	1.295	1.265	0.965	0.733	0.706							
0.441	0.441	0.494	0.551	0.551	1.356	1.104	1.035	0.861	0.758							
0.441	0.494	0.551	0.494	0.494	0.926	0.897	0.912	0.841	0.710							
0.494	0.551	0.391	0.494	0.494	1.151	1.032	0.893	0.804	0.779							
0.441	0.494	0.391	0.391	0.343	1.296	1.026	0.935	0.870	0.739							
0.441	0.343	0.441	0.343	0.343	1.222	1.148	1.148	0.870	0.709							
0.343	0.391	0.343	0.343	0.343	1.261	1.246	0.966	0.796	0.794							
0.391	0.391	0.441	0.391	0.441	1.119	0.970	0.922	0.827	0.672							
0.391	0.391	0.391	0.441	0.441	1.188	0.947	0.797	0.824	0.713							
0.391	0.391	0.391	0.441	0.441	1.171	0.973	0.908	0.705	0.680	1	1	1	1	1	1	1
0.343	0.343	0.343	0.391	0.391	1.221	1.026	0.958	0.786	0.736	1	1	1	1	1	1	1
0.391	0.391	0.391	0.391	0.441	1.106	0.958	0.899	0.811	0.646	1	2	2	1	2	1	2
0.391	0.391	0.441	0.441	0.441	0.947	0.882	0.805	0.737	0.630	1	1	2	2	2	3	3
0.441	0.494	0.441	0.441	0.494	0.919	0.882	0.784	0.686	0.617	2	3	3	2	2	3	4
0.441	0.441	0.441	0.441	0.494	0.867	0.932	0.895	0.676	0.671	2	2	3	2	2	3	4
0.494	0.494	0.441	0.441	0.494	1.157	0.934	0.895	0.728	0.700	1	2	2	1	1	2	3
0.494	0.494	0.494	0.551	0.551	1.271	1.131	1.009	0.938	0.672	1	2	1	1	2	2	2
0.494	0.494	0.494	0.494	0.551	1.218	1.071	0.998	0.892	0.652	1	1	1	1	1	1	1
0.494	0.494	0.551	0.494	0.551	1.392	1.103	1.042	0.925	0.671	1	1	1	1	1	1	1
0.494	0.494	0.441	0.494	0.494	1.292	1.051	1.023	0.902	0.652	1	1	1	1	1	1	1

1	0.343	0.299	0.299	0.256	0.256	1.717	1.442	1038	0.606	0.595							
2	0.343	0.299	0.299	0.256	0.256	1.815	1.409	1.053	0.861	0.584							
3	0.343	0.299	0.256	0.256	0.256	1.680	1.190	0.641	0.613	0.310							
4	0.343	0.299	0.256	0.256	0.220	2.013	1.66	1.176	0.921	0.512							
5	0.391	0.343	0.343	0.299	0.299	1.397	0.95	0.764	0.448	0.231							
6	0.343	0.299	0.256	0.256	0.256	2.030	1.401	0.977	0.629	0.459							
7	0.343	0.299	0.256	0.256	0.256	1.202	0.673	0.626	0.448	0.362							
8	0.343	0.299	0.299	0.256	0.256	1.537	1.401	0.978	0.629	0.459							
9	0.343	0.299	0.299	0.299	0.220	1.789	1.271	1.010	0.749	0.450							
10	0.299	0.256	0.299	0.256	0.256	1.208	1.038	0.919	0.648	0.432	1 1 2	1 1 2	2 1 1	1 1 1	1	1	
11	0.391	0.391	0.391	0.256	0.391	1138	0.854	0.736	0.564	0.408	1 1 2	1 1 1	1 1 1	1 2 2	1	1	
12	0.343	0.343	0.299	0.299	0.299	1.635	1.219	1.023	0.742	0.528	1 1 2	1 1 2	2 2 2	1 1 1	2	1	
13	0.343	0.343	0.299	0.299	0.299	1.479	1.098	0.835	0.728	0.487	1 1 2	1 1 2	2 3 2	1 1 1	2	1	
14	0.343	0.299	0.299	0.299	0.299	1.333	1.022	0.771	0.531	0.505	1 1 1	1 1 2	2 4 2	1 1 1	2	2	
15	0.343	0.299	0.299	0.299	0.256	1.880	1.292	1.083	0.784	0.462	1 1 2	1 1 2	2 4 2	1 2 1	2	3	
16	0.343	0.343	0.299	0.299	0.299	1.630	1.290	1.197	0.797	0.553	1 1 2	1 1 2	3 3 2	1 1 1	3	2	
17	0.343	0.343	0.343	0.343	0.299	1499	1.222	0.976	0.763	0.494	1 1 2	1 1 2	2 2 2	1 1 1	3	2	
18	0.343	0.343	0.343	0.299	0.343	1.552	1.251	0.932	0.774	0.512	1 1 1	1 1 1	1 1 3	1 1 1	2	1	
19	0.391	0.391	0.391	0.391	0.343	1.180	0.875	0.709	0.667	0.477	1 1 1	1 1 1	1 1 1	1 1 1	1	1	
20	0.343	0.299	0.299	0.256	0.256	2.033	1.545	1.301	0.920	0.589	1 1 1	1 1 1	1 1 1	1 1 1	1	1	

1	0.391	0.391	0.441	0.441	0.494	1.897	1.221	1.048	0.940	0.581						
2	0.391	0.391	0.391	0.441	0.494	1.765	1.109	0.996	0.853	0.584						
3	0.391	0.391	0.391	0.441	0.441	1.998	1.531	1.101	0.785	0.564						
4	0.441	0.441	0.441	0.494	0.441	1.829	1.465	1.165	0.837	0.334						
5	0.391	0.391	0.441	0.441	0.494	1.867	1.328	1.178	1.060	0.798						
6	0.391	0.391	0.441	0.441	0.494	1.757	1.493	1.103	0.807	0.782						
7	0.343	0.391	0.391	0.441	0.441	1.922	1.634	1.320	0.752	0.327						
8	0.391	0.391	0.441	0.441	0.494	1.909	1.235	1.018	0.799	0.759						
9	0.441	0.441	0.494	0.494	0.441	1.857	1.593	1.320	0.790	0.882						
10	0.391	0.441	0.441	0.494	0.494	1.799	1.438	1.091	0.691	0.661	1 1 1	1 1 1	1 1 1	1 1 1	7	1
11	0.343	0.391	0.391	0.441	0.441	1.901	1.412	0.963	0.871	0.678	1 1 1	1 1 1	1 1 1	1 1 1	1	1
12	0.391	0.391	0.391	0.441	0.441	1.638	1.201	0.831	0.715	0.532	1 2 3	1 2 3	3 3 3	1 1 1	3	4
13	0.441	0.441	0.441	0.441	0.441	1.701	1.162	0.765	0.613	0.554	1 3 3	1 4 4	3 4 4	1 1 1	3	5
14	0.441	0.494	0.494	0.494	0.551	1.591	1.002	0.742	0.580	0.487	1 5 7	1 5 5	5 6 8	1 1 1	5	8
15	0.441	0.441	0.494	0.551	0.494	1.601	1.242	0.777	0.641	0.493	1 5 8	1 5 6	5 6 9	1 1 1	5	8
16	0.441	0.441	0.441	0.494	0.441	1.845	1.472	1.013	0.951	0.610	1 3 6	1 3 4	2 3 5	1 1 1	4	7
17	0.441	0.391	0.441	0.441	0.494	1.987	1.327	1.263	0.898	0.572	1 2 6	1 3 3	2 4 5	1 1 1	3	7
18	0.391	0.391	0.441	0.391	0.441	1.924	1.750	1.381	1.165	0.898	1 2 3	1 2 2	2 3 3	1 1 1	2	5
19	0.391	0.441	0.391	0.391	0.441	1.996	1.692	1.311	1.191	0.813	1 2 1	1 2 2	2 2 2	1 1 1	2	3
20	0.441	0.391	0.441	0.441	0.441	1.851	1.663	1.102	0.865	0.571	1 1 2	1 1 2	2 2 2	1 1 1	2	2

