

**ROLE OF MASSAGE AND STRETCHING
IN RECOVERY FROM EXERCISE
AND IN DELAYED ONSET MUSCLE SORENESS**

By

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Submitted in part fulfilment of the requirements for the degree

MASTER OF MEDICAL SCIENCE

Department of Physiology

Faculty of Medicine

University of Natal

1997

Abstract

Adequate recovery from intense exercise is essential to optimise performance and reduce the associated symptoms of tiredness, fatigue and lethargy. The purpose of the study was to :-

i. investigate the effects of massage and stretching in delaying the development of fatigue during repeated bouts of dynamic activity and, ii. to investigate the relative effects of massage and stretching on delayed onset muscle soreness (DOMS). Eighteen volunteer males participated in this study. They were randomly allocated into one of six groups of an Orthogonal Latin square design. Subjects performed five repetitions of as many heel raises as possible in 45 seconds. Each repetition was followed by a recovery technique of three minutes duration. This was repeated weekly until each group has had all three recovery techniques. A fatigue index % was calculated from the decline in the number of repetitions from stage 1 to stage 5. Muscle soreness ratings were retrospectively assessed at 12-36 hour after each session. An analysis of variance showed a significant difference in the fatigue indices. Post hoc intergroup comparison using paired T-tests with the Bonferroni adjustment showed a significant difference between rest and massage ($p=0.0001$) and rest and stretching ($p=0.0006$). The differences between massage and stretching were not significant. Fourteen (77.8%) and Fifteen (83.3%) subjects showed an improvement in performance following massage and stretching respectively. Massage is associated with significantly less muscle soreness than stretching ($p<0,001$). DOMS was most frequently found in the stretching group while the lowest incidence and lowest mean ratings of muscle soreness associated with DOMS was found in the massage group. The difference between massage and stretching was marginal ($p=0.0461$) and showed a trend that massage is associated with less DOMS than stretching. This suggests that rest is the least beneficial recovery technique, and that massage may be superior to stretching as there is less muscle soreness.

Preface

This study represents original work by the author and has not been submitted in any form to another University. Where use was made of the work of others, it has been duly acknowledged in the text.

The research described in this commentary was carried out in the Sports Clinic, Department of Physiotherapy, University of Durban-Westville and under the supervision of Prof. Maurice Mars, Department of Physiology, Faculty of Medicine, University of Natal.

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1997

Acknowledgements

I would like to extend my sincere thanks and gratitude to:

Professor M Mars, my supervisor, for his guidance, constructive criticism, expert advice and teaching during the preparation of this commentary.

Eleanor Gouws from the Department of Biomedical Statistics for her assistance with the statistical planning and analysis.

Professor P.Gounden and Mrs.L.Gumede of the Department of Physiotherapy, University of Durban-Westville for their assistance with the use of equipment and venue.

Dr.Y.Coopoo of the Department of Human Movement Studies, University of Durban-Westville for his encouragement and assistance with access to the students and equipment.

Eddie Bodha, Sandra Pillay and Nerosh Pillay for their assistance and support.

The students from the Department of Human Movement Studies, University of Durban-Westville and members of Longcroft Phoenix Aces Soccer club for participating in this study.

My family and friends for their support and encouragement

Ms.Bharti Odhav and Dr.Raj Narainbhai for their moral support, advice and assistance.

My wife Sundrini, for her support and advice, and children, Santhuri and Santhiran, for their patience, tolerance, love and encouragement.

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CHAPTER 1

INTRODUCTION

Everyone has experienced the sensations of fatigue and the increasing difficulty of continuing a given level of physical exercise (Bigland-Ritchie and Woods, 1984). Improved performance may often be related to a reduction of or delay in the onset of fatigue (Kirkendall, 1990). Recovery of physical performance capacity between successive training sessions is considered to be very important in many sports events. Adequate recovery from intense exercise is essential to optimize performance and reduce the associated symptoms of tiredness, fatigue and lethargy.

High intensity activities involve repeated submaximal contractions that may disrupt the coupling between excitation and contraction, disturb the integrity of the muscle cell, upset calcium (Ca^{++}) homeostasis, lead to the accumulation of lactate and H^+ , and cause depletion of glycogen stores (Green, 1996). The rate of recovery for each of these processes varies from a few seconds to a few days depending on the exercise type, intensity and duration. Many physiological processes of recovery from exercise, such as provision of adenosine triphosphate (ATP) and creatine phosphate (CP) resynthesis and removal of metabolites rely to varying degrees on the ability of the cardiovascular system (CVS) and the respiratory system to transport various substances to and from the muscles.

Local blood flow is regulated by the metabolic rate in the surrounding tissue, and cutaneous blood flow during exercise is largely a function of core temperature. Since blood is the medium by which most of the oxygen (O_2), carbon dioxide (CO_2), hormones, glucose, free fatty acids and

thermal energy are transported, anything that alters blood flow will have an effect on the rate at which these substances are delivered or removed (Cafarelli and Flint, 1992). Moreover, the rates of all enzymatic processes are influenced by temperature and pH and later by the substrate concentration. An increase in muscle blood flow will hasten the delivery of oxygen, increase the temperature and buffer acidotic shifts in pH. This would facilitate recovery and aid in the performance of endurance exercise.

Various techniques, both active and passive, are currently used to enhance recovery from muscular fatigue. In active recovery, often called 'cooling down', submaximal aerobic exercise is performed in the belief that this continued movement or activity enhances different lactate removal and utilization patterns (Belcastro and Bonen, 1975; Weltman *et al*, 1979; Gozal *et al*, 1992; Thiriet *et al*, 1993) prevents cramps and stiffness and facilitates the recovery process (McArdle *et al*, 1991).

Brief bouts (5-10 seconds) of intense activity or steady rate aerobic exercise are not associated with lactic acid accumulation and recovery is rapid. However, longer periods of high intensity exercise are performed at the expense of lactate build up in the blood and exercising muscles, and a significant disruption in other physiological processes. Considerably more time is required for complete recovery after lactate accumulation (Belcastro and Bonen, 1975; McArdle *et al*, 1991). Lactic acid inhibits the mobilization of free fatty acids or may retard the rate of glycolysis by inhibiting the activity of glycolytic enzymes such as lactate dehydrogenase and phosphofructokinase (Belcastro and Bonen, 1975).

The removal of lactic acid after intense exercise may therefore be critical for the resumption of subsequent exercise at the previous intensity and for performance enhancement. Inadequate

recovery will lead to fatigue which can reduce work rate during training and during sports such as basketball, hockey, soccer, volleyball and badminton amongst others.

Lactate accumulation is not the only change occurring during and after intense activity (Armstrong, 1990; Kirkendall, 1990; McArdle *et al*, 1991). Most studies have focussed on active recovery and in particular on lactate disappearance and utilization patterns (Weltman *et al*, 1979; Armstrong, 1990; Thieret *et al*, 1993). Whilst there have been convincing reports of the beneficial effect of active recovery on lactate clearance (Belcastro and Bonen, 1975; Gozal *et al*, 1992; Thieret *et al*, 1993) and subsequent performance (Belcastro and Bonen, 1975; Thieret *et al*, 1993), there have also been reports that elevated lactate levels have little effect on subsequent performance (Weltman *et al*, 1979). Furthermore, Gozal *et al* (1992) found that while passive recovery (rest) was associated with higher lactate levels, no significant difference was found between active and passive recovery (rest) on subsequent performance.

It is therefore necessary to examine other methods of passive recovery, which may include rest, massage, cold showers, stretching, and adopting specific body postures (Balke *et al*, 1989; McArdle *et al*, 1991; Zelikovski *et al*, 1993; Viitasalo, 1995). These methods are used at half time during a game, at time out and inbetween repeated bouts of high intensity exercise. Despite widespread use, there is little evidence in the literature to validate the use of massage and stretching as recovery techniques.

Massage

Massage is gaining recognition as an important preventative and treatment modality (Zuluaga *et al*, 1995) and is routinely recommended as an aid for speeding recovery from vigorous exercise (Balke *et al*, 1989; Cafarelli *et al*, 1990; Cafarelli and Flint, 1992). Despite the long history of

practical application of massage, there are very few rigorously obtained scientific data available that would substantiate the widely held belief in its effectiveness (Cafarelli and Flint, 1992).

Many athletes appreciate the efficacy of massage in aiding their recovery from strenuous athletic efforts. Massage of the fatigued limbs is regarded as more effective than passive recovery, but apart from statements by athletes supporting massage, there is a limited amount of scientific evidence to substantiate these claims. Massage has been purported to offset the negative effects of muscular exercise. Some studies have objectively demonstrated that massage is effective in facilitating the recovery from muscular fatigue (Balke *et al*, 1989; Zelikovski, 1993; Viitasalo, 1995). It is suggested that the effect is probably gained by the mechanism of improving the blood circulation of the exercised muscles or by increasing the amount of glycogen and succinate dehydrogenase in the exercised muscle (Ma-Yuhe and Fu-Shiru, 1994), thus restoring the disrupted internal environment back to homeostatic levels.

There are however equivocal reports on the effect of massage on muscle recovery and performance (Samples, 1987; Balke *et al*, 1989; Drews *et al*, 1990).

Kaada and Torsteinbo (1989) suggest that an increase in plasma beta endorphins following connective tissue massage is responsible for the beneficial effects of massage. Possibly the release of beta endorphins is linked with pain relief and a feeling of well-being. Studies on the effect of massage on measurements of blood flow and some blood borne substances are sparse and show extreme variability of the data (table 1, section 2.4.2).

The role of massage in the relief of delayed onset muscle soreness (DOMS) is uncertain. Some studies show massage to provide faster, more graded recovery from DOMS (Hill and Richardson,

1989; Zuluaga *et al*, 1995). However, Wenos *et al* (1990) demonstrated no significant differences in strength reduction and pain perception normally associated with DOMS between subjects who received post event massage and a control group.

Stretching

Stretching is regarded as an important pre-exercise warm-up and a necessity for maintenance and improvement of flexibility and injury prevention (Smith, 1994). Stretching is also recommended as a method of minimising and alleviating muscle soreness (de Vries, 1961; Noakes and Granger, 1990). Although the benefits of stretching are well recognised in the prevention of injuries, there appears to be a paucity of data on the effect of stretching in delaying the development of fatigue.

Electromyography (EMG) data obtained from runners during exercise associated muscle cramps revealed that baseline activity is increased. A reduction in baseline activity correlates with recovery and passive stretching is effective in reducing EMG activity (Schwellnus and Derman, 1996). Muscle stretching causes both mechanical and reflex effects. Abnormal reflex activity of the muscle spindle (increased activity) and the golgi tendon organ (decreased activity) has been observed in fatigue muscle in the cat (Nelson and Hutton, 1985; Hutton and Nelson, 1986). It is proposed that spindle sensitivity can increase tonic and phasic drive to alpha motorneurons. Muscle spindle discharge interacts interneuronally and reflexly with golgi tendon organs which predominantly provide inhibitory input to the same motorneuron pools.

Guissard *et al* (1988) studied motorneuron excitability during stretching of the human soleus muscle. Their results indicate that static stretching not only inhibits excitatory afferents from muscle spindles, but that there is also inhibition of the motorneuron pool. This inhibition could

result from inhibitory afferents originating from the golgi tendon organ or from muscle spindle secondary afferents. Spindle activity has an excitatory effect on alpha motorneuron activity and golgi tendon organ activity has an inhibitory effect on alpha motorneuron activity. Since muscle stretching results in inhibition of the motorneuron pool (Guissard *et al*, 1988), and fatigue is associated with increased alpha motorneuron activity (Nelson and Hutton, 1985), stretching may theoretically have an effect on fatigue.

Studies on the effect of stretching in minimising or alleviating DOMS are few and contradictory. deVries (1961) found that stretching minimised DOMS and hypothesised that muscular distress following unaccustomed exercise is caused at least in part by tonic muscle spasm. Buroker and Schwane (1989) and High and Howley (1989) were however unable to demonstrate that stretching prevented or minimised DOMS.

Despite the popular use of massage and stretching in presumably minimising and alleviating muscle fatigue and DOMS, there is little evidence in the literature to support such use. Increased fatigue resistance would appear to depend on carefully planned programmes designed to adapt the excitation-contraction processes, the cytoskeleton and the metabolic systems to tolerate the changes in the intracellular environment that are caused by intense activity (Green, 1996). However the literature reveals equivocal reports on the efficacy of various recovery techniques. Further research is therefore necessary to validate the various recovery techniques used and to find methods to optimise recovery and subsequent performance.

Thus the aims of this study were:

- To investigate the effects of massage and stretching in delaying the development of fatigue during repeated bouts of dynamic exercise.
- To investigate the relative effects of massage and stretching on DOMS.

The first part of this commentary is an overview of the literature to date covering muscle fatigue, recovery from exercise, delayed onset muscle soreness, massage and muscle stretching. The second part (Methods) describes the exercise protocol used to induce muscle fatigue and the measurement of muscle fatigue, the recovery techniques used and the statistical analysis, followed by the results obtained in this study. This is followed by a discussion of the results in chapter five and the conclusion and recommendations in chapter six.

Chapter 2

Background and Literature Review

2.1 Fatigue

Fatigue imposes limitations on many types of athletic performance and despite the critical role it may play in subsequent performance, it is not known how the many proposed mechanisms of fatigue interact and cause the athlete to stop. Skeletal muscle fatigue is also associated with diminished work productivity as well as increased susceptibility to injury (Williams and Klug, 1995). Improved performance may often be related to a reduction of or delay in the onset of fatigue (Kirkendall, 1990).

2.1.1 Definition

Numerous definitions of fatigue have been offered: 'reductions in the ability to produce a given force or power' (Williams and Klug, 1995), 'an inability of a muscle to sustain the required or expected force' (Bigland-Ritchie and Woods, 1984), failure of the neuromuscular system to carry out a previously determined task (Kroon and Naeije, 1988), 'failure to maintain an expected power output (Kirkendall, 1990). This terminology has led to the concept that the onset of fatigue is delayed or commences only after a protracted period of exercises. However, evidence suggests that the physiological events underlying fatigue commence at the onset of activity, although they cannot always be detected (Bigland-Ritchie and Woods, 1984). Therefore a more appropriate definition would be 'any reduction in the force generating capacity of the total neuromuscular system regardless of the force required in any given situation' (Bigland-Ritchie and Woods, 1984).

