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## **DOCTOR OF PHILOSOPHY**

### **Exercise, neuromuscular control and performance, and stability of the knee joints**

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EXERCISE, NEUROMUSCULAR CONTROL AND PERFORMANCE, AND  
STABILITY OF THE KNEE JOINTS

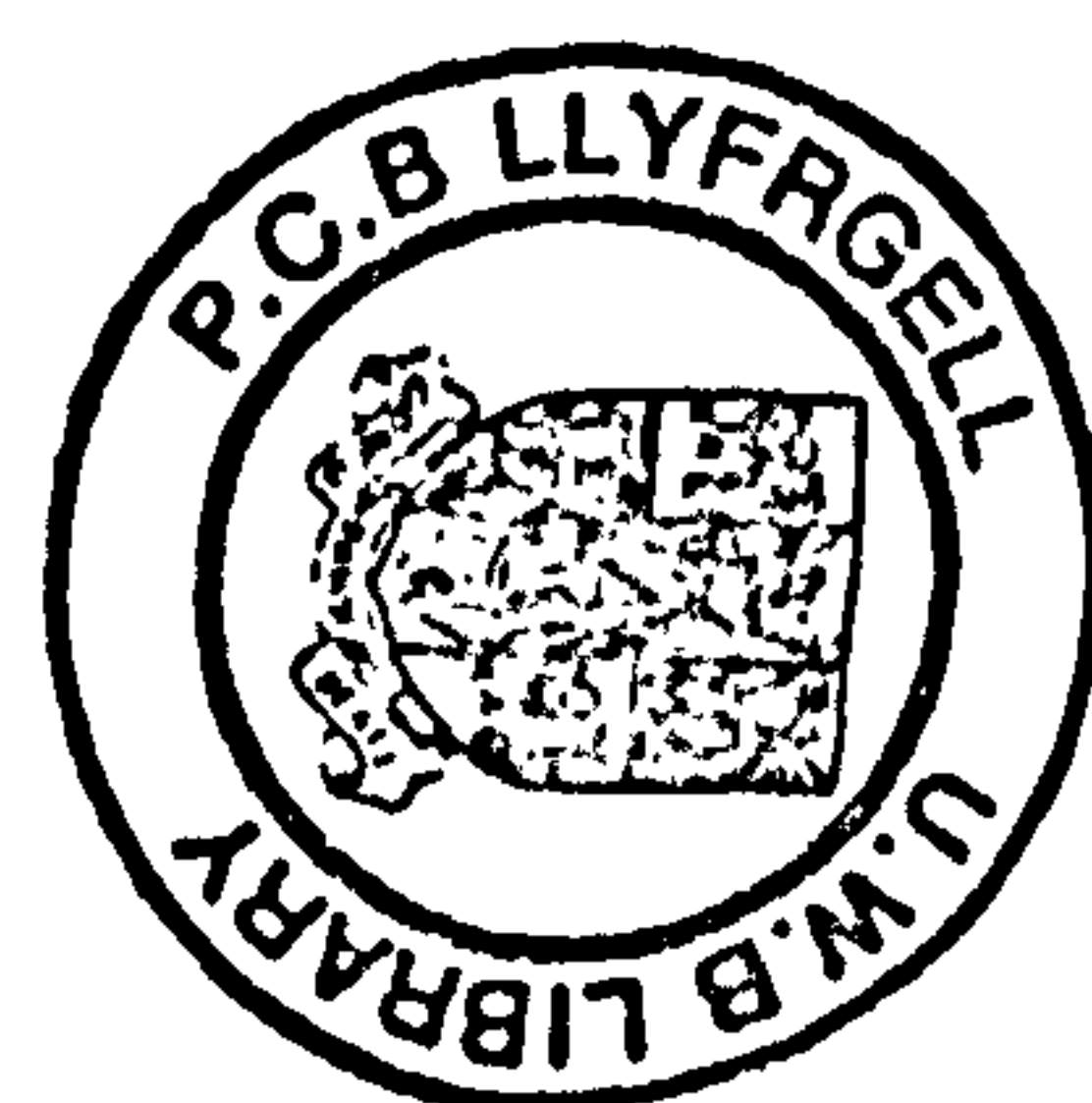
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Thesis submitted for the Degree of Doctor of Philosophy of the University of Wales

School of Sport, Health and Exercise Sciences

Autumn 2004

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# DEDICATION

بسم الله الرحمن الرحيم

## Dedication

( الاهداء )

أهدي هذا الاتجاز العلمي الى والدي ووالدتي  
والى زوجتي وأبنائي وبناتي



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# ABSTRACT

# **EXERCISE, NEUROMUSCULAR CONTROL AND PERFORMANCE, AND STABILITY OF THE KNEE JOINTS**

## **Abstract.**

In spite of an accumulating volume of information about injury to the musculoskeletal system in recent years, little scrutiny has been focused on the cause and prevention of injury. The large number of injuries to the knee joint and the anterior cruciate ligament ACL ligament in particular, is an alarming sign of the seriousness of this problem facing recreational and athletic people around the world.

The large number of injuries appears to arise from an unfavourable interaction between 'static' connective tissue-related and 'dynamic' muscle-related stabilisers of the joint system (Gleeson et al., 1998; Myers and Lephart, 2000). The functional stability is mediated ultimately by the neuromuscular system (Myers and Lephart, 2000) and it is important therefore to understand the function of the neuromuscular system as fully as possible in order to affect the aetiology and likelihood of injury favourably. The neuromuscular system includes biological 'machinery' offering capability for neuromuscular control (proprioception) and neuromuscular performance (motor actions).

The neuromuscular control was quantified by newly-developed and laboratory-based assessments involving the dynamic muscular reproduction of a 'blind' target force and the error associated with such efforts may provide greater efficacy of measurement compared to those tests used in contemporary practice which may not indicate an individual's true functional capability to resist injury threats, in particular, to assess the muscular dynamic intensity associated with sporting endeavours which may functional capability to resist injury threats may be compromised.

This thesis is presented as a series of three studies. The aim of this study was to examine between-day reproducibility and single measurement reliability of objective and self-perceived indices of force error in the knee flexors and extensors in men. Results showed that the reproducibility and single-measurement reliability of objective (CE%, VE %) and self-perceived performance (SPCE% and SPVE %) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 offered compromised precision and efficacy. These indices of performance should be deployed cautiously within both case-study and inter-individual comparisons and must rely on multiple-trial protocols to achieve acceptable levels of measurement precision in such circumstances.

The aim of the second study was to examine the effects of serial bouts of acute fatiguing exercise on objective and self-perceived indices of neuromuscular control and performance of the knee flexors in men. In summary, results showed that despite substantial fatigue-related strength and muscle activation impairments, neuromuscular control as measured objectively by NCA1, NCA3 or RJA was not influenced significantly by the fatigue task intervention and that capability in this aspect of neuromuscular control was preserved in response to this type of exercise stress. Furthermore, constant error associated with self-perceived performance was similarly not influenced significantly by the fatigue task intervention. It is plausible



that in order to provide optimum protection of synovial joints, neuromuscular control of the knee flexors may be preserved preferentially to neuromuscular performance when challenged by fatigue-related exercise stresses.

The third study assessed concomitant effects of an episode of muscle damage interspersed amongst serial bouts of fatigue on objective and self-perceived indices of neuromuscular control and performance of the knee flexors. This type of exercise perturbation reflects that associated with acute, transient and metabolically-focused effects (fatigue) and that during a more prolonged, mechanical type of disruption to the muscle (EIMD). It was likely to mimic some of the patterns of exercise inherent in sports and training practice and may serve to disrupt dynamic stabilization around the knee joint.

Results showed that despite substantial EIMD and fatigue-related strength impairments (the former confirmed in this experiment by means of indirect physiological descriptive variables including a reduced range of movement and an increase in pain upon active movement of the affected limb), neuromuscular control as measured by objective and self-perceived indices of constant and variable error in assessment protocols NCA 2, NCA3 and DNCA was not influenced significantly by the EIMD and fatigue task intervention. It was notable that capability in this aspect of neuromuscular control was preserved in response to this type of exercise perturbation in the three different modes of objective assessments of neuromuscular control used in this study.

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CHAPTER 1

INTRODUCTION  
&  
LITERATURE REVIEW

## 1.0 Introduction and Review of Literature

### 1.1 *Injury to synovial joints.*

The participation of men and women in intercollegiate athletics has been increasing due to the growing numbers of sport activities in the last twenty years around the world; and there has also been an increase in the incidence of sports activity-related injuries (Anthony et al., 1998; Laurie et al., 2001). Injuries have become one of the most significant dilemmas and frustrating matters facing athletes at all levels, despite the advancement of medicine and facilities. For example, the growing number of men and women participating in soccer programmes since 1989 is 9% and 50% respectively (Arendt and Dick, 1995). Furthermore it has been shown that incidence of injury for male outdoor players (> 16 years) are 12 to 35 injuries per 1000 match-hours and 1.5 to 7.6 injuries per 1000 training- hours (Dvorak and Junge, 2000).

These injuries are due to intrinsic and/ or extrinsic factors. Intrinsic and extrinsic factors contribute to the increasing number of knee injuries and subsequently ACL integrity. Intrinsic factors are; anatomic factors such as intercondylar notch, ACL size (Harner et al., 1994), joint laxity (Arendt and Dick, 1995), lack of conditioning or training (Hewett et al., 1999), biomechanical insufficiency (Gleeson et al., 1998) the loss of neuromuscular control (Carafa et al., 1996; Hewitt et al., 1999).

Extrinsic factors are described as adjustable variables such as muscular strength and conditioning, body movement in sport, shoe-surface and level of skill and conditioning (Arendt and Dick, 1995). Both factors contribute directly or indirectly to the body joint injuries such as strains, sprains and dislocations which occur each year.



The knee is the most commonly injured joint, and serious damage to the knee ligament is recurrent (Kelsey, 1982; Miyasaka et al., 2001). The anterior cruciate ligament (ACL) is one of many ligaments present in the knee and is the principle ligamentous restraint to anterior tibio-femoral displacement (Rees, 1994). The ACL is the most frequently injured ligament in the knee joint (Rees, 1994; Griffin et al., 2000).

Athletes may sustain anterior cruciate ligament rupture by one of two mechanisms, either a direct or an indirect setting. However, there is accumulating evidence of an ACL injury epidemic by means of non-contact mechanisms in team sport athletes (Noyes et al., 1983; Anthony and William, 1998). This injury occurs when the flexor muscle group fails to provide stability to the knee joint and subsequently to the ACL by regulating anterior tibio-femoral displacement and tibio-femoral rotation as a result of eccentric contraction exerted by the knee extensor muscle group (Arms et al., 1984). This mechanism of injury is greatest between 30 degrees and near full extension (Anthony and William, 1998; Cerulli et al., 2000).

### *1.2 Stabilisation of a synovial joint and protection from injury.*

The goal of any athlete is to achieve optimal protection and subsequently reduce the number of injuries in the field during training and game situations. This outcome may be achieved in two main ways. First, one of the main roles of the lower extremity is to maintain the knee joint in a position that allows for it to function with maximum protection from injury during physical activity. This maximum protection (functional stability of the joint) depends on a favourable interaction between the static (osseous geometry, capsular structures and menisci) and dynamic

muscle stabilizers (Fu, 1993; Garrett et al., 1992). Second, an appropriate interaction in the communications between the coach, sports medical team and the athlete in considering the consequences of weaknesses in performance capabilities and the associated risks of musculoskeletal injury should ensure agreed perceptions of needs to be addressed by conditioning.

Functional stability has been defined as having adequate stability to perform functional activity and results from the favourable interaction between the static and dynamic muscle stabilizers (Garrett et al., 1992; Joseph and Lephart, 2000). The nature of the interaction between the static and dynamic muscle stabilizers of functional stability is regulated by the neuromuscular system. The neuromuscular system encompasses the potential for precise afferent sensory information (neuromuscular control) that might regulate effectively the capability for neuromuscular performance associated with efferent motor response.

The knee joint is one of the most complex and most commonly injured joints (Rees, 1994). Serious damage to knee ligaments is recurrent (Kelsey, 1982; Miyasaka et al., 2001). The anterior cruciate ligament (ACL) is one of many ligaments present in the knee and is the principle ligamentous restraint to the anterior tibio-femoral displacement (Rees, 1994). The ACL is the most frequently injured ligament in the knee joint (Daniel and Fritschy, 1994; Rees, 1994; Griffin et al., 2000).

There is accumulating evidence of an ACL injury epidemic by means of non-contact aetiologies in team sport athletes (Noyes et al., 1983; Anthony and William, 1998). This injury often occurs during eccentric action of the knee extensors, when the



flexor muscle group fails to assist effectively in the stabilization of the knee joint by not regulating either anterior tibio-femoral displacement or tibio-femoral rotation (Arms et al., 1984). This process is thought to confer levels of mechanical stress on the ACL that may threaten its integrity, especially between 30 degrees and 0 degrees of knee flexion [0 degrees defines full extension of the knee anatomically] (Anthony and William, 1998; Boden et al., 2000; Gleeson et al., 1998).

Each year disruption of the anterior cruciate ligament occurs in one in three thousand individuals in the general population within the United States. This is equivalent to a rate of 250,000 ACL injuries annually (Boden et al., 2000). The numbers of injuries has continued to increase with burgeoning participation in sports and recreation (Laurie et al., 2001).

There are a number of factors that have been associated with to knee joint and anterior cruciate ligament injuries. For example, anatomic factors such as intercondylar notch, ACL size (Harner et al., 1994), lack of conditioning or training (Hewett et al., 1999), biomechanical insufficiency (Gleeson et al., 1998), shoe-surface interface and playing surface (Boden et al., 2000), and losing of neuromuscular control (Caraffa et al., 1996; Hewett et al., 1999). However, little attention has been focused on the underlying mechanisms and prevention of injury in athletes (Boden et al., 2000). Figure 1.1 outlines some of these factors and how they might be interrelated with other integral neuromuscular and musculoskeletal parameters.

Figure 1.1. Locus of anterior cruciate ligament (ACL) injury.

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Figure 1.1: Modified from Minshull (2004). Neuromuscular Performance and Exercise Stress Associated with the Stabilisation of Synovial Joints. Unpublished PhD thesis.

There is no doubt that injury is increasing as population and more specifically elite athletes increase their participation in sport activities, training and recreation (Gandevia et al., 1995; Kelsy, 1992; Gabbett, 2000). Therefore focus on prevention is important. It is important in terms of cost, effort and time (Draganich and Vahey, 1990). Prevention from injury has become a focus among sports medicine teams because of its positive role on athletes in terms of the financial and emotional impact on them (Boden et al., 2000).

Amongst many factors that have been considered as potentially important to the protection and stabilisation of the joint, such as strength, flexibility and ligament compliance or stretchiness, bone geometry, improved proprioception performance, capacity is one of only two such factors that has been consistently linked to a reduction in the incidence of ligamentous injuries in the knee joint.

For example, Caraffa et al., (1996), in a prospective random-allocation control study, demonstrated that the incidence of ACL could be reduced 7-fold with a season-long proprioception training programme. Similar studies have confirmed the role of the neuromuscular system in protection and stabilizing the knee joint (Hewett et al., 1999; Junge et al., 2002)

The neuromuscular system has a significant role in preventing injuries and controlling movement (Barrack et al., 1994 Lephart and Fu, 1995; Hewett et al., 1999, Myers and Lephart, 2000). Every part of the body system has a role to play to fill full the requirement of the body functions in daily life and, more importantly, during sport activities. The function ability, stability and joint stiffness of daily life and sport activities depends on the interaction between static and dynamic muscle stabilizers that provide joint stability (Gleeson et al., 1998; Fu, 1993).

Riomann and Lephart (in press) defined the functional ability as having adequate stability to execute functional activity and result from the interaction between the static and dynamic stabilizers. The interaction between the static and dynamic stabilizers of functional stability is controlled by the neuromuscular system (Myers and Lephart, 2000).

This joint functional stability depends on information from the neuromuscular control (proprioception) which travel from the knee joint and surrounded muscle through afferent pathways to the CNS, where it is integrated with input from other levels of the nervous system, eliciting efferent motor response (neuromuscular regulation process) to maintain and regulate the movement patterns and functional



stability of the knee joint. Sherrington was the first to introduce the term proprioception and defined it as *afferent information arising from the “proprioceptive field” and specifically ‘proprioceptors’* (Sherrington, 1906; Riomann and Lephart in press).

Neuromuscular control (proprioception) originates in the ligament, tendons, capsules, and menisci within the knee joint and muscle surrounding the knee (Lattanzio and Petrella, 1998). There are four different kinds of proprioceptive which have been recognized in the articular structures of the knee: bare nerve endings, ruffini endings, pacinian corpuscles and Golgi tendon organs (Lattanzio and Petrella, 1998). The muscle receptors responsible for the perception of the limb position are the muscle spindles and Golgi tendon organs. For more information on their function please see Table 1.1.

The receptors represented in the knee joint and muscles surrounding the knee are involved in neural reflex, which is defined as *‘involuntary reaction in response to a stimulus applied to periphery and transmitted to the nervous centers in the brain or spinal cord’* (Lattanzio and Petrella, 1998), and this provides stability and stiffness to the knee joint (Beard et al., 1993).

Table 1.1. The principal receptors in the knee joint and muscle surrounding the knee joint.

Proprioceptors in the articular structures and muscle around the knee joint.	Type of receptors	Function
Ruffini	I	Low threshold mechanoreceptors responsible for detecting static and dynamic factors such as joint angle, velocity, and intra-articular pressure.
Pacinian corpuscles	II	Low threshold, rapidly adapting mechanoreceptors responsible for signalling dynamic changes in the deformity of tissues and the initiation, acceleration, and termination of joint motion.
Golgi tendon organs (GTO)	III	High threshold slow adapting mechanoreceptors that are stimulated only at extreme angles of joint displacement.
Bare nerve endings	IV	High threshold and pain receptors.
Muscle spindles	--	Responsible for the perception of limb position
Golgi tendon organs	--	Responsible for the perception of limb position

For example, Solomonow et al. (1987) demonstrated that whilst the anterior cruciate ligament is under stress of a large force, such as when the tibia is displaced anteriorly beyond the normal limits of the ligament, the anterior cruciate ligament receptors are activated and the hamstring contracts. Hamstring contraction will help reduce the anterior displacement of the tibia. Such a mechanism of protection has been reported to occur in similar studies (Baratta et al., 1988; Pitman et al., 1992).

Neuromuscular control (proprioception) within and around the knee joint may be distorted on application of a traumatic force to the joint. This distortion to the neuromuscular control (proprioception) may prevent from not responding quickly



enough or strongly enough to counter a traumatic episode (Jerosh et al., 1995; Gleeson et al., 2000). It has been suggested that muscular fatigue and muscle damage produces neuromuscular system deficiencies within the knee joint and the surrounding muscle which in turn may create joint and muscle injury and ultimately a decrease in athletic performance (Green, 1987; Gleeson et al., 1998; Voight et al., 1996).

### 1.3 *The role of neuromuscular system in knee joint stability.*

Sensory information (neuromuscular control) travels through afferent pathways to central nervous system (CNS), eliciting an efferent motor response (regulation process) vital to coordinate movement pattern and function stability.

Proprioception (neuromuscular control) is defined as the conscious perception of limbs in space (Lattanzio and Petrella, 1998). Proprioceptor information originates within the tendon, joint capsule, ligament, skin and muscle surrounding the knee.

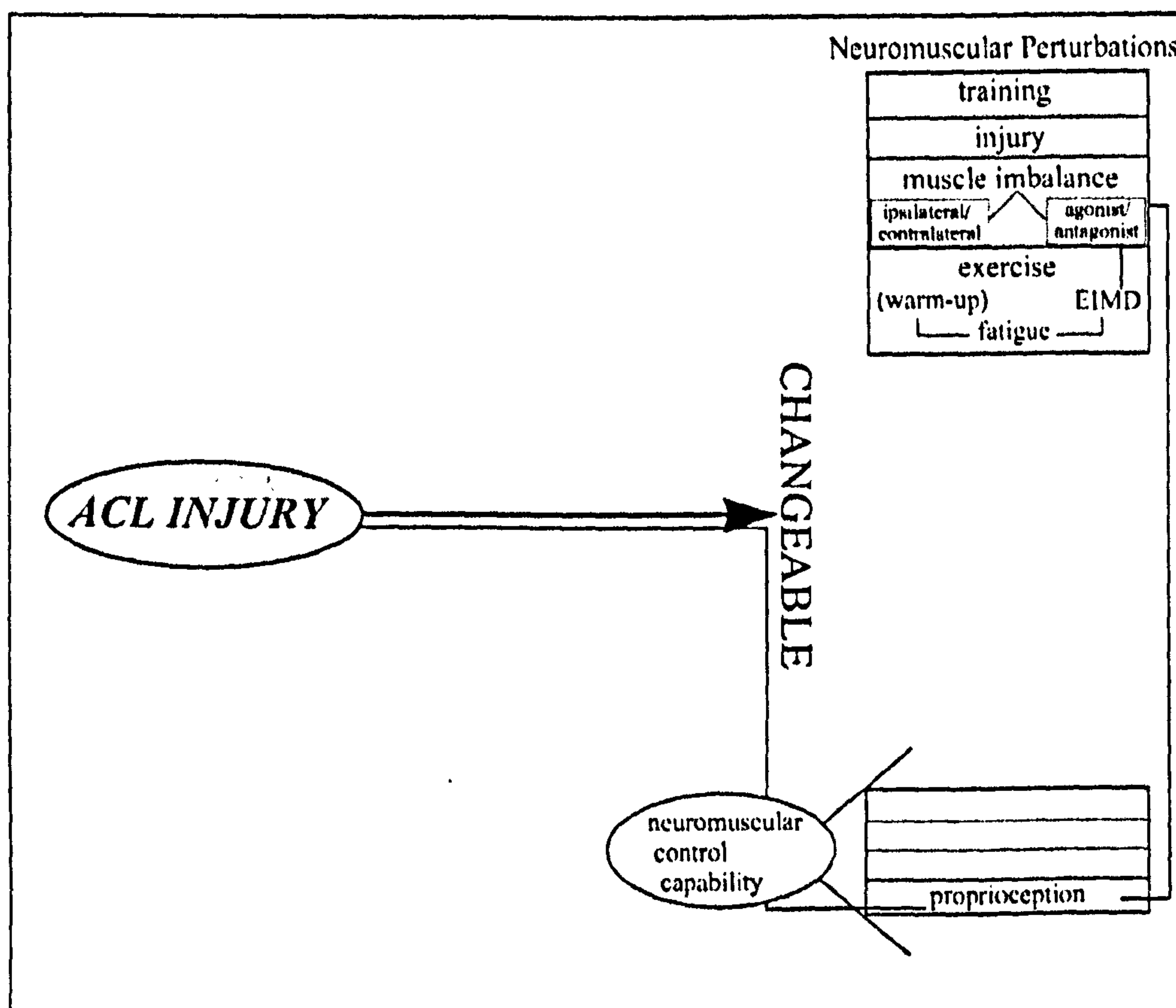
The receptors represented in the knee joint and muscles surrounding the knee are involved in a neural reflex, which is defined as involuntary reaction in response to a stimulus applied to the periphery and transmitted to the nervous centers in the brain or spinal cord, and provide stability and stiffness to the knee joint (Beard et al., 1993).

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mechanism of protection has been reported to occur in similar studies (Baratta et al., 1988; Pitman et al., 1992).

Figure 1.2. Proprioception and ACL injury.



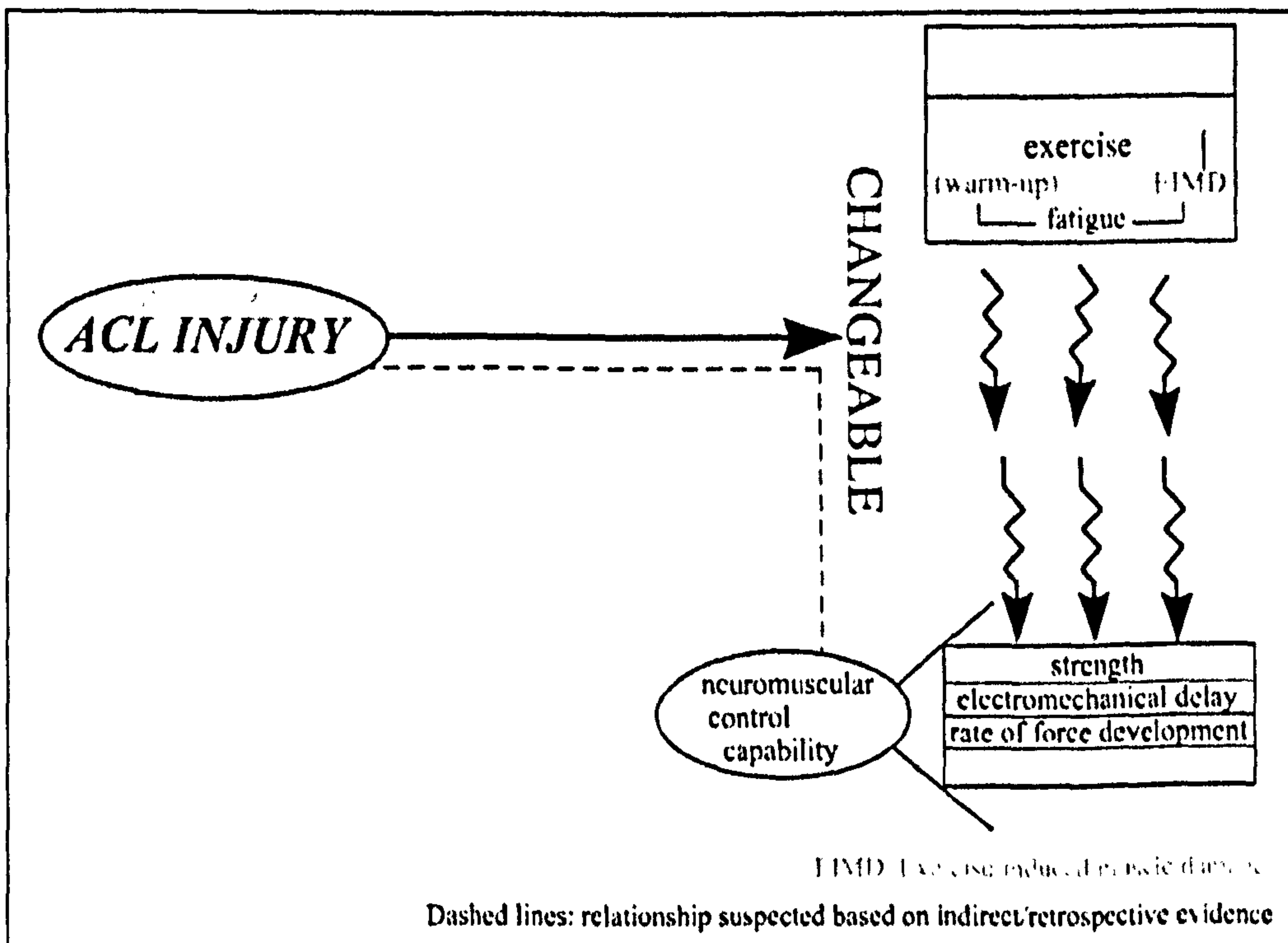
The stabilizing structures (static and dynamic muscle) within and around the knee joint are deformed on application of a traumatic force to the joint, eliciting a feedback and reflexive muscle contraction (Pope et al., 1979). This deformity to the stabilizer structures happens when the mechanoreceptors excitation is not responding quickly enough or strongly enough to counter a traumatic episode (Jerosh et al., 1995; Gleeson et al., 2000). It has been suggested that muscular fatigue produces neuromuscular deficiencies within the muscle, this in return creates joint and muscle injury (deformity) and an ultimate decrease in athletic performance (Green, 1987; Voight et al., 1999; Gleeson et al., 1998).

#### 1.4 Neuromuscular system and fatigue.

The ability of mechanoreceptors to offer proprioceptive information and to initiate protective measurement to the muscles under muscular fatigue has led to the investigation of joint and muscle receptors in the mechanism of knee stability (Lattanzio and Petrella, 1998).

Muscular fatigue has been defined as inability to sustain force output, resulting in a decline in athletic performance (Green, 1987; Ferrell et al., 1987).

Figure 1.3. Possible interrelationships between muscle fatigue, neuromuscular control capability and ACL injury.



It has been shown that muscular fatigue produces neuromuscular deficiencies within the muscle, therefore exposing a joint to injury and an ultimate decrease in athletic performance (Green, 1987; Skinner et al., 1986). Due to the relationship between fatigue and injury, recent researcher undertook a prospective epidemiology study examining injuries sustained in English professional footballs over two seasons (Hawkins et al., 2001). The study showed an increase in injury incidence towards

the latter stages of the first half, and moreover the numbers of injuries recorded in second half is greater than the first half. Other studies reported the same findings (Laurie et al., 2001; Pinto et al., 1999). For example, Pinto et al. (1999) reported that in a prospective study of ice hockey incident injuries at the junior A Level 46.2% of injuries were sustained in the third period, and 46.9% of injuries were sustained in the last 5 minutes of a match-play time.

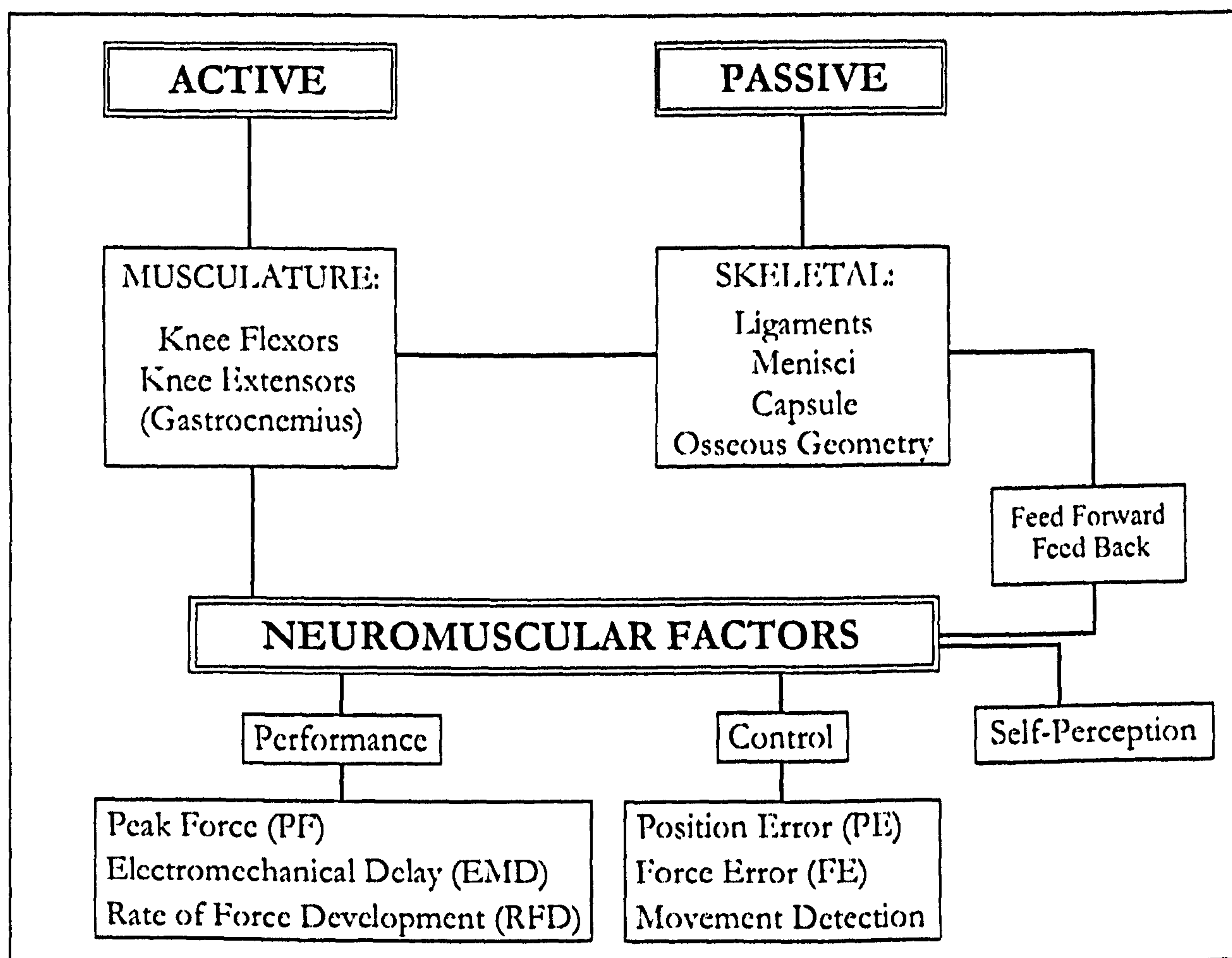
Only a few published experiments have investigated the effect of neuromuscular fatigue on the knee joint. For example, Skinner et al. (1987) found a decline in the ability to reproduce the angle of the knee joint after a series of interval running sprints a of total 3.75 miles in young active men. In contrast, a study by Marks and Quinney (1993) found 20 maximal isokinetic quadriceps of fatigue contraction protocol among young sedentary women and there was no significant reduction in knee proprioception. Both studies have not only presented conflicting information regarding changes in proprioception of the knee joint that occur during fatigue, but differed in fatigue protocol and subjects. Therefore, further studies are needed to measure fatigue intervention and its efficiency on neuromuscular system capacity and subsequently on athlete's ability and performance.

Many epidemiological studies have shown that during sports activities the most common period of time for injury to occur is during the later stage of the game (Pinto et al., 1999; Gabbett, 2000). For example, 46.9% of injuries took place in the last 5 minutes (Pinto et al., 1999). Injuries occurring in the final 5 minutes are due to interference of fatigue that impairs neuromuscular control (proprioception) during sports performance (Skinner et al., 1986; Voight et al., 1996).



The ability of neuromuscular control performance to offer proprioceptive information and to initiate protective measures to the muscles under muscular fatigue has led to the investigation of joint and muscle receptors in the mechanism of knee stability (Lattanzio and Petrella, 1998).

Figure 1.4. Simplified conceptual model for knee joint stability.



Muscular fatigue has been defined as the inability to sustain force output, resulting in a decline in athletic performance (Green, 1987; Ferrell et al., 1987).

It has been shown that muscular fatigue produces neuromuscular deficiencies within the muscle, therefore exposing a joint to injury and an ultimate decrease in athletic performance (Green, 1987; Skinner et al., 1986). Due to the relationship between

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Only three available experiments have investigated the effect of neuromuscular fatigue on the knee joint. The first study was by Skinner et al. (1986), who investigated knee proprioception within healthy young men. The protocol was designed to introduce muscle fatigue to healthy men as follows: subjects alternatively sprinting 1 and ¼ mile with a 90 second rest between each interval for a total distance of 3.75 miles. The examiner used passive reproduction to measure knee joint proprioception. The examiner reports a significant reproduced knee angle following fatigue.

The second study was by Marks and Quinney (1993), who studied maximal isokinetic quadriceps (concentric and eccentric) activation in young sedentary women. The examiners used two separate groups (control and experimental groups). The examiners tested the knee position sense before and after a 5-minutes rest interval for the control group. The experimental group was tested before and following 20 consecutive concentric and eccentric activations of the quadriceps



(fatigue intervention). Neuromuscular control (proprioception) was assessed by reproduction of knee angles by means of active muscle activation in the open kinetic position. The examiners found no significant difference between the control and the experimental groups at the baseline and immediately following exercise. Finally Lattanzio et al. (1997) recently examined the effect of fatigue intervention on knee proprioception. The examiners used three different fatigue protocols and all tests consisted of the lower limb cycling on the computer driven cycle ergometer. An initial standard ramp protocol at 20 / 25 watts/minute to exhaustion was used to decide the subject ( $VO_2$  max) which was used to calculate the work rates for the later two protocols. The continuous test at 80%  $VO_2$  max and interval protocol at 120%: 40%  $VO_2$  max were then carried out to exhaustion. Each of these protocols was carried out on a different day and separated by a week. The result of this study revealed that knee proprioception was statistically reduced for men in all three tests and women in the continuous and interval protocols. Neuromuscular control (proprioception) of the knee joint was examined by active reproduction of the knee angles in the standing position.

