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Exercise-related sensorimotor and neuromuscular performance of the knee joint

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Exercise-related sensorimotor and neuromuscular performance of the knee joint.

Ph.D. thesis

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EXERCISE-RELATED SENSORIMOTOR AND NEUROMUSCULAR PERFORMANCE OF THE KNEE JOINT.

Abstract.

This thesis is presented as a series of five empirical studies. The first study examined the reproducibility and single-measurement reliability associated with two types of dynamic sensorimotor performance (SMP) assessment task. The SMP assessment tasks were thereafter applied to assess the effects of exercise stress in the form of fatigue, an initial and repeated-bout of exercise induced muscle damage and training on sensorimotor and neuromuscular performance associated with the knee flexor musculature of female athletes.

The results from the first study revealed that within-session estimates of both types I and II SMP offered the least level of biological variance or greatest reliability during a single opportunity for assessment. However, such results must be interpreted with caution when used as a clinical outcome measure as the R_1 for both types I and II SMP assessment tasks (within-session, within-day, and between-day) do not approach the clinically acceptable reliability coefficient threshold of greater than 0.80 (Currier, 1984). This represents a limited capacity to discriminate physiological change in types I and II SMP performance, based on a single trial for both within-day and between-day assessments. Therefore, the mean score of 15 and 25 trials is recommended as the basis for estimating sensorimotor performance in order to reduce measurement error during types I and II SMP tasks, respectively.

The second study examined the effects of four bouts of an acute fatigue protocol (4 x 40 seconds of maximal isometric activity in the knee flexor musculature) and acute recovery (at 1, 3 and 6 minutes following the cessation of exercise) on neuromuscular and sensorimotor performance of the knee flexors in female soccer players. A 20% disruption to peak force was observed following the fatigue intervention alongside a significant alteration to the evoked neuromuscular activation response and the accuracy of force perception. This may have significant implications during a 90 minute soccer game when an even greater potential for a loss of strength and dynamic support around the knee joint may exist.

The third and fourth studies investigated the effects of an initial-bout and repeated-bout of exercise induced muscle damage (EIMD) on the physiological, neuromuscular and sensorimotor performance of the knee flexors in female athletes. EIMD was confirmed in both phases of the experiment via several physiological indicators of EIMD and a significant (20%) loss of strength in the knee flexors. Therefore, the repeated-bout effect was not witnessed during a second exposure to an eccentric exercise protocol in the knee flexors. Sensorimotor performance was however preserved during conditions of EIMD which suggests a protective role of force regulation during functional disruption to the lower limb.

Finally, a functional training intervention was assessed for its effect on sensorimotor and neuromuscular performance of the knee flexors in female athletes both at rest and following a single episode of fatigue. No significant alteration to sensorimotor or neuromuscular performance of the knee flexors was witnessed following the two-week intervention period. This may represent either a limited capacity to discriminate a biologically meaningful level of change in the performance indices, or the fact that sensorimotor performance may not be significantly improved in the knee flexors of the 'healthy' female athlete during the chosen period of exposure to the training stimulus.

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This thesis is dedicated to my nanna Marian. *God Bless you.*

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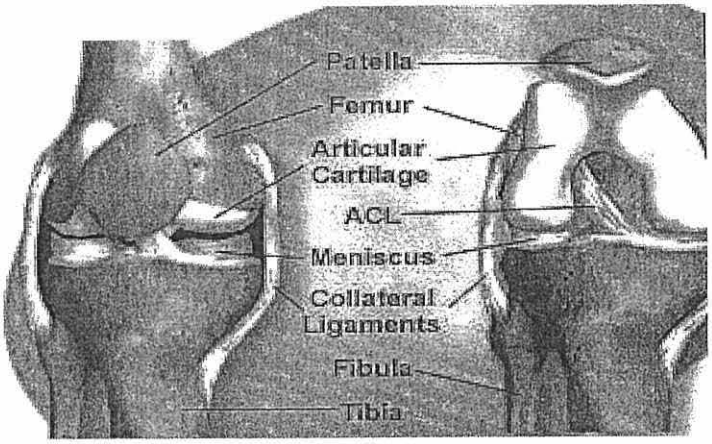
1.0. Introduction.

Lower limb injuries are commonly reported amongst the athletic population, particularly in team sports. Disruption of the anterior cruciate ligament (ACL) accounts for one in 3000 of the many types of injury each year within the USA; equating to 100,000 injuries annually (Huston, Greenfield and Wojtys, 2000). The ACL is the principal ligamentous restraint to anterior tibio-femoral displacement and is the most commonly injured of the major knee joint ligaments (Rees, 1994).



Knee in extension **Knee in flexion**

Figure 1.1. An anatomical diagram of the knee joint, illustrating the ACL and other key structures involved in the movements of flexion and extension.

There is accumulating evidence of an ACL injury epidemic via non-contact aetiologies in team sport athletes (Hutchinson and Ireland, 1995; Huston et al., 2000). In fact, female athletes are suffering ACL disruption two to eight times more frequently than men participating in the same sports (Huston et al., 2000). This may be due to the failure of dynamic stability during fatigue and loss of motor control (Rozzi et al., 1999). Injury to the ACL typically occurs upon rapid deceleration of the body (Huston et al., 2000). For example, during movements which require a rapid change in direction and/or speed, alongside cutting, twisting or pivoting. The prominent mechanism for ACL injury is illustrated in figure 1.2 and involves a slightly flexed knee (25 degrees of flexion) with excessive internal rotation of the tibia at foot strike (Boden, Dean, Feagin and Garrett, 2001).

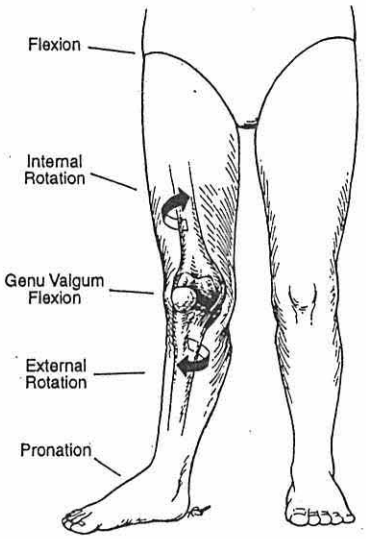


Figure 1.2. The 'position of no return' for the ACL involving hip adduction and internal rotation, 20-30° of knee flexion, external rotation of the tibia and forefoot pronation (Ireland, et al. 2001).

Cruciate ligament disruption may range from a partial to a complete rupture and result in varying degrees of knee joint instability, concomitant exercise dysfunction and reduction in quality of life (Rees, 1994). Even relatively minor ACL trauma may act as a precursor for more severe subsequent ligamentous injury and co-morbidity factors such as meniscal injury and Osteoarthritis (Rees, 1994).

Various causative factors have been investigated in ACL injury and are categorised as intrinsic (non-controllable), extrinsic (potentially-controllable) or both (partially-controllable). Intrinsic factors involve a narrow inter-condylar notch, generalised physiological laxity, mal-alignment of the lower extremity and poor proprioception. Extrinsic factors include an abnormal quadriceps to hamstrings muscle group ratio, shoe-surface interface, playing surface, and the athlete's playing style (Huston et al. 2000).

1.1. Defining 'proprioception'.

The term proprioception is taken from the latin (re)ceptus (the act of receiving) and proprius (one's own) (Ashton-Miller, Wojtys, Huston and Fry-Welch, 2001). Although there is no universally accepted definition of proprioception (Beard, Kyberd, Fergusson and Dodd, 1993) proprioception was first defined by Sherrington (1906) as an individual's ability to sense the

movement and position of a limb through the nerve receptors in the capsule, ligaments, tendons and muscles. Proprioception encompasses both static and dynamic aspects of position sense (Lephart, 1995; Laskowski, 1997). Static sense provides us with conscious orientation of one body part to another whereas dynamic sense gives the neuromuscular system feedback about the rate and direction of movement. Thus, proprioception can be thought of as a complex neuromuscular process that involves both afferent and efferent signals, which allows the body to maintain both stability and orientation.

The presence of neuro-receptors within the human knee joint was first described by Rauber (1874). Palmar (1944) suggested a theory that the ligaments within the knee supply the central nervous system (CNS) with the input that makes neuromuscular control of the knee joints possible. Subsequently, Cohen (1955) popularised the idea of an 'arthrokinetic reflex', suggesting that the origin of important protective afferent input was the knee joint capsule. Proprioceptive input may therefore be derived from mechanoreceptors in the joint capsule, ligaments and musculo-tendinous units, which relay afferent feedback to the CNS for continuous update. Four types of afferent receptors have been identified and are summarised in table 1.1.

Receptor classification	Type	Description	Location	Function
Ruffini	I	Thinly encapsulated, globular corpuscles.	Collateral and cruciate ligaments, knee joint capsule and menisci.	Low threshold mechanoreceptors, which detect static and dynamic joint angle, velocity and intra-articular pressure.
Paciniform	II	Elongated and conical, enclosed in a multi-laminated connective tissue capsule.	Knee joint capsule, both cruciate ligaments, and both meniscii.	Low threshold, rapidly adapting mechanoreceptors responsible for signaling dynamic changes in the tissues and both the initiation of acceleration and the termination of joint movement.
Golgi tendon organs (GTO)	III	Long fusiform shaped receptors enclosed in a fine connective tissue capsule.	Ligaments and horns of meniscii.	High threshold, slowly adapting mechanoreceptors, which function at extreme angles of joint displacement.
Nocioceptive (bare nerve endings)	IV	Fine terminal nerves, non-myelinated.	Various tissues of the joint. e.g. The articular surfaces of the joint capsule and ligaments.	High threshold pain receptors.

Table 1.1 Summary of sensory receptors associated with the knee joint including their classification location and function.

A knee ligament injury is not normally an isolated trauma in terms of other pathology within the knee. Damage to other structures may include the joint capsule, bone surface (articular cartilage) and meniscus, leading to an array of disturbed sensory feedback. Numerous histological studies have provided further evidence that the cruciate ligaments act as a valuable source of sensory input (Kennedy, Alexander and Hayes, 1982; Johannsen, 1991; Barrack, Lund and Skinner, 1994; Jennings, 1994) despite the fact that neuronal tissue constitutes a mere 1.0 - 2.5 % of the total volume of the ACL (Schutte, Dabezies, Zimny and Happel, 1987). The knee joint ligaments are capable of providing the CNS with a varied receptor inflow in most situations. However, this ability may be compromised during rapid movements (Johannsen, 1991).

Recent advances in research using local anaesthetic have also attributed muscle spindles and the sensation of tension as key components in position sense. Both joint and muscle receptors have a role in the proprioceptive system as nervous pathways from the skin, muscle and joint all converge on the same thalmic and cortical neurones (Wyke, 1981; Jennings, 1994). Hence, the proprioceptive awareness of movement and position is due to a combined experience from all kinds of receptors (Jennings, 1994).

Over recent years the term proprioception has been widely applied in describing many distinctly different components of the motor control system. *"It is due to such a conflict in application that the term 'sensorimotor system' has been adopted to unite physiological systems of the complex neuromuscular process involved in maintaining joint homeostasis"* (Lephart and Fu, 2000). This system encompasses both the acquisition and conversion of a sensory stimulus to a neural signal and the transmission of the signal via afferent pathways to the CNS. In addition, the processing and integration of the neural signal by the nervous centres of the CNS into a motor response for locomotion and functional joint stabilisation is involved. This definition of an 'overarching system' shall be hereafter applied within the context of the ensuing experiments.

1.2. Motor control.

Motor control is defined as "control of posture, balance and movement, involving an interaction between the individual, the task, and the environment" whereas motor learning is "the acquisition and/or modification of movement" (Shumway-Cook and Woollacott, 1995). Three main stages of motor learning have been described: cognitive, associative and autonomous. The cognitive stage involves the initial understanding of the task, strategies are devised and accepted or dropped and improvements are very rapid. During the associative stage of learning, the refinement of the skill takes place at a slower pace and variability of performance decreases. At the autonomous stage, the skill has become automatic (Fitts and Posner, 1967). The neural input that is provided by peripheral and visual mechanoreceptors is integrated by the CNS to generate a motor response. Figure 1.3 illustrates the three levels of mechanoreceptor control.

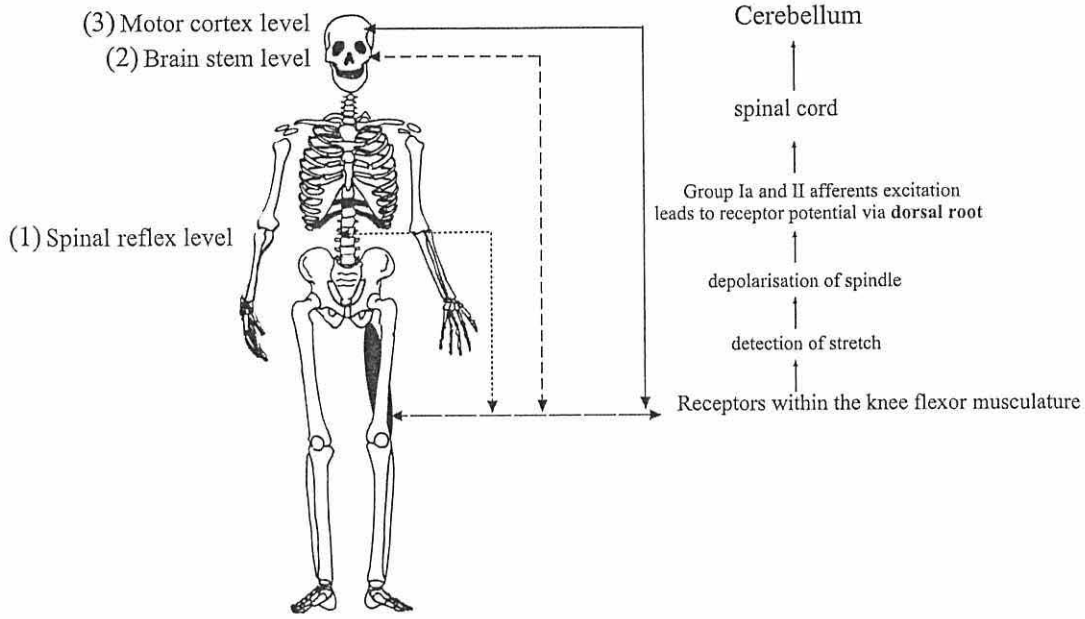


Figure 1.3. The three levels of mechanoreceptor control using the knee flexor musculature as an example.

(1) Spinal reflexes mediate movement patterns (unconsciously) that are received from higher levels of the nervous system providing for reflex joint stabilisation during conditions of abnormal stress. Because they are generated local to the joint, they are characterised as gross, quick movements that require no cortical input or sensory feedback (Wojtys and Huston, 1994).

(2) The brain stem level of control is enabled via input from the joint mechanoreceptors, vestibular centres and visual input, helping to maintain posture and balance of the body.

(3) The motor cortex of the cerebellum is the highest level of function, providing cognitive awareness of body position and movement in which motor commands are initiated for voluntary movements. The dorsal and ventral spinocerebellar tracts contain the most rapidly conducting nerves in the entire human physiological system (~100m/s). Sensory information is therefore provided by muscle spindles, somatic sensors, golgi tendon organs, tactile receptors in the skin and other joint receptors. The cerebellum regulates the intensity and sequence of motor actions of agonist and antagonist muscle groups by comparing the

intentions of the motor control system to actual musculoskeletal movements (Dye, 2000). This level of control is affected by attention and motivation, must be learned and requires practice for perfection. The resulting movement pattern from the cerebellum is stored as a central command, which may be performed without continuous reference to consciousness.

1.3. Neuromuscular performance following ACL reconstruction.

Often, the only means of restoring 'normal' function to a sportspersons knee following an ACL rupture is via surgical reconstruction of the ligament. It has been suggested that despite the choice of graft material, ACL reconstruction improves sensorimotor performance by merely reinstating the physiologically acceptable boundaries of translation within the knee joint, and thus function (Rees, 2002, [personal communication]).

However, results in terms of both the functional outcome and patient's confidence in the affected knee following ACL reconstruction surgery remain to be optimised. Symptoms such as functional instability (the knee 'giving way') and muscular weakness often continue after surgery and indeed throughout the rehabilitation period. This has been associated with a partial de-afferentation of the capsule and ligament, leading to an altered feedback and a mis-match with the 'pre-injury' motor program. Therefore errors in the normal co-ordination pattern of the muscles and joint instability often persist despite an intact cruciate replacement (Johannsen, Sjolander and Sojka, 1991). It is also highly plausible that the musculature of the affected limb does not respond adequately to the drive from the nervous system following injury and ACL reconstruction (Rees, 2002 [personal communication]).

Whether such a phenomenon may be attributed to inhibition or a reduced level of central recruitment is presently unknown. This apparent disruption to neural processing, exaggerated by fatigue during sport, could in fact have predisposed an individual to the original injury. It is also likely that alongside this altered sensorimotor input and disrupted neuromuscular control, a decrease in the level of an athlete's performance may be due to apprehension and

fear of re-injury. Regretfully therefore, a sensorimotor deficit may detract from the functional result of ACL reconstruction and may indeed predispose to reinjury (Lephart, Kocher, Fu and Borsa, 1992).

1.4. Assessment methods for sensorimotor capacity.

Traditional measures of proprioception in relation to the knee joint have included static laboratory-based assessments, namely the threshold to detect passive motion [TDPM] (Barrack, Skinner, Cook and Haddad, 1983; Skinner, Wyatt and Hodgdon, 1986; Barrack, Skinner and Buckley, 1989) the ability to reproduce joint positioning (Barrack, et al., 1983; Skinner, et al., 1986; Barrack, et al., 1989; Lattanzio, Petrella, Sproule and Fowler, 1997) and the reflex hamstrings contraction latency (RHCL) in reaction to a posteriorly applied force (Beard, Kyberd, Fergusson and Dodd, 1993; Jennings and Seedholm, 1994; Huston and Wojtys, 1996).

Many studies have utilised these techniques to assess proprioceptive modification following ACL rupture and reconstruction (Barrack, et al., 1989; Corrigan, Cashman and Brady, 1992; Beard, et al., 1993; MacDonald, Hedden and Pacin, 1996; Borsa, Lephart, Irrang and Safran, 1997; Carter, Jenkinson and Wilson, 1997; Friden, Roberts and Movin, 1998; Beynnon, Ryder and Konradsen, 1999; Pap, Machner and Nebelung, 1999; Roberts, Friden, Zatterstrom and Lindstrand, 1999) as well as during osteo arthritis [OA](Barrack et al., 1983; Barrett, Cobb and Bentley, 1991; Marks and Quinney, 1993; 1997), patello femoral pain syndrome [PFPS] (Kramer et al., 1997) and fatigue (Skinner, et al., 1986; Marks and Quinney, 1993; Lattanzio et al., 1997). However differences between the experimental design and data analysis techniques of all previous studies makes a comparison of results very difficult. Therefore, the consensus of opinion regarding the utility of such assessment techniques is currently equivocal. It is not yet known whether the information obtained from the traditional methods

of proprioceptive assessment provides a true reflection of an individual's ability to perform functional movements. The results from previous investigations into the validity of traditional methods of proprioceptive assessment are explored briefly in chapter 5.0.

While the importance of 'proprioception' as a clinical outcome measure following ACL reconstruction is becoming well recognized (Barrack et al., 1989) the best measurement techniques have yet to be defined (Beynnon et al., 2000). The question arising from the earliest form of research into knee joint proprioception is whether such findings are in fact applicable to the dynamic model of knee joint stability that involves requiring rapid decision-making and neuromuscular feedback. As mechanoreceptors may respond differently during active and passive knee movements, past research may have failed to employ an ecologically valid means of testing sensorimotor interaction during both slow and quick gross movement patterns of the lower limb. Consequently, the applicability of any of such findings to the dynamic scenario is questionable.

1.5. Perceived force and sensorimotor performance.

When observers match the perceived forces of isometric contractions exerted by the reference and indicator arms, the forces produced by the two limbs are approximately equal under non-fatigued conditions (Cafarelli and Bigland-Ritchie, 1979; Jones and Hunter, 1982a). Dynamic sensorimotor performance [SMP] may be defined as *"the ability to regulate volitional force"* and is expressed as the discrepancy between the blinded attainment of a prescribed force (a given percentage of daily volitional peak force, PFv) and the subsequent reproduction of this force (following a standardised delay) (Gleeson et al., 1997).

Two types of sensorimotor performance task have been recently designed. Type I SMP task is time restricted to simulate quick movements in reaction to unexpected conflicts in

movement control. This represents joint preservation within a limited time frame (an open-loop skill). Type II SMP is a slow, self-regulated movement, aimed to mimic the process of muscle re-education following disruption to the neuromuscular system during injury or surgical intervention (a closed-loop skill). The latter type of movement utilizes feedback during the execution of the task to bring about immediate changes in performance, whereas feedback from the open-loop movement may only be used to improve the motor program for the next trial (Cordo, Carlton, Bevan and Carlton, 1994). Please see chapter 4.0 (general methodologies), section 4.6 for a full description of the assessment techniques for each type of task. Dynamic SMP assessment provides a practical, and more importantly, quantifiable means of assessing neuromuscular and sensorimotor capability in terms of force regulation awareness. This type of assessment protocol has received previous application and scientific validation in the ACL deficient population (Gleeson et al., 1997).

The aims of the following thesis are manifold. An initial aim is to assess the single measurement reliability and reproducibility of the two forms of sensorimotor performance task. Where appropriate, these tests will then be applied to assess the influences on sensorimotor and neuromuscular performance of 'negative' exercise stress conditions including fatigue and exercise induced muscle damage. Furthermore, the effects of an acute ('positive') training programme designed to improve neuromuscular and sensorimotor function will be evaluated using these outcome measures. Female athletes were the chosen focus of experiments 6.0, 7.1, 7.2 and 8.0 to investigate the reported higher incidence of injury compared to male counterparts. This research will attempt to offer an expansion of our understanding of dynamic sensorimotor performance and critically evaluate the efficacy of new assessment techniques that may be deployed ultimately in both non-injured and clinical populations.

2.0. Statement of the problem.

This thesis is designed to critically evaluate the measurement utility of methods of dynamic sensorimotor performance assessment in the lower limb and to examine the effects of acute and chronic exercise stress and neuromuscular and sensorimotor training on sensorimotor performance and recovery.

2.1. Aims.

The specific aims are:

(i) To assess the reproducibility and single-measurement reliability of two indices of dynamic sensorimotor performance associated with the knee flexor musculature of the 'healthy' male and female.

(ii) To evaluate the accumulative effects of four bouts of exercise stress and acute recovery on neuromuscular and type I and II indices of SMP associated with the knee flexor musculature of the preferred limb in female athletes.

(iii) To evaluate the effects of eccentric exercise induced muscle damage (EIMD) on neuromuscular and SMP associated with the knee flexor musculature using a single-leg intervention contra-lateral control model over a 5-day follow-up period (female collegiate athletes).

(iv) To investigate the effects of a repeated-bout of eccentric EIMD in the knee flexor musculature using a single-leg intervention contra-lateral control model over a 5-day follow-up period (female collegiate athletes).

(v) To investigate the effects of a single limb neuromuscular training programme on neuromuscular and SMP of the knee flexor musculature using a single-leg intervention contra-lateral control model in female collegiate athletes.

2.2. Objectives.

(i) To describe the measurement reproducibility and single-measurement reliability of two types of SMP assessment tasks during within-day and between-day assessment sessions.

(ii) To describe the effects of four acute bouts of maximal exercise stress on neuromuscular and SMP during two types of assessment task (assessed on separate test occasions) in the knee flexor musculature (preferred limb) of female collegiate athletes.

(iii) To describe the effects of eccentric EIMD on neuromuscular and SMP using a single-leg intervention contra-lateral control model.

(iv) To describe the influence of a repeated-bout of eccentric EIMD on neuromuscular and SMP using a single-leg intervention contra-lateral control model.

(v) To describe the effects of a single-leg training protocol on neuromuscular and SMP in the non-preferred limb of female intercollegiate athletes, compared to an age and sex-matched control (no training) group.

3.0 Review of the literature.

3.1. Sensorimotor performance and injury in the female athlete.

The sensorimotor system intervenes to prevent extreme translation at the knee joint in terms of excessive flexion (>140 °), extension (>180° with up to 10° hyper-extension), internal rotation (>30° with the knee flexed at 30°) and external rotation (>45°) (Thompson and Floyd, 1994). The biceps femoris muscle is illustrated in figure 3.1. This muscle is primarily involved in movement at the knee joint and alongside the semitendinosus and semimembranosus muscles, acts as a dynamic stabiliser against anterior shear of the tibia on the femur.

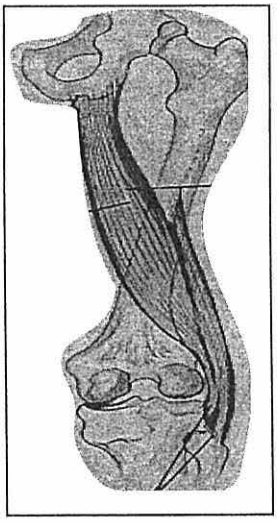


Figure 3.1. The biceps femoris musculature from a posterior view of the right leg (Thompson and Floyd, 1994).

Excessive laxity or compliance within the knee flexor or 'hamstrings' muscle group has been shown to compromise joint homeostasis via a lower passive muscle tone. This may explain the higher incidence of ACL injury in females, given their general tendency for greater overall flexibility (Boden et al., 2001). A 'vicious circle' scenario of repetitive joint instability via loss of mechanoreceptor feed-back from torn knee ligaments contributing to reflex loss, muscular splinting, repetitive major and minor injury and progressive joint laxity (looseness) has been recently proposed (Kennedy, Alexander and Hayes, 1982) and is illustrated in figure 3.2.

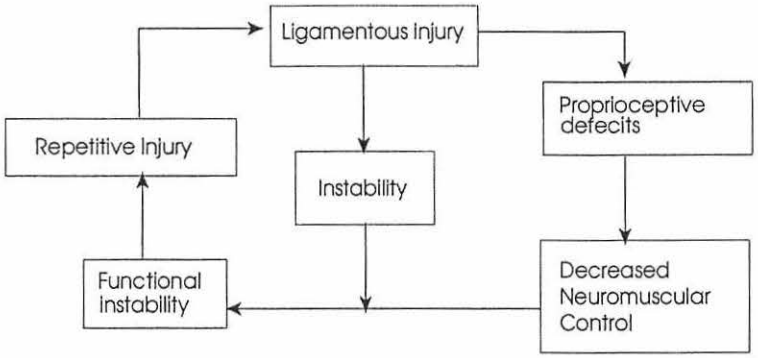


Figure 3.2. The ACL Injury paradigm as described by Kennedy et al., (1982).

The decline in proprioceptive ability of an ACL deficient extremity has not been found to correlate with strength loss, lending support to the belief that proprioceptive loss was the cause rather than the result of ACL injury (Barrack et al., 1989). It is estimated that up to 99% of sensory input may be disregarded at any given moment (Guyton, 1986). This provides a simple explanation of how the slightest alteration in situational play during sports participation may lead to a severe injury. e.g. a divot in the pitch, or a planted foot that is 'caught'. Any pre-activation of the hamstrings may therefore be nullified if the afferent sensory information is confusing or misinterpreted by an athlete. Critical errors in timing, coordination, conditioning and concentration may thus disrupt the dynamic restraint system (Swanik et al., 1997).

3.2. Neuromuscular feed-back and the ACL.

If a particular effort could be relied on to always produce the same muscular force, the need for peripheral force sensing would be reduced (Burgess and Jones, 1997). Sensory awareness in the form of both feed-back and feed-forward is a key aspect in protecting the integrity of the knee joint. Feed-forward neuromuscular control involves planning movements based on sensory information from past experiences via a pre-activated muscle tension (preparation for anticipated loads or activities), whereas the feed-back process continually regulates motor

control through reflex pathways, helping to maintain posture and the regulation of slow movement (reactive muscle activity) (Lephart et al., 2000).

The importance of a mechanism of protection in anticipation of movements and joint loads has been a recent research focus (Swanik et al., 1997). This procedure aims to enable rapid neuromuscular compensation in the injury-producing scenario. Hence, a centrally generated motor command is created and once formed eliminates reliance on reflex pathways producing quick motor commands (Kandell et al., 1996). It is an important point to make that the feed-back and feed-forward mechanisms of motor control are not exclusive to one another but the combined interaction of both mechanisms results in co-ordinated motor skills and dynamic joint stabilisation (Lephart et al., 2000).

It is accepted that both the feed-back and feed-forward pathways are trainable (Guyton et al., 1981). Regrettably however, this fact is frequently neglected in the design of rehabilitation and indeed sports training programmes. The potential for an enhancement in sensorimotor capacity therefore lies in the development of sport-specific movement repertoires as well as preparatory and reactive actions for dynamic restraint (Swanik et al., 1997). Feed-back is essential in the process of re-learning motor programs. Since the sensory feed-back from an injured joint is persistently disturbed, the existing motor programs may need to be modified by regaining coordinated movements or learning new coordinated movement patterns during rehabilitation (Agberg, 2002).

3.3. The muscular system and joint protection (stiffness and force regulation).

The significant contribution of the musculature around the knee has only recently been recognized as an important factor in joint stability. Muscles are dynamic visco-elastic organs that can apply passive visco-elastic effects to a joint when not active (passive tone) and

variable dynamic visco-elastic effects when contracting under voluntary or reflexive control (Solomonow and Krogsgaard, 2001). As the muscles across the knee joint contract, they act to increase joint contact force and decrease tibio-femoral displacement, dissipating potentially dangerous loads and lowering the force carried by the ACL and other passive structures (Huston et al., 2000). Muscle stiffness across the knee has both intrinsic and extrinsic components; the intrinsic being largely dependent on the number of active actin-myosin cross-bridges in the muscles at a specified point and the extrinsic component dependent on the excitation provided by the alpha and gamma motoneurons. The latter component has been recognized as the most trainable of the two aspects to stiffness (Wojtys, Huston, Taylor and Bastian, 1996).

Markolf et al. (1978) recorded that on average, stiffness figures in terms of anterior-posterior drawer of the tibia on the femur were improved by a factor of 2.2 to 4.2 when subjects made a maximal isometric contraction of the knee flexors. Moreover, subjects who considered themselves to be in peak physical condition could enhance their stiffness up to 10.4 times. Therefore an efficient ability to recognise that an alteration in muscular stiffness is required may be the ultimate concern in the preservation of a joint. Much controversy exists with regard to the latency between the recognition of an injury-producing stimulus and the time taken to react, bringing about protection of knee joint structures (Pope, 1979).

Significant gender differences have been observed for active knee stiffness (Wojtys et al., 1998). Bryant and Cook (1988) observed the varus and valgus stiffness of 17 female and 24 male subjects. The knees of the females were found to rotate 66% more than those of the males and were 35 % less stiff than the male comparison group. It has also been suggested that female athletes appear to have adopted compensatory mechanisms of increased hamstring activity to achieve functional stabilisation (Rozzi, Lephart, Gear and Fu, 1999). Pre-activated

muscles therefore offer a greater level of protection against joint perturbation and may facilitate feed-back neuromuscular control mechanisms (Lephart et al., 2000).

3.3.1. Electromyography.

Electromyography (EMG) is the technique for recording the changes in the electrical potential of a muscle when it is caused to contract by a motor nerve impulse (Gleeson, 2001) and offers the only method of objectively assessing when a muscle is active (Grieve, 1975). Electromyography is also an indirect estimate of the excitatory input to a muscle and the magnitude of the descending motor command (Cafarelli and Bigland-Ritchie, 1979). Petrofsky (1980) noted a linear increase in EMG with sustained contractions and also that the rate of increase in EMG signal over time depended on the level of force exerted. This suggests a centrally mediated theory of force perception.

3.3.2. Electromechanical delay.

Electro mechanical delay (EMD) is defined as "*the time delay between the onset of muscle activity and the onset of force generation*" (Norman and Komi, 1979). The EMD is determined using EMG techniques, by the time taken for the contractile component to stretch the series elastic component of the muscle (Winter and Brookes, 1991). The rate of shortening of the series elastic component of muscle may be the primary cause of EMD in a given muscle (Norman and Komi, 1979). Group mean estimates of the reproducibility of the index of EMD and related latencies of muscle activation have ranged between 3.2% and 6.9% for repeated inter-day assessments (Gleeson et al., 1998). Fatigue related slowing of excitation-coupling or altered visco-elastic behavior of collagen within the series elastic component of muscle and ligamentous structures of the knee may be reflected in an increased EMD (Gleeson, 2001).

3.4. Injury and fatigue.

Fatigue may be viewed as the enemy to sporting performance and is defined as '*a decreased force generating capacity, which develops gradually during exercise and is distinct from exhaustion which occurs when the required force or exercise intensity can no longer be maintained*' (Vollestad et al., 1988). The incidence of sporting injury may be associated with fatigue. It would appear that the majority of injuries occur at the end of the first and moreover the end of the second half of the professional soccer game: a time period when most players are experiencing both mental and physical fatigue (Hawkins et al., 2001).

3.4.1. Sensorimotor performance and fatigue.

Only three studies have investigated the direct effects of fatigue on 'proprioceptive' ability to date. Skinner et al. (1986) studied knee proprioception in healthy young men. Fatigue was induced via a protocol of alternate sprints of 1 and 0.25 miles with a 90 second rest between bouts over a 3.75 mile course. The ability to reproduce passive knee joint angles in a seated position was significantly reduced following the fatigue intervention. This was attributed to the negative influence of lactic acid on muscle receptor input.

Marks and Quinney (1993) studied the effects of maximal isokinetic quadriceps contractions on knee position sense in females. No significant differences were observed between an experimental (20 maximal concentric and eccentric quadriceps contractions) and a control group (no exercise). This study suggested that knee joint proprioception was preserved during strenuous active muscle contraction.

A significant reduction in proprioceptive awareness was observed in both male and female subjects during conditions of fatigue (Lattanzio et al., 1997). Three different 'fatigue' protocols for lower limb cycling were utilized and classified as 'ramp', 'continuous' and

'interval' tests. The subject's ability to reproduce knee angles in a standing position was assessed prior to and following such activities for three separate comparisons. Proprioception was impaired following all three test conditions in the male study group and following the continuous and interval tests in the female subjects. The authors of this study could not conclude whether such a reduction in performance was however clinically viable or applicable to the dynamic sporting scenario.

This lack of consensus in findings and widespread variation in assessment protocols suggests that further scientific investigation is warranted into the influence of fatigue on sensorimotor capacity. From a training and rehabilitation perspective, future research is also required to assess the effects of exercise stress on the neuromuscular and sensorimotor systems. If we can assist the sportsperson to approach his/her biological performance capacity or 'ceiling' via an improvement in both neuromuscular and sensorimotor performance, we may ultimately prevent injury.

3.5. Sensorimotor performance and exercise induced muscle damage.

Delayed onset muscle soreness (DOMS) is a common sensation following unaccustomed exercise which results in the symptoms of muscle soreness, stiffness, tenderness and pain on active movement (Byrne and Eston, 1998). This damage is attributed to the muscle lengthening whilst under tension. The most common symptoms of exercise induced muscle damage (EIMD) and DOMS are strength loss, pain on active movement, muscle stiffness, soreness and inflammation.

The initial symptoms of muscle discomfort or soreness are generally witnessed at approximately 8 hours post 'damage–inducing' exercise, which may coincide with increased intra-muscular pressure and inflammation of the connective tissue, sensitising pain receptors.

Pain and loss of normal function tend to peak at 24-48 hours following the EIMD intervention and subside within 5-7 days of the exercise (Cleak and Eston, 1992; Eston, Finney, Baker and Baltzopoulous, 1996). Inflammation and damage to the affected muscles is associated with DOMS alongside elevated serum activities of muscle-specific enzymes such as creatine kinase (CK). Blood plasma CK levels are taken as an indicator of muscle damage with elevated levels being normally apparent within 2 days of EIMD. Peaks in blood plasma CK are typically observed at 4-6 days following the EIMD intervention.

3.5.1. Mechanisms of delayed onset muscle soreness.

The exact mechanism(s) to explain how muscle soreness develops and why there is a delay is not fully understood (Clarkson, Nosaska and Braun, 1992). The symptoms of muscle damage are believed to be caused by damage; yet DOMS and muscle damage share a poor temporal relationship (Byrne and Eston, 1998).

3.5.2. Functional consequences of delayed onset muscle soreness.

Strength loss can have detrimental effects on athletic performance. An inherent loss of force generating capacity and reduction in range of motion are typical functional consequences that accompany DOMS (Byrne and Eston, 1998). Such are accompanied by an elevated physiological response to sub-maximal exercise and may restrict basic daily activities, including the use of stairs (when the lower limb muscle groups are targeted). The majority of prior research has investigated isometric strength reduction in the knee extensors following eccentric exercise (Clarkson et al., 1992; Cleak and Eston, 1992). Isometric force generating capacity was reduced by as much as 50% immediately following eccentric exercise (Jones, Newham and Torgan, 1989; Clarkson et al., 1992; Cleak and Eston, 1992).

3.5.3. Exercise induced muscle damage.

Muscle damage has been referred to as mechanical failure of individual myofibrils consistent with 'materials fatigue', typical of ductile material subjected to cyclic tensile loading (Armstrong, Warren, J.A. and Warren, G.L., 1991). Damage is greater with contractions at long as opposed to short muscle lengths: occurring on the descending limb of the length-tension curve whilst active force is decreasing and passive force is increasing (Armstrong et al., 1991; Byrne and Eston, 1998; Newham, 1998). The explanations offered for this phenomenon include that eccentric contractions generate forces greater than both isometric and concentric contractions (Jones et al., 1993). Also, for a given force production, less motor unit recruitment is required for eccentric versus concentric contractions, which may lead to higher stress per active muscle fibre in eccentric contraction, and structural damage (Adams et al., 1992).

It has been previously suggested that longer muscle lengths increase the magnitude of muscle fibre damage (Jones et al., 1989) and that the velocity of muscle lengthening contributes to the extent of damage (McCully and Faulkner, 1986). DOMS has also been attributed to damage of the non-contractile connective tissue (Jones et al., 1989); as spontaneous muscle shortening and stiffness occur following eccentric muscle activity. Damage to the series elastic components of a muscle such as the myo-tendinous units may prompt an inflammatory response that presents as oedema and stiffening within the affected muscle group.

The 'popping sarcome' theory is a proposed mechanism to describe the loss in force generating capacity during EIMD. This suggests enough sarcomere elongation during eccentric exercise to stretch it beyond filament overlap, reducing the overall number of available cross-bridges. Exercise in the presence of muscle damage involves greater metabolic stress and can result in premature fatigue (Gleeson, Blannin and Zhu, 1995;

Gleeson, Blannin and Walsh, 1998). As outlined in the introduction to this thesis, numerous techniques of 'proprioceptive' assessment exist. Yet the relationship between EIMD, DOMS and sensorimotor system function is presently unclear. Few studies have investigated the effects of EIMD on sensorimotor performance and are restricted to the upper body (Cafarelli and Bigland-Ritchie, 1982; Saxton, Clarkson, James et al., 1995; Brockett, Warren and Gregory et al., 1997).

Saxton et al. (1995) examined the effects of exercise induced muscle damage on joint angle and force sensation in six male and six female right-handed subjects. The ability to reproduce both joint position and force was tested using both experimental as well as the contra-lateral control limb. The damage inducing exercise consisted of 50 eccentric muscle actions, divided into two sets of 25 eccentric muscle actions with a five-minute break between sets. Force sensation was examined with 90° of flexion at the elbow joint prior to, and following the above eccentric exercise protocol. Each subject produced a force using the experimental arm until 35 % of the daily peak force was achieved whilst simultaneously attempting to match the prescribed force using visual feed-back from a pen chart recorder in the control limb. The force was recorded when the subject felt that they had achieved the designated force replication. The mean force for a 3 second period was taken as the criterion score, and results were presented as a constant error (CE) score.

Sensorimotor performance was significantly impaired by the eccentric exercise. Constant error scores decreased immediately after the exercise and remained at a low level over the 5 days post exercise ($p < 0.01$). The bias tended to be towards an under shooting of the target i.e. the subjects over estimated their force production. However, when the experimental arm acted as its own reference, subjects replicated 35 % of the daily reduced peak force,

demonstrating an adjusted force perception capacity, relative to the reduced functional capacity.

Reliance on a feed-forward mechanism and an inability to detect impaired muscle fibre recruitment resulting from structural disturbance may account for some degree for loss of force proprioception between arms (Saxton et al., 1995). This suggestion has profound implications for successful performance of certain motor tasks during recovery from exercise induced muscle damage. The above findings contradict those of Cafarelli et al. (1982) who found that subjects using the same protocol had a tendency to overshoot or underestimate forces in the range of 15 to 20 % peak force.

Brockett et al. (1997) found that eccentric exercise affected force sensation in the elbow over a period of 100 hours [assessed at 20 hour intervals] following activity. For a target force of 10% peak force, force errors were 3.8% of peak force equating to a 40% error (over-estimation) on the target force. This was attributed to an elevated level of activity in the tendon organs (de-sensitisation) during muscle damage. The higher resting muscle tension and disturbed force perception peaked at the 20 hour follow-up occasion. The target force of 10% PFv may have been a difficult level of force to replicate in the upper body. This may explain such a dramatic level of force error.

3.5.4. Factors affecting exercise induced muscle damage.

Musculoskeletal flexibility is a factor affecting the severity of symptoms after eccentric exercise (McHugh, Connoly and Eston, 1999). Following a bout of eccentric exercise, subjects with 'stiff' (within a classification scale of 'stiff',' normal' or 'compliant') muscles had significantly greater loss of strength, pain, muscle tenderness, and elevation of plasma creatine kinase [CK] activity than those with compliant muscles. Greater muscle damage in

the 'stiff' subjects was attributed to the inability of the tendon and aponeurosis of the stiffer muscles to absorb the lengthening imposed by the eccentric contractions (McHugh, 2000). Warm-up and stretching are influential factors in muscle stiffness (Gleim, 1997). Warm-up has been shown to reduce subsequent symptoms of muscle damage (Nosaska and Clarkson, 1997).

