EFFECTS OF ACUTE FATIGUE ON THE VOLITIONAL AND MAGNETICALLY-EVOKED ELECTROMECHANICAL DELAY OF THE KNEE FLEXORS IN MALES AND FEMALES

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Abstract

Neuromuscular performance capabilities, including those measured by evoked responses, may be adversely affected by fatigue; however, the capability of the neuromuscular system to initiate muscle force rapidly under these circumstances is yet to be established. Sex-differences in the acute responses of neuromuscular performance to exercise stress may be linked to evidence that females are much more vulnerable to ACL injury than males. Optimal functioning of the knee flexors is paramount to the dynamic stabilisation of the knee joint, therefore the aim of this investigation was to examine the effects of acute maximal intensity fatiguing exercise on the voluntary and magnetically-evoked electromechanical delay in the knee flexors of males and females. Knee flexor volitional and magnetically-evoked neuromuscular performance was assessed in seven male and nine females prior to and immediately after: (i) an intervention condition comprising a fatigue trial of 30-seconds maximal static exercise of the knee flexors, (ii) a control condition consisting of no exercise. The results showed that the fatigue intervention was associated with a substantive reduction in volitional peak force (PF_V) that was greater in males compared to females (15.0%, 10.2%, respectively, $p < 0.01$) and impairment to volitional electromechanical delay (EMD_V) in females exclusively (19.3%, $p < 0.05$). Similar improvements in magnetically-evoked electromechanical delay in males and females following fatigue (21%, $p < 0.001$), however, may suggest a vital facilitatory mechanism to overcome the effects of impaired voluntary capabilities, and a faster neuromuscular response that can be deployed during critical times to protect the joint system.

Keywords: Fatigue, neuromuscular performance, electromechanical delay, magnetic stimulation

Introduction

During strenuous activities, mechanical loading of the knee joint can often exceed the tensile capacities of the passive structures (Johansson et al. 1991). As a consequence, greater reliance may be placed on the protective capabilities of the surrounding musculature in order to maintain joint integrity (Gleeson et al. 1998a). Evidence of anterior cruciate ligament (ACL) injury by means of non-contact mechanisms in team sports athletes (Ireland et al. 1997; Mandelbaum et al. 2005; Rees 2004) underscores the potentially important contribution of neuromuscular mechanisms to the maintenance of dynamic joint stability and the avoidance of injury. As evidence shows that females are five to eight times more likely to injure their ACL compared to male counterparts given equivalent exposure to sport (Arendt and Dick, 1995; Ireland et al. 1997; Gray et al. 1985), study of factors that might affect the stability of the knee joint in females is important.

Optimal functioning of the knee flexors in particular is considered fundamental to the prevention of ACL injury (Gleeson and Mercer 1996; Johansson et al. 1991; Rees 1994), however, a limited time frame exists whereby potentially harmful dynamic forces must be overcome by the most rapid response of the neuromuscular system in order to protect ligamentous tissue against injury (Gleeson et al. 1998a; Huston and Wojtys 1996; Mercer et al. 1998; Shultz et al. 2001). For the ACL, the time frame from the initial application of such forces to the complete rupture of the ligament has been estimated at 300 ms (Rees, 1994). One aspect of the overall neuromuscular reaction time has been defined as electromechanical delay (EMD). It depicts the time between the onset of electrical activity and the onset of tension in skeletal muscle (Zhou et al. 1996) and is

associated with the propagation of the action potential through the muscle and the stretching of the series elastic component (Norman & Komi, 1979). It represents an important aspect of neuromuscular reaction time, during which there could be unrestrained development of forces of sufficient magnitude to damage ligamentous tissue in synovial joints (Gleeson et al. 2000; Huston and Wojtys 1996; Mercer et al. 1998; Shultz et al. 2001). The importance of this index of performance can be exemplified further by recognising that factors such as muscle fatigue can cause increases in EMD latency of up to 70% (Zhou et al. 1996). The extent of this change in EMD performance may also be influenced by the loading of viscoelastic structures, which can cause creep within the affected tissue and a modulation of the neuromuscular performance characteristics of the associated musculature (Chu et al. 2003; Sbriccoli et al, 2005; Solomonow, 2004; Solomonow et al. 2003). Clearly, any fatigue-related increases in muscle response time within the knee flexors to initiate force, coupled with the effects of increased ligamentous laxity and compliance within muscle-related connective tissue associated with cyclical loading during activity, may result in a hyper-lax system that is more likely to be incapable of restraining high joint loads rapidly enough to prevent ligamentous injury.

Traditionally, neuromuscular performance capabilities have been estimated routinely in the laboratory by means of assessment protocols involving volitional activation of muscle. Recent technological advances, however, have enabled the non-invasive and painless magnetic stimulation of a peripheral motor nerve; the efficacy of this technique has been considered in clinical populations where maximal volitional testing is not appropriate (Polkey et al. 1996; Vivodtzev et al. 2005). Peripheral magnetic stimulation of a nerve root offers the potential to activate the fastest motor units (King

and Chippa 1989; Maertens de Noordhout 1991) and overcome factors associated with the volitional activation of muscle that might otherwise intrude on the proper estimation of an individual's true maximal performance capacity. For example, factors such as autogenic neuromuscular inhibition associated with injury and conditioning status might tend to elicit an underestimation of performance capability even in the most highly motivated of individuals (Gleeson 2001; Hopkins and Ingersoll 2000). A corollary of this interpretation is that assessments of neuromuscular performance by means of magnetic stimulation may offer greater insights into the performance capability that might be available to the sports performer in emergency situations where there is a critical level of threat to the stability of the joint system.