Clearly, these three studies have not only presented conflicting information regarding changes in proprioception of the knee joint that occur during fatigue, but differed in fatigue protocol and subjects. Therefore, further studies are needed to measure fatigue intervention and its efficiency on neuromuscular system capacity and subsequently on athlete's ability and performance.

## *1.5 Indices of neuromuscular performance assessment with regard to fatigue intervention*

### *Peak force index*

Decrease of muscle strength associated with acute functional activities may produce neuromuscular deficiencies within the muscle (Skinner et al., 1986; Green, 1987; Gleeson et al., 1998), hence imposing a threat to knee joint stability and anterior cruciate ligament (ACL) integrity during sports activities (Gleeson et al., 1998). However, Bigland-Ritchie and Woods (1984) reported that one of the most significant signs of fatigue is a decline in maximum force (strength). For example, Avela et al. (2001) demonstrated a decrease in peak force by 18.5%. Similar peak force reductions were reported by Woods et al., 1987; Fellows et al., 1993; Nicol et al., 1991; Gleeson et al. 1998).

Recent research has investigated the time course of loss of force and neuromuscular control performance capacities during simulated soccer 'match-play' and suggests that pattern of injury occurrence correlates strongly with game-related fatigue processes (Gleeson et al., 1998). For example, in a prospective epidemiology study examining injuries sustained in English professional football over two seasons (Hawkins et al., 2001). This study demonstrated an increase in injury incidence towards the latter stages of the first half, moreover the numbers of injuries recorded in the second half was greater than the first half. Other studies reported similar findings (Laurie et al., 2001; Gabbett, 2000; Pinto et al., 1999).

In addition to weakening peak force, muscle fatigue is also believed to negatively affect other indices of neuromuscular performance capacity, such as the rate of force development and electrochemical delay (EMD) (Winter and Brookes, 1991).



*Electromechanical delay (EMD) index performance and its role in sporting injury.*

Electromechanical delay (EMD) is defined as the onset of electrical activity and tension development in human muscle (Zhou et al., 1995).

Cavanagh and Komi (1979) reported that EMD is affected by one of the following factors: first time course propagation of action potential of muscle membrane, second excitation-contraction coupling process, and finally stretching of the series elastic components (SEC) by the contractile component. Thus any reasons that affect the above factors may influence the EMD. However, the literature reports that EMD values range from 10 to 120ms ( Vos et al., 1991). It has been demonstrated that EMD is significantly correlated to the static maximal voluntary contraction (MVC), rate of force development (RFD) (Bell and Jacobs, 1986). In addition, EMD has been reported to be influenced by type of muscle contraction (Norman and Komi, 1979), joint angle (Grabiner, 1986), and more significantly fatigue intervention on knee injury (Nilsson et al., 1977; Zhu et al 1996; Gleeson et al 1998).

Fatigue has been reported to be linked with injury during sports activities (Gleeson et al., 1998). Thus, it is defined as the temporary inability to sustain power output or force during repeated muscle contraction (Gibson and Edward, 1985). Furthermore, it has been demonstrated that fatigue reduces the peak force and prolongs the EMD.

Winter and Brookes (1991) proposed that the time taken for the contractile component to stretch the series elastic component of the muscle primarily determines the length of EMD. However, studies have been controversial in reporting linkages of the lengthening of EMD and fatigue intervention, and subsequently on knee

injury. For example, Stull and Kearney (1979) investigated the effects of fatiguing handgrip exercise on the components of fractionated reaction time of the finger flexors. They found there was a significant increase in the motor time (defined similarly to EMD), from 28.8- 37 ms, while the handgrip strength decreased by 60%. The changes of motor reaction time demonstrated a linear relationship with the level of fatigue. In contrast, Hanson and Lofthus. (1978) found no fatigue effects on the motor time of finger flexors when the handgrip force decreased by 48%. Therefore, further study is needed on the relationship between the fatigue reduce reduces the peak force and prolongs the EMD and subsequently increase the risk of injury to knee joint stability. However, introducing more intense exercise such as eccentric exercise may place knee joint stability under severe consequence of long term of disability.

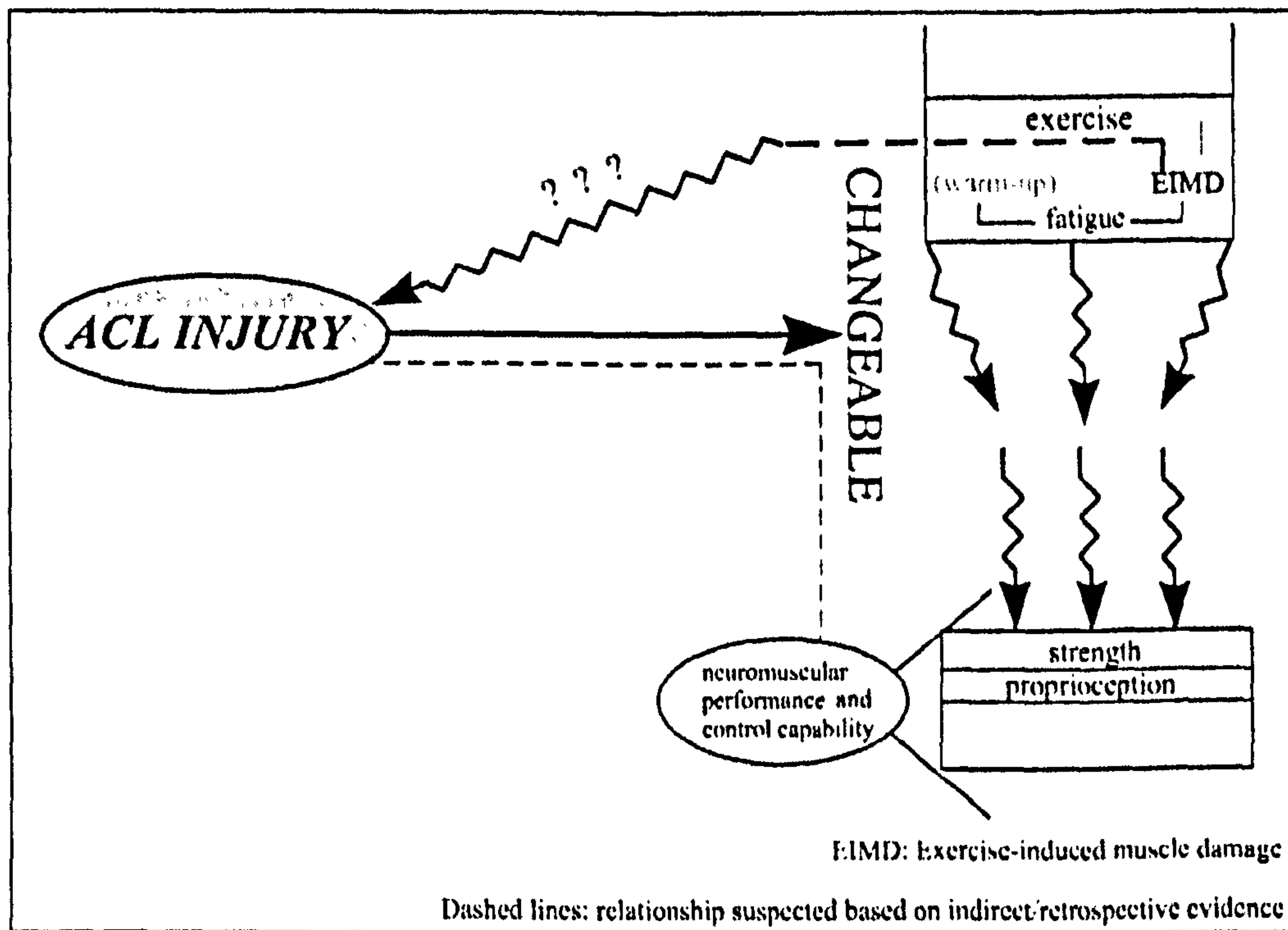
### *1.6 Neuromuscular system, injury and muscle activation*

Among the numerous factors that have been considered as potentially important to the protection and stabilisation of the joint, such as strength, flexibility and ligament compliance or stretchiness, bone geometry, improved neuromuscular system capacity is one of only two such factors that has been consistently linked to a reduction in the incidence of ligamentous injuries in the knee joint. For example, Caraffa et al. (1996), in a prospective random-allocation control study showed that the incidence of ACL could be reduced by 7-fold with a season-long proprioception training programme.



It has been shown that muscular fatigue (Green, 1987; Skinner et al., 1986) and muscle damage (Behm et al., 2001; Saxton et al., 1995) produce neuromuscular control deficiencies within the muscle,

Figure 1.5. Possible interrelationships between exercise-induced muscle damage (EIMD), neuromuscular performance and control, and ACL injury.



therefore influencing knee joint injury, decreasing the athletic performance (Skinner et al., 1986; Green, 1987). This is due to the fact that the neuromuscular system has a limited reaction time to respond effectively, efficiently and very fast, to the dynamic force allied to the knee joint (Gleeson et al 1998; Osternig et al., 1999). Therefore, any dynamic stability to the knee joint structure depends on this time delay (Gleeson et al., 1998; Wojtys, and Huston 1996; Zhou et al., 1996; Stull and Kearney, 1978).

The neuromuscular reaction time is known as electromechanical delay (EMD), which is defined as the time lag between the onset of electrical activity and tension

development (Zhou et al., 1996; Vos et al., 1991). For example, Zhou et al. (1996) proposed that lengthening of EMD is due to extensive changes in the structural and functional properties of the muscle induced by either acute exercise or physical training. Furthermore, Gleeson et al. (1997) and Zhou et al. (1996) demonstrated that the substantive prolongation of this neuromuscular delay after fatigue might permit the unrestrained development of force of sufficient magnitude to damage knee ligamentous tissue.

Stull and Kearney (1979) demonstrated that EMD increased from 28.8 to 37 ms while handgrip strength decreased by 60% after acute fatigue. Zhou et al. (1996) demonstrated a lengthening in EMD from 40.4ms to 63.4ms compared to before fatigue; this was accompanied by a reduction of peak force by 51%-56% after fatigue. Conversely, Viiasalo and Komi (1981) have shown that EMD in the reflex activation were about 7ms longer than that in contraction knee extensions. The outcomes of these studies clearly demonstrate that uncertainty of the effect of acute fatigue on EMD and subsequently on impairment of neuromuscular capacity. Thus more studies are needed on the effect of serial fatiguing on prolonging of EMD.

### *1.7 Muscle damage following eccentric exercise and neuromuscular system.*

During match-play and training activities in the playing-season, at which time athletes may be put through unaccustomed exercise that involves a significant eccentric component which produces damage to the muscle tissue (Newham et al., 1983; Clarkson and Trembly, 1988; Morgan and Allen, 1999). An eccentric contraction is one in which a muscle lengthens as it apply a force and so perform negative work (Balnave and Thompson, 1993). An immediate and long-lasting



decline in muscle strength is observed after eccentric exercise (Byrne and Eston, 2002a; Nosaka et al., 2001). Strength loss is most prominent immediately following eccentric exercise and reduced by 50% (Clarkson et al., 1992) and by 69% (Rinard et al., 2000). Recovery can take up to 7 days (Clarkson et al., 1992; Byrne et al., 2001), in addition to great loss of muscle strength; there is also soreness and pain associated with eccentric exercise (Newham et al, 1983; Warren et al., 1999). In contrast, to the immediate loss of muscle strength, pain and muscle soreness peaks at 24-48 hours (Nosaka and Sakamoto, 2001). Rinard et al. (2000) proposed that recovery from pain and muscle soreness can occur more quickly than restoration of the neuromuscular performance capabilities, which encourages athletes to return to sports activities without full recovery from muscle damage.

Evidence for the effect of exercise- induced muscle injury on neuromuscular control of the lower limb is very limited. However, Saxton et al. (1995), reported significant impairment to neuromuscular control after following eccentric exercise. Obviously, further research is needed given the potential important of the neuromuscular control to the prevention of knee injury after muscle damage, particularly, the shorter EMD founded in the muscle which have a higher percentage of fast twitch fibres which are capable of eliciting protective response to knee joint, but these fibres are most susceptible to damage (Linnamo et al., 2000).

It is important to understand how normal muscle activation works to understand which mechanism becomes weak after sports or daily life activities. The neuromuscular control (proprioception) information reaches the central nervous system (CNS) from the knee joint and surroundings muscles by afferent pathway

through the spinal cord where it is integrated with other information from other levels of the nervous system, eliciting an efferent regulating response to coordinate the pattern movement and functional ability of the knee joint.

Within the muscle fibers, the action potential spreads through the muscle cells by way of the T-tubules. This activates to release  $\text{Ca}^{2+}$  from the sarcoplasmic reticulum (SR).  $\text{Ca}^{2+}$  is then attached (taken up) by the troponin molecules and initiates the cross-bridge cycle that leads to tension development and/or shortening (Morgan and Allen, 1999). The number of tension-producing cross-bridges in each sarcomere depends on the sarcomere length. If the external tension on the muscle exceeds the tension that would be developed at constant muscle length, the muscle stretches while activated, this is known as eccentric exercise (Raven, 1991). Unaccustomed exercise or very intense exercise causes ultrastructural damage or changes to the muscle tissues and is known as eccentric exercise (Clarkson et al., 1992; Morgan and Allen, 1999; Behm et al., 2001).

Eccentric muscle contraction takes place when the muscle is lengthened during contraction, for example when someone tries to extend the elbow while the elbow is in a position of flexion process. In contrast, a concentric exercise muscle takes place when the muscle shortens during contraction, for example when elbow muscle flexors are lifting a weight. The isometric muscle action occurs when tension develops, but there are no changes in the muscle length (Fox et al., 1989).

Isometric, concentric and eccentric muscle action has been compared, the result of the studies constantly show that eccentric action results in evidence of muscle



damage (Golden and Dudley, 1992; Jonhagen et al., 1994). In addition, recently there has been much attention to the role eccentric muscular work plays in the increase of sports injuries (Jonhagen et al., 1994). Furthermore, eccentric muscle actions occur regularly everyday and in sporting activities such as gymnastics, going down hills and running (Komi, 1984).

There are many symptoms associated with exercise induced muscle damage that lead to muscle fibre structural changes, such as strength loss, muscle soreness, swelling, stiffness, pain, tenderness and increased protein release (Armstrong, 1984; Golden and Dudley, 1992; Balnave and Thompson, 1993; Warren et al., 1999; Byrne et al., 2001).

Strength loss, muscle soreness and pain on active movement are the most common indicators for exercise- induced muscle damage (Newham et al., 1983; Warren et al., 1999; Byrne et al., 2001; Byrne and Eston, 2002a).

Many techniques have been used to quantify the proprioceptive assessment explained in the introduction of this thesis and yet the relationship between neuromuscular control (proprioception) and exercise-induced muscle damage (EIMD) is still not clear. A few researchers have examined the effects of EIMD on neuromuscular control (proprioception) and mainly focused on the upper body (Jones et al., 1986; Clarkson et al., 1992; Saxton et al., 1995; Behm et al., 2001). For example, Saxton et al. (1995) evaluated the effect of exercise-induced muscle damage on the flexors of the non-dominant left arm. The protocol consists of each subject performing 50 maximal eccentric exercise actions. This was divided into

two sets of 25 eccentric muscle actions with 5 minutes rest. In this protocol, the subjects forearm extended forcefully by the examiner before being returned to the starting point. Eccentric exercise action takes 3 seconds to finish and 12 seconds rest was given between repetitions. The maximum force was tested at 90° angles. Two isometric muscle activations of 3 s durations were performed and the maximum of two was used as the standard score. The target force in this study was 35% of maximum force.

Each subject had force applied by the counter arm control (i.e. the dominant arm) until it reached the point of 35% of pre-maximum force of the experimental arm, while simultaneously matching the produced forces with experimental arm. This was stopped when subjects felt they produce equal amount of generation of force by both arms. Three trails were recorded on each day and the means of the three trails were used as the criteria score. These scores were presented as a constant error score.

Neuromuscular control (proprioception) was significantly impaired by the eccentric exercise. The result demonstrated that constant error reduced immediately and remained at a low level over the 5 days after exercise ( $P < 0.01$ ). The subjects had overestimated their force production by producing the force below the target force. However, when subjects used their own score as the target force, they re-produced 35% of the force produced in the daily strength of the experimental arm. This is inconsistent with a study by Cafarelli et al. (1982) using the same protocol.

However, a number of researchers pointed out that repeated bouts of muscle damage might not introduce effective muscle damage as the first bout of exercise induced



muscle damage. For example, Clarkson et al. (1992) demonstrated that performance of one bout of high-force eccentric exercise produces an adaptation such that less damage was produced when the same exercise was performed up to several months later. Furthermore, Clarkson and Tremblay. (1988), demonstrated in another study that when participants performed 24 maximal eccentric contractions, this exercise produced an adaptation such that less damage than expected was noted 2 weeks later when subjects performed a second exercise bout consisting of 70 eccentric exercises. Similar result were shown by Ebbeling and Clarkson, 1990; Nosaka et al., 1991.

### *1.8 Indicators of exercise- induced muscle damage.*

Direct or indirect indicators commonly quantify exercise- induced muscle damage. Direct indicators are not discuss in this study. Indirect quantification is achieved by means of different symptoms associated with muscle damage in the following days of muscle damage such as reduction in peak force, delayed-onset muscle soreness, stiffness, swelling and increase of pain (Newham et al., 1983; Clarkson et al., 1992; MacIntyre et al., 1996; Warren et al., 1999; Behm et al., 2001; Byrne and Eston, 2002).

### *1.9 Index of neuromuscular performance assessment*

#### *Peak force index*

Maximal voluntary isometric activations are commonly used as an indicator of muscle damage ( Warren et al., 1999; Behm et al., 2001). There has been a report of immediate and prolonged loss in muscle strength (Clarkson et al., 1992; Byrne et al., 2001; Byrne and Eston, 2002a). Muscles that are involved in the exercise feel weaker, and during the next few days become sore and tender (Morgan and Allen,

1999). Full recovery takes from hours (Newham et al., 1983b), days (Byrne and Esten, 2002) or even more than a week (Clarkson and Nosaka, 1992).

For example, Clarkson et al. (1992) reported a loss of muscle strength of over 50% and it took over 10 days for full recovery. Similar studies reported a loss of muscle strength (Balnave and Thompson, 1993; Gleeson et al., 1998; Newham et al., 2001; Byrne and Eston, 2002a).

### *Theory explaining the reduction of peak force (strength)*

A number of reasons have been suggested to explain the decline of strength associated with muscle damage: many studies demonstrated that eccentric exercise causes damage to the muscle fibres, particularly disruption to the myofibrillar banding pattern (Friden et al., 1983; Newham et al., 1983). However, the ultrastructural damage becomes worse in the days subsequent to exercise, while the strength is recovering (Clarkson et al., 1996).

The first reason is due to the sarcomeres being stretched out by performance of the lengthening actions. If the lengthening actions pull some of the central sarcomeres apart, the overlap between the actin and myosin filament would be reduced, thereby reducing the maximal number of cross bridges that could form. Perhaps subtle changes in sarcomeres length could influence the ability to generate force.

Newham et al. (1988) suggested that larger strength losses were produced when subjects performed an exercise with the muscle at a long length compared to that at a short length. The overstretched sarcomeres could explain the immediate strength



loss after exercise (Clarkson et al., 1992; Byrne et al., 2001; Byrne and Esten, 2002a,b). Following the exercise, the sarcomeres length would gradually recover, reflecting the slow recovery of strength (Byrne et al., 2001; Byrne and Esten, 2002). Second, reduced flexibility of the muscles involved in exercise induced muscle damage is one of the factors contributing to reduce of peak force following muscle damage intervention. Third is that swelling and pain (Jones et al., 1987; Smith, 1991)

### *1.10 Indices of physiology changes following exercise-induced muscle damage.*

#### *Muscle soreness and pain*

Delayed onset muscle soreness (DOMS) is a common sensation of discomfort associated with high intensity of exercise often felt in the skeletal muscle (Balnave and Thompson, 1993; MacIntyre et al., 1996). DOMS may be felt within 8-10h following exercise and peak between 24-72h (Newham et al., 1983; MacIntyre et al., 1996). However, the most common sign of muscle damage is muscle soreness (Clarkson et al., 1992), reduced flexibility (stiffness) (Armiger, 2000), pain on active movement and palpations (Newham et al., 1983; MacIntyre et al., 1996) and inflammation (Smith, 1991). Although, exercise-induced muscle soreness is well documented and well known and frequently experienced phenomenon, the precise mechanisms responsible for its production are not fully understood.

A number of studies pointed out some reasons of (DOMS). For example, Stauber et al. (1990) reported that this phenomenon is due to a complex set of reactions involving disruption of connective tissues and muscle fibers. At the same time Smith (1991) believed it to be due to tissue inflammation.

Muscle pain takes place 24-48 h following the muscle damage (Newham et al., 1983). Clarkson et al. (1992) reported that eccentric exercise is associated with muscle soreness and pain. Smith (1991) reported that pain is experienced during active movement or on muscle palpations.

There are a number of explanations of pain after eccentric exercise; for example Jones and Round (1990) suggested it is due to an inflammation of connective tissue. The inflammation may sensitize mechanoreceptors so they are easily activated with muscle movement and palpation. Another reason is that swelling and edema can cause soreness and pain, due to an increase in muscle pressure (Clarkson et al., 1992).

#### 1.11. *Muscle damage and fatigue exercise*

Fatigue may be seen as a challenge as well as a frustrating issue to sports performance. Fatigue is defined as the inability to maintain force output, resulting in a decrease in performance (Green, 1987; Sterner et al., 1998). Muscle fatigue is thought to affect neuromuscular performance and control (McNair et al., 1992; Skinner et al., 1986; Wojtys et al., 1996; Myers et al., 1999; Lattanzio et al., 1997; Myers et al., 2000). This is due to the fact that fatigue introduces a decrease in proprioceptive input, affecting neuromuscular regulator control (Myers et al., 2000), that response is vital to knee joint stability (Brown et al., 1996; Behm et al., 1997; Yeung et al., 1999). Furthermore, it is believed that muscle fatigue desensitizes muscle spindle threshold, thus affecting neuromuscular control and then performance (Myers et al., 2000), hence it is very significant to joint stability and ACL integrity in sport movements.



For example, Pedersen et al. (1999) demonstrated that increased intramuscular concentration of lactic acid, potassium chloride and serotonin following fatigue intervention may affect the muscle spindle system.

This is supported by Djupsjobacka et al.(1995). Also, exercise induced muscle damage produces great loss of strength and prolonged impairment of force capacity of the involved muscle group (Clarkson et al., 1992). Furthermore, Saxton et al. (1995) reported an increase in tremor and loss of proprioceptive task, which peaked 2-3 days after exercise, induced muscle damage using the forearm flexor of the non-dominant arm. Currently, however, there are few studies, which have investigated neuromuscular performance and control after muscle damage associated with acute fatiguing exercise of the flexor muscles of the lower limb.

Clearly, investigation is needed to understand the changes of neuromuscular performance and control capabilities under high stress exercise conditions and may help to establish appropriate policy to help reduce the risk of injury.

### *1.12 Dynamic joint stability and the neuromuscular system*

Dynamic joint stability has been defined as the ability of appropriately stimulated muscles to offer stability to a joint (Laskowski et al., 2000). Neuromuscular control (proprioception) is a key element which sustains dynamic joint stability (Laskowski et al., 2000). This is due to information from the neuromuscular control (proprioception) in the knee joint and surrounding muscles given to the central nervous system (CNS) through afferent pathways to produce and modulate the efferent motor response to maintain a balance of stability and mobility of the knee



joint. *'In essence, dynamic joint stability is the product of the proprioception system'* (Laskowski et al., 2000).

Wojtyes et al. (1996) reported that it appears that the dynamic restraint system of the knee joint can prevent or reduce injury throughout rigorous physical activity. Wang and Walker. (1974) showed that the capability of the quadriceps and hamstring muscles to reduce external and internal rotation of the tibia by 80% with a corresponding 1% body weight of compressive force. This protection is due to the role of muscle spindle and Golgi tendon capability to stabilise the knee joint during dynamic movement.

Muscle spindles are enclosed in a connective tissue sheath and consist of 2 to 12 specialized muscle fibers that are called intrafusal muscle fibers, connected in parallel to contractile muscle fibers that are called extrafusal muscle fibers (Patton et al., 1989). The primary sensory endings of the muscle spindles are innervated by Ia and II (i.e. afferent), which are fast-conducting to allow the primary sensory to react to dynamic movement.

The other type of receptive surrounding the knee is the Golgi tendon organ, similar to spindles in structure and surrounded by a sensory ending (i.e. Ib afferent) and found close to the musculotendinous junction. It's a very sensitive detector of muscle tension in a localised fraction of a muscle. Thus, it can react to very small force that may change in the muscle tissues (Binder, 1981).

Important information from the afferent groups Ia, Ib and II travels to the CNS and is used to coordinate movement, and elicit the reflex which is very important to joint stiffness (Solomonow and D'Ambrosia, 1994).

Muscle spindles play a strong role in movement regulation throughout stretch reflex (Vallbo, 1979), providing information about limb position and velocity (Sittig, 1985), and loading of the knee joint (Laskowski et al., 2000). In addition the Golgi tendon organ provides information about tension in the muscle inhibition of the muscle by preventing the muscle from contracting so forcefully so as to rupture a tendon through the presence of the II sensory (Gordon and Ghez, 1991). However, very limited studies have been conducted on dynamic performance following muscle damage. Indeed, only one study demonstrated the effect of muscle damage on sprinting performance. This study by Semark et al. (1999) demonstrated that there was no difference either in sprint time or acceleration over 30 meters after 70 drop jumps. Semark et al. (1999) reasoned that by stating the exercise used to induce muscle damage was not powerful enough to induce muscle damage and functional changes in the trained participants that took part in the study. This is certainly an area that requires further investigation, since muscle flexors have a very important role in preventing knee stability and subsequently ACL integrity (Gleeson et al., 1998). Furthermore, assessing joint neuromuscular control (proprioception) in dynamic and thus functional situation may offer more accurate results (Carter et al., 1997). Also dynamic control following fatigue received even less attention during the conditions of muscle damage.



### 1.13 *Assessment methods for neuromuscular control.*

The vast majority of studies measuring neuromuscular control in relation to the knee joint commonly have two different techniques. These are the threshold to detect passive motion (Barrack et al., 1983; Barrack et al., 1984; Skinner et al., 1989) and the reproduction of knee angles, mainly passively (Barrack et al., 1983; Barrack et al., 1984; Barrack et al., 1989; Lattanzio et al., 1997; Fremerey et al., 2000; Barrett, 1991).

The majority of studies have used these techniques to assess neuromuscular control at a constant velocity of 0.5 degree/ second (slow assessment method). For example, following repair and reconstruction of ACL (Fremerey et al., 2000, MacDonald et al., 1996; Barrett, 1991), ACL-deficiency (Fremerey et al., 2000; Beard et al., 1993), osteoarthritic (Barrett et al., 1991), and neuromuscular fatigue (Skinner et al., 1986; Lattazio et al., 1997). However, there are no standard measurement techniques and different data analysis making comparison between studies difficult (Barrack et al., 1989; Fremerey et al., 2000; Beynnon et al., 2000)

While both tests might be considered to assess, in a valid way, the ability to perceive change in or accuracy of joint position using sensory apparatus, it could argued that such 'slow' assessment methods might not best represent or be entirely valid for the need to assess timely, rapid, forceful, precise and efficient responses from the neuromuscular system to threats posed by sporting endeavours. In such a scenario both the capacity of the sensory afferent (neuromuscular control) to offer accurate assessment of need and the motor efferent to respond with timely and forceful



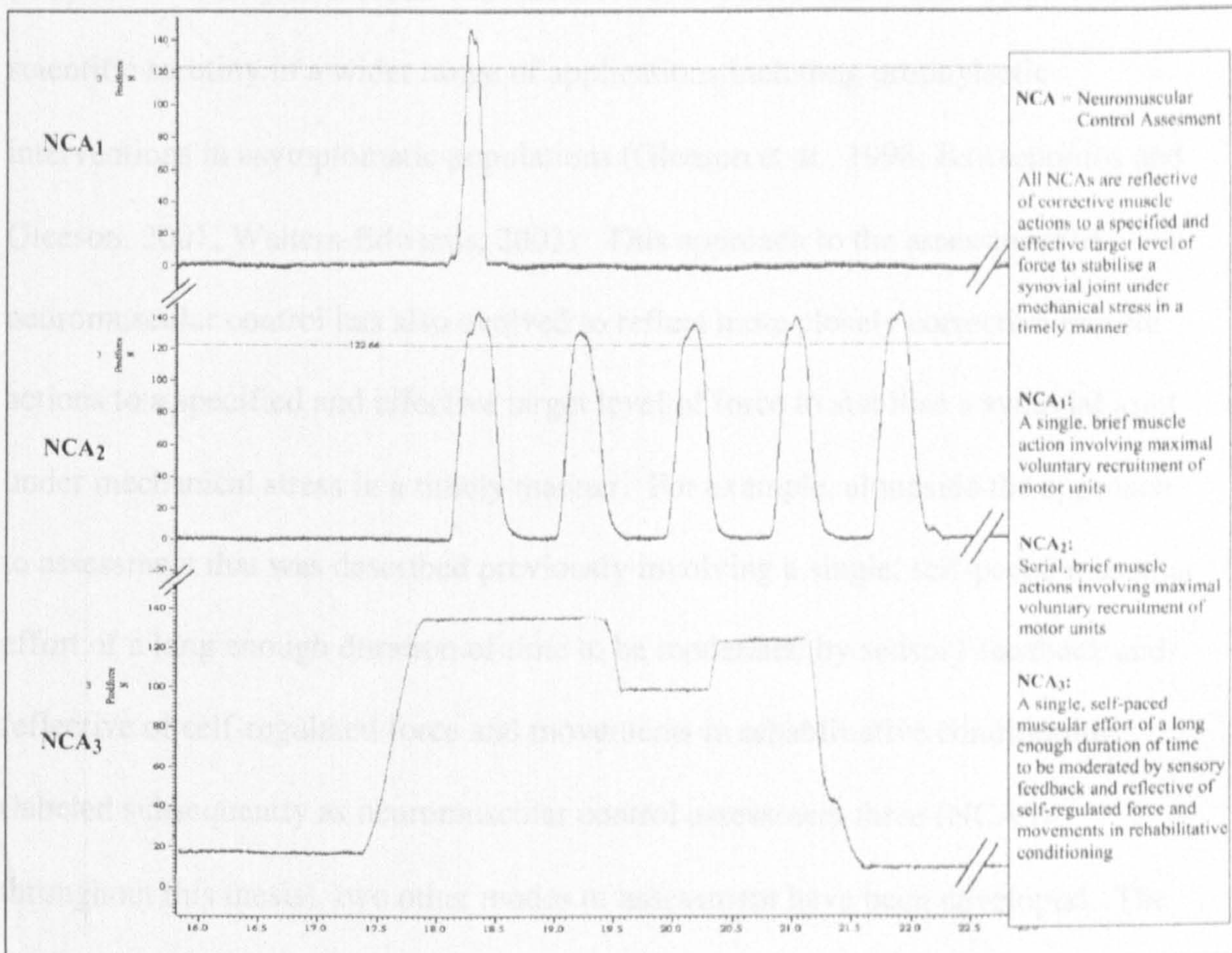
activation patterns to achieve ultimately an efficient outcome (e.g. knee joint protection), should be challenged.

Recent studies (for example, Doyle, 1998; Gleeson, 1999; 2000) have piloted the use of a neuromuscular assessment protocol in which subjects are required to match as precisely as possible, a previously learned 'blind' target force (full details in chapter 2 and relevant following chapters) during static knee extension (refer to chapter 4 only) or flexion (refer to chapters 4, 5 and 6) efforts. The force-time history of the subject's response has been set volitionally but has generally lasted between 2 and 3 seconds. This allows the subject to regulate 'on-line' motor-unit recruitment and ultimately force, on the basis of sensory (proprioception) information. The subject's best match of the prescribed target force has been indicated by a voluntary relaxation of the involved musculature that can be easily distinguished by the test administrator subsequently by interrogation of the force-time record. This type of approach perhaps best mimics a situation during conditioning or rehabilitation of an injured limb in which the subject is required to self-regulate their training intensity involving muscle activation in order to optimise the process.

This approach to the assessment of neuromuscular control and control regulator capacity has shown significant differences between ACL-deficient patients and



Figure 1.6. Example responses associated with three modes of the assessment of neuromuscular control (NCA1, NCA2 and NCA3).



asymptomatic controls, between performance capacities before and after exercise-induced muscle fatigue (Gleeson, 1999) and muscle damage (Gleeson, 2001). The ability of the assessment procedures to discriminate between control capacities in situations and populations where this might be expected to be compromised offers some preliminary support for the utility and validity of this approach.

As previously mentioned, interest has focused on types of assessment of neuromuscular control in the lower limb that may be considered to more closely mimic the patterns of neuromuscular recruitment associated with stabilization of synovial joints in sporting and work-place environments. These assessments were



designed and piloted originally for application in patients with ACL-deficiency (Doyle, 1998; Doyle, Gleeson and Rees, 1998) but have since received preliminary scientific scrutiny in a wider range of applications including prophylactic interventions in asymptomatic populations (Gleeson et al., 1998; Baltzopoulos and Gleeson, 2001; Walters-Edwards, 2003). This approach to the assessment of neuromuscular control has also evolved to reflect more closely corrective muscle actions to a specified and effective target level of force to stabilise a synovial joint under mechanical stress in a timely manner. For example, alongside the approach to assessment that was described previously involving a single, self-paced muscular effort of a long enough duration of time to be moderated by sensory feedback and reflective of self-regulated force and movements in rehabilitative conditioning (labeled subsequently as neuromuscular control assessment three (NCA3) throughout this thesis), two other modes of assessment have been developed. The latter involve single (NCA1) and serial (NCA2), brief muscle actions involving maximal voluntary recruitment of motor units, respectively. The rationale for these approaches to the assessment of neuromuscular control is introduced further in the following sub-section and example responses associated with NCA1, NCA2 and NCA3 are shown in figure 1.5.

Given the nature of the mechanisms for ligament injury, it may be appropriate to consider a test to represent the situation in the field in which the task is completed in a very short time (300ms) and the athlete has no time to moderate the force output and use afferent and efferent (neuromuscular) information to avoid injury (Rees, 1994; Gleeson et al., 1998). Such a task that is encapsulated by the challenges of NCA1, might mimic to some extent the nature of a rapid response to subtle cues or



stimuli needed to limit the threat posed by an external force applied to the joint. This type of response would be expected to involve stiffening of the joint system by muscle action. Furthermore, there may be times within a game in which the stimuli of perceived disordered biomechanics may be registered consciously and the participant has perhaps two or three strides in which to lessen the mechanical stress on the joint system. Such a situation is reflected in NCA2 and in this case, the capability to produce serial, brief controlling force responses may also be vital. The brief nature of the responses required in NCA1 and NCA2 may be expected to permit only limited moderation of performance by the use of feedback. They might require instead a much greater reliance on feed-forward mechanisms of control. This contrasts with the demands of NCA3 where performance may be modulated by predominantly by feedback mechanisms and be reflective of a situation during the conditioning or rehabilitation of an injured limb to increase strength and neuromuscular control.

In this type of assessment of neuromuscular control, the participants have been required to reproduce a blinded prescribed target force set to 50% of their individual capability for maximal voluntary muscle action. The level of performance has been described by the extent of discrepancy observed between the prescribed target force and the participant's blinded reproduction of the target (constant error or bias) and expressed as a percentage relative to the target force. Further quantification of capability for the control of neuromuscular performance may be achieved by assessing the extent of variability of error (variable error) associated with a sequence of performance trials (Schmidt and Lee, 1999).