3.5.5. The repeated-bout effect.

Muscles that have been pre-conditioned with eccentric contractions have been found to be protected against damage from subsequent bouts of eccentric exercise in the same muscle group. This is known as the repeated-bout effect and has been shown with various forms of exercise in both human and animal models (McHugh et al., 1999). This protective effect of prior exercise was first indicated by Highman and Altland (1963) and more recently attributed to eccentric work (Schwane et al., 1983).

Pre-conditioning the muscle with eccentric contractions clearly provides a protective effect and the primary dose of exercise stress may not need to damage the muscle in order to provide protection (McHugh, 2000). Brown et al. (1997) discovered that as few as 10 maximal eccentric contractions of the knee extensors were sufficient to reduce EIMD symptoms appreciably after a subsequent bout of 50 maximal contractions performed three weeks later. A clear repeated-bout effect was observed, despite the fact that the initial dose of exercise did not induce significant symptoms of muscle damage (McHugh, 2000). Therefore, the key elements for the repeated-bout effect are that:

- The pre-conditioning contractions are eccentric.
- High intensity contractions are performed.
- The pre-conditioning contractions affect the same muscle groups that will be working eccentrically in the repeated-bout.

This protective adaptation has been shown to last for approximately 6 weeks and is apparent before full recovery from the initial bout of exercise inducing damage (Nosaska et al., 1995). It is not yet known whether sensorimotor performance is influenced during an initial and repeated-bout of EIMD in the knee flexor musculature.

3.6. The trainability of sensorimotor performance.

The results from preliminary studies investigating whether 'general' proprioceptive training improves joint proprioception are encouraging. In a prospective controlled training study of male semi professional soccer players, Caraffa, Cerulli and Projetti (1996) observed a significant reduction in the incidence of ACL injury in the soccer season following a progressive 'wobble-board' training intervention. The incidence of ACL injury was reduced sevenfold compared to an age matched athletic comparison.

Hewett, Lindenfield, Riccobene and Noyes (1999) also demonstrated a significant decrease in the incidence of knee injury in female athletes after a specific plyometric training program (injury incidence per 1000 athlete exposures was 0.43 in untrained female athletes, 0.12 in trained female athletes, and 0.09 in male athletes. [$p=0.02$, chi- square analysis]). This equates to a 3.6 times higher incidence of knee injury in the untrained compared to the trained athletes ($p=0.05$) which was 4.8 times higher than male athletes ($p=0.03$).

Heidt, Sweeterman and Carlonas (2000) examined the effects of a pre-season training program on the occurrence of soccer injuries in female players. 42 out of 300 female soccer players were randomly selected to participate in a 7-week specialised training regime which included sports specific cardio-vascular conditioning, plyometric work, sport cord drills, strength training and flexibility exercises. The resulting injury statistics for the season (recorded by team trainers) were significantly less within the athletes chosen for the specific

training (14% compared to 33.7%). A functional jump-training program for hamstrings and gastrocnemius muscle groups also produced a subsequent decrease in the rate of ACL injury (Huston et al., 2000).

The latter findings are in agreement with Junge, Rosch and Peterson (2002) who significantly reduced the incidence of soccer injuries in youth amateur players via an improvement of the structure and content of soccer training alongside a greater level of education and supervision of coaches and players. The incidence of injury per 1000 hours of training and playing soccer was 6.7 in the intervention group versus 8.5 in the control group. This equated to 21% less injuries in the trained group. However, a recent risk factor analysis demonstrated that no performance variables other than poor endurance had an influence on the occurrence of an injury (Dvorak, Junge and Chomiak, 2000). The possible improvement in sensorimotor performance of the knee flexor musculature following a specific neuromuscular and sensorimotor training programme has not been assessed to date in terms of using the recently devised types I and II SMP tasks as a means of both constant and variable error assessment.

3.7. Summary.

The relationship between the control of musculo-skeletal movement and an ever-changing athletic environment is complex. Fatigue may provide an added threat to sensorimotor and neuromuscular control of the knee joint during sports participation. Therefore, the anticipation of movement to provide a protective muscular contraction, sufficient in its capacity to prevent damage, may be the only means of injury prevention. One of the current challenges facing sports medicine is to determine whether it is possible to improve an individual's sensorimotor and neuromuscular control; with the intention of achieving optimal performance and ultimately the potential for a reduced risk of injury. Several studies have investigated the effectiveness of a proprioceptive training intervention. However, the success

of such studies has been based on subsequent injury statistics rather than a scientific quantification of functional performance indices. Recently designed tests of dynamic sensorimotor performance may offer a more realistic tool for the clinical assessment of knee joint performance.

4.0. General methodologies.

4.1. Subjects.

Prior to participation in any part of the research activity, each subject completed a 'pre study questionnaire' and signed an 'informed consent to participate in a research project' form in accordance with the University of Wales, Bangor health and safety guidelines. For an example of each type of form, please see appendix 1.

Following habituation procedures, each subject completed a standardised warm-up, which consisted of five minutes on a cycle ergometer (90 Watts for male and 60 Watts for female subjects) and a further five minutes of static stretching of the involved musculature. Participants were instructed to refrain from strenuous physical activity for the 24 hours prior to all test occasions and to maintain constant exercise levels throughout the experimental period, where appropriate. Inter-day assessments of performance were completed as near to the same time of day as possible (± 1 hour). Individuals with any history of orthopaedic or muscular pathology of the involved musculature were excluded from participating in this study. All assessment protocols were approved by the University of Wales, Bangor, Human Performance Ethics Review Committee.

4.2. Instructions to subjects.

All instructions to subjects were given via standardised written instruction cards. Subjects were not given feedback of results until the prescribed number of trials was complete. The same test administrator performed all measurements.

4.3. Application of surface electrodes for EMG.

Estimates of EMD responses were assessed by recording the electromyographic activity (EMG) within the biceps femoris muscle during each PFv contraction. Prior to subject orientation on the dynamometer, rigorous skin preparation including shaving, abradement

(using fine sand paper) and de-greasing (using an alcohol swab) of the skin over the belly of the m.biceps femoris was undertaken. Two self-adhesive bi-polar surface electrodes (AgCl) were placed longitudinally over the mid-line of the distal belly of the m. biceps femoris muscle (to enable the greatest amplitude of signal to be detected) at a fixed inter-electrode distance of 4 cm apart along the line between the ischial tuberosity and the lateral epicondyle of the femur on both the preferred and non-preferred limb (chapters 6.0, 7.1, 7.2 and 8.0 and on the preferred limb only (chapter 5.0). A third or 'reference' electrode was also placed lateral to the m. biceps femoris parallel to the gap between the two detector electrodes.

Skin preparation quality was assessed using a impedance meter with a resistance of less than 5 KΩ being acceptable. The assessment of skin preparation quality using an impedence meter is illustrated in figure 4.1. A standard separation of 10 mm was followed to avoid 'crosstalk' between electrodes (Basmajian and De Luca, 1985). To facilitate signal stability across inter-trial assessments electrode placement was standardised across days, where appropriate, by means of mapping (using acetate paper) and measuring the position relative to anatomical landmarks.

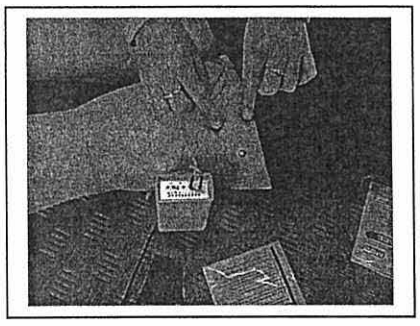


Figure 4.1. The assessment of skin preparation quality using an impedance meter between three bi-polar surface electrodes applied to the knee flexor musculature.

4.4. Subject orientation.

At each assessment occasion, subjects were placed in a prone position on the dynamometer with both lower limbs individually secured using thigh, pelvic and torso straps to limit

extraneous body movements. The lateral femoral epicondyle was used as a bony landmark for the axis of rotation at the knee joint. The load cells of the purpose built dynamometer (RDP Electronics Ltd., Wolverhampton, U.K.: range 1000N) were 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., U.K; 1902 medically isolated programmable amplification/filter [zero amplification] 1401 plus laboratory I/O interface [12-bit ADC sampling frequency 4kHz) and is illustrated in figure 4.2.

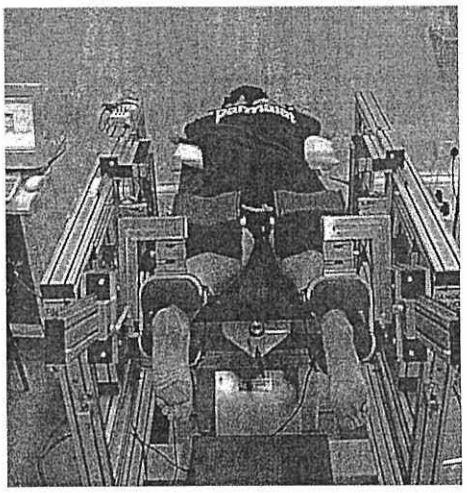


Figure 4.2. Purpose built dynamometer (Gleeson et al., 2000) illustrating the standardised subject positioning for each test occasion. (Note: The right leg is aligned for assessment in this example).

The lever-arm length between the ankle cuff and the axis of rotation was standardised for each participant during inter-day trials, where appropriate. A knee flexion angle of 25° (0.44 rad) was maintained for both knees throughout testing. This has been previously identified as a functionally relevant angle at which key ligamentous structures are under the greatest mechanical strain (Beynnon and Johnson, 1996). The preferred limb was identified as the limb chosen by the subject to kick a soccer ball with maximum force (Klopfer and Greij, 1988; Kubiak, Whitman and Johnson, 1987). All subjects were given verbal encouragement, by the same assessor, for all trials.

4.5. Indices of neuromuscular performance.

4.5.1. PFv warm-up.

Prior to testing, participants were taken through a task specific warm-up for peak force consisting of 2 contractions at 50% of their perceived peak force, followed by 2 efforts at 75% and finally 2 efforts at 95% of their perceived peak volitional force.

4.5.2. Indices of volitional peak force (PFv).

Volitional static peak force (PF_V) was described as the greatest response from three (chapter 5.0) and two (chapters 6.0, 7.1, 7.2 and 8.0) intra-trial replicates of maximal isometric muscle activation of the knee flexor musculature. Peak force was selected as a marker of physiological capability associated with voluntary forceful activation of the knee joint extensors inherent in many activities of daily living.

After a verbal warning, an auditory signal was delivered to the participant randomly within 1-4 seconds. On receipt of the signal, the participant attempted to flex the knee joint as rapidly and forcefully as possible against the immovable restraint offered by the apparatus. Maximal peak force was maintained for 3 seconds, following which another auditory signal was delivered to the participant cueing conscious withdrawal of muscle activation and associated neuromuscular relaxation as quickly as possible. An example of a data recording for PF_V is illustrated in figure 4.3. Data was recorded as the highest gravity-corrected force observed during the single muscle activation. Intra-trial PF_V replicates were each separated by at least 10 seconds to enable neuromuscular recovery (Moore and Kukulka, 1991). Commercially available software (Spike 2 software, version 2.01, Cambridge Electronic Design Ltd., U.K.) was used for all volitional neuromuscular data capture and interpretation.

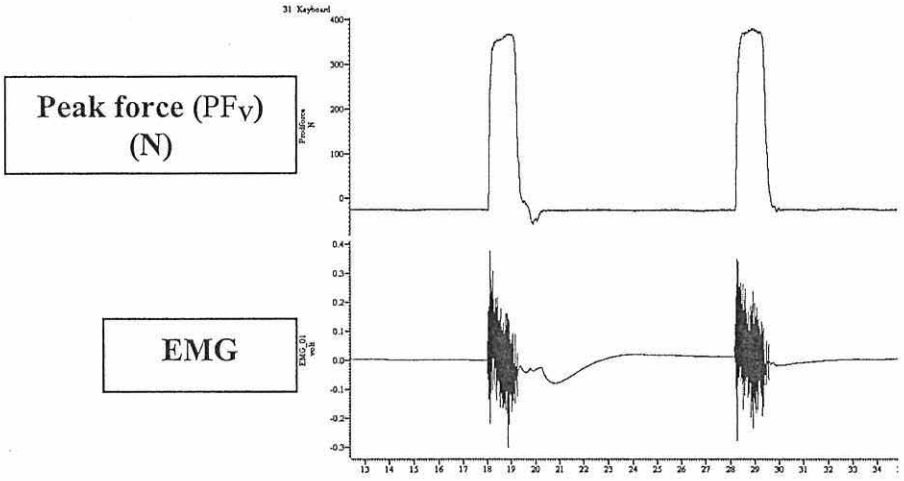


Figure 4.3. An example of a data recording for PFv using Spike 2 software, comprising of two maximal discontinuous isometric efforts (EMG activity recorded).

The technical error associated with force transducers was estimated using ordinary statistical procedures and described by the 95% confidence limits that are likely to include the true force score. Estimates based on the background electrical noise of both the right and left force transducers, calculated using 2-second time periods from within a sample of 40 randomly selected data files, showed that the mean (± SD) 95% confidence limit was 1.06 (± 0.09) N (Minshull, 2003 [unpublished data]).

4.5.3. Apparatus calibration.

The testing equipment was calibrated for precision and linearity of response at several time periods across experiments. Dynamometer forces were recorded during the application of standard forces (10 N, 50 N, 100 N, 500 N) to accommodate the range of biological forces likely to be experienced by the transducer and were compared with expected resultant mechanical forces.

4.5.4 Indices of volitional electromechanical delay.

EMD is defined as the time delay between the onset of muscle activity and the onset of force generation (Norman and Komi, 1979). The EMD is determined using EMG techniques, by

the time taken for the contractile component to stretch the series elastic component of the muscle (Winter and Brookes, 1991).

Volitional EMD (EMDv) was calculated for each PFv. The mean response associated with the two/three intra-trial replicates was used to define EMD_V. The period in time at which the onset of muscular electrical activity occurred was identified visually as the first deviation of the signal away from the background noise and the isoelectric line associated with muscle relaxation. Onset of muscle force was defined as the first point in time at which the force record exceeded consistently the 95% confidence limits associated with the electrical noise amplitude of the load cells. An example of an EMG recording to determine EMD is illustrated in figure 4.4.

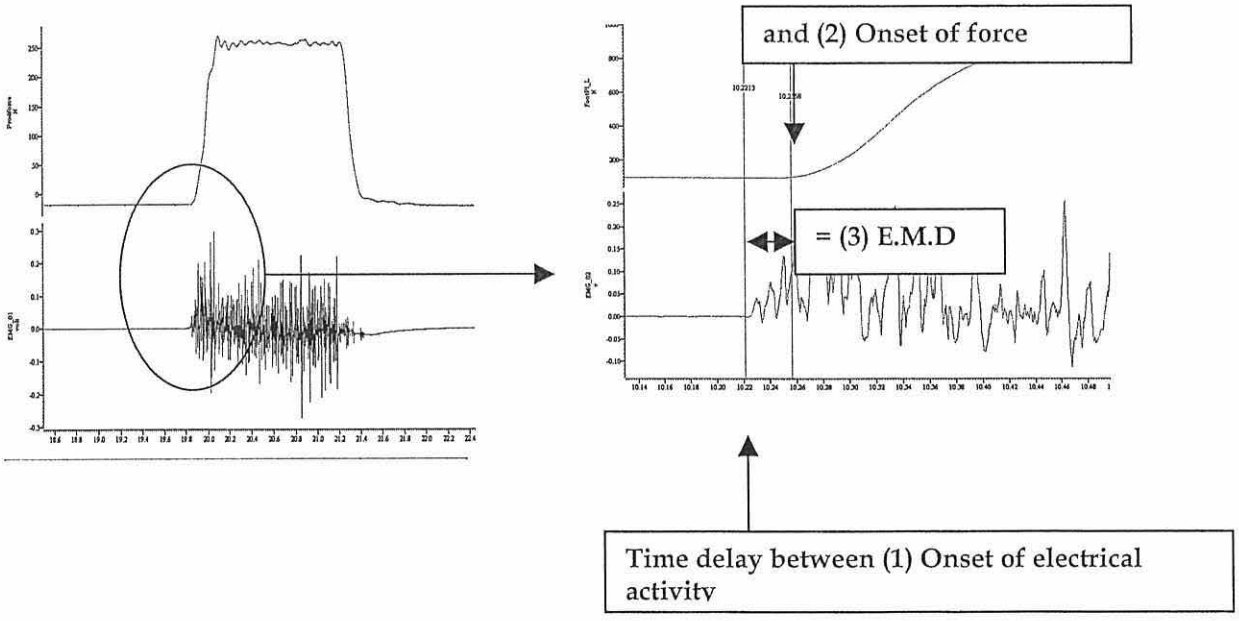


Figure 4.4. An example of an EMG recording to illustrate the determination of volitional electro mechanical delay (EMDv).

4.5.5. Indices of magnetically evoked electromechanical delay.

Voluntary movement control requires afferent and efferent processing time. It is well established that practice of a specific skill will lead to improved efficiency within a given pathway of response, illustrated by a reduced movement time (MT). Evoked response via

magneto stimulation assists the sport scientist by accessing such pathways without voluntary involvement of the individual. Hence, a true reflection of pathway efficiency, and an achievable target response time.

The sciatic nerve root was stimulated by means of a double wound coil (120 mm), powered by a Magstim 200 stimulator (Magstim Co. Ltd., Whitland, Dyfed, Wales) to determine the static neuromuscular performance capacity of the knee flexors associated with a magnetically evoked twitch. The centre of the coil was placed initially in a position 20 mm – 40 mm lateral to the fifth lumbar vertebra on the involved side. Figure 4.5 illustrates the positioning of the coil during magnetic stimulation for an evoked EMD response.

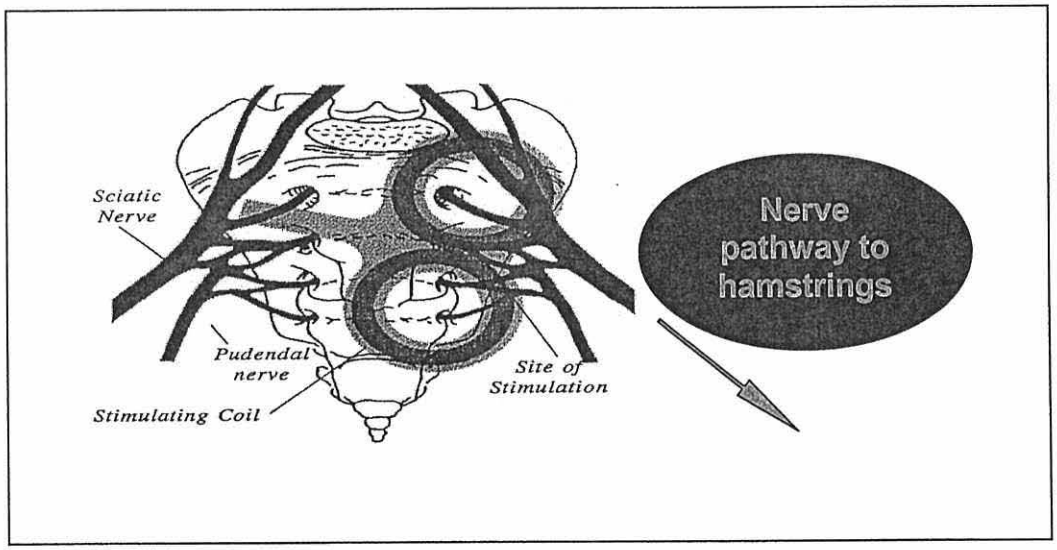


Figure 4.5. The nerve pathway used for magnetically evoked EMD in the knee flexor musculature.

The location of the optimum site for stimulation was denoted by the compound muscle action potential [CMAP] that had the largest amplitude. This was identified by a subsequent procedure in which small positional changes of the coil were made in response to the effects of a series of single stimulations. This optimised coil position was maintained manually throughout the remainder of the test. The standardised laboratory set up during magnetic stimulation is demonstrated in figures 4.6 and 4.7.

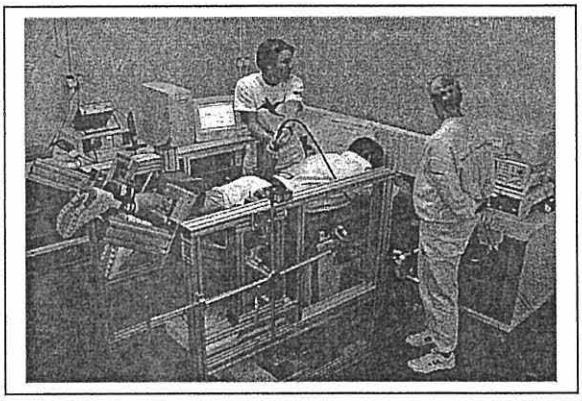


Figure 4.6. The standardised laboratory set up during the assessment of magnetically evoked EMD.

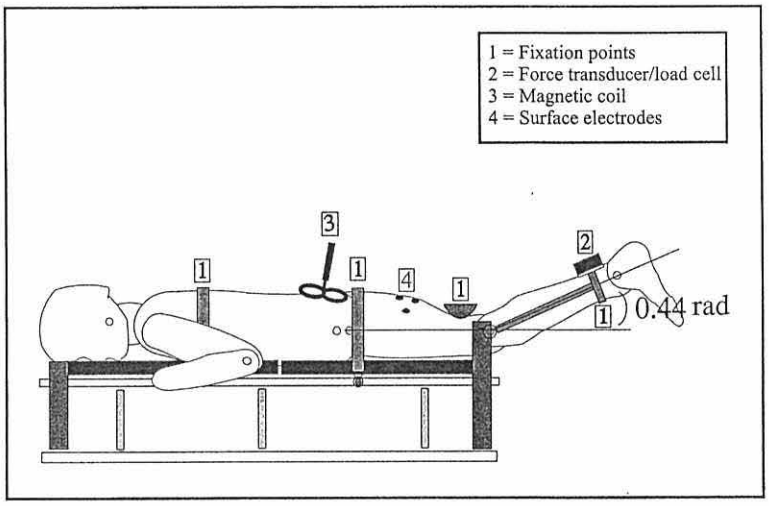


Figure 4.7. Participant and dynamometer orientation for the assessment of magnetically evoked EMD in the knee flexor musculature.

The optimisation procedure for the site of stimulation was repeated on each test occasion in which participants were re-secured to the dynamometer. A sequence of seven stimulations of increasing intensity was performed to identify supramaximal stimulation. This was defined by a plateauing of the CMAP amplitude. The sequence comprised stimuli from 40 to 100%, increased at 10% increments, of the Magstim 200's maximal capacity output to familiarise the subject with the 'feeling' of each stimulation intensity. Sequential stimulations throughout the experimental period were separated by at least 10 seconds to enable neuromuscular recovery (Moore and Kukulka, 1991).

The beginning of the plateau of the CMAP was defined as the intensity at which no more than a 5% increase in CMAP peak amplitude was observed despite a 10% increases in the intensity

of stimulation. This was verified by contemporaneous visual inspection of the data. Supramaximal stimulation was confirmed in12 subjects during a repeated exposure to maximal volitional activation (chapter 6.0), and 6 subjects during conditions of exercise induced muscle damage (chapters 7.1 and 7.2).

Subsequent assessments of magnetically evoked neuromuscular performance in all participants were conducted at a stimulation intensity that was associated with either supramaximal amplitudes of CMAP or peak amplitudes of CMAP limited only by the technological performance of the stimulation system. Estimates of magnetically evoked neuromuscular performance were calculated for each stimulation at the highest intensity. Software (Signal, version 1.81, Cambridge Electronic Design Ltd., U.K.) was used to interrogate the force and electromyographic data records of magnetically evoked muscle activation. The time delay between the onset of electrical activity to the onset of force was calculated for each replicate. The mean response associated with the two intra-trial replicates was used to describe the index of evoked electromechanical delay (EMD_E). Onset of electrical activity was visually identified as the first deviation of the isoelectric line away from the background noise observed during muscle relaxation (or threshold value). Static peak twitch force (P_TF_E) was described as the mean response associated with the two intra-trial replicates where the highest force was recorded. The visual observation of both (EMD_E) and (P_TF_E) using Signal software (version 1.81 CED Ltd) are summarised in figure 4.8.

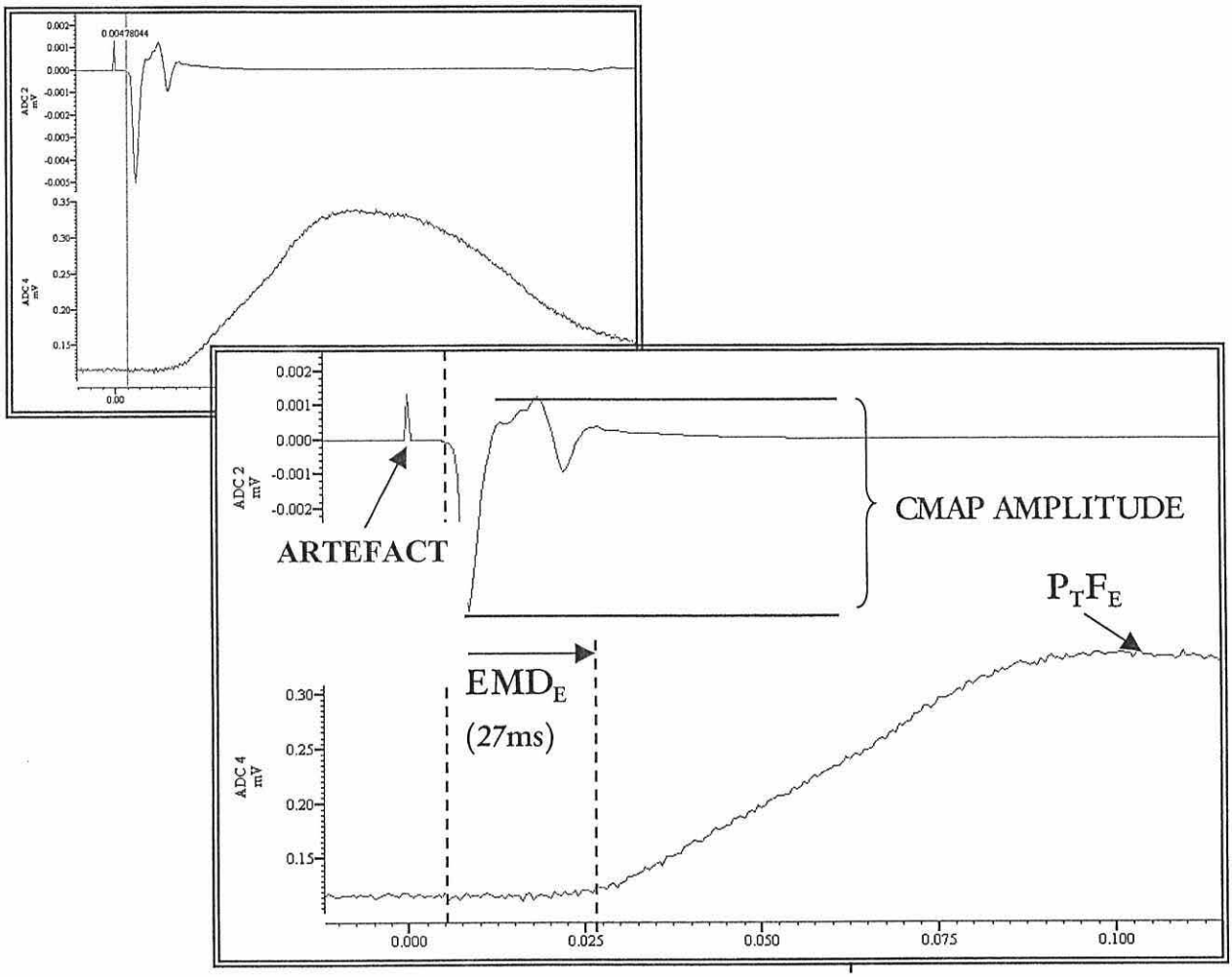


Figure 4.8. An example of a data recording showing; upper trace: force and EMG associated with a single magnetic stimulus; lower trace: magnification of muscle activation to show representative calculation of indices of magnetically evoked neuromuscular performance.

4.6. Indices of dynamic sensorimotor performance.

Dynamic sensorimotor performance [SMP] is defined as "*the ability to regulate volitional force*" and is expressed as the discrepancy between the blinded attainment of a prescribed force (a given percentage of daily volitional peak force, PFv) and the subsequent reproduction of this force following a standardised delay (Gleeson et al. 1997).

Two types of sensorimotor performance task have been recently designed. Type I SMP task is time restricted to simulate quick movements in reaction to unexpected conflicts in movement control for joint preservation within a limited time frame. Type II SMP is a slow, self-regulated movement, aimed to mimic the process of muscle re-education following

disruption to the neuromuscular system during injury or surgical intervention. The latter type of movement utilises feedback from pre-learned strategies of joint protection.

The accuracy of sensorimotor performance during both types of task is described in terms of both constant and variable error [CE, VE]. CE describes the bias around a target or the average magnitude of the response and measures the average direction of the errors (a tendency to over or undershoot a target). The CE does not consider the amount of scatter, variability, or inconsistency in performing such movements. Variable error describes the subject's inconsistency in responding. VE is therefore the variability of the subject around the mean constant error response. A change in VE would indicate little bias; neither over nor undershooting the target, since it is a measure of spread about the subject's own average (but the accuracy of the response was/may be compromised). If a subject always responds very consistently, then the VE will tend to be small. If the subject always receives the same score, even though it is not the correct score, then the VE will be zero (Schmidt, 1988).

Each assessment occasion included a familiarisation session, whereby each subject was blindly familiarised with 50% of his/her daily PFv; described as the target force. This is in accordance with recommendations by Pincivero et al. (2000) that sub-maximal isometric exercise should be based on a relative level within the same day due to normal biological variation. The type of SMP task contraction was 'taught' via subjects producing an isometric resistance in the experimental limb, against the force transducer, which was observed on the computer monitor by the test administrator. Verbal feedback was given as to the nature of the achieved force, for example 'too high, too low'. This process was repeated until the subject could accurately replicate 70 % of all efforts within ± 10 N (68 % confidence limits) of the daily target (in agreement with preliminary pilot study data [unpublished observations]).

For type I SMP assessment subjects were instructed to contract the involved musculature (typical mean rate of response: ~ 1000 Nm.s –1) to the designated target force within a 1 second time frame upon hearing an auditory signal. Subjects were instructed to fully relax the involved musculature between each contraction. This process was assessed four times within each trial. An example of a data recording during the familiarisation period for the type I SMP assessment task is illustrated in figure 4.9.

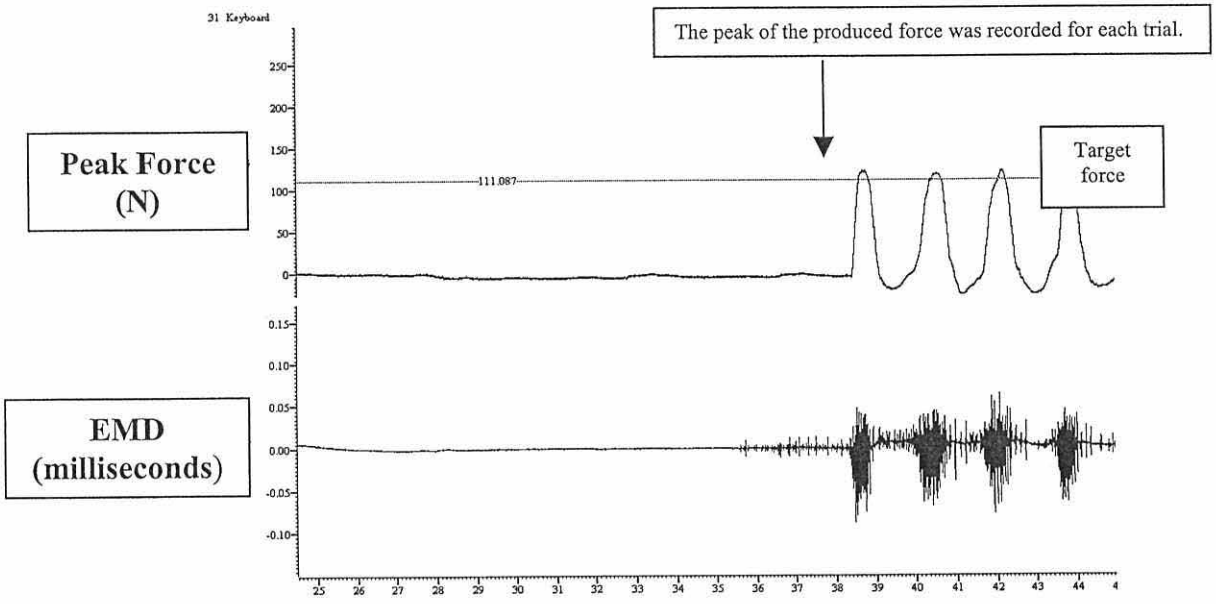


Figure 4.9. An example of a data recording during the familiarisation period for the type I SMP assessment task.

Type II SMP was also 'taught' at the start of each assessment occasion. The subject was verbally instructed to produce the daily target value over a longer time frame (5 seconds with a typical group force response of 200 N.s –1). The subject indicated that the desired force had been perceived and produced by fully relaxing the knee flexor musculature. This process was assessed twice within each data capture for chapter 5.0 and three times for chapters 6.0, 7.1, 7.2 and 8.0. An example of a data recording during both the familiarisation and assessment periods for the type II SMP assessment task is illustrated in figures 4.10 and 4.11, respectively.

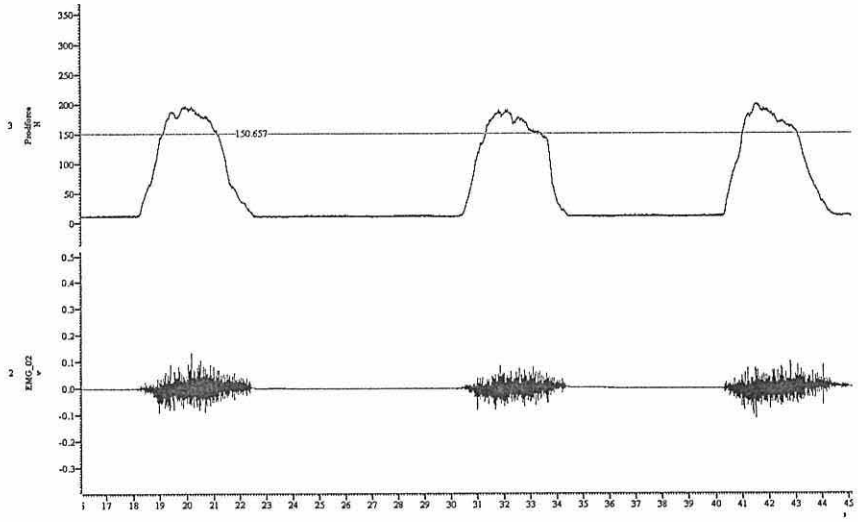


Figure 4.10. An example of a data recording during the familiarisation period for the type II SMP assessment task.

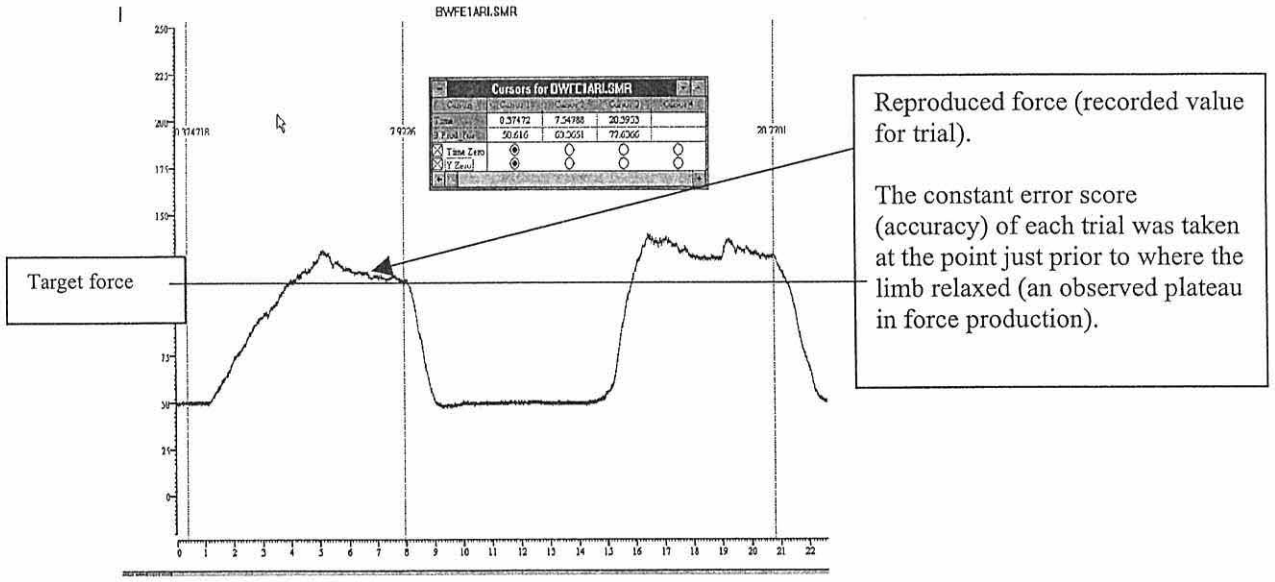


Figure 4.11. An example of a data recording during the assessment period for the type II SMP assessment task.

The above tasks were combined within each data capture for chapter 5.0 in the format of one type II SMP effort followed by two type I SMP efforts, as illustrated in figure 4.12. This procedure was repeated twice within each assessment series. All subjects were blinded to the torque values that they were generating.

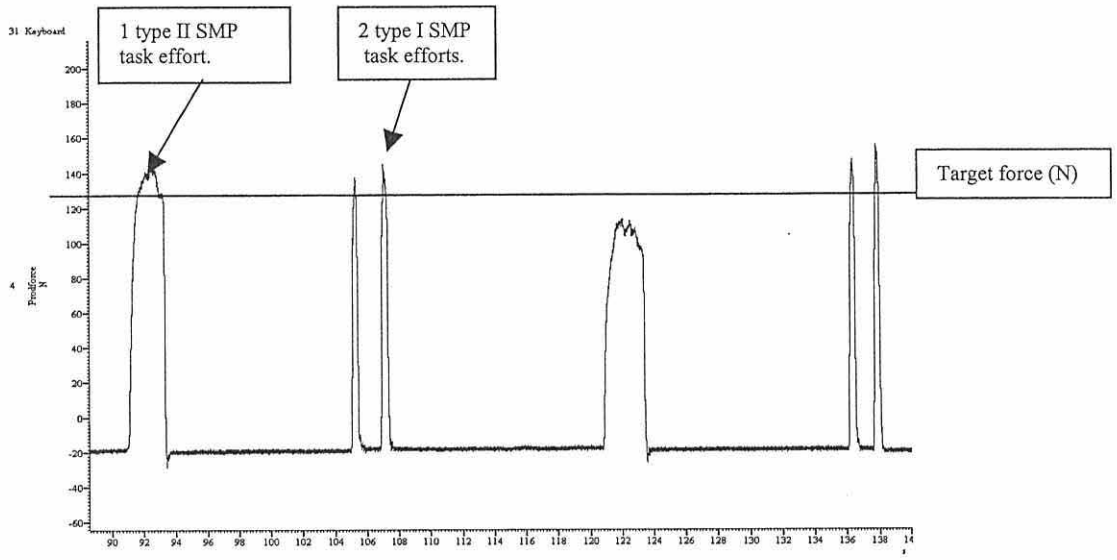


Figure 4.12. An example of the combined assessment procedure for one type II SMP assessment effort followed by two type I SMP assessment efforts, which was repeated twice within each trial (chapter 5.0 only).

The overall error is expressed as the discrepancy between the force produced by the subject and the calculated target force. The raw score for each effort is converted to a percentage score relative to the daily target. For example a daily PFv value of 300 N.s –1 would set the target force at 150 N.s –1 (50%PFv). If the actual reproduced force for the given task was 160 N.s –1, the discrepancy would be +10 N (160 N.s –1 - 150 N.s –1). This score is then transformed to a percentage of the target force by dividing the discrepancy by the target score (10/150 = 0.066 * 100 = 6.6). In this example, the overall error would be +7 %.

The overall error score was calculated for each assessment occasion as the mean of 4 type I SMP trials, or the mean of 2 type II SMP trials. This value was recorded as either positive (overshooting [under-estimation of perceived force]) or negative (under-shooting [over estimation of perceived force]).

Following the initial experiment (chapter 5.0) the assessment of the two types of SMP task was conducted separately. This alleviated any potential conflicts in motor skill strategies given the differences in time restrictions between the two types of test. 5 simultaneous trials

of type I SMP and 3 simultaneous trials of type II SMP were performed at each assessment for chapters 6.0, 7.1, 7.2 and 8.0.

The specific number of exercise sessions, bouts and their duration were chosen to be commensurate with the aims of a given study and are described therein. Also the methods to assess the physiological descriptives of exercise induced muscle damage are described within the relevant chapter (chapters 7.1 and 7.2). The experimental design sensitivity associated with all subsequent data analyses was expected to offer power of 0.8 and above based on a priori estimates of performance change in pilot studies and the scientific literature.