2.1.2 Causes and Location of Fatigue

The causes of fatigue are probably as many as the activities that prompt it. Most studies have sought a single cause, yet the generation of a voluntary (or involuntary) contraction involves a sequence of events, any of which may fail (Bigland-Ritchie and Woods, 1984; Kirkendall, 1990). Muscle fatigue is a complex phenomenon involving biochemical, physiological, functional and psychological elements (Baker *et al*, 1992; Baker *et al*, 1993; de Haan, 1994) and multiple mechanisms may be responsible for moderate fatigue. Furthermore fatigue also depends on the type, duration, and intensity of exercise, the fibre type composition of the muscle, the individual level of fitness, and a number of environmental factors (Fitts, 1993).

The various potential sites of origin of fatigue can be divided into three categories: those that lie within the central nervous system (central fatigue), those concerned with neural transmission from CNS to muscle, and those within the muscle fibre itself (peripheral fatigue) (Bigland-Ritchie and Woods, 1984; Fitts, 1993). Peripheral fatigue may include changes in function of the motoneuron, the neuromuscular junction, excitation-contraction coupling, and the accumulation of metabolites and depletion of fuels (Kirkendall, 1990).

Central fatigue refers to a decline of force that is related to poor motivation, altered central nervous system transmission and or recruitment resulting in a reduced motor drive (Bigland-Ritchie and Woods, 1984; Kirkendall, 1990; McArdle *et al*, 1991; Fitts, 1993).

2.1.3 Central versus Peripheral factors

Bigland-Ritchie *et al* (1986) investigated the role of central and peripheral factors during intermittent submaximal contractions. They suggested that during fatigue induced by intermittent medium intensity isometric exercise of the quadriceps muscle, the central nervous system remains capable of full muscle activation, and that the reduced force in response to maximum voluntary effort can only result from failure of the muscle contractile apparatus. However the behaviour of fatigue of the soleus muscle did not display similar findings. It is suggested that type II muscle fibres in the quadriceps may be more susceptible to excitation contraction failure than are type I fibres that predominate in the soleus muscle.

Belanger and McComas (1981) also found that many of their subjects were unable to contract the soleus muscle maximally even in the unfatigued state. They suggested that these findings may be due to progressive failure of neuromuscular transmission or reduced motor drive and that the central component apparent in fatigue of these exercising muscles may possibly be overcome by more positive and persuasive encouragement.

Due to the complexity of fatigue, it is convenient to discuss the various causes and potential sites of fatigue in the context of the type of activity that induces changes in work output.

2.1.4 Fatigue of Short Duration, High Intensity Exercise

2.1.4.1 Impairment of the Central nervous system

During maximal contractions lasting for 45-60 seconds, central fatigue does not appear to be a major factor (Bigland-Ritchie and Woods, 1984; Fitts, 1993).

2.1.4.2 Neuromuscular Junction (NMJ)

There are no current *in vivo* method of studying the NMJ, and inferences regarding its function are drawn from EMG studies. In all out exercise, when all the motor units are maximally activated, fatigue is accompanied by a decrease in neural activity, as measured by EMG activity (McArdle *et al*, 1991). There are conflicting reports of the NMJ as a potential site of fatigue (Kirkendall, 1990). Possible sites of fatigue at the NMJ include some form of inhibition of the presynaptic endings, limited transmitter substance, or an inability of the postsynaptic membrane to become excited (McLaren *et al*, 1989). The reduced motoneuron firing rate sometimes seen may be related to a peripheral reflex that reduces the firing rate by the CNS. Should this be the case, then the NMJ may not be impaired.

The observation that fatigued muscles generate the same tension whether stimulated directly or by way of the motor nerve argues against NMJ fatigue (Fitts, 1993). Considerable evidence supports post-junctional failure in skeletal muscle fatigue (Nelson and Hutton, 1985). The role of pre-junctional mechanisms involving the motoneuron pool and other central neural factors remain controversial. The role of the NMJ in fatigue remains unresolved.

2.1.4.3 Changes in Sarcolemmal Function

With fatigue, the sarcolemma's action potential (AP) amplitude and duration are depressed and prolonged respectively. A proposed theory is that potassium ion (K^+) efflux and sodium ion (Na^+) influx and inhibition of the sodium potassium (Na^+K^+) pump cause cell membrane depolarisation, a reduced action potential amplitude and in some cells complete inactivation (Fitts, 1993). The membrane potential is dependent on intra- and extracellular Na^+ and K^+ concentrations, and these concentrations are affected by water content. K^+ loss from the exercising muscle may be a major factor in the development of fatigue with maximal exercise (Kirkendall, 1990). Based on the calculations in a study by Sjogaard (1986), the K^+ lost by exercising muscles are taken up by other tissues and delivered back to the fatigued muscles upon relaxation as evidenced by the rapid re-establishment of K^+ concentration in recovery. This could explain in part the rapid recovery of short term power during recovery from exercise while lactate levels remain elevated. It has been suggested that these changes in sarcolemmal function induce fatigue and this hypothesis is known as the membrane mechanism of muscle fatigue.

2.1.4.4 Alterations in Excitation - Contraction Coupling

The action potential produces a charge movement within the T-tubular membrane which subsequently by an unknown process leads to Ca^{2+} release from the sarcoplasmic reticulum (SR). The elevated concentrations of cytoplasmic Ca^{2+} results in Ca^{2+} binding to troponin C, a regulatory protein. A molecular configurational change in the troponin-tropomyosin complex occurs that in turn allows the contractile proteins actin and myosin to bind and to generate force. It has been suggested that a reduction in AP amplitude affects propagation into the T-tubules and thus inhibits subsequent steps in excitation - contraction coupling.

An increase in cytoplasmic Ca^{2+} concentration in fatigued muscles suggests a reduced rate of uptake of Ca^{2+} by the SR. Such changes could be due to alterations in T-tubular charge movement, to changes in the intrinsic ability of the SR to release or remove Ca^{2+} . Prolonged twitch duration and reduced peak rate of tension development is frequently observed in fatigued muscle. Prolonged twitch duration also reflects a similar prolongation in the time course of the increase in intracellular levels of Ca^{2+} concentrations (Fitts, 1993). Kolbeck and Nosek (1994) studied fatigue of rapid and slow onset in isolated perfused rat and mouse diaphragms. They found that rapid fatigue is related to the frequency of stimulation, the time course of mechanical restitution and the time necessary for the Ca^{2+} channels of the SR to recover from inactivation. A reduction in the stimulation frequency caused diminution of the rapid fatigue pattern. Williams and Klug (1995) proposed that these fatigue induced changes in Ca^{2+} exchange may actually be beneficial by reducing the rate of energy utilization by the muscle fibre and preventing irreversible damage to the cell.

2.1.4.5 Metabolic Factors

High intensity exercise involves a high level of anaerobic metabolism. Consequently the concentration of high energy phosphates decrease and the concentration of inorganic phosphate (Pi), adenosine diphosphate (ADP), ammonia, lactate and H^+ ion increase as fatigue develops (Kirkendall, 1990; Fitts, 1993; Zelikovski, 1993). Although the tissue ATP concentration decreases during intense muscular contraction, this reduction does not appear to limit force output or cause muscle fatigue (Fitts, 1993). However, de Haan (1994) found a strong linear relationship between the relative decrease in ATP and the degree of fatigue. This strong relationship between the relative decrease in ATP and degree of fatigue is not necessarily causal and a third factor may be related to ATP decline which is the actual factor causing fatigue. It has also been suggested that declining phosphocreatine (PC) concentrations with contractile activity

could induce fatigue. The reduction in PC concentration and tension in contractile activity, however follow different time courses, making a causal relationship unlikely (Fitts, 1993). Phosphocreatine participates in the movement of ATP from the mitochondria to the cross bridges, a process called the PC - ATP shuttle. A critically low PC concentration may disrupt this shuttle system and slow the rate of ADP rephosphorylation to ATP, thus producing fatigue.

Muscular contraction involves the hydrolysis of ATP, producing energy and yielding ADP, Pi and H⁺ ion as end products. A major source of H⁺ ion production during intense muscular activity is the anaerobic production of lactic acid - the majority which dissociates into lactate and H⁺. The relative importance of ADP and Pi to other potential fatigue agents such as H⁺ is unknown (Fitts, 1993). If an enzyme of glycolysis is inhibited by a declining pH, then a reduction of ATP synthesis will occur (Kirkendall, 1990). An increase in H⁺ concentration could directly inhibit ATP hydrolysis, inhibit phosphofructokinase and thus reduce the rate of glycolysis. A reduction in pH inhibits the binding of Ca²⁺ to troponin C thereby reducing cross-bridge activation. It also reduces Ca²⁺ reuptake and subsequent release by inhibiting SR-ATPase.

Repeated bouts of high intensity activity can also result in depletion of the intracellular substrate, glycogen. Since glycogen is the fundamental fuel to sustain both oxidative phosphorylation and anaerobic glycolysis, fatigue is readily apparent as cellular resources are exhausted (McArdle *et al*, 1991; Fitts, 1993; Green, 1996). Whole muscle glycogen concentration does not appear to be a major factor in fatigue following short-term anaerobic work, yet selective glycogen depletion of type IIb fibres may reduce power output (Kirkendall, 1990).

Baker *et al* (1993) investigated the roles of metabolic and non-metabolic factors in human muscle fatigue in short duration exercise (2 minutes of maximum voluntary contraction) and long

duration exercise (15-20 minutes of intermittent contractions). Their major findings were that after short duration exercise, fatigue correlated closely with increased [Pi], and both force and [Pi] recovered within 5 minutes after exercise. Hydrogen ion concentration recovered more slowly than the recovery of force and [Pi]. There was also no significant difference in the recovery of [H⁺] in short duration exercise and long duration exercise. In short duration exercise, fatigue may be due to metabolic inhibition of contraction. Various studies have demonstrated a close correlation between changes in maximal force development and the metabolites Pi, and H⁺ during fatigue, and suggest that alterations in metabolite concentrations may play a role in regulating force development (Boska *et al* 1990; de Haan, 1994). Theoretically any means of enhancing the removal of the fatigue causing metabolites from the working muscles should delay fatigue in subsequent exercise and improve performance.

2.1.5 Fatigue in Endurance Exercise

Numerous factors have been linked to fatigue resulting from prolonged endurance activity, including depletion of muscle and liver glycogen, decrease in blood glucose concentration, dehydration and increases in body temperature (Kirkendall, 1990; McArdle *et al*, 1991; Fitts, 1993). Circulating blood glucose concentration has been suggested as a fatigue element due to its requirement by the CNS. Hypoglycaemia may affect mood or motivation, which would influence power output (Kirkendall, 1990).

Baker *et al* (1993) found that after exercise of long duration, force recovered more slowly than after short duration exercise, but that metabolic recovery was not slowed. They suggested that in long duration exercise there may be a longer lasting nonmetabolic component to fatigue that acts beyond the cell membrane, possibly at the level of excitation-contraction coupling or centrally.

In exercise of longer duration, a decline in motor drive may well limit force production. Bigland-Ritchie *et al* (1986) showed that the frequency of motor nerve firing decreases during sustained contractile activity. Each of these factors contributes to fatigue to a varying degree, and their relative importance depends on the environmental conditions and the nature of activity.

Since the present study does not involve prolonged endurance activity it is unnecessary to describe in detail the mechanisms of fatigue and the effect of recovery techniques on subsequent performance.

2.1.6 Other Factors Related to Fatigue

2.1.6.1 Effect of Blood Flow Limitation

When a muscle contracts, the intramuscular pressure rises. If this increases above a critical level it causes a progressive restriction of the blood flow and thus limits the rate of nutrient supply. For sustained isometric contractions the fatigue threshold generally corresponds with the onset of blood flow limitation. The longer endurance times associated with the onset of fatigue following intermittent contractions clearly result from unrestricted blood flow and reactive hyperaemia which partly replenish nutrient supply during each period of relaxation. The muscle energy demands are roughly proportional to the time for which the force is held while its replenishment is a function of the period of relaxation (Bigland-Ritchie and Woods, 1984).

2.1.6.2 Effect of Muscle Length

Fitch and McComas (1985) demonstrated the relationship of muscle length to its susceptibility to fatigue. The twitch and tetanic torques of muscles activated at optimum length following a fatiguing procedure were significantly reduced compared to muscles activated at a shortened position. They suggested that fatigue is related to the number of effective cross-bridge interactions during excitation of the muscle fibres. At the optimum length there is sufficient overlap of the actin and myosin filaments for all cross-bridges to be engaged. At short sarcomere lengths, however, opposing actin filaments overlap with each other and presumably interfere with cross-bridge attachment. This possibly explains the reduced tension that is developed in muscles activated at short sarcomere lengths.

It has been shown that torque generated by the fully shortened muscles is less than half that generated by these muscles at their optimum length (Marsh *et al*, 1981). The reduced force and lowered fatigue at shortened positions relative to longer positions were attributed to reductions in sarcomere lengths with a resultant reduction of energy consuming cross bridge interactions. Therefore muscle fatigue may also be related to the number of actin-myosin cross-bridge interactions and is unlikely to be accounted for solely on the basis of changes in the ionic composition of the transverse tubular fluid. If a reduced number of cross bridge attachments is indeed responsible for decreased tension developed at shorter lengths, it follows that there would be less consumption of ATP and precursors and less accumulation of H^+ and other byproducts of contraction (Fitch and McComas, 1985).

However in a similar study by Baker *et al* (1992) using nuclear magnetic resonance spectra, similar rates of ATP utilisation and intracellular concentrations of H^+ were found during contractions at different muscle lengths. They suggest that the reduced force and decreased fatigability of muscles exercised at shortened lengths found in this study and by Fitch and McComas (1985) may not be simply due to fewer cross-bridge interactions and relative ATP utilisation. Their data indicated that changes in inorganic phosphate (P_i) and $H_2PO_4^-$ concentrations correlated closely with force output in both fatigue and recovery. This suggests that phosphate has a role in regulating the rate of cross bridge cycling.