Previous research has identified an effective linkage between changes in objective capability for neuromuscular performance caused by exercise stresses or injury and rehabilitation, and changes in self-perceived performance deficiency using psychometric instruments such as the performance profile (Doyle, 1998; Doyle, Gleeson and Rees, 1998; Doyle, Gleeson and Rees, 2002). This approach offers a 'holistic' insight into inconsistency between perceived and true capability for performance and ultimately into whether or not there may be an avenue for misinformed choice about undertaking actions that have risk associated with them during exercise stress in sporting and work-place endeavours. However, it is noteworthy that there has been no scrutiny of self-perception of the capability for control of neuromuscular performance in comparison to objectively assessed capability for control of neuromuscular performance. Potential interrelationships between these performance parameters are shown in figure 1.1. It is plausible that a discrepancy between perceived and objective capability in the knee extensor and flexor musculature may be important as a contributor to the aetiology of ligamentous injury. This is especially so given the evidence for a causal linkage between injury and proprioceptive capabilities (Caraffa et al., 1996; Hewett et al., 1999). Such assessments may offer further insights into the mechanisms underpinning compromised performance associated with exercise stresses. For example, if self-perception of control was judged to be precise and yet objective performance suggested imprecision in task execution instead, then this would indicate compromised sensory capabilities. Conversely, if self-perception of control was able to mimic objective performance (whether precise or not) then this situation would be expected to be indicative of effective sensory performance with any errors in execution associated with compromised motor performance.



#### 1.14 *Measurement efficacy associated with the assessment of neuromuscular control.*

Given the significant role of the neuromuscular control capabilities to prevent knee joint and ACL integrity, the associated assessment tools should possess adequate precision and reliable measurement to discriminate between performance capacities in a situation where this might expected to be compromised for two important reasons:

The first relates to serial measurements on the same individual in which a lack of reproducibility or excessive random error variability may lead to improper interpretation of data. The second is a situation in which the clinician may wish to quantify the confidence and reliability with which they might differentiate between the performance capabilities of patients or individuals within a group so that they may confidently allocate or offer limited resources to those most in need (Mercer and Gleeson, 2002). However, the intra- and inter-individual measurement efficacy of indices of neuromuscular control has yet to be established for the knee flexion movements.

#### 1.15 *Self-perception and neuromuscular system performance.*

Lephart et al. (1998) reported that many studies have examined the knee performance after ACL injury and these studies have shown that once the injury has happened; the capacity of the knee will always be inferior compared to a normal knee. The major function of ACL is to restrain forward translation of the tibia in relation to the femur and to hyperextension, whereas the secondary function is to resist internal and external rotation at near full extension (Butler, 1989; Fu et al., 1993). Furthermore, the ACL can only maintain loads from 1735 to 2000N (Noyes,



1979), this puts the ACL so vulnerable to any un-balanced contraction between the quadriceps and hamstring muscles, especially under dynamic loading (Ihara, 1986; Solomonow et al., 1987).

The elongation of the ACL is important to maintain normal movement during daily life and sports activities, but if the force exceeds maximum limit of the ligament restraint, there is an increase risk of serious ligament injury or damage.

There are a number of external factors used to prevent injury, such as the development of better training techniques and introducing better equipment (Kerr and Fowler, 1988). Researchers have begun to look to other possible factors contributing to sport injury, such as psychological variables to help predict and prevent athletic injury (Janelle et al., 1999).

Life stress has been examined extensively and researchers found a positive relationship between life stress and injury (Janelle et al., 1999). A new conceptual model by William and Andersen, (1998) demonstrated that higher levels of life stress tend to lead to higher injury incidence. *This relationship tends to be moderated by cognitive appraisal (i.e., the athletic perception/interpretation of the stressful circumstance), physiological changes (in muscle tension/coordination and blood flow) and attentional changes (attentional narrowing/distraction).*

Among the psychosocial moderators of athletic injury, self-concept and individual differences were of interest to early research in the area. For example, Brown, (1971) demonstrated no significant relationship between personal factors and injury.

In contrast, a follow-up study by Young and Cohen. (1981) on female athletes demonstrated a significant relationship between self-concept and injury incidence. However, Smith et al. (1990) specify that *'injuries would appear more likely than many other medical outcomes to be influenced by physical-biomechanical factors, leaving Lee variance to be accounted for by psychosocial factors'* (p.366).

Accordingly, Bramwell et al. (1975) state that *'each individual then must react to the conditions in accordance with his perception of the situation and his ability to response based on that perception'* (p.18). psychological factors may effect self-perception of athletes in the real word, weaker individuals on the field playing could produce internal stress, which may introduce muscle inflexibility, failure of motor coordination, attentional disruption, and a greater chance of injury (Williams and Roepke, 1993). Another psychological implication of negative perception of physical strength is that the athlete tries to overcompensate for his weakness by producing quicker movement and manoeuvrability which may, lead to an increased risk of injury (Williams and Roepke, 1993). Also, injury could be emotional to avoid the embarrassment of being consistently physical dominated by an opposing player. This reaction by the player has been described by Petitpas and Danish (1995) as *'a face-saving mechanism'* (p.265). Finally, Fox and Corbin (1989) defined sports competence as *'perception of sports and athletic ability, ability to learn new skills, and confidence in sports environment'* (p.5). This may lead to an increased the chance of injury. This is also supported by Young and Cohen (1981) who demonstrated a similar conclusion on their study. However, these factors considered as internal issues may or may not all contribute to increase the risk of injury during playing seasons (i.e. pre, during and off seasons), furthermore, past studies on self-



perception and injury gave equivocal results, and indeed more studies are needed to establish a conceptual model of testing in order to control some aspects of the internal psychological elements of athletes during training. Thus, if the players learned to use or control the external and the internal factors associated with self-perceived sense, it would be very important to control and maintain motor unit response to mechanical stabilizers around the knee joint and subsequently reduce threatening situations in sports activities.

### 1.16 *Summary*

This introduction and review of literature highlights the role of the neuromuscular system to minimize and/or protect the knee joint from injury and subsequently ACL integrity during sports activities. Fatigue and muscle damage may increase the risk of injury to athletes during critical phases of play, within-play or training activities. Therefore, the neuromuscular control (proprioception) feedback to the CNS by an afferent pathway is very important to coordinate movement patterns to provide a protective muscular contraction and subsequently knee joint stability. This vital coordination of movement patterns is so important to athletes in terms of significant physiological, psychological and financial impact their life career. Thus, this present a great challenge to coaches, sports medicine teams and all personnel in a sports environment to achieve optimal performance and ultimately to reduce risk of injury. Several studies have investigated the effect of fatigue and muscle damage intervention on the neuromuscular system, however most of these investigations have been limited to the upper part of the body and little attention have has been given to the lower body, and more specifically the flexor muscles (Saxton et al.,



1995). Therefore more studies are needed on the lower body following fatigue and exercise-induced muscle damage.

### 1.17 *General aims of the thesis.*

- (i) To examine inter-day reproducibility and single measurement reliability of indices of force error and self-perception of dynamic neuromuscular control in the knee flexors and extensors in men.
  
- (ii) To examine the effects of serial bouts of acute fatiguing exercise on objective and self-perceived indices of neuromuscular control and performance of the knee flexors in men.
  
- (iii) To examine the concomitant effects of exercise-induced muscle damage and fatigue on objective and self-perceived indices of neuromuscular control and performance of the knee flexors in males.

# CHAPTER 2

## GENERAL METHODS

## 2.0 General Methods.

### 2.1 *Participants*

Following a familiarisation session, each participant completed a standard warm-up which consisted of five minutes of cycle ergometry work at an exercise intensity of 90 W/minute followed by 5 minutes of static stretching of the involved muscular prior to testing for each study. Participants were instructed to refrain from strenuous physical activity for the 24 hours before the test and to maintain regular exercise levels throughout the testing occasions. Day to day assessments of performance was completed as near to the same time of the day as possible (+/- 1 hour). The assessment took place in the human laboratory of the School of Sport, Health and Exercise Science, University of Wales, and protocol assessments were approved by the ethics committee of the University of Wales, Bangor.

### 2.2 *Assessment of the knee flexors: Participant and dynamometer orientation.*

Each participant was placed in a prone position on a specially constructed dynamometer (adapted from Gleeson et al., 1995) with the knee flexed passively to 30°. This angle is thought to produce a mechanical stress that may threaten the knee and subsequently the ACL integrity (Gleeson et al., 1998). The lower leg was supported at a position 10-15 cm proximal to the lateral malleolus by a rigid adjustable system. The lever length between the ankle cuff and the lever arm axis of rotation was standardized for each participant during the testing occasions. To limit the movement to the proper muscle groups, participants were firmly strapped by inflexible strapping across the hip, with an additional restraint applied to the thigh close to the involved joint. The axis of rotation of the dynamometer was aligned midway between the lateral condyle of the tibia and the lateral epicondyle of the



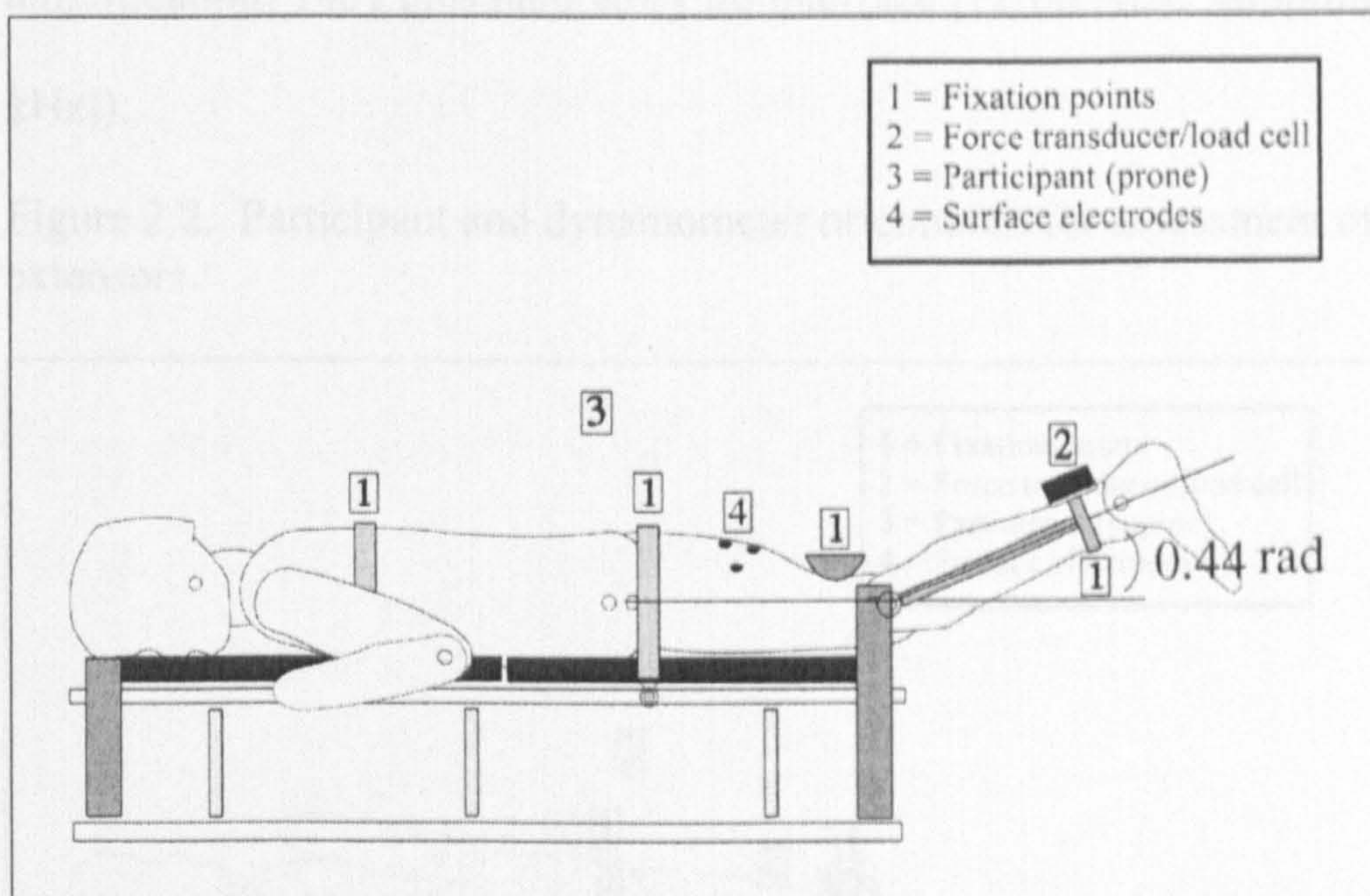
femur consistent with the anatomical axis of the knee joint. The dynamometer (adapted from Gleeson et al., 1995) included a load cell (RDP Electronics Ltd., Wolverhampton, U.K.: range 1000N) and was interfaced to a voltage signal recording system that provided analogue to digital conversion of muscular force interfaced to a data acquisition system (Cambridge Electronic Design Ltd., 1902 medically isolated programmable amplification /filter [zero amplification] 1401 plus laboratory I/O interface [12-bit ADC sampling frequency 4 kHz]).

### *2.3 Volitional muscle actions and establishing target force.*

After a verbal warning (3-2-1-go) from the examiner, each participant attempted to flex the knee joint as forcefully and as quickly as possible against the immovable restraint offered by the apparatus. After an appropriate time of maximal muscle contraction of approximately 2-3 seconds (Saxton et al., 1995) to identify voluntary muscle peak force (i.e. the highest force generated by the single muscle activation), another signal was delivered to the participant to relax as rapidly as possible. A 10 s rest followed, to enable the neuromuscular to recover (Moore and Kukulka, 1991). Following two attempts for practice, this procedure was performed 2 times, separated by 10 seconds, involving musculature (i.e. the knee flexors) at a knee flexion of 30 degree. The target force (acting as blinded target force) was established as 50% of the highest recorded maximal voluntary muscle action at 30° of knee flexion, since maximal voluntary force production following exercise can be reduced by an average of over 50%, according to the expected force-velocity and power-velocity relationship (Hill, 1938). The 'blind' force acted as the target force in all studies in this thesis.



Figure 2.1. Participant and dynamometer orientation for assessment of the knee flexors.



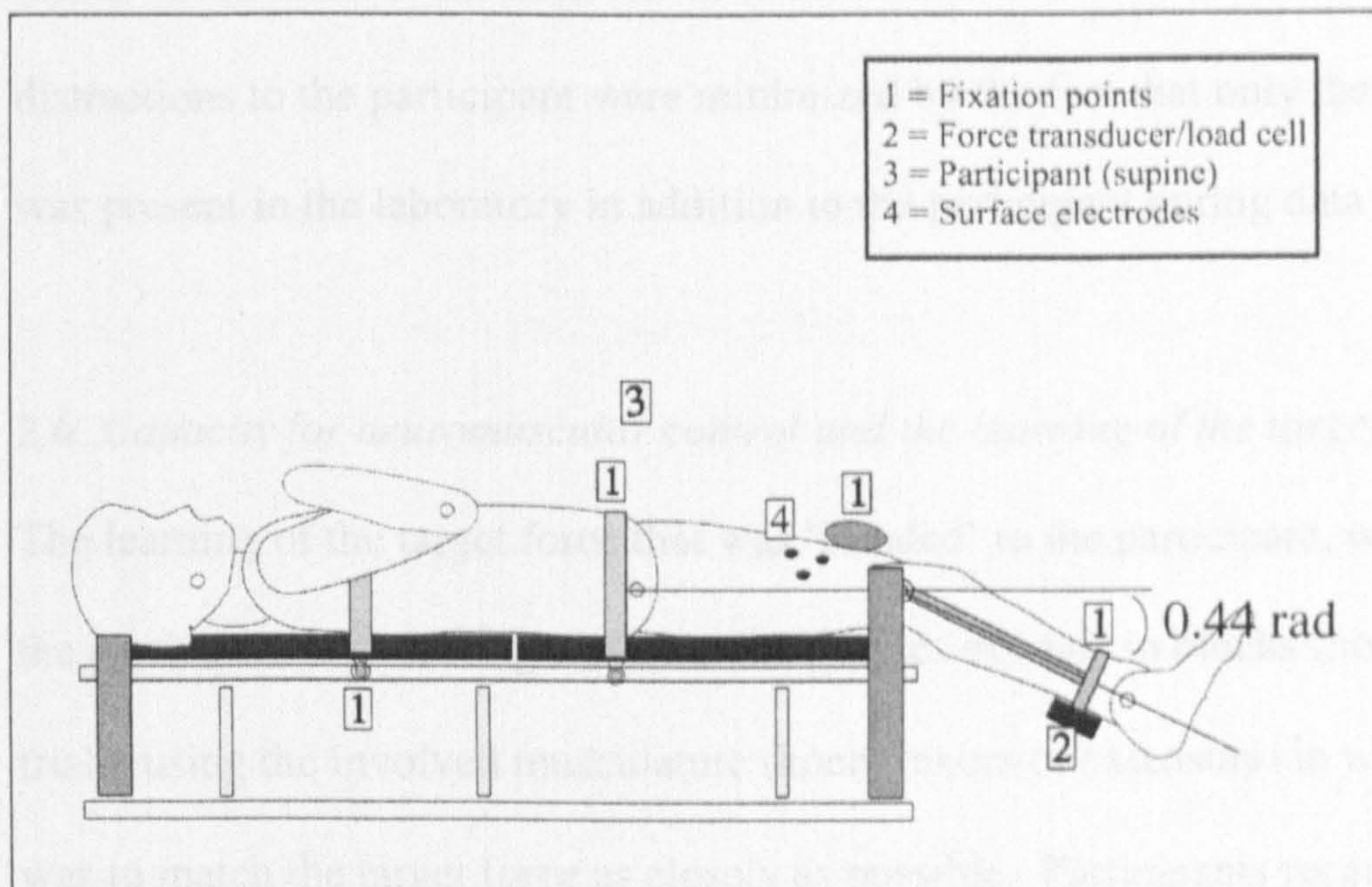
#### 2.4 Assessment of the knee extensors: Participant and dynamometer orientation.

The participant was placed in a supine position on a specially constructed dynamometer (adapted from Gleeson et al., 1995) with the knee extended passively to 30°. The lower leg was supported at a position 10-15 cm proximal to the lateral malleolus by a rigid adjustable system. The lever length between the ankle cuff and the lever arm axis of rotation was standardized for each participant during testing occasions. To limit the movement to the proper muscle groups, participants were firmly strapped by inflexible strapping across the hip, with an additional restraint applied to the thigh close to the involved joint. The axis of rotation of the dynamometer was aligned midway between the lateral condyle of the tibia and the lateral epicondyle of the femur, consistent with the anatomical axis of the knee joint. The dynamometer (adapted from Gleeson et al., 1995) included a load cell (RDP Electronics Ltd., Wolverhampton, U.K.: range 1000N) and was interfaced to a voltage signal recording system that provided analogue to digital conversion of muscular force, interfaced to a data acquisition system (Cambridge Electronic



Design Ltd., 1902 medically isolated programmable amplification /filter [zero amplification] 1401 plus laboratory I/O interface [12-bit ADC sampling frequency 4 kHz]).

Figure 2.2. Participant and dynamometer orientation for assessment of the knee extensors.



### 2.5 Volitional muscle activation.

After a verbal warning (3-2-1-go) from the examiner, each participant attempted to flex the knee joint as forcefully and as quickly as possible against the immovable restraint offered by the apparatus. After an appropriate time of maximal muscle actions of approximately 3 seconds to identify voluntary muscle peak force (i.e. the highest force generated by the single muscle activation), another signal was delivered to the participant to relax as rapidly as possible. A 10 s rest followed to enable the neuromuscular to recover (Moore and Kukulka, 1991). Following two attempts for practice, this procedure was performed 2 times, separated by 10 seconds involving musculature (i.e. the knee flexors) at a knee flexion of 30 degree. This was preceded by a general and a specific warm-up. Achievable target force (acting as



blinded target force) was established as 50% of the highest recorded maximal voluntary muscle action at 30° of knee flexion, since maximal voluntary force production following exercise can be reduced by an average of over 50%, according to the expected force-velocity and power-velocity relationship (Hill, 1938). The 'blind' force acted as the target force in all studies in this thesis. Potential distractions to the participant were minimized by the fact that only the administrator was present in the laboratory in addition to the participant during data collection.

#### *2.6 Capacity for neuromuscular control and the learning of the target force.*

The learning of the target force that was 'blinded' to the participant, was achieved by the participant undertaking a standardised series of trials in blocks (no more than 5 trials) using the involved musculature (knee flexors or extensors) in which the aim was to match the target force as closely as possible. Participants received only contemporaneous standardised verbal feedback from the test administrator to facilitate further improvements in performance precision. In this way, participants were blinded to both the absolute level of the prescribed target force and the scale of measurement used to offer feedback. Participants were effectively learning to self-perceive the performance outcomes in an arbitrary scale of measurement without units. Feedback from the test administrator was offered in standardised terminology such as "25 high", "20 high", "15 high", "10 high", "5 high" and "25 low", "20 low", "15 low", "10 low", "5 low", respectively, depending whether or not the outcome of a trial had been higher or lower than the target (i.e. an underestimation or overestimation of performance, respectively). Trials that showed outcomes that were within  $\pm 2.0$  N of the 'blinded' target force were described in feedback to the participant as having "no error". Verbal feedback from the test administrator was

progressively withdrawn as the participants habituated and accommodated to the requirements of the task. This latter interval corresponded approximately to the 99% confidence limits of the technical error associated with the load cell system and was the least significant difference in performance that could be reliably discriminated by the naked eye of the test administrator from the monitor screen of the computer data acquisition system. The task was deemed to have been learned once the participant was capable of producing a criterion series of ten trials in which seven scores or more showed errors that were within  $\pm 2.0$  N of the 'blinded' target force. This level of performance was achieved once participants had undertaken between 90 and 150 trials. Retention of performance was verified briefly during warm-up and familiarisation trials prior to exercise interventions where appropriate.

For any given performance trial associated with the objective and self-perceived assessments of neuromuscular control NCA1, NCA2 and NCA3, error in performance were computed using the generic expression:  $\text{error} = ((\text{observed performance score} - \text{target performance score}) / \text{target performance score}) \cdot 100\%$

### *2.7 Self-perceived performance.*

During the learning process for the 'blinded' target force, participants were routinely required to report to the test administrator their self-perception of the extent and the direction (positive and negative indicating exceeding and falling-short of the target, respectively) of error associated with individual trials. Initially, participants verbalised these self-perceived scores during preliminary trials using the same arbitrary scale of measurement without units used during the learning process. For example, feedback from the participant was required in standardised terminology



such as “25 high”, “20 high”, “15 high”, “10 high”, “5 high” and “25 low”, “20 low”, “15 low”, “10 low”, “5 low”, respectively, or “no error”, as appropriate. Once the participant had been familiarised with this process of reporting their self-perceived performance outcomes, they were required to transfer these reports to a non-verbal, written format in which a series of miniature visual-analogue scales (using the same unit-less scale of points in the range +25 to -25) were marked appropriately after each block of trials. This contemporaneous approach to the reporting of self-perceived performance capability was adopted throughout all the experimental procedures in this thesis. Serial scores of self-perceived performance capability associated with NCA1, NCA2 and NCA3 were subjected to the same computation procedures to acquire constant and variable error scores as the objectively measured equivalents.

## *2.8 Gravity moment correction.*

Automatic compensation procedures for gravitational error in recorded forces during maximal voluntary muscle actions in the vertical plane were undertaken just prior to testing and on every following testing occasion. Angle-specific force data generated by the effect of gravity acting on the weight of the involved leg of each participant and the weight of the input accessories (including the lever arm) by the software associated with the dynamometer to correct the effects of gravity over the complete range of movement.

## *2.9 Instructions to participants.*

All instructions to participants were given by means of standardized written instruction cards. Participants were not given any information about the results until they had completed all test procedures. The same test administrator performed all testing.

## *2.10 Indices of neuromuscular control and performance..*

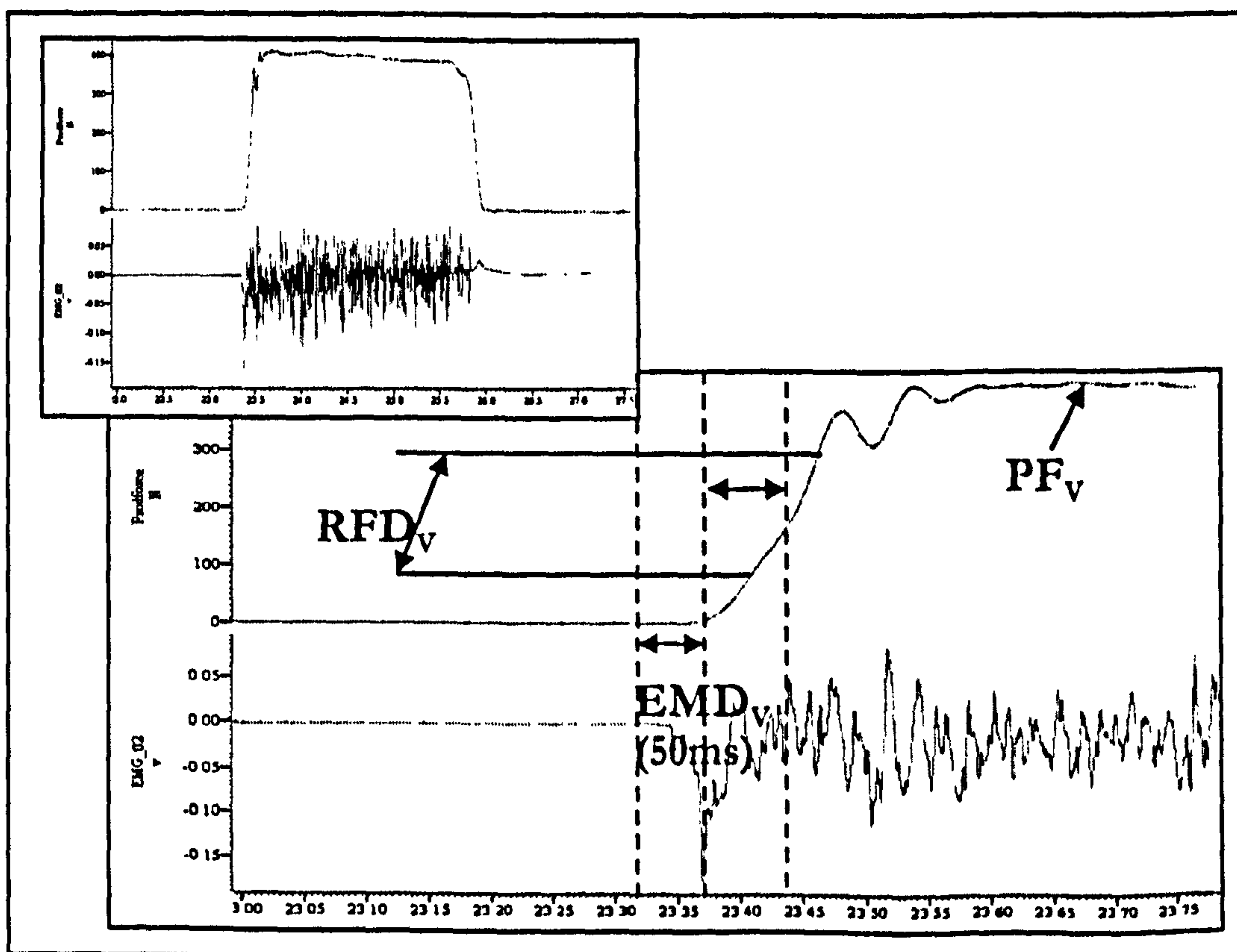
Neuromuscular control was estimated as the difference in force between target and reproduced force, expressed as a percentage of the maximal voluntary muscle activation for the involved muscle group. The mean score of the intra-session estimates of neuromuscular control was used in subsequent statistical analyses to represent individual tests performed in different studies throughout the theses. The specific number of trial sessions at each study was chosen to correspond with the aims of a given study and are described within each study.

The EMDs were recorded with bi-polar surface electrodes (self-adhesive, silver-silver chloride, 10 mm diameter, inter-electrode distance 40 mm centre to centre) applied to the preferred leg following standard skin preparation. The standard skin preparation entailed shaving the concerned area with a razor, abrading the skin with sandpaper and then applying an alcohol swab to remove dead skin, to minimize the resistance between the skin and the electrode (inter-electrode impedance  $<5000 \Omega$ ). Electrodes were placed longitudinally distal to the belly of the biceps femoris muscle on the line between ischial tuberosity and lateral epicondyle of the femur.



The EMG signals were captured by the acquisition system (Cambridge Electronic Design Ltd., UK; Sampling Frequency 4 kHz). The reference electrode was positioned over the lateral femoral epicondyle. The EMGs were archived to hard disk and then analyzed by means of software (Spike 2, version, 3.13, Cambridge Electronic Design Ltd., U.K) to identify the EMD. The latter was defined as the time lag between the onsets of muscle activity (in this case related to biceps femoris muscle) to tension development of force (Gleeson, 2001; defined as the time point at which the force record consistently exceeded the background electrical noise of the load-cell

Figure 2.3. Example data showing; upper trace: example data of force and EMG associated with one MVMA; lower trace: magnification of muscle activation to show representative calculation of indices of volitional neuromuscular performance.



transducer by 0.9 N [i.e. 95% confidence limit of the technical error]). This criterion was consistently used over all the participants.

The index of peak force (PF) was defined as the highest score response in one maximum muscle action. The index of rate of force development (RFD) was calculated as the average rate of force increase associated with the force-time response between 25% PF and 75% PF during one maximum muscle action. The average RFD was used in computations and calculated as the mean responses of repeated trials for each test condition. The index of EMD was calculated from one maximum muscle contraction, based on the definition mentioned above. The EMD was calculated as the mean responses of intra-session trials for each test condition. The numbers of trials used for computational procedures were described in each study.

Each experiment day, peak force (PF), electromechanical delay (EMD) and rate of force development (RFD) responses were assessed by recording the electromyographic activity (EMG) of the biceps femoris muscle of the dominant leg (kick leg) during static maximal voluntary contraction of the knee flexors in prone position. The biceps femoris muscle was chosen for investigation because of its role in restraining anterior- femoral displacement of the knee joint and restraint of the lateral rotation of the femur relative to the tibia, both of which are implicated in the disruption of the ACL integrity (Rees, 1994; Gleeson et al., 1998). The index of peak force was used as a marker of strength performance pre and post experimental intervention and it can provide an important assessment of the extent of fatigue. Similarly, EMD and RFD are markers of the rate of muscle activation and can be initiated by tension development, which gives further insight into the neuromuscular and musculoskeletal performance of a joint system (Gleeson et al., 1998).



# KINANTHROPOMETRY:

## CHAPTER 3

### REPRODUCIBILITY AND SINGLE MEASUREMENT RELIABILITY OF INDICES OF DYNAMIC NEUROMUSCULAR CONTROL OF THE KNEE FLEXORS IN MEN

### **3.0. Reproducibility and single measurement reliability of indices of dynamic neuromuscular control of the knee flexors in men.**

#### **3.1. Introduction**

Contemporary clinical investigations are often concerned with exploring changes in performance abilities measured on different days in which between-day measurements are compared (Beard et al., 1993; Caraffa et al., 1996). For example, during serial clinical visits, such inter-day evaluations of performance may offer the clinician a real opportunity to detect deterioration or rehabilitative advances in an injured limb and any discrepancies between the performance characteristics of injured and contralateral limbs. However, a prerequisite for the proper detection of subtle but potentially important changes in performance in such case-study scenarios is that measurements offer sufficient precision and efficacy. For example, neuromuscular performance in a professional strength athlete may be expected to vary by only  $\pm 5\%$  (95% confidence limits) over the competitive season (Gleeson and Mercer, 1992). Serial measurements on the same individual may lead to improper interpretation of data due to a lack of reproducibility or excessive random error variability.

Another challenge to the efficacy of measurement is the capability to properly differentiate between the capacities for performance of individuals from within the same group (Gleeson and Mercer, 1996). Quantifying the reliability of measurement by intra-class correlations coefficients ( $R_I$ ) and standard error of measurement (SEM) (Thomas and Nelson, 1996) may permit clinicians to quantify the confidence and reliability with which they might differentiate between the performance



capabilities of patients or individuals within a group so that they might confidently allocate or offer limited resources to those most in need (Mercer and Gleeson, 2002).

A primary purpose of any index of neuromuscular function is to offer precision and reliability in estimates of performance (Gleeson and Mercer, 1996; Gleeson et al., 2002). Two aspects of neuromuscular control of the force error (FE %) may be particularly important indicators (indices) of the capability to help regulate knee joint stability and ultimately resistance to injury. These are constant error (CE %), representing the precision of an individual performance relative to the target performance in terms of magnitude and direction, and variability error (VE %), which represents variability or consistency in performing of the same movement during repeated trials (Schmidt, 1988). Furthermore, such indices may also provide indicators of the dynamic protective capacities of a particular joint system during mechanical loading in sports activities (Rees, 1994; Gleeson et al., 1998; Mercer et al., 1998; Gleeson et al., 2000).

Neuromuscular control (proprioception) has been examined in clinical settings by techniques such as the time it takes to detect a threshold of passive motion (kinaesthesia) and joint position sense. In the latter assessment, individuals are required to match with the involved limb the target joint position indicated by ipsilateral and contralateral limb movements. The attempts to match the target position have been undertaken typically without visual feed-back using both active and passive musculature. However, there has been very little validation or comparison of the efficacy of these methods (Beynon et al., 2000). A lack of standardization of assessment protocols and data analysis techniques has led to

difficulties in comparing the outcomes of different studies (Lattanzio et al., 1996; Beynnon et al., 2000; Fischer-Rasmussen et al., 2001). Drouin et al. (2003) investigated the potential relationship between joint position sense and functional hopping tests in athletes and non-athletes. They concluded that the joint repositioning task in both a weight-bearing and non-weight-bearing conditions did not sufficiently estimate the complex integration of peripheral feedback and subsequent efferent response for the performance of dynamic lower limb activities.

Such clinically-based tests might be considered to assess in a valid way the ability to perceive change in or accuracy of joint position using sensory apparatus, it could be argued that such 'slow' assessment methods might not best represent or be entirely valid for the need to assess timely, rapid, forceful, precise and efficient responses from the neuromuscular system to threats posed by sporting endeavours. In such a scenario both the capacity of the sensory afferent (neuromuscular control) to offer accurate assessment of need and the motor efferent to respond with timely and forceful activation patterns to achieve ultimately an efficient outcome (e.g. knee joint protection), should be challenged.

Recent studies have piloted the use of a neuromuscular assessment protocol in which subjects are required to match as precisely as possible, a previously learned 'blind' target force during static knee extension or flexion efforts (Doyle, 1998; Gleeson, 1999; 2000). The force-time history of the subject's response has been set voluntarily but has generally lasted between 2 and 3 seconds. This allows the subject to regulate 'on-line' motor-unit recruitment and ultimately force, on the basis of sensory (proprioception) information. The subject's best match of the prescribed



target force has been indicated by a voluntary relaxation of the involved musculature that can be easily distinguished by the test administrator subsequently by interrogation of the force-time record. This type of approach perhaps best mimics a situation during conditioning or rehabilitation of an injured limb in which the subject is required to self-regulate their training intensity involving muscle activation in order to optimise the process.

This approach to the assessment of neuromuscular control and control regulator capacity has shown significant differences between ACL-deficient patients and asymptomatic controls (Gleeson 1998), between performance capacities before and after exercise-induced muscle fatigue (Gleeson, 1999) and muscle damage (Gleeson, 2001). For example, Gleeson et al. (1998) demonstrated a significant increase in force error (FE %) performance of the injured limb in nine males with unilateral complete ACL rupture (arthroscopically confirmed) before and following reconstructive surgery. There was a significant decrease in force error scores in the injured leg ( $22 \pm 6\%$  before surgery versus  $9 \pm 5\%$  after surgery) compared to the non-injured leg where performance stayed stable across testing times ( $8 \pm 3\%$ ) during a standardised rehabilitation program.

The capability of the assessment procedures to discriminate performance in such circumstances offers some preliminary support for the utility and validity of this approach. These assessments were designed and piloted originally for application in patients with ACL-deficiency (Doyle, 1998; Doyle, Gleeson and Rees, 1998) and form the basis for NCA3. They have since received preliminary scientific scrutiny in a wider range of applications including prophylactic interventions in asymptomatic

populations (Gleeson et al., 1998; Baltzopoulos and Gleeson, 2001; Walters-Edwards, 2003). This approach to the assessment of neuromuscular control has also evolved to reflect more closely corrective muscle actions to a specified and effective target level of force to stabilise a synovial joint under mechanical stress in a timely manner.