5.0. Reproducibility and single-measurement reliability of indices of dynamic sensorimotor performance.

5.1. Introduction.

Joint position sense and joint kinaesthesia are two measurement techniques that have been traditionally used to measure joint 'proprioception'. However there has been very little validation or comparison of these methods (Beynnon et al., 2000). Differences in both experimental design and data analysis of traditional techniques of proprioceptive function make comparison of results between previous studies difficult.

Drouin, Houghlum, Perrin and Gansneder (2003) examined the relationship between joint position sense and a functional hop test in athletes (n = 20 collegiate lacrosse players) and non-athletes (n=20). The authors concluded that "*a joint repositioning task in both a weight bearing and non-weight bearing position did not assess the complex integration of peripheral feedback and subsequent afferent responses necessary for the performance of dynamic lower extremity activities.*" It is therefore possible that the traditional method of knee joint sensorimotor performance assessment by means of a joint angle reproduction task may not actually reflect the challenges to knee joint stability witnessed during functional movement patterns.

Sensorimotor performance is often considered at post-operative assessment periods following ACL reconstruction. However this process often involves 'guess-work' between the surgeon, physiotherapist and patient in judging when it is 'safe' for an athlete to return to competitive or contact sporting situations. In most cases the patient has to rely on how the knee 'feels' in terms of functional capability. A typical 'assessment' of sensorimotor function may merely involve a subjective response to questions such as "does the knee feel like it will give way?" Or, "do you feel like you could return to your sport without the knee feeling unstable?"

Dynamic sensorimotor assessment may provide a practical and more importantly quantifiable way of assessing neuromuscular and sensorimotor capability by means of force regulation awareness; and has received limited previous scientific scrutiny. This method of assessment may eradicate the use of 'guess-work' by providing a scientifically based, safe return to sport that may ultimately prevent re-injury. Often the only means of judging whether a limb is recovered following an injury is by comparison to the non-injured or contra-lateral limb. This is because it is not common practice within professional sports to assess neuromuscular and sensorimotor performance prior to dysfunction.

Gleeson et al. (1998) examined dynamic sensorimotor assessment alongside several neuromuscular performance indices in the ACL deficient population prior to, and following reconstructive surgery. Nine male athletes (age 25 ±3.5 years) with unilateral complete ACL rupture (arthroscopically verified) were assessed at baseline and on three separate testing occasions during the acute phase of a standardised rehabilitation programme (2 weeks prior to surgery and 6, 8 and 10 weeks post surgery). The overall results for Gleeson et al. (1998) are illustrated in figure 5.1 and represent a significant decrease in force error % [FE%] scores in the injured leg between pre and 6, 8 and 10 weeks post surgery. This equated to a 59 % (22 ± 6% pre-surgery versus 9 ± 5 % post-surgery) decrease in FE% (underestimation of volitional force). Scores in the control leg remained constant across test occasions (8± 3%). Improvements in the involved limb post-surgery were attributed to a sub-optimal dose-response relationship in the knee flexors during the early rehabilitation process. However, the single-measurement reliability of this method of assessment was not assessed prior to its application within a clinical population.

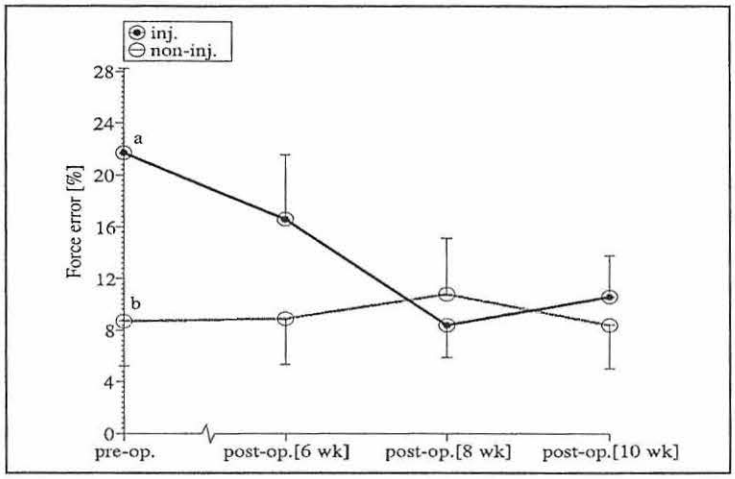


Figure 5.1. The results for type II dynamic sensorimotor performance task assessments in patients prior to and following ACL reconstructive surgery (Gleeson et al., 1998).

Such significant findings within a relatively small and injured sample group, supports the need for further research to determine whether or not subtle differences in muscle re-education can be detected in the 'healthy' population. An assessment protocol that is sensitive enough to detect true differences between the sensorimotor performance of individuals within a group may prove useful in monitoring the effects of an intervention strategy such as exercise stress or training on sensorimotor function.

An important challenge in the design of a scientifically acceptable assessment protocol of dynamic sensorimotor performance is that it possesses sufficient reliability, reproducibility and ultimately sensitivity (i.e. the ability to detect small changes in an individual's sensorimotor performance, or relative changes in an individual's sensorimotor performance within a sub-sample). This will ensure that the test of performance offers at least a minimal level of measurement precision commensurate with its intended use (Gleeson and Mercer, 2002).

Firstly the reproducibility of the index of sensorimotor performance or the measurement error associated with a particular score must be established. The coefficient of variation (V%) may be used to describe the error associated with a score for a given individual · ±V%, or more

accurately ±1.96 ·V% for 68% and 95 % confidence intervals respectively, assuming a normal distribution of performance estimates (Thomas and Nelson, 1990).

Secondly, the single-measurement reliability of types I and II SMP assessment task may be quantified using the intra class correlation co-efficient (R_I). This enables the experimenter to observe the fluctuation of an individual's repeated test scores within the context of sub-sample performance variability. The reliability of repeated tests on the same individual may be estimated and further quantified using the standard error of a single measurement (SEM) in conjunction with R_I and the sub-population standard deviation (Gleeson and Mercer, 2002). This is calculated using the expression SE $\cdot\sqrt{}$ (1-r) where SE is the standard deviation of the sub-sample for the criterion test and r is the reliability coefficient for the criterion test. The SEM may also be expressed as a percentage relative to the mean sub-sample score according to the expression (SEM/mean) · 100% and may control for heterogeneity across conditions. Thus, the quantification of a 'window of stability' for the performance of an individual may offer an indication about the minimum number of replicates required to obtain an acceptable level of measurement error (Gleeson and Mercer, 1996).

The reproducibility of two types of dynamic sensorimotor performance tasks on a single assessment opportunity, alongside within-day and between-day measures, shall be investigated to assess their use as assessment and clinical research tools. Within-session and within-day measures may represent a 'real-life' clinical situation whereby there is a single opportunity or day for assessment and the biological fluctuation in performance within the same patient is considered. Measurement reproducibility associated with between-day assessments may reflect the situation for example, when a patient returns periodically for orthopaedic follow-up prior to and following ACL surgery. This type of measurement reproducibility assessment enables the assessor to know how precisely it is possible to gauge

the effect a particular intervention strategy, such as a physiotherapy program or rehabilitative training regime.

The variability associated with dynamic sensorimotor performance in terms of both constant error (CE) and variable error (VE) is illustrated using type I SMP task in figure 5.2 as an example. Constant error refers to the accuracy of the force production as either above or below the target force (+ or -); whereas variable error describes the consistency of the response (i.e., hitting the same target each time, although inaccurate would lead to a zero variable error score).

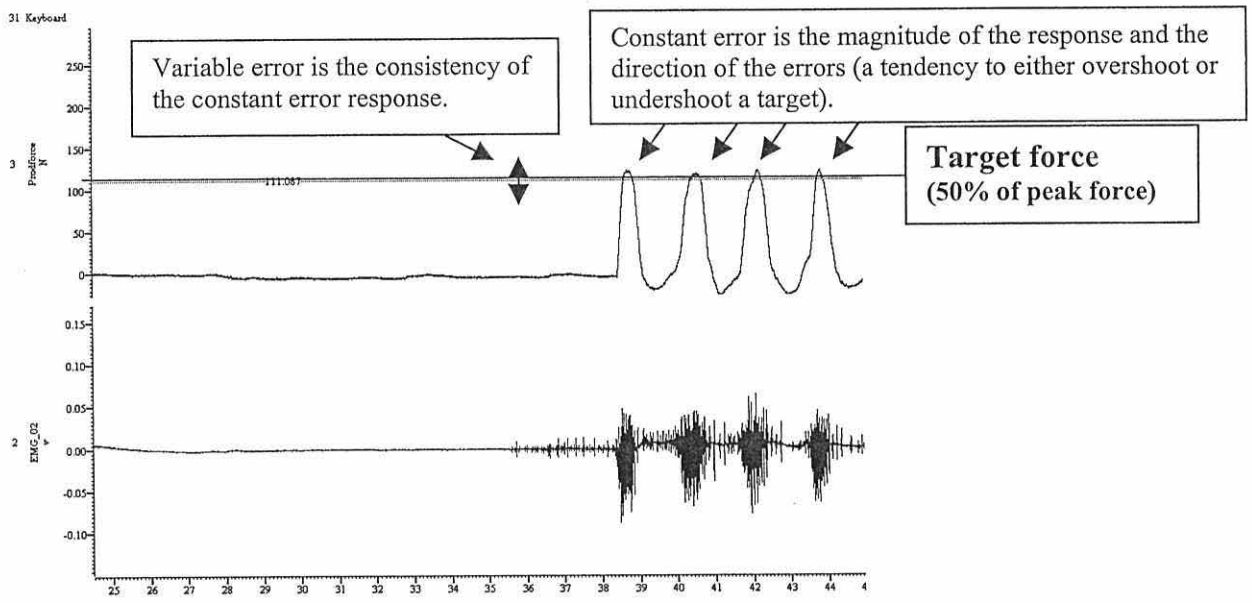


Figure 5.2. An example of a data record to illustrate the variability associated with sensorimotor performance in terms of both constant and variable error.

The variability of SMP scores for each individual in terms of constant error across the selected periods of assessment (within-session, within-day or between-days) may be described using coefficient of variation scores (V%; SD/mean*100%) corrected for small sample bias (Sokal and Rohlf, 1981).

The aim of this study was to document the reproducibility and single-measurement reliability of indices of dynamic sensorimotor performance (type I and type II tasks) associated with within-session, within-day and between-day measurements of both constant and variable error in active male and female volunteers.

5.2. Methods.

Subjects.

Seven adult males (age: 29.6 ±10.4 years; height 1.78 ±0.04 m; mass 77.0 ±7.7 kg [mean ± SD]) and nine females (age 25.2 ±4.2 years; height 1.69 ±0.08 m; mass 62.8 ±8.1 kg) gave their informed consent to participate in this study. All subjects were physically active, healthy volunteers. Subjects were instructed to keep their involvement in sporting activities consistent throughout the experimental period, wherever possible.

Experimental protocol.

Following habituation to procedures, each participant completed three within-day trials, separated by 20 minutes. This process was repeated on three separate days, with at least 3 days between within-day test occasions. Figure 5.3 illustrates the testing protocol for each test occasion. The experimental protocols for the assessment of each index of neuromuscular and sensorimotor performance capacity have been detailed previously (chapter 4.0, section 4.6).

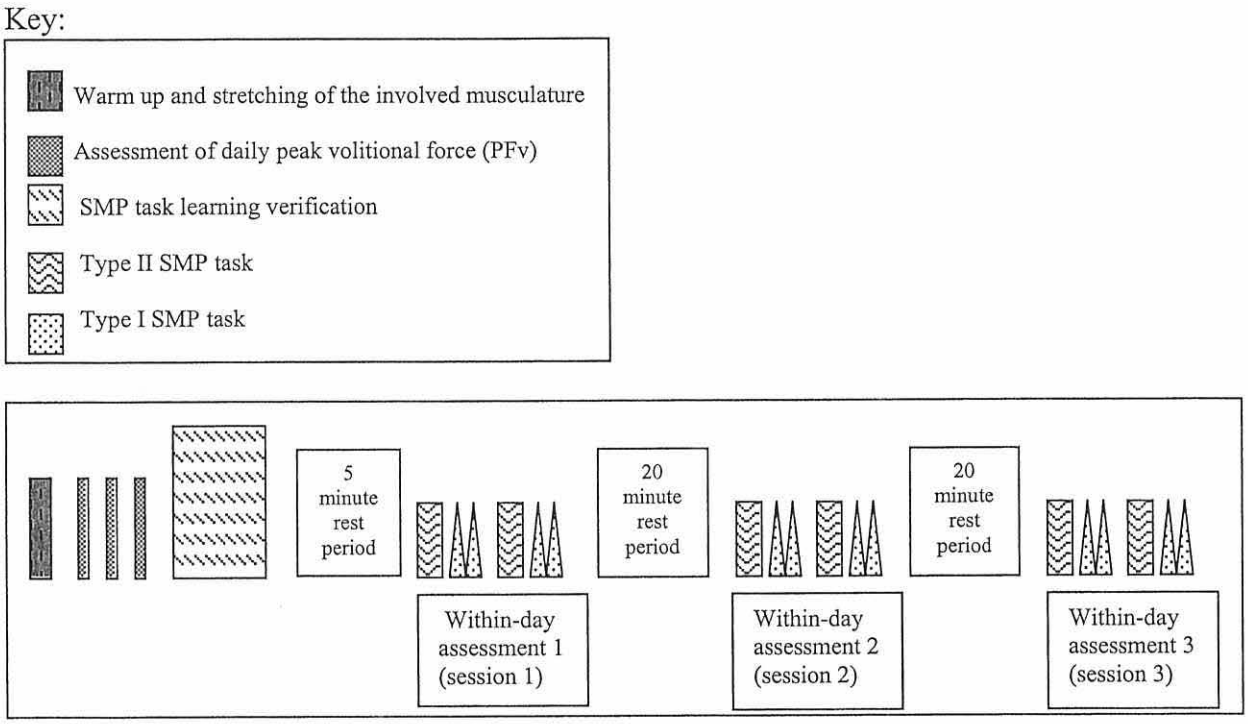


Figure 5.3. Neuromuscular and sensorimotor assessment protocol for the 3 within-day trials. This protocol was repeated over three between-day occasions. There were 9 total within-session occasions (3 sessions per day over three separate days).

Statistical analyses.

Constant error was used to describe the accuracy of sensorimotor performance during each of the two forms of task and variable error was assessed to describe the consistency of the constant error response (Schmidt, 1988). These indices were described using ordinary statistical procedures (mean ± SD). Coefficient of variation (V%) corrected for small sample bias (Sokal and Rohlf, 1981) was used to assess reproducibility separately across estimates derived from the 3 within-day trials and the 3 between-day trials. The latter index was calculated according to the expression (SD/mean)· (1+ [¼ N]) where N is the number of trials.

The reproducibility (V%) of CE and VE scores associated with each of the SMP tasks (type I SMP task, type II SMP task) across the periods of assessment were compared using separate one way ANOVAs for the period of assessment [within-session, within-day, between-day]) with repeated measures.

Intra class correlation co-efficients (R_I) were computed to describe the single-measurement reliability of each type of dynamic sensorimotor performance task (Winer, 1981). Standard error of a single measurement (SEM %) (95% confidence limits, computed as a percentage of the group mean score) was calculated for the chosen indicator of performance (Feldt, 1990).

An a priori alpha level of 0.05 was applied in all statistical procedures. In the event of a significant Mauchly's test of sphericity, the violation to the assumption of ANOVA was corrected for via the Greenhouse-Geisser adjustment of the critical F-value, as indicated by (GG). SPSS/win (V 9.0) was used to perform all statistical procedures (SPSS Inc., 2002).

5.3. Results.

Table 5.1 illustrates the group mean scores for constant error (CE) in relation to the target force (50% PFv) (± SD) during types I and II sensorimotor performance assessment tasks over 9 within-session, 3 within-day and 3 between-day trials.

	Type I SMP task (% of the target force).			**Type II SMP task (% of the target force).**		
	Day 1	**Day 2**	**Day 3**	**Day 1**	**Day 2**	**Day 3**
	CE ± SD	CE ± SD	CE ± SD	CE ± SD	CE ± SD	CE ± SD
Within-day assessment 1	Session 1.	Session 4.	Session 7.	Session 1.	Session 4.	Session 7.
a	45.2 (27.3)	38.7 (28.4)	29.1 (24.6)	a 0 (14.0)	0 (13.1)	-1.3 (12.3)
b	50.1 (23.6)	43.0 (33.1)	31.0 (25.5)	b 3.3 (19.6)	1.9 (22.0)	-2.1 (15.0)
c	40.4 (29.7)	30.2 (30.7)	27.8 (28.8)			
d	49.1 (24.8)	35.8 (29.7)	27.3 (24.4)			
Within-day assessment 2	Session 2.	Session 5.	Session 8.	Session 2.	Session 5.	Session 8.
a	49.5 (15.0)	39.5 (21.1)	40.7 (21.6)	a 11.7 (18.8)	10.6 (16.3)	9.1 (17.1)
b	51.2 (21.8)	42.9 (28.4)	44.3 (22.3)	b 12.3 (21.9)	11.8 (22.0)	12.7 (18.3)
c	42.3 (26.6)	36.0 (32.7)	32.4 (23.0)			
d	44.7 (25.0)	39.5 (30.4)	34.7 (22.7)			
Within-day Assessment 3	Session 3.	Session 6.	Session 9.	Session 3.	Session 6.	Session 9.
a	34.5 (14.8)	31.3 (29.0)	32.6 (20.1)	a 5.8 (16.7)	10.2 (17.5)	3.7 (14.7)
b	42.5 (13.9)	31.5 (26.3)	29.0 (25.6)	b 6.6 (18.1)	10.6 (27.6)	6.7 (17.4)
c	34.1 (20.8)	25.5 (30.8)	22.5 (20.0)			
d	34.5 (17.6)	32.3 (25.0)	22.9 (17.4)			

Table 5.1. Descriptive statistics of single estimates of constant error expressed as a % of the target (mean ± SD) for within-session, within-day and between-day trials for types I and II SMP assessment tasks. Note: a, b, c, d corresponds to the order of the serial measurements within each session (4 trials for type I SMP and 2 trials for type II SMP tasks).

Results from separate ANOVAs suggested that no significant learning trends were observed across the three data collection occasions for each type of SMP task. This suggests that the practice period on the familiarisation day and prior to each assessment occasion was sufficient for subject's to have become accommodated to both types of SMP assessment techniques.

An overall group mean constant error bias of 36.8 ±17.4 % and 6.3 ±11.1 % (relative to the target) was observed for types I and II SMP assessment tasks, respectively and is illustrated in figures 5.4 and 5.5. This demonstrates an overall positive bias (above the target) or a tendency

for subjects to underestimate the amount of force produced in the experimental limb during both types of SMP task.

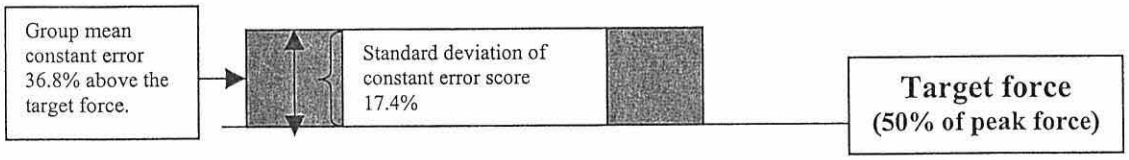


Figure 5.4 Group mean constant error scores during type I SMP assessment task.

The magnitude of the target over-estimation appeared to be greater during the type I SMP task which could be explained by the fact that the type I task requires force reproduction within a restricted time frame (each force replication occurred over a one second time period). The fact that the average response was 37 % above the target may demonstrate the emergency capacity of the high threshold motor units in order to protect the knee joint during an open-loop skill, when there is no opportunity for the integration of feedback.

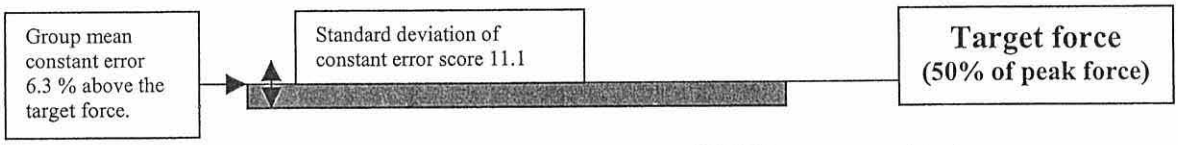


Figure 5.5. Group mean constant error scores during the type II SMP assessment task.

The accuracy of the group mean score for constant error during the type II SMP task was 6 % of the target force. This may be explained by the fact that each subject had a greater period of time to perceive and therefore correct the force output by utilising feedback (closed-loop skill) between efforts for the slower, type II SMP task.

Table 5.2 illustrates the reproducibility of group mean CE scores expressed as a V% (corrected for a small sample size) for types I and II SMP assessment tasks.

Type I SMP assessment task (% of the target).		Type II SMP assessment task (% of the target).
	Group mean constant error scores expressed as a V% [corrected for a small sample size] (±SD)	Group mean constant error scores expressed as a V% [corrected for a small sample size] (±SD)
Within-session [ws]	39.9 (19.9)	62.4 (14.9)
Within-day [wd]	45.1 (16.2)	79.4 (26.9)
Between-day [bd]	51.0 (20.5)	73.9 (23.9)

Table 5.2. The reproducibility of group mean constant error scores expressed as a V% (corrected for a small sample size, Sokal and Rohlf, 1981) for types I and II SMP assessment tasks.

The group mean V% scores for constant error corrected for a small sample size (Sokal and Rohlf, 1981) were 39.9 ±19.9%, 45.1±16.2% and 51.0±20.5 % for within-session, within-day and between-day assessments of the type I SMP task, respectively. Repeated measures ANOVA on the group mean V% scores (corrected for small sample size) revealed no significant difference between assessment occasions [ws, wd, bd]). Therefore the relative amount of constant error was consistent between the chosen time periods for assessment.

Group mean V% scores (corrected for a small sample size, Sokal and Rohlf, 1981) were 62.4 ±14.9%, 79.4 ±26.9% and 73.9 ±23.9 % for within-session, within-day and between-day assessments of the type II SMP task, respectively. Repeated measures ANOVA on the mean V% scores (corrected for small sample size) for the type II SMP assessment task revealed a significant difference between assessment occasions [ws, wd, bd] ($F_{[1, 15]} = 12.6$, $p<0.003$). This suggests that a change in the reproducibility of sensorimotor performance during the type II SMP assessment task was observed across the three periods of data acquisition. Constant error was greatest during within-day measurements of type II SMP which may have contributed to the significant interaction. The lower observed V% score for within-session

assessments may suggest that sensorimotor assessments within the same testing session may be more reproducible compared to within-day and between-day measures for a given patient. Therefore, a patient may replicate a target force with greater accuracy when the trials take place over a relatively short period of time.

Variable error.

Table 5.2 illustrates the combined group mean scores for variable error (VE) or the consistency of the CE response during types I and II sensorimotor performance assessment tasks over 9 within-session, 3 within-day and 3 between-day trials.

	Type I SMP task (% of target).			**Type II SMP task (% of target).**		
	Day 1	**Day 2**	**Day 3**	**Day 1**	**Day 2**	**Day 3**
	VE ± SD	VE ± SD	VE ± SD	VE ± SD	VE ± SD	VE ± SD
Within-day assessment 1	Session 1 26.6 (2.7)	Session 4 30.7 (2.0)	Session 7 26.0 (2.0)	Session 1 16.9 (4.0)	Session 4 17.7 (6.3)	Session 7 13.7 (1.9)
Within-day assessment 2	Session 2 22.3 (5.2)	Session 5 28.4 (5.0)	Session 8 22.6 (0.6)	Session 2 20.0 (1.5)	Session 5 19.3 (4.1)	Session 8 17.8 (0.9)
Within-day assessment 3	Session 3 16.9 (3.1)	Session 6 28.2 (2.6)	Session 9 20.9 (3.5)	Session 3 17.5 (0.9)	Session 6 22.7 (7.2)	Session 9 16.1 (1.9)

Table 5.3. Descriptive statistics of single estimates of variable error as a % of the target (mean ±SD) for within-session, within-day and between-day trials for type I and II SMP tasks. (4 trials for type I SMP and 2 trials for type II SMP tasks).

An overall group mean variable error bias of 24.7 ±5.0% and 18.0 ±3.8 % (of the target force) was observed for types I and II SMP assessment tasks, respectively. The group mean V% scores for variable error (corrected for a small sample size, Sokal and Rohlf, 1981) were 46.9 ±11.5, 51.6 ±15.2 and 43.4 ±8.2 % for within-session, within-day and between-day assessments of the type I SMP task, respectively. Repeated measures ANOVA on the mean V% scores (corrected for small sample size) for the type I SMP assessment task revealed no significant differences between assessment occasions [ws, wd, bd]. This suggests that there

was no difference in terms of the consistency of sensorimotor response during the type II SMP task for within-session, within-day and between-day assessments.

The group mean V% scores for variable error (corrected for a small sample size, Sokal and Rohlf, 1981) were 52.4 ±22.4, 56.1 ±13.8 and 54.9 ±20.9 % for within-session, within-day and between-day assessments of the type II SMP task, respectively. Repeated measures ANOVA on the mean V% scores (corrected for small sample size) for the type II SMP assessment task revealed no significant difference between assessment occasions [ws, wd, bd]. This suggests that the consistency of sensorimotor response was maintained during the type II SMP task for within-session, within-day and between-day assessments.

Table 5.3 illustrates the group mean intra-class correlation coefficient (R_I) and SEM (%) (95 % confidence intervals, expressed as a percentage of the group mean score) for within-session, within-day and between-day assessment of types I and II SMP assessment tasks.

	Type I SMP task		**Type II SMP task**	
	R_I	**SEM (%)**	**R_I**	**SEM (%)**
Within-session	0.69	56.8	0.43	53.1
Within-day	0.48	70.9	0.11	71.7
Between-day	0.34	65.8	0.36	59.8

Table 5.4. Group mean intra-class correlation coefficient (R_I) and standard error of the measurement (SEM [95% confidence limits]) associated with types I and II SMP assessment tasks.

5.4. Discussion.

Constant error (type I SMP task).

The overall group mean score for constant error during the type I task was 36.8 ± 17.4%. The replication of an accurate level of force within the knee flexor musculature may have been difficult given the time restriction placed on this form of assessment. This finding is in agreement with the suggestion by Johannsen et al. (1991) that sensory receptors may not be able to provide receptor inflow during rapid movements. This type of test may well represent the emergency response of the sensorimotor system in that the body protects knee joint stability by over-compensating for force production during conditions of rapid threat when there is minimal opportunity for feedback and therefore the correction of force output. The actual force output for this type of task may have approached maximal force output on occasions (36.8% above the target force of 50% PFv). This may reflect a inefficiency in sensorimotor performance that may contribute to early conditions of fatigue in the knee flexor musculature during competitive athletic participation.

Variable error (type I SMP task).

The consistency of the constant error score for the group was reflected in a variable error value of 24.7 ±5.0%. No significant differences were observed between measures of variable error, corrected for a small sample size across the within-session, within-day and between-day assessments. This suggests that although subjects were not accurate in their force replication, the tendency to over-produce the desired target force was consistent whether the trial occurred within-day or between-days.

Constant error (type II SMP task).

Force production for the type II SMP task occurred over a 3-5 seconds time period. This may have allowed a greater amount of localised feedback from cutaneous receptors within the skin upon pressure on the force cuff. The ability to both judge and correct relative effort with accuracy may be reflected in the low overall group mean score for constant error bias (6.3%). It is very unlikely that an individual would have between 3 and 5 seconds to contract the knee flexors in order to protect the knee joint during the athletic scenario. Therefore, this type of assessment task may be more suited towards force-re-education following, for example, a period of de-training due to injury or surgery.

Variable error (type II SMP task).

The group mean level of consistency associated with the constant error score for the type II SMP task was 18.0 ±3.8 %. No significant differences were observed between the scores for the within-session, within-day and between-day levels of variable error for the type II SMP task. This also illustrates that the overall group performance was consistent between measurement occasions.

Single-measurement reliability.

Consideration of R_I scores suggests that single-measurement reliability for type I SMP assessment task may be slightly better than that of type II SMP assessment task ranging between 0.34 and 0.69 for type I and 0.11 and 0.43 for type II SMP task. The highest reliability of a single measurement was observed for within-session measures of constant error for both types of dynamic SMP assessment task (0.69 and 0.43 for types I and II SMP task respectively). Therefore from a clinical perspective if a single opportunity for assessment is only available for a given patient, within session estimates of sensorimotor performance may offer the least level of biological variance or greatest reliability.

However, such results must be interpreted with caution when used as a clinical outcome measure as the R_I for both types I and II SMP assessment tasks (within-session, within-day, and between-day) do not approach the clinically acceptable reliability coefficient threshold of greater than 0.80 that was recommended by Currier, (1984). The present R_I scores for type I SMP of 0.69, 0.48, and 0.34 may be classified as large, moderate and moderate for within-session, within-day and between-day assessments, respectively (Hopkins, 2002). Whereas for type II SMP task, the observed values of 0.43, 0.11, and 0.36 would be described as moderate, small, and moderate for within-session, within-day and between-day trials, respectively (Hopkins, 2002). The observed results in the present study are however comparable to previous R_I values observed for the 'traditional' measures of joint angle reproduction (0.49-0.82, Saxton, et al., 1995; -0.75, Marks and Quinney, 1996; 0.17-0.79, Kramer, et al., 1997; 0.42 between limbs and 0.85 for repeated trials, Beynnon et al., 2002). These traditional tests of proprioceptive performance have all been utilised as clinical research tools including the 'safe' return of an athlete to competitive sports.

Considering the results from the SEM calculation in the present study, for a single trial an individual's constant error score would have to be at least 56.8% and 53.1% different from the next persons score in the group to be actually detected as different for types I and II SMP assessment tasks, respectively. Overall group mean SEM% scores across the type I SMP assessment task, which range between ± 56.8 % and ± 70.9 % (95% confidence limits) indicate a limited capacity to discriminate physiological change in type I SMP functional performance based on a single trial for both within and between-day assessments. Similarly, the group mean SEM% scores across the type II SMP task ranged between ± 53.1 % and ± 71.7 % (95% confidence limits). This also indicates a limited capacity to discriminate physiological change in type II SMP functional capacity based on a single trial for within and between-day assessments.

Estimated precision or error of the mean score of multiple trials would be expected to vary inversely with the square root of the number of intra subject replicates, assuming a normal distribution of the replicates (Winer, 1981). Therefore, to obtain the minimum amount of assessment error using the Spearman-Brown prophecy formula in conjunction with the calculation of SEM % (Winer, 1981) 15 trials of type I SMP task would be recommended for within-session assessments, 20 trials for within-day and 25 trials for assessments between-days. For the type II SMP assessment, 25 trials would be recommended for within-session measures, >25 trials for within-day and 25 trials for between-day measures.

In summary, using 95% confidence intervals as a criterion, the ability to detect a change in performance to clinically acceptable levels would require 15 within-day trials of the type I SMP task, and 25 trials of the type II SMP task. This suggests that, as in many applications requiring high levels of measurement sensitivity, it is imperative to use a mean score of multiple trials as the basis for estimating SMP in order to reduce measurement error. Therefore a greater number of trials are recommended for subsequent assessments of constant error during types I and II sensorimotor performance tasks.

All potential sources of further variability (heterogeneity of the sample, biological variation and measurement error) were minimised within the present study by maintaining all assessment phases to the same time of day (±1 hour) and ensuring that the subject was 'focused' on the task at hand. The testing equipment was calibrated throughout the period of assessment to minimise technical error. However, the type II sensorimotor performance task was always administered prior to the type I SMP task during this experiment using an integrated approach of assessment (one 'gradual' type II SMP effort followed by two 'rapid' type I SMP efforts). This could possibly have led to a 'conflict' in motor response in terms of the difference between the permitted times for force production between the two tasks. The

five seconds time restriction involved with the type II task may have enabled neuromuscular feedback. Whereas, the muscular contraction of the knee flexors during the 'rapid' type I SMP task had to be performed at a rate of one complete contraction per second over two consecutive contractions. As this possible conflict cannot be eliminated as an influential factor in the results of this experiment, the subsequent experimental protocols within this thesis shall treat both types of SMP task as individual motor skills, whereby the familiarisation and assessment phase of both types of task shall be presented in a random order and separated by both time and test occasion, wherever feasible.

Finally, the heterogeneity of this young healthy recreationally active and 'a-symptomatic' group may not reflect the variation within the elite athletic population or during injured or post surgical, rehabilitative circumstances. As the reproducibility and single-measurement reliability of the two types of dynamic SMP tasks has not been previously examined with regard to the knee flexor musculature, this experiment may act as a guideline for future research that utilises this specialised form of dynamic assessment.

5.5. Summary.

This study documented the intra and inter-day variability and single-measurement reliability of constant error during types I and II sensorimotor performance assessment tasks applied to the knee flexor musculature of seven adult males (age 29.6 ±10.4 yr; height 1.78 ±0.04 m; mass 77.0 ±7.7 kg [mean ± SD]) and nine women (age 25.2 ±4.2 yr; height 1.69 ±0.08 m; mass 62.8 ±8.1 kg). Each subject completed three within-day trials over three test occasions, separated by no less than three days. Dynamic sensorimotor capacity was assessed in terms of active, isometric replication of a self-perceived force (50% daily voluntary peak force [PFv]).

The overall group mean bias or constant error value appeared to be greatest for the type I SMP task (36.8% versus 6.3% for type II SMP). This could be due to the fact that type I SMP task requires force production within a split second time frame, whereas type II task force production occurred over a period of 3-5 seconds, allowing for more localised feedback from cutaneous receptors within the skin upon pressure on the force cuff and also the ability to both judge and correct relative effort in the isometric contraction.

Group mean constant error values were expressed as a coefficient of variation score, corrected for a small sample size (Sokal and Rohlf, 1981). Within-session assessments of sensorimotor performance illustrated less variability and therefore may offer a greater level of measurement reproducibility for both methods of sensorimotor assessment. The observed values for the intra class correlation co-efficient (R_I) data are below the clinically acceptable level of 0.8 that was recommended by Currier, 1984. The overall group mean R_I scores which ranged between 0.69 ±56.8 % and 0.48 ±70.9 % for the type I SMP and 0.43 ±53.11 % and 0.11 ±71.7 % for the type II SMP (95% confidence limits). This suggests a limited capacity to discriminate physiological change in both types of sensorimotor assessment, based on a single trial. Thus, the mean score of multiple trials of both types of sensorimotor performance task would be recommended in order to achieve higher levels of measurement sensitivity in future research studies.

No significant differences were observed for measures of variable error during types I and II SMP task (corrected for a small sample size) across the within-session, within day and between day assessments. This suggests that although subjects were not entirely accurate in their force replication, the tendency to over-produce the desired target force was consistent whether the trial occurred within the same day or between-days. The Spearman-Brown prophecy formula (Winer, 1981) used in conjunction with the calculation of SEM% suggests

that at least 15 trials for type I and 25 replicates of the type II SMP task would be needed to discriminate properly between scores with the same measurement sensitivity. These findings present a slight logistical threat to measurement utility. However, these estimates relate to the average group response; which does not reflect fully the sensorimotor performance heterogeneity of some subjects within this sample.

The dynamic methods of sensorimotor performance assessment allow neuromuscular and sensorimotor performance to be integrated and quantified relative to maximal volitional force production. This form of assessment may therefore be a more transferable measure of lower limb control than slow, static joint reproduction tasks which have minimal transfer to the movement patterns witnessed during functional activities. It may be the case that efficient regulation of both neuromuscular and dynamic sensorimotor control at the knee joint are more important than the detection of joint positioning for injury prevention.

6.0. Effects of exercise stress on neuromuscular and sensorimotor performance of the knee flexors and acute recovery in the female soccer player.

6.1. Introduction.

Fatigue has been previously defined as "*a decrease in force production*" (Gandevia and McCloskey, 1995; Hagberg, 1981; Hawley and Reilly, 1997) and "*an inability to regenerate the original force*" (Bigland-Ritchie, 1981). The relationship between fatigue and acute muscle strain injuries has been documented in previous reports (Heiser, Weber and Sullivan, 1984; Hawkins, Hulse, Wilkinson and Hodson, 2001) although a direct relationship between absolute muscle strength and subsequent injury has not yet been proven (Knapik, Jones, Bauman and Harris, 1992). The mechanism of force 're-scaling' or 'muscle wisdom' during fatigue is unclear (St. Claire, 2001). It may be that force receptors become sensitised during fatigue or that the mechanism is central, perhaps reflecting some facilitation of the force-sensing circuitry by efferent activity (Flanagan, Wing, Allison and Spenceley, 1995). Cain and Stevens (1971) suggested that fatigue does not have a differential effect on the observer's sensitivity to force; instead, its effect appears to be uniform across the range of perceptible forces.

Previous research into the effects of exercise to the point of fatigue on the dynamic sensorimotor system (volitional force replication) is limited and inconsistent. As described in the review of literature, only three studies to date have reported on 'proprioceptive' performance (joint angle replication) following acute exercise induced fatigue (Skinner, et al., 1986; Marks and Quinney, 1993; Lattanzio et al., 1997). Skinner et al. (1986) found a significant decrease in the ability to reproduce joint angles after a series of interval running sprints in which subjects covered a total distance of 3.75 miles.

However, Marks and Quinney (1993) found that 20 maximal isokinetic quadriceps contractions used as a fatigue intervention, did not significantly reduce knee proprioception in young women. This study used an 'open chain' (foot not in contact with a stable surface) angle reproduction task, which differed in terms of fatigue intervention and subject sample when compared to Skinner's (1986) study. The quality of Marks and Quinney's (1993) study may however have been compromised as the fatigue protocol was not standardised according to the fitness status of each subject and thus the heterogeneity of subject response may have led to the non-significant findings.

No previous research has investigated the effects of a repeated exposure to an acute 'fatigue' intervention on neuromuscular and sensorimotor performance of the knee flexor musculature in female athletes. The aim of this study was to document the effects of fatigue inducing exercise and subsequent acute recovery, on indices of neuromuscular performance (PF_V, EMD_V, EMD_E) and both the constant and variable error arising from two types of sensorimotor performance assessment tasks in female soccer players. Repeated episodes of maximal exercise stress exposure were examined to mimic the physiological loading characteristic of a soccer game; 40 seconds bouts of maximal volitional activation of the knee flexors were applied to represent sprints made from penalty area to penalty area (Gleeson et al. 1998).

6.2. Methods.

Subjects.

Twenty females (age 22.0 ±2.8 years; height 165.7 ±0.04 m; mass 66 ±6.5 kg [mean ± SD]) gave their informed consent to participate in this study. All subjects were physically active members of the University of Wales, Bangor, women's soccer team. Subjects were instructed to keep sporting activities consistent throughout the experimental period, wherever possible.

Subject orientation.

Subject orientation and the pre-intervention procedures for this experiment were identical to those described in chapter 4.0.

Experimental protocol.

Following habituation to procedures, each subject completed 8 within day assessment sessions for a control condition consisting of no exercise (pre, post control period $_{A}$, post control period $_{B}$, post control period $_{C}$, post control period $_{D}$ and recovery at 60,180 and 360 seconds post control period $_{D}$ (a time frame which reflected the period where fatiguing exercise would occur subsequently in the experimental condition) and 8 within-day sessions for an experimental condition (pre, post fatigue period $_{A}$, post fatigue period $_{B}$, post fatigue period $_{C}$, post fatigue period $_{D}$ and recovery at 60, 180 and 360 seconds post fatigue period $_{D}$). All trials occurred on the same day and were preceded by a standardised warm-up. Control and experimental trials were separated by a standardised rest interval of 30 minutes to minimise any carry over effects. This protocol was repeated on two test occasions (one session for the type I SMP assessment task and one session for the type II SMP assessment task) with at least 7 days between sessions to eliminate the effects of previous localised fatigue. The order of SMP assessment type was randomly presented.

The experimental task involved contracting the knee flexor musculature of the preferred limb maximally against the immovable resistance offered by the dynamometer. Subjects were given standardised verbal encouragement from the test administrator. Each bout involved a 40 second fatigue task (30 seconds of sustained maximal isometric contraction, followed by two, 5 seconds maximal isometric contractions, separated by a 5 seconds rest interval). This process was repeated four times to represent the type of repeated sprint activity experienced by the athlete during a soccer game. The chosen 'fatigue' protocol was examined in a pilot study (n=16) and four episodes of the protocol were found to produce at least a 20% reduction in PFv.

Indices of neuromuscular and sensorimotor performance.

Peak force, volitional and evoked EMD, constant and variable error.

On each control assessment occasion (pre, post control period $_{A}$, post control period $_{B}$, post control period $_{C}$, post control period $_{D}$ and recovery at 60, 180 and 360 seconds post control period $_{D}$) and within each experimental assessment occasion (pre, post fatigue period $_{A}$, post fatigue period $_{B}$, post fatigue period $_{C}$, post fatigue period $_{D}$ and recovery at 60, 180 and 360 seconds post fatigue period $_{D}$) the peak score of at least two maximal volitional muscle activations of the knee flexor musculature was recorded to estimate volitional peak force and volitional electromechanical delay. Subjects performed 3 and 2 PFv contractions, respectively, to identify any decline in PFv. Subjects were instructed to give a maximal effort for all isometric contractions during the 'fatigue' protocol and subsequent assessments of PFv. Evoked electromechanical delay was also assessed using magnetic stimulation during the immediate time period following the cessation of the final maximal effort of the 'control/fatigue' protocol (complete relaxation was observed from the EMG signal). Finally, subjects completed the sensorimotor assessment task for each particular session. No verbal feedback regarding the achieved PFv or sensorimotor performance was given throughout the

assessment phase. The raw data was collected and interpreted using Spike 2 (version 2.01, Cambridge Electronic design LTD., U.K.) The experimental protocol for each type of assessment is illustrated in figures 6.1 and 6.2.