Sacco *et al* (1994) repeated the original study of Fitch and McComas (1985) with similar experimental findings. However the measurements of ATP cost of the contractions at different lengths and the recovery of force under ischaemic conditions led them to very different conclusions. The mean rates of ATP turnover were not significantly different at the two lengths and this suggests that any differences in the rates of fatigue at the two muscle lengths cannot be explained by different energy costs of the contractions.

The extent of action potential failure will be dependent on a number of factors, including the frequency of stimulation and the volume of the t-tubular lumen. A narrowing of the t-tubule lumen (or t-tubule openings), as might occur with shortening of the muscle fibre, could exacerbate the effects of ion accumulation within the t-tubular spaces, leading to the enhanced force loss that was observed. Also if failure of activation contributes to the additional force loss observed at shortened positions, it would follow that regions of the muscle fibres are effectively quiescent during stimulation, and this could account for the unexpected force recovery observed when the muscle was lengthened. They concluded that the metabolic cost of activity at shortened

and lengthened positions is the same and that excitation failure probably plays an important role in fatigue at shorter position.

2.1.6.3 Spinal Reflex Activity in fatigue

Schwellnus and Derman (1996) hypothesised that abnormal neurological reflex activity may be responsible for exercise associated muscle cramp (EAMC). Electromyographic data obtained from runners during EAMC revealed that baseline EMG activity is increased and that a reduction in baseline EMG activity correlates well with clinical recovery. Furthermore during EAMC the EMG activity is high and passive stretching is effective in reducing EMG activity, probably by invoking the inverse stretch reflex.

Abnormal reflex activity has also been observed in two animal studies. Nelson and Hutton (1985) investigated dynamic and static stretch responses in muscle spindle receptors in fatigued muscle of cats. Both group Ia and group II afferent fibres showed a significant increase in resting discharge and dynamic sensitivity during muscle fatigue. They proposed that spindle stretch sensitivity can increase tonic and phasic drive to alpha motoneurons and provide central neural excitatory compensation for declining isometric contractile tension. These neural and muscle adaptations would reflexly and mechanically result in a muscle more resistive to stretch.

Hutton and Nelson (1986) investigated alterations in Golgi tendon organ (group 1b afferents) in fatigued gastrocnemius muscle of cats. They found that group 1b afferent fibre discharge to a ramp stretch decreased significantly. Muscle spindle discharge interacts interneuronally and reflexly with Golgi tendon organ (tension detectors) which predominantly provide inhibitory input to the same motoneuron pools.

Local muscle fatigue is associated with increased muscle spindle afferent and decreased golgi tendon organ afferent activity (Nelson and Hutton, 1985; Hutton and Nelson, 1986). Muscle spindle activity has an excitatory effect on alpha motoneuron activity and Golgi tendon organ activity has an inhibitory effect on alpha motoneuron activity. Passive stretching invokes afferent activity from the Golgi tendon organ (Smith, 1994; Schwelonus and Derman, 1996) which interacts interneuronally and reflexly with spindle activity, thereby reducing alpha motoneuron activity. The benefits attributed to stretching during fatigue and exercise associated muscle cramps could possibly be explained by these reflexogenic effects. There does not appear to be any scientific evidence in the literature to support the above hypothesis. Considerable evidence supports post-junctional failures in skeletal muscle fatigue. Pre-junctional mechanisms involving the motoneuron pool and other central neural factors remain controversial (Nelson and Hutton, 1985).

More recently, Packer (1996) questioned whether exercise-produced oxidants are directly or at least in part responsible for fatigue, and whether antioxidant supplementation affects short term performance. Strenuous exercise induces physical stress. 'Leaks' in electron transport lead to the production of superoxide radicals. Superoxide radicals can react with each other to produce hydrogen peroxide, which may react with transition metals to produce the hydroxyl radical. During strenuous exercise the antioxidant defences may be overwhelmed and oxidative damage to cellular lipids, protein and or DNA may occur.

Packer (1996) found that when vitamin E-deficient rats ran to exhaustion, they became exhausted more quickly than vitamin E-sufficient rats. Vitamin E is the major membrane bound antioxidant, suggesting an important role for the sarcolemma in fatigue. This suggests that oxidants

and antioxidants do affect fatigue and subsequent performance.

Muscle fatigue is a complex phenomenon involving biochemical, physiological and psychological elements. Several basic mechanisms have been proposed for the fatigue process. One of these is the accumulation of fatigue causing metabolites causing fatigue in the working muscles (Zelikovski, 1993). Theoretically any means of enhancing the removal of the fatigue causing metabolites interfering with the physiological processes in the working muscles should delay fatigue in subsequent exercise and improve performance. Furthermore passive stretching, by altering the abnormal reflex activity could reduce fatigue.

2.2 Recovery

Adequate recovery from intense exercise is essential to optimize performance and reduce the associated symptoms of tiredness, fatigue and lethargy. Recovery from brief bouts (5-10 seconds) of intense activity or steady rate aerobic exercise is not associated with lactic acid accumulation and recovery is rapid. However longer periods of anaerobic exercise are performed at the expense of lactate accumulation in the blood and exercising muscles, and a significant disruption in other physiological processes and considerably more time is required for complete recovery (McArdle *et al*, 1991). The heart rate and cardiac output remain elevated after exercise while the oxygen debt is repaid and temperature returns to normal. Also local metabolites of anaerobic metabolism such as increased $[H^+]$ and $[CO_2]$, and decreased $[O_2]$ are all potent vasodilators and mediators of local blood pressure and thus blood flow control.

Both active and passive recovery methods are being used. In active recovery, often called 'cooling down', submaximal aerobic exercises are performed in the belief that this continued movement prevents cramps and stiffness and facilitates the recovery process. Passive recovery

techniques include rest, massage, cold showers, stretching, and adopting specific body postures. These methods are also used inbetween repeated bouts of high intensity exercise.

Recovery is initially rapid, and recovery time is dependent on the intensity and duration of the exercise.

2.2.1 Active Recovery

Thiriet *et al* (1993) suggested that different recovery strategies following maximal exercise seem to induce different lactate utilisation patterns without significantly affecting performance on one subsequent maximal exercise test. They examined the influence of passive (rest), active leg and active arm exercise with twenty minute recovery periods separating a series of four exhaustive exercises of up to two minutes duration. When the different types of recovery were compared, a more pronounced decrement in performance was found following passive recovery, and this was associated with higher peak blood lactate concentrations. They concluded that the type of recovery has a significant effect on blood lactate elimination kinetics, and that active recovery is beneficial in the preservation of performance during repeated maximal exercise. Furthermore, plasma shifts across the extra and intravascular spaces induced by maximal exercise, appear to closely follow blood lactate kinetics.

Gozal *et al* (1992) studied the effect of different modalities of exercise and recovery on exercise performance in subjects with sickle cell trait (HbAS). They assumed that even if performance capability is equivalent for a maximal exhaustive exercise, among comparable subjects with and without HbAS, sequential repetition of preferentially anaerobic maximal exercise bouts could lead to the accentuation of exercise triggered imbalances and thus help disclose possible differences in recovery processes between the two groups. They found a progressive decrease in performance and effort duration in both subjects with sickle cell trait as well as matched

controls, and that blood lactate concentrations were independent of other variables such as haematocrit, work, or power. Lower concentrations of lactate were observed in trained subjects with sickle cell trait without noticeable differences in power or heart rate responses. This suggests that there may be decreased lactate release from the exercising muscle or that other mechanisms may be involved. Although passive recovery was associated with higher mean blood lactate levels than active recovery, they found that no significant differences in performance were noted in either passive (rest) or active leg recovery, suggesting that the different acid-base kinetics in the exercising muscle as well as in the circulation induced by the type of recovery do not seem to influence subsequent anaerobic performance. Interestingly, significantly higher haematocrit levels with concomitantly lower fluid losses were observed during passive recovery in both groups. This suggests that the type of recovery greatly affects the net water shifts between the intra- and extracellular compartments with better reversal of exercise induced haemoconcentration during active recovery.

This observation may possibly provide some justification for the use of massage as a recovery technique during or after exercise. Massage has been purported to offset the negative effects of muscular exercise and this is probably gained by the mechanism of improving blood circulation of the exercised muscles (Cafarelli and Flint, 1992; Ma-Yue and Fu-Shiru, 1994; Viitasalo *et al*, 1995).

Belcastro and Bonen (1975) studied lactic acid removal rates during controlled and uncontrolled (self-selected) recovery exercise. They found that in controlled recovery periods, lactic acid removal rates were dependent on the intensity of the recovery exercise with optimal removal predicted at 32% VO_2max . Removal rates were faster during self regulated recovery (56.2 and 51.6% VO_2max), and during recovery exercise at 29.7 and 45.3% VO_2max than during rest or exercise at 61.8 and 80.8% VO_2max .

Weltman *et al* (1979) examined the effect of different recovery patterns following maximal exercise on blood lactate disappearance and subsequent performance. Recovery patterns consisted of passive recovery (PR), active recovery below anaerobic threshold, active recovery above anaerobic threshold and active recovery above anaerobic threshold while breathing 100% O₂. Their results indicated that:

- i. Elevated blood lactate levels have little effect on maximal effort exercise of 5 minute duration.
- ii. Exercise recovery following severe exercise will result in enhanced lactate disappearance.
- iii. Exercise recovery below anaerobic threshold is more effective for lactate disappearance than exercise recovery above anaerobic threshold.
- iv. The most effective lactate disappearance pattern was exercise recovery above the anaerobic threshold while breathing oxygen.

This suggests that enhanced delivery of oxygen plays a role in lactate clearance. Since blood is the medium by which O₂ is transported, any improvement of blood flow should therefore have an effect on lactate clearance. There have been reports on improved blood flow during massage (Cafarelli and Flint, 1992) and this effect could possibly account for some reports that massage aids recuperation from fatigue (Balke *et al*, 1989).

2.2.2 Passive recovery

Passive recovery may include rest, massage, cold showers, stretching and adopting specific body postures (McArdle *et al*, 1991; Gozal *et al*, 1992; Thiriet *et al*, 1993). Whilst these are reasonable recommendations, there is little scientific evidence to support these claims, and little theoretical basis for suggesting why these recovery methods may enhance recovery from fatigue.

2.3 Delayed Onset Muscle Soreness

Muscle soreness that occurs during intense exercise is most often caused by muscular compression of capillaries and hence an inadequate blood flow within the contracting muscles. This temporary soreness may persist for a few hours after unaccustomed exercise. Another form of soreness may occur several hours after the cessation of unaccustomed exercise, peaking 24-72 hours after exercise and subsiding after 5-7 days (Lambert and Dennis, 1994). This type of soreness is known as delayed onset muscle soreness (DOMS).

A considerable amount of information exists on DOMS, yet the mechanisms underlying this phenomenon remain unclear (Armstrong, 1990; McArdle *et al*, 1991; Lambert and Dennis, 1994; Smith *et al*, 1994). Although the precise cause is unknown, the degree of discomfort depends to a large extent on the intensity and duration of effort. Eccentric and to some extent isometric muscular contractions generally cause the most muscular soreness (McArdle *et al*, 1991; Lambert and Dennis 1994; Smith *et al*, 1994).

2.3.1 Theories of DOMS

2.3.1.1 The Spasm Hypothesis

A study by De Vries (1961) supported the spasm hypothesis of muscle soreness. Static stretching of the exercised muscles following repeated exercises produced significantly less pain than that occurring in non stretched controls. However Buroker and Schwane (1989) found that postexercise stretching has not consistently been shown to alleviate DOMS.

2.3.1.2 The 'Tear' Theory

The tear theory of muscle soreness proposes that minute tears or ruptures of individual fibres cause the delayed soreness. Because eccentric contractions can place greater strain on connective tissue and muscle fibres compared to concentric muscle action, it is possible that these contractions increase the likelihood of structural changes in muscle fibres. Friden (1984) observed disruptions of myofibrils and streaming of Z lines in muscle biopsies taken from patients with DOMS.

2.3.1.3 Metabolite Accumulation Theory

The excess metabolic theory proposes that prolonged exercise that follows a long layoff causes an accumulation of metabolites in the muscle. This accumulation of metabolites triggers osmotic changes in the cellular environment and fluid is retained. The oedema excites sensory nerve endings and this causes pain. There is little evidence to support this theory (McArdle *et al*, 1991; Lambert and Dennis, 1994) as :-

- i. The time course of metabolites returning to pre-exercise levels and onset and duration of DOMS are different.
- ii. DOMS is more severe after eccentric activity than concentric muscle work (McArdle *et al*, 1991; Lambert and Dennis, 1994; Smith *et al*, 1994), while the metabolic stress of

concentric work is about 5-7 times greater than eccentric work (McArdle *et al*, 1991), and the blood lactate concentration is much lower in eccentric work.

- iii. DOMS may occur after stretching a muscle, despite the fact that blood lactate concentration does not increase (Lambert and Dennis, 1994).
- iv. Patients with McArdle's disease do not show an increase in blood lactate concentrations, yet may still experience DOMS (McArdle *et al*, 1991; Lambert and Dennis, 1994).

2.3.1.4 Connective Tissue and Intramuscular Damage

This is a more scientifically plausible theory and the following observations have been made:

- i. Histological evidence of fibre disruption in muscles of human subjects immediately following eccentric exercise.
- ii. DOMS causes an increased leakage of intramuscular enzymes such as creatine kinase (CK) and lactate dehydrogenase (LDH) into the blood (Clarkson *et al*, 1986; Clarkson *et al*, 1992).
- iii. DOMS is associated with the excretion of high concentration of 3-methylhistidine in urine which is a non-metabolizable amino acid found in the actin protein of the muscle filament.
- iv. After exercise causing DOMS, there is a high concentration of C-reactive protein in the blood which indicates tissue damage.
- v. Following a marathon race, runners' leg muscle have intra- and extracellular oedema with endothelial injury, and dilation and disruption of the T-tubules (Warhol *et al*, 1985).

What remains controversial is the association between tissue injury, and the delayed onset of muscle pain. Smith *et al* (1994) proposed a sequence of events based on various experimental findings to explain DOMS.