Given the nature of the mechanisms for ligament injury, it may be appropriate to consider a test to represent the situation in the field in which the task is completed in a very short time (300ms) and the athlete has no time to moderate the force output and use afferent and efferent (neuromuscular) information to avoid injury (Rees, 1994; Gleeson et al., 1998). Such a task that is encapsulated by the challenges of NCA1, might mimic to some extent the nature of a rapid response to subtle cues or stimuli needed to limit the threat posed by an external force applied to the joint.

This type of response would be expected to involve stiffening of the joint system by muscle action. Furthermore, there may be times within a game in which the stimuli of perceived disordered biomechanics may be registered consciously and the participant has perhaps two or three strides in which to lessen the mechanical stress on the joint system. Such a situation is reflected in NCA2 and in this case, the capability to produce serial, brief controlling force responses may also be vital. The brief nature of the responses required in NCA1 and NCA2 may be expected to permit only limited moderation of performance by the use of feedback. They might require instead a much greater reliance on feed-forward mechanisms of control. This contrasts with the demands of NCA3 where performance may be modulated predominantly by feedback mechanisms and be reflective of a situation during the conditioning or rehabilitation of an injured limb to increase strength and



neuromuscular control. The level of performance has been described by the extent of discrepancy observed between the prescribed target force (50% of maximum voluntary muscle activation force) and the participant's blinded reproduction of the target (constant error or bias) and expressed as a percentage relative to either peak force or the target force.

There has been little scrutiny of the reproducibility and reliability characteristics associated with indices of neuromuscular control that would ensure minimum levels of assessment precision. Furthermore, since a discrepancy between perceived and objective capability in the knee extensor and flexor musculature may be important as a contributor to the aetiology of ligamentous injury, it is notable that little consideration has been given in the literature to the reproducibility and reliability characteristics of indices of self- perception of neuromuscular control.

Although there has been consensus about the importance of the knee flexors to the protection of the knee joint from injury, ipsilateral comparisons of co-activating muscle group neuromuscular performance and control may be important also in the assessment of relative risk to knee joint stability where disordered muscle activation patterns may give rise to escalating net joint moments of sufficient magnitude to damage ligamentous tissue. There has been some suggestion in the scientific literature that assessment protocols investigating the neuromuscular performance of the knee flexors may require more experimental rigour than corresponding protocols for the assessment of the knee extensors in order to ameliorate greater measurement variability and to achieve the same levels of measurement precision (Gleeson and Mercer, 1992). It has been speculated that differential habitual levels of control and

motor unit recruitment patterns in knee extensor and flexor musculature may underpin such differences performance variability. Other research has suggested greater potential for precision for control of movement in the knee flexors due to better afferent input from the muscle-tendon unit compared to the extensor muscles (Friden et al., 1996; Fischer-Rasmussen et al., 2001).

The aim of this study was to examine between-day reproducibility and single measurement reliability of objective and self-perceived indices of force error in the knee flexors and extensors in men.

### *3.2 Methods*

#### *Participants*

Ten adult males (age:  $27.2 \pm 7.61$  years; height:  $1.75 \pm 6.70$  m; body mass:  $72.90 \pm 11.58$  kg [mean  $\pm$  SD]) gave their informed consent to participate in this study. All participants were healthy and engaged in physical activities at least twice a week. Any one with any injuries to the lower part of the body was removed from the study. Participants were instructed not to participate in any exercise 24 hours before testing and on testing days. The ethics committee of the University of Wales, Bangor approved the protocol assessment.

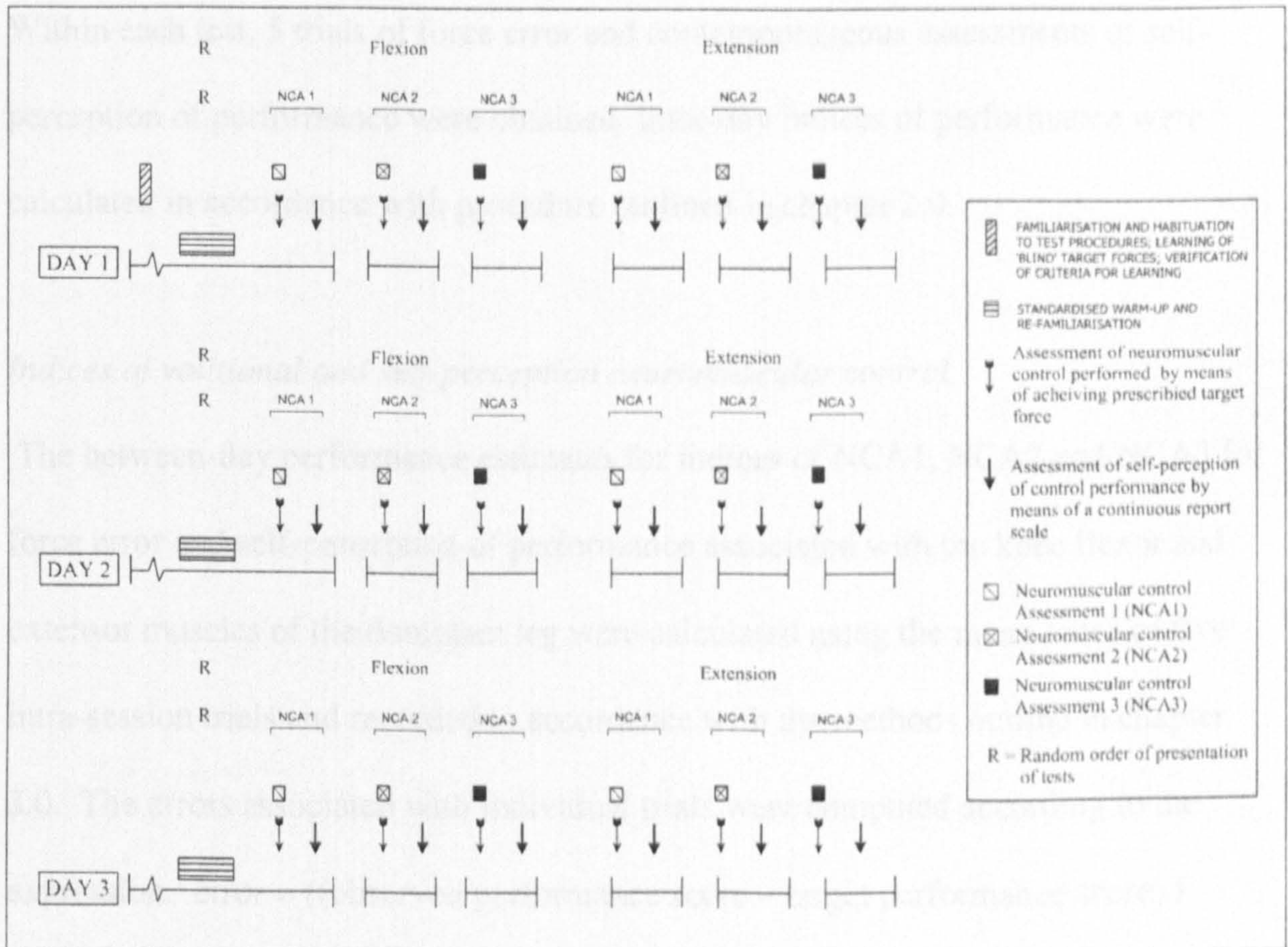
#### *Experimental procedures*

After habituation procedures, each participant completed a general and then a specific warm-up demonstrated by each participant as follows: General warm-up: On each testing occasion participants were requested to carry out a standardised general warm-up consisting of 5 minutes of cycle ergometry work at an exercise



intensity of 90 Watts/ time. *Specific warm-up:* The participant was fixed to the test apparatus (modified from Gleeson et al., 1995) then requested to undertake a specific warm-up consisting

Figure 3.1. Schematic of the protocol for the between-day assessment of indices of reproducibility and single-measurement reliability associated with objective and self-perceived assessments of neuromuscular control NCA1, NCA2 and NCA3 for knee flexor and extensor muscles, respectively.



of 3 x 50%, 2 x 75%, 1 x 95% maximal voluntary static muscle activations of knee flexors against fixed resistance offered by the testing apparatus.

The selected indices of neuromuscular control were described using standardised procedures (Gleeson et al., 2001). One-way repeated measures analysis of variance (ANOVA) was used to assess the likelihood of neuromuscular control strategies being adopted during the between-day trials. Coefficients of variation were used to assess the reliability of the assessments. The establishment of a 'blinded' target force and learning of that target force for the knee flexors and extensors of the dominant leg (kick leg) were undertaken in a random order of presentation and in accordance with the protocol outlined in chapter 3. Assessments of force error (constant and variable error) and self-perception of



neuromuscular control were obtained on three separate days. Inter-day assessment sessions were separated by no less than three days. On each day, neuromuscular control was assessed in six separate tests (flexion or extension and three modes of assessment (NCA1, NCA2 and NCA3); modes of assessment were presented in a random order), each separated by 5 minutes.

Within each test, 5 trials of force error and contemporaneous assessments of self-perception of performance were obtained. Inter-day indices of performance were calculated in accordance with procedure outlined in chapter 2.0.

#### *Indices of volitional and self-perception neuromuscular control.*

The between-day performance estimates for indices of NCA1, NCA2 and NCA3 for force error and self-perception of performance associated with the knee flexor and extensor muscles of the dominant leg were calculated using the mean score of five intra-session trials and recorded in accordance with the methods outline in chapter 3.0. The errors associated with individual trials were computed according to the expression:  $\text{error} = ((\text{observed performance score} - \text{target performance score}) / \text{target performance score}) \cdot 100\%$ .

#### *Statistical Analysis*

The selected indices of neuromuscular control were described using standard statistical procedures (mean  $\pm$  SD). One-way repeated measures analysis of variance (ANOVA) was used to assess the likelihood of intrusion from systematic learning and fatigue effects across inter-day trials. Coefficients of variation (V %), corrected for small sample bias (Sokal and Rohlf, 1981), was used to assess variability of



indices across three inter-day estimates. The latter index was calculated according to the expression  $((SD/mean) \cdot (1 + [1/4N]) \cdot 100)$  and expressed as a percentage of the mean, where N is the number of trials.

Intra-class correlation coefficients ( $R_1$ ) were computed to describe the single-measurement reliability of each objective and self-perceived index of dynamic neuromuscular control and standard error of a single measurement (SEM %)(95% confidence limits), expressed as a percentage of the group mean score according to the formula  $((SD \times \sqrt{(1 - R_1)} / \text{mean}) \times 100)$  (multiplied by 1.96 to compute 95% confidence limits assuming a normal distribution of scores) (Winer, 1981).

Separate 2 x 3 factorial ANOVAs (type of neuromuscular assessment [NCA1, NCA2, NCA3] by muscle group [knee flexors; knee extensors]) with repeated measures were used to assess differences in between-day reproducibility (V% scores) for objective and self-perceived constant error (CE%; SPCE%) and variable error (VE%; SPVE%). Objective and self-perceived performance scores for assessments of neuromuscular control were analysed separately. For all comparisons, the assumption of sphericity was tested by the Mauchly test of sphericity. Any violations of this assumption were corrected using the Greenhouse-Geisser adjustment to raise the critical value of F, as indicated by Stevens (1996). A priori alpha level of 0.05 was applied in all statistical procedures. All statistical analysis was programmed using SPSS (V9.0).

### 3.3 Results

#### Constant error.

Tables 3.1 and 3.2 show group mean (mean  $\pm$  SD) for absolute levels of objective (CE %) and self-perceived (SPCE %) constant error, respectively, expressed as a percentage of target force (%) for assessments of neuromuscular control NCA1, NCA2 and NCA3 for flexor and extensor muscles during inter-day assessments.

A major finding from the present study was that statistical analysis using single-factor ANOVAs with repeated measures revealed no significant difference in absolute performance of neuromuscular control between muscle groups [(flexor and extensor muscles) by mode of test (NCA1, NCA2 and NCA3)], during inter-day assessments. This suggested that learning and other carry-over effects such as fatigue did not intrude in the between-day assessment of neuromuscular control and that variability in performance may be attributed to random error variability (biological variation and technical error).

Table 3.1. Group mean (mean  $\pm$  SD) for constant error (CE %), expressed as a percentage of target force (%) for assessments of neuromuscular control NCA1, NCA2 and NCA3 for flexor and extensor muscles during inter-day assessments.

Inter-day indices assessments						
Days	CE (%) NCA1		CE (%) NCA2		CE (%) NCA3	
	Flex	Ext	Flex	Ext	Flex	Ext
Day-1	1.16 $\pm$ 3.86	-1.09 $\pm$ 2.33	1.26 $\pm$ 4.10	-1.01 $\pm$ 3.90	-0.58 $\pm$ 2.66	-1.07 $\pm$ 1.19
Day-2	0.64 $\pm$ 4.50	-1.12 $\pm$ 1.51	-2.36 $\pm$ 5.10	-1.42 $\pm$ 2.19	-2.68 $\pm$ 2.80	-1.28 $\pm$ 1.84
Day-3	-2.23 $\pm$ 3.96	0.21 $\pm$ 3.27	-1.30 $\pm$ 3.71	-2.08 $\pm$ 3.15	-1.18 $\pm$ 2.10	-1.53 $\pm$ 1.57



Table 3.2. Group mean (mean  $\pm$  SD) for self-perception (SPCE %), expressed as a percentage of target force (%) for assessments of neuromuscular control NCA1, NCA2 and NCA3 for flexor and extensor muscles during inter-day assessments.

Inter-day indices assessments						
Days	SPCE (%) NCA1		SPCE (%) NCA2		SPCE (%) NCA3	
	Flex	Ext	Flex	Ext	Flex	Ext
Day-1	3.20 $\pm$ 1.96	1.47 $\pm$ 2.60	2.55 $\pm$ 2.25	1.49 $\pm$ 2.19	1.93 $\pm$ 2.92	0.63 $\pm$ 2.16
Day-2	2.12 $\pm$ 2.58	0.62 $\pm$ 1.65	-0.83 $\pm$ 2.36	-0.38 $\pm$ 2.40	-0.09 $\pm$ 1.75	1.69 $\pm$ 2.08
Day-3	0.51 $\pm$ 2.10	0.22 $\pm$ 2.87	1.22 $\pm$ 4.24	-0.84 $\pm$ 2.89	1.26 $\pm$ 3.93	2.59 $\pm$ 2.14

The mean and standard deviation (mean  $\pm$  SD) of coefficient of variation (V%, adjusted for small sample bias) for constant error (CE%) and self-perception (SPCE%) for assessments of neuromuscular control NCA1, NCA2 and NCA3 are shown in Tables 3.3 and 3.4 of flexor and extensor muscles respectively, during inter-day assessments.

Table 3.3. Group mean (mean  $\pm$  SD) for of coefficient of variation (V%, adjusted for small sample bias) of constant error (CE%), expressed as a percentage of target force (%), for assessments of neuromuscular control NCA1, NCA2 and NCA3 for flexor and extensor muscles respectively, during inter-day assessments.

Inter-day indices assessments		
V%		
Indices	Flex.	Ext.
CE (%) NCA1	55.7 $\pm$ 188.2	30.8 $\pm$ 284.7
CE (%) NCA2	87.8 $\pm$ 216.7	55.9 $\pm$ 221.5
CE (%) NCA3	124.7 $\pm$ 113.1	155.8 $\pm$ 62.7

Repeated measure ANOVAs on the group mean V% scores (corrected for small sample size, Sokal and Rohlf, 1981) revealed no significant difference between muscle groups by type of test during inter-day assessment times ( $p > 0.05$ ). The results suggested that there were no significant differences in day-to-day variability for neuromuscular control measured by constant error associated with either objective or self-perceived performance in the knee extensor and flexor muscle groups and in the three modes of assessment of neuromuscular control (tables 3.3 and 3.4). The overall group mean coefficients of variability for CE% and SPCE% under these experimental conditions was 85.1% and 133.4%, respectively.



Table 3.4. Group mean (mean  $\pm$  SD) for of coefficient of variation (V%, adjusted for small sample bias) of constant error of self-perceived performance (SPCE%), expressed as a percentage of target force (%), for assessments of neuromuscular control NCA1, NCA2 and NCA3 for flexor and extensor muscles respectively, during inter-day assessments.

Inter-day indices assessments		
V%		
Indices	Flex.	Ext.
SPCE (%) NCA1	188.1 $\pm$ 123.3	127.7 $\pm$ 149.4
SPCE (%) NCA2	153.3 $\pm$ 225.3	46.08 $\pm$ 219.8
SPCE (%) NCA3	118.2 $\pm$ 177.0	166.93 $\pm$ 86.4

Group mean intra-class correlation coefficients ( $R_I$ ) and standard error of the measurement (SEM%)(95% confidence level, expressed as a percentage of the group mean score), for between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 are shown in Tables 3.5 and 3.6 for objective and self-perceived indices of constant error associated with the knee flexor and extensor muscles, respectively.

Table 3.5. Group mean intra-class correlation coefficient ( $R_I$ ) and standard error of the measurement (SEM %) (95% confidence levels, expressed as a percentage of the mean group score), for constant error (CE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 of knee flexor and extensor muscles, respectively.

Inter-day indices assessments				
Indices	$R_I$		SEM%	
	Flex	Ext	Flex	Ext
CE (%) NCA1	0.70	-0.08	97.66	138.87
CE (%) NCA2	0.26	0.18	185.75	137.81
CE (%) NCA3	-0.81	-2.81	139.55	200.09

Table 3.6. Group mean intra-class correlation coefficient ( $R_I$ ) and standard error of the measurement (SEM %) (95% confidence levels, expressed as a percentage of the mean group score), for constant error of self-perceived performance (SPCE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 of knee flexor and extensor muscles, respectively.

Inter-day indices assessments				
Indices	$R_I$		SEM%	
	Flex	Ext	Flex	Ext
SPCE (%) NCA1	-0.59	0.28	158.83	150.19
SPCE (%) NCA2	-0.81	-0.19	119.12	161.07
SPCE (%) NCA3	-0.12	-2.33	160.37	150.07



Intraclass correlation coefficients for constant error of objective (CE%) and self-perceived performance (SPCE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 which ranged between 0.70 and 2.3, did not exceed a clinical acceptable reliability coefficient threshold of greater than 0.80 (Currier, 1984). The findings suggested that the average within-subject variability during between-day assessments exceeded substantively that associated with variability between subjects in this population and therefore, the likelihood of successfully discriminating performance differences amongst individuals within a group on the basis of a single assessment trial would be extremely compromised. Similarly, SEM% scores (95% confidence limits) which offer the potential for moderating the influence of heterogeneity or homogeneity of performance inherent in the sample on  $R_1$  scores, endorse the interpretation of a limited capability to detect changes in neuromuscular control performance based on single-trial assessments. The similarity between objective and self-perceived indices of measurement reproducibility and single-measurement reliability for the assessments of neuromuscular control during knee flexor and extensor activations may be important. It suggest that although they may be compromised for precision and potentially efficacy on the basis of a single measurement, both aspects of performance may be assessed with a similar robustness of (multiple-trial) protocol to achieve a given level of measurement precision.

#### *Variable error.*

The mean and standard deviation (mean  $\pm$  SD) of variable error (VE %) and variable error of self-perceived performance (SPVE%) during between-day assessments

Table 3.7. Group mean (mean  $\pm$  SD) for variable error (VE %), expressed as a percentage of target force (%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 for knee flexor and extensor muscles, respectively.

Inter-day indices assessments						
Days	VE (%) NCA1		VE (%) NCA2		VE (%) NCA3	
	Flex	Ext	Flex	Ext	Flex	Ext
Day-1	5.60 $\pm$ 2.59	3.48 $\pm$ 1.50	4.98 $\pm$ 2.14	3.21 $\pm$ 1.58	2.79 $\pm$ 1.39	2.33 $\pm$ 1.56
Day-2	3.68 $\pm$ 1.66	3.45 $\pm$ 1.14	4.75 $\pm$ 2.63	2.69 $\pm$ 1.72	3.40 $\pm$ 2.35	3.02 $\pm$ 1.58
Day-3	3.34 $\pm$ 1.19	2.67 $\pm$ 0.85	4.00 $\pm$ 1.56	3.46 $\pm$ 1.78	2.87 $\pm$ 1.29	2.66 $\pm$ 1.72

of neuromuscular control NCA1, NCA2 and NCA3 for knee flexor and extensor muscles, are shown in Table 3.7 and 3.8, respectively.



Table 3.8. Group mean (mean  $\pm$  SD) for variable error of self-perceived performance (SPVE %), expressed as a percentage of target force (%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 for knee flexor and extensor muscles, respectively.

Inter-day indices assessments						
Days	SPVE (%) NCA1		SPVE (%) NCA2		SPVE (%) NCA3	
	Flex	Ext	Flex	Ext	Flex	Ext
Day-1	4.20 $\pm$ 2.34	2.26 $\pm$ 2.10	3.98 $\pm$ 2.26	2.20 $\pm$ 1.90	3.16 $\pm$ 2.28	2.40 $\pm$ 1.43
Day-2	3.59 $\pm$ 1.78	2.84 $\pm$ 1.49	3.92 $\pm$ 2.69	2.42 $\pm$ 1.50	4.07 $\pm$ 2.32	1.94 $\pm$ 1.46
Day-3	3.49 $\pm$ 2.03	1.84 $\pm$ 1.34	2.39 $\pm$ 1.76	2.48 $\pm$ 3.46	2.67 $\pm$ 2.79	1.02 $\pm$ 1.01

One-way repeated measure ANOVAs with repeated measures revealed no significant difference ( $P > 0.05$ ) in variable error (VE %) and variable error of self-perceived performance (SPVE%) across inter-day trials. This suggested that day-to-day changes in neuromuscular control performance can be attributed to biological variation and technical error rather than to systematic learning or fatigue effects.

The mean and standard deviation (mean  $\pm$  SD) of coefficient of variation (V%, adjusted for small sample bias) for variable error (VE%) and variable error of self-perceived performance (SPVE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 for knee flexor and extensor muscles are shown in Tables 3.9 and 3.10, respectively.

Table 3.9. Group mean (mean  $\pm$  SD) for of coefficient of variation (V%, adjusted for small sample bias) of variable error (CE%), expressed as a percentage of target force (%), during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 for knee flexor and extensor muscles, respectively.

Inter-day indices assessments		
V%		
Indices	Flex	Ext
VE (%) NCA1	43.21 $\pm$ 24.24	38.32 $\pm$ 18.72
VE (%) NCA2	46.09 $\pm$ 22.27	50.48 $\pm$ 18.50
VE (%) NCA3	40.03 $\pm$ 18.48	43.52 $\pm$ 25.50

Table 3.10. Group mean (mean  $\pm$  SD) for of coefficient of variation (V%, adjusted for small sample bias) of variable error of self-perceived performance of (SPVE%), expressed as a percentage of target force (%), during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 for knee flexor and extensor muscles, respectively.

Inter-day indices assessments		
V%		
Indices	Flex	Ext
SPVE (%) NCA1	43.65 $\pm$ 37.90	73.05 $\pm$ 43.65
SPVE (%) NCA2	53.12 $\pm$ 28.86	91.94 $\pm$ 36.80
SPVE (%) NCA3	75.75 $\pm$ 52.42	83.42 $\pm$ 34.84



Repeated measure ANOVAs of V% scores (corrected for small sample size, Sokal and Rohlf, 1981) revealed no significant differences in variability/reproducibility of neuromuscular control performance between muscle groups and between the type of test during inter-day assessments ( $P > 0.05$ ). Coefficients of variation ranged between 38.2% (VE%, NCA1, extension) to 91.9% (SPVE%, NCA2, extension). This suggested insufficiency of precision of measurement based on single-trial assessments. In a similar way to the findings for CE%, the statistical equivalence of the reproducibility between objective and self-perceived performance suggest that while each may be compromised in terms of absolute precision, protocols of equivalent rigour (i.e. the use of the mean scores of the same number of trials in each protocol to reduce error variability) could be used to establish a given level of measurement precision for both approaches.

Group mean intra-class correlation coefficients ( $R_I$ ) and standard error of the measurement (SEM%)(95% confidence level, expressed as a percentage of the group mean score), for between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 are shown in Tables 3.11 and 3.12 for objective and self-perceived indices of variable error associated with the knee flexor and extensor muscles, respectively.

Intraclass correlation coefficients for both variable error of objective (VE%) and self-perceived performance (SPVE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 exceeded a clinically acceptable reliability coefficient threshold of greater than 0.80 (Currier, 1984) in one experimental condition only (0.84; SPVE%, NCA1). The remainder of conditions

suggested that the average within-subject variability during between-day assessments exceeded substantively those associated with variability between subjects in this population. Therefore, the likelihood of successfully discriminating performance differences amongst individuals within a group on the basis of a single assessment trial would be extremely limited. Similarly, SEM% scores (95% confidence limits) support the interpretation of a limited capability to differentiate amongst the neuromuscular control performance of individuals within a group based on single-trial assessments. The general similarity between the levels of reproducibility and single-measurement reliability of both objective and self-perceived variable error indices of neuromuscular control during knee flexor and extensor activations may be assessed with a similar robustness of (multiple-trial) protocol to achieve a given level of measurement precision.

Table 3.11. Group mean intra-class correlation coefficient ( $R_1$ ) and standard error of the measurement (SEM %) (95% confidence levels, expressed as a percentage of the mean group score), for variable error (VE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 of knee flexor and extensor muscles, respectively.

Inter-day indices assessments				
Indices	$R_1$		SEM%	
	Flex	Ext	Flex	Ext
VE (%) NCA1	-0.16	0.28	48.70	28.07
VE (%) NCA2	0.07	-0.17	53.40	53.25
VE (%) NCA3	0.74	0.79	25.40	30.67



Table 3.12. Group mean intra-class correlation coefficient ( $R_1$ ) and standard error of the measurement (SEM %) (95% confidence levels, expressed as a percentage of the mean group score), for variable error of self-perceived performance (SPVE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 of knee flexor and extensor muscles, respectively.

Inter-day indices assessments				
Indices	$R_1$		SEM%	
	Flex	Ext	Flex	Ext
SPVE (%) NCA1	0.84	0.25	23.27	45.64
SPVE (%) NCA2	0.59	-0.31	47.15	56.69
SPVE (%) NCA3	0.71	0.17	30.70	54.42

### 3.4 Discussion

#### *Constant error and implications for the efficacy of measurement.*

The group mean constant error scores during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 of knee flexor and extensor muscles, were  $-0.14 \pm 3.41\%$ ,  $-0.80 \pm 2.85\%$ ,  $-1.45 \pm 1.32\%$  and  $-0.67 \pm 1.40\%$ ,  $-1.50 \pm 1.90\%$ ,  $-1.29 \pm 0.57\%$ , respectively. The level of error and discrepancy in performance in the current study is broadly in agreement with results from a study by Marks (1994) involving assessment of the neuromuscular control of the knee flexors. The latter study demonstrated that during three inter-session assessments, the absolute error (AE) of position sense was on average  $1.06^\circ$  based on a possible movement range of  $90^\circ$  (i.e. 1.2% error). In the present study involving both objective and self-perceived day-to-day assessments of neuromuscular control,

participants appeared to have consistently over-estimated their performance capability in absolute terms (i.e. produced lower forces than the target performance, table 3.1). In contrast, there is a suggestion that participants self-perceived their performances to be greater than the target force (table 3.2). Such overestimation of objective performance capabilities and inefficiency may leave joint systems vulnerable to injury during functional activities (Boden et al., 2000; Huston and Wojtys, 1996), while discrepancies between objective and self-perceived capabilities may offer an avenue inappropriate choice of intensity of exercise activity.

The reproducibility of neuromuscular control as measured by constant error and variable error of objective performance was statistically similar across knee flexor and extensor conditions and modes of testing (NCA1, NCA2, and NCA3). Overall V% scores of 85.1% (CE%) and 133.4% (VE%), (group mean coefficients of variability), respectively, suggest considerably inflated between-day error variability and correspondingly inferior measurement reproducibility compared to those observed in the assessment of indices of neuromuscular performance. For example, peak force or average peak force in men and women during isokinetic leg strength testing demonstrated low between-day error variability (V%= 2%-4%, approximately) (Gleeson and Mercer, 1992). Reproducibility associated with constant and variable error indices of self-perceived neuromuscular control performance showed similar apparently inflated between-day error variability scores of 43.6% (SPCE%) and 70.1% (SPVE%) (group mean coefficients of variability), respectively, across knee flexor and extensor conditions and modes of testing (NCA1, NCA2, and NCA3).



*Contemporary issues in the interpretation of reproducibility of constant error.*

However, some caution should be exercised when attempting to interpret such apparently large discrepancies in the extent of reproducibility associated with neuromuscular performance and control. Such large discrepancies in reproducibility performance as measured by  $V\%$  may be accounted for by the method of computation of error interacting with differences in the nature of the target score. For example, given the expression for the computation of  $V\%$  (i.e.  $V\% = \text{SD}/\text{mean} \times 100\%$ ), an apparently inflated coefficient of variation of constant error associated with day-to-day testing of some 200% may be accounted for by a moderate error in force production (0.67 N in absolute values, mean of 5 trials) in attempting to achieve the target force, in combination with relatively homogenous day-to-day variability (SD) of approximately  $\pm 1.4$  N. An equivalent expression of the day-to-day variability of peak force (mean score 200 N, for example) exhibiting the same extent of homogeneity of scores ( $\pm 1.4$  N) would be less than 0.7%. To further contextualise absolute day-to-day variability of constant errors of  $\pm 1.4$  N, ACL-deficient patients have demonstrated changes in blinded constant error (equivalent to NCA3) of up to 40 N (or 25% of peak force, approximately) (Doyle, Gleeson and Rees, 1998). The latter suggests that although  $V\%$  scores for day-to-day variability in constant error of indices of neuromuscular control appear to be relatively gross, the likely raw effect size of change in populations of interest may be several-fold larger. Thus such indices of performance might offer the possibility of appropriate precision of measurement in some circumstances. Furthermore, it should be noted also that technical error may contribute some 0.9 N (95% confidence limits) of the  $\pm 1.4$  N used in this example of absolute day-to-day variability of constant errors.

Overall, the perception of the extent of error and performance variability associated with day-to-day assessments of neuromuscular control is likely to be dependent on the criterion by which the score is expressed. In this thesis, the method of computation was selected to offer consistency with that broadly adopted within the area of study involving motor control.

The computation of 95% confidence limits of V% scores used in conjunction with the Central Limit Theorem suggests that the mean score of more than 350 inter-day would be required to achieve a level of measurement precision of arbitrarily better than  $\pm 5\%$ . This level of precision would facilitate the detection of subtle but potentially important day-to-day changes to intra-individual (case-study) neuromuscular control performance capabilities for a given individual. Since the analysis of variability revealed no significant differences between muscle groups and the mode of testing during inter-day assessments in this study, this type of protocol rigour would be required for all objective assessments of knee flexor and extensor performance using NCA1, NCA2 and NCA3. These scores present a logistical threat to measurement efficacy especially during intra-subject assessments. Furthermore, these estimates related to group mean response that does not reflect fully the indices on both muscle groups the heterogeneity of performance of participants within this sample (Gleeson et al., 1995). Corresponding findings for the reproducibility of the constant error of self-perceived performance suggest that the mean score of more than 700 inter-day trials would be required to achieve equivalent levels of measurement precision.



*Variable error and implications for the efficacy of measurement.*

The variable error reflects the consistency of the constant error score around a particular target in terms of undershooting or overshooting (Saxton et al., 1995).

The analyses of V% scores for both objective and self-perceived indices of variable error of neuromuscular control revealed no significant differences between muscle groups and mode of testing. The level of reproducibility associated with these indices was similar to those associated with constant error indices of neuromuscular control. This suggested that indices of neuromuscular control involving variable error would be subject to similar concerns about measurement efficacy.

It is interesting to note that while the  $R_1$  scores for CE%, VE%, SPCE% and SPVE% indices of NCA1, NCA2 and NCA3 under most experimental conditions in this study have not exceeded a clinically acceptable coefficient threshold of greater than 0.80 (Currier, 1984), the general levels of single measurement reliability approximate to those reported in the literature for reproduction of knee angle assessments used in contemporary clinical practice (0.49-0.82; -0.75 ; 0.17-0.79) reported by Saxton et al., 1995, Marks and Quinney, 1996 and Kramer et al., 1997, respectively.

The observed  $R_1$  scores in the present study suggested that the average within-subject variability during between-day assessments exceeded substantively those associated with variability between subjects in this population. Therefore, the likelihood of successfully discriminating performance differences amongst individuals within a group on the basis of a single assessment trial would be extremely limited.

Similarly, SEM% scores (95% confidence limits) support the interpretation of a

limited capability to differentiate amongst the neuromuscular control performance of individuals within a group based on single-trial assessments.

Constant and variable error indices of objective and self-perceived performance during between-day assessments of neuromuscular control may be compromised for precision and potentially efficacy on the basis of a single measurement when attempting to discriminate between the performances of individuals within a group. However, the similarity of single measurement reliability scores in this study between objective and self-perceived indices of neuromuscular control suggest that both aspects of performance may be assessed with a similar robustness of (multiple-trial) protocol to achieve a given level of measurement precision.

In this context, these indices of performance would need to be deployed with caution in clinical applications. For example, Spearman-Brown prophecy formula (Winer, 1981) used in conjunction with both  $R_1$  and  $SEM\%$  scores suggested that in general, individuals within a group would need to accrue some 900 inter-day trials of NCA1, NCA2 or NCA3 in order to achieve discrimination of better than  $\pm 5\%$  (of the group mean score) between individuals with 95% confidence levels. While such protocol requirements seem to be excessive, there are other precedents in the scientific literature for this type of logistical threat to measurement precision. For example, some 2000 trials have been required to detect a small  $\pm 3$  degree change in joint angles within a target zone (Michale et al., 2002).



### *Conclusions.*

In conclusion, the reproducibility and single-measurement reliability of objective (CE%, VE%) and self-perceived performance (SPCE% and SPVE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3 offered compromised precision and efficacy. These indices of performance should be deployed cautiously within both case-study and inter-individual comparisons and must rely on multiple-trial protocols to achieve acceptable levels of measurement precision in such circumstances. However, appropriate criteria of experimental design sensitivity should ensure that such indices of neuromuscular control may be used effectively in the testing of experimental hypotheses associated with inter-group comparisons and repeated-measures designs of performance differences. Such considerations will be investigated within the thesis' subsequent studies that will investigate the effects of fatiguing tasks and exercise-induced muscle damage on selected indices of objective and self-perceived performance of neuromuscular control.

INTERVENTIONS:

CHAPTER 4

THE EFFECT OF SERIAL  
FATIGUE TASKS ON  
NEUROMUSCULAR CONTROL  
AND PERFORMANCE AT THE  
KNEE FLEXORS IN MEN



## **4.0 The effect of serial fatigue tasks on neuromuscular control and performance of the knee flexors in men**

### **4.1 Introduction**

The first study (reproducibility and single measurement reliability) in this thesis has focused attention on the potential efficacy and measurement precision associated with a relatively new approach to the assessment of neuromuscular control that might mimic the responses to stresses inherent in the competitive sporting environment. The neuromuscular control assessment protocols [NCA1, NCA2 and NCA3] involved single and serial brief muscle actions, and a single self-paced muscle action, respectively and involved both objective and self-perceived assessments of the extent of error in performance of force-matching tasks. Results from the first study showed no significant differences between these assessments in terms of their reproducibility and reliability characteristics. This indicated that each would offer equivalent suitability for inclusion within subsequent experimental studies based on the selection of appropriate criteria for experimental design sensitivity.

Injuries have become one of the most significant dilemmas facing athletes on all levels, despite the advancement of medicine and facilities. The numbers of injuries have continued to increase with increasing participation in sports and recreation (Laurie et al., 2001). The knee is the most commonly injured joint, and serious damage to the knee ligament is recurrent (Kelsey, 1982; Miyasaka et al., 2001). The anterior cruciate ligament (ACL) is one of many ligaments present in the knee and is the principal ligamentous restraint to the

anterior tibio-femoral displacement (Rees, 1994). The ACL is the most frequently injured ligament in the knee joint (Daniel and Fritschy, 1994; Rees, 1994; Griffin et al., 2000).