Type I SMP assessment occasion.

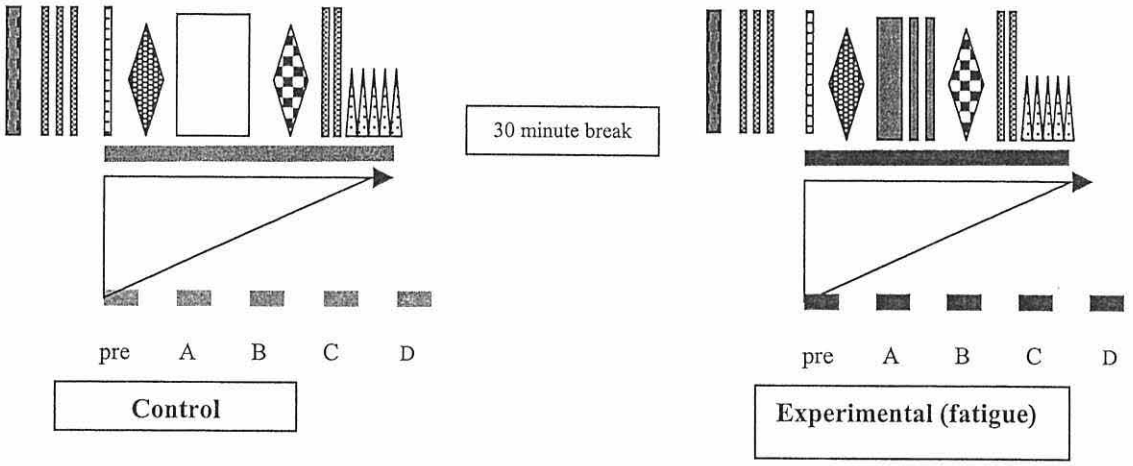


Figure 6.1. Neuromuscular and sensorimotor assessment protocol during the type I SMP task assessment occasion to determine constant and variable error; pre control and post control A, B, C and D and pre experimental and post experimental A, B, C, and D conditions.

Type II SMP assessment occasion.

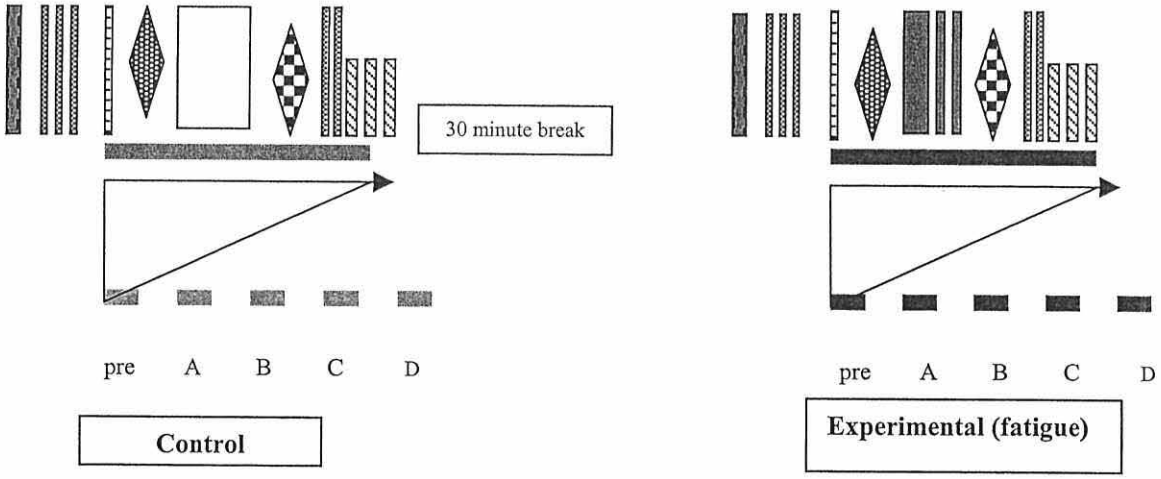
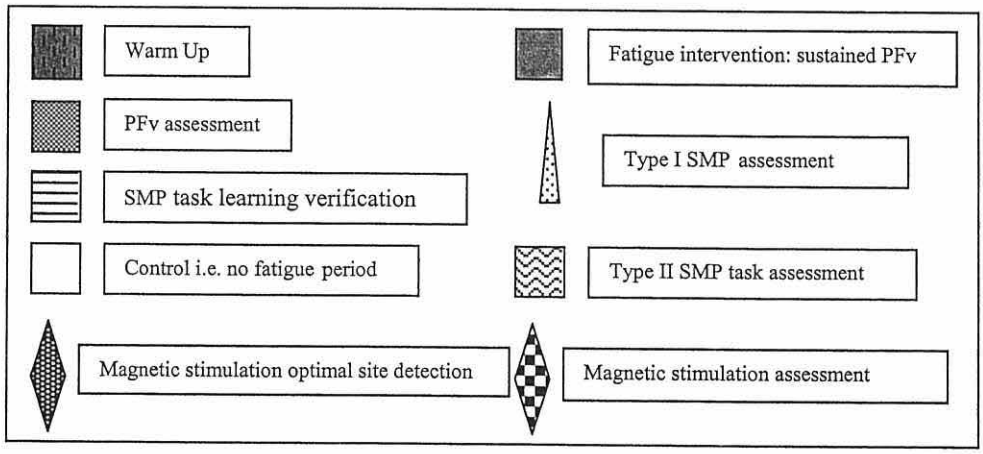


Figure 6.2. Neuromuscular and sensorimotor assessment protocol during the type II SMP task assessment occasion to determine constant and variable error; pre control and post control A, B, C and D and pre experimental and experimental A, B, C and D conditions.

Key:



Neuromuscular and sensorimotor performance was also assessed in terms of acute recovery following both the control and fatigue intervention at 60, 180 and 360 seconds post (post recovery 60, 180 and 360) as illustrated in figures 6.3 and 6.4.

Type I SMP post control/fatigue acute recovery assessment protocol.

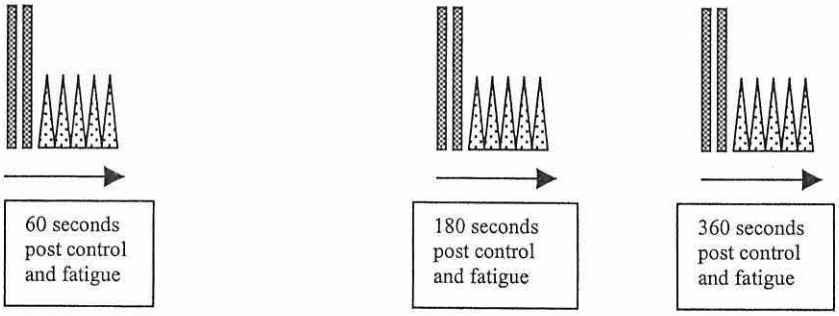


Figure 6.3. Neuromuscular and sensorimotor assessment protocol during the acute recovery phase (type I SMP task) to determine constant and variable error; following control D and experimental D conditions.

Type II SMP post control/fatigue acute recovery assessment protocol.

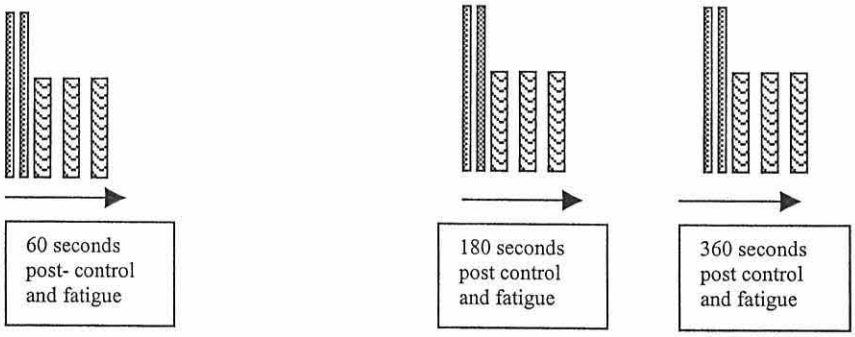
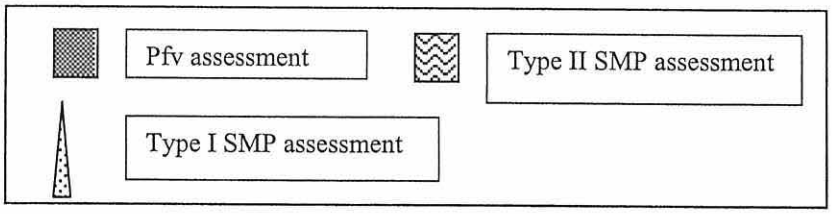


Figure 6.4. Neuromuscular and sensorimotor assessment protocol during the acute recovery phase (type II SMP task) to determine constant and variable error; following control D and experimental D conditions.

Key:



Statistical analyses.

Descriptive statistics (mean ±SD) were used to describe the indices of neuromuscular (PFv, EMD_E, EMD_v) and sensorimotor performance (constant and variable error in types I and II assessment tasks) associated with the knee flexor musculature. No significant correlation was previously observed between the two indices of sensorimotor performance assessment (n=16).

Therefore, separate two-factor ANOVAs (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures were used to analyse the effects of fatiguing exercise over time for each dependent variable.

Similarly, separate two-factor ANOVAs (condition [control; recovery] by time [post $_D$; recovery $_{60}$; recovery $_{180}$; recovery $_{360}$) with repeated measures were used to analyse the acute recovery responses in performance following the cessation of fatiguing exercise.

In the event of a significant Mauchly's test of sphericity, the violation to the assumption of ANOVA was corrected for using the Greenhouse-Geisser adjustment of the critical F-value, as indicated by (GG). SPSS (V 9.0) was used to perform all statistical procedures.

6.3. Results.

Volitional peak force [PFv] (type I SMP assessment occasion).

Group means and standard deviations for volitional peak force (type I SMP assessment occasion) at pre, post A, B, C, D and also at 60, 180 and 360 seconds post control and experimental conditions are illustrated in figure 6.5.

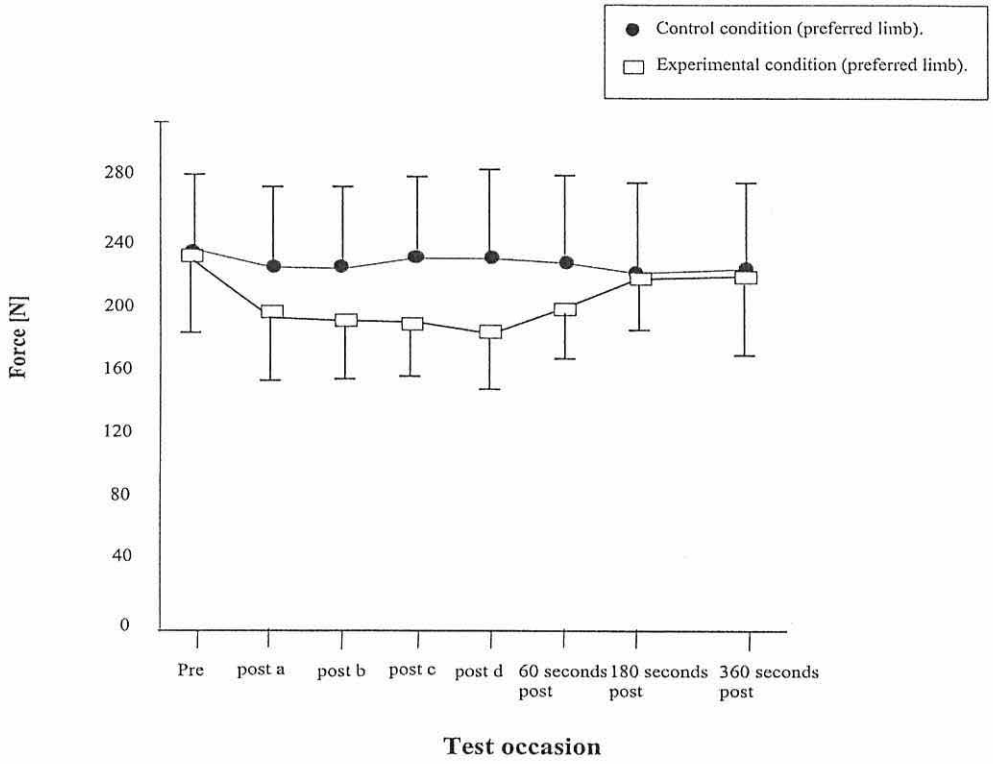


Figure 6.5. Group mean volitional peak force (N) [± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type I SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures showed a significant condition by time interaction ($F_{[4, 76]} = 18.27$, $p<0.002$). This suggests that while performance during the control trial was maintained, strength performance associated with the end of the fatigue trial was altered following the fatigue intervention. This reduction in PFv equated to a 22.3% decline when post $_D$ (fatigue) values were compared to pre-trial values (185.4 ± 43.3 N vs. 238.6 ± 48.3 N, respectively).

A second two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures also showed a significant condition by time interaction

($F_{[3, 57]} = 5.5$, $p<0.002$) for recovery. This suggests that while performance during the control trial was maintained, strength performance between the cessation of the fatigue trial and the end of the acute recovery period were different. The observed strength values following 360 seconds of recovery represented 96% of the pre fatigue strength measures (229.5 ± 49.0 N vs. 238.6 ±48.3 N, respectively).

Volitional peak force [PFv] (type II SMP assessment occasion).

Group means and standard deviations for volitional peak force (type II SMP assessment occasion) at pre, post a, b, c, d and also at 60, 180 and 360 seconds post control and experimental conditions are illustrated in figure 6.6.

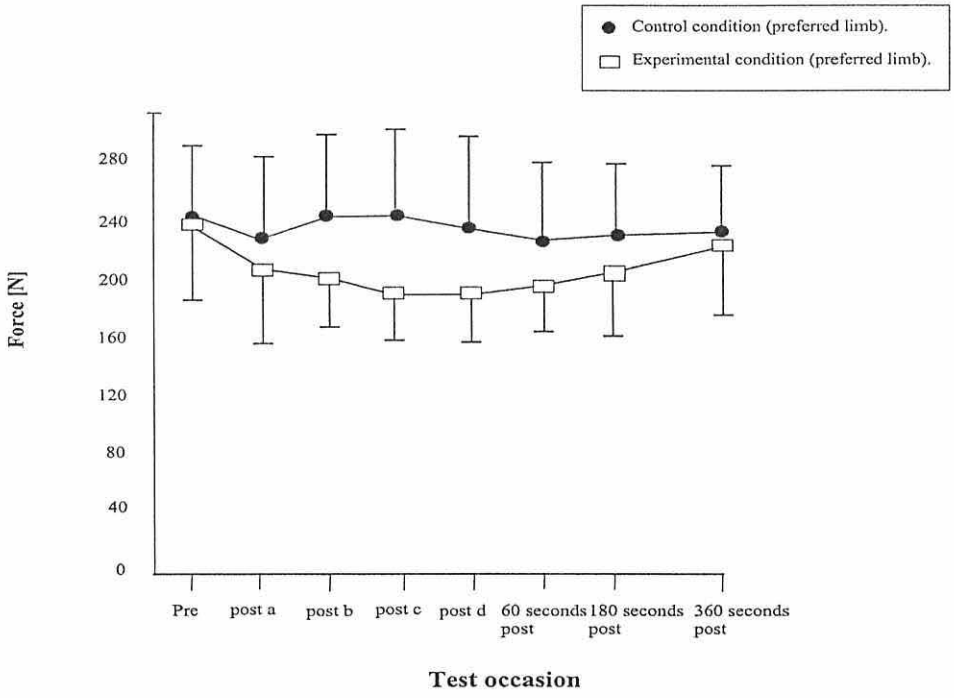


Figure 6.6. Group mean volitional peak force (N) [± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type II SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures showed a significant condition by time interaction ($F_{[4, 76]} = 13.49$, $p<0.001$). This suggests that while performance during the control trial was maintained, strength performance associated with the end of the fatigue trial was significantly different than pre fatigue measures. The observed reduction to peak force following the final bout of

fatigue equated to an 18.7% loss of PFv when compared to pre-trial levels (199.8 ± 44.3 N vs. 245.6 ± 55.6 N, respectively). This may have contributed to the significant interaction.

A second two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures also showed a significant condition by time interaction ($F_{[3, 57]} = 10.78$, $p<0.00$) for recovery. This suggests that whilst performance during the control trial was maintained PFv performance associated with the final episode of the fatigue trial altered between the final bout of fatigue and during the subsequent acute recovery up to 360 seconds following cessation of the maximal isometric activity. Group mean PFv values at the final assessment occasion during the acute recovery period equated to 92% of pre fatigue strength measures (226.1 ± 51.6 N vs. 245.6 ± 55.6 N, respectively). This illustrates almost a full recovery for PFv by 360 seconds post fatigue-inducing exercise.

Volitional EMD (type I SMP assessment occasion).

Group means and standard deviations for volitional electromechanical delay (type I SMP assessment occasion) at pre, post a, b, c, d and also at 60,180 and 360 seconds post control and experimental conditions are presented in table 6.1.

	Volitional EMD (EMDv) (ms)	
Test occasion	Control [Mean ±SD]	Fatigue [Mean ±SD]
Pre	58.1 (9.7)	58.1 (9.7)
Post a	58.4 (13.4)	60.0 (18.4)
Post b	59.3 (10.6)	57.7 (12.8)
Post c	58.0 (11.7)	56.7 (12.8)
Post d	57.5 (9.6)	56.1 (14.3)
Recovery 60 seconds	58.0 (13.2)	63.2 (17.9)
Recovery 180 seconds	54.7 (12.8)	58.2 (13.4)
Recovery 360 seconds	56.7 (12.4)	60.0 (16.6)

Table 6.1. Group mean volitional EMD (ms) [± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type I SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures showed no significant condition by time interaction and no significant main effects. This suggests that volitional electromechanical delay was preserved in the preferred limb during both the control and fatigue trials across all time periods during the type I SMP assessment occasion.

A second two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures, showed no significant condition by time interaction or main effects during recovery. This suggests that performance was consistent between both the control and fatigue trials and subsequent acute recovery periods of up to 360 seconds for the type I SMP assessment occasion.

Volitional EMD (type II SMP assessment occasion).

Group means and standard deviations for volitional electromechanical delay (type II SMP assessment occasion) at pre, post a, b, c, d and also 60, 180 and 360 seconds post control and experimental conditions are presented in table 6.2.

	Volitional EMD (EMDv) (ms)	
Test occasion	Control [Mean ±SD]	Fatigue [Mean ±SD]
Pre	62.1 (6.2)	62.1 (6.2)
Post a	61.2 (5.0)	60.5 (6.9)
Post b	62.9 (3.4)	58.1 (5.4)
Post c	60.3 (5.5)	55.3 (4.9)
Post d	60.7 (6.3)	52.6 (4.6)
Recovery 60 seconds	59.7 (5.7)	54.7 (6.5)
Recovery 180 seconds	61.8 (4.9)	59.4 (5.6)
Recovery 360 seconds	60.6 (5.3)	62.8 (5.8)

Table 6.2. Group mean volitional EMD (ms) [± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type II SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures showed no significant condition by time interaction and no significant main effects. This suggests that volitional electromechanical delay was preserved during both the control trial and fatigue trials across all time periods in the preferred limb.

A second two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures showed no significant condition by time interaction or main effects during recovery. This suggests that volitional EMD performance was also consistent between both the control and fatigue trials and subsequent acute recovery periods of up to 360 seconds.

Evoked EMD (type I SMP assessment occasion).

Group means and standard deviations for evoked electromechanical delay (type I SMP assessment occasion) at pre, post a, b, c, d and also 60,180 and 360 seconds post control and experimental conditions are illustrated in figure 6.7.

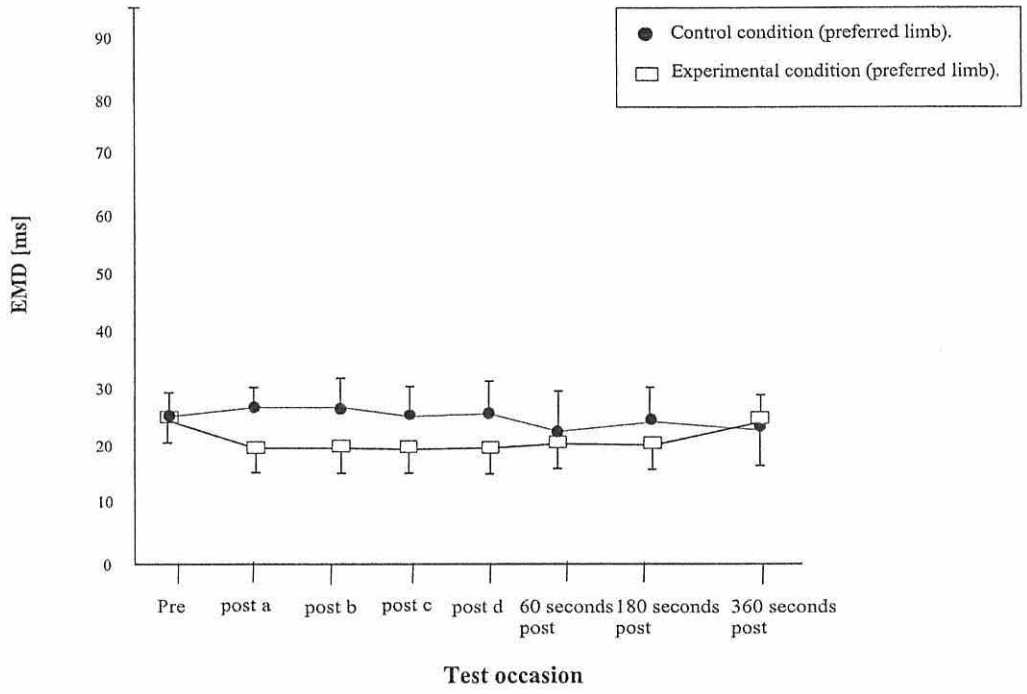


Figure 6.7. Group mean evoked EMD (ms) [± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type I SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures showed a significant condition by time interaction ($F_{[4, 76]} = 5.71$, $p<0.001$). This suggests that while performance during the control trial was maintained, evoked electromechanical delay values during the fatigue trials showed a significant difference when compared to pre test values (21.9 ± 2.9 ms, 21.9 ± 4.0 ms, 21.7 ± 4.1 ms and 22.0 ± 3.7 ms vs. 24.5 ±4.1 ms, respectively).

A second two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures, showed no significant condition by time interaction or main effects for the recovery of evoked EMD between the final bout of fatigue and subsequent recovery period. This illustrates that evoked EMD values were consistent during the final control period assessment, the final bout of fatigue and corresponding acute recovery periods of up to 360 seconds.

Evoked EMD (type II SMP assessment occasion).

Group means and standard deviations for evoked electromechanical delay (type II SMP assessment occasion) at pre, post a, b, c, d and at 60, 180 and 360 seconds post control and experimental conditions are illustrated in figure 6.8.

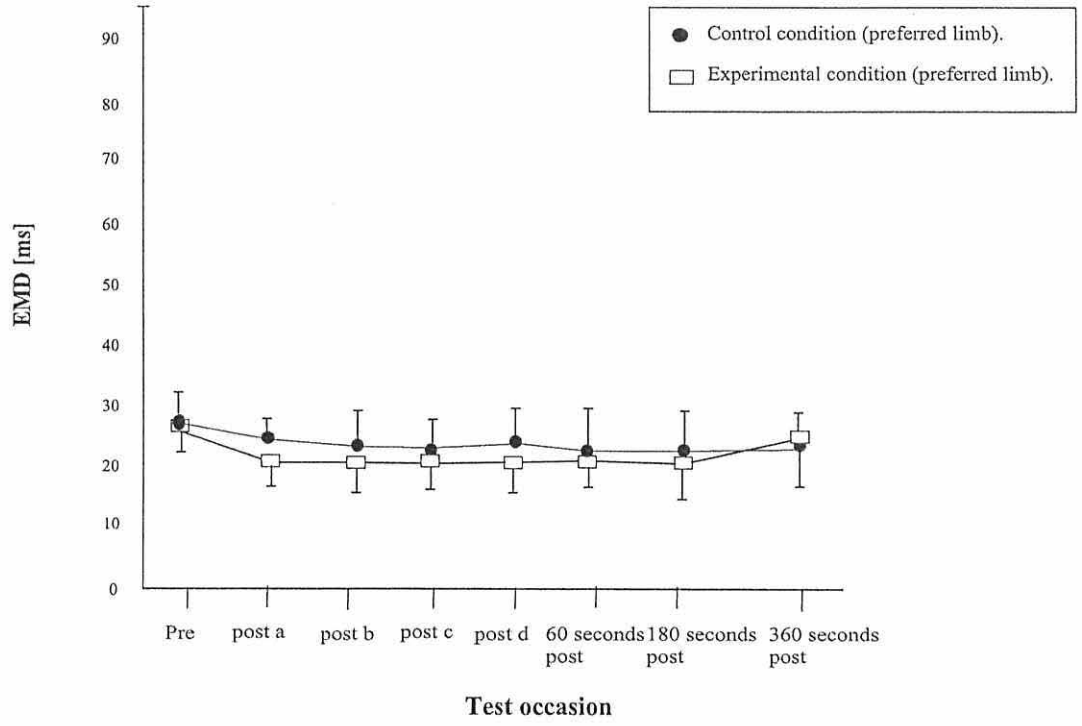


Figure 6.8. Group mean evoked EMD (ms) [± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type II SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures showed a significant condition by time interaction ($F_{[4, 76]} = 2.56$, $p<0.002$). This suggests that while performance during the control trial was maintained, evoked electromechanical delay values following the initial and subsequent fatigue trials were different when compared to pre intervention values (21.8 ±4.4 ms, 21.4 ±3.2 ms, 21.8 ±6.0 ms and 22.3 ±4.3 ms vs. 26.7 ±6.6 ms respectively).

A further two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures also showed a significant condition by time interaction ($F_{[3,57]} = 4.14$, $p<0.01$) for recovery. This suggests that while performance during the control trial was maintained, evoked EMD times altered during the acute recovery phase up to and

including 360 seconds in comparison to those values immediately following the final bout of fatigue (25.8 ±5.8 ms, 24.6 ±6.1 ms, 25.9 ±5.9 ms vs. 22.3 ±4.3 ms, respectively).

Constant error (type I SMP assessment occasion).

Group means and standard deviations for constant error (type I SMP assessment occasion) at pre, post a, b, c, d and also 60, 180 and 360 seconds post control and experimental conditions are illustrated in figure 6.9.

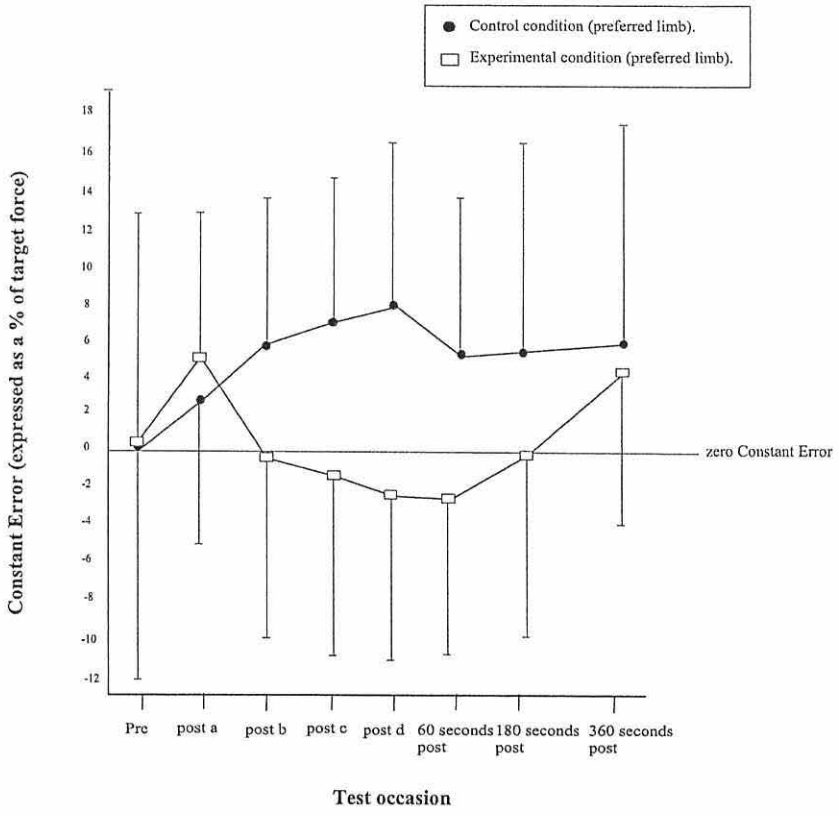


Figure 6.9. Group mean constant error (as a % of target force)[± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type I SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures showed a significant condition by time interaction ($F_{[4, 76]} = 6.79$, $p<0.003$). This suggests that constant error scores during the fatigue condition were significantly different than constant error values during the control trial.

Secondly, a two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures showed a significant condition by time interaction ($F_{[3,}$

57] = 5.59, p<0.001) for recovery. This suggests that performance during the control trial was maintained but constant error scores for the type I SMP task were altered during the acute recovery phase up to and including 360 seconds when compared to the group mean scores immediately following the final bout of fatigue (-2.5 ±9.5%, 0.3 ±10.1% and 4.1 ± 9.6% vs. – 2.2 ± 11.9% [of target force], respectively).

Constant error (type II SMP assessment occasion).

Group means and standard deviations for constant error values (type II SMP) at pre, post a, b, c, d and also 60, 180 and 360 seconds post control and experimental conditions are illustrated in figure 6.10.

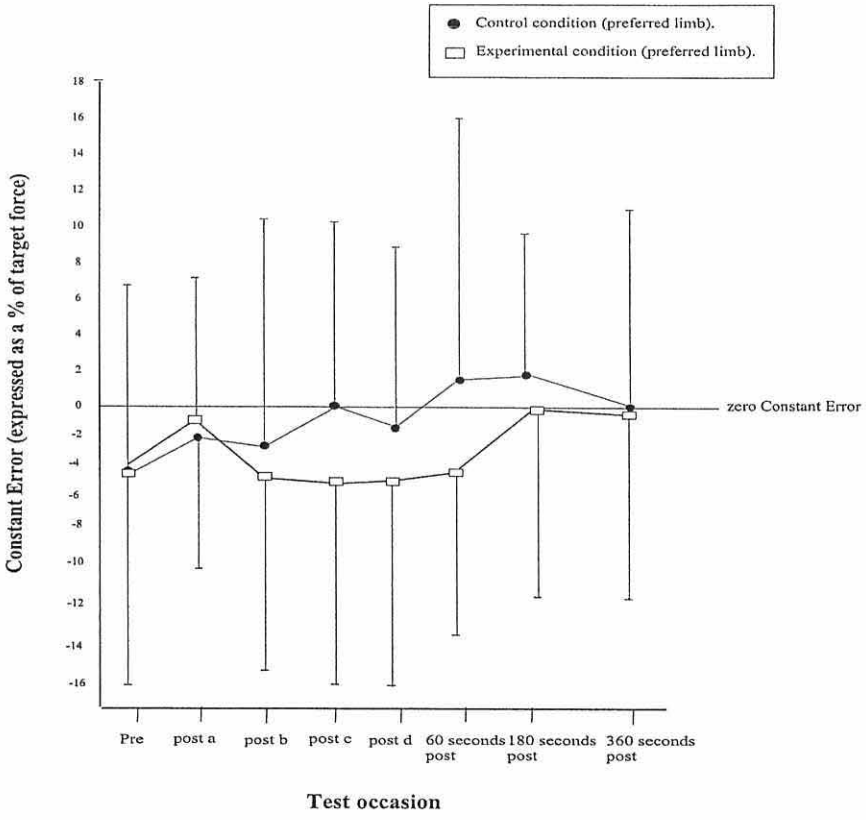


Figure 6.10. Group mean constant error (as a % of target force)[± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type II SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures showed a significant condition by time interaction ($F_{[4, 76]} = 4.22$,

$p<0.002$). Thus, constant error performance during the control trial was different than that during the fatigue trial.

A subsequent two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures showed no significant condition by time interaction for measures between the final bout of fatigue and following acute recovery phase. This suggests that the pattern of constant error response for the type II SMP task was similar during both the control and fatigue assessments.

Variable error (type I sensorimotor performance assessment).

Group means and standard deviations for variable error (type I SMP) at pre, post a, b, c, d and also at 60, 180 and 360 seconds post control and experimental conditions are presented in table 6.3.

	Variable error (% of target force)	
Test occasion	**Control [Mean ±SD]**	**Fatigue [Mean ±SD]**
Pre	9.1 (3.0)	9.1 (3.0)
Post a	12.5 (6.4)	13.3 (8.8)
Post b	12.5 (6.3)	11.0 (7.5)
Post c	12.8 (5.5)	9.2 (4.5)
Post d	10.5 (4.0)	10.5 (3.6)
Recovery 60 seconds	13.8 (5.1)	9.2 (4.6)
Recovery 180 seconds	12.3 (7.6)	9.9 (4.8)
Recovery 360 seconds	10.9 (3.2)	12.2 (5.3)

Table 6.3. Group mean variable error [± SD] (as a % of target force) at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type I SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$]) with repeated measures revealed no significant condition by time interaction or main effects. This suggests that performance during both the control and fatigue trials was comparable and that the level of variable error (consistency of response) was not significantly altered as a

result of the fatigue intervention, despite a significant alteration to the accuracy of the force reproduction.

A further two-factor ANOVA (condition [control; fatigue] by time [post $_D$, post 60, 180, 360 seconds]) with repeated measures showed a significant condition by time interaction for measures of variable error between the final bout of fatigue and following acute recovery phase ($F_{[3, 57]} = 4.22$, $p<0.001$). Therefore, performance in terms of variable error associated with the group mean response for the type I SMP task differed between the control and experimental recovery occasions (13.8 ±5.1%, 12.3 ±7.6%, 10.9 ±3.2% vs. 10.5 ±4.0% (pre) and 9.2 ±4.6%, 9.9 ±4.8%, 12.2 ±5.3% vs. 10.5 ±3.6% (pre) [% of target force] for the for control and experimental conditions, respectively).

Variable error (type II sensorimotor performance assessment).

Group means and standard deviations for variable error during the type II SMP assessment occasion at pre, post a, b, c, d and also at 60, 180 and 360 seconds post control and experimental conditions are presented in table 6.4.

	Variable error (% of target force)	
Test Occasion	**Control [Mean ±SD]**	**Fatigue [Mean ±SD]**
Pre	11.5 (11.8)	11.5 (11.8)
Post a	12.8 (11.8)	10.6 (5.6)
Post b	11.8 (7.4)	11.1 (8.5)
Post c	12.3 (8.0)	10.7 (5.3)
Post d	12.0 (7.6)	12.2 (6.6)
Recovery 60 seconds	10.6 (5.2)	10.0 (6.5)
Recovery 180 seconds	10.1 (5.3)	8.0 (4.2)
Recovery 360 seconds	13.9 (7.1)	9.5 (5.2)

Table 6.4. Group mean variable error [± SD] at pre, post A, B, C, D and 60, 180 and 360 seconds recovery for control and fatigue occasions (type II SMP assessment).

A two-factor ANOVA (condition [control; fatigue] by time [pre; post $_A$; post $_B$; post $_C$; post $_D$) with repeated measures revealed no significant condition by time interaction or main effects. This suggests that performance of the type II SMP task during both the control and fatigue

trials was similar and therefore the consistency of sensorimotor response was not significantly altered by the fatigue intervention.

Furthermore, a two-factor ANOVA (condition [control; fatigue] by time post $_D$, post 60, 180, 360 seconds with repeated measures showed no significant interaction or main effects for measures of variable error during for the post $_D$ to post 360 seconds phases of recovery. Thus the consistency of the constant error response for the type II SMP assessment task did not differ between the control and experimental recovery occasions. This suggests that although the force replication was not 100% accurate, the consistency of this response was maintained across both a period of control and maximal isometric activity of the knee flexor musculature.

6.4. Discussion.

Peak force.

Peak force was preserved during the control conditions for both assessment days in the experimental (preferred) limb. However, the fatigue intervention achieved a significant (23%) reduction in strength of the knee flexors for both type I and II SMP assessment sessions when compared to the corresponding pre fatigue values. These findings are in agreement with Gleeson et al. (1998) who reported that prior endurance activity impaired the potential for tension development in subsequent resistance activity. The observed strength loss may be attributed to both ultra-structural damage and chemical (metabolic) stressors within the skeletal muscle tissue during the acute bouts of fatigue inducing activity. Recovery to 94-96% (± 2%) (95% confidence limits) of group mean 'pre' strength values was observed at the 360 seconds stage of recovery on both fatigue occasions. The potential for a loss of dynamic support may be far greater for soccer players during a game scenario that requires sustained neuromuscular performance for a 90-minute period.

De Vries (1968) suggested that the peak volitional force exerted by a fatigued limb was perceptually different from that exerted by an unfatigued limb. The recovery rate from fatigue is suggested to be fastest at higher forces and slowest at lower forces (Petrofsky and Phillips, 1981) depending on the rate of removal of waste products, which accumulate in higher concentrations during low force (30% MVC) contractions (Karlsson, Funderbunk, Essen and Lind, 1975, Vollestad, Sejersted and Saugen, 1997). These observations are in agreement with the current findings in that the recovery of PFv was almost complete within 360 seconds and would probably oppose the findings of Newham et al. (1991) that maximal voluntary force of the quadriceps did not recover to pre-exercise values until 24 hours following fatiguing concentric and eccentric exercise. However, as this study examined the knee flexor musculature, a direct comparison to the relatively larger (in terms of cross

sectional area) antagonistic muscle group may be misleading, as the removal of metabolic waste products following fatigue may indeed be a lengthier process in the quadriceps.

The chosen fatigue protocol was considered suitable for the current study in that the observed cumulative loss of force capability in the knee flexors of up to 20% compared to baseline levels, would be entirely compatible with valid assessments of sensorimotor performance involving blinded target forces of no more than 50% of maximum strength performance i.e. Subjects were still physically capable of producing 50% of pre PFv values following the final bout of fatigue inducing exercise, enabling a true examination of sensorimotor function. The observed disruption to the accuracy of force replication during both types I and II sensorimotor tasks could be attributed to the significant loss of overall strength and also a failure of the force 're-scaling' mechanism during the sustained fatigue protocol. The observed trend to under-achieve the target force during the type II form of assessment could therefore be attributed to the disruption of maximal strength capacity remaining undetected by both the afferent (muscle spindles) and therefore the efferent feedback systems.

Volitional EMD.

Volitional EMD was preserved across both control and fatigue conditions during types I and II SMP assessment occasions. The non significant difference between the control and experimental conditions may be explained by either an overall increase in the compliance of the musculotendinous unit (Westgaard and De Luca, 1999) or that motor efferent recruitment patterns are altered in fatigue to prevent metabolic derangement from damaging the muscle fibres (St.Clair, 2001) although it is unclear how well orderly recruitment is preserved under conditions of fatigue (Enoka, 1995). A subconscious neuromuscular reserve has also been witnessed during maximal sprinting activities (Kay et al., 2001) and may exist in order to protect the human body from over exertion.

It was expected that the EMD_V would be prolonged or increased during fatigue conditions (Mercer and Gleeson, 1996, Zhou, McKenna, Lawson, et al. 1996; Mercer, Gleeson, Claridge, and Clement, et al. 1998). Fatigue related impairment of EMD_v may be attributed to a complex interaction of neuromuscular and biomechanical factors including the rate of shortening of the series elastic component of muscle, accompanied by the limb segment orientation and moment of inertia (Gleeson, 2001). As no significant interaction was observed for volitional EMDv, it is plausible that certain other factors may in some way have moderated or diluted the expected effects of fatigue on EMDv within this particular study. It is possible that the intensity of the chosen fatigue task may not have been reflective of the real life scenario despite the observed loss of strength and thus failed to elicit a significant disruption to the switch on time of the knee flexors. This suggestion may be reinforced by the fact that the consistency of sensorimotor performance response (variable error) was maintained during the accumulative effects of the exercise stress protocol.

Evoked EMD.

A significant condition by time interaction was observed for evoked electromechanical delay values during both sensorimotor task assessment occasions. As this type of stimulation does not involve conscious regulation by the subject, it may be that the maximal isometric contractions led to an increased localised muscle temperature and thus the propagation speed of the impulse along the sciatic nerve. The contractile speed of skeletal muscle has been previously shown to be sensitive to temperature (Stein, Gordon and Shriver, 1982). A 3-4 °C increase in muscle temperature during 30% and 60 % MVC repetitive isometric exercise to exhaustion has been witnessed (Vollestad et al. unpublished observations). An increase in central drive to the sciatic nerve during the fatigue intervention may also explain the preservation of evoked conduction velocities (Pincivero, Coelho and Erikson, 2000). A significant interaction was observed for magnetically evoked EMD between the final bout of

fatigue and the acute recovery phase during the type II SMP assessment session. An apparent increase in evoked EMD values towards the final recovery phase assessments may be attributed to a relative 'cooling down' period in recovery and thus a decrease in both temperature and compliance of the involved musculotendinous units.

Sensorimotor performance.

The precise relationship between the physiological and psychological mechanisms involved in the perception of force remains unclear. It has been previously suggested that isometric forces feel larger during fatigue, in spite of the instruction to ignore the increased effort associated with fatigue and focus only on force matching (Jones, 1983). This overestimation of muscular forces during fatigue has led to the idea that more importance is placed on sensing the effort put into the contraction rather than on the achieved muscular tension (Gandevia and McCloskey, 1977). Sensorimotor performance during both types of dynamic assessment task of the knee flexors has not been previously assessed in terms of both constant and variable error, thus an expected outcome could not be predicted for this study. However, as the type I SMP task is a 'rapid' style of muscular response, recruiting predominantly type II muscle fibres, it was hypothesised that disruption to this style of sensorimotor performance would be the most pronounced during situations of acute fatigue and subsequent loss of volitional peak force. However, the accuracy of force replication was significantly altered during *both* SMP assessment sessions.