- Connective and/or contractile tissue disruption occur during exercise involving unaccustomed eccentric muscle action. This evokes an inflammatory response. Within a few hours, there is significant elevation in circulating neutrophils, which migrate to the site of injury and predominate for several hours. The next wave of white blood cells, the monocytes, migrate to the injured area within 6-12 hours. They peak in number at 48 hours and are no longer seen at 72 hours.

- On exposure to the inflammatory environment, macrophages begin synthesizing large quantities of prostaglandin E (not established in the literature), which results in the significant increases in serum prostaglandin E (PGE). PGE sensitizes type III and IV 'pain' afferents (not established in the literature).

In explaining why DOMS is not experienced during rest but in response to movement the following is proposed. Oedema associated with DOMS does not produce a significant increase in intramuscular pressure at rest. However movement or palpation may exacerbate even small increases in pressure and thus provide a mechanical stimulus for pain receptors.

2.3.2 Physiological consequences of DOMS

2.3.2.1 Accumulation of calcium in the sarcoplasm

The excessive contraction at rest is thought to occur as a result of an influx of Ca^{2+} into the sarcoplasm, either through the Ca^{2+} channels or through physical disruption of the cell membrane. Uncontrolled rises in cytoplasmic Ca^{2+} concentrations activate a Ca^{2+} dependent proteolytic enzyme which preferentially degrades Z-discs, troponin and tropomyosin. A Ca^{2+} activated phospholipase also damages cell and other membranes.

2.3.2.2 Elevated plasma Creatinine Kinase (CK) activity

The mechanism causing increased plasma CK activity is not entirely understood. It appears to be exercise dependent and the delayed appearance in the plasma is thought to be due to the slow increase in membrane permeability or due to the delay caused by diffusion into the interstitial fluid and subsequent removal by the lymphatic system.

Delayed onset muscle soreness (DOMS) occurs in individuals in response to unaccustomed exercise. DOMS interferes with athletic performance, activities of daily living, adherence to exercise programmes etc. and a number of strategies to reduce DOMS are being employed. There does not appear to be a superior method to treat the muscle soreness. Both rest and low intensity exercise are commonly practiced by sports participants. The effect of massage after unaccustomed exercise shows somewhat equivocal results on DOMS (Wenos *et al*, 1990; Smith *et al*, 1994; Vitasalo *et al*, 1995).

The lay literature has recommended several possible ways to prevent DOMS, including static stretching before exercise (deVries, 1961), warm up prior to exercise, and alternate treatments

of heat and cold following a workout. While these are reasonable recommendations, there is little scientific evidence to support these claims and little theoretical basis for suggesting why these treatments might prevent DOMS (High *et al*, 1989).

Static stretching has been proposed as a mode to prevent or treat DOMS (deVries, 1961). If the muscles are damaged in the acute phase of DOMS, then stretching the muscle may exacerbate the injury, and be counter-productive. If tonic muscular spasms cause the temporary and residual soreness, then stretching may alleviate some form of soreness.

2.4 Massage

Massage is probably the most ancient treatment of the human body. More than 3000 years ago, systematic manipulations of the body tissues called massage were used in China (Hovind and Nielsen, 1974). The root of the word massage is derived from either the Arabic word 'mass'- 'to touch', the Greek word 'massein'- 'to knead' or the Latin word 'manus' - 'hand' (Cafarelli and Flint, 1992). Massage can be defined as ' the act of rubbing, kneading, or stroking the superficial parts of the body with the hand or an instrument.

Despite the long history of practical application of massage as an aid to preparing for exercise, speeding recovery and enhancing performance, there are very few rigorously obtained scientific data available in the literature that would substantiate the widely held belief in its effectiveness (Balke *et al*, 1989; Drews *et al*, 1990; Cafarelli and Flint, 1992; Viitasalo, 1995). This may be due to the inability to conduct a double blind study on the effects of massage both in preparation for and recovery from exercise. Furthermore, there is also a paucity of data and equivocal reports on the physiological changes that are thought to occur in massage (Hansen and Kristensen, 1973; Hovind and Nielsen, 1974; Samples, 1987). Sports massage is gaining recognition as an

important preventative and treatment modality (Zuluaga *et al*, 1991). Studies into the physiological effects of massage are continuing as improved sensitivity for measuring those parameters used as indices become available.

2.4.1 Techniques

In this study only two of the massage techniques were used viz. Effleurage and Petrissage (kneading). Effleurage is the technique that accustoms the subject to the physical contact of the therapist. It is composed of light gliding movements over the skin with no attempt to manipulate the deeper tissues. The technique is used to enhance relaxation and prepare the subject for further manipulation. When performed on the limbs, it is done from the distal to proximal direction so as to enhance venous return and lymphatic drainage (Zuluaga *et al*, 1991; Cafarelli and Flint, 1992; Norris, 1993).

Petrissage is a kneading and working of the tissues that is accomplished through a pressing and rolling action using repeated grasping, pressure and lifting or rolling. Connective tissue and muscles are gently squeezed and rolled with the fingers in a milking fashion (Zuluaga *et al*, 1991; Cafarelli and Flint, 1992; Norris, 1993).

2.4.2 Physiological effects

The physiological effects are achieved through mechanical, physiological and psychological processes. Some of the mechanical effects are reported to include reduction of induration, breakdown of adhesions and stretching of contracted tendons or ligaments. Physiologically there are reports of an increase in circulation and lymphatic flow (Norris, 1993). A secondary effect of massage comes from the heat of friction. Local heating is thought to increase venous compliance which would not necessarily augment blood flow (Cafarelli and Flint, 1992). The psychological effects may be related to a feeling of well-being following massage and the positive mood state. Kaada and Torsteinbo (1989) reported a moderate mean increase of 16% in beta-endorphin levels from 20.0 to 23.2 pg/0.1ml, lasting for about an hour after the termination of massage.

Table 1. Effects of Massage on Some Physiological Parameters

References	Effect
Wakim <i>et al</i> (1949)	50%↑.VOP, 7%↑.RBC
Ebel and Wisham (1952)	↔ VOP
Wakim <i>et al</i> (1954)	20%↑.VOP
Hansen and Kristensen (1973)	5%↑.VOP
Hovind and Nielsen (1974)	35%↑.VOP
Dubrovsy (1992)	42%↑ ¹³³ Xe, 2% ↑.O2 saturation
Arkko <i>et al</i> (1983)	2%↓.[Hb],4.5%↑.[K ⁺]
Tomasik (1983)	↔[La],↔[K ⁺],↔[Na ⁺]
Puustjarvi (1986)	↔ endorphins
Day <i>et al</i> (1987)	↔ endorphins
Kaada and Torsteinbo(1987)	↔ endorphins
Kaada and Torsteinbo (1989)	↔ VIP
Mortimer <i>et al</i> (1990)	16% ↑.endorphins
Boone <i>et al</i> (1991)	100%↑.lymph flow(ICT) ↔VO ₂ max.

Adapted from (Cafarelli and Flint 1992)

Key

↑ increase

↓ decrease

↔ no change

VOP- venous occlusion plethysmography

¹³³Xe- labelled xenon washout

ICT- isotope clearance technique

VIP- vasoactive intestinal peptide

2.4.2.1 Effect of Massage on Blood Flow

Studies on the effect of massage on measurements of blood flow and some blood borne substances are sparse and show extreme variability of the data (table 1, section 2.4.2). Most of the studies used venous occlusion plethysmography in which changes in blood flow are inferred from changes in limb circumference. Using this method it is difficult to distinguish between muscle and skin flow. While ^{133}Xe washout indicates some change in local tissue circulation, it is still not possible to obtain a quantitative expression for the blood flow in a single tissue.

When massage is applied to a limb, the manual pressure increases arteriolar pressure as well as emptying the veins. Momentarily there is a slight negative pressure in the veins which tends to draw blood through the capillaries. The rate of flow is therefore transiently increased without an increase in metabolism (Cafarelli and Flint, 1992). It is also uncertain whether the reported changes in blood flow are related to therapeutic effects of massage, but the squeezing effect of petrissage might be important for lymphatic drainage (Hovind and Nielsen, 1974).

Hovind and Nielsen (1974) measured skeletal blood flow during and after short application of massage (petrissage and tapotement). Following the application of these techniques, a very limited and brief increase in blood flow was observed with tapotement, whereas petrissage caused insignificant changes in blood flow. This difference might be ascribed to a more traumatic effect of the hacking movements of tapotement, probably mediated by the release of histamine like substances.

Hansen and Kristensen (1973) studied the effects of massage, short wave diathermy (SWD), and ultrasound (U/S) upon ^{133}Xe clearance from muscle and subcutaneous tissue. Effleurage, SWD and U/S was applied for 5 minutes to the calves of 12 human subjects. They found that during

the massage period the ^{133}Xe clearance in the muscle increased. SWD and U/S induced no significant changes in the ^{133}Xe clearance. In the subcutaneous tissue no changes occurred in the ^{133}Xe clearance during and after any of the treatments suggesting that the use of massage leads to an increased blood flow in the muscle. Effleurage may assist the blood flow by emptying the capillary bed and the veins, explaining why the increased rate of ^{133}Xe clearance was observed.

Compression and squeezing of the soft tissues will improve venous and lymphatic drainage. Interstitial pressure is increased and fluid absorption aided. Deep stroking and kneading of the calf muscle for a 10 minute period has shown to increase blood volume for 40 minutes (Bell, 1964).

Because the literature is sparse and data variable, it is difficult to come to a definitive conclusion about the effect of massage on peripheral blood flow.

2.4.2.2 Effects on blood borne substances

Similarly the data does not appear to demonstrate an effect of massage on blood borne substances (Cafarelli and Flint, 1992; Zelikovski, 1993). Kaada and Torstienbo (1989) found a 16% increase in plasma endorphin concentration after connective tissue massage. This may account for the feeling of well-being (Wiktorsson-Moller, 1983) and euphoria following massage (Kaada and Torstienbo, 1989).

2.4.3 Effect on Recovery

Despite the long history of application, both as an aid to physical performance and as a modality for facilitating recovery from the effects of vigorous exercise, the scientific data available to substantiate the widely held belief in its effectiveness are few and equivocal. Massage of fatigued limbs is regarded as a more effective technique than passive recovery. Sports massage is routinely recommended as an aid for speeding recovery from vigorous exercise (Balke *et al*, 1989; Cafarelli and Flint, 1992; Viitassalo, 1995) and has been purported to offset the negative effects of muscular exercise (chapter 1).

Massage techniques have been shown to be two or three times more likely to promote recovery following exercise than resting. Balke *et al* (1989) evaluated the effects of manual massage, mechanical massage and rest (no treatment) on recovery from overall physiological fatigue and muscular fatigue. This study consisted of two experimental designs. Overall physiological fatigue was assessed by a gradual exercise test on a treadmill and the subject's perceived level of maximal performance capacity (to a rating of 20 on the Borg scale), and this was corroborated with decreasing pulse pressure at maximal heart rates and ventilatory efforts. In the second experiment, muscular fatigue was assessed by evaluating maximum strength and maximum endurance of the right upper leg on the Lido exercise unit. Their results indicated that both manual and mechanical massage aid recuperation from fatigue more effectively than rest alone.

More recently, Viitasalo *et al* (1995) studied the effects of warm underwater water-jet massage on recovery from intense physical training. Neuromuscular function, selected biochemical parameters (serum creatinine kinase, lactic dehydrogenase, serum carbonic anhydrase, myoglobin, urine urea and creatinine) and muscle soreness were evaluated to assess recovery. The main findings of the study showed that a smaller decline in jumping power and a greater

increase in continuous jumping contact time occurred when underwater jet massage was used as a treatment. The serum creatinine kinase and serum myoglobin concentrations were significantly higher than in the control group, indicating an increased and more rapid leakage of proteins from the cells into the blood. The greater release of serum myoglobin during the treatment week, may be due to an accelerated wash-out of the protein already present in the extracellular spaces or that more damage was caused because they were able to jump more.

Zelikovski *et al* (1993) also studied the effects of the modified intermittent sequential pneumatic device (MISPD) on exercise performance following an exhaustive exercise bout. The metabolic blood data failed to show a corresponding change in the blood concentrations of the metabolites (lactate, pyruvate, ammonia, bicarbonate and pH) measured. The possibility that peripheral venous blood does not always reflect muscle metabolic changes has been proposed. There was however a marked improvement in the subjects' ability to perform subsequent exercise bouts. After exercise there is an accumulation of fluid in the interstitial spaces. This is due to an increase in blood flow to the working muscles along with an increase in blood pressure, as well as an increase in the osmotic pressure of the tissues from the accumulation of metabolites. The accumulation of fluid in the interstices can cause several effects:

- i. an increase in the diffusion distance between the capillaries and the muscle cells.
- ii. changes in membrane characteristics and,
- iii. an increase in venous pressure due to an increase of fluid with a resultant reduction in blood flow.

The effective removal of excess fluid that accumulates in the interstitial space after exercise could prevent the disturbance of muscle function and possibly explain the improvement in performance. The MISPD effected a 45% improvement in the subjects ability to perform the subsequent exercise bout. The MISPD creates a pleasant sensation on the muscles and

psychological reasons for improved performance cannot be ruled out.

Cafarelli *et al* (1990) studied the effects of percussive vibratory massage on recovery from repeated submaximal contraction. Their results indicated that there was no significant difference in the rate of fatigue in the control and vibrated groups and concluded that short term recovery from intense muscular activity is not augmented by percussive vibratory massage.

Drews *et al* (1990) studied the effects of post-event massage therapy on muscle recovery and performance in repeated ultra-endurance cycling. Their results indicated that post-event massage therapy does not expedite muscle recovery from ultra-endurance cycling as evaluated by muscle and liver enzyme efflux or performance.

The above studies indicate that there are equivocal reports on the effect of post-exercise massage on recovery from muscle fatigue. This may possibly be due to the variability of exercise protocols and massage techniques that were used in each study. Considering the widespread use of post event massage and the few equivocal reports of its effect in accelerating relief from muscular exercise, there is still a need to test the various techniques of massage on different exercise intensities and duration.