Acute muscle fatigue induced by means of maximal voluntary muscle activation (MVMA) has the potential to cause dramatic increases in EMD; of between 42% to 70% longer compared with pre-fatigue values (Horita and Ishiko 1987; Zhou et al. 1996) and concomitant decreases in the capacity for generating peak force. Temporal impairments of this type to the dynamic muscle stabilisers of the knee joint, may affect the ability to stabilise the knee during competitive match-play and place the sports performer at increased risk of injury. Studies of fatigue-related changes to EMD measured using electrically-evoked activation of muscle, however, have yielded conflicting findings of impaired (Zhou 1996), unchanged (Strojnik and Komi 1998) and even improved performance (Sahlin and Seger 1995). Given the potential inhibitory effects on performance that pain may elicit under conditions of electrical stimulation, an evoked assessment of the neuromuscular system by means of magnetic stimulation**,** a technique that minimises intrusion of noxious stimuli, may offer a truer insight into the maximal

physiological capacity for rapid muscle activation. No studies have yet investigated the effects of acute muscle fatigue on the magnetically-evoked EMD of the knee flexors.

The aim of this investigation was to examine the effects of an acute bout of maximal intensity static fatiguing exercise on the voluntary and magnetically-evoked electromechanical delay in the knee flexors of males and females.

Methods

Subjects

Seven men (age: 29.6 (± 10.4) yrs; height 1.78 (± 0.04) m; body mass 77.0 (± 7.7) kg (mean [± SD]) and nine women (age 25.2 (± 4.2) yrs; height 1.69 (± 0.08) m; body mass 62.8 (± 8.1) kg) gave their informed consent to participate in this study. All participants were regularly involved in exercise (at least 3 times per week) and were asymptomatic at the time of assessment. Participants were instructed to refrain from strenuous physical activity for the 24 hours prior to the test. Assessment protocols were approved by the Ethics Committee for Human Testing of the University of Wales, Bangor.

Experimental procedures

Following habituation procedures, participants completed a standardised warm-up of five minutes cycle ergometry (90 watts for males, 60 watts for females) and a further five minutes of static stretching of the involved musculature. This warm-up was equivalent to that used in other recent studies within this laboratory examining the effects of various interventions on indices of volitional neuromuscular performance (Gleeson 2001; Gleeson et al. 2000; Gleeson et al. 1998a; Gleeson et al. 1998b; Mercer et al. 1998). Participants were then secured in a prone position on a custom-built dynamometer (Gleeson et al. 1995).

The experimental design comprised two treatment conditions: (i) an intervention condition that required participants to perform a fatigue trial of 30 seconds maximal static fatiguing exercise of the knee flexors of the preferred leg; (ii) a control condition of equivalent duration to the intervention consisting of no exercise. Treatment conditions were separated by twenty minutes. The control condition was performed first in order to

avoid any potential carry-over effects. Participants were verbally encouraged during periods of maximal muscle activation. Estimates of knee flexor volitional and magnetically-evoked neuromuscular performance were obtained prior to and immediately after each treatment condition. The protocol is illustrated schematically in figure 1.