A significant interaction was also observed for measures of constant error between the post $_D$ to post 360 seconds (type I SMP session only). During this time period, pre fatigue values were not re-established until 180 seconds following the final fatigue bout (94-96% of pre PFv values ± 2% [95% confidence limits]). This may have alarming implications for the sporting scenario in terms of knee joint control and the maintenance of homeostasis. Using soccer as

an example, if a player is required to make multiple end to end sprints of the pitch within a short time frame, neuromuscular feedback from the fast twitch muscle fibres may be increasingly compromised if recovery to resting values requires up to a three-minute period. Thus, the rapid neuromuscular compensatory mechanism outlined via feed forward (which was assessed via the type I SMP task) may be jeopardized during conditions of 'acute' fatigue. This contradicts the suggestion by Cafarelli et al. (1982) that the effects of fatigue may be counterbalanced by an efficient feedback/ feed forward system of information processing, preventing injury from unexpected perturbation. This potential for joint injury is heightened by the fact that dynamic support was reduced by up to 23% during fatigue.

However, the observed performance in both types of sensorimotor task may also be viewed as highly accurate when put into context. As the recorded constant error values were expressed relative to 50% of PFv the observed scores should be reduced by half to be expressed relative to maximal strength performance. Thus, a constant error score of between 1% and 4% (of the target force) may indeed reflect a refined sensitivity of the sensorimotor system and could alternatively be interpreted as a preservation of muscle spindle performance capability during strength losses of up to 23%.

Fast twitch fibres are dominant during maximal isometric contractions but easily fatigued, leaving slow twitch muscle fibres to be recruited first under conditions of fatigue according to the 'size-principle' (Gabriel, Basford and An, 2001; Gleeson, 2001). A significant disruption to variable error (during the type I SMP task) was only observed during the recovery phase of assessment. This may reflect an actual fine-tuning or honing of the sensorimotor afferent feedback strategy during recovery as the amount of variability in performance stayed relatively constant during the ongoing fatigue. It can be assumed that the degree to which a muscle generates force is, in part, a product of a centrally generated motor command (Guyton

and Hall, 1996). The influence of muscle fatigue may have provided a significant degree of input for mediating perceived effort, as an increase in central drive would have been necessary to sustain a given force level, with longer sustained contractions (Pincivero, Coelho and Erikson, 2000). This enhancement of effort awareness may have thus 'masked' any actual disruption to the muscle spindles.

There are many factors to take into consideration when evaluating the effects of fatigue on neuromuscular performance including the specific demands of the exercise, type of muscle action, velocity of movement, frequency of activation and subject motivation. Acute recovery of the neuromuscular and sensorimotor system following repeated isometric contractions has received very limited research with regard to the knee flexor musculature. The only disadvantage to having the subjects perform this test twice was the potential for a pacing strategy to be adopted for the second test because subjects had prior experience of the fatigue condition during their initial assessment. The influence of this possible strategy on the style of SMP task was minimised as the type of task administered for each visit was randomly presented.

The chosen study population was female athletes as it has been previously suggested that females harbor poor proprioceptive capabilities and are more influenced by fatigue (Huston and Wojtys, 1996). The fatigue intervention significantly affected volitional peak force, evoked EMD, and the accuracy of sensorimotor performance. However a transient 2% loss of sensorimotor performance during fatigue may not be biologically meaningful in comparison to the 23% loss of PFv. Further research is therefore required in order to investigate the effects of a more permanent type of interruption to the muscle spindles and therefore the body's afferent apparatus such as during conditions of exercise induced muscle damage (EIMD). This may represent/ reflect a more realistic sporting situation, for example

during prolonged physical exercise or indeed tournament conditions when there is limited recovery between prolonged bouts of rigorous exercise.

6.5. Summary.

This study documented the effects of repeated bouts of exercise stress and acute recovery on indices of neuromuscular (PF_v, EMD_V, EMD_E) and two types of sensorimotor performance assessment of the knee flexor musculature (preferred limb) in active female soccer players. Volitional peak force was significantly disrupted following four bouts of maximal isometric activity in the preferred limb and also over an acute recovery period of 360 seconds for both assessment occasions. The overall loss of strength in the knee flexor musculature equated to 23% across the two test occasions. Strength recovery was observed following the cessation of exercise with 94-96% ± 2% (95% confidence limits) of the group mean 'pre' strength values being achieved by the 360 seconds on both assessment days.

Volitional EMD performance appeared to be preserved across all test occasions. This may be explained by either an overall increase in the compliance of the musculotendinous unit (Westgaard and De Luca, 1999) or that efferent motor unit recruitment patterns are altered in fatigue, preventing metabolic derangement and damage to the muscle fibres (St.Clair, 2001). However, it is also possible that the laboratory based experimental protocol may not have been potent enough to have significantly disrupted volitional EMD within this study. A significant alteration to the group mean evoked EMD response was observed following the fatigue protocol on both assessment days. This disruption was also evident during the recovery phase of the type II SMP assessment occasion. The alteration to evoked EMD may be attributed to an increase in the localised muscle temperature during maximal isometric activity of the knee flexors and thus the conduction velocity of the action potential at the neuromuscular junction.

The accuracy of sensorimotor performance was significantly disrupted by the fatigue intervention during both the time-regulated and self-regulated tasks. Pre test levels of sensorimotor accuracy were not fully re-instated until 180 seconds following the cessation of the fatigue protocol; which has significant implications during athletic performance. The consistency of the constant error response (variable error) was also significantly affected during the acute recovery phase of the type I SMP assessment occasion. This may in fact represent a fine-tuning of force regulation during conditions of strength loss and metabolic stress within the affected musculature. The observed sensorimotor performance during both styles of assessment may alternatively be viewed as efficient when the 4% loss of sensorimotor performance is compared to the 23 % loss of strength. The functional implication of this magnitude of disruption to sensorimotor and neuromuscular control of the knee joint requires further investigation. The potential for knee joint disruption may be far greater considering that the observed findings are in relation to a 3-minute period of maximal activity rather than the 90-minute scenario of a soccer game.

On balance, the recovery of volitional peak force may be the greatest threat to knee joint stability during situations of acute fatigue. Further research into the effects of a more permanent disruption to knee flexor homeostasis on neuromuscular and sensorimotor performance is required, such as during conditions of exercise induced muscle damage. This will help to determine how the body may adapt in order to maintain protection of the lower limb, during a more permanent alteration to the primary factors of knee joint control.

7.1. The effects of exercise induced muscle damage on neuromuscular and sensorimotor performance in the knee flexors of female athletes.

7.1.1. Introduction.

In the previous chapter (6.0) the effects of an acute fatigue intervention protocol on neuromuscular and sensorimotor and performance of the knee flexors was examined. Neuromuscular performance in terms of absolute strength was significantly disrupted by up to 23% in the experimental (preferred) limb following four bouts of a maximal isometric activity. The fatigue intervention also led to a significant alteration to the overall group evoked neuromuscular response and the accuracy of force replication during *both* types of sensorimotor assessment task. Volitional EMD was however preserved following the fatigue intervention, despite the significant loss of strength. Up to a 6-minute period of recovery was required to restore baseline levels of strength, evoked EMD and sensorimotor performance following the fatigue intervention. This reflects a transient disruption to both neuromuscular and sensorimotor performance during acute conditions of exercise stress.

Sensorimotor performance during a more prolonged, mechanical type of disruption to the muscle i.e. during conditions of exercise induced muscle damage has received a limited amount of previous investigation (Hutton and Attwater, 1992) 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 unfamiliar exercise, primarily involving eccentric contractions (McHugh, 2000) and that this type of exercise stress frequently results in symptoms of delayed onset muscle soreness (DOMS). In humans, exercise induced muscle damage (EIMD) is usually described via symptoms of strength loss, pain with activity, muscle tenderness and elevated muscle enzyme activity (McHugh et al., 1999). The peak in symptoms is expected to occur between four and six days following the muscle-damaging episode (Byrne and Eston, 1998). The majority of previous research into EIMD in the lower limb has had a restricted focus on

the 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).

Little is currently known about how the body copes from a neuromuscular *and* sensorimotor perspective, whilst the lower limb musculature is experiencing conditions of EIMD. The assessment of both volitional and evoked EMD may offer an insight into the recruitment pattern of motor fibres during conditions of muscle damage. The potential for disruption to the pattern of type II motor fibre recruitment may be greater during conditions of EIMD and could be reflected in an alteration to the consistency of performance (variable error) during the type I SMP assessment task which requires a time orientated pattern of force delivery. Sensorimotor performance may be an important factor in the preservation of joint integrity during muscle damage and disruption to the capacity for muscle spindle feedback. For example, in a tournament situation for any given sport, repeated exposure to exercise stress may cause micro-damage of the muscle fibres, progressing to more major muscle tears of the involved muscle groups (Proske and Morgan, 2001). Sensorimotor efficiency may be compromised if the effect of this damage is detrimental to joint awareness and the neuromuscular control of the involved joint(s).

The primary aim of this experiment was to document any moderation to both neuromuscular and sensorimotor performance during conditions of EIMD in the knee flexors of the non-preferred limb in female athletes. Secondly, it is not yet known whether the findings from previous research which describe a less pronounced neuromuscular interruption to EIMD following a second or repeated-bout exposure to EIMD in the knee extensors (Brown, et al., 1997; McHugh, et al., 1999; McHugh, 2000) are applicable to the neuromuscular and sensorimotor performance of the knee flexors. Therefore, any further moderation to

neuromuscular and sensorimotor performance in the knee flexors following a repeated-bout of the same EIMD intervention will be examined in a supplementary section to study 7.1.

7.1.2. Methods.

Subjects.

10 adult females (age 21.3 ±2.4 years; height 1.67 ± 0.07 m; body mass 67.7 ±7.04 kg [mean ± SD]) gave their informed consent to participate in this study. The sample comprised collegiate and semi-professional team-game athletes. All physiological, neuromuscular and sensorimotor assessments were performed as near to the same time of day as possible (±1h).

Physiological indices of performance capacity.

Each subject's active flexibility of the involved muscle group, blood plasma creatine kinase (CK) levels and perceived muscle soreness (pain rating) were assessed at each test occasion during the study (prior to and at 1, 24, 48, 72 and 120 hours following the EIMD protocol) to provide descriptive evidence of EIMD.

CK assessment.

Blood plasma creatine kinase levels were determined by a finger-prick blood sample. The finger was warmed, cleaned with alcohol and dried. After puncture, the initial sample of blood was removed. A 30 µl sample was then collected in a capillary tube and immediately pipetted onto a test strip for analysis. CK activity was analysed by colorimetric assay procedure (Reflotron, Boehringer Mannheim, Indianapolis, Indiana). This system uses a plasma separation principle incorporated in the reagent carrier on the test strip.

Perceived pain (muscle soreness) rating scale.

Pain on active movement utilising the hamstrings musculature of the affected limb was assessed using a specifically designed self-perceived pain rating scale (Marginson et al. 2001). This pain scale was designed and validated for use with DOMS and EIMD (Marginson et al. unpublished PhD data) and requires the subject to rate his/her perceived pain by moving a pointer on a sliding scale (0-10) to the most suitable from a series of statements which include 'my muscles don't feel sore at all', 'my muscles feel sore when I move them', and 'my muscles feel so sore that I don't want to move them', corresponding to a numerical rating of 0, 5 and 10, respectively. Subjects were asked to stand in an upright position with their legs shoulder width apart. With the majority of body weight taken through the experimental (non-preferred) limb, subjects were asked to bend forward as if trying to touch their toes, and then move the sliding pointer to the corresponding level of pain that was experienced in the 'hamstrings' *during* the movement.

Flexibility.

Subject's active hamstrings flexibility was assessed using the 'sit and reach' test. This requires the subject to sit on the floor with legs out straight ahead. The feet (shoes off) are placed flat against the 'sit and reach' box. Both knees are held flat against the floor by the evaluator. The athlete leans forward slowly as far as possible and holds the greatest stretch for two seconds. Ballistic movement is not permitted, and the fingertips must remain level and the legs flat. The score is recorded as the distance before (negative) or beyond (positive) the toes (cm). This procedure was repeated three times and the best score recorded prior to the warm-up, which enabled a comparison of results to 'norm' values. (Normative values for the sit and reach test are presented in appendix 2).

Indices of neuromuscular and sensorimotor performance: Peak force, volitional and evoked EMD, constant and variable error.

On each assessment occasion (pre, 1h, 24h, 48h, 72h and 120h post EIMD) neuromuscular and sensorimotor performance indices were examined in both the control (preferred) and experimental (non-preferred) limbs in a random order. The peak score of three maximal volitional isometric muscle contractions of the knee flexor musculature was recorded to identify any alteration to PFv and associated indices of volitional electromechanical delay. The methodology for skin preparation and electrode placement was identical to that described in the general methodologies chapter (chapter 4.0). Types I and II sensorimotor performance assessment tasks were performed to examine the accuracy and consistency of sensorimotor performance during conditions of EIMD. Evoked electromechanical delay was also assessed during the immediate time period following the cessation of the final sensorimotor assessment task (complete relaxation was confirmed by the observation of the EMG signal). Please see the general methods chapter (chapter 4.0) for a full description of the assessment techniques for PF_V, EMD_E, EMD_V, and both constant and variable error during types I and II SMP tasks.

The same testing dynamometer was used for all neuromuscular assessments and the assessment co-ordinators executed the same role during test procedures. All motivational and verbal feedback was standardised to that of previous experiments. The raw data was collected and interpreted using Spike 2 (version 2.01, Cambridge Electronic Design LTD., U.K.) The experimental protocol is illustrated in figure 7.1.1.

Warm-up.

Prior to all isokinetic and/or neuromuscular assessment activity and following blood plasma CK, sit and reach and perceived pain rating assessments, a standardised warm-up was carried out on a cycle ergometer for 5 minutes with 60 Watts of resistance. This was followed by 5

minutes of stretching in the involved musculature. Further task-specific warm-up's were performed and are described in chapter 4, section 4.5.1. Following the initial baseline assessment protocol (pre), the experimental limb (non-preferred) was exercised to provoke EIMD using an eccentric isokinetic exercise protocol. Subsequent neuromuscular and sensorimotor assessments for the previously described performance indices were undertaken in both the control and experimental limbs at 1, 24, 48, 72, and 120 hours following the exercise intervention.

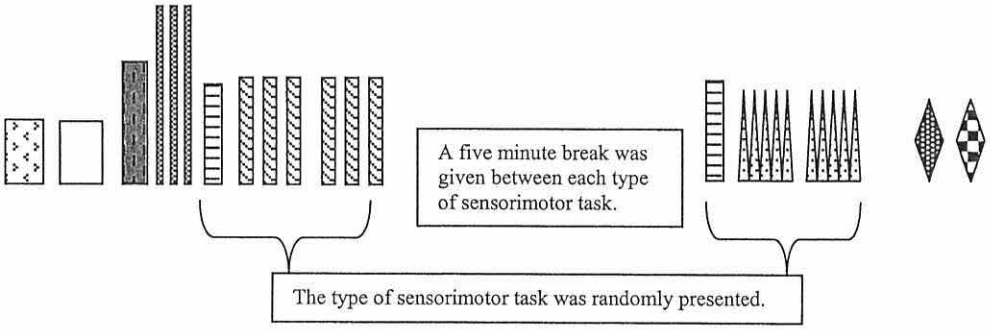
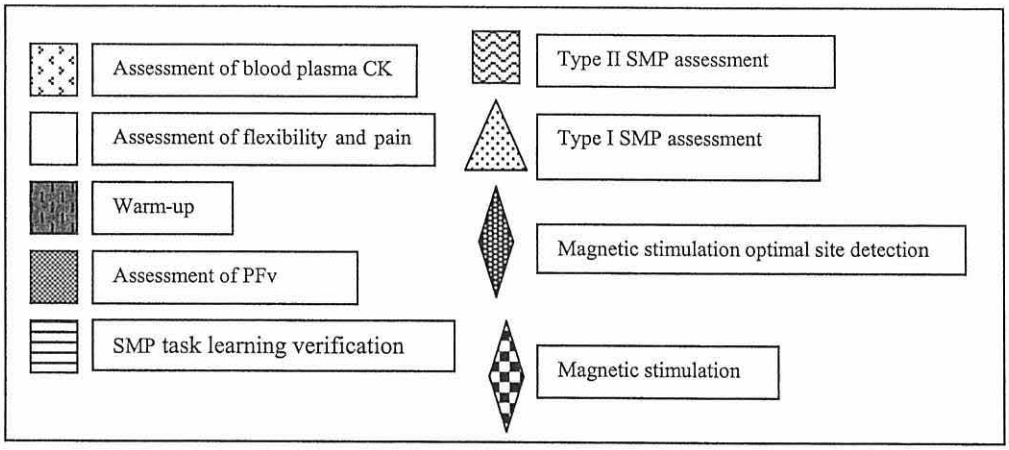


Figure 7.1.1. Physiological, neuromuscular and sensorimotor (type I and II tasks) assessment protocol for prior to and at 1, 24, 48, 72 and 120 hours following the eccentric isokinetic exercise protocol intervention.



Eccentric isokinetic exercise protocol for EIMD.

Subjects were familiarised with the Kin-Com© isokinetic dynamometer (500H, Chattecx, Chattanooga, TN, USA) and were securely positioned in an upright position on the equipment. A standardised hip flexion angle of 1.6 rad (90°) was measured using a goniometer. The pelvis and abdomen were stabilised to minimise rotation and standardise

body position, by means of velcro straps. The thigh of the experimental limb was also strapped to prevent hip flexion during testing. The axis of knee joint rotation was taken as the space between the lateral tibial condyle and lateral femoral condyle of the experimental knee and was aligned with the axis of rotation of the dynamometer. The general orientation of the participant on the dynamometer is illustrated in figure 7.1.2.

The ankle of the experimental limb was secured within a cuff incorporating a load transducer, to allow the assessment of the produced force and movement angle (the ankle cuff position [read from the scale on the lever arm] was recorded in order to accurately replicate subject orientation during the repeated-bout experiment, chapter 7.2). The isokinetic dynamometer was used to determine the working range of motion between 10° and 70° of flexion for each individual (0° being the subject's full extension). Further to a full description of the exercise protocol and a sub-maximal warm up routine, subjects undertook the EIMD protocol as validated by McHugh et al. (1999).

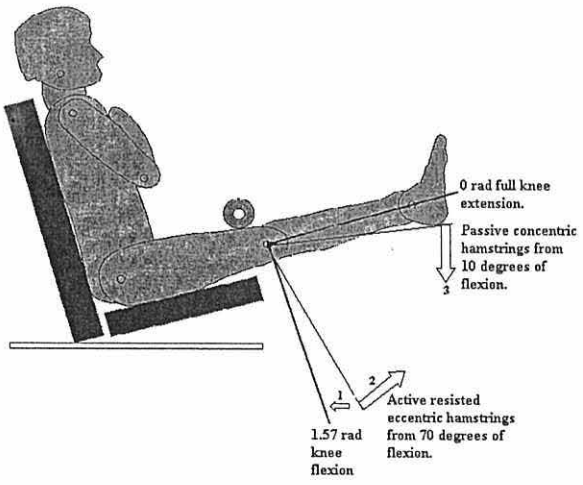


Figure 7.1.2. Standardised subject positioning on the isokinetic dynamometer illustrating the phases of limb movement during the eccentric exercise protocol (exercise induced muscle damage).

For the exercise induced muscle damage protocol, angular velocity was set at 60° per second through a range of motion starting from 70° to 10° knee flexion. The subject instigated

movement by actively contracting the hamstrings [1]. The isokinetic exercise protocol consisted of resisted isotonic eccentric hamstrings at 100% of concentric strength and (passive) eccentric quadriceps (i.e. subjects resisted the leg being moved towards full extension [2] and the test supervisor returned the leg to a flexed starting position [3]). The EIMD protocol consisted of 6 sets of 10 maximal repetitions, with a standardised one- minute rest between sets. Although the lever arm was repositioned by the experimenter, the Kin Com© maintained a constant 60° per second velocity and therefore the time between contractions was constant for each person. Participants were verbally encouraged by the experimenter to give a maximal effort at all times and target force values were observed on the testing equipment monitor as a source of visual feedback in order for the subject to maintain maximal output. This protocol has been previously employed and validated by Clarkson et al. (1987) and utilised by McHugh et al. (1999), Williams et al. (2002) (unpublished MSc data) and Marginson, (2003) (unpublished Ph. D data).

Statistical analyses.

Descriptive statistics (mean ±SD) were used to describe the indices of physiological (CK, pain and flexibility), neuromuscular (PFv, EMD_E, EMD_v) and sensorimotor performance (constant and variable error during types I and II assessment tasks) associated with the knee flexor musculature.

The descriptive markers for EIMD of blood plasma CK, perceived pain and flexibility were analysed by a fully within subjects repeated measures ANOVA to investigate the effects of EIMD over time [pre and 1, 24, 48, 72, and 120 hours post EIMD].

Separate two-factor ANOVAs (limb [control, experimental] by time [pre and 1, 24, 48, 72 and 120 hours post EIMD]) with repeated measures were used to analyse the effects of EIMD over time for each dependent variable (peak force, volitional and magnetically evoked EMD, constant and variable error).

In the event of a significant Mauchly's test of sphericity, the violation to the assumption of ANOVA was corrected for via the Greenhouse-Geisser adjustment of the critical F-value, as indicated by (GG). SPSS (V 9.0) was used to perform all statistical procedures.

7.1.3. Results.

Descriptive markers for exercise induced muscle damage.

Plasma creatine kinase [CK].

Group means and standard deviations for creatine kinase activity at pre, 1h, 24h, 48h, 72h and 120h post EIMD are illustrated in figure 7.1.3. Repeated measures ANOVA revealed a significant main effect for time ($F_{[1, 9]} = 8.0$, $p < 0.008$). CK levels increased dramatically between 48 and 72 hours post intervention and continued to rise towards the final assessment occasion (120 hours post intervention) compared to pre values (3314.0 ±3634.5 µl and 3778.5 ±3725.1 µl vs. 127.6 ±107.0 µl, respectively).

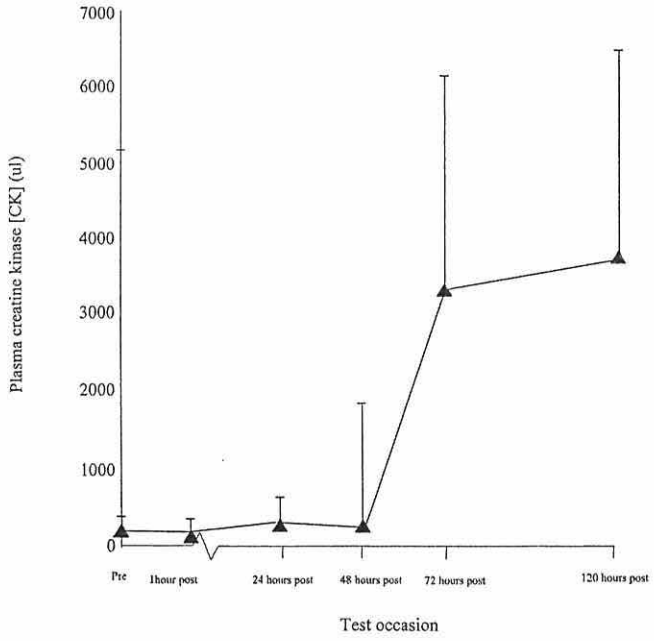


Figure 7.1.3. Group mean plasma creatine kinase (µl) [± SD] at pre and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

Perceived pain.

Group means and standard deviations for pain at pre, 1h, 24h, 48h, 72h and 120h post EIMD are presented in figure 7.1.4. Repeated measures ANOVA revealed a significant main effect for time ($F_{[1, 9]} = 15.0$, $p < 0.002$). It appears that the EIMD protocol induced a significant pain response in the affected knee flexor musculature when compared to pre levels, with an observed peak and plateau in response between 48 and 72 hours post intervention (a pain rating of 5.0 ±1.4 and 5.0 ±2.3 vs. 0.0 ±0.1 on the pain rating scale for measures at 48h, 72h

and pre EIMD intervention, respectively). The group mean pain rating appeared to be returning towards pre values at the final assessment occasion. This suggests either a lessening of symptoms or a tolerance of the discomfort (a group mean pain rating of 2.8 ±2.1 vs. 0.4 ±0.8 for 120h and pre EIMD, respectively).

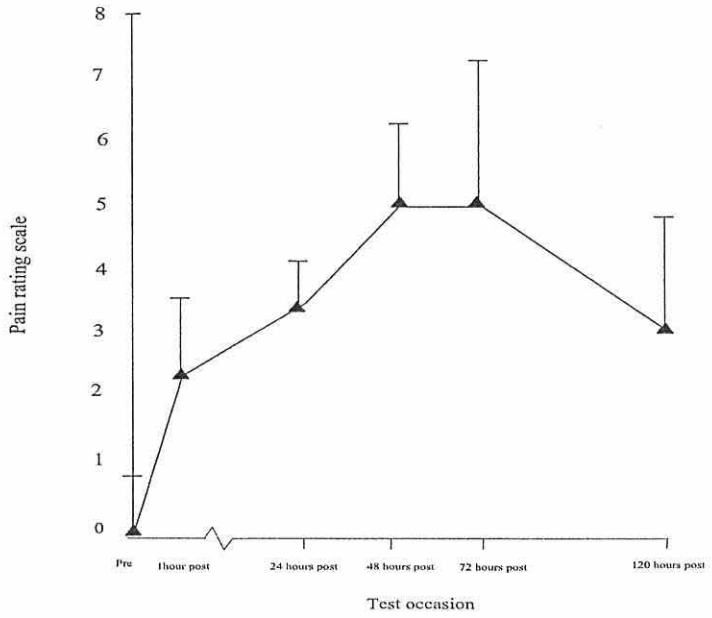


Figure 7.1.4. Group mean pain rating [± SD] at pre and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

Flexibility.

Group means and standard deviations for flexibility at pre, 1h, 24h, 48h, 72h and 120h post EIMD are presented in figure 7.1.5. Repeated measures ANOVA, revealed a significant main effect for time ($F_{[1, 9]} = 3.2$, $p<0.01$). Up to a 10 cm group mean impairment to flexibility associated with the affected knee flexor muscle group was observed at the 72 hours post intervention occasion when compared to pre values (21.0 ±13.7 cm vs. 30.0 ±5.9 cm, respectively) which coincided with the peak in pain response.

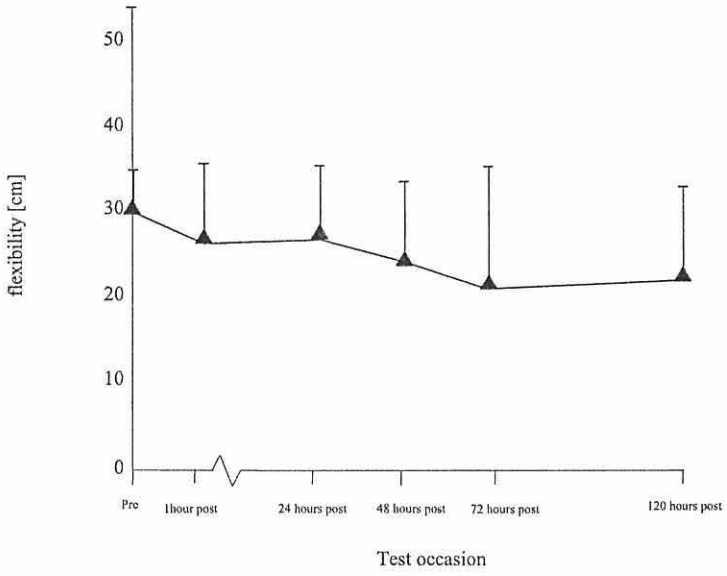


Figure 7.1.5. Group mean flexibility scores using the sit and reach test (cm) [± SD] at pre and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

Neuromuscular and sensorimotor parameters.

Peak force.

Group means and standard deviations for peak force at pre, 1h, 24h, 48h, 72h and 120h post EIMD are illustrated in figure 7.1.6.

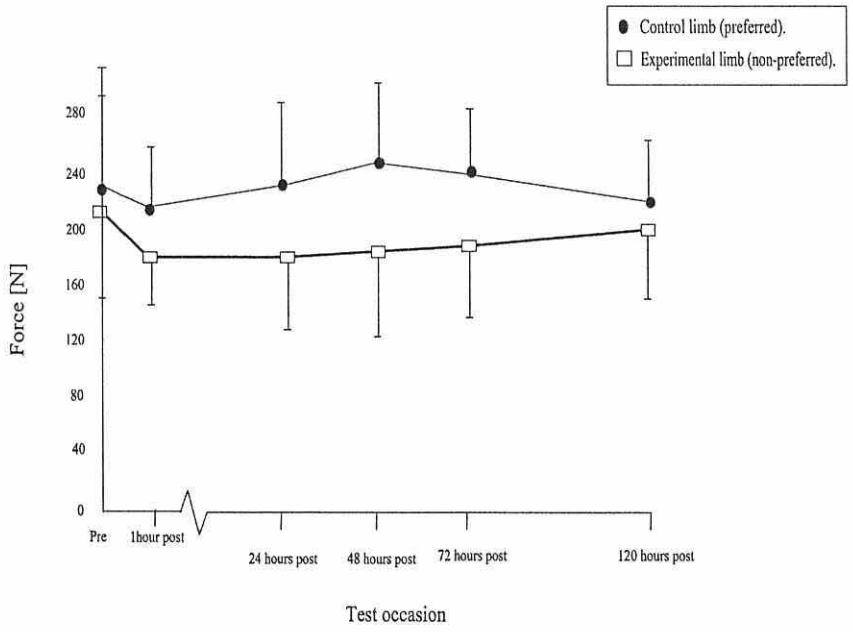


Figure 7.1.6. Group mean peak force (PFv) [N] [± SD] in both the control and experimental limbs at pre and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

A two factor ANOVA (limb [control; experimental] by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed a significant limb by time interaction ($F_{[1, 9]} = 5.4$, $p<0.001$). This suggests that peak force was preserved across all assessment phases in the control limb, but was altered in the experimental limb across assessment conditions.

Volitional EMD.

Group means and standard deviations for volitional EMD at pre, 1h, 24h, 48h, 72h and 120h post EIMD are presented in table 7.1.1.

	Volitional electromechanical delay [EMDv] (ms)	
Test occasion	**Control limb [Mean ±SD]**	**Experimental limb [Mean ±SD]**
Pre	63.2 (13.3)	52.2 (14.9)
1 hour post	61.7 (13.0)	57.5 (11.6)
24 hours post	60.4 (13.5)	55.7 (11.4)
48 hours post	58.4 (13.5)	56.3 (8.5)
72 hours post	59.5 (13.6)	57.8 (14.1)
120 hours post	56.9 (15.2)	57.9 (8.2)

Table 7.1.1. Group mean volitional EMD (ms) [± SD] in both the control and experimental limbs at pre, and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

A two factor ANOVA (limb [control; experimental] by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed no significant limb by time interaction. This suggests that volitional EMD was consistent in both the control and experimental limbs across all periods of assessment.

Magnetically evoked EMD.

Group means and standard deviations for magnetically evoked EMD at pre, 1h, 24h, 48h, 72h and 120h post EIMD are presented in table 7.1.2.

	Magnetically evoked electromechanical delay [EMD_E] (ms)	
Test occasion	Control limb [Mean ±SD]	Experimental limb [Mean ±SD]
Pre	27.8 (4.3)	26.4 (3.6)
1 hour post	26.1 (1.4)	26.0 (1.9)
24 hours post	26.3 (3.7)	28.2 (4.7)
48 hours post	29.2 (3.4)	29.3 (5.1)
72 hours post	26.4 (3.4)	25.1 (2.6)
120 hours post	25.9 (4.0)	25.8 (2.8)

Table 7.1.2. Group mean magnetically evoked EMD (ms) [± SD] in both the control and experimental limbs at pre, and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

A two factor ANOVA (limb [control; experimental] by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed no significant limb by time interaction. This suggests that magnetically evoked EMD was comparable in both the control and experimental limbs across the observed time periods.

Constant error (type I sensorimotor performance task).

Group means and standard deviations for constant error during the type I sensorimotor performance assessment task at pre, 1h, 24h, 48h, 72h and 120h post EIMD are presented in table 7.1.3.

	Constant error [type I SMP task] (% of target force)	
Test occasion	Control limb [Mean ±SD]	Experimental limb [Mean ±SD]
Pre	6.8 (9.5)	0.9 (10.1)
1 hour post	5.4 (17.2)	6.9 (14.8)
24 hours post	2.0 (10.3)	-10.2 (17.4)
48 hours post	7.8 (16.1)	-6.9 (35.0)
72 hours post	0.6 (16.1)	-6.5 (12.6)
120 hours post	5.4 (10.1)	-4.2 (8.9)

Table 7.1.3. Group mean constant error (expressed as a % of target force) [± SD] for the type I SMP task assessment in both the control and experimental limbs at pre, and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

A two factor ANOVA (limb [control; experimental] by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed no significant limb by time interaction. This

suggests that the eccentric exercise intervention did not significantly affect the accuracy of sensorimotor performance in the experimental limb.

Variable error (type I sensorimotor performance task).

Group means and standard deviations for variable error during the type I sensorimotor performance assessment task at pre, 1h, 24h, 48h, 72h and 120h post EIMD are presented in table 7.1.4.

	Variable error [type I SMP task] (% of target force)	
Test occasion	Control limb [Mean ±SD]	Experimental limb [Mean ±SD]
Pre	10.2 (2.8)	12.0 (3.8)
1 hour post	8.0 (2.0)	8.9 (5.9)
24 hours post	8.8 (3.4)	8.5 (6.6)
48 hours post	8.1 (4.7)	7.1 (3.7)
72 hours post	10.6 (5.8)	7.8 (3.4)
120 hours post	9.9 (3.9)	10.6 (7.4)

Table 7.1.4. Group mean variable error (expressed as a % of target force) [± SD] for the type I SMP task assessment in both the control and experimental limbs at pre, and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

A two factor ANOVA (condition [limb; control, experimental] by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed no significant limb by time interaction. This suggests that the consistency of sensorimotor performance during this type of SMP task was maintained between limbs and across all assessment occasions despite the presence of EIMD and the associated loss of PFv in the affected limb.

Constant error (type II sensorimotor performance task).

Group means and standard deviations for constant error during the type II sensorimotor assessment task at pre, 1h, 24h, 48h, 72h and 120h post EIMD are presented in table 7.1.5.

	Constant error [type II SMP task] (% of target force)	
Test occasion	Control limb [Mean ±SD]	Experimental limb [Mean ±SD]
Pre	-6.3 (5.5)	-8.1 (13.1)
1 hour post	-14.0 (11.0)	-8.2 (10.8)
24 hours post	-6.1 (12.0)	-11.6 (17.8)
48 hours post	-5.1 (11.7)	-12.7 (16.1)
72 hours post	-6.3 (10.4)	-8.2 (9.4)
120 hours post	-11.8 (8.6)	-12.8 (16.5)

Table 7.1.5. Group mean constant error (expressed as a % of target force) [± SD] for the type II SMP task assessment in both the control and experimental limbs at pre, and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

A two factor ANOVA (limb [control; experimental] by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed no significant limb by time interaction. This suggests that the accuracy of force replication during this type of SMP task was not disrupted by the presence of EIMD.

Variable error (type II sensorimotor performance task).

Group means and standard deviations for variable error during the type II sensorimotor performance assessment task at pre, 1h, 24h, 48h, 72h and 120h post EIMD are presented in table 7.1.6.

	Variable error [type II SMP task] (% of target force)	
Test occasion	Control limb [Mean ±SD]	Experimental limb [Mean ±SD]
Pre	12.8 (8.4)	9.4 (2.8)
1 hour post	7.9 (4.9)	10.9 (7.9)
24 hours post	9.0 (6.6)	7.7 (3.6)
48 hours post	12.4 (4.9)	5.8 (3.6)
72 hours post	8.9 (3.4)	6.9 (3.2)
120 hours post	5.5 (3.9)	7.2 (4.0)

Table 7.1.6. Group mean variable error (expressed as a % of target force) [± SD] for the type II SMP task assessment in both the control and experimental limbs at pre, and 1, 24, 48, 72 and 120 hours following the exercise induced muscle damage intervention.

A two factor ANOVA (condition [limb; control, experimental]) by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed no significant interaction. This

suggests that the variability in performance during this type of SMP task was consistent between limbs and was maintained across all assessment occasions. Thus, the presence EIMD did not appear to affect the consistency of the force replication response in the experimental limb.

7.1.4. Discussion.

Physiological indices of performance.

Creatine kinase.

This study demonstrated strong (indirect) evidence of muscle damage following an isokinetic eccentric exercise protocol on the knee flexor musculature of the non-preferred limb in female athletes. EIMD was confirmed via several descriptive markers of performance including a significant increase in plasma CK, which has been attributed to leakage of the muscle specific protein from damaged structures (Byrne and Eston, 1998).

Perceived pain.

The significant increase in delayed perceived soreness is consistent with a DOMS theory, as a peak was evident at 48 hours post EIMD. Muscles that experience DOMS usually show evidence of muscle fibre damage (z-band streaming), yet there is no evidence that muscle fibre damage causes DOMS (Byrne and Eston, 1998). The most likely explanation for DOMS is muscle damage and/or inflammation of the connective tissue, which leads to heightened pain sensitivity. The reduction in PFv coincided with the observed peak in perceived pain. Previous studies that have employed electrically super-imposed twitches to ensure maximal voluntary contraction, have generally found that subjects can fully activate painful muscles (Rutherford, et al., 1986; Newham, et al., 1987; Saxton et al., 1996). Therefore the pain experienced within the affected musculature may not have had a dramatic influence on the maximal activation of the knee flexors.

Flexibility.

Passive hamstring stiffness has been shown to correlate with the sit and reach test (Magnusson et al., 1997). A significant increase in stiffness of the experimental limb (reduced

flexibility) was observed following the intervention. Females have been shown to be generally more flexible than males (Hutchinson and Ireland, 1995; Huston and Wojtys, 1996). However, the present values may have been influenced by a general variation in inter-individual general flexibility. The degree of muscle compliance has been shown to influence the extent of damage, where stiffer individuals are more susceptible as a result of greater mechanical strain on the muscle fibres during active lengthening (McHugh et al., 1999). It was a clear observation in the present study that the more flexible subjects had a lower pain rating during the recovery from EIMD.

The findings from the physiological markers for EIMD are comparable to those of Williams et al. (2002) (unpublished MSc data) who observed a peak in CK at 72 hrs [7168 ± 6991.8μl], perceived pain at 48 hours post EIMD [pain 7.0 ±2.0 on a pain rating scale of 0 to 10] and stiffness (reduced flexibility) at 72 hours post EIMD [sit and reach value of 6.1± 8.0 cm] in male collegiate athletes (n=7) using the same EIMD protocol.

Neuromuscular and sensorimotor indices of performance.

Peak force.

Strength was maintained in the control limb following the EIMD protocol. However, up to a 20% reduction in PFv was observed for the experimental limb following the EIMD intervention. These results are in agreement with the findings of Komi, (2000), Byrne et al., (2001), McHugh et al., (2000) and Williams et al., 2002 (unpublished MSc data) that strength of the knee extensors was impaired following prolonged eccentric activation.

Ultra-structural changes within skeletal muscle fibres alongside metabolic and mechanical stressors may have contributed to the observed strength impairment following the EIMD protocol within the present study (Gleeson et al. 1998). The physiological mechanisms underpinning the observed deterioration in performance may include the 'popping sarcomere

theory' (Morgan, 1990) and perhaps the fact that the fast twitch fibres may have been preferentially damaged by the eccentric exercise stress, leading to an impaired rapid and forceful contraction of the affected muscle group. Clarkson and Newham (1995) described an assumption that relative changes in isometric force are comparable to those in concentric and eccentric torque, yet there is little evidence to substantiate this (Byrne and Eston, 1998). It could be the case that the observed isometric strength loss at the chosen assessment angle of 25 degrees of knee flexion does not fully reflect all aspects of force loss during dynamic knee stabilisation.

Volitional and magnetically evoked EMD.

Volitional EMD appeared to be preserved in the experimental limb during conditions of EIMD and a significant loss of PFv. This conservation of EMDv occurred despite the potential loss of the type II motor units during conditions of muscle damage. The relative preservation of the muscle switch on time in the affected limb may be attributed to oedema within the affected muscle and thus an increase in both intra-muscular pressure and temperature, which may have actively 'stiffened' the affected muscle group. Such changes to the intra-muscular environment may therefore have counteracted any actual changes to the conduction of electrical activity and ultimately the excitation-coupling process (Winter and Brookes, 1991). Thus, EMD could play an important role in joint preservation, in that the rapid EMD response was not compromised during disruption to peak force.

Since magnetically evoked EMD is not restricted by inhibition from the central and peripheral nervous system (Gleeson, 2001) it was anticipated that this type of neuromuscular performance would be unaffected during conditions of muscle damage. This expectation was confirmed in the present findings. Therefore, it appears that the availability of the fast twitch muscle fibres was not compromised during evoked supra-maximal stimulations of the knee

flexor musculature. This finding may be attributed to the magnetic stimulation having accessed a greater pool of motor units during passive muscular activation and also the increase in intra-muscular temperature having facilitated the conduction of the nerve impulse to the knee flexors of the experimental limb.

Constant error (type I sensorimotor performance task).

