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INVESTIGATIONS INTO THE FATIGUE-RELATED REDUCTION IN TORQUE, SHORTENING VELOCITY, AND JOINT RANGE OF MOTION IN HUMANS

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INVESTIGATIONS INTO THE FATIGUE-RELATED REDUCTION IN TORQUE, SHORTENING VELOCITY, AND JOINT RANGE OF MOTION IN HUMANS

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ABSTRACT

Studies in humans and animals have derived much understanding of neuromuscular function from isometric (static) contractions. In comparison, fewer studies have evaluated dynamic contractions, which are relevant to everyday movements and activities of daily living. The primary purpose of this thesis was to investigate and compare the contributing factors to fatigue during different voluntary contraction tasks. The interpolated twitch technique is commonly used to assess voluntary activation, but with changes in muscle length, musculotendinous slackness can diminish the amplitude of electrically-evoked twitches used to calculate voluntary activation. This might result in erroneous measurements of voluntary activation. Chapter 2 describes an experiment in which at the short muscle lengths, when voluntary activation is 80% or lower, actual activation will be underestimated. Maximum voluntary isometric contraction (MVC) torque is often used to assess overall neuromuscular function, and any activity-induced decline in MVC torque is indicative of fatigue. However, a reduction in shortening velocity is also an important feature of fatigue. Results from Chapter 3 indicate that shortening velocity was an important and perhaps more sensitive measure of fatigue following both isometric and dynamic contraction tasks than MVC torque per se. These findings are further supported in Chapter 4, in which, following comparable repetitive shortening contraction tasks in two different muscles, shortening velocity was reduced to a greater extent at task failure but was restored more rapidly than MVC torque. Shortening contractions are also characterized by a fatigue-related reduction in joint range of motion (ROM) and it was suggested that the reduction in ROM might be due to length-dependent alterations in torque or contractile slowing with fatigue. Results

presented in Chapter 5, suggest that length-dependent alterations in torque or contractile slowing cannot explain the fatigue-related reduction in dorsiflexion ROM. Thus, in addition to fatigue-related reductions in torque, decreases in shortening velocity and joint range of motion are important indicators of a fatigue-induced impairment in muscle shortening capacity.

Keywords: isometric contraction, dynamic contraction, fatigue, voluntary activation, torque, shortening velocity, range of motion, contractile slowing, tibialis anterior, triceps brachii, soleus

CO-AUTHORSHIP

This thesis contains material from published manuscripts (Chapters 2 and 3). On all manuscripts, Arthur J. Cheng was the first author and Charles L. Rice was a co-author. Andrew W. Davidson was also a co-author for Chapter 5. All experimental data presented in this thesis were collected and interpreted by Arthur J. Cheng.

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GLOSSARY OF TERMS

Contractile Slowing – a reduction in muscle contraction and relaxation speed

Dynamic - a contraction mode that involves limb movement

Fatigue – any reduction in torque, angular velocity, or joint range of motion as a result of prolonged intermittent or sustained muscle contraction(s)

Isokinetic – the dynamic contraction mode in which joint angular velocity is held constant but the torque can vary

Isotonic – the dynamic contraction mode in which a load is held constant and the joint angular velocity at which the joint moves through the range of motion is variable

Isometric – the contraction mode in which there is no appreciable change in muscle length and joint angle does not change

M-wave – the electrical response of all muscle fibres in a given muscle or muscle group which is elicited via supramaximal transcutaneous electrical stimulation of the peripheral nerve

Post-activation Potentiation – phosphorylation of myosin light-chains after a muscle contraction that increases electrically-evoked muscle twitch force, and can increase torque and shortening velocity during a voluntary contraction

Power – the product of torque (N·m) and joint angular velocity (rad/s) expressed as Watts (W)

Range of Motion – the angular displacement of a limb around the joint fulcrum expressed in degrees (°)

Task Failure – the termination time-point of a fatiguing contraction task, defined as a percentage reduction in torque, angular velocity, or joint range of motion

Torque – the product of force (N) and moment arm length relative to the fulcrum (m) expressed in N·m

Velocity-dependent - the dynamic contraction mode in which a load is held constant and the angular velocity at which the joint moves through the range of motion is variable

Voluntary activation - the level of voluntary drive to the muscle during a contractile effort