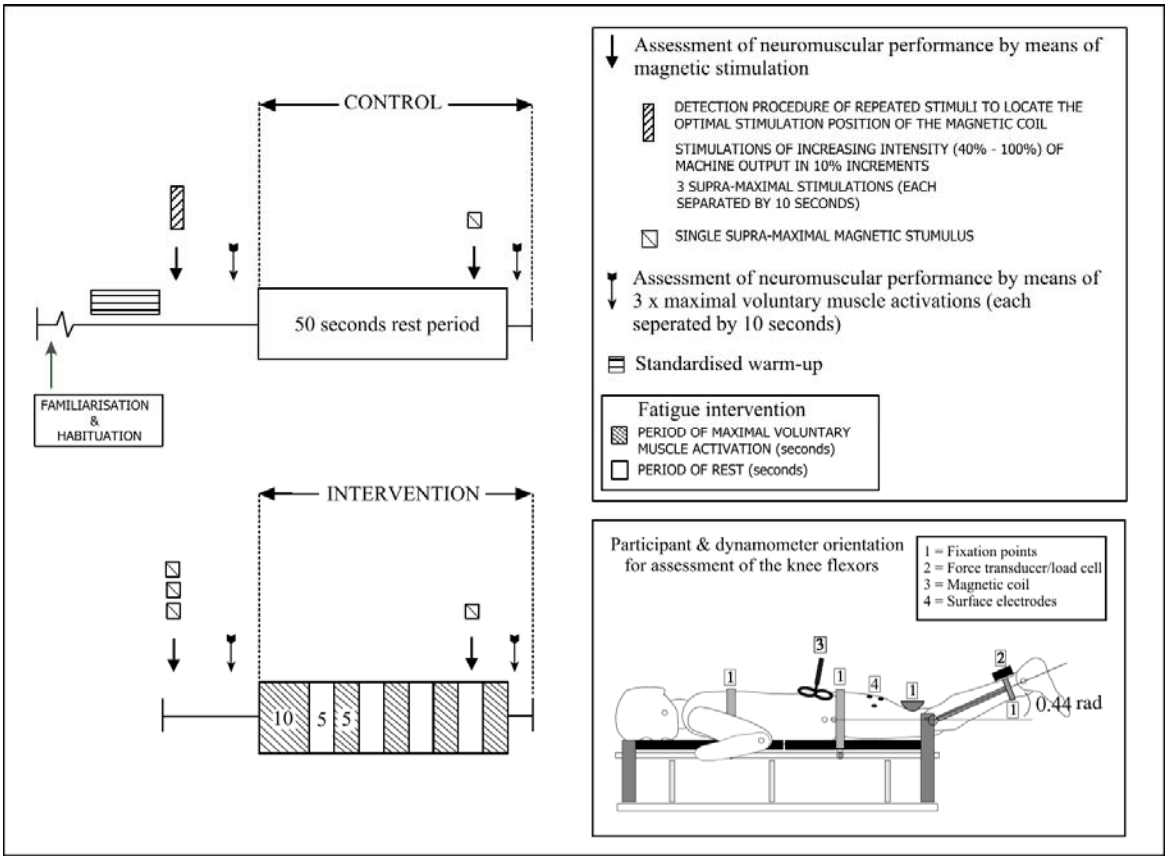

Figure 1. A schematic of the protocol to assess the effects of an acute fatiguing task on the volitional and magnetically-evoked neuromuscular performance of the knee flexors.

Adjustable strapping across the mid-thoracic spine, pelvis and posterior thigh proximal to the knee localised the action of the involved musculature. A functionally relevant knee flexion angle of 25 degrees (0.44 rad) associated with the greatest mechanical strain on key ligaments (Beynnon and Johnson 1996), was maintained throughout testing. This angle was identified for each participant during activation of the involved musculature using a goniometer system. Once secured into position and prior to testing, participants were required to perform a series of warm-up muscle activations, comprising of 2 x 25%, 50%,

75% and 100% of subjectively-judged maximal voluntary peak force. Each of the activations was sustained for three seconds and was separated from the next by 10 seconds. The orientation of the participant and dynamometer is illustrated schematically in figure 1.

Assessment of neuromuscular performance

Maximal volitional muscle activation (MVMA)

On receipt of an auditory signal, given randomly within 1-4 seconds, the participants attempted to activate their musculature as rapidly and forcefully as possible by attempting to flex the knee joint against the immovable restraint offered by the apparatus. Another auditory signal was given to the participant after 2 - 3 seconds of MVMA to cue neuromuscular relaxation. Intra-trial MVMA replicates were each separated by at least 10 seconds to enable neuromuscular recovery (Moore and Kukulka 1991).

Magnetically-evoked muscle activation

Supra-maximal magnetic stimulation of the sciatic nerve (L4-L5) and associated activation of the knee flexors was achieved by means of double wound coil (120 mm) that was powered by a Magstim 200 stimulator (Magstim Co. Ltd., Whitland, Dyfed, Wales). The optimum site for stimulation of the nerve was defined as the site that offered the largest amplitude of the compound muscle action potential (CMAP). This was identified by a procedure in which the centre of the magnetic coil was placed in a position 20 mm to 40 mm lateral to the fifth lumbar vertebra on the involved side and then small iterative positional changes of the coil were made that were commensurate with increasing CMAP responses during a series of discrete stimulations. This optimised coil position was maintained manually throughout the remainder of the test.

There appears to be no standardised way described in the literature that systematically verifies the attainment of supra-maximal magnetic stimulation of a peripheral motor nerve. Protocols to elicit supra-maximal stimulation of the femoral nerve have been described briefly in the literature (Polkey et al. 1996; Vivodtzev et al. 2005). However, these protocols have been limited to the verification of a supra-maximal response by changes in peak twitch force data only due to the intrusion of stimulation artefact compromising the quality of muscle EMG recordings. As such, CMAP amplitude responses have not previously been used in a verification process. A protocol was developed for the current study in which supra-maximal stimulation was defined as the intensity of stimulation at which there was subsequently no more than a 5% increase in CMAP peak amplitude despite a 10% or greater increase in the intensity of stimulation, and verified using a procedure that would mimic the approach to the physiological verification of the attainment of maximal oxygen uptake. Thus supra-maximal stimulation was verified by contemporaneous visual inspection of the data during a sequence of seven discrete stimulations of increasing intensity that commenced at 40% of the Magstim 200's maximal capacity output with subsequent increments of 10% to 100% of capacity. Retrospective analyses of CMAP amplitude and peak twitch force demonstrated proportionate and linear increases when plotted against one another. In the four participants from the present study whose CMAP amplitude did not by definition reach supra-maximal proportions, supplementary criteria that were based on minimal simultaneous increases in the performance of peak twitch force (P_TF_E) and electromechanical delay (< 5% increase in performance elicited by stimulations of increasing intensity between 80% and 100% of the Magstim 200's maximal capacity output) were used to verify that 'peak' amplitudes of CMAP had occurred (Minshull et al. 2002a; Minshull et al. 2002b). The latter instances were associated with limitations of the

technological capability of the stimulation system. Sequential stimulations throughout the experimental period were separated by at least 10 seconds to ensure neuromuscular recovery (Moore and Kukulka 1991).

Indices of neuromuscular performance

Peak force

Volitional static peak force (PF_V) was recorded as the mean response of three intra-trial replicates in which the highest force was recorded in each trial. Compensation procedures for gravitational errors in forces recorded in the vertical plane were undertaken immediately prior to testing.