Strength in terms of peak force was re-assessed at the start of every follow up test occasion, allowing the sensorimotor target to be adjusted relative to the maximal daily performance of each limb. This allowed the subject to reproduce an achievable target force during EIMD. No significant interaction was observed for measures of constant error during the type I SMP task across all test occasion comparisons. The force reproduction task may have been mediated by the pain experienced during active isometric contraction in the experimental limb following EIMD. This strategy may therefore have assisted subjects in the judgement of force by replicating a certain level of discomfort during each contraction of the affected musculature. Several subjects did report the fact that they had utilised the level of pain to assist in the judgement of isometric force, but did not perceive the force to have been under target. The type I SMP task is time restricted, and therefore subjects had minimal opportunity to judge or indeed alter the level of force being produced via feedback control. This type of sensorimotor response was consistent across assessments. The sensorimotor system may therefore utilise sensory feedback from pain receptors in order to detect any disruption to the internal environment of the muscle. The restricted threshold for maximal force production during conditions of EIMD did not impair the ability to reproduce 50% of peak force relative to the daily PFv for each assessment session during the type I style of SMP task.

Constant error (type II sensorimotor performance task).

No significant disruption to constant error during the type II SMP task was observed despite a significant disruption to peak force in the experimental limb. Colebatch and McCloskey (1987) suggested that a stiffer muscle effectively facilitates feedback neuromuscular control mechanisms. Thus, a relative improvement in feedback may explain the lack of significant differences following the eccentric damage in the experimental limb.

Variable error types I and II SMP task.

Sensorimotor performance in terms of variable error during both types I and II SMP tasks was not significantly disrupted despite the presence of EIMD in the experimental (non-preferred) limb. Thus the loss of PFv did not affect the sensorimotor awareness of the fast twitch muscle fibres during conditions of EIMD for the type I SMP task which requires rapid activation of the musculature.

7.1.5. Summary.

This study investigated the effects of EIMD on sensorimotor and neuromuscular performance indices in both the experimental (non-preferred) and control (preferred) limbs of ten female athletes. Peak force, volitional and magnetically evoked EMD, constant and variable error during types I and II SMP tasks was assessed prior to and 1, 24, 48, 72 and 120 hours following an eccentric isokinetic exercise protocol in the non-preferred hamstrings musculature. Contra-lateral (control) limb comparisons were also undertaken using the hamstrings musculature of the preferred limb at all test occasions. EIMD was confirmed via a significant increase in both plasma CK and perceived pain alongside a significant reduction in both the flexibility and peak volitional force associated with the knee flexors of the experimental limb.

Both volitional and evoked electromechanical delay values were consistent across assessment occasions and limbs. This suggests that either EMD is a significant factor in the maintenance of joint stability during conditions of EIMD or that EIMD does not affect volitional and evoked neuromuscular activation. A rotation of the high threshold motor units may also explain how the switch on time of the knee flexors was seemingly unaffected during muscle damage. Constant error values during the type I SMP task showed a consistent pattern of response between limbs and test occasions. Pain may have been utilised to moderate isometric force in both types of sensorimotor test as the body's innate defence against further injury and damage to muscular structures within the limb. The use of pain for sensory feedback may therefore have assisted in the maintenance of a consistent level of force reproduction during conditions of EIMD thus 'masking' any actual alteration to sensorimotor performance. The loss of PFv did not appear to affect sensorimotor awareness and communication with the fast twitch muscle fibres: even during the type I SMP task that requires rapid activation of the musculature.

In summary, the presence of EIMD was confirmed in this experiment via several physiological descriptives including elevated levels of creatine kinase within the blood, a reduced range of movement and an increase in pain upon active movement of the affected limb. Sensorimotor performance in terms of both the accuracy and consistency of response to types I and II dynamic assessment tasks did not change at a knee flexion angle of 25° despite a 20% loss of overall strength following the eccentric exercise intervention. This may reinforce the fact that sensorimotor performance is the body's underlying apparatus for the preservation of joint integrity that functions despite significant internal disruption the muscle tissue. This sensory protection may be complemented by consistent neuromuscular activation enabling the body to activate a 'damaged' limb in a timely enough manner to prevent further disruption to the joint and surrounding structures.

7.2. The effects of a repeated-bout of exercise induced muscle damage on neuromuscular and sensorimotor performance of the knee flexors in female athletes.

7.2.1. Introduction.

It was established in chapter 7.1 that an initial-bout exposure to an EIMD protocol in the knee flexors of the experimental (non-preferred) limb in ten female athletes induced a significant physiological response for the parameters of blood plasma creatine kinase, perceived pain and flexibility of the affected muscle group. Neuromuscular performance in terms of volitional peak force was also significantly affected by means of a 20% group mean reduction in performance of the experimental limb following the intervention. Both the volitional and evoked EMD response was however preserved in both limbs across all assessment occasions. Similarly, sensorimotor performance in terms of both the accuracy and consistency of force replication during types I and II SMP tasks was not significantly affected in the knee flexors of the non-preferred limb, despite the presence of muscle damage.

Previous research has shown a less pronounced physiological and neuromuscular response during a second or 'repeated-bout' exposure to the same exercise intervention protocol on the knee extensor or 'quadriceps' musculature (Brown, et al., 1997; McHugh, et al., 1999; McHugh, 2000). This protective effect has been found to last up to six weeks (Nosaska et al., 1995). The physiological mechanisms involved in the repeated-bout effect have been recently explored; for a comprehensive review of such mechanisms the reader is directed to McHugh et al., (1999).

It was not known whether the non-significant influence of EIMD on sensorimotor performance and neuromuscular switch on times of the knee flexor musculature which was observed in chapter 7.1 would also be evident during a second or repeated-bout exposure to

the same chronic type of neuromuscular stress (EIMD). This study therefore investigated whether the findings of Nosaska et al., (1995), Brown et al., (1997), McHugh et al., (1999) and McHugh, (2000) are indeed applicable to the knee flexor musculature. A further aim of this experiment was to determine whether a repeated-bout exposure to types I and II SMP assessment tasks during potential EIMD could actually improve sensorimotor performance of the knee flexors in female athletes.

7.2.2. Methods.

Subjects.

Unfortunately one subject from the initial-bout EIMD study (chapter 7.1) received an injury to the hamstrings musculature of her experimental limb immediately prior to the repeated-bout study and was therefore unable to participate in the second phase of assessment. Nine adult females (age 21.3 ±2.5 years; height 1.66 ±0.07 m; body mass 67.1 ±7.2 kg [mean ± SD]) gave their informed consent to participate in this study. The sample comprised collegiate and semi-professional team-game athletes.

The experimental design for this section of the chapter is identical to that described in chapter 7.1.1 as an exact replication of the EIMD and sensorimotor assessment protocol was used to mimic the initial-bout of exercise induced muscle damage and follow-up assessment criteria. This enabled a direct comparison between both neural and sensorimotor responses to a repeated-bout exposure to eccentric exercise stress.

Physiological, neuromuscular and sensorimotor indices of performance capacity.

All physiological, neuromuscular and sensorimotor indices were repeated as outlined in section 7.1.2. All physiological, neuromuscular and sensorimotor assessments were performed as near to the same time of day as possible (±1h).

Repeated-bout EIMD.

The isokinetic protocol for the repeated-bout of EIMD during knee extension and flexion movements was undertaken in accordance with the protocol and experimental conditions outlined in chapter 7.1. The repeated-bout was undertaken within the six-week window of effect as described by Nosaska et al., (1995) and the EIMD protocol was conducted at the same time of day as in chapter 7.1 (morning assessment occasions to within ±1hour).

Electrode placement for the assessment of EMG was standardised for the repeated-bout by accurate measurement and recording of the electrode placement during the initial phase of the experiment (chapter 7.1) as the distance from the posterior crease in knee to the centre of first electrode. The inter-electrode distance was also accurately monitored in accordance with the values taken from chapter 7.1. This aimed to reduce the potential for error in inter-trial assessments of the same muscle (Gleeson, 2001).

Statistical analyses.

Descriptive statistics (mean ±SD) were used to describe the indices of physiological (CK, perceived pain and flexibility) neuromuscular (PFv, EMD_E, EMD_v) and sensorimotor performance (constant and variable error in types I and II assessment tasks) associated with the knee flexor musculature.

Separate two factor ANOVAs (condition [initial-bout, repeated-bout] by time [pre and 1, 24, 48, 72 and 120 hours post EIMD]) with repeated measures were used to analyse the effects of EIMD over time for the descriptive markers for EIMD of blood plasma CK, perceived pain and flexibility.

Separate three factor ANOVAs (limb [control, experimental] by time [pre and 1,24,48,72,120 hours post EIMD] by bout [initial-bout, repeated-bout]) with repeated measures were used to analyse the effects of the EIMD over time for each dependent variable (PFv, EMD_E, EMD_v, constant and variable error during types I and II SMP assessment tasks).

In the event of a significant Mauchly's test of sphericity, the violation to the assumption of ANOVA was corrected for via the Greenhouse-Geisser adjustment of the critical F-value, as indicated by (GG). SPSS (V 9.0) was used to perform all statistical procedures.

7.2.3. Results.

Initial-bout versus repeated-bout plasma creatine kinase.

Group means and standard deviations for creatine kinase activity at pre, 1h, 24h, 48h, 72h and 120h post both the initial-bout and repeated bout of EIMD are presented in figure 7.2.1. A two factor ANOVA (condition [initial-bout, repeated-bout]) by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed a significant condition by time interaction ($F_{[5,40]} = 4.71$, $p<0.02$). This suggests that a difference was observed between the blood plasma creatine kinase results from the initial-bout and repeated-bout of the EIMD protocol. Upon visual inspection of the data, it appears that the group mean blood plasma CK response was less pronounced during the repeated-bout follow-up occasions when compared to that of the initial-bout of EIMD at 72 and 120h post intervention (803.1± 1135.6 µl vs. 3314.6 ± 3634.5µl and 740.8 ±924.0 µl vs. 3778.5 ± 3725.1µl, respectively). This contributed to the observed significant interaction. A repeated-bout effect may therefore have been witnessed in terms of the severity of CK response following EIMD in the knee flexors.

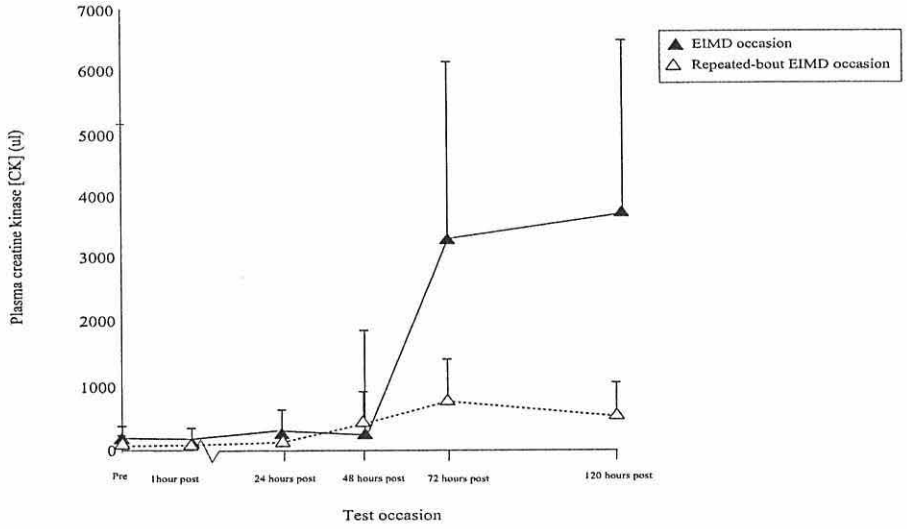


Figure 7. 2. 1. Group mean plasma creatine kinase (µl) [± SD] at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Initial-bout versus repeated-bout perceived pain.

Group means and standard deviations for perceived pain at pre, 1h, 24h, 48h, 72h and 120h post both the initial-bout and repeated bout of EIMD are presented in table 7.2.1. A two factor ANOVA (condition [initial-bout, repeated-bout]) by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed no significant condition by time interaction. This suggests that the pattern of pain response was not significantly different between bouts of EIMD and therefore a repeated-bout effect was not evident in terms of pain response. The peak in pain rating was observed 48h post intervention for both intervention occasions.

	Perceived pain	
Test occasion	**Initial-bout [Mean ±SD]**	**Repeated-bout [Mean ±SD]**
Pre	0 (0.8)	0 (0.9)
1 hour post	2.3 (1.7)	1.0 (1.0)
24 hours post	3.5 (0.7)	2.8 (0.8)
48 hours post	5.0 (1.4)	3.7 (0.9)
72 hours post	5.0 (2.3)	2.9 (1.5)
120 hours post	2.7 (2.0)	1.4 (1.2)

Table 7. 2.1. Group mean pain rating [± SD] at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Initial-bout versus repeated-bout flexibility.

Group means and standard deviations for flexibility (sit and reach) measures at pre, 1h, 24h, 48h, 72h and 120h post the initial-bout and repeated-bout of EIMD are presented in table 7.2.2. A two factor ANOVA (condition [initial-bout, repeated-bout]) by time [pre, 1h, 24, 48, 72, 120 hours post EIMD]) with repeated measures revealed no significant condition by time interaction. This suggests that the group mean flexibility was not significantly different between bouts of EIMD and that the repeated-bout theory was not evident in terms of preserved flexibility within the affected musculature during the repeated –bout EIMD occasion. The greatest loss of flexibility was observed between 48h and 72h post intervention for both the initial-bout and repeated-bout occasions.

	Flexibility (cm)	
Test occasion	Initial-bout [Mean ±SD]	Repeated- bout [Mean ±SD]
Pre	30 (5.9)	27 (7.7)
1 hour post	28 (7.3)	27 (8.1)
24 hours post	27 (7.2)	26 (8.2)
48 hours post	25 (8.7)	25 (9.5)
72 hours post	21(13.7)	25 (11.0)
120 hours post	25(12.3)	26 (9.3)

Table 7.2.2. Group mean flexibility (cm) [± SD] at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Neuromuscular and sensorimotor parameters.

Initial-bout versus repeated-bout peak force.

Group means and standard deviations for volitional peak force at pre, 1h, 24h, 48h, 72h and 120h post initial-bout and repeated-bout of EIMD in both the control and experimental limbs are presented in table 7.2.3. A three factor ANOVA (limb [control, experimental] by time [pre and 1, 24, 48, 72, 120 hours post EIMD] by bout [initial-bout, repeated-bout]) with repeated measures revealed no significant interaction. This suggests that while the performance of the control limb remained constant, both episodes of eccentric exercise provoked a loss of strength performance in the experimental limb.

The interaction for time*limb was found to be significant ($F_{[1, 8]} = 5.19$, $p<0.01$). Thus, the repeated-bout effect did not appear to be evident in terms of protection against the severity of damage to the knee flexor musculature (experimental limb) during a second exposure to the EIMD protocol.

	Peak force (PFv) (N)			
Test occasion	**Experimental limb Initial-bout [Mean ±SD]**	**Control limb Initial-bout [Mean ±SD]**	**Experimental limb Repeated-bout [Mean ±SD]**	**Control limb Repeated-bout [Mean ±SD]**
Pre	226 (59.7)	238 (47.7)	223 (64.5)	234 (52.6)
1 hour post	187 (36.5)	231 (39.5)	203 (76.6)	215 (38.2)
24 hours post	180 (51.2)	239 (46.3)	204 (66.3)	242 (54.4)
48 hours post	187 (55.4)	247 (46.7)	189 (66.4)	237 (51.7)
72 hours post	190 (58.4)	242 (42.6)	199 (43.5)	239 (67.7)
120 hours post	211 (52.7)	222 (42.6)	208 (55.1)	225 (58.3)

Table 7.2.3. Group mean peak force (PFv) [N] [± SD] in both the control and experimental limbs at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Initial-bout versus repeated-bout volitional EMD.

Group means and standard deviations for volitional EMD at pre, 1h, 24h, 48h, 72h and 120h post initial-bout and repeated-bout of EIMD in both the control and experimental limbs are presented in table 7.2.4. A three factor ANOVA (limb [control, experimental] by time [pre and 1, 24, 48, 72, 120 hours post EIMD] by bout [initial-bout, repeated-bout]) with repeated measures revealed no significant interaction. This suggests no significant difference in volitional EMD response between limbs and bouts across time. The two-way interaction for limb*bout was however found to be significant ($F_{[1, 8]} = 6.62$, $p<0.03$) which suggests that there was a difference in the pattern of EMDv over the two separate bouts of EIMD. The control limb was the dominant limb within this study, which could explain the difference between EMD responses and therefore the observed interaction.

	Volitional EMD (EMDv) (ms)			
Test occasion	Experimental limb Initial-bout [Mean ±SD]	Control limb Initial-bout [Mean ±SD]	Experimental limb Repeated-bout [Mean ±SD]	Control limb Repeated-bout [Mean ±SD]
Pre	52.2 (14.9)	63.2 (13.3)	53.6 (10.8)	57.6 (10.8)
1 hour post	57.5 (11.6)	61.7 (13.0)	59.9 (10.6)	54.2 (10.6)
24 hours post	55.7 (11.4)	60.4 (13.5)	58.2 (8.2)	55.0 (8.2)
48 hours post	56.3 (8.5)	58.4 (13.5)	57.5 (18.0)	54.0 (18.0)
72 hours post	57.8 (14.1)	59.5 (13.6)	54.6 (11.8)	53.6 (11.8)
120 hours post	57.9 (8.2)	56.9 (15.2)	50.5 (13.3)	52.5 (13.3)

Table 7.2.4. Group mean volitional EMD (EMDv) [ms] [± SD] in both the control and experimental limbs at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Initial-bout versus repeated-bout magnetically evoked EMD.

Group means and standard deviations for magnetically evoked EMD at pre, 1h, 24h, 48h, 72h and 120h post initial-bout and repeated-bout of EIMD in both the control and experimental limbs are presented in table 7.2.5. A three factor ANOVA (limb [control, experimental] by time [pre and 1, 24, 48, 72 and 120 hours post EIMD] by bout [initial-bout, repeated-bout]) with repeated measures revealed no significant interaction. This suggests no significant difference in evoked EMD response between limbs and bouts across time.

No further significant interactions were observed for either time*limb or limb*bout suggesting no apparent difference between the pattern of EMD_E response in the control and experimental limbs across time. This indicates a similar pattern of evoked neuromuscular activation was observed in the experimental limb during both bouts of EIMD.

Test occasion	Evoked EMD (EMD_E) (ms)			
	Experimental limb Initial-bout [Mean ±SD]	Control limb Initial-bout [Mean ±SD]	Experimental limb Repeated-bout [Mean ±SD]	Control limb Repeated-bout [Mean ±SD]
Pre	26.4 (3.6)	27.8 (4.3)	25.3 (2.6)	27.5 (4.7)
1 hour post	26.0 (1.9)	26.1(1.4)	28.0 (6.0)	25.5 (3.9)
24 hours post	28.2 (4.7)	26.3(3.7)	27.7 (3.0)	26.0 (3.5)
48 hours post	29.3 (5.1)	29.2(3.4)	26.0 (3.2)	26.3 (3.0)
72 hours post	25.1 (2.6)	26.4(3.4)	24.2 (4.3)	25.2 (4.8)
120 hours post	25.8 (2.9)	25.9(4.0)	24.6 (4.1)	25.6 (4.4)

Table 7.2.5. Group mean magnetically evoked EMD (EMD_E) [ms] [± SD] in both the control and experimental limbs at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Initial-bout versus repeated-bout constant error (type I sensorimotor performance task).

Group means and standard deviations for constant error during the type I (time regulated) sensorimotor performance assessment in both the control and experimental limbs at pre, 1h, 24h, 48h, 72h and 120h post the initial-bout and repeated-bout of EIMD are presented in table 7.2.6. A three factor ANOVA (limb [control, experimental] by time [pre and 1, 24, 48, 72, 120 hours post EIMD] by bout [initial-bout, repeated-bout]) with repeated measures revealed no significant interaction. This suggests a preservation of sensorimotor performance in that the pattern of response during the initial-bout and repeated-bout of EIMD was not dissimilar (no time*limb interaction) and thus there was no significant disruption to the accuracy of force replication in the type I task during both bouts of EIMD. However, the limb*bout interaction was found to be significant ($F_{[1, 8]} = 5.88$ ($p < 0.04$) suggesting that the repeated-bout effect was not witnessed for sensorimotor performance in the experimental limb. Therefore, no significant enhancement of time-regulated force replication response was witnessed during a second exposure to 'negative' conditions of EIMD.

	Constant error (type I SMP task) (% of target force)			
Test occasion	Experimental limb Initial-bout [Mean ±SD]	Control limb Initial-bout [Mean ±SD]	Experimental limb Repeated-bout [Mean ±SD]	Control limb Repeated-bout [Mean ±SD]
Pre	0.9 (10.1)	6.8 (9.5)	1.1 (18.4)	1.8 (12.6)
1 hour post	6.9 (14.8)	5.4 (17.2)	6.3 (22.2)	7.6 (14.9)
24 hours post	-10.2 (17.4)	2.0 (10.3)	5.9 (12.8)	-0.5 (9.5)
48 hours post	-6.9 (35.0)	7.8 (16.1)	-5.9 (11.4)	-2.7 (10.8)
72 hours post	-6.5 (12.6)	0.6 (16.1)	0.0 (13.1)	0.5 (20.2)
120 hours post	-4.1 (8.9)	5.4 (10.1)	-2.7 (11.0)	2.7 (19.3)

Table 7.2.6. Group mean constant error (% of target force) [type I SMP assessment] [± SD] in both the control and experimental limbs at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Initial-bout versus repeated-bout variable error (type I sensorimotor performance task).

Group means and standard deviations for variable error during the type I (time regulated) sensorimotor performance at pre, 1h, 24h, 48h, 72h and 120h post repeated-bout of EIMD in both the control and experimental limbs are presented in table 7.2.7. A three factor ANOVA (limb [control, experimental] by time [pre and 1, 24, 48, 72, 120 hours post EIMD] by bout [initial-bout, repeated-bout]) with repeated measures revealed no significant interaction. This suggests that the variability in performance was similar between limbs and bouts. Therefore, the repeated-bout occasion did not lead to any enhancement of sensorimotor response in terms of an improvement in the consistency of the 'rapid' force replication performance in the experimental limb during conditions of EIMD.

	Variable error (type I SMP task) (% of target force)			
Test occasion	Experimental limb Initial-bout [Mean ±SD]	Control limb Initial-bout [Mean ±SD]	Experimental limb Repeated-bout [Mean ±SD]	Control limb Repeated-bout [Mean ±SD]
Pre	12.0 (3.8)	10.2 (2.8)	11.2 (5.5)	13.3 (5.1)
1 hour post	8.9 (5.9)	8.0 (2.0)	9.8 (5.5)	11.0 (5.5)
24 hours post	8.5 (6.6)	8.8 (3.4)	7.1 (5.6)	9.1 (3.9)
48 hours post	7.1 (3.7)	8.1 (4.7)	8.9 (4.3)	11.9 (5.6)
72 hours post	7.8 (3.4)	10.6 (5.8)	9.4 (5.5)	8.0 (8.0)
120 hours post	10.6 (7.4)	9.9 (3.9)	7.4 (5.0)	11.9 (6.2)

Table 7.2.7. Group mean variable error (% of target force) [type I SMP assessment] [± SD] in both the control and experimental limbs at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Initial-bout versus repeated-bout constant error (type II sensorimotor performance task).

Group means and standard deviations for constant error during the type II (self regulated) sensorimotor performance assessment in both the control and experimental limbs at pre, 1h, 24h, 48h, 72h and 120h post the initial-bout and repeated-bout of EIMD are presented in table 7.2.8. A three factor ANOVA (limb [control, experimental] by time [pre and 1, 24, 48, 72, 120 hours post EIMD] by bout [initial-bout, repeated-bout]) with repeated measures revealed no significant interaction. This suggests a preservation of sensorimotor performance (constant error during the type II SMP task) in that the pattern of response during the initial-bout and repeated-bout of EIMD was not dissimilar (no significant time*limb or limb*bout interaction). However, a significant main effect for limb was observed ($F_{[1,8]} = 8.14$, $p < 0.02$) suggesting that the response for one limb was significantly different than the other across trials. This finding may be attributed to the fact that the control limb was the preferred limb and may have demonstrated a more refined level of accuracy during the self-regulated force replication task.

The significant loss of peak volitional force did not therefore appear to have a significant impact on the accuracy of sensorimotor performance during the initial-bout and repeated-bout

of EIMD in the experimental limb. Subjects may have utilised pain to reproduce a level of discomfort rather than force during the familiarisation period. This strategy may have prevented the detection of any actual differences in sensorimotor performance between limbs during conditions of EIMD.

	Constant error (type II SMP task) (% of target force)			
Test occasion	**Experimental limb** **Initial-bout [Mean ±SD]**	**Control limb** **Initial-bout [Mean ±SD]**	**Experimental limb** **Repeated-bout [Mean ±SD]**	**Control limb** **Repeated-bout [Mean ±SD]**
Pre	-8.1 (13.1)	-6.3 (5.5)	-19.0 (7.6)	-10.9 (7.6)
1 hour post	-8.2 (10.8)	-14.0 (11.0)	-5.3 (13.5)	-5.1 (13.5)
24 hours post	-11.6 (17.8)	-6.1 (12.0)	-7.3 (17.4)	-2.3 (17.4)
48 hours post	-12.7 (16.1)	-5.1 (11.7)	-10.1 (7.9)	-3.7 (7.9)
72 hours post	-8.2 (9.4)	-6.3 (10.4)	-10.3 (14.0)	-3.8 (14.0)
120 hours post	-12.8 (16.5)	-11.8 (8.6)	-11.9 (14.4)	-8.0 (14.4)

Table 7.2.8. Group mean constant error (% of target force) [type II SMP assessment] [± SD] in both the control and experimental limbs at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

Initial-bout versus repeated-bout variable error (type II sensorimotor performance task).

Group means and standard deviations for variable error during the type II (self regulated) sensorimotor performance at pre, 1h, 24h, 48h, 72h and 120h post the initial-bout and repeated-bout of EIMD in both the control and experimental limbs are presented in table 7.2.9. A three factor ANOVA (limb [control, experimental] by time [pre and 1, 24, 48, 72, 120 hours post EIMD] by bout [initial-bout, repeated-bout]) with repeated measures revealed no significant interaction. This suggests that the variability in performance was similar between bouts. The limb*time interaction did however prove significant ($F_{[5, 40]} = 3.00$, $p<0.02$). This suggests a difference in the pattern of variable error response across assessment occasions. The presence of pain following eccentric damage in the knee flexor musculature of the non-preferred limb may have facilitated the consistency of force judgement during conditions of EIMD, contributing to the observed interaction.

	Variable error (type II SMP task) (% of target force)			
Test occasion	Experimental limb Initial-bout [Mean ±SD]	Control limb Initial-bout [Mean ±SD]	Experimental limb Repeated-bout [Mean ±SD]	Control limb Repeated-bout [Mean ±SD]
Pre	9.4 (2.8)	12.8 (8.4)	7.9 (6.9)	8.4 (5.0)
1 hour post	10.9 (7.9)	7.9 (4.9)	6.5 (4.6)	9.5 (5.1)
24 hours post	7.7 (3.6)	9.0 (6.6)	7.0 (4.2)	10.5 (6.7)
48 hours post	5.8 (3.6)	12.4 (4.9)	6.7 (4.7)	9.3 (5.0)
72 hours post	6.9 (3.2)	8.9 (3.4)	8.8 (4.7)	8.4 (4.8)
120 hours post	7.2 (4.0)	5.5 (3.9)	8.8 (4.1)	7.4 (3.7)

Table 7.2.9. Group mean variable error (% of target force) [type II SMP assessment] [± SD] in both the control and experimental limbs at pre and 1, 24, 48, 72 and 120 hours following the initial-bout and repeated-bout of exercise induced muscle damage intervention.

7.2.4. Discussion.

Physiological indices of performance.

Creatine kinase, flexibility and perceived pain.

The results from the second phase of this experiment suggest that overall the repeated-bout effect may not have been witnessed following an isokinetic eccentric exercise intervention on the hamstrings musculature of the non-preferred limb in female athletes. Blood plasma creatine kinase was the only physiological marker to have demonstrated a significantly different pattern of response between the two bouts of EIMD. The level of blood plasma creatine kinase reflects leakage of the muscle specific protein from damaged structures (Byrne and Eston, 1998). The magnitude of plasma CK response was less pronounced during the 72h and 120h follow-up occasions during the second exposure to EIMD. The pattern of response for the indices of perceived pain and flexibility were not statistically different between the initial-bout and repeated-bout of EIMD. This lack of a repeated-bout effect is somewhat in opposition to previous research that found a lesser physiological and neuromuscular response during a repeated-bout of EIMD in the knee extensor musculature (Brown et al., 1997; McHugh et al., 1999; McHugh, 2000).

Neuromuscular and sensorimotor indices of performance.

Peak force.

Several authors have attributed the repeated-bout effect to either a change in neural recruitment during the repeated-bout which limits the extent of damage or to the belief that the workload for the repeated-bout is distributed over a greater number of active fibres (Golden, Graves, Buchanan, and Dudley, 1991; Mair, Mayr and Muller, 1995; Nosaska and Clarkson, 1995; Pizza, Davis and Henrickson, 1996; Byrne and Eston, 1998). The outcome from the present experiment does not appear to concur with such theories as volitional peak force [PFv] in the knee flexors (non-preferred limb) was significantly disrupted during *both*

follow-up phases of the EIMD interventions. Thus, the repeated-bout effect was not witnessed in the knee flexors following the second exposure to EIMD. The observed decline in PFv coincided with the peak in perceived pain and reduction in flexibility of the affected muscle group for both bouts of EIMD and is consistent with symptoms of DOMS. The intensity of the chosen EIMD protocol, taken from previous application to the knee extensor muscle group, was reduced prior to its current application to the knee flexors (due to the severity of EIMD witnessed in pilot studies). However, a substantial amount of muscle damage was still reported in terms of physical symptoms (anecdotally, subjects were physically impaired whilst trying to walk and use stairs) during both phases of the intervention. As there is limited previous research into the repeated-bout effect associated with the knee flexor musculature of female athletes, the current EIMD protocol was perhaps too aggressive given the cross-sectional area of the hamstrings musculature. Hence, the repeated-bout effect may not have been witnessed due to the magnitude of damage caused by the eccentric exercise protocol used in this experiment.

Also, the time period between the initial and repeated-bout of EIMD intervention within this study coincided with the University of Wales, Bangor's Christmas break, a reportedly inactive period athletically for most subjects. Therefore it is possible that the potential for muscular 'de-training' could have also exaggerated the effects of the repeated-bout of EIMD. Furthermore, the six weeks window of protection previously suggested for the quadriceps musculature (Nosaska et al., 1995) may not be applicable directly to the knee flexors.

It is hereby recommended that future research into the effects of EIMD in the hamstring musculature be performed at least seven days in advance of both pre-season training and the competitive soccer season to allow the recovery of strength in all players. This schedule would be important in minimising any increased risk of knee injury associated with

participation during sub-optimal strength of the knee flexors. This muscle group functionally prevents anterior-tibial translation of the tibia on the femur and also counteracts excessive shear of the joint that is caused during contraction of the more dominant knee extensor muscle group. Coaches and athletic trainers must be made aware of the functional implications of an athlete participating in competitive sports during conditions of EIMD, when a reduced level of dynamic stabilisation from the musculature may compromise knee joint integrity.

Volitional and magnetically evoked EMD.

A significant (limb*bout) difference was observed for volitional EMD values during the EIMD intervention. This suggests that the repeated-bout effect was not witnessed in terms of any greater preservation of volitional EMD; the experimental limb showed the same degree of change relative to the control limb during the initial-bout and repeated-bout conditions of EIMD. The potential for timely recruitment of the fast-twitch motor fibres did not appear to have been disrupted during either bout of EIMD.

The magnetically evoked activation of the knee flexor musculature was also unaffected by the presence of EIMD despite the significant loss of PFv in the experimental limb. It therefore appears that sufficient quantities of fast twitch muscle fibres could be recruited during both phases of assessment, despite the presence of muscle damage.

Constant error (type I sensorimotor performance task).

To the author's knowledge, no previous studies have explored the effects of the repeated-bout effect on sensorimotor performance in the knee flexor musculature. The initial-bout and repeated-bout of exercise induced muscle damage were compared in order to summarise the implications of this type of eccentric exercise stress on real-life athletic performance. The possibility that the previously witnessed 'protective' repeated-bout effect for the knee

extensors (Brown et al., 1997, McHugh et al., 1999, McHugh, 2000) could perhaps lead to an actual improvement or potentiation of sensorimotor performance in the knee flexors was investigated as a secondary objective of this study.

No significant disruption to the accuracy of the type I force replication task was observed despite a significant loss of peak force in the experimental limb during the repeated-bout. The pattern of sensorimotor response in the experimental limb was similar during both conditions of EIMD and therefore a repeated-bout effect was not observed for constant error during the type I SMP task. Pain may have been utilized, and possibly better tolerated in order to mediate the desirable force response during the repeated-bout intervention but this did not lead to a significant improvement to force recognition.

Constant error (type II sensorimotor performance task).

The accuracy of force replication during the type II task was not significantly disrupted during both the initial-bout and repeated-bout of EIMD. The observed significant main effect for limb may be explained by the fact that the control limb was the dominant limb, which may possess a slightly more refined sensorimotor capacity. Performance of the type II SMP task was unaffected despite the significant loss of strength during the presence of EIMD in the experimental limb. The presence of pain during the relatively slow movement may either have altered the subjects' ability to hold the isometric contraction or alternatively may have assisted in the reproduction of the given force thereby masking any significant differences in sensorimotor performance between limbs. Several subjects did report the fact that they reproduced the level of pain that they had previously associated with the target force during the task familiarisation.

Variable error types I and II SMP task.

No significant interaction was observed for variable error during both types I and II SMP task. This reflects a consistency in response during both the self and time regulated tasks despite the presence of muscle damage and a loss of maximal force production capability in the non-preferred limb. This study therefore concludes that either the feedback from the damaged muscle spindles during conditions of long-term exercise stress (EIMD) did not adversely affect force judgement or that the sensorimotor control mechanism possesses the sensitivity to *adapt* under *prolonged* conditions of extreme mechanical disruption to the intra-muscular environment in order to protect the limb from further deformation in the form of an injury when strength is compromised. This pattern of response is different to that witnessed during conditions of acute fatigue and a relatively transient disruption to sensorimotor information processing.

Given the current findings, it appears that strength is the factor in knee joint protection that is most threatened during conditions of EIMD. Both volitional and evoked EMD were preserved during conditions of prolonged exercise stress, which implies that an efficient neuromuscular 'switch-on' time is a protective mechanism, inherent in the reaction to unforeseen joint perturbation whilst the body is experiencing pain, loss of flexibility and indeed function for up to 120 hours following eccentric exercise. This effective mechanism is complemented by a sensorimotor system that remains functional despite internal disruption to the muscle spindle apparatus and the potential for a disturbance to afferent feedback.

The findings from the current study may be taken a stage further to investigate whether the 'blue-print' of neuromuscular control and sensorimotor performance of the lower limb may be improved by an alternative type of exercise stress in the form of a 'positive' physical training regime which utilises functional movement patterns.

7.2.5. Summary.

This study monitored the effects of an initial and repeated-bout of EIMD on sensorimotor and neuromuscular performance of the knee flexors in the non-preferred limb of female athletes. Blood plasma creatine kinase, pain and flexibility were observed as physiological indicators of EIMD. The neuromuscular parameters of peak force, volitional and magnetically evoked EMD and both constant and variable error during types I and II sensorimotor performance tasks were also examined. All indices of performance were assessed prior to and 1, 24, 48, 72 and 120 hours following an eccentric isokinetic exercise protocol in the non-preferred hamstrings musculature. Contra-lateral (control) limb comparisons were undertaken using the preferred limb at all test occasions. The order of limb assessment was randomly presented at all pre and post EIMD assessment occasions.

The index of plasma creatine kinase was the only factor to show a significant difference between bouts of EIMD. The protective mechanism associated with the repeated-bout effect was therefore *not* witnessed in the knee flexors, as a similar pattern of PFv loss was evident in the experimental limb following both bouts of EIMD. Both volitional and magnetically evoked EMD responses were preserved during the initial-bout and repeated-bout of EIMD despite a 20% loss of peak force and disruption to the fast twitch muscle fibres. Similarly, sensorimotor performance capacity in terms of constant error during types I (time-regulated) and II (self-regulated) assessment tasks was unaffected by the presence of long-term 'negative' exercise stress. Pain may have been used to moderate the intensity of muscular contraction in the affected limb which may have contributed towards the lack of significant differences for the experimental limb during conditions of an initial-bout versus a repeated-bout of EIMD.

In consideration of all results of the present study it appears that the neuromuscular factor of strength may be the most compromised during conditions of EIMD in the knee flexor musculature. However, it is not known what magnitude of strength loss may constitute a significant threat to knee joint protection. The recovery of strength to baseline levels may take at least five days following eccentric exercise stress. It is hereby recommended that future research into the effects of EIMD in the hamstring musculature be performed at least seven days in advance of both pre-season training and the competitive soccer season to allow the recovery of strength and functional performance indices of knee joint performance (range of motion and movement without pain) to be restored in all athletes. Efficient neuromuscular activation may act as a protective mechanism, inherent in the reaction to unforeseen joint perturbation whilst the body is experiencing pain, loss of flexibility and indeed function during a mechanical disruption to the muscle. This neuromuscular control mechanism is complemented by a sensorimotor system that remains functional for up to 5 days during conditions of EIMD and the ensuing loss of 20% peak force in the knee flexor musculature.

8.0. The effects of an acute training programme on sensorimotor and neuromuscular performance of the knee flexors in the female athlete.

8.1. Introduction.

In the previous chapters the effects of two forms of exercise stress on neuromuscular and sensorimotor performance of the lower limb in female athletes was investigated. The chosen conditions of 'negative' exercise stress have ranged from an acute fatigue intervention with recovery up to six minutes following the cessation of exercise to a more permanent type of exercise stress in the form of exercise induced muscle damage. The rationale behind this 'continuum of exercise stress' was to identify any potential risk factors that may predispose female athletes to the previously reported increased risk of lower limb injury. Fatigue and exercise induced muscle damage are both commonly encountered forms of 'real-life' stress that may affect both the neuromuscular and sensorimotor systems during athletic performance.

The recent rise in injury statistics concerning female athletes has raised concern regarding sports that require jumping and cutting manouvres. In fact, up to a four-fold higher incidence of knee injury has been reported for females compared to male athletes given the equivalent exposure to the same sports (Gray, 1985; Arendt and Dick, 1995). It is also possible that the non-preferred or less dominant limb may be the most susceptible to both ACL injury (Rees, 2003 [personal communication]) and hamstring injury (Knapik, Bauman, Jones and Harris, 1991). Since the majority of injuries are reported to occur during a non-contact situation, the failure of intrinsic (within the body) factors such as skill, neuromuscular co-ordination, sensorimotor control and an abnormal quadriceps to hamstrings muscle group ratio are implicated alongside extrinsic (outside of the body) factors for injury that include, shoe-surface interface, playing surface, and the athlete's playing style (Huston et al., 2000). This

injury risk may be heightened during conditions of fatigue when up to a 23% loss of knee flexor strength has been observed up to only 3 minutes of maximal isometric activity. The typical two-week period of pre-season training may be the only access that a coach or athletic trainer can have with a team prior to the competitive season. This relatively short time frame offers a challenge to the coach of how best to impact the vulnerable areas of a player's physical performance, which may threaten an entire seasons competitive edge. In the worse-case situation a physiotherapist has an even greater time pressure to return an injured athlete to the game in as little time as possible. This is especially true for professional athletes when there may be financial pressure associated with absence from competition. The goal in improving the functional performance of the lower limb by addressing sensorimotor and neuromuscular performance is to improve the underlying motor program through repeated and appropriate stimulation of both the afferent and efferent pathways. Therefore it is important to investigate what quantity or 'dose' of functional training is potent enough to have a 'positive' impact on both neuromuscular control and the integration of sensorimotor feedback for improved performance in the lower limb.

A preventative training program to improve neuromuscular and sensorimotor function of the lower limb musculature and thus knee joint stabilisation may be a cost effective approach to the prevention of injury. If the efficiency of such intricate control systems can be improved by functional intervention strategies, the reduced incidence of sports injuries may significantly impact many lives. Various types of training intervention have already been applied as a means to reduce this sex-specific injury prevalence (Caraffa et al., 1996; Hewett et al., 1999; Heidt et al., 2000; Huston et al., 2000; Junge et al., 2002) and have utilised co-ordination, plyometric, agility and strength training approaches. In a prospective controlled training study of 600 semi professional soccer players (the sex of the participants was not specified) over three soccer seasons, Caraffa et al. (1996) observed a significant reduction in

the incidence of ACL injury. The training regime utilised progressive balance board training. Three hundred players were asked to train for 20 minutes per day with five different phases of wobble-board training of increased difficulty. The remaining 300 players (age matched athletic control comparison group) were asked to train according to their normal team routine. The incidence of ACL injury was reduced sevenfold (0.15 injuries per team per year) in the 'proprioceptively trained' group compared to the age matched athletic comparison (1.15 ACL injuries per team per year). Hewett et al. (1999) also demonstrated a significant decrease in the incidence of knee injury in female athletes after a specific plyometric training program (injury incidence per 1000 athlete exposures was 0.43 in untrained female athletes, 0.12 in trained female athletes, and 0.09 in male athletes. [p=0.02, chi- square analysis]). This equates to a 3.6 times higher incidence of knee injury in the untrained compared to the trained female athletes (p=0.05) and was 4.8 times higher than male athletes (p=0.03). Huston et al. (2000) also utilised a functional jump-training program for both the hamstrings and gastrocnemius muscle groups in female athletes that produced a subsequent decrease in the rate of ACL injury.