2.4.4 Effect of Massage in the Prevention and Treatment of DOMS

Delayed onset muscle soreness (DOMS) occurs in trained and untrained individuals in response to unaccustomed exercise (Lambert and Dennis, 1994; McArdle *et al*, 1991; Smith *et al* 1994). If massage is to alleviate DOMS, it must either interrupt the pathways involved in the sensation or alter the underlying process of cellular damage. Moderate to severe DOMS may interfere with athletic performance. This is most likely due to swelling, a reduction in the range of motion of the involved joint and a reduction in force output.

Massage has been shown to provide faster, more graded recovery from DOMS than rest (Smith *et al*, 1994; Viitasalo *et al*, 1995). Smith *et al* (1994), studied the effects of athletic massage on DOMS, creatinine kinase, and neutrophil count. They reported lower soreness scores in the massage group than in the control group. In this study massage was administered 2 hours after the exercise. The rationale for administering the massage 2 hours after the exercise bout was related to the neutrophil activity associated with the onset of acute inflammation. They hypothesised that massage had an impact on neutrophil accumulation and its effect in subsequent inflammatory events. Since massage appears to increase blood flow through the vascular bed (section 2.4.2), this increased flow rate in the area of microtrauma could interfere with neutrophil activity and subsequent inflammatory processes. Also the mechanical action of massage could shear marginated neutrophils from vessels' walls, and hinder migration of the cells from the circulation into the tissue spaces. They proposed that vigorous sports massage rendered two hours after the termination of unaccustomed activity, reduces the intensity of DOMS.

Wenos *et al* (1990) examined the effect of massage treatments on DOMS and the strength decrements normally associated with DOMS. Their results indicated that the treatment and control groups did not differ significantly on soreness perceptions. This study did not assess if massage had any effect in minimising or alleviating DOMS.

2.5 Muscle Stretching

In sports medicine, stretching can be beneficial in several areas. Increased flexibility resulting from stretching activities (Wiktorsen-Moller *et al*, 1983) may decrease the incidence of musculotendinous injuries (Shellock and Prentice, 1985; Norris, 1993; Smith, 1994; Brukner and Kahn, 1995), minimize and alleviate muscle soreness (de Vries, 1961; Noakes and Granger, 1990), and improve athletic performance (Shellock and Prentice, 1985; Taylor *et al*, 1990; Bandy and Irion, 1994; Smith, 1994; Worrel *et al*, 1994).

The role of stretching in alleviating or minimising muscle fatigue and DOMS has not been convincing (Section 2.5.6). Stretching is an important component of flexibility and overall fitness. Dynamic flexibility is important in athletic performance because it is essential for an extremity to be capable of moving through a non-restricted range of motion (Shellock and Prentice, 1985). If a muscle does not have enough elasticity to compensate for this additional stretch, it is likely that injury will occur in the musculotendinous unit.

2.5.1 Methods of stretching

The various stretching techniques can be summarised into ballistic stretching, static stretching, contract relax stretching and contract relax- agonist contract (Shellock and Prentice, 1985; Taylor *et al*, 1990; Smith, 1994; Magnusson *et al*, 1996).

2.5.1.1 Static Stretching

The static stretch is a method in which the muscle is slowly elongated to tolerance (comfortable stretch, short of pain) and the position is held with the muscle at the greatest tolerated length. Static stretching techniques are recommended because they are easy to perform and have little associated injury risk. The literature supports the fact that static stretch will increase the

flexibility of muscle. However, variability exists on the duration of the static stretch (Taylor *et al*, 1990; Bandy *et al*, 1994; Smith, 1994).

Duration of stretch

In vitro analysis has observed resistance decline to be less after the initial 12-18 seconds of static stretch (Taylor *et al*, 1990). Bandy and Irion (1994) studied the effect of time of static stretch on the flexibility of the hamstring muscles. Their results indicated that the change in flexibility was dependent on the duration of stretching. Thirty and 60 seconds of stretching were more effective at increasing flexibility than stretching for 15 seconds. In addition, they found no significant difference between stretching for 30 seconds and 1 minute, indicating that 30 seconds of stretching the hamstring muscle was as effective as the longer duration of one minute. Smith (1994) proposed that the static stretch should be held for 15-20 seconds and each muscle group should be stretched three to five times for maximum benefit.

2.5.1.2 The Ballistic Stretch

The ballistic stretch uses bouncing or jerking movements imposed on the muscle to be stretched. The quick, jerking motion that occurs during the ballistic stretch can theoretically exceed the extensibility limits of the muscle in an uncontrolled manner and cause injury (Taylor *et al*, 1990; Bandy and Irion, 1994). Ballistic stretching is also associated with an increased incidence of injuries (Norris, 1993; Brukner and Kahn, 1995).

2.5.1.3. Proprioceptive neuromuscular techniques

Proprioceptive neuromuscular techniques (PNF) are also being used for increasing flexibility (Shellock and Prentice, 1985). There are a number of different PNF techniques currently used for stretching, including slow reversal-hold, contract-relax, and hold-relax. All involve some combination of altering contraction and relaxation of both agonist and antagonist muscles. The rationale for hold-relax is that successive maximal excitations of motoneurons reflexly promote their subsequent inhibition (Condon and Hutton, 1987).

Condon and Hutton (1987) found that the different stretching procedures had no significant differential effect on the range of movement (ROM) achieved. The static procedure produced a range of movement comparable to that achieved with the more complicated procedures, and elicited little or no active resistance. Furthermore an antagonist contraction before stretching did not increase myoelectric activity to stretch. Therefore a more complicated procedure is not required. This provides a reason for the choice of static stretching procedure in this study.

2.5.2 Biophysical properties of stretching

Despite the widespread use of various stretching techniques in sport and rehabilitation, limited knowledge exists with respect to mechanisms and the efficacy of stretching of human muscle-tendon unit *in vivo* (Magnusson *et al*, 1996). The effectiveness has been based on neurological and mechanical factors. The biophysical properties responsible for resistance to stretch include both passive resistance components (visco-elastic properties of muscle, joint, subcutaneous and cutaneous structures) and active components (active muscle contractions of volitional and reflex origin). Any stretching effects mediated by reflex activity must involve the active component. The passive component resides in part in the connective tissue of the muscle-tendon unit. Muscle is considered to have both elastic and viscous properties (Taylor *et al*, 1990). Elasticity implies

that length changes, or deformations, are directly proportional to the applied forces, or load. Viscous properties are characterised as time-dependent and rate change dependent, where the rate of deformation is directly proportional to the applied forces.

Taylor *et al* (1990) studied the visco-elastic behaviour of the muscle-tendon unit and considered the clinical applications of these visco-elastic properties. They found that the behaviour of muscle in response to stretch can be explained by visco-elastic properties alone, exclusive of reflex effects. Denervated muscles respond similarly to innervated muscles suggesting that there is no significant force contribution from the stretch reflex in their model.

Increases in ROM produced by muscle stretching are often attributed to a decrease in active resistance produced reflexly, or volitionally induced inhibition to motoneurons.

The maintenance of stretch after the limit of joint range of motion is achieved by influences of the creep response of connective tissue. Creep is a visco-elastic property of biological tissues and is characterised by continued deformation at a fixed load. Hysteresis is the variation in the load-deformation relationship that takes place between loading and unloading (Taylor *et al*, 1990).

2.5.3 Spinal reflex activity during and after stretching

The musculoskeletal system has an inherent, built in protective device made up of the muscle spindles and Golgi tendon organs, which are highly sensitive receptors acting to prevent overstretch of the passive joint structures and muscle tendon unit respectively (Smith, 1994). The muscle spindle is attached to the intrafusal and extrafusal muscle fibres and is sensitive to active or passive stretch of the muscle. It detects the degree of stretch applied to the muscle and control its dynamic length via the stretch reflex.

The Golgi tendon organ located in the tendons (stretch receptors) are sensitive to tension and prevent overstretching in the opposite way to the stretch reflex. Activation of the golgi tendon organ receptors inhibits muscle contraction and induces relaxation via the the inverse stretch reflex.

The neurophysiological factors are based on neural inhibition of the muscle undergoing stretch. Guissard *et al* (1988) studied motoneuron excitability during three basic modalities of slow or static stretching of the human soleus muscle. Tendon and Hoffmann reflexes were analysed during static stretching, contraction - relaxation and contraction of the antagonistic muscles. Their results indicate that the static stretching not only avoids the excitatory afferents from muscle spindles, but that there is also inhibition of the motoneuron pool. The inhibition could result from inhibitory afferents originating from the Golgi tendon organs or from muscle spindle secondary afferents.

It has also been proposed that motoneuron pool excitability can be indirectly depressed by group Ia presynaptic inhibition of group Ia afferents. They have concluded that maximal mobilisation observed during static stretching is closely controlled by the excitability of the motoneuron pool. Furthermore inhibition of the motoneuron pool excitability is present in all three stretching manouvres and inhibition of the motoneuron pool stops as soon as the stretching is interrupted. The neurophysiological factors are based on neural inhibition of the muscle undergoing stretch: a decreased reflex activity resulting in reduced resistance to stretch (Magnusson *et al*, 1996).

2.5.4 Spinal reflex activity after exercise and in fatigue

In recovery from exercise, stretching is frequently being used. In a recent unpublished study, EMG data obtained from runners during exercise associated muscle cramps revealed that baseline activity is increased. A reduction in baseline activity correlates with recovery and, passive stretching is effective in reducing EMG activity (Schwellnus and Derman, 1996). He hypothesised that exercise associated muscle cramps are associated with abnormal spinal reflex activity which appears to be secondary to muscle fatigue. Local fatigue is associated with increased alpha motoneuron activity. Spindle activity has an excitatory effect on alpha motoneuron activity and Golgi tendon organ activity has an inhibitory effect on alpha motoneuron activity. Slow sustained stretch increases golgi tendon organ activity which has an inhibitory effect on alpha motoneuron activity. Abnormal spinal reflex activity has been observed in two animal studies (Section 2.1.6.3)

2.5.5. Effect of Muscle Stretching as a Method of Recovery

There does not appear to be any scientific evidence on the effects of stretching as a recovery technique. The relationship of flexibility and performance has been established (Shellock and Prentice, 1985; Worrel *et al*, 1994). The observation that fatigue is associated with increased alpha motoneuron activity and that static stretching results in inhibition of the motoneuron pool (Guissard *et al*, 1988), suggests that stretching may therefore theoretically have an effect on fatigue through reflex mechanisms.

2.5.6. Effect of Stretching on DOMS

Studies on the effect of stretching in minimising or alleviating DOMS are few and contradictory. If stretching is to alleviate DOMS, it must either interrupt the pathways involved in the sensation or alter the underlying processes of cellular damage (Buroker and Schwane, 1989). If spasm occurs in sore muscles and contributes to the pain, stretching could inhibit the spasm via the Golgi tendon organ reflex.

deVries (1961) studied the effects of static stretching following exercise soreness that was produced in both arms by repeated wrist hyperextensions. Muscles of the nondominant arm were stretched by the static stretching technique. He found that significantly greater levels of soreness were found in the dominant arm that was not stretched. This study supports the hypothesis that muscular distress following unaccustomed exercise is caused at least in part by tonic muscle spasm.

High *et al*, (1989) studied the effects of static stretching and/or warm up on the level of pain associate with DOMS. They found that static stretching and/or warm up does not aid in the prevention of DOMS associated with exercise. Similar findings were reported by Buroker and Schwane (1989) in a study on post-exercise static stretching and its effect on DOMS. This variation in findings may be due to that differences existing in the static stretch time and exercise protocols. The results of Buroker and Schwane (1989) and High *et al* (1989) do not prove that stretching is not effective in alleviating DOMS. However, they indicate that stretching does not always reduce soreness and further research is needed to clearly define the conditions under which DOMS is reduced by stretching.

The paucity of literature and contradictory reports on the efficacy of massage and stretching as recovery techniques suggests that further research is necessary to validate the popular use of these recovery techniques.

CHAPTER 3

Methods

3.1 Subjects and Experimental Design

Eighteen volunteer, moderately active subjects participated in the study. They were male between the ages 20 and 31 years and their characteristics are outlined in table 2. Subjects were informed of the potential risks and discomforts, and benefits of the study. Written consent was obtained from each subject prior to participation in the study (see appendix A). They were instructed to abstain from any vigorous activity during the three week period of the study.

Table 2. Characteristics of experimental subjects n= 18

	Age (years)	Height (cm)	Weight (kg)	Body Mass Index (kgm ⁻²)	Training (hours/week)
Mean	23.4	175	69.4	22.8	4.8
Std. Dev.	3.9	5.9	8.4	2.3	2.3

The study was designed on a 6 x 3 orthogonal latin square design to avoid bias due to treatment (massage, rest, or stretching) or week effect (table 3). Three subjects were randomly allocated into one of six groups.

Table 3 Orthogonal Latin square design

	Group 1	Group 2	Group 3	Group 4	Group 5	Group 6
Week 1	rest	rest	massage	massage	stretching	stretching
Week 2	massage	stretching	rest	stretching	rest	massage
Week 3	stretching	massage	stretching	rest	massage	rest

The subjects attended each session once a week, at the same time and venue. During each session the subjects performed five stages or sets of maximal heel raises of 45 seconds each. In between each stage of exercise, a recovery method was used.

The order in which the recovery methods (Rest, Massage and Stretching) were used depended on the group to which the subjects were randomly allocated. The following week, the same exercise protocol and another recovery technique was used, and on the following week, the remaining recovery technique was used.

3.2 Exercise Protocol

The exercise protocol consisted of the subjects performing as many heel raises as possible in 45 seconds. The subjects stood on a step with the forefoot placed at a fixed distance from the edge of the step. The amplitude of the heel raise was standardised for each subject. Therefore the amount of work done was dependent on the maximum number of repetitions per unit time.

Before the start of each session, the subject performed a single heel raise adjacent to a height stand and a horizontal bar was placed at a fixed height above the subject's head. The bottom end of the amplitude was delineated by a piece of soft foam placed under the subject's heel. The

subjects were instructed to perform the maximum number of heel raises possible in 45 seconds. During each heel raise, the subject was instructed to touch the horizontal bar placed above the subject's head and then to touch the foam below without bouncing off the foam. Failure to reach the horizontal bar above or foam below constituted an incomplete heel raise and was not counted.

3.3 Recovery Techniques

The effects of rest, massage and stretching as recovery techniques were tested in this study. The order of the three recovery techniques varied in the six groups to which the subjects were randomly allocated. The recovery period between each stage of exercise was three minutes.