Electromechanical delay

Electromyographic activity (EMG) was recorded from the m. biceps femoris during the estimation of PF_V and subsequent to supra-maximal stimulation. The EMG was recorded using bipolar surface electrodes (self-adhesive, silver-silver chloride, 10 mm diameter) that were applied longitudinally over the belly of m. biceps femoris, on the line between the ischial tuberosity and the lateral epicondyle of the femur. The m. biceps femoris was selected as an important contributor to restraint of anterior tibio-femoral displacement and lateral rotation of the femur relative to the tibia since both processes have been implicated in ACL injury (Fu et al. 1993).

The raw unfiltered EMG signals was passed through a differential amplifier, input impedance 10,000 MOhms , CMMR 100 dB, and a gain of 1000 (Cambridge Electronic Design,UK). The signal, which incorporated minimal intrusion from induced currents associated with external electrical and electromagnetic sources and noise inherent in the

remainder of the recording instrumentation, was analogue-to-digitally converted at 2.5 kHz sample rate, ensuring a significant margin of reserve between the highest frequency expected in the EMG signal and the Nyquist frequency and minimal intrusion from aliasing errors (Gleeson, 2001). The EMG signals remained unfiltered during subsequent analyses. The inter-electrode distance was 30 mm and a reference electrode was placed 30 mm lateral and equidistant from the recording electrodes. Standardised skin preparation techniques yielded inter-electrode impedance of less than 5 kΩ.

Volitional and magnetically-evoked EMD (EMD_V and EMD_E, respectively) were computed as the mean response of three intra-trial muscle activations in which the time delay between the onset of electrical activity and the onset of force was recorded. Post-fatigue EMD_E was estimated on the basis of performance in a single trial to minimise the potential intrusion of neuromuscular recovery on recorded scores. The superior reproducibility (coefficient of variation expressed as a percentage of the mean group score) and single measurement reliability (intra-class correlation coefficient) characteristics associated with EMD_E compared to the equivalent volitional estimates of performance have been described previously (8.1%; 0.84 vs. 10.1%; 0.80 for EMD_E and EMD_V, respectively) (Minshull et al. 2002b). The onset of electrical activity was defined as the first point in time at which the electrical signal exceeded consistently the 95% confidence limits of the isoelectric line associated with the background electrical noise amplitude and quiescent muscle, and which was the first deviation of the recorded electrical signal that was congruent with physiological activation of the muscle. 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 (see figures 2 and 3). 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 (see figures 2 and 3).

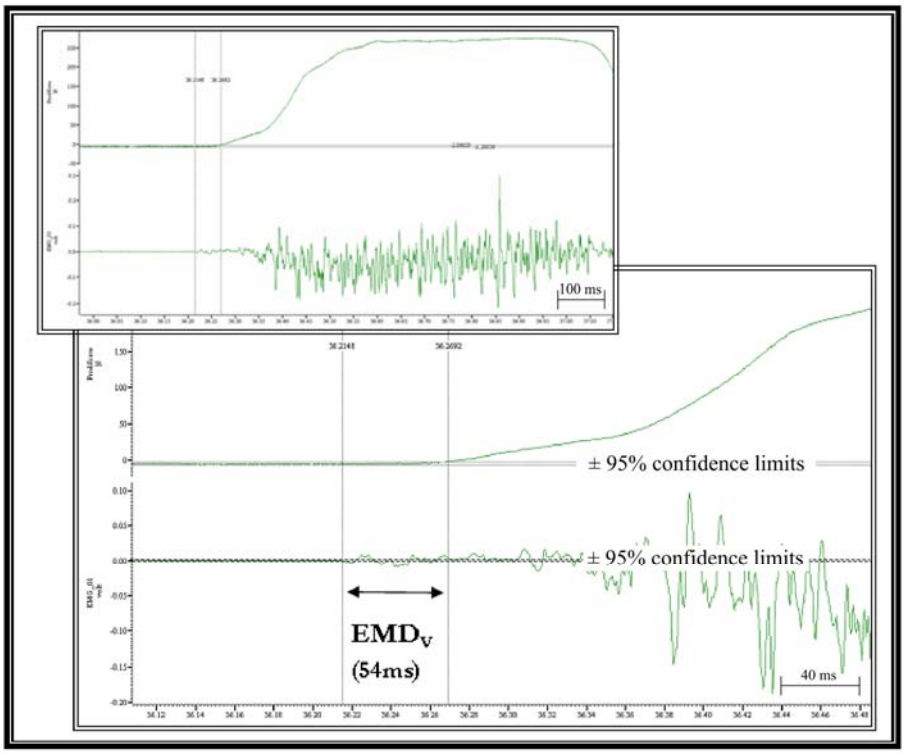

Figure 2. Example raw data showing: upper trace: example data of force and EMG associated with one MVMA; lower trace: magnification of muscle activation to show representative calculation of volitional electromechanical delay (EMD_V).