1	0·391	0·391	0·441	0·441	0·391	1·534	1·217	0·964	0·721	0·535										
2	0·343	0·391	0 391	0·3 91	0 ·441	1·621	1·281	0·983	0·743	0·601										
3	0·441	0·441	0 441	0·441	0·494	1·609	1·311	0·997	0·822	0·694										
4	0·343	0·441	0·494	0·441	0·494	1·522	1·204	0·931	0·685	0·517										
5	0·391	0·391	0·441	0·441	0·391	1·551	1·267	0·935	0·672	0·532										
6	0·494	0·494	0·494	0·494	0·551	1·447	1·237	0·886	0·532	0·499										
7	0·391	0·343	0·391	0·441	0·441	1·566	1·283	1·017	0·752	0·598										
8	0·441	0·441	0·494	0·494	0·494	1·611	1·252	0·951	0·786	0·612										
9	0·494	0·494	0·441	0·494	0·494	1·597	1·186	1·093	0·744	0·603										
10	0·391	0·441	0·441	0·494	0·494	1·562	0·993	0·884	0·683	0·507	1 1 1	1 1 1	1 1 1	1 1 1	1	1				
11	0·551	0·551	0·610	0·610	0·673	1·789	1·247	0·967	0·792	0·634	1 1 1	1 1 1	1 1 1	1 1 1	1	1				
12	0·551	0·551	0·551	0·610	0·673	1·621	0·995	0·838	0·688	0·451	1 2 3	1 2 4	3 3 3	1 1 1	2	3				
13	0·610	0·610	0·673	0·673	0·738	1·598	0·921	0·792	0·617	0·432	1 2 4	1 2 4	3 3 4	1 1 1	2	4				
14	0·610	0·673	0·673	0·673	0·738	1·442	0·886	0·655	0·542	0·387	1 4 6	1 4 7	5 6 8	1 1 1	3	6				
15	0·551	0·551	0·610	0·673	0·610	1·334	0·892	0·627	0·598	0·382	1 4 6	1 5 7	5 6 8	1 1 1	3	6				
16	0·551	0·551	0·551	0·610	0·673	1·447	0·935	0·845	0·775	0·461	1 3 5	1 3 7	3 4 6	1 1 1	3	5				
17	0·610	0·551	0·551	0·610	0·610	1·503	1·111	0·876	0·774	0·653	1 3 5	1 3 7	3 4 6	1 1 1	3	5				
18	0·494	0·494	0·551	0·551	0·610	1·621	1·205	0·911	0·747	0·662	1 2 3	1 3 5	2 4 5	1 1 1	2	4				
19	0·494	0·494	0·494	0·551	0·551	1·615	1·238	0·946	0·693	0·529	1 1 2	1 2 3	2 4 4	1 1 1	2	4				
20	0·551	0·494	0·551	0·610	0·610	1·634	1·284	0·924	0·722	0·601	1 1 2	1 1 2	2 3 3	1 1 1	1	3				

1	0.494	0.551	0.494	0.391	0.494	1.359	1.294	0.739	0.466	0.335														
2	0.391	0.441	0.494	0.441	0.494	1.528	1.146	0.936	0.648	0.612														
3	0.494	0.551	0.551	0.551	0.551	1.418	0.953	0.812	0.551	0.407														
4	0.441	0.551	0.610	0.551	0.551	1.612	1.457	1.387	0.864	0.759														
5	0.494	0.494	0.494	0.551	0.610	1.549	1.349	0.997	0.913	0.740														
6	0.551	0.551	0.551	0.610	0.610	1.430	1.269	0.883	0.572	0.540														
7	0.551	0.551	0.610	0.551	0.610	1.622	1.108	0.855	0.632	0.561														
8	0.494	0.494	0.494	0.551	0.610	1.573	0.962	0.753	0.495	0.387														
9	0.494	0.551	0.494	0.551	0.551	1.641	1.225	0.910	0.693	0.666														
10	0.494	0.551	0.551	0.610	0.610	1.653	1.122	0.831	0.602	0.486	1	1	1	1	1	1	1	1	1	1	1	1	1	1
11	0.673	0.673	0.673	0.673	0.673	1.770	1.187	0.921	0.795	0.721	1	1	1	1	1	1	1	1	1	1	1	1	1	1
12	0.673	0.610	0.673	0.673	0.738	1.693	0.995	0.792	0.665	0.527	1	1	2	1	1	3	5	6	5	1	1	1	2	5
13	0.673	0.673	0.673	0.738	0.673	1.541	1.113	0.815	0.672	0.512	1	1	2	1	1	3	5	6	6	1	1	1	4	6
14	0.673	0.673	0.673	0.673	0.738	1.370	0.881	0.652	0.557	0.410	1	1	4	1	1	6	5	7	8	1	1	1	6	8
15	0.610	0.610	0.673	0.610	0.673	1.32	0.791	0.557	0.413	0.382	1	1	5	1	1	6	5	6	10	1	1	1	6	8
16	0.610	0.610	0.673	0.610	0.610	1.453	0.926	0.731	0.661	0.459	1	1	4	1	1	6	5	6	8	1	1	1	5	8
17	0.551	0.551	0.610	0.673	0.610	1.410	0.887	0.751	0.592	0.461	1	1	4	1	1	5	3	5	7	1	1	1	4	8
18	0.551	0.551	0.551	0.610	0.610	1.731	1.112	0.910	0.738	0.681	1	1	2	1	1	4	2	4	6	1	1	1	2	5
19	0.551	0.610	0.551	0.610	0.610	1.654	1.210	0.85	0.673	0.552	1	1	2	1	1	4	2	4	6	1	1	1	2	5
20	0.551	0.494	0.551	0.551	0.551	1.724	1.183	0.872	0.569	0.491	1	1	1	1	1	3	1	3	3	1	1	1	1	3

APPENDIX D

ANOVA TABLES FOR STUDY TWO (CHAPTER 5)