3.3.1 Rest

Following each stage of exercise the subject sat on a chair for three minutes.

3.3.2 Massage

Following each stage of exercise, the subject lay prone over a treatment couch with a pillow placed under the distal leg. Vigorous massage was performed on both the triceps surae muscles by a qualified physiotherapist. The techniques used were effleurage and petrissage (section 2.4.1) and were performed for ninety seconds on each leg. No lubricating cream or powder was used during massage.

3.3.3 Stretching

The static stretching technique was used in this study (section 2.5.1). Prior to commencing with the exercise, the static stretching procedure was demonstrated to the subject. This technique involved stretching the triceps surae muscles in the walk-standing position whilst resting both hands forward on an examination couch. Both feet were placed flat on the floor. With the rear knee extended the subject was instructed to lean forward by slowly flexing the forward knee until a comfortable stretch, just short of pain was experienced in the rear calf muscle.

This method of static stretch was chosen because it is a relatively simple technique and is commonly used by athletes. Furthermore whilst the emphasis is on stretching the rear calf muscle, flexing the forward knee with the foot flat on the floor also stretches the lower calf muscle on the same side. The stretch was then maintained for thirty seconds. The subjects were then instructed to stretch the alternate side for another thirty seconds. The same stretching procedure was repeated but the stretch was maintained for one minute for each side.

3.4 Recording and Measurements

3.4.1 Calculation of Fatigue Index

The number of completed repetitions for each stage was counted and recorded on a record chart. One of the ways of measuring the decline in force output is to measure fatigue as a fatigue index. In this study the fatigue index can be defined as a percentage decline of number of repetitions from the first stage of exercise to the fifth stage of exercise.

$$\text{Fatigue index \%} = \frac{\text{no. of repetitions in stage 1} - \text{no. of repetitions in stage 5}}{\text{no. of repetitions in stage 1}} \times 100$$

The greater the decline in the number of repetitions from stage 1 to stage 5, the greater will be the fatigue index. Where there was no decline in the number of repetitions but an increase in the number of repetitions, the index was expressed as a negative percentage index. This negative index can be interpreted as a percentage increase in the number repetitions from the first stage to the fifth stage and indicates improvement.

3.4.2 Assessment of Muscle Soreness

Subjective ratings of soreness were assessed retrospectively at 0-12, 12-24, and 24-36 hours after each exercise session (see appendix C). Subjects rated the soreness if any, on a modified soreness scale: -1 = no pain, 10 = unbearable soreness. The reliability and validity of this in measuring experimentally induced soreness has been established in other studies (Buroker and Schwane, Lucille *et al*).

Subjects were also questioned on the recovery techniques that caused the least amount of soreness and those that caused the most amount of soreness during each exercise session.

3.5 Statistical Analysis

The design of the study was based on an orthogonal latin square design to avoid any bias or training effect. A series of statistical tests were done to determine if there was any training/week effect, treatment effect or group effect in the study.

$$Y_{ijk} = \mu + \alpha_i + T_j + \beta_k + \xi_{ijk}$$

Where Y_{ijk} is the observation in the i^{th} row
 k^{th} column,
for the j^{th} treatment

$\alpha_i = i^{\text{th}}$ week effect (ie. week 1,2 or 3)

$T_j = j^{\text{th}}$ treatment effect (massage, rest or stretch)

$\beta_k = k^{\text{th}}$ group effect (group 1,2, or 6)

Figure 1 Statistical model for a latin square

- i. A series of paired t-tests were used to compare the effects of static stretching, massage and rest during repeated bouts of dynamic exercise. Bonferroni adjustment was used to protect from type I error. In addition, Repeated Measures Analysis of Variance was used to compare the 3 methods with respect to the Index and total number of repetitions. These tests were also used to compare differences of DOMS in the three experimental groups.

- ii. Pearson correlation coefficients were used to determine if there was any correlation between the fatigue index and the total number of repetitions to the number of training hours per week and the body mass index.
- iii. The McNemar's Chi-square test was used to determine if there were significant differences in DOMS between the three weeks. This test was also used to compare the recovery techniques that caused the most and least amount of discomfort.

CHAPTER 4

Results

4.1 Study Group and Design

4.1.1 Characteristics of experimental group

The Mean and Standard deviation of characteristics of the experimental group listed in Table 2 indicated that the subjects trained for an average of 4.7 ± 2.3 hours/week and had a mean body mass index (BMI) was $22.83\text{kgm}^{-2} \pm 2.3$.

4.1.2 Correlation between fatigue index (%) to training(hrs/week) and BMI(kgm^{-2})

Table 4 shows the correlation between the fatigue index and the number of training hours for each of the three possible treatments sequences. There was a poor, non significant correlation. A similar poor correlation was shown between BMI and the fatigue indices.

Table 4. Correlation of fatigue index to training and BMI using Pearson's Correlation

Index	Training		Body mass index	
	r =	p =	r =	p =
rest-massage	-0.02	NS	0.12	NS
rest-stretching	-0.15	NS	-0.03	NS
massage-stretching	-0.11	NS	-0.12	NS

Pearson correlation: $r > 0.7$ - strong correlation

$r < 0.3$ - weak correlation

4.1.3 Correlation between the total number of repetitions to training and BMI

Poor correlation was found between the total number of repetitions, and training and BMI. The results are listed in table five.

Table 5. Correlation of total number of repetitions to training and BMI using Pearsons Correlation

Total no. of Repetitions	Training		BMI	
	r =	p =	r =	p =
Rest	0.03	NS	-0.31	NS
Massage	0.11	NS	0.03	NS
Stretching	0.08	NS	-0.11	NS

Pearson correlation: $r > 0.7$ - strong correlation

$r < 0.3$ - weak correlation

4.1.4 Analysis of study design

The Orthogonal Latin Square design was chosen to eliminate any bias, due to weekly exercise and group effect (ie. sequence of recovery methods). Analysis of variance was used to determine if there was any week or group effect. This design was effective because no significant differences were found in the three weeks of exercise or the six groups of the Latin Square design. This indicates that there was no influence on the sequence of modalities used. Furthermore there was no training benefit from the previous week/s. The results are listed in table six.

Table 6. Week and group effect on fatigue index and total number of repetitions

Variable	Week			Group		
	DF	F	P =	DF	F	P =
Index	2	1.80	NS	5	1.66	NS
Total Reps	2	0.56	NS	5	0.85	NS

DF= degrees of freedom

F= F statistic

4.2 Results of Variables

The fatigue index was used to measure the decline in the number of repetitions from stage one to stage five. A negative fatigue index indicates an increase in the number of repetitions from stage one to stage five and indicates improvement. The fatigue indices for each subject are shown in table eight. The mean values of the number of repetitions in stage 1, number of repetitions in stage 5, total number of repetitions and fatigue index are listed in table seven.

Table 7. Mean values and one sd of different variables in rest, massage and stretching

Variable	Rest	Massage	Stretching
Stage 1	54.83 (6.40)	48.72 (5.63)	49.72 (6.84)
Stage 5	50.11 (6.69)	56.0 (8.898)	53.72 (8.16)
Index %	7.78 (14.03)	-14.89 (12.92)	-8.556 (13.54)
Total Reps	266.28 (23.52)	269.33 (35.61)	262.61 (31.67)

Table 8. Fatigue Index (%) for each subject in the three experimental groups

Subject	Rest	Massage	Stretching
1	19	-34	-8
2	13	-23	-3
3	19	-25	13
4	4	-22	-10
5	7	5	17
6	9	-25	-26
7	-8	-29	-17
8	-21	7	-16
9	-12	-8	-23
10	-2	-20	-2
11	27	-2	-2
12	16	-5	-30
13	13	-29	-8
14	4	-6	2
15	38	3	-2
16	8	-10	-32
17	8	-21	0
18	-2	-24	-7
MEAN	7.78	-14.89	-8.56
SD	14.03	12.9	13.54
Improvement	27.8%	83.3%	77.8%
Fatigue	72.2%	16.7%	16.7%

Improvement (negative fatigue index) indicates the percentage of subjects that showed an increase in performance.

4.3 Effect of rest, massage and stretching on muscle fatigue

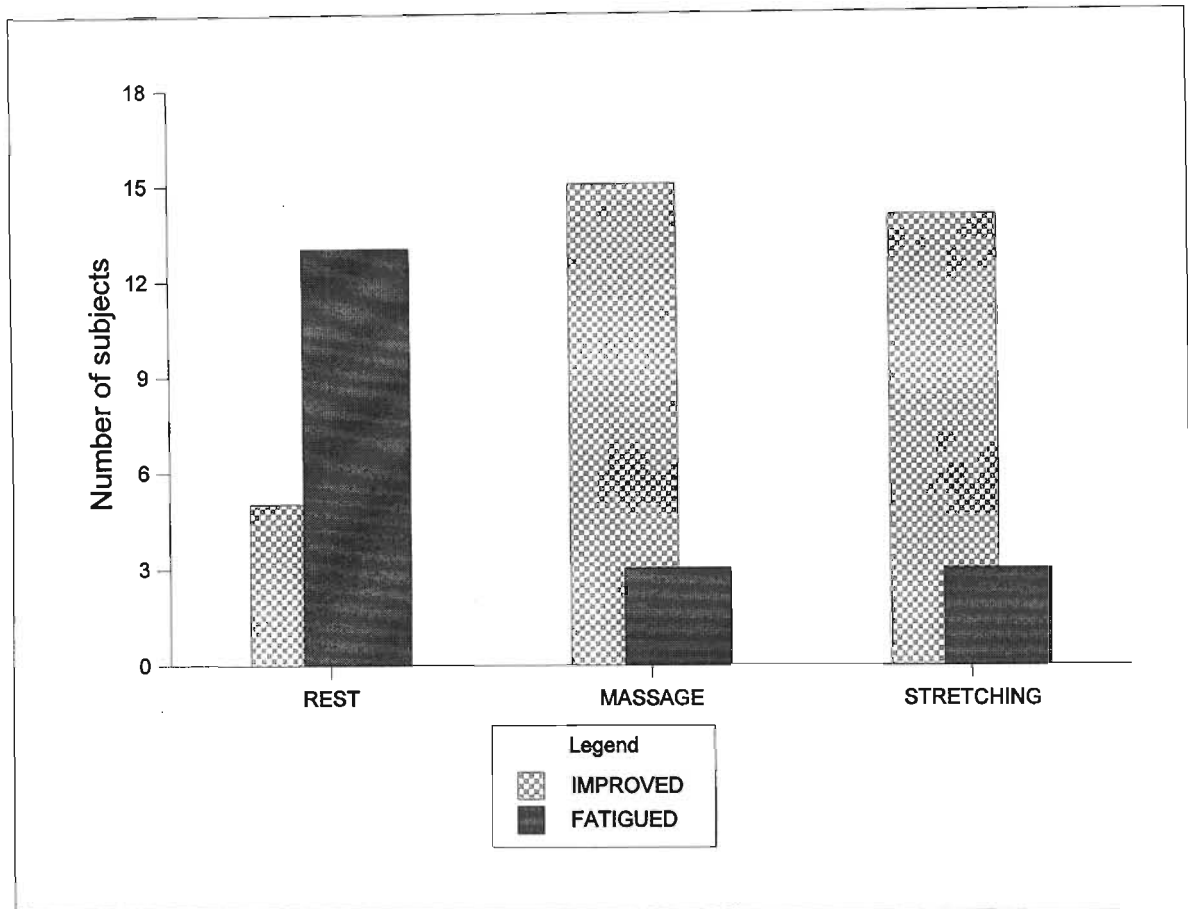


Figure 2. Subjects that improved or fatigued.

In all three experimental groups, there was both improvement (increase in number of repetitions from stage 1 to stage 5) and fatigue (decline in number of repetitions). Five (27.8%) subjects showed an improvement in the control group, whilst 14 (77.8%) and 15 (83.3%) subjects showed an improvement in the stretching and massage groups respectively. The difference between rest and the latter two groups was significant (McNemar's Chi-square test, $p < 0.001$).

Conversely, the recovery from fatigue varied, in that 13 (72.2%) out of 18 subjects showed a decline in the number of repetitions (fatigue) from stage 1 to stage 5 in the control (rest) group, while only 3 (16.7%) subjects showed a decline in the number of repetitions in both the massage and stretching groups. The mean fatigue index % for each method shows that there was greater fatigue in rest (control group) when compared to the massage and stretching groups (table 8).

Intergroup comparison was by Paired T-tests with the Bonferroni adjustment to protect from type I error. The results are listed in table nine.

Table 9. Statistical differences between mean fatigue indices

Recovery method	T test	P value
Rest and massage	5.03	0.0001
Rest and stretching	4.19	0.0006
Massage and stretching	-1.47	NS

Significance set at $P < 0.016$

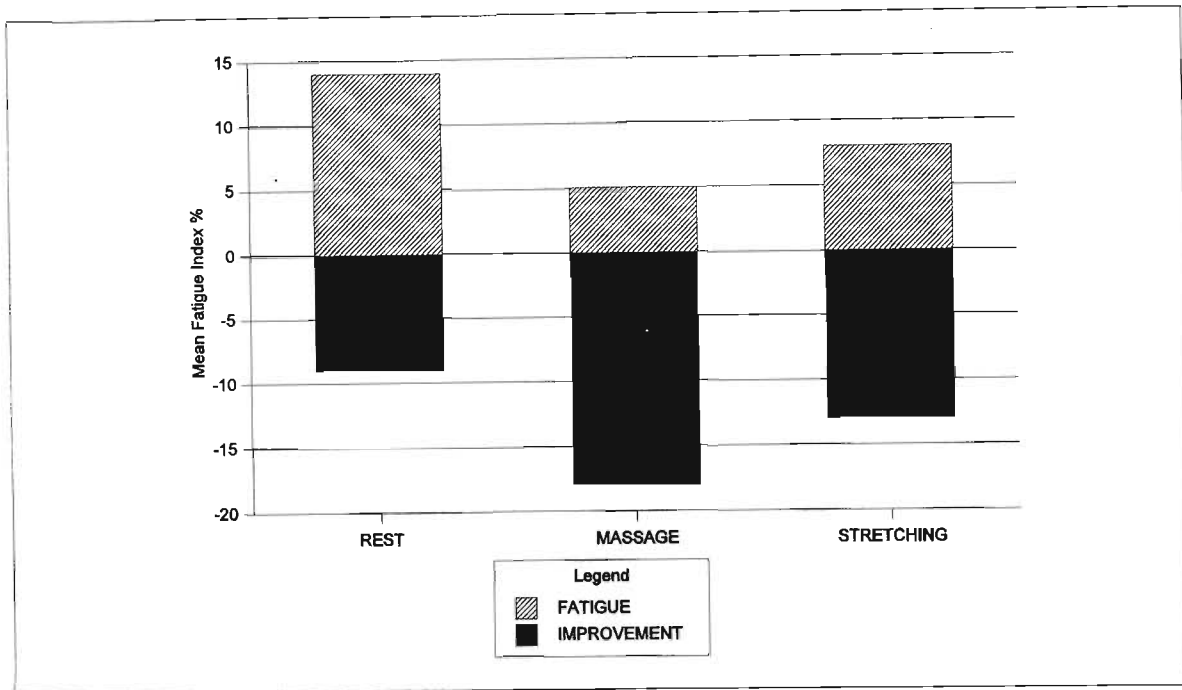


Figure 3. Graphical representation of mean fatigue indices in rest, massage and stretching

4.4 Delayed Onset Muscle Soreness

Figure 4 shows the number of subjects who developed DOMS in week one, two and three. The number of subjects in each experimental group who developed DOMS are listed in table 10.