CHAPTER 1

GENERAL INTRODUCTION AND PURPOSE

1.0 GENERAL INTRODUCTION

1.0.1 Task-Dependency

A great deal of our understanding about neuromuscular function in humans and animals has been gained from evaluating isometric (static) contractions, and many useful measures have been developed from isometric contractions to evaluate the central and peripheral contributions to voluntary torque (or force) generation and fatigue. Central contributions typically consist of the supraspinal, spinal, and axonal components involved in the ability to voluntarily activate muscle, whereas peripheral contributions refer to portions distal to the neuromuscular junction including the excitation-contraction coupling processes as well as contractile and non-contractile machinery of the musculotendinous system (1, 12, 14, 17, 24-26, 72, 77).

One of the most widely employed measures is the assessment of maximum voluntary isometric contraction (MVC) torque during isometric contraction tasks, because MVC torque provides a good indication of impaired neuromuscular function following fatigue induced by an isometric contraction task (14). Fatigue, which can be defined as any exercise-induced reduction in the ability of muscle to produce torque (2, 14, 77), is a valuable model to evaluate neuromuscular function because fatigue places stress on the system, allowing various factors in the pathway and their influence to be identified. Furthermore fatigue is detrimental to athletic performance, and is an important concern in occupational, clinical, and everyday situations because it will impair

the ability to perform any task involving muscle contraction. However, MVC as the sole measure of fatigue may be too simplistic, especially during tasks involving movement (2, 14, 77).

In comparison to isometric contractions, fatigue during dynamic contractions is necessarily more complex and in addition to torque loss, includes two other key components: fatigue-induced reductions in shortening velocity (2, 16), and reductions in joint range of motion (16, 41-43, 50, 73). Because power loss is the overall product of impairments in both torque and velocity, and work is reduced by both impairments in torque and joint displacement (i.e., range of motion), these factors contribute to a greater fatigue-induced decline in muscle power output and work generating capacity than torque alone. Largely because of the complexity involved in studying dynamic contractions however, the mechanisms responsible for the impairment in muscle shortening capacity remain poorly understood (2, 14). In addition to the measurement of MVC torque, investigations of shortening velocity and joint range of motion may provide insight into the mechanisms of fatigue during dynamic contractions, but no studies in humans have purposely investigated each of these concerns.

1.0.2. Voluntary Activation

Since the study by Merton in 1954 (56), the interpolated twitch technique has become a widely accepted method to elucidate the central contributions to voluntary torque production. The interpolated twitch technique is typically applied to MVCs to help discriminate whether central-mediated factors are responsible for changes in MVC torque, as a result of fatigue, training status, or from alterations in disease progression (26, 72). The interpolated twitch technique has been shown to be the best technique to

assess voluntary activation (75), although it has some limitations. For example, the interpolated twitch technique has been employed previously during isometric (10, 11, 13, 21, 28, 32, 45, 46, 74) and isokinetic contractions (6, 7, 27, 60), but the technique cannot be applied to velocity-dependent contractions. The technique is based on measurements of evoked torque, but when used during velocity-dependent contractions (i.e., in which the contraction load is essentially constant), there remain difficulties in interpreting changes in shortening velocity with the application of an interpolated stimulus (Cheng and Rice, unpublished observations). Instead, some studies have employed MVCs to enable the use of the interpolated twitch technique to assess voluntary activation following dynamic contraction tasks (16, 43, 62). In these studies, voluntary activation was assessed during an MVC performed at a single static joint angle. However, when using the interpolated twitch technique, the muscle length at which voluntary activation is assessed should be considered, because it may affect the calculation of voluntary activation. For example, when muscle length is changed, especially when it is shortened, slackness of the musculotendinous system might lead to methodological error when assessing voluntary activation (45, 52, 58). Previous studies have not comprehensively assessed the effect of muscle length on the calculation of voluntary activation. This is important because many tests of voluntary activation could be assessed at different joint angles (5, 9, 28, 45, 47, 58, 65, 74), and understanding these limitations may improve the validity of the technique. Because of the limitations of velocity-dependent tasks used in this thesis, understanding the length-dependent limitations in assessing voluntary activation during isometric contractions was an important first step.