Analysis of Variance Table for RESONANT FREQUENCY (squared)

Source	DF	SS	MS	F	P
SUBJ	15	23.28313	1.55221	928.11	0.000
DAY	11	2.02093	0.18372	10.78	0.000
TORQUE	4	0.05322	0.01330	0.97	0.429
SUBJ*DAY	165	2.81232	0.01704	10.19	0.000
DAY*TORQUE	44	0.09289	0.00211	1.26	0.124
SUBJ*TORQUE	60	0.82011	0.01367	8.17	0.000
Error	660	1.10381	0.00167		
Total	959	30.18641			

Analysis of Variance Table for AMPLITUDE OF MOVEMENT

Source	DF	SS	MS	F	P
SUBJ	15	11.6147	0.7743	51.84	0.000
DAY	11	2.6630	0.2421	5.56	0.000
TORQUE	4	101.5777	25.3944	403.88	0.000
SUBJ*DAY	165	7.1880	0.0436	2.92	0.000
DAY*TORQUE	44	0.7521	0.0171	1.14	0.246
SUBJ*TORQUE	60	3.7726	0.0629	4.21	0.000
Error	660	9.8577	0.0149		
Total	959	137.4258			

Analysis of Variance Table for SORENESS

Source	DF	SS	MS	F	P
SUBJ	15	860.488	57.366	51.71	0.000
DAY	11	254.485	23.135	1.88	0.046
SITE	13	1966.796	151.292	25.71	0.000
SUBJ*DAY	165	2035.360	12.336	11.12	0.000
DAY*SITE	143	185.213	1.295	1.17	0.100
SUBJ*SITE	195	1147.501	5.885	5.30	0.000
Error	2145	2379.775	1.109		
Total	2687	8829.619			

Analysis of Variance Table for LIMB GIRTH

Source	DF	SS	MS	F	P
Between Subjects	15	836.596	55.773	121.288	0.001
Within Subjects	176	80.932	0.46		
Treatments	11	34.499	3.5	13.61	0.001
Residual	165	42.432	0.257		
Total	191	917.528			

APPENDIX E

PAGE'S L TREND TEST

PAGE'S L TREND TEST
(Holliday & Cohen 1984, p226-228)

This test is appropriate when the researcher is looking at trends, when, as a result of previous theories, it is possible to predict the ordering of results under the experimental treatments.

In the case of the present study the null hypothesis is that the peak level of serum creatine kinase is not related to the time between performing eccentric exercise and taking the sample.

Page's L is given by: $L = \sum (t_c \times c)$

where t_c = the rank totals for each column

c = the numbers allotted to the conditions from left to right

To compute L the procedure is as follows:-

1) The conditions are arranged in the order predicted.

(In this case the predicted order is as follows:-
Day 5, Day 4, Day 6, Day 7, Day 8, Day 9, Day 10, Day 3, Day 11, Day 2, Day 12, Day 1.

That is, the highest level of CK is expected on Day 5 of the study, the second highest value is expected on Day 4, etc.)

2. Rank the scores across each row separately

3. Sum the ranks for each column

4. Substitute into the formula $L = \sum(t_c \times c)$

In this case:-

$L = (28.5 \times 1) + (43 \times 2) + (64 \times 3) + (87 \times 4) + (70.5 \times 5) + (105.5 \times 6) + (124 \times 7) +$
$(105 \times 8) + (136 \times 9) + (145.5 \times 10) + (162.5 \times 11) + (171 \times 12)$

$L = 9865.5$

(See tables in following pages for the ranked scores)

5. Consult statistical tables to determine significance level

Page's L is significant at the 0.01 level so the null hypothesis is rejected. There is therefore a **trend** in the direction predicted.

Subjects	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8	Day9	Day10	Day11	Day12
A	53	103	324	1434	1489	1667	547	355	188	167	77	55
B	70	68	90	153	383	273	150	160	111	70	59	63
C	103	68	82	104	58	51	57	72	64	62	67	58
D	84	342	342	424	1093	1012	892	765	410	232	135	82
E	61	68	68	77	78	62	63	68	61	64	72	59
F	137	131	454	2045	3172	1785	827	916	561	311	277	274
G	226	205	783	6589	7990	4804	3694	2135	1052	779	332	243
H	55	205	1220	18005	13488	13076	8606	6630	3382	1613	642	320
I	96	171	3531	5176	6892	8353	4263	2134	1949	497	543	467
J	55	153	78	352	472	56	63	132	223	160	111	67
K	117	199	5601	7653	13511	5926	4523	2949	1668	849	975	843
L	96	111	104	1183	977	1052	418	398	341	167	171	234
M	176	143	752	3061	4850	2517	1230	1434	780	421	383	314
N	69	125	175	184	324	254	113	175	106	115	36	79
O	150	273	307	3690	3826	2733	1362	820	409	403	763	352
P	62	96	199	78	1012	970	349	258	150	119	68	59

Subjects	1 Ranks (Day5)	2 Ranks (Day4)	3 Ranks (Day6)	4 Ranks (Day7)	5 Ranks (Day8)	6 Ranks (Day9)	7 Ranks (Day10)	8 Ranks (Day3)	9 Ranks (Day11)	10 Ranks (Day2)	11 Ranks (Day12)	12 Ranks (Day1)
A	1	2	3	4	5	7	8	6	10	9	11	12
B	1	4	2	5	3	6	8·5	7	12	10	11	8·5
C	9·5	1	12	11	4	7	8	3	6	5	9·5	2
D	1	5	2	3	4	6	9	7·5	10	7·5	12	11
E	1	2	9	8	5	10·5	7	5	3	5	12	10·5
F	1	2	3	5	4	6	8	7	9	12	10	11
G	1	2	3	4	5	6	8	7	9	12	10	11
H	2	1	3	4	5	6	7	8	9	11	10	12
I	2	3	1	4	6	7	9	5	8	11	10	12
J	1	2	11	10	6	3	4	8	7	5	9	12
K	1	2	3	5	6	7	9	4	8	11	10	12
L	3	1	2	4	5	6	9	11	8	10	7	12
M	1	2	3	5	4	6	8	7	9	12	10	11
N	1	3	2	8	4·5	9	7	4·5	12	6	10	11
O	1	2	3	4	5	7	8	10	6	11	9	12
P	1	9	2	3	4	6	7	5	10	8	12	11
Rank Totals	28·5	43	64	87	70·5	105·5	124	105	136	145·5	162·5	171

APPENDIX F

DATA FOR STUDY TWO
(CHAPTER 5)