Table 10. Subjects who had DOMS in week one, two and three

Week	Rest	Massage	Stretching	Total
1	4	5	6	15
2	2	0	4	6
3	1	1	2	4
Total	7	6	12	25

The McNemar's Chi-Square test was used to determine if there were significant differences of muscle soreness between the three weeks. Significant difference was found between week one and week two, and between week one and week three ($p < 0.01$). No significant difference was found between weeks two and three. This is an expected finding in that DOMS is usually induced by unaccustomed exercise.

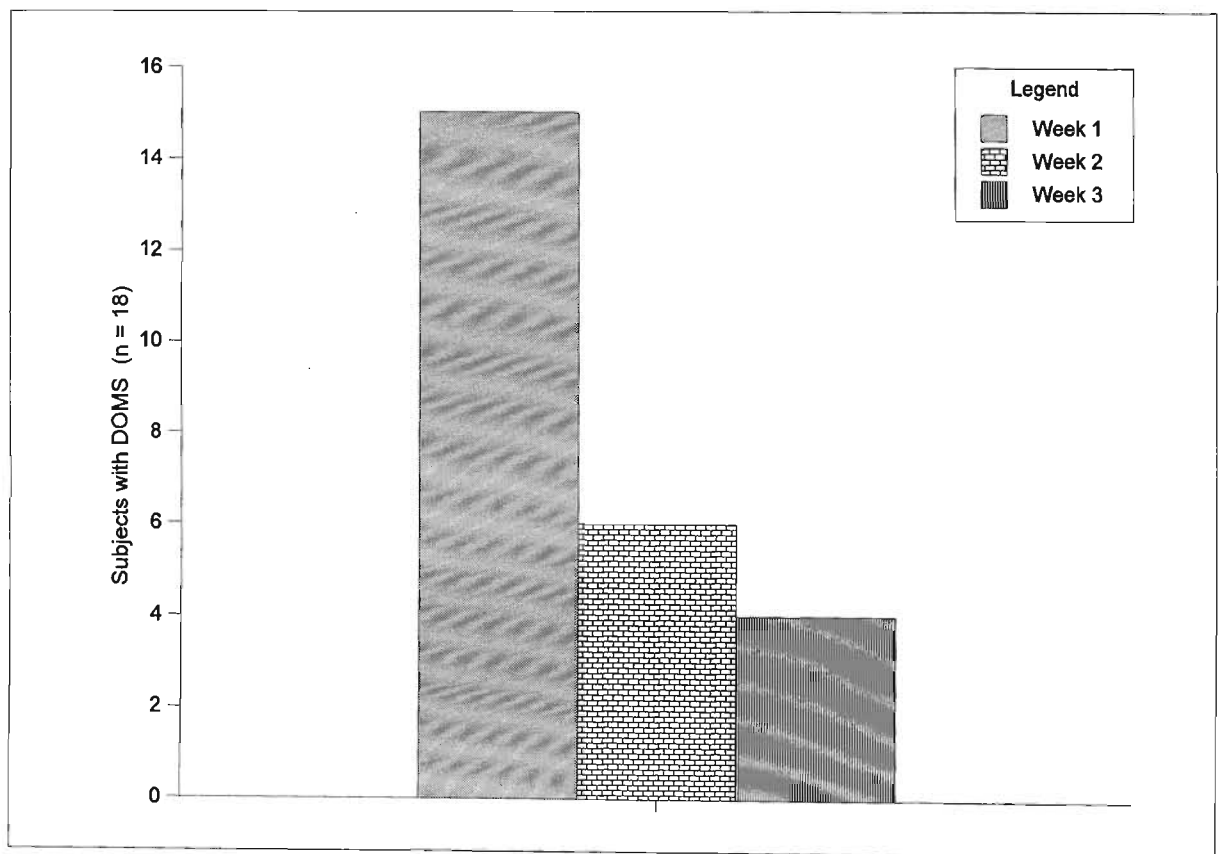


Figure 4 Number of subjects who had muscle soreness in week one, two and three.

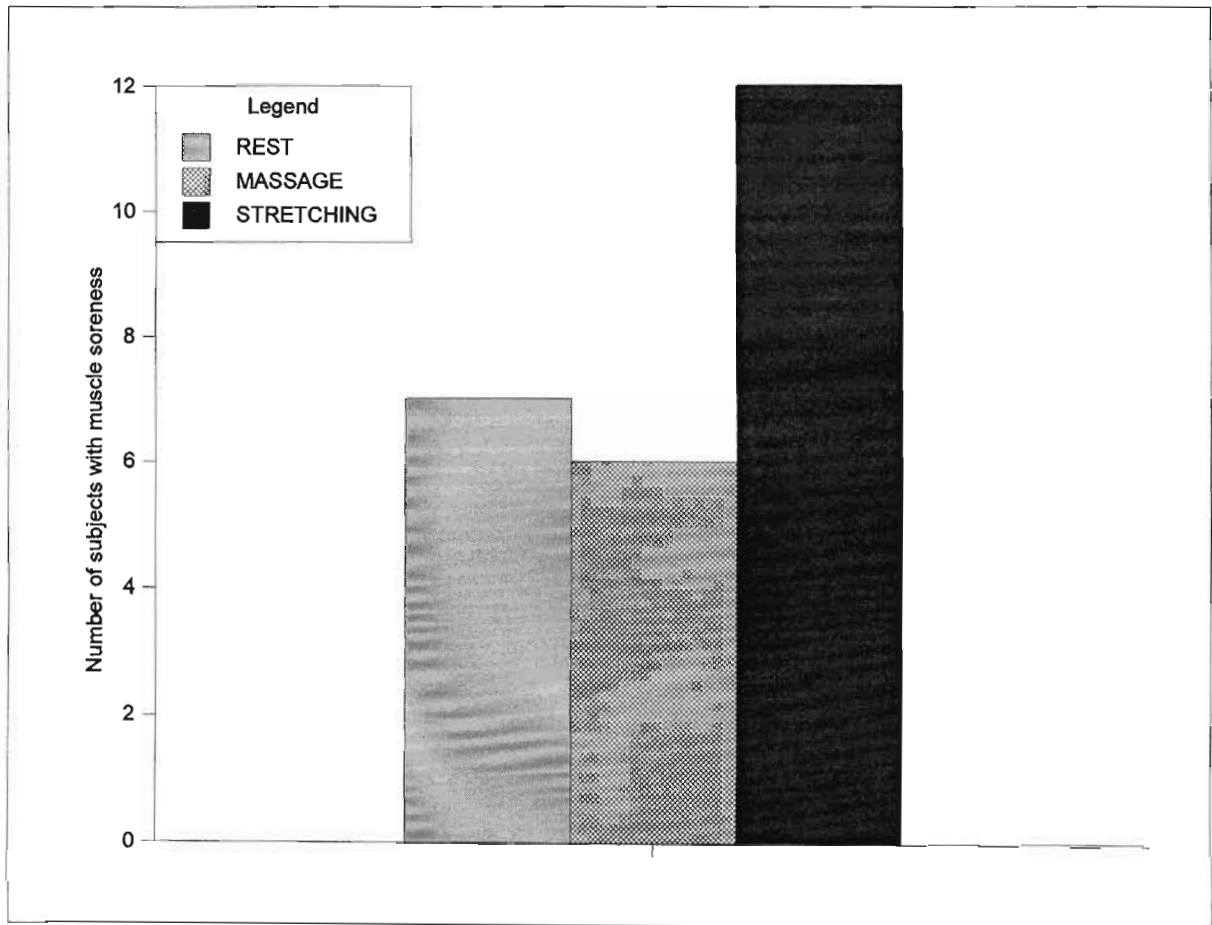


Figure 5. Number of subjects with DOMS after rest, massage and stretching

A representation of the frequency with which DOMS was associated with rest, massage and stretching is depicted in figure 5. This indicates that DOMS was most frequently observed with static stretching.

Statistical analysis of DOMS and the three experimental groups is listed in table 11.

Table 11 Statistical analysis of DOMS and rest, massage and stretching

Variable	T	P
Rest - Massage	1.26	NS
Rest - Stretching	-0.95	NS
Massage - Stretching	-2.15	<0.05

The differences of the mean ratings of muscle soreness between rest, massage and stretching were not significant. The difference between massage and stretching ($p < 0.05$) was not significant when the Bonferroni correction is applied. This could possibly be due to the small sample size. DOMS was associated most often with stretching and least often with massage. This finding is clinically significant because it indicates that massage might reduce the incidence of DOMS or that stretching may cause or exacerbate DOMS.

Ratings of mean muscle soreness in subjects who experienced DOMS, on a scale of 1-10, is shown in table 12. Higher soreness ratings were reported for rest (6.14) than massage (3.83) and stretching (5.33). The lowest rating of soreness was reported following massage. The above findings indicate that massage is associated with fewer number of subjects who developed muscle soreness as well as lower ratings of muscle soreness. This indicates the benefit of massage in alleviating or minimising the effects of DOMS.

Table 12. Mean soreness ratings and sd of different recovery methods between 12 and 36 hours

RECOVERY METHODS	MEAN
Rest	6.14(1.81)
Massage	3.83(1.07)
Stretching	5.33(2.17)

The onset of muscle soreness varied between 12 and 36 hours, with 16 subjects experiencing muscle soreness between 12-24 hrs after the exercise and 8 subjects between 24-36 hrs. In the majority of the subjects with DOMS subsided between 24 and 36 hours.

4.4 Muscle soreness during each experimental session

On retrospective analysis, the experimental sessions that caused the least amount of muscle soreness was massage, whereas stretching caused the most sorenes. These results are tabulated in Table13.

Table 13. Experimental sessions that caused the least and most amount of discomfort. n= 18

	Rest	Massage	Stretching
Least Soreness	1	16	1
Most Soreness	4	1	13

Least discomfort was associated with massage whilst most discomfort was associated with stretching. The Chi-square test ($p < 0.001$), showed significant difference between massage and stretching ($p=0.0005$).

Chapter 5

Discussion

Recovery from fatigue has important implications on subsequent exercise or activity. Facilitating the recovery process will result in improved performance in subsequent exercise. Various studies have demonstrated the effects of different methods to facilitate recovery from fatigue.

Most studies have focussed on active recovery (Belcastro and Bonen, 1975; Weltman et al, 1979; Gozal et al, 1992; Thiriet et al, 1993) and its role in facilitating lactate disappearance following an exhaustive bout of exercise. This is based on the concept that an increase in metabolites (lactate and H^+) are the causes of fatigue. These studies have demonstrated that active recovery (submaximal exercise) is more effective than passive recovery (rest) in facilitating lactate removal. Elevated lactate levels however, have been shown to have no demonstrable effect on subsequent maximal effort performance (Weltman et al, 1979). This indicates that other mechanisms apart from lactate clearance are also involved in active recovery. Recovery from fatigue not only involves re-establishment of metabolic homeostasis, but also recovery of non-metabolic factors such as spinal reflex activity, fluid homeostasis and the resting membrane potential.

Fatigue is due to a combination of factors that contribute to the sequence of events that result in decreased performance (section 2.1). Gozal et al (1992) and Thiriet et al (1993) found significantly higher haematocrit levels with significantly lower fluid losses in passive recovery (rest), and better reversal of exercise induced haemoconcentration during active recovery.

This indicates that active recovery also influences plasma shifts across the extravascular and intravascular spaces. At the end of exercise fluid accumulates in the muscles and in the interstitial spaces. The extracellular oedema can cause disturbance in membrane function and consequent disturbance in muscle function. The effective removal of interstitial oedema after exercise could therefore prevent the disturbance of muscle function with resultant improvement in function. The benefit associated with active recovery may be attributed to restoration of fluid homeostasis.

Massage and stretching are often used following exhaustive exercise in an attempt to facilitate the recovery process. Despite their frequent use, the mechanisms by which they work are somewhat unclear. Studies on the role of massage and stretching in recovery are few and contradictory (Balke, 1989; Cafarelli et al, 1990; Drews et al, 1990; Viitasalo et al, 1995). Also the role of massage or stretching in minimising or alleviating DOMS show equivocal reports in the literature (de Vries, 1961; Wiktorsson-Moller et al, 1983; Hill and Richardson, 1989; Buroker and Schwane, 1989; Wenos et al, 1990; Smith et al, 1994). These equivocal findings are probably due to the differences in the exercise protocols used to induce muscle fatigue as well as variability in the type and duration of massage and stretching.