Heidt et al. (2000) examined the effects of a pre-season training program on the occurrence of ACL injuries in elite female soccer players. 42 out of 300 female soccer players were randomly selected to participate in a 7-week specialised training regime which included sports specific cardio-vascular conditioning, plyometric work, sport cord drills, strength training and flexibility exercises. The resulting ACL injury statistics for the season (recorded by team trainers) were significantly less within the athletes chosen for the specific training compared to the age-matched control group (14% versus 33.7%, respectively). The findings by Heidt et al. (2000) are in agreement with Junge et al. (2002) who significantly reduced the incidence of injuries in male youth amateur soccer players (aged 14-19 yrs) over two soccer seasons. Both the intervention and control groups consisted of three 'high skill' and three 'low skill' teams

to avoid any potential influence of expertise. The training intervention consisted of "*exercises to improve stability of the ankle and knee joints, the flexibility and strength of the trunk, hip and leg muscles, as well as to improve co-ordination, reaction time and endurance*". Alongside this physical intervention, the team coaches were educated on injury prevention strategies. Each player was also given individual feedback regarding his baseline physical assessment and informed on how to improve his 'weaknesses'. The incidence of injury per 1000 hours of training and playing soccer was 6.7 in the intervention group versus 8.5 in the control group. This equated to 21% less injuries in the trained group. Interestingly, the low-skill intervention group had 37% fewer injuries following the training when compared to the corresponding control group, as opposed to only 6% fewer injuries within the high-skill group. This suggests that the high-skill or more experienced players may have a refined ability to avoid injury during soccer because they have been exposed to the threat of joint injury on a regular basis. Therefore, there may be a greater potential for improvement within a low-skill group of athletes. Whether the refined ability of the high-skill group was due to a superior level of sensorimotor and neuromuscular performance was not investigated.

It is an important point to make that the above studies assessed the outcome of their training intervention in terms of both muscular and ligamentous injuries over the subsequent soccer season(s) (Caraffa, et al. 1996; Heidt, et al. 2000; Hewett, et al. 1999; Junge et al., 2002). Dramatic reductions in terms of injury incidence were seen when the intervention lasted at least one soccer season. The investigators within each of the previously described experiments did not however use any form of specific neuromuscular or sensorimotor analysis to determine what aspect of performance may have contributed most to the observed reduction in injury statistics.

An injury-producing scenario may be viewed as time critical for the neuromuscular system because the protective motor response requires a certain fixed time interval to execute. Fixed

sensory transmission latencies limit the time remaining for the neuromuscular system to complete that motor response within what has been described as the 'available response time' (Chen et al., 1994). This concept may be demonstrated by the fact that ligamentous sprains occur whereby the external challenge exceeds the capability of the neuromuscular system to protect the joint (Ashton-Miller et al., 2001). Therefore an efficient muscle reaction time may be required for injury prevention. Tests that examine a subject's dynamic sensorimotor ability may be more able to assess how the body copes during the real-life scenario than slow joint reproduction tasks or ensuing injury statistics.

The possibility of training proprioception during 'rapid' joint loading conditions has not yet been tested (Ashton-Miller et al. 2001). Therefore the aim of this final study was to investigate the potentially 'positive' effects of a training intervention on neuromuscular and sensorimotor performance of lower extremity, utilising a brief dose-response approach over two weeks. This time period was chosen to represent a typical pre-season period in soccer. The transfer effect of this training regime on sensorimotor performance was assessed using the previously implemented dynamic sensorimotor performance tasks.

8.2. Methodology.

Subjects.

Twelve adult females (age 22 ±1.8 years; height 1.65.8 ±0.05 m; body mass 65.3 ±7.0 kg [mean ± SD]) gave their informed consent to participate in this study. The sample comprised collegiate soccer (n=7), volleyball (n=3) and dance (n=2) athletes. Subjects were instructed to keep sporting activities consistent throughout the experimental period, wherever possible.

All subjects were randomly assigned to either a control [n=5] (no training) or experimental [n=7] (additional sensorimotor and neuromuscular training) group. The experimental group trained their non-preferred limb as it was hypothesised that a greater improvement to neuromuscular and sensorimotor function and therefore a greater effect size may be witnessed in the least developed limb. This would therefore increase the likelihood of detecting a biologically meaningful change in performance. All exercises were designed to provide a stimulus for the improvement of sensorimotor performance and to mimic the time critical dynamic threats to joint integrity that may be encountered during the sporting situation as recommended by Caraffa et al. (1996), Hewett et al. (1996), Laskowski et al. (1997), DeMont and Lephart, (1998) and Nottingham et al. (2001).

The training intervention took place over a two-week period, all subjects participating in a total of 6 exercise sessions per week. Following a standardised warm-up and stretching of the lower limbs, each training session consisted of six exercises that were performed in a random order for a period of two minutes each. A one minute rest period was allowed between each exercise and subjects were instructed to rest for a further 10-15 seconds if they considered the quality of their exercise performance to be jeopardized by symptoms of 'fatigue'. The key objective for each exercise was to focus on both the accuracy and /or speed of movement depending on the particular task. During the second week of training, the exercises were

made progressively more challenging by refining the boundaries of permissible movement i.e. a smaller target area to hop onto, alongside an increase in the target number of repetitions of each exercise (working on an individual basis of improvement) within the designated time limit. A description of each exercise station and subsequent progression over the training period of two weeks is included within appendix 3.

Neuromuscular and sensorimotor performance indices were assessed prior to and post intervention, with a two week interval between pre and post intervention occasions using a single leg intervention (non-preferred) contra-lateral control (preferred) model of assessment. Furthermore, the non-preferred (experimental) limb was also assessed on the above test occasions following a previously applied ipsilateral control (no fatigue) and single bout of fatigue intervention protocol (chapter 6.0). This enabled the effects of fatigue on neuromuscular and sensorimotor performance to be monitored prior to and following the training intervention in the non-preferred limb.

Because of logistical limitations to the study and to protect against any interaction between training and fatigue, the non-preferred limb of the control group was also assessed during conditions of fatigue. Furthermore, as it was difficult to guarantee that each participant in the experimental group also maintained their 'normal' level of sports activity throughout the experimental period, a control group was utilised to reduce the potential limitations to the study.

Subject orientation.

Subject orientation for the baseline and pre and post control/experimental intervention assessments are described in the general methodologies chapter (chapter 4.0). The assessment protocol for both experimental and control group is illustrated in figure 8.1.

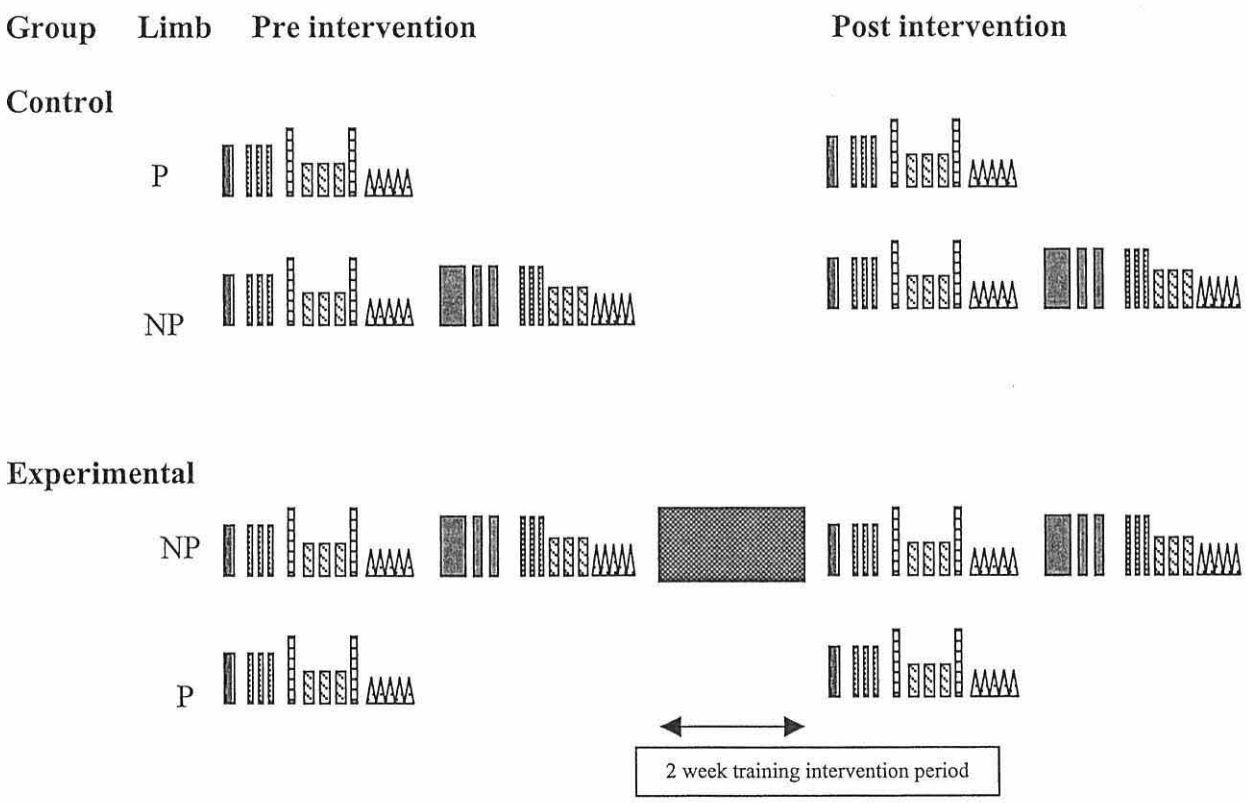
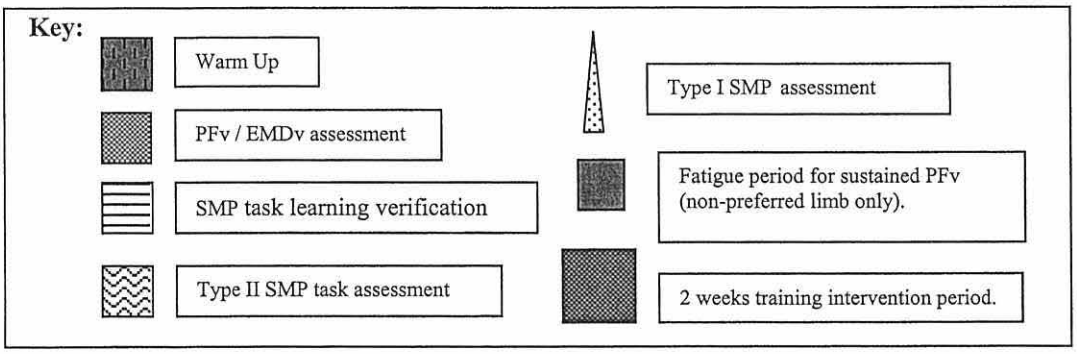


Figure 8.1 Neuromuscular and sensorimotor assessment protocol for the non-preferred (experimental) and preferred (control) limb prior to and following a fatigue protocol at pre and post intervention assessment periods for the control (no training) and experimental (training intervention) groups. Note: NP denotes non-preferred (experimental) limb and P denotes preferred (control) limb.



Statistical analyses.

(1) The effects of the training intervention.

Separate three way mixed model ANOVAs, with repeated measures on time [pre intervention, post intervention] and limb [experimental, control] with a between-subjects factor of group [control (no training); experimental (training intervention]) were used to test the null hypothesis that training had no effect on the dependent variables of PF_v, EMD_v, constant and variable error (expressed as a % of the target force) during types I and II SMP tasks.

(2) The effects of a fatigue intervention.

Further 3 way mixed model ANOVAs, with repeated measures on time [pre, post fatigue] and occasion [pre training intervention, post training intervention] with group [control (no training), experimental (training intervention)] as a between subjects factor were used to test the null hypothesis that training had no effect on performance during fatigue for the dependent variables of PF_v, EMD_v, constant and variable error (expressed as a % of the target force) during types I and II SMP tasks for the experimental limb only.

In the event of a significant Mauchly's test of sphericity, the violation to the assumption of ANOVA was corrected for using the Greenhouse-Geisser adjustment of the critical F-value, as indicated by (GG). SPSS (V 9.0) was used to perform all statistical procedures.

8.3. Results.

Sensorimotor performance.

The effects of the training intervention on constant error during the type I SMP task.

Group means and standard deviations for constant error during the type I SMP task at pre and post intervention for the control (preferred) and experimental (non-preferred) limbs in the control and experimental groups are presented in table 8.1.

A three way mixed model ANOVA with repeated measures on time (pre intervention, post intervention) by limb (control [preferred], experimental [non-preferred]) with group (control, experimental) as a between subjects factor revealed no significant interaction (time*limb*group). No further two-way interactions were observed. This suggests that constant error values were comparable between both the control and training groups. Therefore there is no evidence to suggest an improvement in the accuracy of sensorimotor response during the type I task as a result of the training intervention.

Constant error (type I SMP task) (% of target force)			
Time	**Limb**	**Control group [Mean ± SD]**	**Experimental group [Mean ± SD]**
Pre control	P	1.7 (19.1)	-17.4 (20.9)
Post control	P	4.7 (20.1)	0.12 (13.9)
Pre experimental	NP	1.4 (34.5)	-4.1 (23.6)
Post experimental	NP	10.8 (41.2)	2.0 (22.2)

Table 8.1. Group mean constant error [% of target force] [± SD] for the type I SMP task at pre and post control and pre and post experimental conditions for the control (preferred) limb and experimental (non-preferred) limb of the control and experimental groups. Note: NP denotes non-preferred limb (experimental) and P denotes preferred limb (control).

The effects of a fatigue intervention on constant error during the type I SMP task.

Group means and standard deviations for constant error during the type I SMP task at pre and post intervention prior to and following a single episode of a fatigue task for the experimental limb of both the control and experimental groups are presented in table 8.2.

A three way mixed model ANOVA with repeated measures on time (pre fatigue, post fatigue) by occasion (pre training intervention, post training intervention) with group (control, experimental) as a between subjects factor revealed no significant interaction (time*occasion*group) for measures on the non-preferred limb. This suggests that the training intervention did not significantly affect the accuracy of sensorimotor performance represented by constant error values during fatigue in the non-preferred limb.

Constant error (type I SMP task) (% of target force)				
Time	**Occasion**	**Limb**	**Control group [Mean ± SD]**	**Experimental group [Mean ± SD]**
Pre fatigue	Pre training intervention	NP	1.4 (34.5)	-4.1 (23.6)
Post fatigue	Pre training intervention	NP	10.4 (34.8)	-1.8 (22.2)
Pre fatigue	Post training intervention	NP	10.8 (41.2)	2.0 (22.2)
Post fatigue	Post training intervention	NP	12.7 (24.2)	13.3 (21.3)

Table 8.2. Group mean constant error [% of target force] [± SD] for the type I SMP task at pre and post fatigue (prior to the control/training intervention period) and pre and post fatigue (following the control/ training intervention period) for the experimental (non-preferred) limb in both the control and experimental group. Note: NP denotes non-preferred limb (experimental).

The effects of the training intervention on constant error during the type II SMP task.

Group means and standard deviations for constant error during the type II SMP task at pre and post intervention for the control and experimental limbs of both the control and experimental groups are presented in table 8.3.

A three way mixed model ANOVA with repeated measures on time (pre intervention, post intervention) by limb (control, experimental) with group (control, experimental) as a between subjects factor revealed no significant interaction (time*limb*group). This suggests that sensorimotor performance in terms of constant error during the type II SMP task was not improved as a result of a short-term training intervention programme in the experimental limb.

Constant error (type II SMP task) (% of target force)			
Time	**Limb**	**Control group [Mean ± SD]**	**Experimental group [Mean ± SD]**
Pre control	P	0.7 (6.8)	-9.8 (8.4)
Post control	P	-22.2 (28.4)	-16.6 (22.5)
Pre experimental	NP	4.0 (26.4)	-9.4 (19.1)
Post experimental	NP	9.0 (12.3)	-5.8 (14.2)

Table 8.3. Group mean constant error [% of target force] [± SD] for the type II SMP task at pre and post control and pre and post experimental conditions for the control (preferred) limb and the experimental (non-preferred) limb of the control and experimental groups. Note: NP denotes non-preferred limb and P denotes preferred limb.

The effects of a fatigue intervention on constant error during the type II SMP task.

Group means and standard deviations for constant error during the type II SMP task at pre and post intervention prior to and following a single episode of a fatigue task for the experimental limb of both the control and experimental groups are presented in table 8.4.

A three way mixed model ANOVA with repeated measures on time (pre fatigue, post fatigue) by occasion (pre training, post training) with group (control, experimental) as a between subjects factor revealed no significant interaction (time*occasion*group) for measures on the experimental limb. This suggests that the training intervention did not significantly influence

the accuracy of sensorimotor performance in terms of constant error prior to and following conditions of fatigue in the experimental limb.

Constant error (type II SMP task) (% of target force)				
Time	**Occasion**	**Limb**	**Control group [Mean ± SD]**	**Experimental group [Mean ± SD]**
Pre fatigue	Pre training intervention	NP	4.0 (26.4)	-9.4 (19.1)
Post fatigue	Pre training intervention	NP	-0.2 (20.5)	1.0 (22.9)
Pre fatigue	Post training intervention	NP	9.0 (12.3)	-5.8 (14.2)
Post fatigue	Post training intervention	NP	1.7 (27.4)	-13.0 (18.5)

Table 8.4. Group mean constant error [% of target force] [± SD] for the type II SMP task at pre and post fatigue (prior to the control/training intervention period) and pre and post fatigue (following the control/ training intervention period) for the experimental (non-preferred) limb in both the control and training group. Note: NP denotes non-preferred (experimental) limb.

Neuromuscular performance indicators.

The effects of the training intervention on peak force.

Group means and standard deviations for volitional peak force at pre and post intervention for the control limb and experimental limb of both the control and experimental groups are presented in table 8.5.

A three way mixed model ANOVA with repeated measures on time (pre intervention, post intervention) by limb (control [preferred], experimental [non-preferred]) with group (control, experimental) as a between subjects factor revealed no significant interaction (time*limb*group). This suggests that strength remained constant in both the control and experimental groups i.e. the training intervention did not significantly improve strength in the experimental limb for the training group.

Peak force (PFv) (N)			
Time	**Limb**	**Control group [Mean ±SD]**	**Experimental group [Mean ±SD]**
Pre control	P	222.6 (37.1)	246.2 (80.7)
Post control	P	222.8 (22.1)	229.0 (71.5)
Pre experimental	NP	198.8 (32.8)	196.6 (43.0)
Post experimental	NP	181.8 (45.5)	224.4 (74.1)

Table 8.5. Group mean peak force (N)[± SD] at pre and post control and pre and post experimental conditions for the control (preferred) limb and the experimental (non-preferred) limb of the control and experimental groups. Note: NP denotes non-preferred limb and P denotes preferred limb.

The effects of a fatigue intervention on peak force.

Group means and standard deviations for peak force at pre and post intervention prior to and following a single episode of a fatigue task for the experimental limb of both the control and experimental groups are presented in table 8.6.

A three way mixed model ANOVA with repeated measures on time (pre fatigue, post fatigue) by occasion (pre training, post training) with group (control, experimental) as a between subjects factor revealed no significant interaction (time*occasion*group) for measures on the experimental limb. This suggests that the results for pre and post fatigue measures of peak force were comparable between both the control and experimental groups. Therefore, there is not enough evidence to suggest that the training intervention had affected the performance of the experimental limb in terms of strength during conditions of fatigue.

Peak force (PFv) (N)				
Time	**Occasion**	**Limb**	**Control group [Mean ±SD]**	**Experimental group [Mean ±SD]**
Pre fatigue	Pre training intervention	NP	198.8 (32.8)	196.6 (43.0)
Post fatigue	Pre training intervention	NP	191.0 (31.2)	185.9 (39.0)
Pre fatigue	Post training intervention	NP	181.8 (45.5)	224.4 (74.1)
Post fatigue	Post training intervention	NP	170.8 (21.6)	201.2 (67.8)

Table 8.6. Group mean peak force (N) [± SD] at pre and post fatigue (prior to the control/training intervention period) and pre and post fatigue (following the control/ training intervention period) for the experimental (non-preferred) limb in both the control and training group. Note: NP denotes non-preferred limb.

The effects of the training intervention on volitional electromechanical delay.

Group means and standard deviations for volitional electromechanical delay at pre and post intervention for the control limb and pre and post intervention prior to and following a single episode of a fatigue protocol in the experimental limb are presented in table 8.7.

A three way mixed model ANOVA with repeated measures on time (pre intervention, post intervention) by limb (control, experimental) with group (control, experimental) as a between subjects factor revealed no significant interaction (time*limb*group). However a significant main effect for group was observed ($F_{[1,1]} = 11.1$, $p<0.008$). This suggests that there was a difference between group mean values when the control (no training) and experimental (training) group are compared across the factor of time (pre intervention and post intervention). As the control group had not participated in the training, the changes to the group mean values cannot be attributed to the physical intervention. Factors that may have influenced neuromuscular activation of the knee flexors include a difference in limb temperature and therefore muscular compliance. This difference may possibly have been

apparent between assessment occasions for volitional EMD and could have contributed to the observed interaction.

Volitional EMD (EMDv) (ms)			
Time	**Limb**	**Control group [Mean± SD]**	**Experimental group [Mean± SD]**
Pre control	P	59.2 (25.5)	48.2 (14.5)
Post control	P	44.2 (15.0)	56.4 (19.2)
Pre experimental	NP	65.8 (11.5)	58.9 (20.6)
Post experimental	NP	44.9 (15.9)	43.1 (12.4)

Table 8.7. Group mean volitional EMD (ms)[± SD] at pre and post control or experimental conditions for the control (preferred) limb and the experimental (non-preferred) limb of the control and experimental groups. Note: NP denotes non-preferred limb and P denotes preferred limb.

The effects of a fatigue intervention on volitional EMD.

Group means and standard deviations for volitional EMD at pre and post intervention prior to and following a single episode of a fatigue task for the experimental limb of both the control and experimental groups are presented in table 8.8.

A three way mixed model ANOVA with repeated measures on time (pre fatigue, post fatigue) by occasion (pre training, post training) with group (control, experimental) as a between subjects factor revealed no significant interaction (time*occasion*group) for measures on the experimental limb. This suggests that there was no significant difference in terms of neuromuscular activation of the knee flexors between the control and experimental groups prior to and following the fatigue intervention.

Volitional EMD (EMDv) (ms)				
Time	**Occasion**	**Limb**	**Control group [Mean ±SD]**	**Experimental group [Mean ±SD]**
Pre fatigue	Pre training intervention	NP	65.8 (11.5)	58.9 (20.6)
Post fatigue	Pre training intervention	NP	64.8 (12.0)	73.1 (19.0)
Pre fatigue	Post training intervention	NP	44.9 (15.9)	43.1 (12.4)
Post fatigue	Post training intervention	NP	87.1 (39.8)	62.2 (24.9)

Table 8.8. Group mean volitional EMD [± SD]at pre and post fatigue (prior to the control/training intervention period) and pre and post fatigue (following the control/ training intervention period) for the experimental (non-preferred) limb in both the control and training group. Note: NP denotes non-preferred limb and P denotes preferred limb.

8.4. Discussion.

Sensorimotor performance.

The aim of this final chapter of the thesis was to investigate the potential for improvement in both neuromuscular and sensorimotor performance following a dose-response training intervention on the lower limb musculature in female athletes. Types I and II dynamic sensorimotor assessment tasks have never been applied to determine the outcome of a training intervention. Each test is specifically designed to reflect a different type of muscular response. Type I SMP is designed to target a more timely response for joint protection (fast-twitch muscle fibres), whereas the type II SMP task is associated with force re-education following muscular disruption or injury (slow-twitch muscle fibres).

It has been previously suggested that training may act as a sensorimotor stimulus by systematically increasing fusimotor drive to the muscle spindles during challenging tasks as well as increasing the gain of the spinocerebellar and dorsal column networks which receive spindle afference (Ashton-Miller et al., 2001). The observed results did not provide sufficient evidence to suggest either an improvement or disruption to the accuracy of performance during either the type I or II task following the training intervention. This may suggest that there was a limited capacity for improvement within the chosen population or that the sample sizes for the control [n=5] and experimental [n=7] groups may have been insufficient and therefore the ability to detect a significant difference in performance may have been threatened. Finally, the transfer between the training and assessment methods may have been minimal (Baker et al., 1994). This may be due to the fact that the majority of the training exercises were weight bearing which may provide greater mechanical deformation of the soft tissues around the knee joint, greater levels of muscle activity and also more input from other joints which are involved in the movement.

The amount of training exposure that is required to elicit a biologically meaningful improvement in sensorimotor capacity has not been conclusively proven. This may be due to the fact that previous training intervention studies have used injury statistics as an outcome measure (Caraffa, et al. 1996; Heidt, et al. 2000; Hewett, et al. 1999; Junge et al. 2002). This experiment utilised a dose-response training approach that represented the typical time period that soccer players would be exposed to during pre-season preparatory training. This may suggest that either the potential to improve the accuracy and efficiency of an individual's motor program is restricted by factors such as their level of sports participation, or that the amount of 'exposure' or training within this study was not sufficient in its capacity to bring about an alteration to the intricate afferent and efferent sensory feedback systems of every individual. The observed results for the sensitivity of the test which was examined in chapter 5.0 suggested that an individual's sensorimotor performance would have to be 65.8% and 59.8% different from the rest of the group (for between day measures) to be detected as different for types I and II sensorimotor tasks, respectively. Therefore a true difference in sensorimotor performance may have been difficult to detect given the short time frame and thus scope for improvement within this study.

Furthermore, it was hypothesised that by using the non-preferred limb as the experimental limb, a greater potential for both sensorimotor and neuromuscular gain may have been expected. This theory may not have been upheld as the training exercises may have either proven to be a difficult challenge for the non-preferred limb or the training regime may have resulted in a 'negative-transfer' effect: whereby the training has actually interfered with the performance of a related task (Caroll et al., 2000). This may be explained by the fact that most of the exercises required co-activation of the knee flexors and extensors, but the assessment protocol merely focused on the activation of the knee flexors. From a skill acquisition point of view, the subjects may not quite have achieved an autonomous level of

performance given the relatively low number of training sessions. Ashton-Miller et al. (2001) suggested that exercises that are intended to train 'proprioceptive' abilities in a given joint may do so by merely focusing the individual's attention to cues from the brain and autonomous control centers which ultimately improves performance. Therefore, there may be an interaction between the athlete's attention and an improvement in performance as opposed to trying to improve a neuromuscular or sensorimotor indicator of performance.

Peak force.

Adequate lower extremity conditioning may be the key to establishing an effective time sequence for muscle recruitment (Huston et al. 1996). A significant improvement in peak force was not expected due to the short time frame of this intervention and also given that the exercise regime did not utilise heavy resistance exercises (leading to type II muscle fibre hypertrophy) (MacDougall et al. 1980). The overall strength loss observed during the fatigue intervention was comparable between occasions and groups, equating to an approximate 10% loss of strength in the non-preferred limb following a single bout of maximal volitional isometric activation of the knee flexors. Such effects are consistent with the 20% reduction in strength changes that was witnessed in chapter 6.0 following four bouts of the same fatigue protocol. The training intervention did not appear to reduce the impact of the fatigue protocol on the fast twitch muscle fibres that are essential to maximal volitional activation of the knee flexor musculature. The observed loss of ~10% strength did not also disrupt sensorimotor performance during the reproduction of 50% PFv in types I and II sensorimotor assessment tasks.

Volitional EMD.

The current training intervention did not have any significant effects on rapid volitional activation of the hamstrings musculature. The chosen exercises for the intervention may not

have been sufficiently challenging to the neuromuscular switch-on process in the lower limb. Wojtys et al. (1996) reported an improvement in muscle reaction times of the lower extremity following a six-week agility training programme in 16 male and 16 female subjects. The training regime entailed the use of slide-boards, bounding, cariocas, figure of eight runs and backward running. The non-significant effect of the present study may also suggest that volitional EMD cannot be improved during such a short period of time.

It has been previously suggested that neuromuscular training may be viewed as 'motor learning', as the subject has to learn co-ordinated movements or patterns associated with optimal performance (Hakkinen, Alen and Kallinen, 2000). The repetitive nature of the current training protocol may, given adequate time for practice, improve an athlete's ability to detect afferent cues with greater probability whilst refining motor responses to standard cues from the proprioceptive systems (Ashton-Miller et al., 2001).

In conclusion, this dose-response intervention was not potent enough to have significantly impacted sensorimotor performance in the knee flexors. This may reflect the fact that within the 'healthy' athlete, the scope for improvement is minimal as regular participation may provide a sufficient stimulus for neuromuscular adaptation to a threatening situation for knee joint stability, or that a greater exposure to the training protocol is required. This study does however provide guidance for a physiotherapist who may only have the opportunity to treat a patient once a week. The short dose response approach within this study (12 minutes of intensive activity, six times per week) did not significantly impact sensorimotor performance and neuromuscular activation. Therefore, further research is required which assesses a greater magnitude of training over a longer period of time.

The fatigue protocol used within this study may also not have been reflective of the real-life threat to joint homeostasis and therefore not potent enough to have significantly altered PFv. Four bouts of the fatigue protocol were found to elicit a significant (20%) decrease in strength and a disruption to constant error during both types of SMP assessment tasks in chapter 6.0. Training neuromuscular activation and sensorimotor factors *during* conditions of fatigue is a recommended avenue for future research. This would perhaps offer a realistic insight into the intricate mechanism of joint protection when strength the knee joints primary protective mechanism is compromised. This is a crucial time period when the athlete actually requires optimal performance of the dynamic stabilizers in order to avoid damage to the knee joint.

8.5. Summary.

This final study investigated the effects of a potentially 'positive' form of intervention on sensorimotor and neuromuscular performance of the knee flexors in female athletes (n=7). Sensorimotor and neuromuscular performance indices were assessed in both the preferred and non-preferred limbs prior to and following either a training (n=7) or control (n=5) period with a two-week interval between assessment sessions. The effects of fatigue were also assessed in the non-preferred limb of the control and training groups. The response to a fatigue task was also assessed in the control group in order to protect against any potential interaction between training and fatigue.

Dynamic sensorimotor assessment tasks have not previously been used to determine the outcome of a training intervention. The results from the present study do not provide sufficient evidence to support an improvement in sensorimotor performance during both types I and II SMP tasks following the exercise intervention. The fatigue protocol elicited a 10% reduction in PFv for both groups but this loss of strength did not affect sensorimotor performance of the non-preferred limb prior to and following the training intervention.

Similarly the neuromuscular switch-on times of the knee flexors were not affected by either the training or fatigue interventions. The lack of significant findings within this study may have been influenced by several factors including a small sample size, an insufficient number of training sessions, and/or a lack of transfer between the training and assessment methods. Alternatively, the lack of significant alteration to sensorimotor performance may suggest that there is a limited scope for improvement within the 'healthy', athletic individual.

As this is the first study to use dynamic sensorimotor assessment techniques to assess the effects of an acute training intervention, it may also be possible that this form of sensorimotor performance may not be altered in the knee flexor musculature by such an 'acute' training intervention strategy. Therefore a pre-habilitative (preventative) approach may be beneficial to the novice or recreational athlete who may place themselves at a greater risk of sports injury due to a low level of neuromuscular conditioning and sensorimotor awareness. The current training intervention may also be applicable to either the post surgical or sports injury population for rehabilitation, whereby a greater opportunity for improvement may be evident. It seems that future application of this form of dynamic sensorimotor assessment as a means of an outcome measure would be useful following a long-term prospective intervention which spans at least one whole soccer season.

9.0. Synthesis of work and clinical implications.

The specific objectives of the thesis were stated in chapter 2.0. The interrelationship between studies in this thesis and recommendations for future research are shown schematically in figure 9.1.

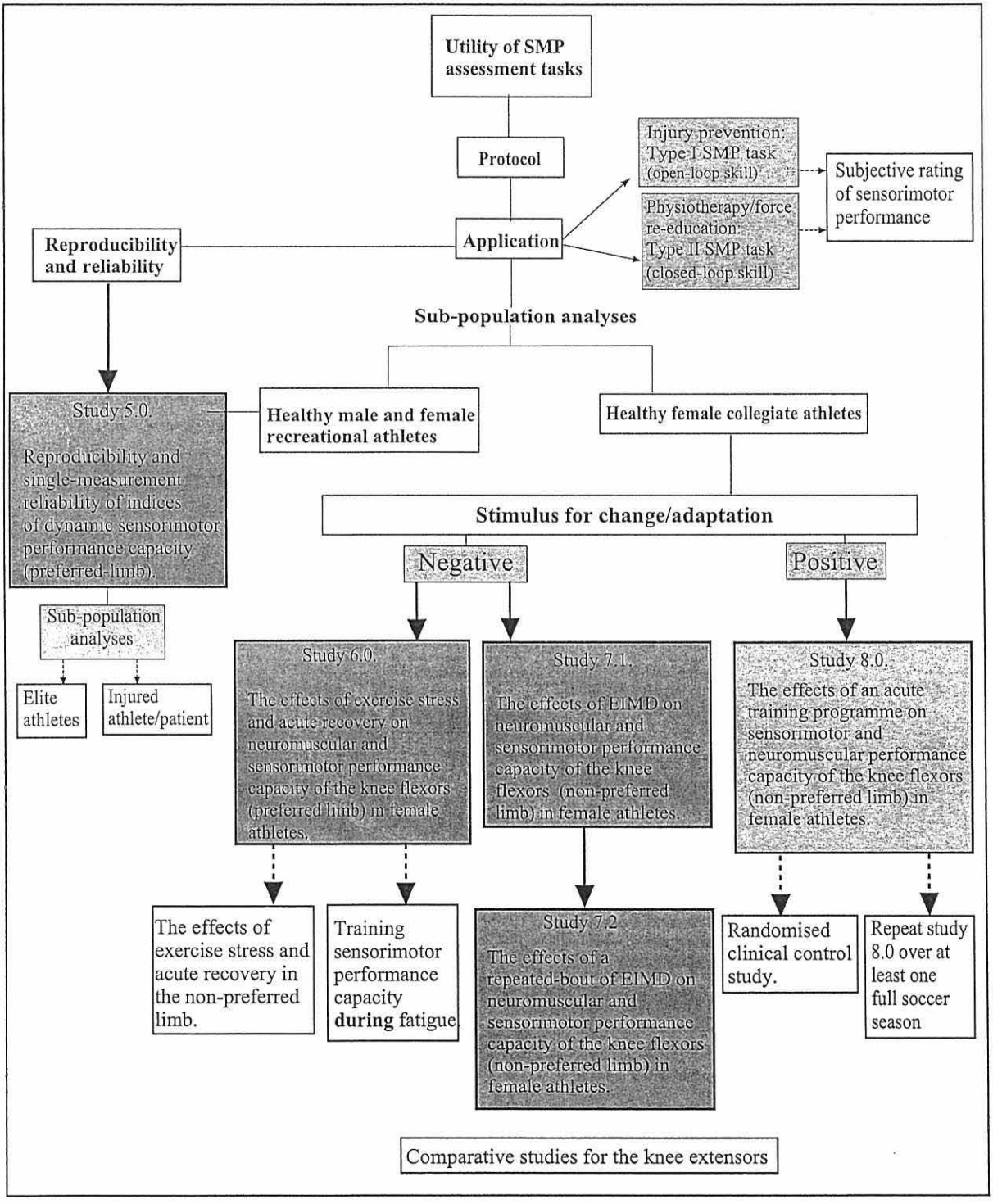


Figure 9.1. A schematic overview illustrating the interrelationship between studies of sensorimotor and neuromuscular performance associated with the knee flexor musculature in this thesis. Note: broken lines represent recommended future studies.

9.1. The clinical application of dynamic sensorimotor performance assessment techniques.

It is fundamental that a sports scientist or health professional (practitioner) working with an individual patient or team has a reliable measure of sensorimotor performance. The first study within the thesis was designed to evaluate the reproducibility and single-measurement reliability of two recently designed types of dynamic sensorimotor performance assessment tasks using both male and female recreational athletes. The main reason for employing this strategy was to identify the utility and appropriateness of the sensorimotor assessment tasks prior to their application in the 'healthy' (non-injured) athletic population. The use of a mean score from multiple trials (n=15 for the type I SMP task and n=25 for the type II SMP task) was recommended as the basis for estimating constant error during both types of SMP assessment task in order to minimize measurement error. The information gained from this preliminary research into the reproducibility and single-measurement reliability of two types of sensorimotor performance assessment tasks provides direction for future clinical studies, for example, in athletes following lower limb dysfunction and surgical intervention; where there may be more cause for the case-study approach to sensorimotor assessment.

Sensorimotor performance and fatigue during the athletic scenario.

The maximal isometric fatigue protocol that was applied in chapter 6.0 led to a significant alteration of both the isometric strength of the knee flexors (20% loss) and sensorimotor awareness (a greater tendency to under-achieve [over-estimate] the target force]) at 25° of flexion following only four bouts of sustained activity (4x 40 seconds of maximal activity). The potential for strength loss is therefore far greater during a 90 minute game of soccer. Complete recovery of knee flexor strength and SMP was also found to take up to six minutes following cessation of the activity. This is an alarming finding when the athletic scenario is considered. For example, if a major tackle, stumble or sprint occurred during this vulnerable

time period, the risk of injury to the knee joint could be amplified as a greater stress would be placed on the secondary structures involved in knee joint support.

Sensorimotor performance and exercise induced muscle damage.

The longer lasting, 'negative' effects of muscle damage that were observed in chapters 7.1 and 7.2 did not result in a loss of sensorimotor performance. Both the accuracy and consistency of force reproduction during types I and II SMP assessment tasks was maintained over a 5-day follow-up period during conditions of confirmed EIMD in the knee flexors. There are several important points to be made from the investigation into the effects of EIMD on neuromuscular and sensorimotor control. Coaches, physiotherapists and sports scientists must be aware of the potential for strength loss during conditions of EIMD, and therefore the basis for neuromuscular support during functional activities involving the knee joint. This compromised level of dynamic stabilisation may be evident up to 5 days following substantial eccentric activity. As this study is possibly the first of it's kind to address the implications of a combined interruption to neuromuscular and sensorimotor control during EIMD, the actual amount of strength loss required to significantly threaten functional sensorimotor performance and secondary knee joint protection remains to be quantified. It may perhaps be the case that a highly trained athlete can indeed cope with a 20% loss of PFv without any further repercussion.

Training sensorimotor performance: Future directions.

It is not yet known what magnitude of training exposure is required to significantly improve sensorimotor performance in the lower limb. The results from the final exercise stress intervention did not provide sufficient evidence to support an improvement in constant and variable error associated with performance of the type I and II SMP assessment tasks. As this was the first study to use types I and II SMP assessment techniques as an outcome measure

following an acute training intervention, it is possible that the dynamic SMP assessment methods do not reflect functional improvements to limb performance and/or that sensorimotor performance may not be altered in the knee flexor musculature of 'normal' athletic individuals. This could imply that most injuries do indeed occur during conditions of acute fatigue, when there is a reduction to both strength and sensorimotor control of the knee joint, leaving the lower limb vulnerable in terms of the ability to both detect and correct disruption to dynamic support.

9.2. Recommendations and future work.

The observed variation in sensorimotor performance assessment indices within the 'normal' recreationally active subject group that were observed in study 5.0 may not reflect the variation within the elite athletic population or during clinical, rehabilitative circumstances. Further research into such specific sub-groups of the population would help to further this preliminary research study into the reproducibility and single-measurement reliability of dynamic sensorimotor performance assessments.

Fatigue elicited a transient disruption to the performance indices of strength, neuromuscular activation and also the accuracy of sensorimotor performance of the knee flexors in chapter 6.0. This alteration to both isometric force production and awareness was sustained for up to 6 minutes following the cessation of exercise. Such significant effects from an acute fatigue intervention may be even more pronounced if this experiment is repeated in the future using the non-preferred limb. Future injury prevention strategies may also benefit from 'training' sensorimotor performance via force recognition and reproduction *during* conditions of fatigue, when dynamic support, neuromuscular activation and force response awareness around the knee joint are compromised. A replication of this study using a population of male

soccer players would provide an interesting comparison of sensorimotor and neuromuscular performance of the knee flexor musculature during fatigue.

It is recommended that future research into the effects of EIMD should re-assess the intensity of eccentric exercise that is required to induce significant muscle damage in the hamstrings musculature. The use of a subjective rating system following each isometric force production could also help to determine whether an individual can perceive the accuracy of his/her sensorimotor performance; and if this ability is indeed enhanced during the presence of pain. Objective assessments of sensorimotor performance may also help to individualize progression throughout a given training intervention. This confirms the need to progress beyond the paradigm of error detection and to focus on the ability of an athlete to integrate neuromuscular and sensorimotor feedback into functional movements. The question of whether an athlete can actually improve his/her neuromuscular and sensorimotor performance via the described training techniques may be examined in the future by using a non-athetes vs. athletes prospective intervention study design or alternatively by comparing the capacity for improved performance in elite athletes vs. recreational athletes. The incorporation of functional movements as part of the outcome assessment criteria may also benefit future training intervention studies.

As all studies within this thesis have focused exclusively on the knee flexors, it would be interesting to assess the effect of fatigue, EIMD and training on neuromuscular and sensorimotor performance in *both* the knee flexors and extensors. This rationale may be justified by the fact that individual muscles are seldom required to generate force in isolation (Caroll et al. 2001).