	RESONANT FREQUENCY (SQUARED)					AMPLITUDE OF MOVEMENT							
	Torque 1	Torque 2	Torque 3	Torque 4	Torque 5	Torque 1	Torque 2	Torque 3	Torque 4	Torque 5	Total Soreness / Flexion + Extension Soreness	Limb Girth	Creatine Kinase
1	0·34	0·30	0·34	0·34	0·39	1·06	0·83	0·82	0·75	0·57	12	28·5	53
											1,1		
2	0·55	0·49	0·49	0·44	0·39	0·82	0·65	0·57	0·44	0·39	101	28·7	103
											14,14		
3	0·39	0·44	0·39	0·44	0·39	1·23	0·93	0·69	0·63	0·53	107	30·0	324
											16,16		
4	0·44	0·44	0·49	0·39	0·49	1·42	1·06	0·89	0·76	0·58	75	31·0	1434
											14,16		
5	0·50	0·44	0·44	0·50	0·50	1·36	1·05	0·98	0·72	0·54	18	30·5	1489
											4,14		
6	0·34	0·39	0·44	0·49	0·49	1·39	1·03	0·84	0·67	0·52	14	30·0	1667
											2,14		
7	0·34	0·39	0·44	0·44	0·44	1·38	1·01	0·90	0·70	0·52	12	30·0	547
											1,8		
8	0·44	0·44	0·44	0·39	0·44	1·26	1·00	0·83	0·72	0·47	12	30·2	355
											1,4		
9	0·34	0·30	0·30	0·30	0·30	1·32	1·05	0·90	0·76	0·49	12	30·0	188
											2,2		
10	0·34	0·34	0·39	0·39	0·39	1·33	0·96	0·87	0·68	0·57	12	29·5	167
											1,2		
11	0·34	0·34	0·39	0·39	0·34	1·30	0·94	0·88	0·71	0·52	12	28·9	77
											1,1		
12	0·34	0·34	0·34	0·34	0·39	1·34	1·00	0·83	0·72	0·54	12	29·0	55
											1,1		

1	0·34	0·34	0·30	0 34	0·34	1·10	0·84	0·54	0·53	0·37	12	24·7	70
											1, 1		
2	0·391	0·44	0·39	0·39	0·39	0·75	0·73	0·60	0·38	1·0	53	25·0	68
											6, 8		
3	0·44	0·44	0·44	0·39	0·39	0·59	0·51	0·44	0·38	0·25	50	25·0	96
											6, 8		
4	0·39	0·34	0·34	0·34	0·34	1·45	1·02	0·62	0·41	0·33	30	25·6	163
											4, 6		
5	0·39	0·39	0·39	0·39	0·39	1·138	0·97	0·72	0·55	0·29	29	25·4	383
											3, 4		
6	0·39	0·39	0·39	0·39	0·39	1·45	0·95	0·64	0·53	0·37	15	25·0	273
											1, 3		
7	0·39	0·39	0·34	0·34	0·34	1·26	0·99	0·77	0·60	0·54	15	25·2	150
											1, 2		
8	0·39	0·39	0·34	0·34	0·34	1·21	1·05	0·83	0·59	0·36	18	25·5	160
											2, 3		
9	0·39	0·34	0·34	0·34	0·34	1·18	0·71	0·46	0·28	0·28	16	25·5	111
											2, 2		
10	0·34	0·34	0·34	0·34	0·30	1·38	0·98	0·78	0·46	0·29	12	25·0	70
											1, 1		
11	0·34	0·34	0·34	0·30	0·30	1·25	0·88	0·64	0·40	0·30	12	24·8	59
											1, 1		
12	0·30	0·30	0·34	0·34	0·34	1·34	0·99	0·75	0·50	0·36	12	25·0	63
											1, 1		

1	0.61	0.55	0.74	0.61	0.67	1.65	0.98	6.87	0.78	0.45	12	24.0	103
											1,1		
2	0.74	0.67	0.61	0.61	0.67	1.51	1.08	0.83	0.67	0.46	13	24.0	68
											2,1		
3	0.74	0.74	0.74	0.67	0.74	1.49	1.06	0.84	0.66	0.47	12	25.0	82
											1,1		
4	0.49	0.55	0.55	0.49	0.55	1.63	1.19	0.85	0.75	0.46	12	24.9	104
											1,1		
5	0.55	0.49	0.49	0.49	0.49	1.53	1.15	0.82	0.68	0.47	12	24.0	58
											1,1		
6	0.55	0.55	0.49	0.49	0.49	1.65	1.15	0.88	0.78	0.46	12	24.0	51
											1,1		
7	0.49	0.49	0.49	0.49	0.49	1.60	1.13	0.88	0.77	0.45	12	24.6	57
											1,1		
8	0.55	0.55	0.49	0.55	0.49	1.54	1.06	0.86	0.70	0.50	16	24.8	72
											2,2		
9	0.55	0.55	0.55	0.55	0.55	1.58	1.10	0.89	0.74	0.48	12	25.0	64
											1,1		
10	0.55	0.55	0.61	0.55	0.55	1.66	1.13	0.88	0.72	0.45	12	23.8	62
											1,1		
11	0.55	0.49	0.55	0.49	0.49	1.60	1.09	0.83	0.70	0.43	12	23.8	67
											1,1		
12	0.49	0.49	0.49	0.49	0.44	1.66	1.06	0.88	0.77	0.51	12	23.6	58
											1,1		

1	0·39	0·39	0·39	0·39	0·44	1·12	0·88	0·63	0·43	0·28	13	22·8	84
											1, 1		
2	0·44	0·44	0·49	0·44	0·44	1·07	0·82	0·58	0·38	0·24	43	23·0	342
											5, 6		
3	0·49	0·55	0·55	0·55	0·55	1·01	0·79	0·54	0·34	0·22	49	23·7	342
											6, 7		
4	0·55	0·49	0·55	0·55	0·55	1·05	0·86	0·56	0·41	0·25	32	23·7	424
											3, 6		
5	0·49	0·49	0·49	0·49	0·49	1·18	0·94	0·70	0·48	0·39	24	23·6	1093
											2, 5		
6	0·44	0·44	0·49	0·44	0·49	1·13	0·90	0·66	0·49	0·32	18	23·5	1012
											2, 4		
7	0·44	0·44	0·49	0·44	0·44	1·10	0·86	0·60	0·41	0·26	14	23·5	892
											1, 2		
8	0·44	0·44	0·44	0·44	0·44	1·07	0·82	0·62	0·41	0·25	24	23·7	765
											2, 3		
9	0·44	0·39	0·39	0·44	0·44	1·12	0·88	0·59	0·39	0·26	18	23·1	410
											2, 3		
10	0·39	0·39	0·39	0·44	0·44	1·18	0·93	0·65	0·44	0·29	14	23·0	232
											2, 2		
11	0·39	0·39	0·39	0·39	0·44	1·15	0·89	0·65	0·47	0·37	14	23·0	135
											1, 1		
12	0·39	0·39	0·39	0·39	0·39	1·13	0·84	0·65	0·58	0·29	12	22·9	82
											1, 1		