The present study investigated the effects of massage and stretching in facilitating recovery during repeated bouts of dynamic exercise as well as on DOMS. Correlation analysis revealed that there was no correlation between training status and BMI of the subjects and the fatigue index or total number of repetitions performed. The orthogonal Latin square design enabled the effect of training during the study and the effect of the sequence of treatments to be investigated. No significant differences were noted between the three weeks of exercise sessions and the six groups of the study design on the variables fatigue index and total number of repetitions. This

indicates that there was no influence on the sequence of recovery method (rest, massage, and stretching). Furthermore there was no training benefit from the previous week/s.

The results of this study indicated that significant differences were found in the different recovery methods on the variable fatigue index (table 9). An analysis of variance showed a significant difference in the fatigue indices. Post hoc intergroup comparison using paired T-tests showed a significant difference between rest and massage ($P = 0.0001$) and rest and stretching ($P = 0.0006$). The differences between massage and stretching were not significant.

Of the eighteen subjects who participated in this study, fourteen (77.8%) and fifteen (83.3%) subjects showed an improvement in performance following stretching and massage respectively, whilst only five (27.8%) showed an improvement in the control (rest) group. The improvement following stretching and massage was significantly better than that following rest ($P < 0.001$).

The pattern of the fatigue between the three experimental groups also varied. Thirteen (72.2%) subjects showed a decline in the number of repetitions from stage 1 to stage 5 in the control group, while only three subjects fatigued in the massage and stretching groups ($P < 0.001$).

No other study has used the massage techniques (effleurage and petrissage) during repeated bouts of dynamic exercise. Bigland-Ritchie and Woods (1984) have suggested that the physiological events underlying fatigue are complex and commence at the onset of activity. Thus if massage is to have any effect on the physiological consequences of muscular fatigue, it would be logical to implement it early and during repeated bouts of exercise. The use of massage following a protracted period of exercise might not effect such changes, as the physiological change might be too great to be influenced by massage.

These results indicate that both massage and stretching are effective in delaying the development of fatigue during repeated bouts of exercise. The finding of this study that massage facilitates recovery and results in an improvement in subsequent performance has also been demonstrated by other studies (Zelikovski et al, 1993; Viitasalo et al, 1995). However the exercise protocols differed and the massage was done by mechanical devices. The finding of this study is in contrast to the findings of Cafarelli et al (1990) and Drew et al (1990). The differences in findings are probably due to the fact that different massage techniques were used. Cafarelli et al (1990) found that short term recovery from intense muscular activity is not augmented by percussive vibratory massage. Percussive vibratory massage is thought to increase local blood flow and facilitate the removal of fatigue causing metabolites. However, review of the literature on the effect of massage on blood flow is equivocal and not convincing. Drews et al (1990) studied the effect of massage on ultra-endurance events. The mechanisms of fatigue in endurance events are different to those of short duration, high intensity exercise. Furthermore the mechanisms of fatigue that are associated with endurance events also depend on the environmental conditions and may mitigate against the efficacy of massage on such fatigue.

Massage is postulated to have a similar effect to that of active recovery in restoring and reversing exercise induced haemoconcentration. Massage by virtue of its squeezing effect may affect fluid shifts across the intra- and extracellular compartments. The effective removal of fluid that accumulates in the interstitial spaces after exercise could therefore prevent the disturbance of muscle function which result in an improvement in performance. This effect has not been shown scientifically.

Other benefits of massage may also be attributed to the placebo effect and the release of beta-endorphins. Kaada and Torsteinbo (1989) demonstrated a 16% increase in beta-endorphins

following connective tissue massage. It is assumed that the release of beta-endorphins is linked with pain relief and a feeling of well being. This would have an effect on positive mood states and would exert a positive effect on performance.

Stretching has been shown to be significantly more effective ($p < 0.01$) than rest during recovery. Despite its frequent use during and after exercise, there does not appear to be any other scientific evidence on the effect of stretching during recovery following exercise. The observation that fatigue is associated with increased alpha motorneuron activity and that static stretching results in inhibition of the motorneuron pool, stretching may therefore theoretically have an effect on fatigue through reflex mechanisms (section 2.1.6.3). Muscle fatigue is associated with a feeling of tightness and sometimes cramping of the affected muscles. Stretching is frequently used to reverse such effects, by stimulating Golgi tendon organ activity and inducing relaxation via the inverse stretch reflex.

Both massage and stretching showed similar findings in delaying fatigue and improving performance, yet the mechanisms by which they are thought to affect muscle function differ. This does not indicate that either one is questionable in their effects. Muscle fatigue is a complex phenomenon and the various physiological events may be affected by a number of different factors, both metabolic and non-metabolic. Whilst there may not be parameters and means to validate the above assumptions, we must remember that it is clinically more important to find methods that would optimise recovery from exercise and that would result in the enhancement of performance.

A retrospective analysis of the recovery techniques that caused the least and most discomfort during recovery revealed that 89% of subjects associated massage with the least amount of

discomfort, while 72% associated stretching with the most amount of discomfort ($p < 0.001$). This suggests that massage may be more favourable as a recovery technique. The discomfort that is associated with stretching may cause athletes to be reluctant to stretch sore muscles and this may affect compliance with stretching routines following exercise.

DOMS is associated with unpleasant sensations, tightness and soreness of muscles and this would undoubtedly have an effect on exercise performance. Possible means to minimise or alleviate this soreness warrants further investigation. The effect of massage and stretching on DOMS showed very different findings. As would be expected significant differences were found between week one and week two, and between week one and week three ($p < 0.001$). Fifteen (83%) subjects developed DOMS after the first week, six subjects had muscle soreness in week two and four subjects had muscle soreness in week three. This indicates that the exercise protocol was of sufficient intensity to induce DOMS in the first week of unaccustomed exercise. Some short term adaptation would have occurred resulting in the significantly lower incidence of DOMS in the subsequent weeks.

DOMS was most frequently found in the stretching group. Twelve subjects developed DOMS in the stretching group with a mean rating score of 5.33. Seven subjects developed DOMS in the rest group with the highest mean rating score of 6.14. Only four subjects developed DOMS in the massage group with the lowest mean rating score of 3.83. These results indicate that the highest incidence associated with stretching and lowest incidence with massage. No significant differences were noted between the mean ratings of DOMS in the three groups. However, the difference between massage and stretching was not significant when corrected for possible Type I error ($p < 0.05$). This may possibly be due to the small sample size.

The lowest incidence and the lowest mean ratings of muscle soreness were found in the massage group. These findings are similar to those of Smith et al (1994) and in contrast to Wenos et al

(1990). Wenos et al (1990) did not evaluate the effects of massage on DOMS, but found that massage did not ameliorate the strength decreases normally seen with DOMS. The benefits associated with massage may be due to the fact that the effect of massage interferes with the sequence of events that lead the inflammatory processes found in DOMS. Other mechanisms may be due to beta-endorphin release and their role in minimising the sensation of pain or placebo effects.

The findings of this study indicate that post exercise stretching has no beneficial effect on exercise induced DOMS. This finding is similar to those of Buroker and Schwane (1989) and in contrast to that of de Vries (1961). A comparison to this study might not be valid because there were variations in the stretching time as well as the muscle disorders of his subjects. There have also been suggestions that stretching may in fact cause DOMS (Lambert and Dennis, 1994). Stretching sore muscles following exercise may actually exacerbate the intramuscular damage. This may possibly be the reason for the highest incidence of DOMS in the stretching group.

Whilst there was no statistical difference between massage and stretching on DOMS, clinically these findings may be significant. Post exercise massage was shown to minimise the effects associated with DOMS. This may certainly have an effect on performance. Stretching following exercise does not appear to have any beneficial effects on DOMS and may in fact exacerbate the effects or physiological processes associated with DOMS.

Both stretching and massage are better than rest in maintaining function during repeated bouts of exercise. Massage is associated with significantly less muscle soreness than stretching. A trend is shown that massage is associated with less delayed onset muscle soreness than stretching.

Limitations

In the exercise protocol that was used in this study, the bouncing effect could not be totally eliminated. Furthermore electromyography recordings could have been used to monitor the effects of fatigue and static stretching on myoelectric activity.

Chapter 6

Conclusion and Recommendations

6.1 Conclusions

- The findings of this study suggest that massage (effleurage and petrissage) and static stretching are more effective than rest in delaying the development of fatigue.
- Both massage and stretching have shown to be more effective than rest in facilitating recovery from exercise and improving subsequent performance.
- Massage is associated with a lower incidence, as well as lower ratings of muscle soreness in DOMS.
- Static stretching following exercise does not appear to have any beneficial effects on DOMS
- Massage is associated with significantly less muscle soreness than stretching during recovery from exercise.

6.2 Recommendations

- Massage and stretching (if tolerable) should be used at time-out, at half time during a game and during training to facilitate recovery and to enhance subsequent performance.
- Massage should be used routinely following unaccustomed, eccentric or strenuous activity to minimise or alleviate DOMS.

The results of this pilot study provides a baseline from which to persue further reseach on this subject.

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Appendix A

E.8 INFORMED CONSENT FOR INCLUSION IN A CLINICAL TRIAL[§]

E.8.1 I,(Name)

hereby consent to the following Procedure and/or Treatment being conducted on myself or the person indicated in (iv) below:

E.8.2 I acknowledge that I have been informed by:

(Name)

concerning the possible advantages and possible adverse effects which may result from the abovementioned procedure and/or treatment and of the ways in which it is different from the conventional procedure and/or treatment.

I,(Name)

hereby acknowledge that I understand and accept the "Information to Patients" leaflet handed to me in connection with this trial.

8.3 I agree that the above procedure and/or treatment will be carried out and/or supervised by

(Name)

8.4 I acknowledge that I understand the contents of this form, including the information provided in the "Information to Patients" leaflet and as the

*SUBJECT PARENT GUARDIAN OTHER

(Specify) freely consent to the above procedure and/or treatment being conducted on:

(Name

Signed

Date:

Subject/Parent/Guardian

Signed

Date:

Witness

Signed

Date:

Informat

Signed

Date:

Researcher

8.5 I am aware that I may withdraw my consent at any time without prejudice to further care.

[§]With the exception of the names and signatures in paragraphs 8.1, 8.4 and 8.5, please provide the above information.

E.9 INFORMATION GIVEN TO SUBJECTS

Please indicate what you will tell the subjects in simple language and addressed to them. The procedure or treatment which will be applied should be described and reference should be made to possible side effects, discomfort, complications and/or benefits.

NB. A translation into the home language of the patient must also be provided where subjects will be Zulu speaking. If no Zulu speaking subjects are to be included, please mention this below the English version.

It must be made clear to the patient that he/she is free to decline participate or to withdraw at any time without suffering any disadvantage or prejudice

The purpose of this study is to determine the effects of rest, massage and stretching in delaying the development of fatigue as well as on muscle soreness that follows unaccustomed exercise. This study has important implications as it will attempt to indicate the best recovery technique to use during and inbetween high intensity sports such as basketball and volleyball. By establishing the best form of recovery technique in delaying the development of fatigue, ones performance will be enhanced.

You will be required to attend 3 exercise sessions on three consecutive weeks. There must be no change in the routine during the 3 weeks of participation in the study and it will have to be performed at exactly the same time and same day for the next 3 weeks. The exercise will consist of a maximum number of heel raises 45 seconds followed by 3 minutes of a recovery technique (massage ,rest or stretching). This procedure will be repeated 5 times per exercise session. A heart rate monitor will be strapped over your chest to record yor heart rate.

The only complication that might possibly arise is some muscle soreness which you must have experienced after a heavy training session following a long lay off. The benefit is that this study may determine the best form of recovery technique that delays the development of fatigue which will definately help to enhance performance. These techniques could be used during time-off or half time to recover from fatigue so that performance is enhanced.

Appendix B

Personal Details and Record Sheet

Name :- Age :-
 Occupation :- Sport :-
 Height :- Weight :-
 Training (Hrs/week) :-
 Group :-

Session 1 - Recovery technique _____

Myoelectric activity at rest (standing) :- uV
 Myoelectric activity on heel raise (MEAHR) :- uV
 Heart rate at rest :-

Stage	No. of Reps	MEA start	MEA end	MEA ave	Heart Rate
Stage 1					
Stage 2					
Stage 3					
Stage 4					
Stage 5					

$$\text{Fatigue index} = \frac{\text{no. of lifts in stage 1} - \text{no. of lifts in stage 5}}{\text{no. of lifts in stage 1}} \times \frac{100}{1}$$

=

Notes

Session 2 - Recovery technique _____

Myoelectric activity at rest (standing) :- uV

Myoelectric activity on heel raise :- uV

Heart rate at rest :-

Stage	No. of Reps.	MEA start	MEA end	MEA ave	Heart Rate
Stage 1					
Stage 2					
Stage 3					
Stage 4					
Stage 5					

$$\text{Fatigue Index} = \frac{\text{no. of reps. in stage 1} - \text{no. of reps in stage 5}}{\text{no. of reps. in stage 1}} \times \frac{100}{1}$$

Session 3 - Recovery technique _____

Myoelectric activity at rest :- uV

Myoelectric activity on heel raise :- uV

Heart rate at rest :-

Stage	No. of Reps	MEA start	MEA end	MEA ave	Heart rate
Stage 1					
Stage 2					
Stage 3					
Stage 4					
Stage 5					

$$\text{Fatigue Index} = \frac{\text{no. of reps. in stage 1} - \text{no. of reps in stage 5}}{\text{no. of reps in stage 1}} \times 100$$

=

Appendix c

Questionnaire- muscle soreness

1. Did you experience any muscle soreness following each session of exercise?

	Yes	No
Rest	_____	_____
Massage	_____	_____
Stretching	_____	_____

2. How long after the exercise did the muscle soreness come on?

	0-12 Hrs	12-24Hrs	24-36Hrs
Rest	_____	_____	_____
Massage	_____	_____	_____
Stretching	_____	_____	_____

3. How would you rate the muscle soreness on a scale of 1-10

(1=no pain, 10= unbearable pain)

Rest	/10
Massage	/10
Stretching	/10

4. How long did it take for the muscle soreness to completely subside?

	0-12Hrs	12-24Hrs	24-36Hrs
Rest	_____	_____	_____
Massage	_____	_____	_____
Stretching	_____	_____	_____

5. Which technique caused the least amount of discomfort during the exercise protocol?

Which technique caused the most amount of discomfort during the exercise protocol?

Rest	_____	_____
Massage	_____	_____
Stretching	_____	_____