There is accumulating evidence of an ACL injury epidemic by means of non-contact aetiologies in team sport athletes (Noyes et al., 1983; Anthony and William, 1998). This injury often occurs during eccentric action of the knee extensors, when the flexor muscle group fails to assist effectively in the stabilisation to the knee joint by not regulating either anterior tibio-femoral displacement or tibio-femoral rotation (Arms et al., 1984). This process is thought to confer levels of mechanical stress on the ACL that may threaten its integrity, especially between 30 and 0 degrees of knee flexion (Anthony and William, 1998).

There are a number of factors that are thought to contribute to anterior cruciate ligament (ACL) injuries; for example, anatomic factors such as intercondylar notch, ACL size (Harner et al., 1994), lack of conditioning or training (Hewett et al., 1999), biomechanical insufficiency (Gleeson et al., 1998) which have already received considerable attention in the literature. However, while loss of neuromuscular control has been considered previously as an important factor in the risk of ACL injury (Carafa et al., 1996; Hewitt et al., 1999), the influence of fatigue on neuromuscular control has received only limited scrutiny.

To date, the findings on the effects of fatigue on knee neuromuscular control have been somewhat ambiguous. For example, Skinner et al., (1986) studied knee proprioception



performance in healthy young men. They found a decrease in the ability to reproduce knee joint angles after a series of interval running sprints (3.75 miles in total). In contrast, a study by Marks and Quinney (1993) found that 20 maximal isokinetic quadriceps contractions that acted as the fatigue intervention in young sedentary women did not significantly reduce knee proprioception performance. In both studies, knee proprioception was measured using a popular technique consisting of testing a participant's ability to reproduce specific knee joint angles in an open-kinetic sitting position (Barrack et al., 1991; Marks & Quinney, 1993; Skinner et al., 1986). While these two studies have produced conflicting results, comparisons of their findings should be undertaken with caution since they differed in fatigue intervention protocols and in the characteristics of the participants.

There is accumulating evidence that fatigue processes have been associated with risk of injury (Hawkins et al., 2001; Gleeson et al., 1998). Muscular fatigue has been defined as the temporary inability to sustain power output or force during repeated muscle contraction (Gibson and Edward, 1985). Bigland-Ritchie and Woods (1984) reported that one of the most significant signs of fatigue is a decline in maximum force (strength). It has also been reported that strength is reduced after sustained isometric contraction (Fellows et al., 1993; Woods et al., 1987) and with the performance of long-lasting, low-intensity efforts (Avela et al., 1999a; Nicol et al., 1991).

This extent of reduction in force (strength) capabilities associated with acute functional activities (Gleeson et al., 1998) may produce sufficient neuromuscular deficiencies within the muscle (Skinner et al., 1986; Green, 1987) to impose a threat to knee joint stability

during sports performance and is thought to correlate strongly with injury occurrence (Gleeson et al., 1998). More recently, researchers have undertaken prospective epidemiology studies to investigate the hypothesised relationship between fatigue and injury further (Laurie et al., 2001; Gabbett, 2000; Pinto et al., 1999; Hawkins et al., 2001). For example, Hawkins et al., (2001) examined injuries sustained in English professional football over a period of two seasons. The study showed a prominent increase in the incidence of injury towards the latter stages of the first half and especially in the second half.

In addition to weakening peak force, muscle fatigue is also believed to negatively affect other indices of neuromuscular performance capacity such as the rate of force development and electrochemical delay (EMD) (Winter and Brookes, 1991). Electrochemical delay (EMD) is defined as the time lag between the onset of muscle activity and tension development (Zhou et al., 1996). Winter and Brookes (1991) proposed that the time taken for the contractile component to stretch the series elastic component of the muscle primarily determines the length of EMD. Subsequently, recent studies proposed a link between a prolonged EMD and injury (Wojtys et al., 1996; Gleeson et al., 1998).

Interest has focused on types of assessment of neuromuscular control in the lower limb that may be considered more closely corrective muscle actions to a specified and effective target level of force to stabilise a synovial joint under mechanical stress in a timely manner. These assessments were designed and piloted originally for application in patients with ACL-deficiency (Doyle, 1998; Doyle, Gleeson and Rees, 1998) but have since received



preliminary scientific scrutiny in a wider range of applications including prophylactic interventions in asymptomatic populations (Gleeson et al., 1998; Baltzopoulos and Gleeson, 2001; Walters-Edwards, 2003). Single (NCA1) and serial (NCA2), brief muscle actions involving maximal voluntary recruitment of motor units, respectively, have been developed subsequently alongside single, self-paced muscular efforts of a long enough duration of time to be moderated by sensory feedback and reflective of self-regulated force and movements in rehabilitative conditioning (NCA3). In these assessments, the participants have been required to reproduce a blinded prescribed target force set to 50% of their individual maximal voluntary muscle action. The discrepancy observed between the prescribed target force and the participant's reproduction of the target (constant error) was expressed as percentage relative to peak force. Variability error represents variability or consistency in the performing of the same movement during repeated trials (Schmidt, 1988). These two aspects of neuromuscular control may be particularly important indicators of the capability to help regulate knee joint stability and ultimately resistance to injury.

Few researchers have investigated changes to neuromuscular performance and neuromuscular control during serial episodes of fatiguing exercise. Furthermore, no one has investigated whether or not an individual's self-perception of neuromuscular control is affected by serial fatiguing episodes of exercise. It is likely that imprecision of self-perception may contribute to a process by which injury becomes more likely. For example, overestimation of performance may leave the participant partially unprotected during an episode of externally imposed mechanical stress.

The aim of this study was to examine the effects of serial bouts of acute fatiguing exercise on objective and self-perceived indices of neuromuscular control and performance of the knee flexors in men.

## *4.2 Methods*

### *Participants*

Twelve adult males (age  $28.6 \pm 6.1$  years; height  $183.0 \pm 8.5$  cm; body mass  $72.3 \pm 13.0$  kg [mean  $\pm$ SD]) from the student population at the University of Wales, Bangor volunteered to take part in this study. Inclusion and exclusion criteria were that participants needed to be healthy and engaged in physical activities at least twice a week and that any potential participant with current musculoskeletal injury to the lower part of the body would be routinely excluded from the study. Participants were instructed not to participate in any exercise 24 hours prior to testing and on the days of testing. All participants gave written consent to participate in this study. The assessment protocols were approved by the Ethics Committee for Human Testing of the School of Sport, Health and Exercise Sciences.

### *Procedures*

#### *Familiarisation and preliminary tests*

The initial meeting in this study entailed the familiarisation of participants to the criterion test and testing environment. In this initial meeting, the participant's peak force was established as follows. After a general and specific warm-up, a verbal warning from the examiner ('3-2-1-go'), each participant attempted to flex the knee joint as forcefully and as quickly as possible against the immovable restraint offered by the apparatus. After an



appropriate time of maximal muscle activity of approximately 3 seconds to identify maximum voluntary muscle activation (MVMA) force (i.e. the highest force generated by the single muscle activation), another signal was delivered to the participant to relax as rapidly as possible. A recovery period (10 s) followed. Following another practice attempt, this procedure was performed a further 2 times, separated by 10 seconds of rest using the involved musculature (i.e. the knee flexors) at a knee flexion of 30 degree. The 'blinded' target force for assessments of neuromuscular control was established as 50% of the highest recorded maximal voluntary muscle action at 30° of knee flexion. This level of force corresponds approximately to that associated with peak muscular power outputs during sporting endeavours according to the expected force-velocity and power-velocity relationships (Hill, 1938). The force acted as the 'blind' target force for all-subsequent assessments of neuromuscular control. Potential distractions to the participant were minimised by the fact that only the administrator was present in the laboratory in addition to the participant during data capture.

Following the establishment of MVMA force and blinded target force, participants were familiarised to all assessment procedures of neuromuscular control that had been selected for inclusion in this study (NCA1, NCA3 and a reproduced joint angle task (RJA) Lattanzio and Petrella ,(1998), together with protocols for the assessment of neuromuscular performance. The NCA1 and NCA3 assessments of neuromuscular control required participants to produce a muscle action eliciting a blinded target force of 50% MVMA. On achievement of the blinded target force, participants were instructed to either relax immediately after they felt the prescribed force had been attained in the case of NCA1 or to

maintain the blinded target force for 2-3 seconds before relaxing to signal task completion. Participants were familiarised to the reproduced joint angle (RJA) task passively using a 'blinded' 30 degree target angle and initiation of movement from various reference angles. All assessments of neuromuscular control followed a specific procedures for learning the prescribe target force as described below.

### *Learning of target force for NCA1 and NCA 3*

The learning of the target force that was 'blinded' to the participant, was achieved by the participant undertaking a standardised series of trials in blocks (no more than 5 trials) using the involved musculature (knee flexors) in which the aim was to match the target force as closely as possible. Participants received only contemporaneous standardised verbal feedback from the test administrator to facilitate further improvements in performance precision. In this way, participants were blinded to both the absolute level of the prescribed target force and the scale of measurement used to offer feedback. Full details of these procedures are described in chapter 2 (General methods, section 2.6). The task was deemed to have been learned once the participant was capable of producing a criterion series of ten trials in which seven scores or more showed errors that were within  $\pm 2.0$  N of the 'blinded' target force. Retention of performance was verified briefly during warm-up and familiarisation trials prior to exercise interventions.



*Self-perceived performance (NCA1, NCA3).*

During the learning process for the 'blinded' target force, participants were routinely required to report to the test administrator their self-perception of the extent and the direction (positive and negative indicating exceeding and falling-short of the target, respectively) of error associated with individual trials. Initially, participants verbalised these self-perceived scores during preliminary trials using the same arbitrary scale of measurement without units used during the learning process. For example, feedback from the participant was required in standardised terminology such as "25 high", "20 high", "15 high", "10 high", "5 high" and "25 low", "20 low", "15 low", "10 low", "5 low", respectively, or "no error", as appropriate. Once the participant had been familiarised with this process of reporting their self-perceived performance outcomes, they were required to transfer these reports to a non-verbal, written format in which a series of miniature visual-analogue scales (using the same unit-less scale of points in the range +25 to -25) were marked appropriately after each block of trials. Full details of these procedures are described in chapter 2 (General methods, section 2.7). Similar procedures to these for self-perceived performance during NCA1 and NCA3 assessments were adopted for the self-perception of performance during the RJA (please see below).

Assessment of reproduced joint angle (RJA).

This assessment used in this study for the reproduction of knee angles as was described by Lattanzio<sup>1</sup> and Robert, (1998). This involves evaluating the participant's ability to reproduce specific knee angles, as determined by the examiner. From a reference angle, the participant's lower extremity was moved to a specific angle for a few seconds and then returned to the reference angle. With angle vision out of range (blind), the participants were asked to reproduce the test angle. The angular error (ignoring the direction of the error) between the test angle and the participant's perceived angle was recorded. Reproduction of knee angles in this present study were performed in open kinetic chain manner (sitting), and the examiner performed movements passively.

*Reproduction of knee joint angle and familiarisation procedures.*

A MIE (Leeds, UK) goniometer was used in this experiment to record the knee angles. The MIE goniometer was positioned in the lever arm of a specially constructed dynamometer (adapted from Gleeson et al., 1995). The familiarisation of the target angle was attained within an accuracy of  $\pm 3^\circ$ . The  $\pm 3^\circ$  was chosen based on a study involving 2000 experimental trial performed by Michale (2002). Each participant was given two trials to become familiar with the procedure.

In this study, participants passively reproduced joint angles of  $30^\circ$  (i.e. target angle), from three reference angles  $15^\circ$ ,  $45^\circ$  and  $60^\circ$ , once. The examiner passively moved the dominant leg to the target angle ( $30^\circ$ ), from selected reference angles in random order, to target angles and held the leg in this position for 3 seconds, then returned it to the reference

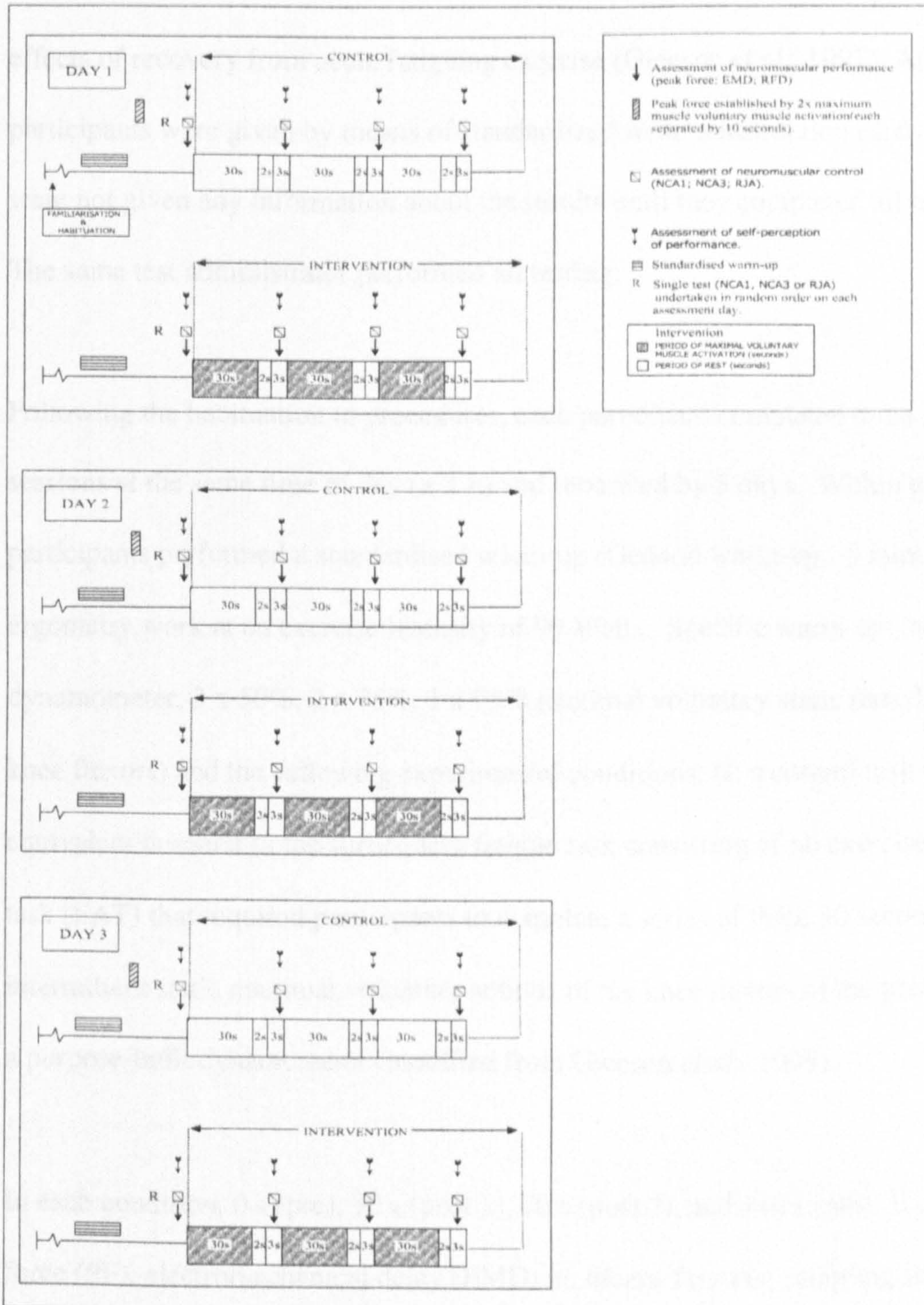
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<sup>1</sup> The reproduced matching angle only applied to the fatigue study.



position. While blinded, the participants were asked to reproduce passively the target angle from one of these reference angles once. The reproduced matching angle was repeated two more times as described above and the examiner used the above reference angles in random order. Participants were then required to record their self-perceived accuracy of effort within 5 seconds.

Figure 4.1. Schematic of the protocol for the assessment of the effects of serial an acute fatiguing task on objective and self-perceived volitional neuromuscular control and performance of the knee flexors.





### *Experimental protocol*

An inter-day experimental design involving repeated measures was chosen to facilitate the assessment of serial estimates of neuromuscular control while avoiding the intrusion of the effects of recovery from acute fatiguing exercise (Gleeson et al., 1997). All instructions to participants were given by means of standardized written instruction cards. Participants were not given any information about the results until they completed all test procedures. The same test administrator performed all testing.

Following the habituation to procedures, each participant completed three assessment sessions at the same time of day ( $\pm 1$  h) and separated by 3 days. Within each session, participants performed a standardised warm-up (General warm-up: 5 minutes of cycle ergometry work at an exercise intensity of 90 Watts. Specific warm-up: while fixed to dynamometer, 3 x 50%, 2 x 75%, 1 x 95% maximal voluntary static muscle activations of knee flexors) and the following experimental conditions: (i) a control task (CON) of equivalent duration to the subsequent fatigue task consisting of no exercise; (ii) a fatigue task (FAT) that required participants to complete a series of three 30 second periods of intermittent static maximal voluntary actions of the knee flexors of the preferred limb using a purpose-built dynamometer (modified from Gleeson *et al.*, 1995).

In each condition, 0 s (pre), 30 s (post 1), 70 s (post 2), and 110 s (post 3) estimates of peak force (PF), electromechanical delay (EMD; m. biceps femoris; sampling frequency 4 kHz; mean score derived from 2 discontinuous efforts) and rate of force development (RFD) were evaluated during prone maximal voluntary muscle actions of the knee flexors (knee

flexion angle 0.44 rad) (please see figure 4.1). In addition, a contemporaneous assessment of neuromuscular control (NCA1, NCA3 or RJA) was undertaken alongside the estimates of neuromuscular performance. In the latter, participants were required to reproduce as accurately and as rapidly as possible the peak of the previously learned 'blind' target performance (force or angle) in a single discrete episode of muscle activation, followed by full muscle relaxation. A further trial was completed. Participants were then required to record their self-perceived precision of performance.

For any given performance trial associated with the objective and self-perceived assessments of neuromuscular control (NCA1, NCA3 and RJA), error in performance was computed using the expression:  $\text{error} = ((\text{observed performance score} - \text{target performance score}) / \text{target performance score}) \cdot 100\%$ . Serial scores of self-perceived performance capability associated with NCA1, NCA3 and RJA were subjected to the same computation procedures to acquire constant and variable error scores as the objectively measured equivalents. Verbal encouragement was given throughout testing.

*Indices of peak force (PF), electromechanical delay (EMD) and rate of force development (RFD).*

Electromyograms were recorded with bi-polar surface electrodes (self-adhesive, silver-silver chloride, 10mm diameter, inter-electrode distance 40mm centre to centre) applied to the preferred leg following standard skin preparation. The standard skin preparation entailed shaving the concerned area with a razor, abrading the skin with sandpaper and



then applying an alcohol swab to remove dead skin, to minimize the resistance between the skin and the electrode (inter-electrode impedance  $<5000 \Omega$ ). Electrodes were placed longitudinally distal to the belly of the biceps femoris muscle on the line between ischial tuberosity and lateral epicondyle of the femur.

Peak force (PF), electromechanical delay (EMD) and rate of force development (RFD) responses were assessed by recording the electromyographic activity (EMG) of the biceps femoris muscle of the dominant leg (kick leg) during static maximal voluntary contraction of the knee flexors in prone position. The biceps femoris muscle was chosen for investigation because of its role in restraining anterior- femoral displacement of the knee joint and restraint of the lateral rotation of the femur relative to the tibia, both of which are implicated in the disruption of the ACL integrity (Rees, 1994; Gleeson et al., 1998). The index of peak force was used as a marker of strength performance pre and post experimental intervention and it can provide an important assessment of the extent of fatigue. Similarly, EMD and RFD are markers of the rate of muscle activation can be initiated by tension development, which gives further insight into the neuromuscular and musculoskeletal performance of a joint system (Gleeson et al., 1998).

The lower leg was supported at a position 10-15 cm proximal to the lateral malleolus by a rigid adjustable system. The lever length between the ankle cuff and the lever arm axis of rotation was standardized for each participant during the three days of testing. To limit the movement to the proper muscle groups, participants were firmly strapped by inflexible strapping across the hip, with an additional restraint applied to the thigh close to the

involved joint. The axis of rotation of the dynamometer was aligned midway between the lateral condyle of the tibia and the lateral epicondyle of the femur consistent with the anatomical axis of the knee joint. The included a load cell interfaced to the recording system (computer system) to provide related measurement of muscle force and EMG activity. The EMG signals were captured by the acquisition system (Cambridge Electronic Design Ltd., UK; Sampling Frequency 4KHZ). The reference electrode was positioned over the lateral femoral epicondyle.

The EMGs were archived to hard disk and then analysed by means of software (Spike 2, version, 3.13, Cambridge Electronic Design Ltd., U.K) to identify the EMD, defined as the time lag between the onset of muscle activity (in this case related to biceps femoris muscle) to tension development of force (Zhou et al., 1996) (defined as 0.9 N (based on 0.1% of PF), this was selected to exceed technical error [95% confidence limits]). This criterion was consistently used over all the participants. The index of PF was calculated as the highest score in the one maximum muscle contraction. The index of EMD was calculated from one maximum muscle contraction. The index of RDF was calculated as the average rate of force change between 25% PF and 75% PF associated with one maximum muscle contraction.

#### *Gravity moment correction*

Automatic compensation procedures for gravitational error in recorded forces during maximal voluntary muscle actions in the vertical plane were undertaken just prior to testing. Angle-specific force data generated by the effect of gravity acting on the mass of



the involved leg of each participant and the mass of the input accessories (including the lever arm) was utilized by dynamometer software to correct the effects of gravity over the complete range of movement.

### *Statistical analysis*

The selected indices of neuromuscular control and performance were described using standard statistical procedures (mean  $\pm$  SD). Separate factorial ANOVAs (condition [CON; FAT] by time [pre; post 1; post 2; post 3]) with repeated measures were used to assess differences in objective and self-perceived constant error (CE%; SPCE%) and variable error (VE%; SPVE%) scores for each assessment of neuromuscular control (NCA1, NCA3, RJA). Objective and self-perceived performance scores for assessments of neuromuscular control were analysed separately.

Further separate factorial ANOVAs (condition [CON; FAT] by time [pre; post 1; post 2; post 3]) with repeated measures were used to assess differences in neuromuscular performance indices of peak force (PF), electromechanical delay (EMD) and rate of force development (RFD) using scores for each inter-day assessment.

For all comparisons the assumption of sphericity was tested by the Mauchly test of sphericity. Any violations of this assumption were corrected using the Greenhouse-Geisser adjustment to raise the critical value of F, as indicated by Stevens (1996). Performance indices across assessment occasions were compared using *post hoc* Tukey (*HSD*) tests where appropriate. An a priori alpha level of 0.05 was applied in all statistical analysis in

this study. The experimental design was expected to offer an approximate 0.70 - 0.80 power to avoid *Type-II* error. All statistical analysis was programmed using SPSS (V9.0).



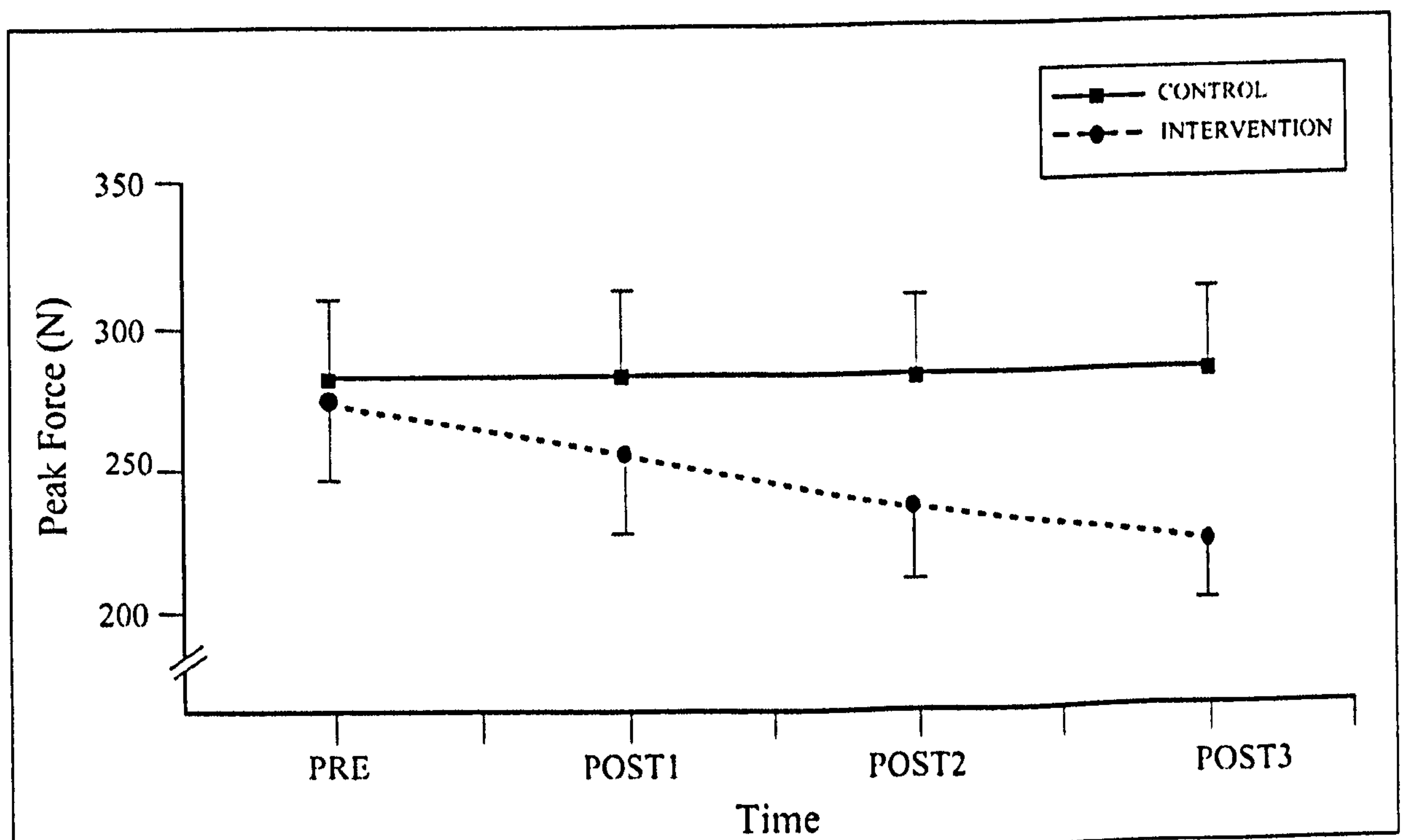
### 4.3 Results

#### *Indices of neuromuscular performance assessment.*

#### *Peak force.*

The mean and standard deviation (mean  $\pm$  SD) of PF associated with assessments NCA1, NCA3 and RJA are shown in tables 4.1, 4.2 and 4.3, respectively. The ANOVAs of peak force scores for NCA1, NCA3 and RJA revealed significant condition by time interactions ( $F_{[3, 33]} > 4.3, 10.7; p < 0.05$ ), respectively. The results in general indicated that while strength performance associated with the control condition was preserved, performance during the fatigue condition was reduced by 25.7 % compared with baseline scores.

Figure 4.2. The effects of serial acute fatiguing exercise protocol on the volitional peak force performance (PF) of the knee flexors (group mean  $\pm$  SD).



### *Electromechanical delay.*

The mean and standard deviation (mean  $\pm$  SD) of EMD associated with assessments NCA1, NCA3 and RJA are shown in tables 4.1, 4.2 and 4.3, respectively. The ANOVAs of EMD scores for NCA1 and NCA3 revealed significant condition by time interactions ( $F_{[3, 33]} > 4.3, 10.7; p < 0.05$ ), respectively. The results in general indicated that while strength performance associated with the control condition was preserved, performance during the fatigue condition was reduced by more than 20% compared with baseline scores (i.e. an increase in EMD times). However, the corresponding analysis associated with RJA did not show this interaction.

### *Rate of force development.*

The mean and standard deviation (mean  $\pm$  SD) of RFD associated with assessments NCA1, NCA3 and RJA are shown in tables 4.1, 4.2 and 4.3, respectively. The ANOVAs of RFD scores for NCA1 and NCA3 revealed no significant condition by time interactions ( $p > 0.05$ ), respectively. The results showed that RFD was not influenced by the fatigue task intervention.

### *Indices of objective neuromuscular control associated with assessments NCA1, NCA3 and RJA.*

The mean and standard deviation (mean  $\pm$  SD) of objectively measured constant error (CE %) and variable error (VE %) associated with assessments NCA1, NCA3 and RJA are shown in tables 4.1, 4.2 and 4.3, respectively. The ANOVAs of CE% scores for NCA1, NCA3 and RJA revealed no significant condition by time interactions ( $p > 0.05$ ), respectively. The results showed that CE% was not influenced significantly by the fatigue task intervention and that capability in this



aspect of neuromuscular control was preserved in response to this exercise stress. Objective overall mean scores for CE% of 1.34% ( $\pm 6.6\%$ ) and 3.6% ( $\pm 5.3\%$ ) associated with NCA1 and RJA suggested that in these tests of neuromuscular control, participants produced greater force than was required in general and that they underestimated their performance capability. In contrast, an objective overall mean score for CE% of -6.6% ( $\pm 6.2\%$ ) associated with NCA3 suggested that participants produced less force than was required and that they overestimated their performance capability during this test.

*Indices of self-perceived neuromuscular control associated with assessments NCA1, NCA3 and RJA.*

The mean and standard deviation (mean  $\pm$  SD) of constant error (SPCE%) and variable error (SPVE%) of self-perceived performance associated with assessments NCA1, NCA3 and RJA are shown in tables 4.4, 4.5 and 4.6, respectively. The ANOVAs of SPCE% scores for NCA1, NCA3 and RJA revealed no significant condition by time interactions ( $p > 0.05$ ), respectively. The results showed that SPCE% was not influenced significantly by the fatigue task intervention and that capability in this aspect of neuromuscular control was preserved in response to this exercise stress. Self-perceived overall mean scores for SPCE% of 1.3% ( $\pm 1.6\%$ ), 0.7% ( $\pm 1.5\%$ ) and 5.1% ( $\pm 7.2\%$ ) associated with NCA1, NCA3 and RJA suggested that in these tests of neuromuscular control, participants perceived consistently that they had produced greater force than had been required to match the target force/angle.

Similarly, the results showed that SPVE% for each assessment of neuromuscular control (NCA1, NCA3) was not influenced significantly by the fatigue task intervention and that capability in this aspect of neuromuscular control was also preserved in response to this exercise stress. Self-perceived overall mean scores for SPCE% of 0.7% ( $\pm 0.7\%$ ) and 0.6% ( $\pm 0.8\%$ ) associated with NCA1 and NCA3 suggested that in these tests of neuromuscular control, participants' self-perception of performance showed a similar level of consistency.

Table 4.1. Group mean (mean  $\pm$  SD) constant error (CE %), variable error (VE %) expressed as a percentage of target (50% of peak force), and peak force (PF), electrical mechanical delay (EMD) and rate of force development (RFD) associated with neuromuscular control assessment NCA1, n =12.

Experimental Conditions								
Control					Fatigue			
Variable	pre	post1	post2	post3	pre	post1	post2	post3
CE (%)	6.25 $\pm$ 8.8	2.18 $\pm$ 8.8	2.25 $\pm$ 7.5	1.34 $\pm$ 6.9	1.34 $\pm$ 6.9	2.25 $\pm$ 6.2	-3.31 $\pm$ 5.9	-1.02 $\pm$ 9.5
VE (%)	5.29 $\pm$ 5.2	4.17 $\pm$ 2.85	5.48 $\pm$ 3.92	4.50 $\pm$ 3.21	4.50 $\pm$ 3.21	6.44 $\pm$ 2.7	5.81 $\pm$ 4.5	3.97 $\pm$ 4.8
PF (N)	273 $\pm$ 72	275 $\pm$ 68	276 $\pm$ 69	272 $\pm$ 70	272 $\pm$ 70	257 $\pm$ 47	231 $\pm$ 44	226 $\pm$ 49
EMD (ms)	46 $\pm$ 91	45 $\pm$ 13	44 $\pm$ 11	43 $\pm$ 12	43 $\pm$ 12	55 $\pm$ 23	54 $\pm$ 24	55 $\pm$ 13
RFD (N)	2430 $\pm$ 1283	2662 $\pm$ 1223	2992 $\pm$ 1337	2896 $\pm$ 1399	2896 $\pm$ 1399	2456 $\pm$ 1175	2226 $\pm$ 962	2346 $\pm$ 1411

Notes: negative values indicate score that participant underestimate the target force.



Table 4. 2 Group mean (mean  $\pm$  SD) constant error (CE %), variable error (VE %) expressed as a percentage of target (50% of peak force), and peak force (PF), electrical mechanical delay (EMD) and rate of force development (RFD) associated with neuromuscular control assessment NCA3, n =12.

Experimental Conditions								
Control					Fatigue			
Variable	pre	post1	post2	post3	pre	post1	post2	post3
CE (%)	-6.12 $\pm$ 6.1	-5.75 $\pm$ 5.4	-3.80 $\pm$ 7.4	-4.95 $\pm$ 5.0	-4.95 $\pm$ 5.0	-6.42 $\pm$ 5.3	-9.28 $\pm$ 7.3	-11.35 $\pm$ 9.7
VE (%)	2.88 $\pm$ 1.8	1.72 $\pm$ 1.6	3.80 $\pm$ 3.3	3.19 $\pm$ 2.2	3.19 $\pm$ 2.2	2.67 $\pm$ 2.4	2.55 $\pm$ 2.6	3.50 $\pm$ 2.4
PF (N)	271 $\pm$ 78	273 $\pm$ 83	279 $\pm$ 79	281 $\pm$ 79	281 $\pm$ 79	281 $\pm$ 79	281 $\pm$ 79	207 $\pm$ 25
EMD(ms)	46 $\pm$ 13	45 $\pm$ 13	45 $\pm$ 14	45 $\pm$ 12	45 $\pm$ 12	49 $\pm$ 17	58 $\pm$ 25	57 $\pm$ 12
RDF(N)	2576 $\pm$ 1099	4483 $\pm$ 5053	3038 $\pm$ 1334	2883 $\pm$ 1385	2883 $\pm$ 1385	2424 $\pm$ 1129	2005 $\pm$ 1259	1968 $\pm$ 1134

Notes: negative values indicate score that participant underestimate the target force.

Table 4.3. Group mean (mean  $\pm$  SD) constant error (CE %), variable error (VE %) expressed as a percentage of target (50% of peak force), and peak force (PF), electrical mechanical delay (EMD) and rate of force development (RFD) associated with neuromuscular control assessment RJA, n =12.

Experimental Conditions								
Control					Fatigue			
Variable	pre	post1	post2	post3	pre	post1	post2	post3
CE (%)	6.34 $\pm$ 5.2	5.28 $\pm$ 4.1	1.67 $\pm$ 7.6	3.06 $\pm$ 4.4	3.06 $\pm$ 4.4	4.72 $\pm$ 8.8	1.94 $\pm$ 7.9	2.78 $\pm$ 6.48
PF (N)	284 $\pm$ 79	266 $\pm$ 70	276 $\pm$ 78	274 $\pm$ 76	274 $\pm$ 76	237 $\pm$ 55	240 $\pm$ 53	228 $\pm$ 65
EMD (ms)	43 $\pm$ 11	50 $\pm$ 15	45 $\pm$ 16	40 $\pm$ 17	40 $\pm$ 17	53 $\pm$ 18	56 $\pm$ 19	46 $\pm$ 16
RDF (N)	2794 $\pm$ 1476	2642 $\pm$ 1208	2829 $\pm$ 1219	2846 $\pm$ 1209	284 $\pm$ 1209	2444 $\pm$ 1091	2342 $\pm$ 957	2217 $\pm$ 1001

Notes: negative values indicate score that participant underestimate the target force.

Table 4.4. Group mean (mean  $\pm$  SD) constant error (SPCE %) and variable error (SPVE %) associated with self-perceived performance during neuromuscular control assessment NCA1, n =12.

Experimental Conditions								
Control					Fatigue			
Variable	pre	post1	post2	post3	pre	post1	post2	post3
SPCE (%)	0.75 $\pm$ 2.1	1.58 $\pm$ 1.4	2.25 $\pm$ 7.5	0.66 $\pm$ 0.7	0.66 $\pm$ 0.7	1.96 $\pm$ 1.4	1.58 $\pm$ 1.1	1.12 $\pm$ 1.6
SPVE (%)	0.79 $\pm$ 0.5	0.63 $\pm$ 0.37	0.98 $\pm$ 0.6	1.12 $\pm$ 0.7	1.12 $\pm$ 0.7	0.71 $\pm$ 0.5	0.87 $\pm$ 1.1	0.52 $\pm$ 0.7

Notes: negative values indicate score that participant underestimate the target force.