1.0.3. Shortening Velocity

With the advent of commercially available isokinetic dynamometers for use as testing, training, or rehabilitation devices, many previous investigations of fatigue during dynamic contraction tasks have used these dynamometers to study isokinetic "constant-velocity" contractions (6, 7, 15, 27, 29, 33, 36, 38, 40, 49, 53, 60, 62). Joint angular velocity is kept constant during isokinetic contractions, but intuitively, it is clear that everyday movements are rarely performed at a constant velocity. Rather, human locomotion and other activities are characterized by fluctuations in muscle shortening (and lengthening) velocities (14). Because lengthening contractions can add other complex factors to fatigue due to muscle damage (76), I chose shortening contractions for all studies in my thesis centred on exploring velocity-dependent contractions and fatigue. Compared with studies of fatigue using isometric or isokinetic contractions, relatively few studies have evaluated fatigue in humans using velocity-dependent contractions (i.e., velocity is allowed to vary) (16, 38, 55, 63, 70, 73). The fatigue-related reduction in power is much greater than the comparable reduction in MVC torque (1, 3, 16, 38, 39), because power is impaired by both reductions in torque and shortening velocity. Muscle shortening involves a large amount of crossbridge cycling activity, and this consumes more myofibrillar ATP than isometric contractions (22, 35, 48, 59). With increased ATP utilization during repetitive shortening contractions of a fatiguing task, additional metabolites are produced and metabolite accumulation inhibits crossbridge kinetics. This reduces maximum unloaded shortening velocity in vitro (2). Thus, it appears that fatigue during dynamic contractions is greater than during isometric contractions, and that shortening velocity may be a more suitable parameter than MVC torque to evaluate fatigue following shortening contractions in humans.

An important difference between studies in animals and humans is that maximum unloaded shortening velocity cannot be measured directly in humans, because in-vivo muscle contractions are performed with at least some resistance supplied by the limbs to which these muscles are attached. Thus, loaded shortening velocity would be a more physiologically representative fatigue parameter to evaluate muscle function in humans. Also, loaded shortening velocity may be more greatly affected by fatigue than maximum unloaded shortening velocity because some metabolites such as H^+_i (44), and P_i (19) largely impair loaded shortening velocity.

Furthermore, many studies in animals that investigated fatigue following shortening contractions have been performed in artificial conditions and at non-physiological temperatures (4, 18, 67, 71, 78-80). More recently, temperature has been shown to dramatically influence muscle fatigue (19, 20, 44, 57, 68), and this may result in different findings in animals compared to humans.

In humans, voluntary activation failure and impairments in neuromuscular transmission have also been shown to contribute to the reduction in MVC torque during isometric contractions (26). However, the fatigue-related contribution of voluntary activation and neuromuscular transmission to fatigue-induced changes in dynamic contraction task performance remain unclear.

1.0.4. Fatigue in Different Muscles

Due to physiological or functional disparities in different muscles, intrinsic differences might result in muscle-specific fatigue responses during dynamic contraction tasks. Muscles differ in fatigue resistance and post-activation potentiation capabilities, both of which are coexisting properties that can potentially affect the amount and rate of

reduction in shortening velocity. For example, slow twitch fibres are fatigue-resistant whereas fast-twitch fibres are highly fatigable because the lower mitochondrial density of fast-twitch fibres places a greater reliance on anaerobic energy utilization (81). The fatigability of human muscles may thus depend on the relative proportion of slow and fast fibres amongst muscles or muscle groups. At the same time, post-activation potentiation, through the mechanism of myosin light-chain phosphorylation (69), is known to increase loaded shortening velocity (8). Post-activation potentiation is greater in fast- compared to slow-twitch fibres (69) and might help compensate to some extent for the greater fatigability of muscles with a predominant fast-twitch fibre composition. The coexisting influences of fatigue and post-activation potentiation could result in different fatigue profiles for the fatigue-related reduction in shortening velocity in specific muscles, but this has not been investigated previously.