It has not been possible to pinpoint a single factor as *the* most important in knee joint control within the time constraints of this research. It appears that the strength of the knee flexor musculature was compromised by up to 20% in the chosen female population during both acute and long-term exercise-related interventions. However, it is not yet known what level of maximal force performance may actually represent a physical threat to knee joint support. Likewise, a relatively small loss of sensorimotor performance was observed in comparison to strength loss within the chosen research studies (~4% vs. ~20% for SMP and PFv, respectively) but this does not necessarily mean that less importance should be placed on sensorimotor performance for the preservation of joint homeostasis. This theory is supported by the fact that both neuromuscular switch on times and the accuracy of force recognition were preserved during prolonged intra-muscular disruption of knee flexors. There is a distinct need for several further research projects within this chosen area of study, as identified in figure 9.1. An equal emphasis on *both* efficient neuromuscular control and accurate dynamic sensorimotor response may be the key to maintaining optimal joint protection and ultimately the prevention of sports injuries associated with the lower limb in female athletes during functional movement patterns.

GLOSSARY.

ACL:	Anterior cruciate ligament.
ANOVA:	Univariate analysis of variance.
DOMS:	Delayed onset muscle soreness.
EIMD:	Exercise induced muscle damage.
EMD:	Electromechanical delay is defined as the time delay between the onset of muscle activity and onset of acceleration (Norman and Komi, 1979).
p:	Attained significance level.
PFv:	Volitional peak force.
R_I:	Intraclass correlation coefficient. This statistical index describes single-measurement reliability (Winer, 1981).
SD:	Standard deviation.
SEM:	Standard error of the measurement
SEM%:	The standard error of a single measurement (computed as a percentage of the group mean score at 95% confidence limits).
SMP:	Sensorimotor performance.
V%:	Coefficient of variation.

References.

Abbott, L.C., Saunders, J.B., Bost, F.C. and Anderson, C.E. (1944). Injuries to the ligaments of the knee joint. *Journal of Bone and Joint Surgery,* **26** A, 503 - 521.

Ageberg, E. (2002). Consequences of a ligament injury on neuromuscular function and relevance to rehabilitation using the anterior cruciate ligament-injured knee as a model. *Journal of Electromyography and Kinesiology,* **12**, 205-212.

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

Ashton-Miller, J.A., Wojtys, E.M., Huston, L.J., Fry-Welch, D. (2001). Can proprioception really be improved by exercises? *Knee Surgery, Sports Traumatology, Arthroscopy*, **9**, 128-136.

Baker, D., Wilson, G., Carlyon, B. (1994). Generality versus specificity: a comparison of dynamic and isometric measures of strength and speed-strength. *European Journal of Applied Physiology*, **68**, 350-355.

Barrack, R.L., Skinner, H.B., Cook, S.D. and Haddad, R.J. (1983). Effect of articular disease and total knee arthroplasty on knee joint-position sense. *Journal of Neurophysiology.* **50**, 684-687.

Barrack, R.L., Skinner, H.B., Brunet, M.E. and Cook, S.D. (1984). Joint kinesthesia in the highly trained knee. *Journal of Sports Medicine*, **24**, 18-20.

Barrack, R.L., Buckley, S.L. and Skinner, H.B. (1989). Proprioception in anterior cruciate deficient knee. *American Journal of Sports Medicine*, **17**, 1-6.

Barrack, L., Lund, P.J. and Skinner, H.B. (1994). Knee joint proprioception revisited. *Journal of Sports Rehabilitation,* **3**, 18 – 42.

Barrett, D.S., Cobb, A.G. and Bentley, G. (1991). Joint proprioception in normal osteoarthritic and replaced knees. *Journal of Bone and Joint Surgery (British).* **73**, 53-56.

Basmajian, J.V. and De Luca, C.J. (1985). Muscles Alive: Their functions revealed by electromyography. (Williams and Wilkins, Baltimore. MD).

Beard, D., Kyberd, R., Fergusson, C. and Dodd, C. (1993). Proprioception after rupture of the Anterior Cruciate Ligament. *Journal of Bone and Joint Surgery,* **75B**, 311-315.

Beynnon, B.D. and Johnson, R.J. (1996). Anterior cruciate ligament injury rehabilitation in athletes. *Sports Medicine*, **22**, 54-64.

Beynnon, B.D., Ryder, S.H., Konradsen, L. et al. (1999). The effect of antertior cruciate ligament trauma and bracing on knee proprioception. *American Journal of Sports Medicine*, **27** (2), 150-155.

Beynnon, B.D., Renstrom, P.A., Konradsen, L., Elmqvist, L.G., Gottleib, D., Dirks, M. (2000). Validation of techniques to measure knee proprioception. In Lephart, S.M and Fu, F. (eds) (2000). Proprioception and Neuromuscular Control in Joint Stability. Chapter 12. Human Kinetics. USA.

Bigland-Ritchie, B., Cafarelli, E. and Vollestad, N.K. (1986). Fatigue of submaximal static contractions. *Acta Physiologica Scandanavica*, **128**,137-148.

Boden, B.P., Dean, G.S., Feagin, J.A. and Garrett, W.E. (2001). Mechanisms of anterior cruciate ligament injury. *Orthopaedics*, **23**(6), 573-578.

Borsa, P.A., Lephart, S.M., Irrang, J.J. and Safran, M.R. (1997). The effects of joint position and direction of joint motion on proprioceptive sensibility in anterior cruciate ligament-deficient athletes. *American Journal of Sports Medicine*, **25**(3), 336-340.

Brockett, C., Warren, N., Gregory, J.E., Morgan, D.L., Proske, U. (1997). A comparison of the effects of concentric versus eccentric exercise on force and position sense at the human elbow joint. *Experimental Brain Research,* **771**, 251-258.

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

Burgess, P.R., and Jones, L.F. (1997). Perceptions of effort and heaviness during fatigue and during the size weight illusion. *Somatosensory & Motor Research*, **14** (3), 189-202.

Byrne, C. and Eston, R.G. (1998). Exercise induced muscle damage and delayed onset muscle soreness. *Sports, Exercise and Injury*, **4**, 69-73.

Byrnes, W.C., Clarkson, P.M., Spencer-White, J. and Hseih, S.S. (1985). Delayed onset muscle soreness following repeated-bouts of downhill running. *Journal of Applied Physiology,* **59**, 710-715.

Cafarelli, E. and Bigland-Ritchie, B. (1979). Sensation of static force in muscles of different length. *Experimental Neurology*, **65**, 511-525.

Cafarelli, E. (1982). Peripheral contributions to the perception of effort, *Medicine in Science, Sports and Exercise*, **14** (5), 382-9.

Cain, W.S. and Stevens, J.C. (1971). Effort in sustained and phasic handgrip contractions. *American Journal of Psychology*, **84**, 52-65.

Caraffa, A., Cerulli, G., Projetti, M. et al. (1996). Prevention of anterior cruciate ligament injuries in soccer. A prospective controlled study of proprioceptive training. *Knee Surgery, Sports Traumatology, Arthroscopy*, **4**, 19-21.

Caroll, S.R. and Carson, R.G., (2001). Neural adaptations to resistance training: Implications for movement control. *Sports Medicine*, **31,** (12), 829-840.

Carter, N.D., Jenkinson, T.R., Wilson, D. et al. (1997). Joint position sense and rehabilitation in the anterior cruciate deficient knee. *British Journal of Sports Medicine*, **31** (3), 209-212.

Clarkson, P.M., Byrnes, W.C., Gillison, E. et al. (1987). Adaptation to exercise-induced muscle damage. *Clinical Science,* **73,** 383-386.

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

Clarkson, P.M. and Newham, D.J. (1995). Associations between muscle soreness, damage and fatigue. In: Gandevia, S.C., Enoka, R.M., McComas, A.J., Stuart, D.G., Thomas, C.K., eds. Fatigue: Neural and Muscular Mechanisms. New York: Plenum press. 457-569.

Cleak, M.J. and Eston, R.G. (1992). Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. *British Journal of Sports Medicine*, **26**, 267-272.

Cohen, L.A. (1955). Activity of knee joint proprioceptors recorded from posterior articular nerve. *Yale Journal of Biological Medicine* **28**, 225 - 232.

Colebatch, J.G. and McCloskey, D.I. (1987). Maintenance of constant arm position or force: Reflex and volitional components in man. *Journal of Physiology,* **386**, 247-261.

Corrigan, J.P., Cashman, W.F. and Brady, M.P. (1992). Proprioception in the cruciate deficient knee. *British Journal of Bone and Joint Surgery,* **74**(2), 247-250.

De Vries, H.A. (1968). Method for the evaluation of muscle fatigue and endurance from electromyographic fatigue curves. *American Journal of Physical Medicine*, **58**, 70-85.

Drouin, J.M., Houghlum, P.A., Perrin, D.H., and Gansneder, B.M. (2003). Weight-bearing and non-weight-bearing knee-joint reposition sense and functional performance. *Journal of Sport Rehabilitation.* **12**, 54-66.

Dvorak, J., Junge, A., Chomiak, J. et al. (2000). Risk factor analysis for injuries in football players: Possibilities for a prevention program. *American Journal of Sports Medicine,* **28** (Suppl): S69-S74.

Dye, S.F. (2000). Functional Anatomy of the Cerebellum. In Lephart, S.M and Fu, F. (Eds) (2000). Proprioception and Neuromuscular Control in Joint Stability. Chapter 3. Human Kinetics. USA.

Enoka, R.M. (1995). Neuromechanical Basis of Kinesiology (2nd edition). Champaign, IL. Human Kinetics. Pp. 193-200.

Eston, R.G., Finney, S., Baker, S. and Baltzopoulous, V. (1996). Muscle tenderness and peak torque changes after downhill running following a prior bout of isokinetic eccentric exercise. *Journal of Sports Science*, **14**, 291-299.

Eston, R.G. and Byrne, C. (1998). Exercise, muscle damage and delayed onset muscle soreness. *Sports, Exercise and Injury*, **4**, 69-73.

Feldt, L.S. (1990). The sampling theory for the intraclass reliability co-efficient. *Applied Measurement in Education*, **3** (4), 361-7.

Fitts, P.M. and Posner, M.I. (1967). Human Performance. Pacific Grove, CA: Brooks/Cole.

Flanagan, J.R., Wing, A.M., Allison, S., and Spenceley, A. (1995). Effects of surface texture on weight perception when lifting objects with a precision grip. *Perceptual Psychophysics*, **53**, 315-324.

Friden, T., Roberts, D., Roberts, D., Movin, T. et al. (1998). Function after anterior cruciate ligament injuries. Influence of visual control and proprioception. *Acta Orthopedica Scandanavica,* **69**(6), 590-594.

Gabriel, D.A., Basford, J.R. and An, K. (2001). Neural adaptations to fatigue: implications to muscle strength and training. *Medicine in Science, Sports and Exercise*, **33** (8), 1354-1360.

Gandevia, S.C. and McCloskey, D.I. (1977). Effects of related sensory inputs on motor performances in man studied through changes in perceived heaviness. *Journal of Physiology*, **272**, 653-672.

Gandevia, S.C., and McCloskey, D.I. (1977b). Sensations of heaviness. *Brain*. **100**, 345-354.

Gandevia, S.C., and Rothwell, J.C. (1987). Knowledge of motor commands and the recruitment of human motoneurons, *Brain.* **110**, 1117-1130.

Gleeson, M., Blannin, A.K., Zhu, B. et al. (1995). Cardiorespiratory, hormonal and haematological responses to submaximal cycling performed 2 days after eccentric or concentric exercise bouts. *Journal of Sports Science*, **13**, 471-479.

Gleeson, M. Blannin, A.K., Walsh, N.P. et al. (1998). Effect of exercise-induced muscle damage on the blood lactate response to incremental exercise in humans. *European Journal of Applied Physiology.* **77**, 292-295.

Gleeson, N.P., and Mercer, T.H. (1996). The utility of isokinetic dynamometry in the assessment of human muscle function. *Sports Medicine*, **21**(1), 18-34.

Gleeson, N.P., Mercer, T., Morris, K. et al. (1997). Influence of a fatigue task on electromechanical delay in the knee flexors of soccer players [abstract]. *Medicine in Science, Sports and Exercise*, **29**, S281.

Gleeson, N.P., Rees, D., Doyle, J., Walters, M., Minshull, C. and Bailey, A. (1998). The effects of anterior cruciate ligament reconstructive surgery and acute physical rehabilitation on neuromuscular modelling associated with the knee joint. British Association of Sports and Exercise Sciences Annual Conference, Worcester College, November.

Gleeson, N.P., Mercer, T.H., *et al.* (1998). The influence of acute endurance activity on leg neuromuscular and musculoskeletal performance. *Medicine and Science in Sports and Exercise*, **30**, 596-608.

Gleeson, N.P. (2001). Assessment of neuromuscular performance using electromyography. In Eston, R. and Reilly, T. (Eds). Kinanthropometry and Exercise Physiology Laboratory Manual: Tests, procedures and data. Second Edition. Volume 2: Exercise Physiology. Pp 47-50. Routledge. New York.

Gleeson, N.P., and Mercer, T.H. The efficacy of measurement and evaluation in evidence-based clinical practice. *Physical Therapy in Sport*, (in press).

Gleim, G.W., McHugh, M.P. (1997). Flexibility and its effects on sports injuries and performance. *Sports Medicine*, **24**, 289-299.

Golden, C.L., Graves, J.E., Buchanan, P. and Dudley, G. (1991). Eccentric and concentric strength after repeated bouts of intense exercise. *Medicine and Science in Sports and Exercise*, **23** (Suppl), 655A.

Grieve, D.W. (1975). Electromyography. In: Techniques for the Analysis of Human Movement. Eds. D.W. Greive, D.L Miller, D, Mitchelson, *et al.* (Lepus Books, London).

Guyton, A.C. (Eds). (1986). Textbook of Medical Physiology. 7th Edition. Philadelphia: Saunders.

Guyton, A.C. and Hall, J.E. (1996). Textbook of Medical Physiology. 9th edition. Philadelphia, PA: WB Saunders Co. 699-712.

Hawkins, R.D., Hulse, M.A., Wilkinson, C., Hodson, A., and Gibson, M. (2001). The association football medical research programme: an audit of injuries in professional football. *British Journal of Sports Medicine,* **35**, 43-47.

Heidt, R.S., Sweeterman, L.M., Carlonas, R.L. et al. (2000). Avoidance of soccer injuries with pre-season conditioning. *American Journal of Sports Medicine*, **28**, 659-662.

Heiser, T.M., Weber, J., Sullivan, G., et al. (1984). Prophylaxis and management of hamstring muscle injuries in inter collegiate football players. *American Journal of Sports Medicine*, **12,** 368-370.

Hewett, T.E., Lindenfield, T.N., Riccobene, J.V. and Noyes, F.R. (1999). The effect of neuromuscular training on the incidence of knee injury in female athletes. *The American Journal of Sports Medicine*, **27** (6), 699-706.

Hopkins, W.G. (2002). A New View of Statistics.
www.sportsci.org/resource/stats/stdev.html.

Huston, L.J., and Wojtys, E. (1996). Neuromuscular performance characteristics in elite female athletes. *American Journal of Sports Medicine*, **24**(4), 427-436.

Huston, L.J., Greenfield, M.L. and Wojtys, E.M. (2000). Anteror cruciate ligament injuries in the female athlete: potential risk factors. *Clinical Orthopaedics and Related Research,* **372**, 50-63.

Hutchinson, M.R. and Ireland, M.L. (1995). Knee injuries in female athletes. *Sports Medicine*, **19** (4), 288 - 302.

Ireland, M.J. (2000). Proprioception and neuromuscular control related to the female athlete. In Lephart, S.M and Fu, F. (Eds) (2000). Proprioception and Neuromuscular Control in Joint Stability. Chapter 26. Human Kinetics. USA.

Jennings, A.G. and Seedhom, B.B. (1994). Proprioception in the knee and reflex hamstrings contraction latency. *British Journal of Bone and Joint Surgery*, **76B** (3), 491-494.

Jennings, A.G. (1994). A proprioceptive role for the anterior cruciate ligament: A review of the literature. *Journal of Orthopaedic Rheumatology*, **7**, 3-13.

Jerosch, J. and Prymka, M. (1996). Knee proprioception and joint stability. *Knee Surgery, Sports Traumatology, Arthroscopy*, **4**, 171-179.

Johannsen, H. (1991). Role of knee ligaments in proprioception and regulation of muscle stiffness. *Journal of Electromyography and Kinesiology*, **1** (3), 158 - 179.

Johannsen, H. (1991). A sensory role for the cruciate ligaments. *Clinical Orthopaedics and Related Research,* **268**, 161-178.

Jones, L.A. and Hunter, I.W. (1982a). Force sensation in isometric contractions: A relative force effect. *Brain Research,* **244**: 186-189.

Jones, L.A. and Hunter, I.W. (1983). Percieved force in fatiguing isometric contractions. *Perception and Psychophysics*, **33** (4) 369-374.

Jones, D.A., Newham, D.J. and Torgan, C. (1989). Mechanical influences on long-lasting human muscle fatigue and delayed onset pain. *Journal of Physiology*, **412**, 415-427.

Jones, D.A. and Round, J.M. (1993). Skeletal Muscle in Health and Disease. A textbook of muscle Physiology. Manchester; Manchester University Press.

Junge, A., Rosch, D., Peterson, L. et al. (2002). Prevention of soccer injuries: A prospective intervention study in youth amateur players. *American Journal of Sports Medicine*, **30** (5), 652-659.

Kakuda, N., Wessberg, J. and Vallbo, A.B. (1997). Is human muscle spindle afference dependent on perceived size of error in visual tracking? *Experimental Brain Research*, **114**, 246-254.

Karlsson, J., Funderburk, C.F., Essen, B. and Lind, A.R. (1975). Constituents of human muscle in isometric fatigue. *Journal of Applied Physiology*. 38: 208-211.

Kay, D., Marino, F.E., Cannon, J. et al. (2001). Evidence for neuromuscular fatigue during high-intensity cycling in warm, humid conditions. *Journal of Appied Physiology*, **84** (1-2), 115-21.

Kennedy, J.C., Alexander, I.J. and Hayes, K.C. (1982). Nerve supply of the human knee and its functional importance. *American Journal of Sports Medicine*, **10**, 329-35.

Kent-Braun, J. A. and Le Blanc, R. (1996). Quantification of central activation failure during maximal voluntary contractions in humans. *Muscle and Nerve*, **19** (7), 861-9.

Knapik, J.J., Bauman, C.L., Jones, B.H., Harris, J.M. and Vaughan, L. (1991). Pre-season strength and flexibility imbalances associated with athletic injuries in female collegiate athletes. *American Journal of Sports Medicine,* **19**, 76-81.

Knapik, J.K., Jones, B.H., Bauman, C.L., and Harris, J. 1992. Strength, flexibility and athletic injuries. *Sports Medicine*, **14** (5), 277-288.

Kramer, J., Handfield, T., Kiefer, G., Forwell, L. and Birmingham, T. (1997). Comparisons of weight bearing and non-weight bearing tests of knee proprioception performed by patients

with P-F syndrome and asymptomatic individuals. *Clinical Journal of Sports Medicine*, **7**, 113-118.

Lattanzio, P.J., Petrella, R.J., Sproule, J.R. and Fowler, P.J. (1997). The effects of fatigue on knee proprioception. *Clinical Journal of Sports Medicine*, **7**, 22-27.

Lephart, S.M., Fu, F.H., Borsa, P.A., et al. (1991). Proprioception of the knee and shoulder joint in normal, athletic, capsule-ligamentous pathological and post-reconstruction individuals. *Orthopaedic Transcripts*, **18**, 1157.

Lephart, S.M., Kocher, M.S., Fu, F.H., Borsa, P.A. and Harner, C.D. (1992). Proprioception following anterior cruciate ligament reconstruction. *Journal of Sports Rehabilitation,* **1**, 188-196.

Lephart,S.M., Kocher, M.S., Harner, C.D., and Fu, F.H. (1993). Quadriceps strength and functional capacity after anterior cruciate ligament reconstruction. Patellar tendon autograft versus allograft. *American Journal of Sports Medicine,* **21**(5), 738-743.

Lephart, S.M and Fu, F. (Eds) (2000). Proprioception and Neuromuscular Control in Joint Stability. Chapter 12. Human Kinetics. USA.

MacDougall, J.D., Elder, G.C.B., Sale, D.G., Moroz, J.R. and Sutton, J.R., (1980). Effects of strength training and immobilization on human muscle fibres. *European Journal of Applied Physiology*, **43**, 25-34.

MacDonald, P.B., Hedden, D., Pacin, O. et al. (1996). Proprioception in anterior cruciate ligament deficient and reconstructed knees. *American Journal of Sports Medicine*, **24**(6), 774-778.

Mair, J., Mayr, M. and Muller, E. (1995). Rapid adaptation to eccentric exercise-induced muscle damage. *International Journal of Sports Medicine*. **16**, 352-356.

Marks, R. and Quinney, H.A. (1993). Effect of fatiguing maximal isokinetic quadriceps contractions on ability to estimate knee position. *Perceptual Motor Skills,* **77**, 1195-1202.

Marks, R. and Quinney, A.H. (1996). Position sense perception in healthy persons and persons with chronic degenerative arthritis. *Clinical Kinesiology*, **50** (4), 77 - 82.

Marks, R. and Quinney, A.H. (1997). Position sense perception at the knee in healthy persons and persons with chronic degenerative arthritis. *Clinical Kinesiology,* **50** (4), 76-82.

McCloskey, D.I. (1980). Kinaesthetic sensations and motor commands in man. In J.E. Desement (Eds). Spinal and supraspinal mechanisms of voluntary motor control and locomotion (Vol. 8). Basel: Karger.

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

McHugh, M.P., Connolly, D.A., Eston., R.G. and Gleim, G.W. (1999). Exercise-induced muscle damage and potential mechanisms for the repeated-bout effect. *Sports Medicine*, **27** (3), 157-170.

McHugh, M.P., Connoly, D.A.J., and Eston, R.G. (1999). The role of passive muscle stiffness in symptoms of exercise induced muscle damage. *American Journal of Sports Medicine,* **27**, 594 -599.

McHugh, M.P. (2000). Can exercise-induced muscle damage be avoided? *Western Journal of Medicine*, **172**, 265 – 266.

Mercer, T.H., and Gleeson, N.P. (1996). Prolonged intermittent high intensity exercise impairs neuromuscular performance of the knee flexors. *Physiologist*, **39**, A-62.

Mercer, T.H., Gleeson, N.P., Claridge, S. and Clement, S. (1998). Prolonged intermittent high intensity exercise impairs neuromuscular performance of the knee flexors. *European Journal of Applied Physiology and Occupational Physiology*, **77,** 560-2.

Minshull, C.M. (2003). Unpublished Ph.D., data observations. University of Wales, Bangor. U.K.

Moore, M.A. and Kukilka, C.G. (1991). Depression of Hoffman reflexes following voluntary contraction and implications for proprioceptive neuromuscular facilitation therapy. *Physical Therapy*, **71**, 35-41.

Mulder, T., and Hulstyn, W. (1984). Sensory feedback therapy and theoretical knowledge of motor control and learning. *American Journal of Physical Medicine.* **63** (5), 226-244.

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

Newham, D.J. (1988). The consequences of eccentric contractions and their relationship to delayed onset muscle pain. *European Journal of Applied Physiology*, **57**, 353-359.

Norman, R.W. and Komi, P.V. (1979). Electromechanical delay in skeletal muscle under normal movement conditions. *Acta Physiologica Scandanavica*, **106**, 241-248.

Nosaska, K., Clarkson, P.M., McGuiggin, M.E. and Byrne, J.M. (1991). Time course of muscle adaptation after high force eccentric exercise. *European Journal of Applied Physiology*. (63) 70-76.

Nosaska, K. and Clarkson, P.M. (1995). Muscle damage following repeated-bouts of high force eccentric exercise. *Medicine in Science, Sports and Exercise*. 27:1263-1269.

Nosaska, K. and Clarkson, P.M. (1997). Influence of previous concentric exercise on eccentric exercise-induced muscle damage. *Journal of Sports Science,* **15**, 477-483.

Nosaska, K., Sakamoto, K., Newton, M. and Sacco, P. (2001). The repeated bout effect of reduced-load eccentric exercise on elbow flexor muscle damage. *European Journal of Applied Physiology,* **85**, 34-40.

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

Nosse, L.J. (1982). Assessment of selected reports on the strength relationship of the knee musculature. *Journal of Orthopaedic and Sports Physical Therapy*, **4**, 78-85.

Palmar, I. (1944). Pathophysiology of the medial ligament of the knee joint. *Acta Chir Scandinavica*, **91**, 37-48.

Pap, G., Machner, A., Nebelung, W. et al. (1999). Detailed analysis of proprioception in normal and ACL deficient knees. *Journal of Bone and Joint Surgery (British),* **81** (5), 764-768.

Pertofsky, J.S. (1980). Computer analysis of the surface EMG during isometric exercise. *Computers in Biology and Medicine*, **10,** 83-95. .

Pincivero, D.M., Coelho, A.J., and Erikson, W.H. (2000). Perceived exertion during isometric quadriceps contraction. A comparison between men and women. *Journal of Sports Medicine and Physical Fitness*, **40**, 319 – 326.

Pizza, F.X., Davis, B.H. and Henrickson, S.D. (1996). Adaptation to eccentric exercise: effect on CD64 and CD11b/CD18 expression. *Journal of Applied Physiology*, **80**, 47-55.

Pope, M.H., Johnson, R.J., Brown, D.W. and Tighe, C. (1979). The role of the musculature in injuries to the medial collateral ligament. *Journal of Bone and Joint Surgery*, **61A**, 398-402.

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

Rauber, A. (1874). Uber die Vater'schen Korper der Gelenkkapseln. *Centr Med Wiss*. **12**, 305-306.

Rees, D. (1994). ACL reconstructions: Possible modes of failure. **Proceedings of the Royal College of Surgeons [Edinburgh] Football Association Sixth Joint Conference on Sports Injury.** Lilleshall, 2nd-3rd July.

Roberts, D., Friden, T., Zatterstrom, R., Lindstrand, A. et al. (1999). Proprioception in people with anterior cruciate ligament deficient knees: comparison of symptomatic and asymptomatic patients. *Orthopaedic and Sports Physical Therapy*, **29**(10), 587-594.

Rozzi, S.L., Lephart, S.M., Gear, W.S., and Fu, F. (1999). Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. *American Journal of Sports Medicine,* **27** (3), 312-319.

Rutherford, O.M., Jones, D.A. and Newham, D.J. (1986). Clinical and experimental application of the percutaneous twitch superimposition technique for the study of human muscle activation. *Journal of Neurology, Neurosurgery and Psychiatry*, **49**, 1288-1291.

Saxton, J.M., Clarkson, P.M., James, R., Miles, M., Westerfer, M., Clark, S. and Donnelly, A.E. (1995). Neuromuscular Dysfunction Following Eccentric Exercise. *Medicine and Science in Sports and Exercise*, **27** (8), 1185 – 1193.

Saxton, J. and Donnelly, A.E. (1996). Length-specific impairment of skeletal muscle contractile function after eccentric muscle actions in man. *Clinical Science,* **90**, 119-125.

Schmidt, R.A. (1988). Motor Control and Learning. Human Kinetics. Champaign, Illinois. 55 – 61.

Schutte, M.J, Dabezies, E.J., Zimny, M.L. and Happel, L.T. (1987). Neural anatomy of the human cruciate ligament. *Journal of Bone Joint Surgery* (American), **69**, 243 - 247.

Schwane, J.A., and Armstrong, R.B. (1983). Effects of training on skeletal muscle injury from downhill running in rats. *Journal of Applied Physiology*. **55** (3), 969-75.

Sherrington, C.S. (1906). (Eds). The integrative action of the nervous system. New Haven, CT: Yale University Press.

Shumway-Cook, A. and Woollacott, M.M. (1995). Motor Control. Theory and Practical Applications, 1st edition. Baltimore: Williams &Wilkins.

Sjolander, P., Johannsen, H. and Djupsjobacka. (In Press). Spinal and supraspinal effects of activity in ligament afferents. *Journal of Electromyography and Kinesiology.*

Skinner, H.B., Wyatt, M.P. and Hodgdon, J.A. et al. (1986). Effect of fatigue on joint position sense of the knee. *Journal of Orthopaedic Research,* **4,** 112-118.

Sokal, R. and Rohlf, F. (1981). Biometry. (2nd Ed.), Oxford, U.K. W.H. Freeman.

Solomonow, M., Baratta, B.H., Zhou, B.H., et al. (1987). The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. *American Journal of Sports Medicine*, **15**, 207-213.

Solomonow, M. and Krogsgaard, M. (2001). Sensorimotor control of knee stability: A Review. *Scandanavian Journal of Medicine & Science in Sports*, **11**, 64-80.

Stein, R.B., Gordon, T. and Shriver, J. (1982). Temperature dependence of mammalian muscle contractions and ATPase activities. *Journal of Biophysics,* **40,** 97-107.

Stupka, N., Lowther, S., Chorneyko, K. et al., (2000). Gender differences in muscle inflammation after eccentric exercise. *Journal of Applied Physiology,* **89,** 2325-2332.

Teghtsoonian, R., Teghtsoonian, M., and Karlsson, J.G. (1977). The effects of fatigue on the perception of muscular effort. In: Borg, G. (Eds.), Physical Work and Effort. Oxford: Pergamon Press.

Thomas, J.R. and Nelson, J.K. (1990). Introduction to Research in Health, Physical Education, Recreation and Dance (2nd edition). Champaign, Illinois: Human Kinetics.

Thompson, C.W. (deceased) and Floyd, R.T. (1994). Manual of Structural Kinesiology. Twelfth Edition. Mosby. Pp 115-119.

Vollestad, N.K., Sejersted, I., and Saugen, E. (1997). Mechanical behaviour of skeletal muscle during intermittent voluntary isometric contractions in humans. *Journal of Applied Physiology*, www.jap.org.

Vollestad, N.K. and Sejersted, O.M. (1988). Biomechanical correlates of fatigue: A brief review. *European Journal of applied Physiology,* **57**, 336 - 347.

Walla, D.J., Albright, J.P., McAuley, E. et al. (1985). Hamstring control and the unstable anterior cruciate ligament-deficient knee. *American Journal of Sports Medicine*, **13**, 34-39.

Westgaard, R.H. and De Luca, C.J. (1999). Motor unit substitution in long duration contractions of the human trapezius muscle. *J Neurphysiol*, **82**, 501-504.

Williams, K.M. (2001). Neuromuscular performance and the extent of fatigue in the knee flexors during periods of eccentric exercise-induced muscle damage. Unpublished M.Sc., dissertation. University of Wales, Bangor.U.K.

Winer, B.J. (1981). Statistical Principles in Experimental Design (2nd Ed.). McGraw-Hill. New York.

Winter, E.M. and Brookes, F.B.C. (1991). Electromechanical response times and muscle elasticity in men and women. *European Journal of Applied Physiology*, **63**, 124-8.

Wojtys, E,M., and Huston, L.J. (1994). Neuromuscular performance in normal and anterior cruciate ligament deficient lower extremities. *The American journal of Sports Medicine*, **22** (1), 89 –103.

Wojtys, E.M., Huston, L.J., Taylor, P.D. and Bastian, S.D. (1996). Neuromuscular adaptations to isokinetic, isotonic, and agility training programs. *American Journal of Sports Medicine* **24**, 187-192.

Wojtys, E.M., Huston, L.J. and Fry-Welch, D. (2001. Can proprioception really be improved by exercises? *Knee Surgery, Sports Traumatology, Arthroscopy*. **9**, 128-136.

Wyke, B. (1981). The neurology of joints: a review of general principles. *Clinics in Rheumatology,* **7**, 223-241.

Zhou, S., McKenna, M.J., Lawson, D.L. et al., (1996). Effects of fatigue and sprint training on electromechanical delay of knee extensor muscles. *European Journal of Applied Physiology,* **72**, 410-6.

Appendix 1.

Pre-study questionnaire.
Physiology informed consent and medical questionnaire.
Informed consent to participate in a research project form.

Form 6

PRE-STUDY QUESTIONNAIRE

NAME:

DATE:

RESEARCHER

		YES/NO
1.	HAVE YOU HAD ANY KIND OF ILLNESS OR INFECTION IN THE LAST 2 WEEKS?	.....
2.	ARE YOU TAKING ANY FORM OF MEDICATION?	.....
3	DO YOU HAVE ANY FORM OF INJURY?	.....
4.	HAVE YOU EATEN IN THE LAST HOUR?	.....
5.	HAVE YOU CONSUMED ANY ALCOHOL IN THE LAST 24 HOURS?	.....
6.	HAVE YOU PERFORMED EXHAUSTIVE EXERCISE WITHIN THE LAST 48 HOURS?	.....

IF THE ANSWER TO ANY OF THE ABOVE QUESTIONS IS YES, THEN YOU MUST CONSULT A MEMBER OF STAFF BEFORE UNDERGOING ANY EXERCISE TEST.

SIGNATURE OF PARTICIPANT .....

Form 4ii

PHYSIOLOGY INFORMED CONSENT & MEDICAL QUESTIONNAIRE

Name: .....

Age:.....

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

How would you describe your present level of activity? Tick intensity level and indicate approx. duration.

vigorous moderate low intensity

Duration (Min).

How Often:
< once per month
once per month
2-3 times per week
4-5 times per week
> 5 times per week

Have you 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

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

PLEASE READ THE FOLLOWING CAREFULLY

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

have a fever, suffer from fainting spells or dizziness;

have suspended training due to a joint or muscle injury;
have a known history of medical disorders, i.e. high blood pressure, 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, hemorrhoids, or any other condition of the rectum;

DECLARATION
I agree that I have none of he above conditions and I hereby volunteer to be a participant in experiments/investigations during the period of
.....20___.

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.

I understand that 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.

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.

I undertake to obey the laboratory/study regulations and the instructions of the experimenter regarding safety, subject only to my right to withdraw declared above.

Signature of Participant.....

Date:.....

Signature of Experimenter.....

Date: .....

Form 4i

INFORMED CONSENT TO PARTICIPATE IN A RESEARCH PROJECT OR EXPERIMENT

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: _____

DATE: _____

Appendix 2.

'Sit and reach test' normative values table.

Appendix 2.

Sit and reach 'Norm' scores for population.

	Men (cm)	Women (cm)
Super	> +27	> +30
excellent	+17 to +27	+21 to +30
good	+6 to +16	+11 to +20
average	0 to +5	+1 to +10
fair	-8 to –1	-7 to 0
poor	-19 to –9	-14 to -8
very poor	< -20	< -15

http://topendsports.com/testing/tests/sitandreach.htm

Appendix 3.

Training exercises for study 8.0.

Appendix 3.

Exercise details for the sensorimotor and neuromuscular training programme.

Exercise 1.

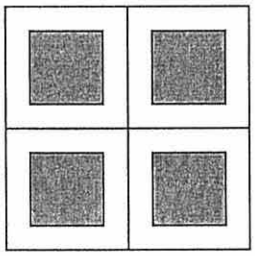
(i) Wobble board with eyes open/closed **(ii) Wobble board and ball catching**

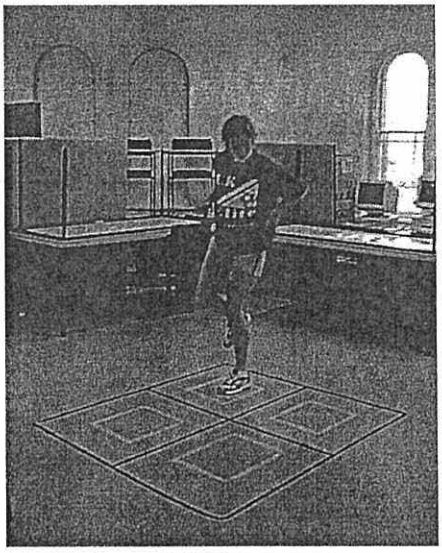
Progression:

Exercise	Wk 1 (sessions 1-6)	Wk 2 (sessions 7-12)
Wobble board whilst standing on the non-preferred limb.	Wobble board (eyes open) Wobble board (eyes closed)	Wobble board whilst receiving and returning a ball using the arms.

Exercise 2.

Grid hopping.





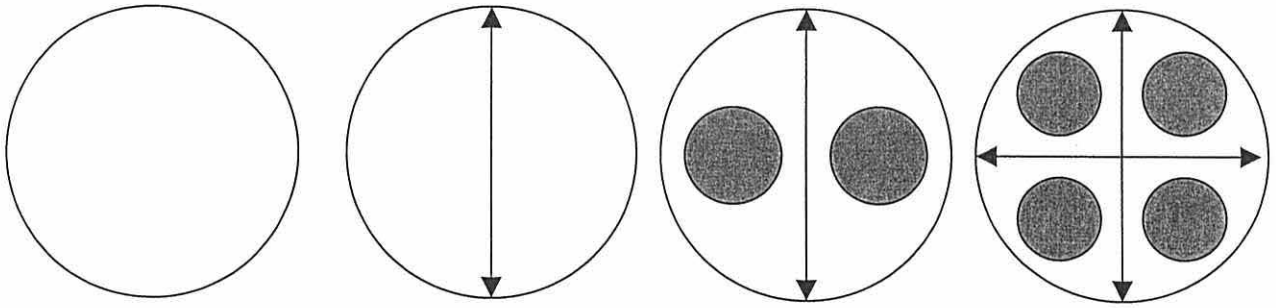
	Progression	
Exercise	**Wk 1 (sessions 1-6)**	**Wk 2 (sessions 7-12)**
Hopping on the non-preferred limb between the squares.	Hopping within large area of squares (random movement) in all directions. Emphasis on the **accuracy** of the movement.	Hopping within refined central area of square (random movement) in all directions. Emphasis on the **speed** of movement.

Exercise 3.

Trampoline hopping.

Exercise	Wk 1 (sessions 1-6)	Wk 2 (sessions 7-12)
Hopping on the trampoline using the non-preferred limb.	(1) Hopping within the whole area of the trampette. Emphasis on the **speed** of the hopping. (2) Hopping over chalk line from left to right. Emphasis on the **accuracy** of the movement.	(3) Hopping within refined central area on both sides of the trampette. Emphasis on **accuracy and height** of movement. (4) Hopping onto a refined central area of the targets within each quarter of circle in a random order. Emphasis on both **speed and accuracy**.

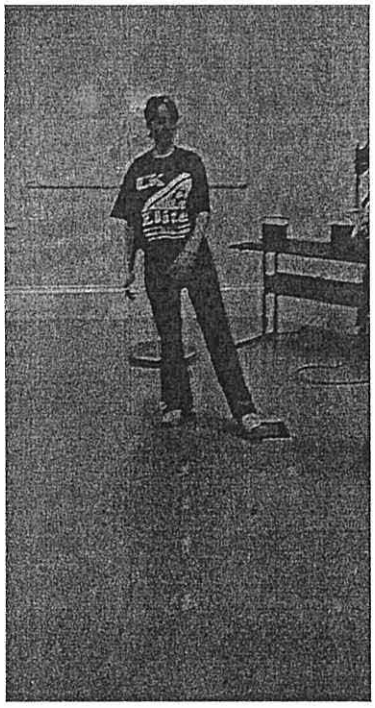
1. **2** **3** **4**

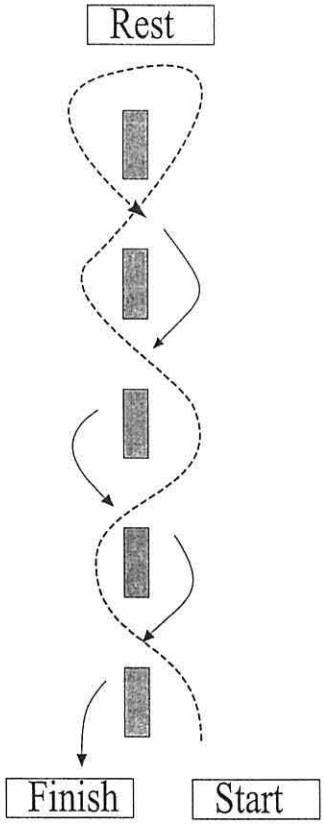


Exercise 4.

Hopping with movement.

Exercise	Wk 1 (sessions 1-6)	Wk 2 (sessions 7-12)
Agility hops between markers. Both the preferred and non-preferred limbs (on alternate attempts).	Hopping on non-preferred limb between floor markers in a figure of eight fashion.	As sessions 1-6 but with less space between the markers.

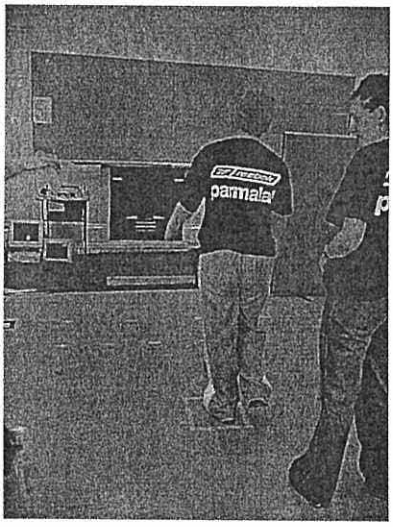




Exercise 5.

Unexpected perturbation from behind.

Exercise	Wk 1 (sessions 1-6)	Wk 2 (sessions 7-12)
Plyometric jumping. Non-preferred limb.	Jumping up in the air with an emphasis on soft landing and **accuracy** to land within the designated box on the floor.	As sessions 1-6 but with a peturbation to the back of the subject to unbalance her whilst she is off the ground. **Accuracy** of landing.



Exercise 6.

Backward jumping.

Exercise	Wk 1 (sessions 1-5)	Wk 2 (Sessions 6-10)
Jumping backwards onto one of three target boxes. Hopping forwards to return to starting position. (Non-preferred limb)	Hopping backwards onto target area with an emphasis on the **distance** of movement.	Hopping within a refined central area of the target boxes. Emphasis on the **accuracy and height** of movement.