1	0·55	0·55	0·67	0·67	0·67	1·24	1·10	0·91	0·61	0·44	12	24·8	61
											1, 1		
2	0·55	0·55	0·55	0·67	0·67	1·27	1·15	1·03	0·58	0·28	12	25	68
											1, 1		
3	0·67	0·67	0·67	0·81	0·74	1·03	0·76	0·68	0·48	0·29	33	25	68
											3, 3		
4	0·67	0·67	0·67	0·74	0·74	1·05	0·76	0·58	0·52	0·65	24	24·6	77
											3, 4		
5	0·67	0·67	0·55	0·67	0·67	0·96	0·56	0·51	0·51	0·49	18	24·8	78
											3, 2		
6	0·67	0·67	0·55	0·55	0·67	1·09	0·90	0·71	0·36	0·15	17	25·0	62
											1, 2		
7	0·67	0·55	0·67	0·67	0·55	1·15	0·96	0·77	0·44	0·38	14	24·8	63
											1, 1		
8	0·55	0·55	0·55	0·67	0·67	1·18	1·07	0·75	0·42	0·34	16	25·0	68
											1, 2		
9	0·55	0·55	0·49	0·55	0·55	1·16	1·03	0·74	0·44	0·37	18	25·0	61
											2, 3		
10	0·55	0·55	0·55	0·55	0·55	1·22	1·10	0·78	0·49	0·43	15	24·8	64
											1, 2		
11	0·55	0·55	0·55	0·55	0·55	1·20	1·05	0·89	0·60	0·39	14	24·6	72
											1, 2		
12	0·55	0·55	0·55	0·55	0·55	1·17	0·99	0·90	0·60	0·45	12	24·6	59
											1, 1		

1	0·39	0·39	0·39	0·39	0·39	1·67	1·25	0·96	0·70	0·50	12	24·6	137
											1,1		
2	0·44	0·39	0·44	0·39	0·44	1·58	1·16	1·04	0·78	0·27	36	25·4	131
											4,5		
3	0·39	0·39	0·39	0·34	0·39	1·77	1·28	0·91	0·66	0·55	42	25·4	454
											5,3		
4	0·39	0·39	0·39	0·39	0·39	1·57	1·20	0·95	0·70	0·46	41	25·7	204·5
											4,5		
5	0·39	0·39	0·39	0·39	0·39	1·74	1·22	1·10	0·54	0·48	30	25·4	3172
											3,4		
6	0·61	0·55	0·55	0·55	0·55	1·27	0·99	0·88	0·72	0·23	19	25·1	1785
											2,4		
7	0·44	0·49	0·44	0·44	0·49	1·36	1·00	0·54	0·52	0·44	12	25·0	827
											1,2		
8	0·44	0·44	0·44	0·44	0·44	1·48	1·29	0·96	0·75	0·46	34	25·7	916
											4,3		
9	0·44	0·44	0·44	0·39	0·39	1·66	1·15	0·93	0·69	0·45	30	25·6	561
											3,3		
10	0·39	0·44	0·39	0·39	0·39	1·79	1·45	1·00	0·84	0·48	26	25·6	301
											4,3		
11	0·39	0·44	0·39	0·39	0·39	1·68	1·26	0·96	0·66	0·50	15	25·1	277
											2,2		
12	0·49	0·44	0·49	0·49	0·49	1·61	1·21	0·94	0·65	0·49	12	24·7	274
											1,1		

1	0·55	0·61	0·67	0·61	0·61	1·63	1·45	1·23	0·89	0·60	12	24·5	2.26
											1,1		
2	0·81	0·67	0·67	0·67	0·74	1·65	1·19	1·10	0·67	0·64	49	24·7	205
											7,7		
3	0·95	0·95	0·95	0·95	0·95	1·42	1·07	0·96	0·62	0·50	38	26·9	783
											7,8		
4	1·28	0·95	1·03	1·03	1·03	0·93	0·76	0·79	0·62	0·45	32	27·9	6589
											7,9		
5	0·95	0·81	0·81	0·81	0·81	1·21	0·84	0·78	0·68	0·45	26	27·3	7990
											3,6		
6	0·74	0·67	0·67	0·67	0·67	1·74	1·51	1·08	0·75	0·46	19	27·2	4804
											3,3		
7	0·67	0·67	0·74	0·67	0·61	1·38	1·01	0·84	0·66	0·58	19	27·0	3694
											2,3		
8	0·55	0·55	0·61	0·61	0·61	1·07	1·02	0·91	0·62	0·5	17	27·3	2135
											2,2		
9	0·67	0·61	0·61	0·49	0·55	1·64	1·32	1·13	0·78	0·64	14	27·6	1052
											1,2		
10	0·61	0·55	0·49	0·49	0·55	1·66	1·36	1·21	0·87	0·62	12	26·4	779
											1,1		
11	0·61	0·61	0·61	0·74	0·61	1·53	1·42	0·96	0·78	0·64	12	24·8	332
											1,1		
12	0·61	0·61	0·61	0·61	0·67	1·56	1·43	1·15	0·86	0·61	12	24·6	243
											1,1		

1	0·49	0·44	0·44	0·49	0·44	1·62	1·37	1·05	0·79	0·58	12	28·0	55
											1, 1		
2	0·88	0·81	0·74	0·74	0·67	1·57	1·20	0·88	0·70	0·54	15	28·9	205
											5, 6		
3	0·81	0·81	0·81	0·81	0·81	0·96	0·76	0·58	0·52	0.40	22	30·0	1220
											6, 8		
4	0·61	0·61	0·61	0·67	0·67	0·99	0·84	0·74	0·59	0·45	27	31·0	18005
											7, 10		
5	0·67	0·67	0·61	0·61	0·61	1·57	1·17	0·91	0·75	0·57	24	31·6	13488
											4, 7		
6	0·55	0·55	0·55	0·55	6·55	1·58	1·41	0·99	0·81	0·59	14	32·8	13076
											3, 6		
7	0·61	0·61	0·61	0·61	0·61	1·70	1·18	1·06	0·81	0·57	13	31·5	8606
											2, 4		
8	0·61	0·61	0·55	0·55	0·55	1·60	1·33	1·08	0·83	0·62	15	31·0	6636
											2, 3		
9	0·49	0·49	0·49	0·55	0·55	1·62	1·31	1·04	0·79	0·57	13	30·7	3382
											1, 3		
10	0·55	0·51	0·55	6·55	0·55	1·65	1·38	1·06	0·82	0·54	12	29·5	1613
											1, 2		
11	0·61	0·61	0·61	0·55	0·49	1·58	1·34	1·03	0·77	0·54	12	30·3	642
											1, 1		
12	0·55	0·49	0·49	0·49	0·55	1·63	1·36	1·07	0·80	0·56	12	28·4	320
											1, 1		