A comparison of the triceps brachii and soleus muscle in humans may be a useful model to evaluate the influence of fatigue and post-activation potentiation on the fatigue profile of different muscles during velocity-dependent contraction tasks. These muscles represent opposite ends of the morphological and physiological spectrum. The soleus has the greatest slow-twitch fibre composition among human limb muscles, whereas the triceps brachii has the lowest slow-twitch fibre composition (31, 37). Indeed, the soleus is also more fatigue-resistant than the triceps brachii during isometric contraction tasks (31), and this may be a result of both its high fatigue-resistant slow-twitch fibre composition (31) and the fact that it is a chronically-trained antigravity muscle (30). The triceps brachii has a much greater post-activation potentiation capacity than the soleus, but it is likely to offset only to a modest extent any fatigue-induced reduction in

shortening velocity. Thus, I expected that the soleus would have a greater endurance capacity than the triceps brachii during velocity-dependent contraction tasks.

1.0.5. Joint Range of Motion

In addition to the decline in torque output and shortening velocity, a reduction in joint range of motion (ROM) is a feature of fatigue following velocity-dependent contraction tasks. The maintenance of joint ROM may have important occupational and functional implications for tasks that require human limbs to move over a distance, including lifting objects, and performing tasks of daily living such as walking and climbing stairs. Only a few studies have assessed joint ROM failure after fatiguing dynamic contraction tasks (16, 41-43, 50, 73); the fatigue-related mechanisms related to the reduction in ROM remain uncertain.

The ability of muscle to generate torque at different muscle lengths seems important because muscles always contract against some resistance in vivo (i.e., provided by the human limbs, body mass, mass of objects being carried), and limb movement occurs only when the muscles that act across this joint are able to shorten effectively against this resistance.

Whereas previous studies have suggested that the reduction in ROM during fatigue is related to length-dependent alterations in torque-generating capacity (43), in comparison, fatigue-related contractile slowing may be a more important fatigue parameter following dynamic contraction tasks (16, 50). Furthermore, one previous study speculated that ROM failure may also be related to more impaired contractile kinetics at the short compared to long muscle length (43).

Previous studies have largely compared isometric contraction tasks performed at the short and long muscle length to evaluate the influence of muscle length on fatigue (23, 34, 46, 51, 54, 61, 64, 66). However, because fatigue is task-dependent, it seems inappropriate to deduce findings from isometric contraction tasks performed at different muscle lengths to explain fatigue resulting from repetitive shortening contraction tasks. Consequently, investigations of length-dependent alterations in torque and contractile slowing following repetitive shortening contraction tasks may provide new knowledge of the fatigue-related factors involved in the reduction in ROM.

1.1. PURPOSE

Collectively, the purpose of this thesis is to extend our understanding of fatigue by using various proposed models involving both isometric and velocity-dependent contractions. Chapter 2 is a methodological study to determine whether using the interpolated twitch technique to calculate voluntary activation at different muscle lengths results in erroneous measurements of voluntary activation. This may help determine, for both isometric and dynamic contraction tasks, whether muscle length is an important factor when employing this technique. In Chapter 3, I investigate whether there are apparent differences in the fatigue-induced changes in MVC torque and shortening velocity following isometric and velocity-dependent contraction tasks. In comparison with the isometric contraction task, this study will help determine whether shortening velocity is reduced more following velocity-dependent contraction tasks, possibly due to greater fatigue-induced contractile slowing from the increased metabolic cost of muscle shortening (22, 35, 48, 59). In Chapter 4, I extend these ideas of fatigue-related factors during velocity-dependent contractions by comparing fatigue in two different human

muscles. Due to the known fatigue resistance of the soleus muscle during isometric contraction tasks, I expect that the soleus will also show a longer time to task failure compared with the highly potentiating triceps brachii during the velocity-dependent contraction-tasks. Finally, in Chapter 5, I examine whether the fatigue-induced reduction in ROM following repetitive shortening contractions is related to length-dependent impairments in torque and contractile slowing. As suggested in previous papers, I hypothesize that greater slowing of contractile kinetics and greater reductions in torque at the short versus the long muscle length will be related to the fatigue-induced reduction in ROM.