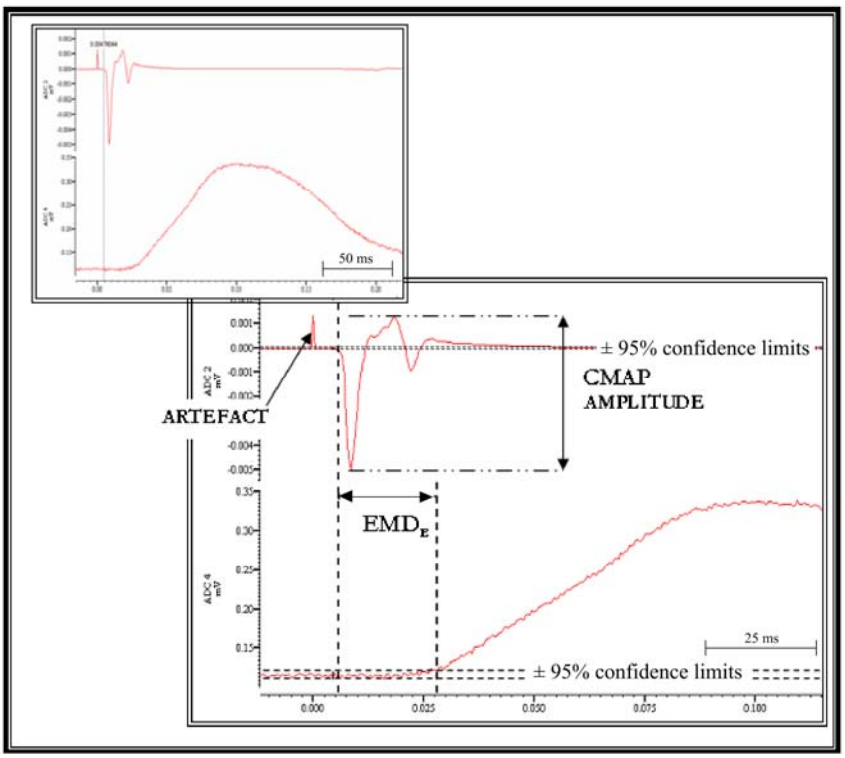

Figure 3. Example data showing; upper trace: example data of force and EMG associated with a single magnetic stimulus; lower trace: magnification of muscle activation to show representative calculation of magnetically-evoked electromechanical delay (EMD_E).

Statistical analysis

The effect of the fatiguing exercise intervention was assessed for each index of performance (PF_V; EMD_V; EMD_E) using separate two (condition: control; fatigue) by two (time: pre; post) by two (group: male; female) mixed-model ANOVAs with repeated measures on the first two factors. The assumptions underpinning the use of repeated measures ANOVA were checked and violations corrected by the Greenhouse-Geisser adjustment of the critical F-value, as indicated by $_{GG}$. Statistical significance was accepted at $p < 0.05$.

The experimental design offered an approximate .80 power of avoiding a Type-II error when employing a least detectable difference of 16 N, 8 ms and 3.5 ms for PF_V, EMD_V and EMD_E, respectively.

Results

Volitional muscle activation

Volitional peak force (PF_V)

A significant three-factor interaction showed that while absolute strength performance was preserved during the control task, the fatiguing exercise task elicited a reduction in absolute strength performance in both males and females ($F_{[1,14]} = 14.0$, $p < 0.05$). However, the absolute strength performance (group mean score (± SD)) was impaired to a greater extent in males than in females compared to baseline scores (265.1 (± 52.0) N vs. 311.8 (± 52.8) N [15% impairment] and 171.4 (± 33.9) N vs. 190.8 (± 48.6) N [10.2% impairment], respectively).

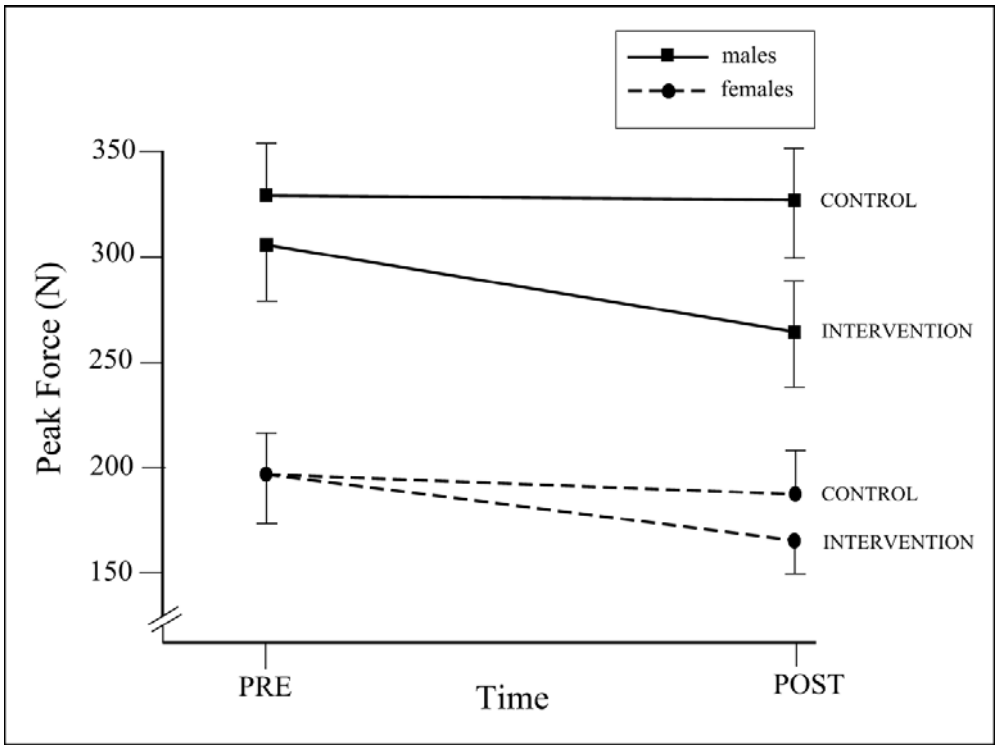

Figure 4. The effects of the fatigue task on the volitional peak force (PF_V) of the knee flexors (group mean ± SD).