1	0·39	0·39	0·39	0·44	0·44	1·58	1·38	1·16	0·76	0·53	12	21·3	96
											1,1		
2	0·44	0·44	0·44	0·44	0·49	1·58	1·22	1·02	0·65	0·58	38	21·8	171
											4,7		
3	0·55	0·49	0·55	0·55	0·55	1·35	1·01	0·84	0·59	0·45	49	22·1	3531
											5,9		
4	0·55	0·49	0·49	0·55	0·55	1·07	0·85	0·77	0·57	0·41	45	22·0	5176
											4,10		
5	0·49	0·49	0·49	0·49	0·55	1·18	0·82	0·71	0·56	0·35	27	21·8	6892
											3,7		
6	0·49	0·44	0·44	0·44	0·39	1·58	1·28	0·99	0·63	0·41	22	21·6	8353
											2,5		
7	0·44	0·44	0·39	0·39	0·39	1·38	1·05	0·83	0·61	0·42	28	21·8	4243
											3,6		
8	0·44	0·44	0·44	0·39	0·44	1·28	1·08	0·90	0·65	0·41	22	21·6	2134
											2,5		
9	0·39	0·44	0·44	0·39	0·39	1·54	1·30	1·04	0·73	0·57	19	21·6	1949
											2,4		
10	0·39	0·39	0·39	0·44	0·49	1·62	1·25	1·09	0·78	0·47	16	21·4	497
											2,3		
11	0·44	0·44	0·44	0·44	0·44	1·55	1·24	0·88	0·70	0·47	16	21·4	543
											1,2		
12	0·49	0·44	0·44	0·39	0·44	1·58	1·27	1·02	0·76	0·47	16	21·4	467
											1,2		

1	0·34	0·29	0·29	0·26	0·26	1·54	1·40	0·98	0·63	0·44	12	22·7	55
											1, 1		
2	0·34	0·34	0·34	0·34	0·34	1·51	1·26	0·94	0·66	0·52	23	23·2	153
											2, 3		
3	0·34	0·30	0·30	0·34	0·34	1·28	0·95	0·72	0·56	0·39	26	23·6	78
											4, 3		
4	0·34	0·34	0·34	0·34	0·30	1·22	0·94	0·74	0·53	0·36	16	24·1	352
											2, 2		
5	0·30	0·26	0·26	0·26	0·26	1·16	0·81	0·64	0·53	0·25	13	24·0	472
											1, 1		
6	0·26	0·26	0·26	0·26	0·26	1·42	1·05	0·91	0·51	0·36	12	23·8	56
											1, 1		
7	0·30	0·30	0·30	0·30	0·25	1·37	1·10	0·83	0·57	0·25	12	24·0	63
											1, 1		
8	0·34	0·30	0·30	0·30	0·30	1·48	1·14	0·89	0·68	0·31	19	24·2	132
											3, 2		
9	0·34	0·30	0·34	0·30	0·30	1·44	1·27	0·94	0·68	0·50	21	23·8	223
											3, 3		
10	0·34	0·30	0·30	0·26	0·30	1·59	1·15	0·97	0·68	0·32	16	23·3	166
											2, 2		
11	0·34	0·30	0·30	0·30	0·30	1·58	1·05	0·80	0·63	0·31	12	23·0	111
											1, 1		
12	0·30	0·30	0·26	0·30	0·30	1·60	1·11	0·89	0·67	0·32	12	23·0	67
											1, 1		

1	0·34	0·30	0·30	0·30	0·30	1·53	1·12	0·92	0·70	0·47	12	28·5	117
											1, 1		
2	0·49	0·61	0·61	0·55	0·49	1·37	0·97	0·81	0·63	0·54	36	29·1	199
											5, 10		
3	0·55	0·55	0·61	0·61	0·49	1·10	1·00	0·84	0·52	0·41	35	29·4	5601
											9, 14		
4	0·55	0·55	0·55	0·49	0·44	1·21	0·96	0·89	0·56	0·44	30	29·7	7653
											6, 9		
5	0·44	0·44	0·39	0·34	0·34	1·44	1·06	0·87	0·55	0·53	16	29·9	13511
											2, 3		
6	0·39	0·39	0·34	0·34	0·30	1·44	1·08	0·91	0·69	0·45	12	29·6	5926
											1, 1		
7	0·39	0·34	0·34	0·34	0·39	1·46	1·10	0·91	0·72	0·50	12	29·6	4523
											1, 1		
8	0·34	0·30	0·34	0·34	0·34	1·34	1·12	0·89	0·69	0·45	12	29·5	2949
											1, 1		
9	0·39	0·39	0·39	0·39	0·39	1·47	1·11	0·87	0·75	0·47	12	29·1	1668
											1, 1		
10	0·39	0·34	0·39	0·34	0·39	1·40	1·15	0·93	0·71	0·45	12	28·8	849
											1, 1		
4	0·39	0·34	0·34	0·34	0·34	1·42	1·12	0·92	0·69	0·47	12	28·8	875
											1, 1		
12	0·34	0·30	0·30	0·34	0·34	1·48	1·11	0·94	0·69	0·47	12	28·6	843
											1, 1		

1	0·22	0·22	0·26	0·26	0·22	1·46	1·20	0·95	0·78	0·62	12	24·0	96
											1, 1		
2	0·39	0·39	0·44	0·39	0·39	1·30	1·16	0·98	0·70	0·51	57	24·4	111
											5, 7		
3	0·39	0·44	0·44	0·39	0·39	1·26	1·04	0·77	0·58	0·46	60	26·0	104
											5, 11		
4	0·39	0·39	0·39	0·34	0·34	0·99	1·03	0·87	0·45	0·44	44	26·5	1183
											4, 11		
5	0·39	0·39	0·39	0·39	0·39	0·92	0·98	0·95	0·90	0·57	35	26·8	977
											3, 5		
6	0·39	0·39	0·39	0·39	0·34	1·31	1·05	0·96	0·76	0·58	22	25·8	1052
											2, 3		
7	0·34	0·34	0·34	0·34	0·34	1·32	1·10	0·93	0·78	0·59	17	24·5	418
											2, 2		
8	0·39	0·30	0·30	0·30	0·30	1·58	1·17	1·08	0·82	0·57	28	25·0	398
											2, 3		
9	0·30	0·30	0·30	0·30	0·30	1·23	1·26	0·99	0·85	0·71	18	25·2	341
											2, 2		
10	0·30	0·30	0·30	0·30	0·30	1·36	1·16	0·95	0·80	0·54	12	24·8	167
											1, 1		
11	0·30	0·30	0·30	0·30	0·30	1·46	1·17	0·92	0·76	0·55	12	24·7	171
											1, 1		
12	0·30	0·26	0·36	0·26	0·26	1·48	1·16	0·97	0·80	0·62	12	24·7	234
											1, 1		

1	0.79	0.39	0.39	0.44	0.39	1.37	1.20	1.00	0.68	0.43	12	25.8	176
											1,1		
2	0.49	0.61	0.61	0.61	0.61	1.21	1.06	0.75	0.51	0.29	21	26.8	143
											4,4		
3	0.61	0.61	0.61	0.67	0.67	1.13	0.94	0.72	0.45	0.27	28	27.2	752
											7,10		
4	0.55	0.55	0.61	0.61	0.67	1.35	1.07	0.92	0.90	0.47	19	28.0	3061
											3,5		
5	0.55	0.49	0.55	0.55	0.55	1.38	1.17	0.97	0.73	0.44	18	28.0	4850
											3,4		
6	0.49	0.49	0.49	0.49	0.55	1.40	1.16	0.98	0.70	0.41	14	27.9	2517
											2,3		
7	0.49	0.49	0.49	0.55	0.55	1.36	1.26	0.99	0.68	0.45	15	27.5	1230
											2,2		
8	0.39	0.39	0.39	0.39	0.39	1.14	1.01	1.00	0.79	0.65	14	28.0	1434
											1,1		
9	0.39	0.39	0.39	0.39	0.39	1.23	1.12	1.02	0.78	0.57	12	27.5	780
											1,1		
10	0.34	0.39	0.39	0.39	0.39	1.36	1.19	0.96	0.70	0.52	12	26.8	421
											1,1		
11	0.39	0.39	0.39	0.39	0.44	1.34	1.20	1.02	0.75	0.54	12	26.6	383
											1,1		
12	0.39	0.34	0.34	0.34	0.34	1.39	1.16	1.00	0.68	0.53	12	25.7	314
											1,1		