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CHAPTER 2

VOLUNTARY ACTIVATION IN THE TRICEPS BRACHII AT SHORT AND LONG MUSCLE LENGTHS

2.0 INTRODUCTION

The interpolated twitch technique (ITT) has been used extensively to evaluate voluntary activation in skeletal muscle (for review see 17, 34) and is at present the most direct method to assess the extent of voluntary drive to the muscle (22, 36). The ITT quantifies percent voluntary activation using the formula: (1- interpolated twitch on voluntary contraction / potentiated twitch evoked at rest) x 100, which compares as a ratio, an interpolated twitch evoked during a voluntary contraction, to the twitch evoked in a relaxed muscle following a maximum voluntary contraction (MVC) (i.e., post-MVC twitch) (1, 8). A limitation of voluntary activation calculated in this manner is that musculotendinous slack can diminish the amplitude of the post-MVC twitch (25, 27, 28). Unless the muscle is in a stretched position and made more rigid to reduce this slack, voluntary activation could be underestimated. In many studies, voluntary activation is not always tested at the longest muscle length, and in fact, some studies have compared voluntary activation at shortened and long muscle lengths (2, 4, 18, 25, 26, 28, 31, 35). The influence of this mechanical component needs to be further addressed because musculotendinous slack may play a role in the assessment of voluntary activation that is independent from changes in central drive.

Typically, the primary interest of studies is to assess voluntary activation during

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maximal efforts, and during these high-intensity efforts, previous research has shown that the ITT technique is not affected by substantial reductions in post-MVC twitch torque at shortened muscle lengths if maximal or near maximal voluntary activation is achieved (25, 28). This occurs because at high activation levels the interpolated twitch is very small or non-existent compared with the post-MVC twitch amplitude. However, during submaximal voluntary activation, the rather large interpolated twitch would be compared to the post-MVC twitch whose amplitude may be blunted mechanically, resulting in an underestimation of voluntary drive. This is important because submaximal voluntary activation may be expected in certain clinical or special populations (23, 32), due to fatigue-induced voluntary drive failure (17), or in particular muscle groups with known muscle length-dependent deficits in voluntary drive (11, 14, 26, 31).

To avoid potential issues of muscle slackness, many studies have used the relationship between interpolated twitches superimposed on voluntary torques of increasing contraction intensities to estimate, at 100% activation, the MVC torque of the muscle (5, 7, 12, 16, 18, 30, 32). The percentage difference between estimated and actual MVC torque is then used to provide a measure of voluntary activation without including the post-MVC twitch. However, a number of inherent factors can distort the voluntary and interpolated torque relationship at high torque (or force) levels including: the influence of muscle synergists, small muscle length changes, and inadvertent antagonist stimulation, all of which can lead to errors in the extrapolated MVC torque estimate (1, 17, 34, 36). In contrast, maximum voluntary activation in the quadriceps was calculated using the ITT in one study by extrapolating interpolated twitches obtained from three contraction intensities (40%, 60%, and 100% of MVC) to predict the post-MVC twitch

amplitude at rest (4). Although not the main point of the study, their results indicated a potential advantage of using extrapolation to predict the resting post-MVC twitch torque, which would be less affected by the inherent limitations related to estimating MVC torque (17, 34). Yet, the utility of this method of extrapolation to account for the potentially lower post-MVC twitch amplitude at shortened muscle lengths has not been evaluated systematically.

Thus, the aim of this study was to assess whether length-induced changes in post-MVC twitch amplitude cause changes in the calculation of voluntary activation in the elbow extensors. The comparison of voluntary activation values calculated using evoked versus predicted post-MVC twitch amplitudes (from extrapolation) will evaluate indirectly the influence of musculotendinous slack on voluntary activation, and assess the potential utility of this extrapolation technique. Compared to other muscle groups, voluntary activation of the elbow extensors has been studied infrequently (9, 13, 19, 24). The elbow extensors provide a useful model in assessing voluntary activation at short and long muscle lengths because it has no major synergists (38) to complicate predictions from extrapolation. In addition, previous studies in the triceps surae (2, 28) and quadriceps muscle (35) that assessed voluntary activation at short and long lengths used less suitable models because both muscles operate largely on the ascending portion of the torque-length relationship (4, 33, 35), whereas the elbow extensors have a relatively flat torque length-relationship (13, 15, 29) (see Appendix A). Using paired stimuli to minimize the influences of potentiation (21) and to maximize the sensitivity of twitch interpolation at submaximal contraction intensities (1), I hypothesized that the post-MVC doublet amplitude would be lower at short compared to long muscle lengths, which

would result in systematic error and underestimation of voluntary activation values at the short muscle length. Furthermore, accounting for the reduction in post-MVC doublet amplitude at shortened lengths using predicted post-MVC doublet torque values from extrapolation would more closely match voluntary activation values obtained at shortened as compared to those obtained at long muscle lengths.