Table 4. 5. Group mean (mean  $\pm$  SD) constant error (SPCE %) and variable error (SPVE %) associated with self-perceived performance during neuromuscular control assessment NCA3, n =12.

Experimental Conditions								
Control					Fatigue			
Variable	pre	post1	post2	post3	pre	post1	post2	post3
SPCE (%)	0.41 $\pm$ 1.4	0.88 $\pm$ 1.0	0.63 $\pm$ 1.4	0.32 $\pm$ 1.5	0.32 $\pm$ 1.5	0.69 $\pm$ 1.9	1.07 $\pm$ 1.5	0.20 $\pm$ 1.8
SPVE (%)	0.56 $\pm$ 0.5	0.58 $\pm$ 0.6	0.68 $\pm$ 0.4	0.39 $\pm$ 0.4	0.39 $\pm$ 0.4	0.74 $\pm$ 0.9	0.70 $\pm$ 0.9	0.72 $\pm$ 0.6

Notes: negative values indicate score that participant underestimate the target force.



Table 4.6. Group mean (mean  $\pm$  SD) constant error (SPCE %) associated with self-perceived performance during neuromuscular control assessment RJA, n =12.

Experimental Conditions								
Control					Fatigue			
Variable	pre	post1	post2	post3	pre	post1	post2	post3
SPCE (%)	4.4 $\pm$ 9.5	5.56 $\pm$ 8.3	3.33 $\pm$ 5.1	8.06 $\pm$ 5.4	8.06 $\pm$ 5.4	4.12 $\pm$ 8.4	2.50 $\pm$ 9.1	4.72 $\pm$ 6.4

#### 4.4 Discussion

The present study attempted to assess the effect of acute serial fatiguing on neuromuscular control and performance that may disrupt dynamic stabilization around the knee joint. Some of the muscles concerned in stabilising the knee joint are the knee flexors and extensors (Swanik et al., 1997). However it has also been thought that an unfavourable interaction between the activations of the knee extensors and flexors may threaten the integrity of the ACL since an aggressive activation of the extensors can initiate an anterior shear force (Grood et al., 1984; Arms et al., 1984).

#### *Neuromuscular performance*

The present study found that the fatigue intervention elicited substantial reductions in aspects of neuromuscular performance. For example, there was a group mean average 25.7% reduction in the volitional force generating capacity of the neuromuscular system compared to baseline. These findings of a reduction in the force generating capacity of neuromuscular performance, following serial bouts of fatigue static exercise are consistent with previous reports in the literature. For

example peak force was reduced by 20-30% of initial peak force (James et al., 1995), by 20 to 60% of the baseline strength of the knee extensors and flexors during concentric actions (Gleeson et al., 1995) and in a more recent study, by 18.5% compared to baseline (Avelo et al., 2001) following laboratory-based fatigue interventions. A similar reduction of force has been shown following sustained isometric contractions (Fellows et al. 1993; Woods et al., 1987), and after exercise interventions involving stretch-shortening cycles and long-lasting low-intensity efforts (Avela et al., 1999a; Nicol et al., 1991).

A number of reasons have been suggested to explain the decline of strength associated with muscle fatiguing exercise: (1) many studies demonstrated that fatiguing exercise may cause damage to the muscle fibres, particularly disruption to the myofibrillar-banding pattern (Friden et al., 1983; Newham et al., 1983). (2) increased stiffness following exercise induced muscle fatigue is one of the major factors contributing to reduce of peak force (Armiger, 2000). (3) formation of swelling in muscle tissues and pain may contribute to the reduction of peak force after exercise induced muscle fatigue (Jones et al., 1987; Smith, 1991).

Further evidence that the fatigue intervention had compromised neuromuscular performance was shown by an increase in group mean EMD scores of some 19.3% compared to baseline. This result is comparable to those from other research which showed that EMD decreased by between 19% and 60% following various fatigue intervention protocols (Zhou et al., 1996; Gleeson et al. 1998; Mercer et al., 1998).



The fatigue-related reduction of force generating capacity with concomitant compromised EMD in the knee flexors at 30 degrees of knee flexion may be adequate to threaten knee stability during a critical phase of play within competitive match or training activities. It also appears to correlate with an emerging pattern of injury occurrence (Gleeson et al., 1998).

While such decrements to the capacity for neuromuscular performance associated with fatigue would be expected to reduce the capability of stabilising a synovial joint system and contribute to an increased risk of musculoskeletal injury, this risk may be amplified substantially should there be concomitant fatigue-related disruption to the capability for neuromuscular control.

### *Neuromuscular control*

A major finding from the current study was that irrespective of the method of assessment of neuromuscular control (i.e. NCA1, NCA3 or RJA), it was not influenced significantly by the fatigue task intervention and that capability in this aspect of neuromuscular control was preserved in response to this type of exercise stress. This finding may have important implications for appreciating mechanisms by which the body is protected in adverse and stressful conditions. For example, despite fatigue-related strength and muscle activation impairments, the risk of injury may be reduced if the athlete was still able to maintain neuromuscular control and sustain a proper technique, such as landing from a jumping manoeuvre properly with favourable mechanical advantage on the balls of his feet with the knee flexed and the chest over the knees (Boden et al., 2000).

While it could be argued that a more potent fatigue intervention might exceed this apparent capability of the neuromuscular system to compensate for exercise stress, the level of decrement to neuromuscular performance in this study was in fact, able to mimic those associated with ecologically-valid threats (Gleeson et al., 1998). Thus it is plausible that this finding illustrates an important principle that the 'control' system may be preserved in preference to the physiological machinery for performance when challenged by exercise stresses. Enoka et al., (1984) suggested that this type of acute adaptation may be due the fact that the body system has tried to improve its ability to cope with the ecological demands placed on it by means of potentiation. It may be that various mechanisms contribute collectively and interactively to increase both the electrical and mechanical output capabilities of the sensory-motor systems above the usual resting values. Examples of these capabilities include monosynaptic responses, miniature end-plate potentials, m-wave amplitudes and post-tetanic potentiation (Enoka, 1994).

### *Self-perception of performance*

As intimated earlier, no research has investigated whether or not an individual's self-perception of neuromuscular control is affected by serial fatiguing episodes of exercise. It was interesting to note that alongside the preservation of objectively measured neuromuscular control, the results showed that self-perceived performance as indicated by constant error (SPCE%) for NCA1, NCA3 and RJA was not influenced significantly by the fatigue task intervention and that capability in this aspect of neuromuscular control was similarly preserved in response to this exercise stress.



In general, whether fatigued or not, participants appeared to perceive consistently that they had produced greater force than had been required to match the target force or angle. The latter finding was consistent with the corresponding objectively measured performance for NCA1 and RJA in particular. Again this finding may be important because it could be hypothesised that any imprecision of self-perception may contribute to a process by which injury becomes more likely. For example, self-perceived overestimation of performance capability relative to true performance capability may leave the participant partially unprotected during an episode of externally imposed mechanical stressors. Indeed, this scenario may provoke a willingness to choose actions in competitive match-play that may have inherent extra risk when in conjunction with fatigue-related reduced neuromuscular performance capabilities.

An overshooting of a target under conditions of exercise stress may be a mechanism of protection. For example, Burke et al. (1976) proposed that moderate to high intensity muscle contractions may result in excitatory response in spinal segmental proprioceptive circuits, resulting in an involuntary enhancement of sub-maximal muscle tension to following motoneuronal activation. Therefore, in conditions of sub-maximal voluntary muscle contraction, the sensory discharge may well contribute to a potential error (overshooting) in the regulation of force, mainly in the absence of visual feedback of the force being produced.

Although the this study showed similar responses to fatigue-related exercise stresses amongst the selected indices of neuromuscular control (NCA1, NCA3 and RJA), it was interesting to note that retrospective correlational analyses (Pearson product-

moment) showed no significant relationships amongst these indices either prior to or after fatigue ( $p > 0.05$ ). This finding suggests that each protocol had been assessing different aspects of neuromuscular control commensurate with its design characteristics.

### *Summary*

In summary, this study assessed the effect of acute serial fatiguing tasks on objective and self-perceived neuromuscular control and performance. Results showed that despite substantial fatigue-related strength and muscle activation impairments, neuromuscular control as measured objectively by NCA1, NCA3 or RJA was not influenced significantly by the fatigue task intervention and that capability in this aspect of neuromuscular control was preserved in response to this type of exercise stress. Furthermore, constant error associated with self-perceived performance was similarly not influenced significantly by the fatigue task intervention. It is plausible that in order to provide optimum protection of synovial joints, neuromuscular control of the knee flexors may be preserved preferentially to neuromuscular performance when challenged by fatigue-related exercise stresses.

In order to investigate further critical levels of threat to the stability of the joint system, the next study will focus on unaccustomed high intensity exercise in which skeletal muscle is susceptible to ultrastructural damage (Armstrong et al., 1983; Jones et al., 1989; Byrne and Eston, 1998) and which may be experienced by the sports performer who recommences training, match play or even rehabilitation following a prolonged period of inactivity or injury. The effects of muscle damage on the susceptibility to further injury remains relatively unknown but it may be



hypothesised that the protective capability offered by neuromuscular control and performance may also be compromised by a relatively greater exercise stress compared to fatigue alone.

INTERVENTIONS:

## CHAPTER 5

THE EFFECTS OF EXERCISE-  
INDUCED MUSCLE DAMAGE  
AND FATIGUE ON  
NEUROMUSCULAR CONTROL  
AND PERFORMANCE OF THE  
KNEE FLEXORS IN MEN.



## **5.0 The effects of exercise-induced muscle damage and fatigue on neuromuscular control and performance of the knee flexors in men.**

### **5.1 Introduction**

The previous study considered the acute, transient and metabolically-focused effects of fatigue on neuromuscular control and performance. However, neuromuscular control during a more prolonged, mechanical type of disruption to the muscle i.e. during conditions of exercise induced muscle damage has received only limited scrutiny to date and has only focused on the upper limb (Saxton, et al., 1995; Brockett, et al., 1997).

It is well accepted that muscle damage occurs with unaccustomed exercise, primarily involving eccentric contractions (Clarkson et al., 1992; Balnave and Thompson, 1993; Morgan and Allen, 1999; McHugh, 2000; Behm et al 2001) and that this type of exercise stress frequently results in symptoms of delayed onset muscle soreness (DOMS). Exercise induced muscle damage (EIMD) is usually described by means of symptoms of strength loss, pain with activity, muscle tenderness and elevated muscle enzyme activity (McHugh et al., 1999; Warren et al., 1999).

The peak response of these symptoms would be expected to occur between four and six days following the muscle-damaging episode but symptoms and performance deficits may persist for longer periods (Byrne and Eston, 1998; Clarkson et al., 1992; Rinard et al., 2000; Nokasa et al., 2001), requiring several days to recovery fully after strenuous eccentric exercise interventions.

The majority of previous research into EIMD in the lower limb has restricted its focus to neuromuscular performance indices associated with strength (Byrne and Eston, 1998; McHugh et al. 1999) and only recently volitional EMD in the knee extensor (quadriceps) musculature (Williams et al. 2000)

There is only limited knowledge of how neuromuscular performance and control is moderated while the lower limb musculature is experiencing conditions of EIMD. Moreover, the sports performer who recommences training, match play or even rehabilitation following a prolonged period of inactivity or injury is faced frequently by the need to undertake unaccustomed high intensity exercise in which skeletal muscle is susceptible to ultrastructural damage (Armstrong et al., 1983 ; Jones et al., 1989; Byrne and Eston, 1998), together with the likelihood of the occurrence of concomitant fatigue. As alluded to earlier, the effects of muscle damage alone or concomitant effects of muscle damage and fatigue, on the susceptibility to further injury remains relatively unknown. However, it may be hypothesised that the protective capability offered by neuromuscular control and performance may also be compromised by relatively greater exercise stress compared to fatigue alone.

In particular, the potential for disruption to the pattern of type II motor fibre recruitment (MacPherson et al., 1996; McHugh et al., 2000) may be greater during conditions of EIMD and could be reflected in an alteration to the extent of performance (constant error) and consistency of performance (variable error) during the NCA1 task that requires a very rapid and time-orientated pattern of force delivery. Neuromuscular control may be an important factor in the preservation of joint integrity during muscle damage and disruption to the capacity for muscle



spindle feedback. Muscle spindles play a key role in movement regulation throughout the stretch reflex (Pattern et al., 1989), provide information about limb position and velocity (Sittig, 1985) and the loading of the knee joint (Laskowski et al., 2000).

It is plausible that in a sports tournament situation, repeated doses of exercise stress may cause micro-damage of the muscle fibres which may progress to more major muscle tears of the involved muscle groups (Proske and Morgan, 2001). The efficiency of neuromuscular control may be compromised if the effect of damage in conjunction with fatigue is detrimental to joint awareness and the neuromuscular control of the involved joints. Similarly, it may be that such potential disruptions to the biological machinery for sensory-motor performance may influence detrimentally objective performance on NCA3-type tasks. The latter involve single, self-paced muscular efforts of a long enough duration of time to be moderated by sensory feedback and reflective of self-regulated force and movements in rehabilitative conditioning. Furthermore, any potential disruptions to the biological machinery for sensory-motor performance may elicit compromised self-perception of the capability for neuromuscular control. Imprecision of self-perception may contribute to a process by which injury becomes more likely. For example, overestimation of performance capability may leave the participant partially unprotected during an episode of externally imposed mechanical stress.

The aim of this study was to examine the concomitant effects of exercise-induced muscle damage and fatigue on objective and self-perceived indices of neuromuscular control and performance of the knee flexors in males.



## 5.2 Methods

### *Participants*

Twenty-six adult males who were selected from the student population at the University of Wales, Bangor, gave written consent to participate in this study which had received approval from the University's ethics committee. Participants were allocated randomly to experimental (n = 14; age:  $25.8 \pm 5.8$  years; height:  $1.78 \pm 0.05$  m; body mass:  $74.9 \pm 11.7$  kg [mean  $\pm$  SD]) and control (n = 12; age:  $25.6 \pm 5.6$  years; height:  $1.73 \pm 0.06$  cm); body mass ( $70.7 \pm 11.4$  kg) [mean  $\pm$  SD]) groups. Participants were healthy and physically active but had not participated in any resistance training of the lower limb during the last 6 months prior to the testing.

### *Experimental procedures.*

Following habituation procedures, participants completed a standardised warm-up of five minutes cycle ergometry at an exercise intensity of 90 W/ time and a further five minutes of static stretching of the involved musculature. Participants were then secured in the appropriate position on a custom-built dynamometer to undergo a specific warm-up consisting of 3 x 50%, 2 x 75%, 1 x 95% maximal voluntary static muscle activations of knee flexors. All instructions to participants were given by means of standardized written instruction cards. Participants were not given any information about the results until they completed all test procedures. The same test administrator performed all testing.

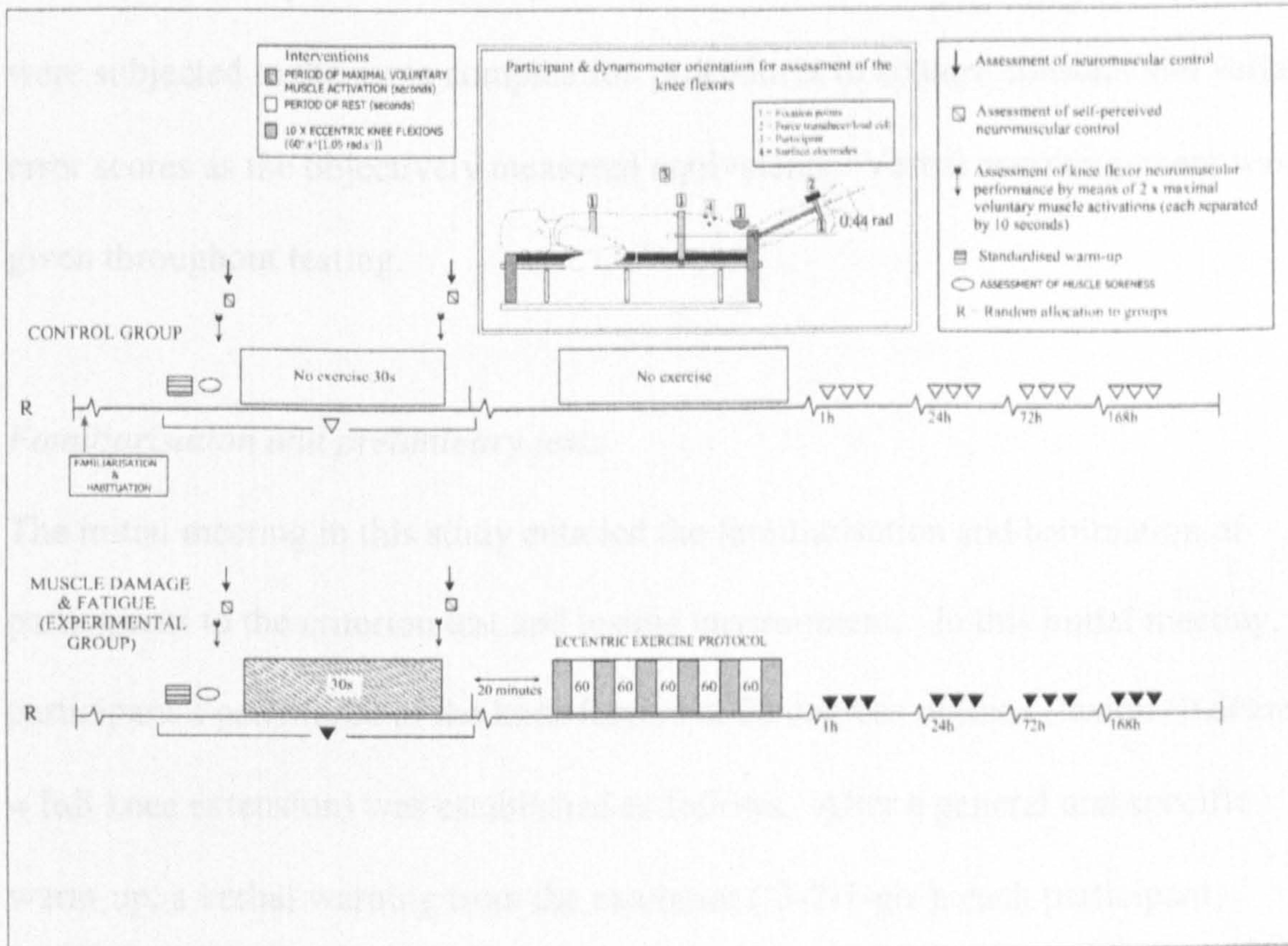
The experimental design comprised two independent treatment conditions undertaken by the two separate groups of participants: (i) a concomitant exercise-induced muscle damage and fatigue condition (EIMD-FAT) in which a fatigue task involving static maximal voluntary muscle activation of the knee flexors of the

preferred leg, was performed prior to (pre) and at 1 h, 24 h, 72 h and 168 h following a single EIMD intervention protocol. The static fatiguing exercise task consisted of 30 seconds sustained maximal activation of the musculature. The EIMD intervention protocol consisted of a warm-up involving 5 sub-maximal and 5 maximal eccentric activations of the knee flexors of the preferred leg, followed by 6 sets (each separated by 1 minute) of 10 repetitions of maximal eccentric activations using an isokinetic dynamometer (Kin-Com, Chattecx, Chattanooga, USA). The system's lever-arm moved at an angular velocity of  $60^\circ \text{ s}^{-1}$  ( $1.05 \text{ rad s}^{-1}$ ) through a range of  $70^\circ$  to  $10^\circ$  of knee flexion ( $0^\circ = \text{full extension}$ ); (ii) a control condition (CON) of consisting of no exercise equivalent duration to the EIMD-FAT conditions consisting of exercise.

The CON and EIMD-FAT treatment conditions were undertaken independently to minimise the potential intrusion of (i) contralateral limb adaptation effects associated with a single-leg, repeated measures model and (ii) any potential sequencing effects (a highly extended period of recovery [wash-out] would be expected to be required to ensure total restoration of muscle function following eccentric exercise-induced damage in a ipsilateral, repeated measures design (Brown et al., 1997) and (iii) any waning participant motivation due to the volume of assessments involved.



Figure 5.1. Schematic of the protocol to assess the effects of exercise-induced muscle damage and fatigue on objective and self-perceived indices of neuromuscular control and performance of the knee flexors in males. Also depicted is participant and dynamometer orientation.



Measures of objective and self-perceived neuromuscular control and performance of the knee flexors were taken prior to and immediately following each static fatiguing exercise task (or equivalent period of rest) within the treatment conditions. The protocol is illustrated schematically in figure 5.1.

Each of the selected testing conditions for neuromuscular control (NCA1, NCA3 and DNCA) was separated by 5 minutes. NCA2, NCA3 and DNCA were presented in random order on each test occasion to offset any sequencing effects.

For any given performance trial associated with the objective and self-perceived assessments of neuromuscular control (NCA2, NCA3 and RJA), error in



performance was computed using the expression:  $\text{error} = ((\text{observed performance score} - \text{target performance score}) / \text{target performance score}) \cdot 100\%$ . Serial scores of self-perceived performance capability associated with NCA2, NCA3 and DNCA were subjected to the same computation procedures to acquire constant and variable error scores as the objectively measured equivalents. Verbal encouragement was given throughout testing.

### *Familiarisation and preliminary tests*

The initial meeting in this study entailed the familiarisation and habituation of participants to the criterion test and testing environment. In this initial meeting, the participant's peak force of the knee flexors at 30 degrees of knee flexion (0 degrees = full knee extension) was established as follows. After a general and specific warm-up, a verbal warning from the examiner ('3-2-1-go'), each participant attempted to flex the knee joint as forcefully and as quickly as possible against the immovable restraint offered by the apparatus. After an appropriate time of maximal muscle activity of approximately 3 seconds to identify maximum voluntary muscle activation (MVMA) force (i.e. the highest force generated by the single muscle activation), another signal was delivered to the participant to relax as rapidly as possible. A recovery period (10 s) followed. Following another practice attempt, this procedure was performed a further 2 times, separated by 10 seconds of rest using the involved musculature (i.e. the knee flexors) at a knee flexion of 30 degree. The 'blinded' target force for assessments of neuromuscular control was established as 50% of the highest recorded maximal voluntary muscle action at 30° of knee flexion. This level of force corresponds approximately to that associated with peak muscular power outputs during sporting endeavours according to the expected force-velocity



and power-velocity relationships (Hill, 1938). The force acted as the 'blind' target force for all-subsequent assessments of neuromuscular control. Potential distractions to the participant were minimised by the fact that only the administrator was present in the laboratory in addition to the participant during data capture.

Following the establishment of MVMA force and blinded target force, participants were familiarised to all assessment procedures of neuromuscular control that had been selected for inclusion in this study (NCA2, NCA3 and a dynamic neuromuscular control assessment task [DNCA]), together with protocols for the assessment of neuromuscular performance. The NCA2 and NCA3 assessments of neuromuscular control required participants to produce a muscle action eliciting a blinded target force of 50% MVMA. On achievement of the blinded target force, participants were instructed to either relax immediately after they felt the prescribed force had been attained in the case of NCA2 or to maintain the blinded target force for 2-3 seconds before relaxing to signal task completion. All assessments of neuromuscular control followed a specific procedures for learning the prescribe target force as described below. The dynamic neuromuscular control assessment task required participants to match as rapidly and as precisely as possible the position of a previously learned target knee angular position. The participant's completion of the DNCA task was indicated by the peak amplitude of knee movement prior to the joint's return to its starting position using optical encoders and a computerised system for the registration of angular position.

### *Learning of target force for NCA2 and NCA 3*

The learning of the target force that was 'blinded' to the participant, was achieved by the participant undertaking a standardised series of trials in blocks (no more than 5 trials) using the involved musculature (knee flexors) in which the aim was to match the target force as closely as possible. Participants received only contemporaneous standardised verbal feedback from the test administrator to facilitate further improvements in performance precision. In this way, participants were blinded to both the absolute level of the prescribed target force and the scale of measurement used to offer feedback. Full details of these procedures are described in chapter 2 (General methods, section 2.6). The task was deemed to have been learned once the participant was capable of producing a criterion series of ten trials in which seven scores or more showed errors that were within  $\pm 2.0$  N of the 'blinded' target force. Retention of performance was verified briefly during warm-up and familiarisation trials prior to exercise interventions.

### *Self-perceived performance (NCA2, NCA3).*

During the learning process for the 'blinded' target force, participants were routinely required to report to the test administrator their self-perception of the extent and the direction (positive and negative indicating exceeding and falling-short of the target, respectively) of error associated with individual trials. Initially, participants verbalised these self-perceived scores during preliminary trials using the same arbitrary scale of measurement without units used during the learning process. For example, feedback from the participant was required in standardised terminology such as "25 high", "20 high", "15 high", "10 high", "5 high" and "25 low", "20 low", "15 low", "10 low", "5 low", respectively, or "no error", as appropriate. Once



the participant had been familiarised with this process of reporting their self-perceived performance outcomes, they were required to transfer these reports to a non-verbal, written format in which a series of miniature visual-analogue scales (using the same unit-less scale of points in the range +25 to -25) were marked appropriately after each block of trials. Full details of these procedures are described in chapter 2 (General methods, section 2.7). Similar procedures to these for self-perceived performance during NCA2 and NCA3 assessments were adopted for the self-perception of performance during the DNCA (please see below).

*Dynamic neuromuscular control assessment task [DNCA].*

Each participant was placed in a prone position on a specially constructed dynamometer-goniometer system (adapted from Gleeson et al., 1995) with the dominant leg (kick leg) moving actively toward the target angle. The target angle was 30 degrees. This angle is associated with inherent anatomical and mechanical stresses that load ligamentous tissue and may threaten the knee and subsequently the ACL integrity (Anthony and William, 1998; Gleeson et al., 1998). The lower leg was supported 10-15 cm proximal to the lateral malleolus by an adjustable rigid system in a movement initiation position adjusted to 10° of joint flexion (0° = full knee extension). In a pilot study, participants had been unable to initiate movement from anatomical full knee extension due to the inertia inherent in the system's lever-arm. The lever length between the ankle cuff and the lever arm axis of rotation was standardized for each participant during the testing occasions. To limit the movement to the proper muscle groups, participants were firmly secured by strapping across the hip, with additional restraints applied to the upper and lower back. The axis of rotation of the dynamometer was aligned midway between the

lateral condyle of the tibia and the lateral epicondyle of the femur consistent with the anatomical axis of the knee joint. The lever-arm of the computerised dynamometer-goniometer system included high-precision optical encoders of angular position that were interfaced to a computerised data acquisition system and recording system to provide a time-history of lever-arm and associated knee joint position in relation to the specified target angle. The technical precision offered by this goniometry system was better than 0.001 degrees

### *Learning of the target angle*

The learning of the target angle that was 'blinded' to the participant, was achieved by the participant undertaking a standardised series of trials in blocks (no more than 5 trials) using the involved musculature (knee flexors) in which the aim was to match the target angle of joint flexion as precisely and as rapidly as possible. The process of habituation and learning of the target angle was similar to that used for the learning of the target force in NCA2 and NCA3. Participants received only contemporaneous standardised verbal feedback from the test administrator to facilitate further improvements in performance precision. In this way, participants were blinded to both the absolute level of the prescribed target angle and the scale of measurement used to offer feedback. Full details of this approach are described in chapter 2 (General methods, section 2.6). Trials that were within  $\pm 3.0$  degrees of the 'blinded' target angle were described as having 'no error', whereas trials that exceeded or fell below the target angle were described as '5' below', '4' high', and '6' high' for example, as appropriate.



The task was deemed to have been learned once the participant was capable of producing a criterion series of ten trials in which seven scores or more showed errors that were within  $\pm 3.0$  degrees of the 'blinded' target angle. This level of precision was deemed to reflect the likely biological variability (95% confidence limits) associated with this type of performance (Mickle et al., 2002). Retention of performance was verified briefly during warm-up and familiarisation trials prior to exercise interventions. While blinded, the participants were asked to reproduce the target angle in five discrete and brief episodes of effort (1 s) separated by five periods of muscular relaxation from the reference initial angle. Participants were then required to record their self-perceived accuracy of effort within 5 seconds.

#### *Indices of neuromuscular performance assessment*

In addition to contemporaneous assessment of neuromuscular control (NCA2, NCA3 or DNCA), estimates of peak force (PF) were evaluated during prone maximal voluntary muscle actions of the knee flexors (knee flexion angle 0.44 rad) (please see figure 5.1). More complete descriptions of the assessment procedures for neuromuscular performance assessment of the knee musculature of the dominant leg is outlined in chapter 2.

#### *Indirect markers of muscle damage*

Subjective assessments of soreness of the knee flexors of the preferred leg were obtained prior to the evaluation of performance on each assessment occasion. Participants were required to rate their soreness on a 100 cm visual-analogue scale (Gleeson et al., 2003) on stretching and active flexion of the knee of the preferred leg. The statements on the scale included 'my muscles don't feel sore at all' and 'my

muscles feel so sore that I don't want to move them', which corresponded to numerical ratings of 0 and 10, respectively.

### *Flexibility*

For each participant active flexor muscle flexibility was assessed using the 'sit and reach' test. Participants sat on the floor, extended both his knees and the feet (shoes off) were positioned flat against the 'sit and reach' test box. The participant bent forward slowly as far as possible, with the knee joints maintained in a fully extended position. No ballistic movement was allowed and maximum flexibility (i.e. maximum distance in (cm) held for two seconds) was recorded (in cm) as the furthest position of the tips of the fingers as they touched on the testing box relative to the feet.

The protocol was completed twice before the warm-up procedure on each test occasion which allowed for standardised comparisons of scores. The better of two scores was recorded for subsequent evaluation.

### *Statistical analysis*

The selected indices of neuromuscular control and performance were described using standard statistical procedures (mean  $\pm$  SD). The concomitant effects of exercise-induced muscle damage and fatigue was evaluated for each index of performance (constant error [CE%; SPCE%] and variable error [VE%; SPVE%] scores associated with objective and self-perceived performance for each assessment of neuromuscular control [NCA1, NCA3, DNCA] and neuromuscular performance index of peak force [PF] using separate two (group: CON; EIMD-FAT) by five (time: pre; 1 h; 24 h; 72 h; 168 h) mixed-model ANOVAs with repeated measures on the latter factor.



Flexibility and perceived soreness were assessed by means of separate two (condition: CON; EIMD-FAT) by five (time: pre; 1 h; 24 h; 72 h; 168 h) mixed-model ANOVAs. The cumulative effects of having undertaken repeated fatigue tasks was assessed by evaluating pre-fatigue and post-fatigue performance scores separately.

Violations to the assumptions underpinning the proper use of repeated measures ANOVA were corrected by the Greenhouse-Geisser adjustment of the critical F-value. This was indicated by the suffix *GG*. Statistical significance was accepted at  $p < 0.05$ . The experimental design offered an approximate 0.70 - 0.80 power of avoiding a Type-II error when employing a least detectable difference of 5.0% - 7.0% for CE%, SPCE% and VE%, SPVE% scores, respectively and 5.0 N for PF.

### 5.3 Results

*Indices of neuromuscular performance and indirect markers of muscle damage.*

#### *Peak force*

Group mean scores (mean  $\pm$  SD) for the index of peak force (PF) of the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to* and *after* the static fatiguing exercise task are shown in table 5.1.

Table 5.1. Group mean scores for the index of peak force (PF) of the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

Index		Time				
		Pre	1h	24h	72h	168h
PF (N) (pre-fatigue)	a	255.2 $\pm$ 69.8	224.4 $\pm$ 71.4	193.2 $\pm$ 78.9	222.7 $\pm$ 68.9	241.4 $\pm$ 63.3
	b	251.1 $\pm$ 87.8	241.7 $\pm$ 67.5	253.8 $\pm$ 88.2	242.1 $\pm$ 56.6	246.7 $\pm$ 43.7
PF (N) (post-fatigue)	a	192.3 $\pm$ 50.0	163.5 $\pm$ 59.8	145.7 $\pm$ 71.2	157.3 $\pm$ 53.6	172.3 $\pm$ 50.7
	b	175.5 $\pm$ 93.2	171.6 $\pm$ 73.2	187.0 $\pm$ 74.1	186.7 $\pm$ 72.4	200.3 $\pm$ 43.5

The ANOVAs of peak force scores at each assessment juncture prior to fatiguing exercise revealed significant group by time interactions ( $F_{[4, 96]} = 2.5$ ;  $p < 0.05$ ), respectively. The results in general indicated that while strength performance associated with the control group was preserved, performance during the concomitant EIMD-FAT condition was reduced by more than 24% (at 24 h) compared with baseline scores. Strength performance appeared to be still depressed compared to baseline at seven days after the exercise stress. A similar pattern of differential performance deficit over time between groups was noted in performance after the fatigue task ( $F_{[4, 96]} = 2.6$ ;  $p < 0.05$ ).



### *Flexibility*

Group mean scores for the index of flexibility (sit-and-reach) associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to* the static fatiguing exercise task is shown in table 5.2. Repeated measure ANOVA revealed a significant interaction between the control group and the experimental group by time ( $F_{[4, 96]} = 4.3$ ;  $p < 0.05$ ). While the flexibility in the control group remained relatively constant over the total period of time, the EIMD experimental group was most prominent in terms of flexibility at 24h following eccentric exercise ( $7.79 \pm 7.37$  cm vs.  $9.21 \pm 7.98$  cm [24 h vs. pre; table 5.2]) that also coincided with the peak force lost.

Table 5.2. Group mean scores for the index of flexibility (sit-and-reach) associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

Index		Time				
		Pre	1h	24h	72h	168h
Flexibility (cm)	a	9.21 $\pm$ 7.98	10.50 $\pm$ 7.35	7.79 $\pm$ 7.37	9.71 $\pm$ 7.50	10.50 $\pm$ 7.78
	b	11.33 $\pm$ 6.71	10.50 $\pm$ 5.55	11.00 $\pm$ 5.56	12.25 $\pm$ 5.50	13.67 $\pm$ 6.07

### *Perceived soreness*

A significant condition by time interaction ( $F_{[4, 96]} = 6.3$ ;  $p < 0.001$ ) showed that while perceived soreness levels remained constant for the control group(CON), the intervention group and concomitant EIMD-FAT condition was associated with an increase in group mean values. This increase was most prominent at 24 h during the

concomitant EIMD-FAT exercise intervention ( $7.29 \pm 1.49$  vs.  $0.3 \pm 0.20$  [24 h vs. pre; table 5.3]).

Table 5.3. Group mean scores for the index of perceived pain associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

Index		Time				
		Pre	1h	24h	72h	168h
Pain (no units)	a	$0.3 \pm 0.20$	$2.50 \pm 0.52$	$7.29 \pm 1.49$	$4.00 \pm 1.47$	$1.86 \pm 0.77$
	b	$0.2 \pm 0.20$	$1.33 \pm 0.49$	$1.33 \pm 0.49$	$1.33 \pm 0.49$	$1.42 \pm 0.51$

*Indices of objectively measured neuromuscular control associated with assessments NCA2, NCA3 and DNCA.*

The mean and standard deviation (mean  $\pm$  SD) of objectively measured constant error (CE%) and variable error (VE%) associated with assessments NCA1, NCA3 and DNCA are shown in tables 5.4, 5.5 and 5.6, and 5.7, 5.8 and 5.9, respectively.