Electromechanical delay (EMD_V)

A significant three-factor interaction ($F_{[1,14]} = 5.9$, $p < 0.05$) suggested that EMD_V performance was maintained during the control task for both groups and in the

experimental condition for males. However, the fatiguing exercise task elicited a 19.5% impairment in EMD_V performance compared to baseline levels in females (61.2 (± 19.0) ms vs. 51.2 (± 13.1) ms, respectively) (see figure 5). A-priori Helmert contrasts between group mean scores for males and females at baseline revealed no significant differences in EMD_V performance.

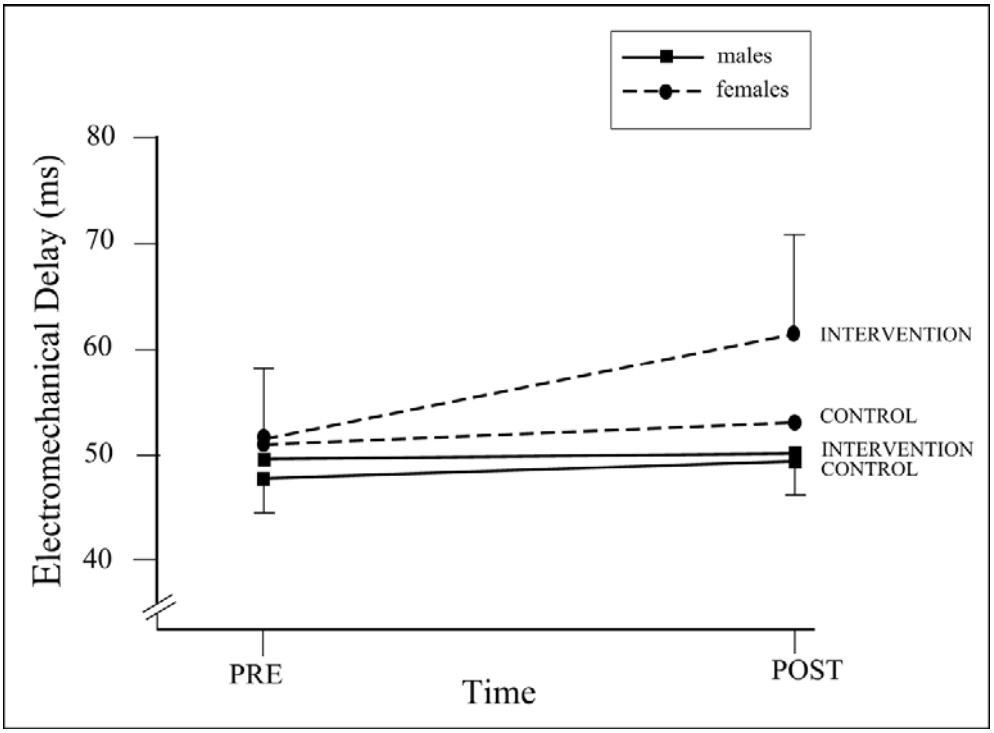

Figure 5. The effects of the fatigue task on the volitional electromechanical delay (EMD_V) of the knee flexors (group mean ± SD).

Magnetically-evoked muscle activation

Evoked electromechanical delay (EMD_E)

A significant two-factor interaction of condition (control; fatigue) by time (pre; post) on EMD_E showed that while absolute EMD_E performance was preserved during the control task, the fatiguing exercise task elicited a potentiation (21% decrease) in EMD_E latencies in both males and females ($F_{[1,14]} = 27.3$, $p < 0.001$) (see figure 6). A-priori Helmert contrasts between males and females at baseline revealed significantly shorter absolute EMD_E values in females compared to males ($F_{[1,14]} = 7.3$, $p < 0.05$)