1	0·74	0·88	1·03	1·03	1·20	1·36	1·08	0·74	0·14	0·09	12	25·2	69
											1, 1		
2	0·95	0·95	1·03	0·95	0·95	1·33	1·05	0·87	0·78	0·13	30	25·4	125
											6, 9		
3	0·88	0·88	0·88	1·03	1·03	0·84	0·77	0·80	0·43	0·12	30	25·4	175
											6, 8		
4	0·95	0·81	0·95	0·88	1·50	1·37	1·00	0·63	0·26	0·12	22	25·4	184
											4, 4		
5	0·95	0·95	1·03	1·11	1·11	1·46	1·01	0·46	0·26	0·05	18	25·2	324
											3, 3		
6	0·81	0·88	0·88	0·81	0·95	1·27	1·06	0·46	0·22	0·13	15	25·2	254
											2, 2		
7	0·81	0·74	0·74	0·81	1·03	1·16	0·73	0·44	0·17	0·06	12	25·2	113
											1, 1		
8	0·81	0·74	0·74	0·81	1·03	1·31	1·06	0·87	0·36	0·10	14	25·2	175
											1, 2		
9	0·88	0·74	0·81	0·88	1·28	1·40	1·12	0·68	0·35	0·08	12	25·0	106
											1, 1		
10	0·95	0·95	1·11	1·11	1·20	1·10	0·95	0·37	0·27	0·13	12	25·2	115
											1, 1		
11	0·81	0·74	0·88	0·88	0·95	1·40	1·16	0·45	0·31	0·13	12	25·2	86
											1, 1		
12	0·81	0·74	0·81	0·95	1·37	1·37	0·84	0·54	0·31	0·10	12	25·2	79
											1, 1		

1	0·34	0·34	0·34	0·34	0·30	1·26	1·09	0·75	0·46	0·21	12	24·7	150
											1, 1		
2	0·39	0·39	0·34	0·34	0·34	1·12	0·48	0·33	0·43	0·18	40	25·2	273
											3, 4		
3	0·44	0·44	0·44	0·39	0·39	1·18	0·80	0·54	0·44	0·17	41	25·5	307
											5, 6		
4	0·44	0·39	0·44	0·39	0·44	1·23	1·14	0·80	0·31	0·26	19	25·9	3690
											3, 4		
5	0·39	0·39	0·39	0·39	0·39	1·29	1·20	0·85	0·59	0·32	14	25·8	3826
											1, 2		
6	0·39	0·39	0·39	0·39	0·39	1·34	1·19	0·84	0·66	0·27	14	25·0	2733
											1, 2		
7	0·39	0·39	0·39	0·39	0·34	1·62	1·15	0·78	0·44	0·24	12	25·2	1362
											1, 1		
8	0·39	0·39	0·39	0·39	0·34	1·40	1·08	0·80	0·43	0·16	12	25·1	820
											1, 1		
9	0·39	0·39	0·39	0·34	0·39	1·38	1·06	0·75	0·41	0·33	12	25·0	409
											1, 1		
10	0·39	0·34	0·39	0·34	0·39	1·39	0·95	0·50	0·61	0·29	12	25·0	403
											1, 1		
11	0·39	0·39	0·39	0·39	0·34	1·34	1·04	0·81	0·48	0·22	12	25·0	763
											1, 1		
12	0·39	0·34	0·34	0·39	0·34	1·38	1·09	0·87	0·68	0·26	12	24·9	352
											1, 1		

1	0·39	0·39	034	0·39	0·39	1·22	1·06	0·85	0·54	0·29	12	24·8	62
											1, 1		
2	0·44	0·49	0·44	0·44	0·44	1·07	0·96	0·61	0·36	0·26	22	25·4	96
											4, 4		
3	0·49	0·49	0·49	0·44	0·44	0·98	0·80	0·56	0·42	0·24	29	26·1	199
											7, 10		
4	0·44	0·39	0·39	0·39	0·39	1·08	0·95	0·68	0·50	0·26	20	26·1	78
											3, 5		
5	0·44	0·44	0·44	0·44	0·44	1·13	0·98	0·73	0·49	0·26	19	25·8	1012
											3, 4		
6	0·44	0·44	0·39	0·44	0·44	1·20	1·06	0·75	0·50	0·28	15	25·2	970
											2, 3		
7	0·44	0·44	0·39	0·39	0·39	1·26	1·04	0·74	0·50	0·26	16	25·0	349
											2, 2		
8	0·44	0·44	0·39	0·39	0·39	1·12	0·93	0·71	0·44	0·26	15	25·2	258
											1, 1		
9	0·44	0·39	0·39	0·39	0·39	1·14	1·01	0·87	0·53	0·26	13	25·2	150
											1, 1		
10	0·39	0·39	0·39	0·39	0·34	1·30	1·10	0·90	0·56	0·33	12	25·0	117
											1, 1		
11	0·39	0·39	0·39	0·34	0·34	1·21	1·07	0·89	0·62	0·28	12	24·9	68
											1, 1		
12	0·34	0·34	0·39	0·39	0·39	1·22	0·94	0·78	0·76	0·34	12	24·9	59
											1, 1		

APPENDIX G

CALCULATION OF CREATINE KINASE VALUES (CHAPTER 5)

Determine CK activity as follows:

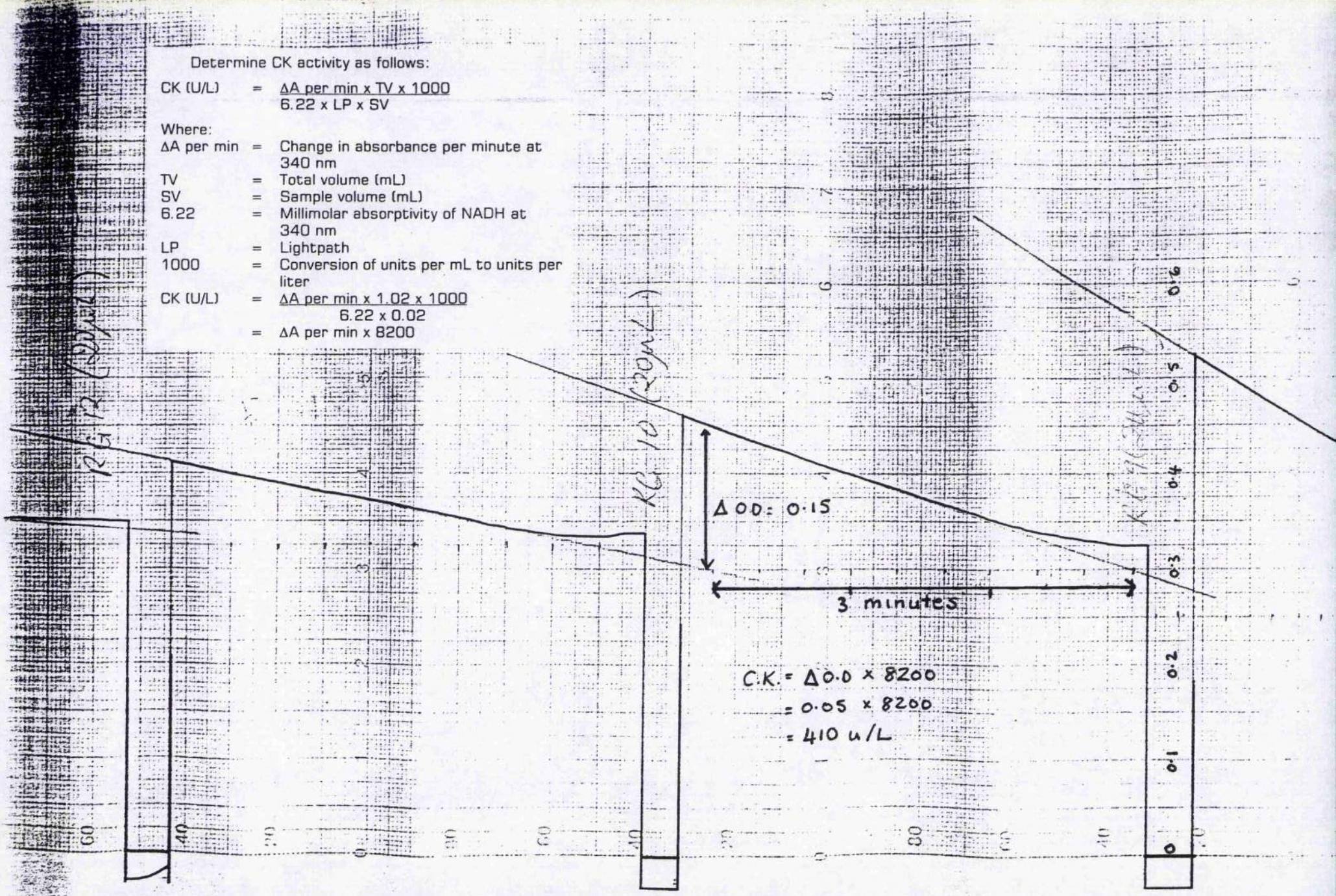
$$\text{CK (U/L)} = \frac{\Delta A \text{ per min} \times TV \times 1000}{6.22 \times LP \times SV}$$

Where:

- ΔA per min = Change in absorbance per minute at 340 nm
- TV = Total volume (mL)
- SV = Sample volume (mL)
- 6.22 = Millimolar absorptivity of NADH at 340 nm
- LP = Lightpath
- 1000 = Conversion of units per mL to units per liter

$$\text{CK (U/L)} = \frac{\Delta A \text{ per min} \times 1.02 \times 1000}{6.22 \times 0.02}$$

$$= \Delta A \text{ per min} \times 8200$$



APPENDIX H

ANOVA TABLE FOR STUDY THREE
(CHAPTER 6)

Analysis of Variance for SORENESS

Source	DF	SS	MS	F	P
SITE	13	674.607	51.893	7.98	0.000
DAY	1	745.723	745.723	137.14	0.000
SUBJ	15	81.062	5.404	0.83	0.640
SITE*DAY	13	662.214	50.940	7.86	0.000
SITE*SUBJ	195	1268.250	6.504	1.00	0.492
DAY*SUBJ	15	81.562	5.437	0.84	0.634
Error	195	1264.500	6.485		
Total	447	4777.919			

APPENDIX I

T-TEST ON RESTING ELBOW FLEXION ANGLE (CHAPTER 6)

T - TEST FOR THE RESTING ANGLE OF FLEXION

The null hypothesis is that there is no difference between the pre- exercise and post-exercise angle of elbow flexion

Subjects	Pre-exercise	Post-exercise	D	D2
A	165	155	10	100
B	168	160	8	64
C	170	152	18	324
D	155	157	-2	4
E	166	150	16	256
F	172	149	23	529
G	168	163	5	25
H	162	145	17	289
I	160	157	3	9
J	168	149	19	361
K	167	155	15	144
L	165	165	0	0
M	168	152	16	256
N	170	161	9	81
O	162	161	1	1
P	166	160	6	6
Total			161	2479

$$t = \frac{\Sigma D}{\sqrt{\frac{(N\Sigma D^2 - (\Sigma D)^2)}{N - 1}}}$$

$$t = \frac{161}{\sqrt{\frac{(16 \times 2479 - 25921)}{15}}}$$

$$t = \frac{161}{30.27}$$

$$t = 5.32$$

degrees of freedom = 15

The obtained value for t exceeds the critical value at the 1% level. The null hypothesis is therefore rejected. There is a significant difference between the pre- exercise and post-exercise angle of elbow flexion ($p < 0.01$).

APPENDIX J

T-TEST ON PERCEPTION TEST

T - TEST FOR THE PERCEPTION TEST

The null hypothesis is that there is no difference between the pre- exercise and post-exercise ability to perform the perception test.

Subjects	Pre-exercise	Post-exercise	D	D2
A	1.5	3.5	-2	4
B	5.5	5.7	-0.2	0.04
C	8.7	5.0	-3.7	13.69
D	3.6	3.8	-0.2	0.04
E	9.4	10.1	-0.7	0.49
F	7.5	5.2	2.3	5.29
G	3.7	2.9	0.8	0.64
H	6.2	3.9	2.3	5.29
I	5.7	10.1	-4.4	19.36
J	9.2	9.8	-0.6	0.36
K	1.7	1.8	-0.1	0.01
L	3.0	3.5	-0.5	0.25
M	5.6	9.1	-4.5	20.25
N	4.4	8.6	-3.8	14.44
O	2.5	2.6	-0.1	0.01
P	3.1	4.3	-1.3	1.69
Total			-16.7	85.85

$$t = \frac{\Sigma D}{\sqrt{\frac{(N\Sigma D^2 - (\Sigma D)^2)}{N - 1}}}$$

$$t = \frac{-16.7}{\sqrt{\frac{(16 \times 85.85 - 278.89)}{15}}}$$

$$t = \frac{-16.7}{2.206}$$

$$t = 7.57$$

degrees of freedom = 15

The obtained value for t does not exceed the critical value at the 5% or 1% level. The null hypothesis is therefore accepted. There is no difference between the pre- exercise and post-exercise ability to perform the perception test.