Table 5.4. Group mean scores for the index of objectively measured constant error (CE%) for assessment NCA1 in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

		Time				
Index [NCA1]		Pre	1h	24h	72h	168h
CE (%) (pre-fatigue)	a	3.03 $\pm$ 7.8	0.64 $\pm$ 6.4	2.80 $\pm$ 5.8	2.71 $\pm$ 7.27	3.54 $\pm$ 5.6
	b	6.31 $\pm$ 9.2	3.16 $\pm$ 8.7	3.84 $\pm$ 9.5	2.54 $\pm$ 3.01	2.89 $\pm$ 4.4
CE (%) (post-fatigue)	a	3.97 $\pm$ 8.6	-1.00 $\pm$ 9.3	-1.60 $\pm$ 6.3	1.82 $\pm$ 9.3	-2.92 $\pm$ 5.7
	b	3.44 $\pm$ 7.7	1.90 $\pm$ 8.2	-0.83 $\pm$ 9.5	1.93 $\pm$ 6.3	5.49 $\pm$ 8.1

Table 5.5. Group mean scores for the index of objectively measured constant error (CE%) for assessment NCA3 in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

		Time				
Index [NCA3]		Pre	1h	24h	72h	168h
CE (%) (pre-fatigue)	a	-5.9 $\pm$ 12.0	-9.9 $\pm$ 10.5	-10.2 $\pm$ 8.8	-8.7 $\pm$ 9.6	-9.7 $\pm$ 6.9
	b	-7.0 $\pm$ 11.0	-8.7 $\pm$ 5.7	-8.7 $\pm$ 10.4	-7.9 $\pm$ 8.6	-4.0 $\pm$ 5.2
CE (%) (post-fatigue)	a	-11.3 $\pm$ 11.7	-11.4 $\pm$ 12.0	-14.7 $\pm$ 10.1	-13.8 $\pm$ 12.2	-13.8 $\pm$ 10.6
	b	-8.5 $\pm$ 8.8	-5.3 $\pm$ 11.7	-13.2 $\pm$ 6.7	-10.9 $\pm$ 8.6	-9.0 $\pm$ 6.8

Table 5.6. Group mean scores for the index of objectively measured constant error (CE%) for assessment DNCA in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

Index [DNCA]		Time				
		Pre	1h	24h	72h	168h
CE (%) (pre-fatigue)	a	3.6 $\pm$ 4.7	1.9 $\pm$ 3.4	2.9 $\pm$ 5.4	-0.81 $\pm$ 4.2	0.8 $\pm$ 4.6
	b	3.2 $\pm$ 4.9	4.0 $\pm$ 5.3	0.8 $\pm$ 2.7	3.15 $\pm$ 4.4	2.2 $\pm$ 3.2
CE (%) (post-fatigue)	a	2.9 $\pm$ 5.6	3.0 $\pm$ 4.9	3.7 $\pm$ 4.8	1.76 $\pm$ 6.1	2.9 $\pm$ 3.3
	b	2.7 $\pm$ 4.5	4.4 $\pm$ 6.1	0.4 $\pm$ 5.3	5.46 $\pm$ 5.1	5.1 $\pm$ 5.3

The ANOVAs of pre- and post-fatigue CE% scores for NCA2, NCA3 and DNCA revealed no significant group by time interactions ( $p > 0.05$ ), respectively. The results showed that CE% was not influenced significantly by the concomitant exercise-induced muscle damage and fatigue task intervention and that capability in this aspect of neuromuscular control was preserved in response to this degree of exercise stress. Objective overall mean scores for CE% of 3.0% ( $\pm 7.1\%$ ) and 1.2% ( $\pm 7.6\%$ ), and 8.1% ( $\pm 8.3\%$ ) and 11.2% ( $\pm 8.8\%$ ), for pre- and post-fatigue conditions and assessments NCA2 and DNCA, respectively, suggested that in these tests of neuromuscular control, participants produced greater force than was required in general. Corresponding objective overall mean scores for CE% of -8.1% ( $\pm 8.3\%$ ) and -11.2% ( $\pm 8.8\%$ ), for pre- and post-fatigue measurements associated with NCA3 suggested that participants produced less force than was required and that



they had overestimated their performance capability during this test. In general, objectively measured VE% which ranged between 2.1% and 8.1% across the experimental conditions for NCA2, NCA3 and DNCA, respectively, was similarly unaffected by the concomitant exercise-induced muscle damage and fatigue task ( $p > 0.05$ ).

Table 5.7. Group mean scores for the index of objectively measured variable error (VE%) for assessment NCA1 in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

Index [NCA1]		Time				
		Pre	1h	24h	72h	168h
VE (%) (pre-fatigue)	a	4.83 $\pm$ 1.9	5.94 $\pm$ 2.3	7.23 $\pm$ 2.3	6.01 $\pm$ 2.6	6.01 $\pm$ 2.2
	b	5.98 $\pm$ 1.7	6.72 $\pm$ 2.4	5.06 $\pm$ 2.00	5.56 $\pm$ 2.6	6.94 $\pm$ 1.8
VE (%) (post-fatigue)	a	7.69 $\pm$ 3.0	7.22 $\pm$ 2.3	8.07 $\pm$ 3.4	7.52 $\pm$ 3.0	6.84 $\pm$ 3.1
	b	6.00 $\pm$ 1.9	7.86 $\pm$ 3.3	6.00 $\pm$ 1.5	8.14 $\pm$ 3.4	5.91 $\pm$ 2.1

Table 5.8. Group mean scores for objectively measured variable error (VE%) for assessment NCA3 in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

		Time				
Index [NCA3]		Pre	1h	24h	72h	168h
VE (%) (pre-fatigue)	a	6.40 $\pm$ 3.3	7.78 $\pm$ 4.3	6.66 $\pm$ 3.49	7.90 $\pm$ 3.9	7.70 $\pm$ 3.7
	b	7.20 $\pm$ 3.1	6.50 $\pm$ 2.7	6.42 $\pm$ 1.4	5.54 $\pm$ 2.5	7.38 $\pm$ 3.9
VE (%) (post-fatigue)	a	7.40 $\pm$ 3.5	5.81 $\pm$ 3.2	7.23 $\pm$ 3.3	6.80 $\pm$ 2.5	7.20 $\pm$ 3.7
	b	7.16 $\pm$ 4.4	6.24 $\pm$ 1.9	6.19 $\pm$ 3.2	5.62 $\pm$ 1.8	5.72 $\pm$ 2.2

Table 5.9. Group mean scores for the index of variable error of performance (VE%) for assessment DNCA in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

		Time				
Index [DNCA]		Pre	1h	24h	72h	168h
VE% (pre-fatigue)	a	4.86 $\pm$ 2.28	2.04 $\pm$ 0.99	4.87 $\pm$ 3.07	5.17 $\pm$ 2.27	5.66 $\pm$ 4.19
	b	5.55 $\pm$ 2.25	2.50 $\pm$ 1.34	5.42 $\pm$ 2.19	4.52 $\pm$ 1.95	4.63 $\pm$ 2.90
VE% (post-fatigue)	a	4.87 $\pm$ 2.84	2.07 $\pm$ 0.90	5.26 $\pm$ 2.38	5.49 $\pm$ 1.55	4.76 $\pm$ 2.06
	b	5.71 $\pm$ 3.65	2.31 $\pm$ 1.51	4.43 $\pm$ 1.67	5.45 $\pm$ 1.55	4.76 $\pm$ 2.06

*Indices of self-perceived neuromuscular control associated with assessments NCA2, NCA3 and DNCA.*

The mean and standard deviation (mean  $\pm$  SD) of constant error (SPCE%) and variable error (SPVE%) of self-perceived performance associated with assessments NCA2, NCA3 and DNCA are shown in tables 5.10, 5.11 and 5.12, and 5.13, 5.14 and 5.15, respectively.

Table 5.10. Group mean scores for the index of constant error of self-perceived performance (SPCE%) for assessment NCA3 in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

Index [NCA1]		Time				
		Pre	1h	24h	72h	168h
SPCE% (pre-fatigue)	a	2.10 $\pm$ 2.3	1.38 $\pm$ 1.5	1.39 $\pm$ 3.8	2.15 $\pm$ 2.9	2.08 $\pm$ 3.3
	b	0.56 $\pm$ 1.5	1.64 $\pm$ 0.9	0.08 $\pm$ 2.00	0.51 $\pm$ 1.3	0.66 $\pm$ 0.9
SPCE% (post-fatigue)	a	0.89 $\pm$ 5.3	1.06 $\pm$ 1.7	1.50 $\pm$ 3.7	1.90 $\pm$ 3.1	1.65 $\pm$ 3.4
	b	0.84 $\pm$ 1.6	1.22 $\pm$ 1.5	0.62 $\pm$ 1.5	0.77 $\pm$ 0.7	1.46 $\pm$ 1.7



Table 5.11. Group mean scores for the index of constant error of self-perceived performance (SPCE%) for assessment NCA3 in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

		Time				
Index [NCA3]		Pre	1h	24h	72h	168h
SPCE% (pre-fatigue)	a	2.09 $\pm$ 1.7	0.75 $\pm$ 1.3	2.02 $\pm$ 3.2	1.24 $\pm$ 3.39	1.53 $\pm$ 3.2
	b	1.01 $\pm$ 1.1	1.33 $\pm$ 1.4	0.61 $\pm$ 1.2	1.20 $\pm$ 0.90	1.59 $\pm$ 0.7
SPCE% (post-fatigue)	a	2.01 $\pm$ 2.3	0.58 $\pm$ 1.7	2.12 $\pm$ 3.4	2.14 $\pm$ 2.8	1.36 $\pm$ 3.4
	b	0.79 $\pm$ 1.4	1.11 $\pm$ 1.3	0.60 $\pm$ 1.8	0.85 $\pm$ 1.2	0.98 $\pm$ 1.1

Table 5.12. Group mean scores for the index of constant error of self-perceived performance (SPCE%) for assessment DNCA in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

		Time				
Index [DNCA]		Pre	1h	24h	72h	168h
SPCE% (pre-fatigue)	a	4.05 $\pm$ 5.02	2.05 $\pm$ 3.99	4.62 $\pm$ 2.98	3.76 $\pm$ 3.94	2.71 $\pm$ 3.23
	b	2.89 $\pm$ 1.91	1.72 $\pm$ 4.49	1.33 $\pm$ 4.92	3.17 $\pm$ 3.75	3.89 $\pm$ 2.85
SPCE% (post-fatigue)	a	4.38 $\pm$ 5.08	1.14 $\pm$ 6.59	2.71 $\pm$ 3.86	2.90 $\pm$ 3.17	3.43 $\pm$ 3.17
	b	1.22 $\pm$ 4.73	3.06 $\pm$ 4.23	1.00 $\pm$ 6.81	0.61 $\pm$ 6.16	2.67 $\pm$ 5.26

The ANOVAs of SPCE% scores for NCA2, NCA3 and DNCA revealed no significant group by time interactions ( $p > 0.05$ ), respectively. The results showed that SPCE% was not influenced significantly by the concomitant exercise-induced muscle damage and fatigue task intervention and that the participants' capability to self-perceive neuromuscular control in relation to the precision of force output or knee positioning was preserved in response to this exercise perturbation. Overall mean scores for SPCE% of 1.3% ( $\pm 2.6\%$ ) and 1.2% ( $\pm 2.9\%$ ), 1.4% ( $\pm 2.7\%$ ) and 1.3% ( $\pm 2.4\%$ ), and 3.0% ( $\pm 4.9\%$ ) and 2.3% ( $\pm 5.3\%$ ) associated with pre- and post-fatigue for NCA2, NCA3 and DNCA suggested that in these tests of neuromuscular control, participants perceived consistently that they had possibly produced greater force than had been required to match the target force or joint angle.

Similarly, the results showed that SPVE% for each assessment of neuromuscular control (NCA2, NCA3, and DNCA) was not influenced significantly by the fatigue task intervention and that capability in this aspect of neuromuscular control was also preserved in response to this exercise stress. Overall mean scores for SPVE% of 1.4% ( $\pm 0.7\%$ ) and 1.7% ( $\pm 1.4\%$ ), 1.5% ( $\pm 1.3\%$ ) and 1.4% ( $\pm 1.4\%$ ), and 5.4% ( $\pm 2.7\%$ ) and 5.6% ( $\pm 3.2\%$ ) associated with pre- and post-fatigue task measurements of NCA2, NCA3, and DNCA, respectively, suggested that in these tests of neuromuscular control, participants' self-perception of performance was of a similar level of consistency for those assessments of neuromuscular control requiring precision of force output in particular.

Table 5.13. Group mean scores for the index of variable error of self-perceived performance (SPVE%) for assessment NCA1 in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

		Time				
Index [NCA1]		Pre	1h	24h	72h	168h
SPVE% (pre-fatigue)	a	1.34 $\pm$ 1.2	1.70 $\pm$ 0.89	1.28 $\pm$ 0.7	1.18 $\pm$ 0.9	1.23 $\pm$ 0.7
	b	1.86 $\pm$ 1.6	1.60 $\pm$ 1.2	1.25 $\pm$ 0.7	1.17 $\pm$ 0.7	1.72 $\pm$ 0.6
SPVE% (post-fatigue)	a	3.77 $\pm$ 0.9	1.57 $\pm$ 1.1	1.48 $\pm$ 1.1	1.65 $\pm$ 0.7	1.60 $\pm$ 0.8
	b	1.32 $\pm$ 1.4	1.59 $\pm$ 1.5	1.15 $\pm$ 1.2	1.66 $\pm$ 0.6	1.33 $\pm$ 1.5

Table 5.14. Group mean scores for the index of variable error of self-perceived performance (SPVE%) for assessment NCA3 in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

		Time				
Index [NCA3]		Pre	1h	24h	72h	168h
SPVE% (pre-fatigue)	a	1.36 $\pm$ 0.9	2.05 $\pm$ 1.1	1.81 $\pm$ 0.9	1.67 $\pm$ 0.85	1.54 $\pm$ 0.7
	b	1.77 $\pm$ 0.9	1.56 $\pm$ 2.1	1.69 $\pm$ 1.2	1.16 $\pm$ 0.9	0.98 $\pm$ 0.7
SPVE% (post-fatigue)	a	1.07 $\pm$ 1.0	1.96 $\pm$ 1.0	1.43 $\pm$ 0.98	1.65 $\pm$ 0.7	1.56 $\pm$ 0.8
	b	1.28 $\pm$ 0.89	1.58 $\pm$ 2.1	1.08 $\pm$ 1.3	1.66 $\pm$ 0.6	1.30 $\pm$ 1.0



Table 5.15. Group mean scores for the index of variable error of self-perceived performance (SPVE%) for assessment DNCA in the knee flexors associated with the concomitant exercise-induced muscle damage and fatigue (EIMD-FAT) treatment condition *prior to and after* the static fatiguing exercise task: (a) experimental group and, (b) control group (mean  $\pm$  SD).

Index [DNCA]		Time				
		Pre	1h	24h	72h	168h
SPVE% (pre-fatigue)	a	5.38 $\pm$ 2.99	5.89 $\pm$ 2.71	5.24 $\pm$ 3.04	5.47 $\pm$ 2.77	5.71 $\pm$ 2.11
	b	5.65 $\pm$ 2.63	4.93 $\pm$ 2.61	6.20 $\pm$ 2.16	5.13 $\pm$ 1.96	4.45 $\pm$ 3.58
SPVE% (post-fatigue)	a	5.00 $\pm$ 3.37	5.45 $\pm$ 2.28	5.86 $\pm$ 2.59	6.19 $\pm$ 2.26	6.03 $\pm$ 2.19
	b	5.60 $\pm$ 2.21	6.59 $\pm$ 3.34	5.04 $\pm$ 2.55	5.29 $\pm$ 1.67	4.82 $\pm$ 3.08

#### 5.4 Discussion

The purpose of the EIMD condition in the present study was to investigate the concomitant effects of an episode of muscle damage interspersed amongst serial bouts of fatigue on objective and self-perceived indices of neuromuscular control and performance of the knee flexors. This type of exercise perturbation mimics some of the patterns of exercise inherent in sports and training practice and may serve to disrupt dynamic stabilization around the knee joint. The knee flexors have been considered of paramount importance to the rotational and anterior-posterior stabilisation of the knee joint in particular (Swanik et al., 1997; Grood et al., 1984; Arms et al., 1984).

### *Neuromuscular performance*

The absence of change over the control condition for each index of performance indicates that there were no systematic or learning effects and that observed changes in performance throughout the other treatment conditions can be attributed to the associated exercise intervention(s). The present study found that an exercise perturbation involving concomitant exercise-induced muscle damage and fatigue elicited substantially reduced capability in objectively measured indices of neuromuscular performance. For example, there was a group mean average 24.3% reduction in the volitional force generating capacity of the neuromuscular system compared to baseline ( $193.2 \pm 78.9$  N vs.  $255.2 \pm 69.8$  N [24 h vs. pre, table 5.1; pre-fatigue data]). It is of interest that at the completion of an acute bout of fatiguing exercise at 24 h, this deficit in neuromuscular performance becomes even more prominent (42.9%;  $145.7 \pm 71.2$  N vs.  $255.2 \pm 69.8$  N [24 h post-fatigue vs. pre, pre-fatigue data, table 5.1]). The decrements to neuromuscular performance in key musculature caused by fatigue have been interpreted as representing a potentially compromised capability to contribute to dynamic stabilisation of synovial joints and increased risk of musculoskeletal injury (Chan et al., 2001; Gleeson et al., 1998; Mercer et al., 1998). However, prolonged impairment to neuromuscular performance capabilities following eccentric exercise-induced muscle damage (Byrne et al., 2004) could promote extended periods of elevated threat to the effective stabilisation of synovial joints during critical episodes of mechanical loading.

Overall, the findings from this study strongly suggest that the eccentric exercise protocol induced muscle damage that was characterised indirectly by a significant

and prolonged decrease in PF of the knee flexors associated with the EIMD-FAT condition, together with increases in ratings of perceived soreness and reduced flexibility (tables 5.2 and 5.3, respectively). Reductions in flexibility as measured by the sit-and-reach test correlate with increased passive hamstring stiffness (Magnusson et al., 1997; McHugh et al., 1999). Muscle soreness that reached a peak between 24 h and 48 h (Clarkson et al., 1992), and 24 h -72 h (MacIntyre et al., 1996) after eccentric muscular loading is well established and is a commonly experienced phenomenon associated with exercise-induced muscle damage.

It is plausible that increased tissue stiffness consequent to EIMD may have evolved as a compensatory mechanism within the muscle opposing reduced contractile efficiency and slower transmission of internal protective contractile forces.

However, the extent of increased muscle soreness coupled with the substantive impairment to objectively measured PF performance may present substantial challenges to the neuromuscular system of the knee flexors to generate sufficient levels of force in response to dynamic loading of the knee and accordingly, preserve joint integrity.

The impairment to PF associated with the knee flexors following eccentric exercise is likely to reflect a complex interaction of several factors. These may include possible contributions of structural damage to a proportion of muscle fibres and associated organelles (Hortobagyi et al., 1998; Stupka et al., 2001), possible disruption to intra-muscular  $Ca^{2+}$  dynamics (Morgan and Allen, 1999), increased recruitment of slow-twitch muscle fibres (Warren et al., 2000) and potential conscious/unconscious inhibition as a result of sensitisation of nociceptors (Latash,



1998) associated with the pain response. Furthermore, any relative increase in the recruitment of slow-twitch fibres (i.e. preferential damage to fast-twitch fibres) would make it difficult to apportion properly the relative contributions of fatigue and EIMD to such changes since both would offer a similarity of response in this respect.

Compromised capacity for neuromuscular performance associated with EIMD and fatigue would be expected to reduce the capability of stabilising a synovial joint system and contribute to an increased risk of musculoskeletal injury. This risk may be amplified substantially should there be concomitant disruption to the capability for neuromuscular control. Mechanical disruption of muscle fibres resulting from exercise-induced muscle damage has been hypothesised to be capable of severely jeopardising proprioceptive function (Brockett et al., 1997; Miles et al., 1997; Saxton et al., 1995). For example, muscle spindles play a vital role in movement regulation throughout the stretch reflex (Pattern et al., 1989), provide information about limb position and velocity (Sittig, 1985) and loading of the knee joint (Laskowski et al., 2000). In addition, the Golgi tendon organs provide information about tension in the muscle and regulate inhibition (Gordon and Ghez, 1991).

There may have been adaptations following eccentric exercise involving the shortening of some sarcomeres as compensation for the over-stretched and irreversibly damaged sarcomeres in series (Morgan and Allen, 1999) that may have offered a degree of compensation for functional performance that would otherwise have been compromised. However, the extent of these potential changes to the contractile and connective machinery that would have accompanied EIMD may have been expected to be such that neuromuscular control might have been compromised.

### *Neuromuscular control*

Thus a major finding from the current study was that irrespective of the objective method of assessment of neuromuscular control (i.e. NCA2, NCA3 or DNCA), this aspect of performance was not influenced significantly by the EIMD-fatigue task intervention. In this way, capability in this aspect of neuromuscular control was preserved in response to the imposed exercise perturbation. This finding which mimics that for the isolated effects of serial fatigue tasks reported in Chapter 4, may once again have important implications for appreciating mechanisms by which the body is protected in adverse and stressful conditions. For example, despite EIMD and fatigue-related strength impairments, the risk of injury may be reduced if the athlete was still able to maintain neuromuscular control, sustain a proper technique and his/her reactive (feedback) or proactive (feedforward) capability to external threats to joint stability. As was noted previously, acute physiological potentiation responses (Enoka et al., 1984) may have enhanced performance in response to ecologically-valid exercise stresses that facilitated preservation of the 'control' system in preference to the physiological machinery for performance. Some caution should be used when interpreting these findings. There is a possibility that Type II error may have intruded into the analyses of some of the data in this study since although *a priori* estimates of power associated with the selected experimental design sensitivity appeared sufficient, observed power was compromised in some circumstances with a type II error rate expectancy of some 0.40 rather than the acceptable rate of 0.20.

This interpretation of the experimental findings in this study for objectively measured neuromuscular performance is supported to some extent by the

observations that functional performance is preserved even in the presence of exercise-induced muscle damage. For example, Semark et al. (1999) demonstrated that there was no difference either in sprint time or acceleration over 30 meters after 70 drop jumps to induce muscle damage. However, findings in this study may also conflict with the sparse information in the literature that has investigated the effects of muscle damage on neuromuscular control directly. For example, Saxton et al. (1995) reported an increase in tremor and loss of performance in a proprioceptive task involving the forearm flexors of the non-dominant arm that peaked 2 to 3 days after exercise-induced muscle damage. Overall, such a preservation of performance suggests that neuromuscular control is either a significant factor in the maintenance of joint stability during conditions of concomitant EIMD and fatigue or that concomitant EIMD and fatigue does not affect volitional neuromuscular control.

It is conceivable that the sensorimotor system may have utilised sensory feedback from pain receptors in order to detect and/or compensate for any disruption to the internal environment of the muscle. Thus it is plausible that the performance on neuromuscular control tasks that involve the reproduction of target forces may have been mediated by the pain experienced during active isometric contraction in the experimental limb following EIMD. This strategy may therefore have been used to assist participants in the judgement of force by replicating a certain level of discomfort during each contraction. Several participants did report that they had utilised the level of pain to assist in the judgment of isometric force. This may have been a contributor to NCA3 in particular where self-paced performance of the task facilitated the use of feedback. In contrast, NCA2 required only very brief activations of the involved musculature and therefore participants had only minimal



opportunity to judge or indeed alter the level of force being produced by means of feedback control.

As alluded to earlier, Colebatch and McCloskey (1987) suggested that a stiffer muscle effectively facilitates feedback neuromuscular control mechanisms. Both these latter potential compensatory mechanisms may help to explain the lack of significant differences in neuromuscular control following the EIMD and fatigue intervention.

#### *Self-perception of performance*

As intimated earlier, no previous research has investigated the concomitant effects of an episode of muscle damage interspersed amongst serial bouts of fatigue on self-perceived indices of neuromuscular control and performance of the knee flexors. It was interesting to note that alongside the preservation of objectively measured neuromuscular control, the results showed that self-perceived neuromuscular control as indicated by constant error (SPCE%) for NCA2, NCA3 and DNCA was not influenced significantly by the EIMD-FAT task intervention. In general, both control and intervention groups appeared to have perceived that they had produced greater force than had been required to match the target force or angle. The latter finding was consistent with the corresponding objectively measured performance for NCA2 and DNCA in particular but not necessarily so for NCA3 where objective performance tended to indicate an overestimation of performance capability and an undershooting of the target (table 5.5).

Again this finding may be important because it could be hypothesised that any imprecision of self-perception may contribute to a process by which injury becomes more likely. For example, self-perceived overestimation of performance capability relative to true performance capability may leave the participant partially unprotected during an episode of externally imposed mechanical stressors. Indeed, this scenario may provoke a willingness to choose actions in competitive match-play that may have inherent extra risk when in conjunction with fatigue-related reduced neuromuscular performance capabilities.

An overshooting of a target under conditions of exercise stress may be a mechanism of protection. For example, Burke et al. (1976) proposed that moderate to high intensity muscle contractions may result in excitatory response in spinal segmental proprioceptive circuits, resulting in an involuntary enhancement of sub-maximal muscle tension to following motoneuronal activation. Therefore, in conditions of sub-maximal voluntary muscle contraction, the sensory discharge may well contribute to a potential error (overshooting) in the regulation of force, mainly in the absence of visual feedback of the force being produced. However, it could also be argued that such an overshooting of the target force offers a route ultimately for an inefficient deployment of energy resources in which accelerated fatigue processes may lead to increased risk of injury. Neuromuscular deficiencies have been thought to correlate strongly with injury occurrence (Gleeson et al., 1998; Hawkins et al., 2001).

Importantly, any discrepancies between objective and self-perceived neuromuscular control capabilities may also have offered insights into the contributions of

underlying neurophysiological mechanisms. For example, situations where perception of significant error in performance has been congruent with objectively measured performance might suggest that while calibration of sensory pathways has been preserved, motor performance has been compromised. In contrast, errors in objective performance accompanying perceptions of high precision would be expected to indicate compromised sensory performance. The latter potentially reflected the finding in the present study for NCA3 where there appeared to be a incongruence between self-perceived constant error scores (no error) and corresponding objective performance scores (undershooting).

The results showed that SPVE% for each assessment of neuromuscular control (NCA2, NCA3) was not influenced significantly by the EIMD and fatigue task intervention and that capability in this aspect of neuromuscular control was also preserved in response to this exercise perturbation. Self-perceived overall mean scores for SPVE% of 1.4% ( $\pm 1.1\%$ ) and 1.5% ( $\pm 0.9\%$ ) associated with NCA2 and NCA3 suggested that in these tests of neuromuscular control, participants' self-perception of performance showed a similar level of consistency. Thus in situations where the performer is likely to experience unaccustomed high intensity exercise such as recommencement of training, match play or even rehabilitation following a prolonged period of inactivity or injury and in which skeletal muscle is susceptible to ultrastructural damage (Armstrong et al., 1983; Jones et al., 1989; Byrne and Eston, 1998), as well as a preservation of self-perceived precision (bias) in neuromuscular control, the perception of consistency is similarly maintained.



### *Summary*

In summary, this study assessed concomitant effects of an episode of muscle damage interspersed amongst serial bouts of fatigue on objective and self-perceived indices of neuromuscular control and performance of the knee flexors. This type of exercise perturbation reflects that associated with acute, transient and metabolically-focused effects (fatigue) and that during a more prolonged, mechanical type of disruption to the muscle (EIMD). It was likely to mimic some of the patterns of exercise inherent in sports and training practice and may serve to disrupt dynamic stabilization around the knee joint.

Results showed that despite substantial EIMD and fatigue-related strength impairments (the former confirmed in this experiment by means of indirect physiological descriptive variables including a reduced range of movement and an increase in pain upon active movement of the affected limb), neuromuscular control as measured objectively by the indices of constant and variable error in assessment protocols NCA2, NCA3 and DNCA was not influenced significantly by the EIMD and fatigue task intervention. It was notable that capability in this aspect of neuromuscular control was preserved in response to this type of exercise perturbation in the three different modes of objective assessments of neuromuscular control used in this study. Furthermore, constant and variable error associated with self-perceived performance was similarly not influenced systematically by the EIMD and fatigue task intervention. Overall, such a preservation of neuromuscular control suggests that it is either an important factor in the maintenance of joint stability during conditions of concomitant EIMD and fatigue or that concomitant EIMD and fatigue does not affect volitional neuromuscular control substantively. However, it

is plausible that in order to provide optimum protection of synovial joints, neuromuscular control of the knee flexors may be preserved preferentially to neuromuscular performance when challenged by fatigue-related exercise stresses.

# CHAPTER 6

## FINAL DISCUSSION AND CONCLUSIONS



## **6.0 Synthesis of work.**

*The specific aims of this thesis were: -*

- (i) To examine inter-day reproducibility and single measurement reliability of indices of force error and self-perception of dynamic neuromuscular control in the knee flexors and extensors in men.
- (ii) To examine the effects of serial bouts of acute fatiguing exercise on objective and self-perceived indices of neuromuscular control and performance of the knee flexors in men.
- (iii) To examine the concomitant effects of exercise-induced muscle damage and fatigue on objective and self-perceived indices of neuromuscular control and performance of the knee flexors in males.

The focus of studies in this thesis is shown schematically in figure 6.1. These aims were chosen to further the understanding of performance changes associated with stressful exercise and concomitant injury-related threats to the integrity of synovial joints. These aims were investigated by means of three empirical investigations. The following sections will outline how such aims were examined and detail any new contributions to the literature.

Figure 6.1. A schematic overview illustrating the interrelationship between studies of indices of neuromuscular control and neuromuscular performance associated with the knee flexor musculature in this thesis.

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Figure 6.1: Modified from Minshull (2004). Neuromuscular Performance and Exercise Stress Associated with the Stabilisation of Synovial Joints. Unpublished PhD thesis.

### 6.1. *General discussion.*

The first study within the thesis was designed to evaluate the reproducibility and single-measurement reliability of three types of recently designed assessment tasks of dynamic neuromuscular control in male recreational athletes. The main reason for employing this strategy was to identify the efficacy and appropriateness of the assessment tasks of neuromuscular for future application to healthy (non-injured) populations. The assessment tasks NCA1 and NCA2 were designed with injury prevention in mind.

This approach to the assessment of neuromuscular control has evolved to reflect more closely corrective muscle actions to a specified and effective target level of force to stabilise a synovial joint under mechanical stress in a timely manner. The

latter involve single (NCA1) and serial (NCA2), brief muscle actions involving maximal voluntary recruitment of motor units, respectively. The third approach (NCA3) involved a single, self-paced muscular effort of a long enough duration of time to be moderated by sensory feedback and reflective of self-regulated force and movements in rehabilitative conditioning. This may be reflective of a rehabilitative situation following injury to the knee joint where afferent feedback is required for muscle re-education in order to restore joint awareness and ultimately a safe return to sport.

The assessment of constant error scores enabled the precision of force regulation to be described in relation to a pre-determined target force (50% of peak force capability) and representative of the level of forces associated with peak power outputs during sporting endeavours. An accurate level of force regulation is an important factor in knee joint protection as the knee flexor musculature, together with the knee extensors offers the primary defence against excessive translation of the tibia on the femur. The relative inconsistency of the constant error response was also quantified using the index of variable error. For example, this facilitated the description of the overall pattern of neuromuscular control capability in the knee flexors in terms of whether or not there was either inaccuracy with consistency or inaccuracy with inconsistency. The latter type of neuromuscular control response in particular may reflect a threat to joint instability by means of an inefficient and therefore potentially compromised pattern of sensorimotor detection and integration.

It is fundamental that a sports scientist or health professional (practitioner) working with an individual patient or team has a reproducible and reliable protocol of



measurement for the assessment of neuromuscular control. This will enable the effectiveness and progression of a particular rehabilitation or training strategy to be monitored. Furthermore, the health professional working with a team or group of sports performers also needs to be confident that the tests of neuromuscular control are reliable in order to be able to focus efforts and resources effectively on those most deserving of help.

However, it is noteworthy that there has been no scrutiny of self-perception of the capability for control of neuromuscular performance in comparison to objectively assessed capability for control of neuromuscular performance. New instruments to assess self-perceived performance capability contemporaneously with corresponding objective assessments of neuromuscular control were used throughout this thesis. It is plausible that a discrepancy between perceived and objective capability in the knee extensor and flexor musculature may be important as a contributor to the aetiology of ligamentous injury. This is especially so given the evidence for a causal linkage between injury and proprioceptive capabilities (Caraffa et al., 1996; Hewett et al., 1999). Such assessments may offer further insights into the mechanisms underpinning compromised performance associated with exercise stresses. For example, if self-perception of control was judged to be precise and yet objective performance suggested imprecision in task execution instead, then this would indicate compromised sensory capabilities. Conversely, if self-perception of control was able to mimic objective performance (whether precise or not) then this situation would be expected to be indicative of effective sensory performance with any errors in execution associated with compromised motor performance.

There has been little scrutiny of the reproducibility and reliability characteristics associated with indices of neuromuscular control that would ensure minimum levels of assessment precision. Furthermore, since a discrepancy between perceived and objective capability in the knee extensor and flexor musculature may be important as a contributor to the aetiology of ligamentous injury, it is notable that little consideration has been given in the literature to the reproducibility and reliability characteristics of indices of self-perception of neuromuscular control.

The results from chapter 4 detailing the reproducibility and single-measurement reliability of objective (CE%, VE %) and self-perceived performance (SPCE% and SPVE%) during between-day assessments of neuromuscular control NCA1, NCA2 and NCA3, suggested that these indices of performance offered compromised precision and efficacy. For example, computation of 95% confidence limits of V% scores used in conjunction with the Central Limit Theorem suggests that the mean score of more than 350 inter-day trials would be required to achieve a level of measurement precision of arbitrarily better than  $\pm 5\%$ . This level of precision would facilitate the detection of subtle but potentially important day-to-day changes to intra-individual (case-study) neuromuscular control performance capabilities for a given individual. Similarly for inter-individual comparisons within a group, Spearman-Brown prophecy formula (Winer, 1981) used in conjunction with both  $R_i$  and SEM% scores suggested that in general, individuals within a group would need to accrue some 900 inter-day trials of NCA1, NCA2 or NCA3 in order to achieve discrimination of better than  $\pm 5\%$  (of the group mean score) between individuals with 95% confidence levels.

Thus, these indices of performance should be deployed cautiously within both case-study and inter-individual comparisons and must rely on multiple-trial protocols to achieve acceptable levels of measurement precision in such circumstances.

However, appropriate criteria of experimental design sensitivity should ensure that such indices of neuromuscular control may be used effectively in the testing of experimental hypotheses associated with inter-group comparisons and repeated-measures designs of performance differences.

While the extent to which neuromuscular performance and control must decline in order to constitute a threat to knee joint stability and protection, the remaining focus of the thesis was to investigate the potentially negative effects of two forms of exercise stress on both objective and self-perceived neuromuscular control and performance associated with the knee flexor musculature.

The findings of study 2 (chapter 4) showed that despite substantial fatigue-related strength and muscle activation impairments (for example, there was a group mean average 25.7% reduction in the volitional force generating capacity of the neuromuscular system compared to baseline), neuromuscular control as measured objectively by NCA1, NCA3 or RJA (the latter test involved a passive reproduction of a 'blinded' target knee joint angle) was not influenced significantly by a fatigue task intervention involving three serial, 30 s episodes of static maximal effort, and that capability in this aspect of neuromuscular control was preserved in response to this type of exercise stress. Furthermore, constant error associated with self-perceived performance was similarly not influenced significantly by the fatigue task intervention. It is plausible that in order to provide optimum protection of synovial



joints, neuromuscular control of the knee flexors may be preserved preferentially to neuromuscular performance when challenged by fatigue-related exercise stresses. Results from this study also suggested that each protocol of assessment had been assessing different aspects of neuromuscular control commensurate with its design characteristics. For example, retrospective correlation analyses (Pearson product-moment) showed no significant relationships amongst indices of constant or variable error for each protocol either prior to or after fatigue ( $p > 0.05$ ).

While the second study focused on the acute, transient and metabolically-focused effects of fatigue, the third study attempted to extend our understanding of the extent to which neuromuscular control and performance might be affected by a more prolonged, mechanical type of disruption to the muscle i.e. that associated with exercise-induced muscle damage. The effects of this type of intervention on lower limb performance have received only limited scrutiny to date.

The third study assessed the concomitant effects of an episode of muscle damage interspersed amongst serial bouts of fatigue on objective and self-perceived indices of neuromuscular control and performance of the knee flexors. This type of exercise perturbation reflects that associated with both acute, transient and metabolically-focused effects (fatigue) and that during a more prolonged, mechanical type of disruption to the muscle (EIMD). It was considered likely to mimic some of the patterns of exercise inherent in sports and training practice that may serve to disrupt dynamic stabilization around the knee joint.