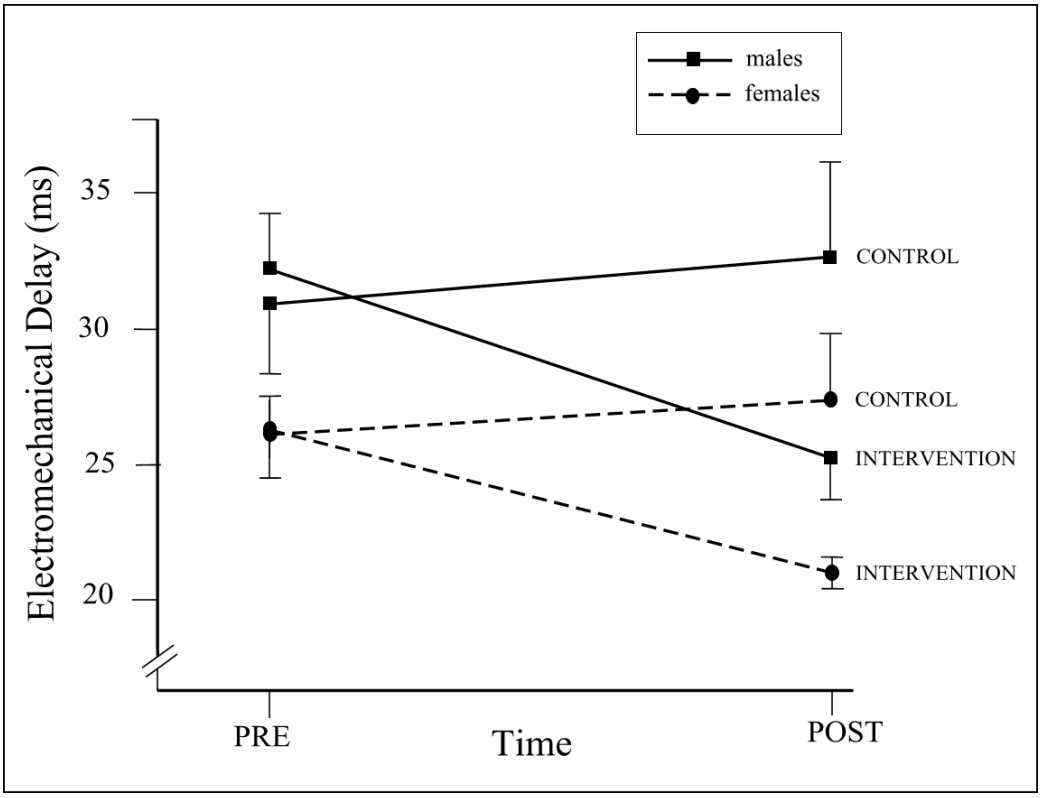

Figure 6. The effects of the fatigue task on the magnetically-evoked electromechanical delay (EMD_E) of the knee flexors (group mean ± SD).

Discussion

The absence of change over the control condition for each index of performance indicates that there were no systematic or learning effects and that performance variation can be attributed to the exercise intervention.

Volitional neuromuscular performance

The exercise intervention induced fatigue in the knee flexors, characterised by a significant decrease in PF_V from pre- to post-fatiguing exercise levels. The magnitude of PF_V performance decrement observed in the current study (15% for males and 10% for females) is congruent with the extent of performance loss associated with match play in team games such as soccer (Gleeson et al. 1998b). These findings, together with corroborating findings from other studies (e.g. Gleeson et al. 2000; Gleeson et al. 1998b; Zhou et al. 1996) may suggest a reduced capability of the dynamic stabilisers to provide forceful corrective responses to mechanical loading of the knee. Such fatigue-related changes in neuromuscular performance may be interpreted to represent an increased risk of injury (Chan et al. 2001; Gleeson et al. 1998b; Mercer et al. 1998), which may be amplified particularly at knee angles where key ligamentous structures are already under greatest mechanical strain (Beynnon and Johnson 1996).

Recent research has demonstrated that loading of viscoelastic structures in isolation can cause creep within the affected tissue and a modulation of the neuromuscular performance characteristics of the associated musculature (Chu et al. 2003; Sbriccoli et al. 2005; Solomonow, 2004; Solomnow et al. 2003). For example, cyclical loading (150-200N) of the anterior cruciate ligament has been associated with an

approximate 10% reduction in knee extensor peak force (Sbriccoli et al, 2005). Furthermore, outcomes of testing in animal models have reported increases in shear creep of up to 27% and 53% respectively, compared to baseline following ten minutes and thirty minutes of intermittent bouts of feline spinal flexion (Solomnow et al. 2003). Sporting pursuits involving cyclical loading of viscoelastic tissue may contribute to increased injury risk because compliance characteristics and reflexive muscular activity may be adversely affected (Solomonow, 2004). However, the magnitude of the loading applied cyclically on viscoelastic tissue within the present study was probably low. For example, the loading effect of gravity in the current study would have created a relatively small passive anterior shear force on the knee of approximately 10-15N. This force is likely to have been moderated further by the frequent periods of muscle activation performed by participants, shielding relevant tissue from mechanical stress. It is likely that the cyclical application of such forces will have contributed an effect to baseline performance by means of the duration of the standardised warm-up (5 minutes of cyclical loading) and a small effect to the experimental changes in EMD performance following the acute 30 second fatigue-task, plus time spent in static maximal voluntary muscle actions.

While the decrements to PF_V capabilities of males exceeded that experienced by females (PF_V: 15% vs. 10%, respectively), a group mean increase in EMD_V latencies from pre- to post-fatigue levels (19.3%) was observed exclusively in females. Recent research that has indicated that the reaction time of the neuromuscular system to imposed dynamic forces may be fundamental to the protection of the joint system (Gleeson et al. 2000; Gleeson et al. 1998a; Gleeson et al. 1998b; Huston and Wojtys 1996; Mercer et al. 1998; Shultz et al. 2001) may suggest such concomitant increases in EMD_V may affect the timely

correction of joint forces and be associated with exacerbation of injury risk. Indeed, the current results may provide a new insight into the complex phenomenon that describes a five- to seven-fold increase risk of ACL injury in the female athlete compared to male counterparts (Gray et al. 1985; Ireland et al. 1997).