Results showed that despite substantial EIMD and fatigue-related strength impairments (the former confirmed in this experiment by means of indirect physiological descriptive variables including a reduced range of movement and an increase in pain upon active movement of the affected limb), neuromuscular control as measured objectively by the indices of constant and variable error in assessment protocols NCA2, NCA3 and DNCA was not influenced significantly by the EIMD and fatigue task intervention. It was notable that capability in this aspect of neuromuscular control was preserved in response to this type of exercise perturbation in the three different modes of objective assessments of neuromuscular control used in this study. The DNCA assessment was a sophisticated and function-related rapid limb positioning task using a specially-constructed computerised goniometer system that included high-precision optical encoders of angular position to provide a time-history of lever-arm and associated knee joint position in relation to a specified target angle. Furthermore, constant and variable error associated with self-perceived performance was similarly not influenced systematically by the EIMD and fatigue task intervention.

A  $\geq 20\%$  reduction in peak force for the experimental limb was confirmed during conditions of EIMD and was comparable to the transient loss of strength observed during conditions of fatigue in chapter 4. However, it was notable that the longer lasting effects of muscle damage did not result in a loss of neuromuscular control capability. Thus, both the precision and consistency of force and joint positioning reproduction tasks were maintained over a 5-day follow-up period during conditions of confirmed EIMD and fatigue in the knee flexors.

Overall, these findings may demonstrate the protective role of the neuromuscular system whilst the limb is experiencing long-term pain, loss of function, oedema and a significant loss of maximal force capacity. It is plausible that in order to provide optimum protection of synovial joints, neuromuscular control of the knee flexors may be preserved preferentially to neuromuscular performance when challenged by fatigue-related exercise stresses. Alternatively, it may be that concomitant EIMD and fatigue does not affect volitional neuromuscular control substantively.

There are several important implications that might be drawn from the investigation into the effects of EIMD and fatigue on neuromuscular control and performance. Coaches, physiotherapists and sports scientists should be aware of the potential for strength loss during conditions of EIMD because of its importance to neuromuscular support of functional activities involving the knee joint. This compromised level of dynamic stabilisation may be evident up to 5 days following substantial eccentric activity.

Ultimately, the findings that neuromuscular control was not influenced significantly despite substantial EIMD and fatigue-related strength and muscle activation impairments may be of particular importance. For although neuromuscular control has been linked causally to musculoskeletal injury, it may be that changes to neuromuscular performance and not control, might have the greatest influence on joint protection and avoidance of injury: Neuromuscular performance has been shown to decrease by up to 50% during ecologically-valid exercise perturbations whereas there had been homeostasis of neuromuscular control.



## *6.2 Limitations*

### *Sample size*

The sample size of all studies in this thesis was designed to offer appropriate levels of experimental design sensitivity where logistically feasible to avoid the likelihood of Type II errors. Occasionally, there was reluctance on the part of potential participants to undertake the considerable time commitment required of each individual within the various studies (> 15 hours) which ultimately limited the volume of participants. Various aspects of the data including an inability to verify normality of performance responses in small samples and unpredictable heterogeneity of response to the EIMD protocol may have ordinarily warranted the deployment of non-parametric methods of data analysis. However, studies 2 and 3 in particular represented exploratory investigations, examining aspects of neuromuscular control and performance not previously considered following EIMD and fatigue intervention. As such, the application of parametric statistics (with their robustness to minor violations of assumptions underpinning their use) seemed reasonable as they were likely to have afforded greater experimental power to detect subtle changes in performance compared to the equivalent non-parametric techniques. Ultimately, this may have helped to provide an indication of whether or not more detailed prospective research might be warranted in the future.

## *6.3 Testing conditions*

Throughout this thesis, emphasis has been placed on the temporal capability of the individual to muster meaningful levels of muscle force to provide adequate dynamic protection to the knee during critical periods of joint loading. Some aspects of capabilities for control and performance have currently been estimated

by means of static maximal voluntary muscle activations within a laboratory setting. While these experimental conditions enabled control of the testing environment and minimisation of associated performance variability, they were not representative of a true 'emergency scenario'. Accordingly, future investigations attempting to replicate such scenarios within assessment settings may obtain a truer estimation of the emergency response and knee injury avoidance capabilities.

#### *6.4 Clinical importance*

It is very desirable to have sophisticated testing equipment in sports medicine clinics to be able to perform different types of testing on muscle tissues following exercise induced fatigue or muscle damage. Muscle tissues after intense exercise demonstrate many different physiological changes such as swelling, deformity, pain and increases in the temperature of muscular tissue, while this may decrease athlete's performance capability for at least days, it will sometimes decrease performance for years if not a permanent disability. Clinical testing presents sports medicine team with a clear idea of the best way to tackle these problems in order to save money, time and effort for those concerned with 'safe' return of athletes to sports activities. Therefore, aspects of clinical testing of athletes need to be developed to further understand the behaviour of muscle tissue associated with fatigue or damage in order to establish more specific clinical treatments and /or conditioning or rehabilitation programs compared to what is already found in the literature.

### *6.5 Recommendations for future study*

The review of literature identified several areas of research which, although not directly addressed in this thesis, should be examined in future work to elucidate further the understanding of the potential relationship between neuromuscular performance, neuromuscular control, perception of performance and injury risk. This relationship should be examined by appropriate prospective randomly-allocated trials, similar to those conducted for the effects of proprioception conditioning (Caraffa et al., 1996). Prospective trials will help to explore whether or not a causative relationship may exist between neuromuscular control, perception of performance and musculoskeletal injury. In addition, examination of indices of objective and self-perceived neuromuscular control during conditions of injury and rehabilitation may reveal whether or not a greater relative difference between these performance capabilities is implicated in ACL injury risk. The neuromuscular performance capabilities of the knee extensor muscle group and the agonist-antagonist in general may also be worthy of further research scrutiny given the potential for close interactive effects in the maintenance of joint stability. However, these types of prospective investigations would require large study population numbers and resources to realise the research aims.

More research is required to examine the mechanisms by which mechanical and metabolic stresses may contribute to potential neuromuscular performance and control impairments, which may be associated with knee injury. Such research may consider more 'functionally-relevant' fatiguing protocols involving muscle activation patterns associated with team sports. Future research may wish to examine the possible effects of neuromuscular inhibitory mechanisms (Zhou et al.,



1995; Gleeson, 2001) on physiological and functional performance relating to knee injury avoidance capabilities following exercise.

Future research may wish to examine the possible effects of exercise induced muscle fatigue and muscle damage on physiological changes within the muscle tissues such as increases in tissues temperature following exercise, and how these physiological changes may contribute to reduce functional capability of athletes during sporting endeavours and increase the chance of injury.

# GLOSSARY

## Glossary

ACL:	Anterior cruciate ligament.
ANOVA:	Univariate analysis of variance.
EMD:	Electromechanical delay is defined as the time delay between the start of electrical activity and tension development in human muscle (Zhou et al.,1995).
MVMA:	Maximal voluntary muscle activation.
PF <sub>v</sub> :	Index of maximal voluntary muscle activation force.
R <sub>i</sub> :	Intraclass correlation coefficient. This statistical index describes single-measurement reliability (Winer, 1981).
SD:	Standard deviation.
SEM	Standard error of measurement.
SEM%:	The standard error of a single measurement (computed as a percentage of the group mean score at 95% confidence limits).
NCA	Neuromuscular control assessment.
NCA-1	Neuromuscular control assessment-1, which represents where the participants is completed the task in a very short time, and the participant has no time to moderate the force output and use afferent and efferent information to avoid injury (Rees, 1994; Glesson et al., 1998).
NCA-2	Neuromuscular control assessment-2, Represent a situation where the participant has perhaps two or three strides in which to lessen the mechanical stress on the joint system; in which case the capability to produce serial, brief controlling force responses may also be vital to prevent injury.
NCA-3	Neuromuscular control assessment-3; this assessment represents a situation during the conditioning or rehabilitation of an injured limb to increase strength.
V%:	Coefficient of variation.
P:	Achieved significance level.
M:	Mean.
PF:	Peak force.
Reliability:	The statistical reliability linked with an index of neuromuscular control. Within a given measurement environment, suitable reliability characteristics linked with a given index of neuromuscular control will ensure that the index is likely to be able to rank the performance capability of members of a group (Gleeson and Mercer,1992).



Reproducibility:	The relative consistency with which an index of neuromuscular control is measured during repeated inter-participant testing. Within a given measurement environment, appropriate reproducibility characteristics connected with a given index of neuromuscular control will ensure that the index is likely to be able to detect relatively small changes in performance capability (Gleeson and Mercer, 1992).
RFD	Rate of force development.
RFD <sub>v</sub> :	Index of rate of force development following to maximal voluntary muscle activation, calculated as the average rate of force increase connected with the force-time response between 25 percent and 75 percent of volitional static peak force.
Sensitivity:	The ability of an index of performance to detect with confidence relatively small changes in performance capability (Gleeson and Mercer, 1992).
EIMD:	Exercise-induce muscle damage.

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# APPENDICES

## Appendix A

### Example informed consent and medical questionnaire

**SCHOOL OF SPORT, HEALTH AND EXERCISE SCIENCES**

**UNIVERSITY OF WALES, BANGOR**

**INFORMED CONSENT**

Name: \_\_\_\_\_

Age: \_\_\_\_\_

Are you in good health? Yes/No  
If no, please explain:

How would you describe your present level of vigorous activity?  
Vigorous (please circle):

	less than once per month
	Once per month
	2-3 times per week
	4-5 times per week
	>5 times per week

Have you ever suffered from a serious illness or accident? Yes/No  
If yes, please give particulars:

Do you suffer, or have you ever suffered from:

Asthma	Yes/No
Diabetes	Yes/No
Bronchitis	Yes/No
Epilepsy	Yes/No
High blood pressure	Yes/No
High cholesterol	Yes/No
Are you currently taking medication?	Yes/No

If yes, please give particulars:

Are you currently attending your GP for any condition or have you consulted your doctor in the last three months? Yes/No  
If yes, please give particulars:

Have you, or are you presently taking part in any other laboratory experiment? Yes/No  
Do you smoke? Yes/No



If you smoke, how many cigarettes do you smoke per day  
If you have smoked previously, approximately how long ago did you stop?

### **PLEASE READ THE FOLLOWING CAREFULLY**

Persons will be considered unfit to do the experimental exercise if they:

- Have a fever, suffer from fainting spells or dizziness;
- Have suspended training due to joint or muscle injury;
- Have a known history of family history of medical disorders, i.e. high blood pressure, high cholesterol, heart or lung disease;
- Have had hyper/hypothermia, heat exhaustion, or any other heat or cold disorder;
- Have anaphylactic shock symptoms to needles, probes, or other medical-type equipment;
- Have chronic or acute symptoms of gastrointestinal bacterial infections (e.g. dysentery, salmonella);
- Have a history of infectious diseases (e.g., HIV, Hepatitis B); and if appropriate to the study design, have a known history of rectal bleeding, anal fissures, haemorrhoids, or any other condition of the rectum.

#### **DECLARATION**

I hereby volunteer to be a participant in experiments/investigations during the period of \_\_\_\_\_2001.

My replies to the above questions are correct to the best of my belief and I understand that they will be treated with the strictest confidence. The experimenter has explained to my satisfaction the purpose of the experiment and possible risks involved.

Furthermore, if I am a student I am aware that taking part or not taking part in this experiment, will neither be detrimental to, or further my position as a student. For my own protection and the protection of others, I undertake to obey the laboratory/study regulations and the instructions of the experimenter regarding the health and safety issues pertaining to this experiment.

I understand I may withdraw from the experiment at any time and that I am under no obligation to give reasons for withdrawal or to attend again for experimentation.

Signature of participant \_\_\_\_\_

Date \_\_\_\_\_

Signature of experimenter \_\_\_\_\_

Date \_\_\_\_\_

## Appendix B

<b>INFORMED CONSENT TO PARTICIPATE IN A RESEARCH PROJECT OR EXPERIMENT</b>
--

The researcher conducting this project subscribes to the ethics conduct of research and to the protection at all times of the interests, comfort, and safety of participants. This form and the information it contains are given to you for your own protection and full understanding of the procedures. Your signature on this form will signify that you have received information which describes the procedures, possible risks, and benefits of this research project, that you have received an adequate opportunity to consider the information, and that you voluntarily agree to participate in the project.

Having been asked by \_\_\_\_\_ of the School of Sport, Health and Exercise Sciences at the University of Wales Bangor to participate in a research project experiment, I have received information regarding the procedures of the experiment.

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

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

I also understand that I may register any complaint I might have about this experiment to Dr Roger Eston Head of the School of Sport Health and Exercise Sciences, and that I will be offered the opportunity of providing feedback on the experiment using standard report forms.

I may obtain copies of the results of this study, upon its completion, by contacting:

\_\_\_\_\_

I confirm that I have been given adequate opportunity to ask any questions and that these have been answered to my satisfaction.

I have been informed that the research material will/will not [SELECT ONE] be held confidential by the researcher.

I agree to participate in the study

\_\_\_\_\_

NAME (please type or print legibly): \_\_\_\_\_

ADDRESS: (Optional) \_\_\_\_\_

\_\_\_\_\_

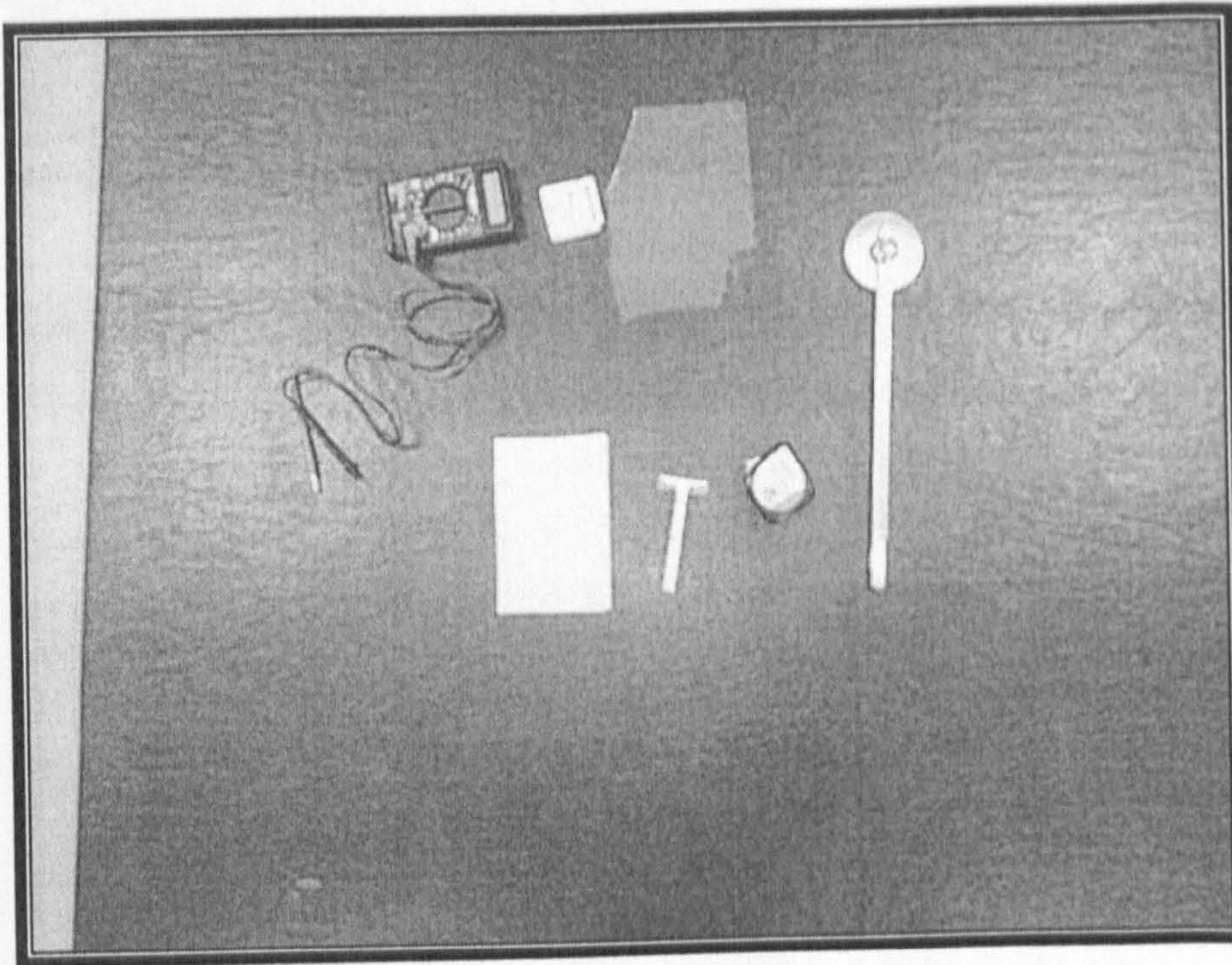
SIGNATURE: \_\_\_\_\_

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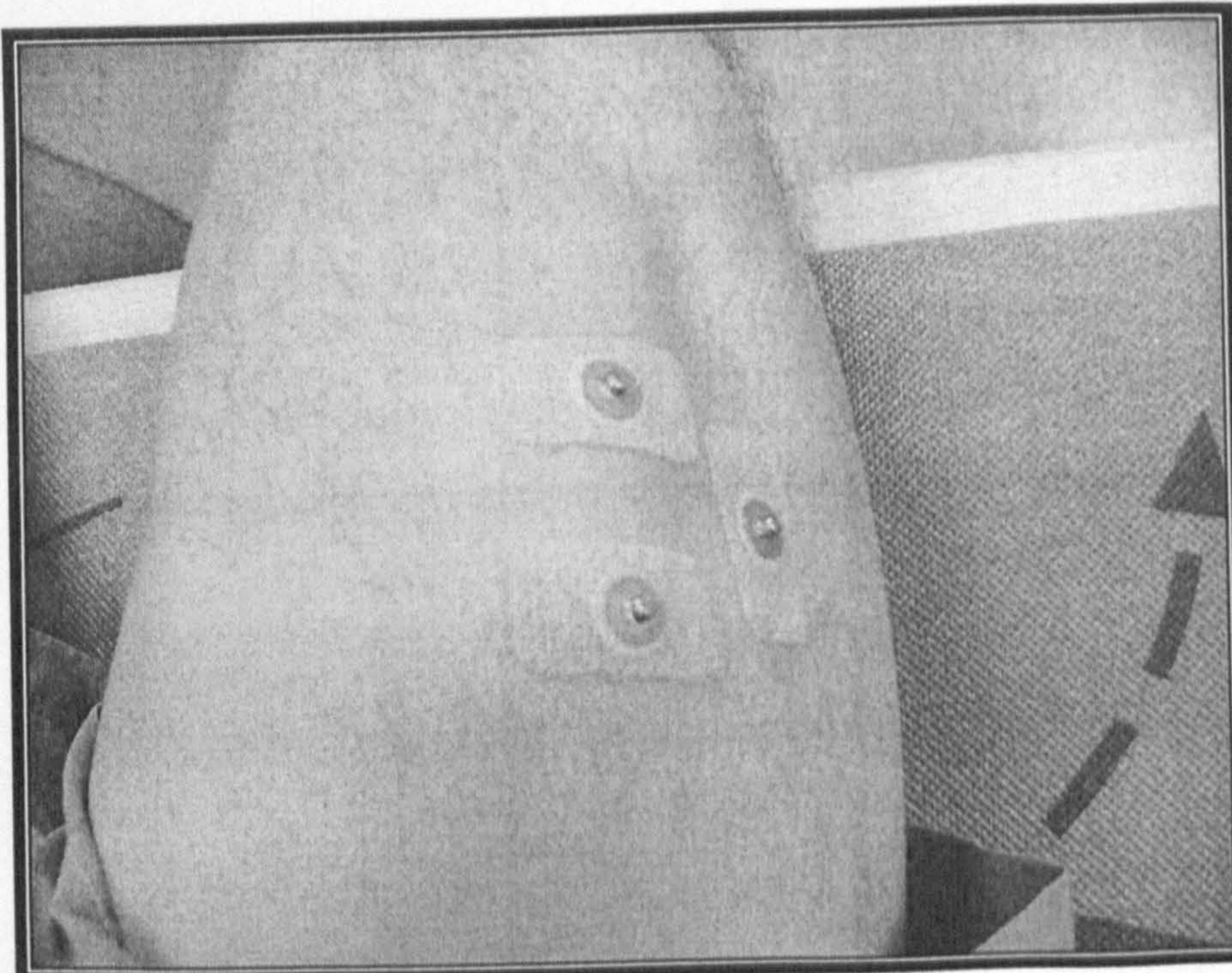


Appendix c

Photographs of selected experimental procedures



Types of tools used in the studies through out the thesis



Three electrode position on lateral vasties muscle of the quadriceps muscle to connected to load cell.



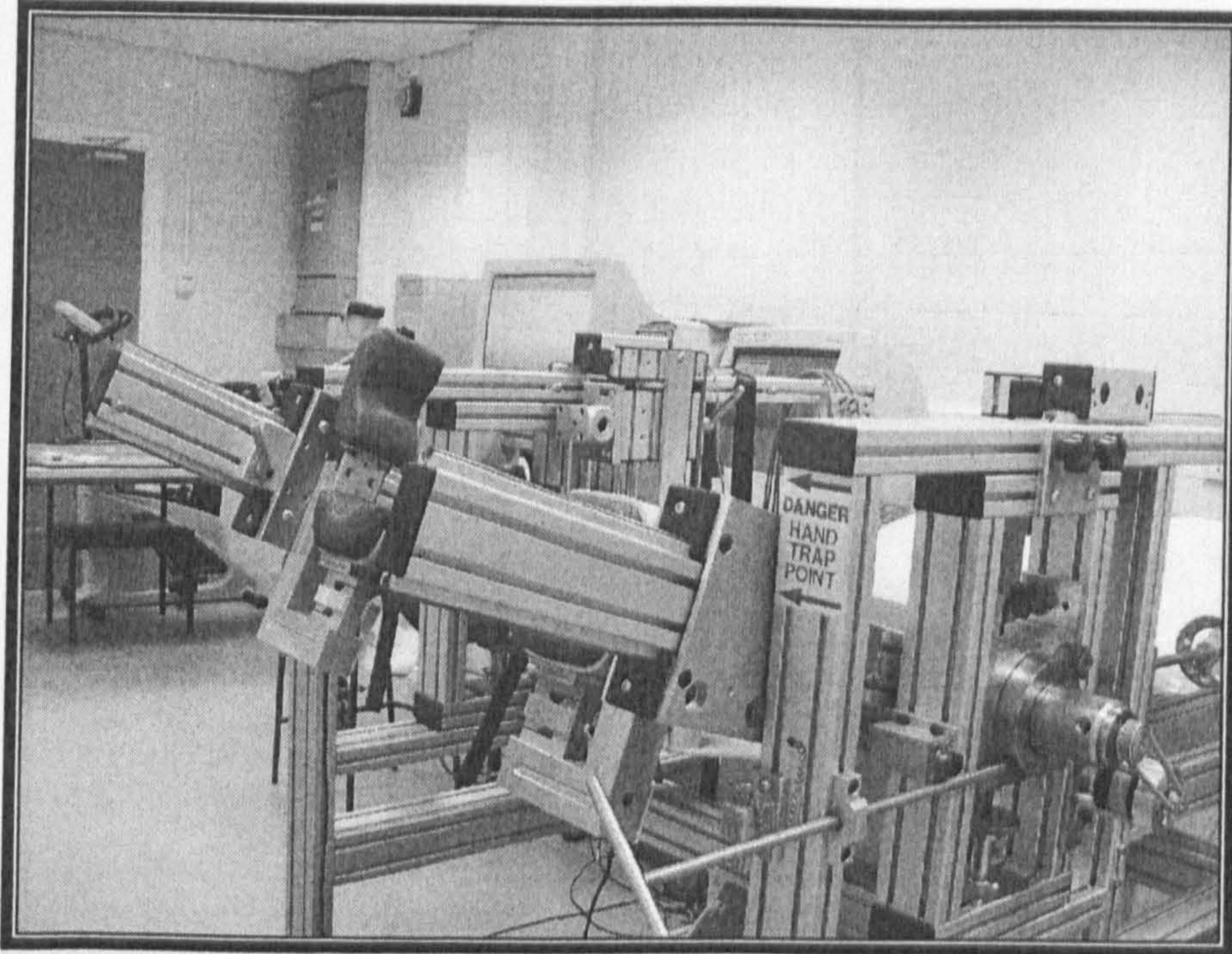
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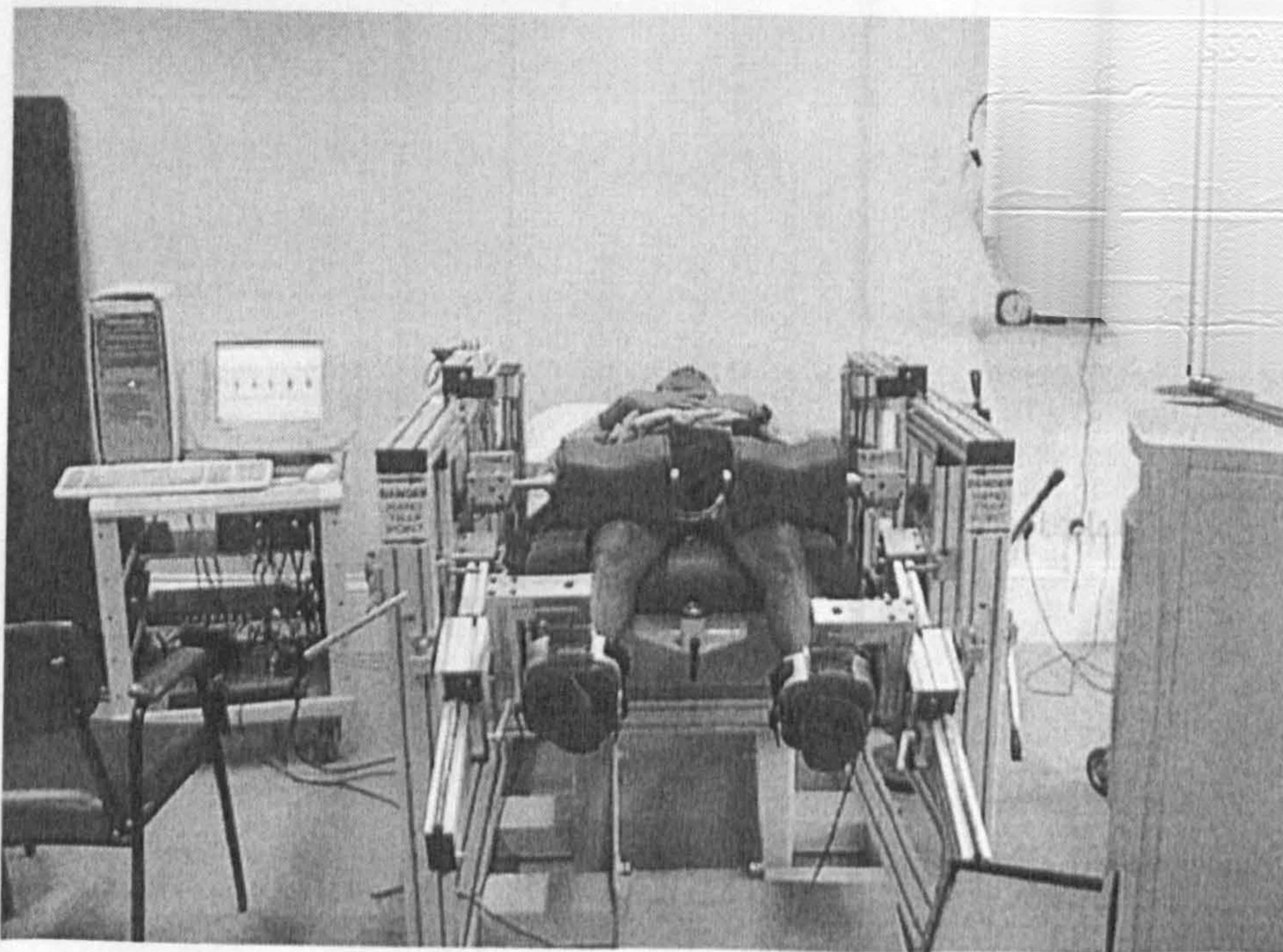
One of the participants is in an apposition to demonstrate set and reach test.

The participant is performing the set and reach test



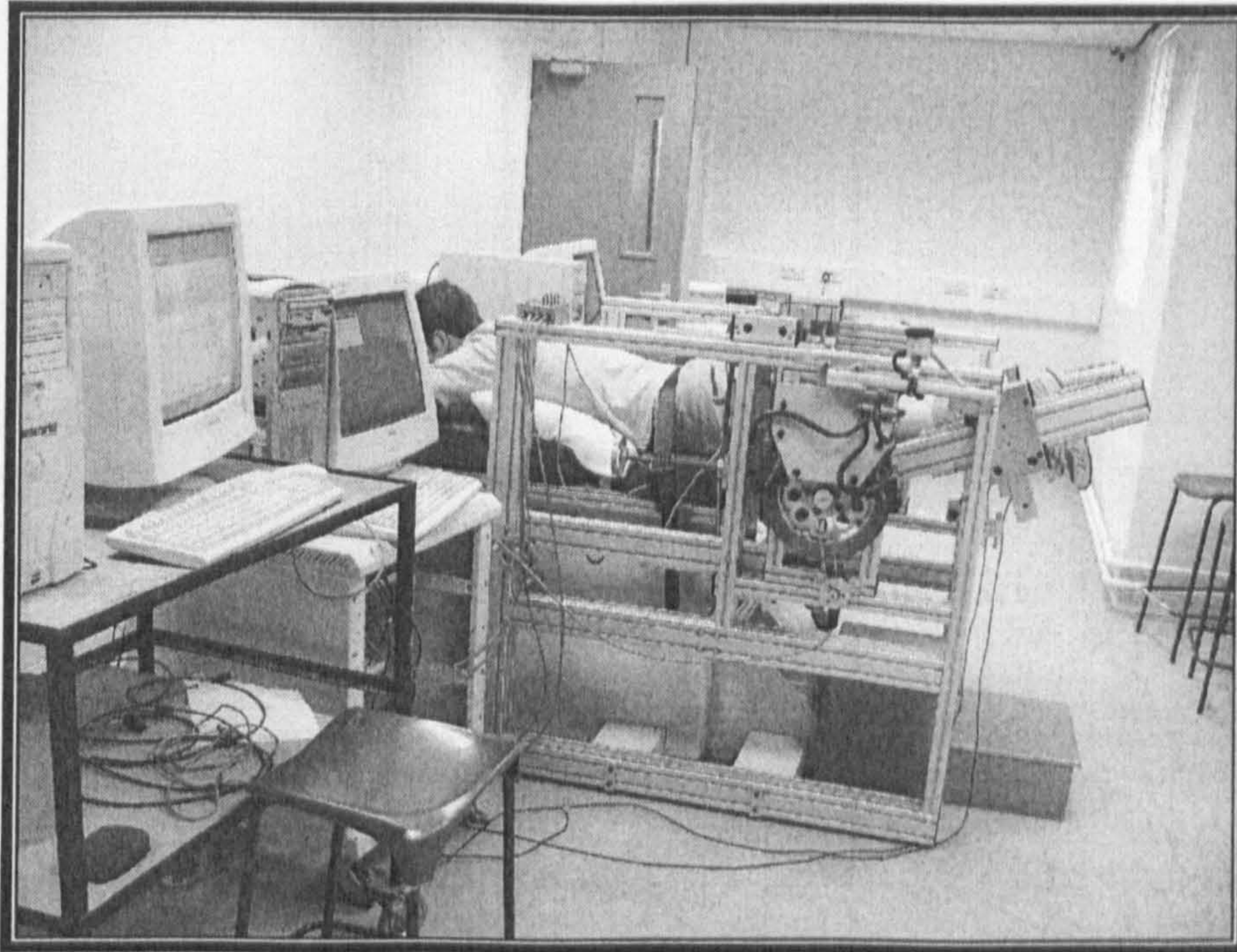


Custom-built  
dynamometer  
(modified from  
Gleeson et al., 1995)

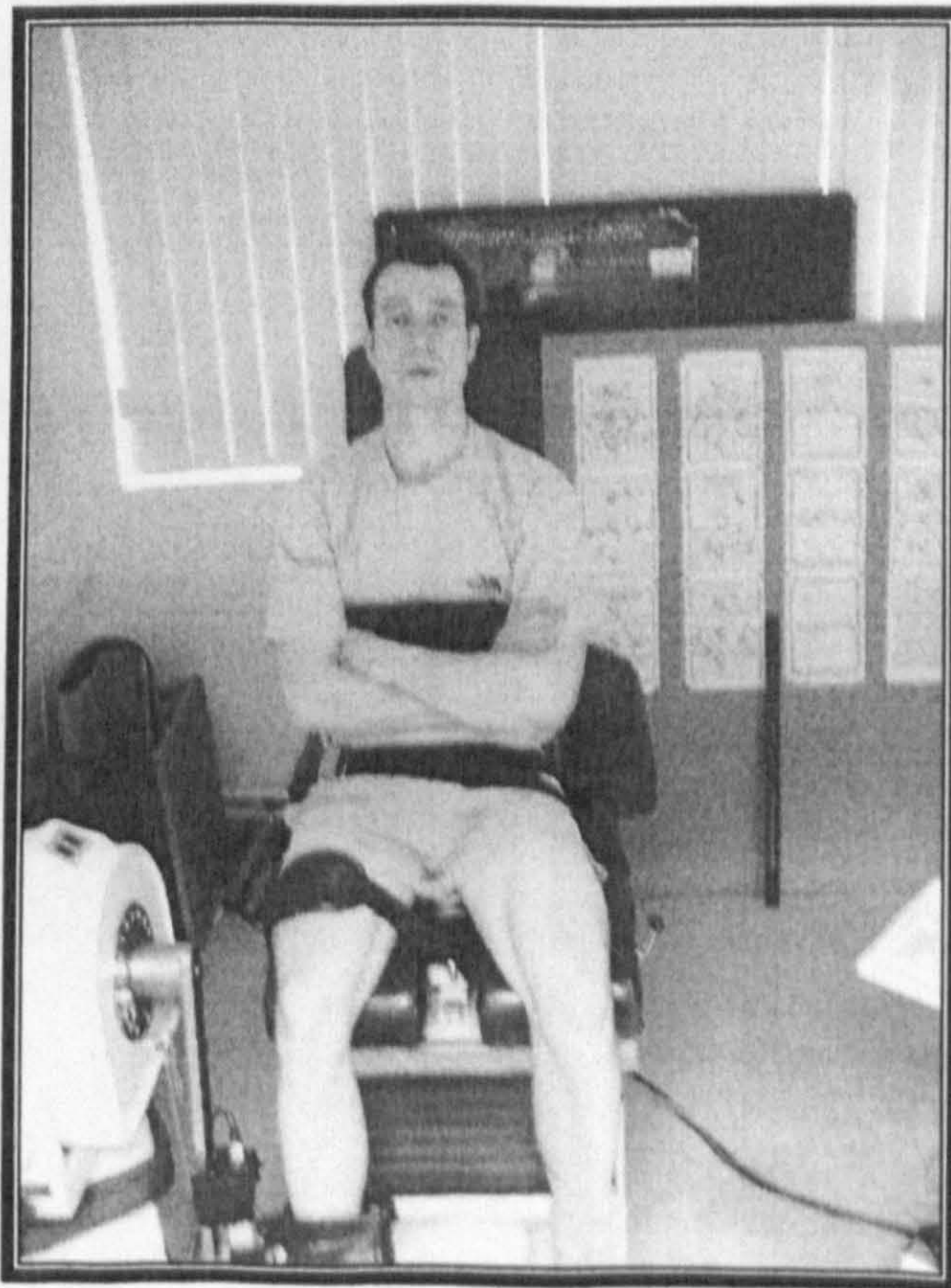


The participant performing  
quadriceps test to assist  
neuromuscular control  
assessment.





The participant performing hamstring test to assist neuromuscular control assessment.



Participant placed on the isokinetic dynamometer (KinCom) used to educe exercise-induced muscle damage by via of eccentric exercise of the knee flexors.