The processes involved in the conversion of excitation into muscle force can potentially contribute to the fatigue-related changes in the force-generating capability observed in the current study. However, it has been proposed that the majority of the EMD is determined by the time taken to stretch the series elastic component (SEC) (Cavanagh and Komi 1979), most of which is situated at the connective tissue attachments at the end of the muscle fibres (McComas 1996). The differential changes in EMD_V performance between sexes in the current study could be partially explained by a generally greater compliance in biologic tissue in females (Wojtys et al. 1998), exacerbated by muscle temperature increases associated with the fatiguing exercise (Zhou et al. 1998). Given the many injury risk factors experienced by females, habituated exposure to scenarios where knee joint stability may be under threat might condition the neuromuscular system of the healthy female athlete at functional joint angles. The subsequent formation of pre-programmed responses that provide fast compensatory reactions to joint perturbations (Latash 1998) may quickly harness the SEC and account for the parity in EMD_V performance observed between the sexes at baseline. Under conditions of muscle fatigue and sustained loading, however, this capability may be diminished due to a reduction of the effectiveness of the fastest most powerful motor units, impairing the temporal capability of the muscle to ‘gather in’ a more compliant SEC.

Magnetically-evoked neuromuscular performance

Despite the fatiguing exercise intervention causing fatigue and impairment to indices of volitional neuromuscular performance, the ultimate temporal physiological capacity of the neuromuscular system (EMD_E), as measured by magnetic stimulation, was potentiated by similar amounts in both males and females.

Our understanding of aspects of the nature of fatigue may be challenged somewhat by the observed differences in fatigue-related changes to EMD_V and EMD_E. However, the apparent paradoxical coexistence of fatigue of volitional performance and potentiation of evoked performance has been documented previously subsequent to exercise. Improvements of electrically-evoked peak twitch force (Rassier and MacIntosh 2000) and EMD (Sahlin and Seger 1995) have been described following acute and prolonged exercise protocols, respectively. It is plausible that these changes facilitate a biological conservation of resources during energy-costly volitional exercise efforts, while simultaneously offering enhanced reflex and 'emergency' capabilities to resist mechanical threats to musculoskeletal integrity. While metabolically mediated increases in sensitivity of muscle contractile proteins to Ca^{2+} may represent the processes underlying potentiated muscle force characteristics (Rassier and MacIntosh 2000), exercise-related changes to the compliance characteristics of the musculoskeletal system may represent the principal potentiating processes in the present study. This may be particularly true considering that the major proportion of EMD is accounted for by lengthening of the SEC (Komi 1979; Zhou et al. 1995).

Connective tissue and muscle-tendon units subjected to a constant stress elongate over time (stress-relaxation), eliciting an increased length at a given load (Stone and

Karatzaferi 1992). Recently, this creep effect has been shown to elicit a 'disordering' of the neuromuscular reflex response and, coupled with the concomitant increase in connective tissue compliance and ligamentous laxity, has been interpreted as representing a major knee injury risk factor (Chu et al. 2003; Solomonow, 2004). In is interesting to note that results from the current investigation, show an improvement in magnetically-evoked EMD following fatigue. This shortening of evoked latency may suggest that the exercise-related stress and assessment characteristics elicited a decrease in compliance within the knee joint system. It is conceivable that the strong static activation of muscle induced reactive hyperemia (McComas 1996) and a potential distension of the muscle. These latter processes may have contributed substantially to the facilitated post-fatigue EMD_E when coupled with comparably reduced levels of muscle force that would be required to stretch stress-relaxed viscoelestic structures.

The implications of the potentiation of EMD_E performance may be commensurate with the potential to overcome the fatigue-related impairments of the volitional performance capabilities during critical times. The net result following acute volitional muscle fatigue may be a 'reserve capacity' of unused motor units that can be deployed during perceived threat to the joint system. The utility of this preserved emergency capacity to the individual athlete may be dependent entirely, however, on the down-regulation of these potential protective central and peripheral neuromuscular inhibitory mechanisms (Hopkins and Ingersoll 2000) that appear to limit access to the full capacity of large high threshold motor units under voluntary conditions (Tsuji and Nakamura 1988; Zhou et al. 1995). This inhibition may be exemplified by the longer latencies associated with EMD_V (e.g. 51.2 ms) compared to EMD_E (e.g. 27.0 ms) in this study. A further corollary of this interpretation suggests that methods of assessment of

performance capacity must be carefully considered, since utilisation of solely volitional means of assessment may predispose a gross underestimation of the true capacity of the neuromuscular system.

In summary, the substantive decrement to the force-generating capacity of the knee flexors in males and females following acute fatigue (10% and 15% decreases in PF_V, respectively) may demonstrate a reduced capability to provide adequate dynamic restraint in response to mechanical loading of the knee joint at functional joint angles. In addition, the significant increase of EMD_V in females following acute muscle fatigue (19%) may be congruent with a reduced temporal capability to harness stabilising or resistive forces at the knee and place the female sports performer at increased risk of injury compared to male counterparts. Potentiation of magnetically-evoked EMD following fatigue in both males and females may suggest a capability to overcome the effects of impaired voluntary neuromuscular performance. Yet, the efficacy of a preserved temporal performance capacity to avoid synovial joint injury may be dependent entirely on whether the neuromuscular recruitment strategies observed subsequent to magnetic stimulation can be replicated under non-evoked conditions. Ultimately, increased risk of injury is likely to reflect the complex interaction of several factors, some of which may include neuromuscular conditioning, susceptibility to fatigue and an ability to deploy the full motor unit capacity of the neuromuscular system at crucial times.

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