2.1 METHODS

2.1.1 Subjects

Twelve recreationally-active young men [26.9 (4.3) y, 179.9 (6.9) cm, 77.8 (8.2 kg)] were recruited for this study. All subjects were asked to refrain from exercising the upper body at least 48 h before the testing session, nor consume caffeine on the day of testing. Informed written consent was obtained from all subjects. The University of Western Ontario Human Research Ethics Board approved the study and this experiment was performed in accordance with the Declaration of Helsinki of 1975.

2.1.2 Testing Overview

The single testing session consisted of isometric submaximal contractions and MVCs of the elbow extensors performed at short (20° of elbow flexion) and long (120° of elbow flexion) muscle lengths. The testing order of the joint angles was randomized, and at each joint angle, voluntary contractions were completed first at 100% of elbow extension MVC and then in a randomized order at 5%, 10%, 20%, 40%, 60%, and 80% of elbow extension MVC torque.

2.1.3 Experimental Setup

All testing was performed on a Biodex System 3 multi-joint dynamometer (Shirley, New York, USA), which provided measures of elbow extension torque and joint

position. Subjects were secured in an upright chair with a hip angle of 85°. The right arm was rotated externally 40° from the sagittal plane, and the arm was abducted approximately 40° from the torso. In order to minimize extraneous movement of the shoulder, body, and arm, two inelastic straps fixed the right shoulder, one strap was placed tightly around the mid-torso, one strap secured both thighs, and one strap secured the arm to an elbow rest. Each subjects' forearms were placed in a neutral position and a custom designed aluminum plate allowed a strap to secure the wrist to the arm attachment of the dynamometer. The rotation of the dynamometer was aligned with the lateral epicondyle of the humerus.

2.1.4 Experimental Procedures

Two aluminum foil electrode pads (4.5 x 5 to 4.5 x 8 cm in size) coated with conductive gel were customized to each subject to ensure stimulation of a significant portion of the triceps brachii without interference from the antagonists. These stimulation electrodes were placed transversely over the muscle belly of the triceps brachii to electrically evoke doublets (50μs pulse width, 10ms interpulse interval) using a Digitimer stimulator (DS7AH, Digitimer Ltd, Welwyn Garden City, Hertfordshire, UK). The cathode was centered over the proximal section of the triceps brachii ~ 1 cm distal to the posterior deltoid muscle and the anode was centered midway between the distal border of the deltoid muscle to the olecranon. During pilot testing, this placement gave the greatest amplitude for doublets evoked in a relaxed muscle. A ground electrode was placed on the acromion of the right scapula. To ensure that stimulation was primarily to the triceps brachii muscle, electrically evoked doublets were incrementally increased in stimulation intensity at the start of testing at either the short or long length, until doublets

reached a plateau in peak torque. If doublet torque decreased with an increase in stimulation intensity, the electrode pads were repositioned or a different size of electrode was used until a plateau in doublet torque was achieved with increasing stimulation intensity. Stimulation intensity (300-480 mA) was then set at ~10% above that needed to achieve peak doublet torque. After 5 min of rest, subjects performed 5 s duration MVCs (with 3 min rest between MVCs) to determine 100% MVC torque. If MVC torque for the first two MVCs varied by more than 5%, then up to two additional MVCs were performed. Subjects were provided with visual torque feedback on a computer display monitor and were verbally encouraged during all maximal efforts. An interpolated doublet was evoked during each MVC upon reaching a plateau in peak MVC torque. A doublet was also evoked in the potentiated but relaxed muscle ~ 3 s after each MVC (i.e., the actual post-MVC doublet). Five minutes after the MVCs, subjects were asked to target torque output on a computer monitor for 3-5 s at several contraction intensities (5, 10, 20, 40, 60, and 80% MVC) in a randomized order. Each targeting contraction was repeated at least two times to obtain average voluntary/interpolated torque values; sometimes the higher contraction intensities (60% and 80%) required up to four repetitions to target the contraction intensity accurately. To prevent fatigue, extra rest was provided as necessary. After 10 min of rest, the measurements were repeated at the other joint angle.

All torque data obtained from the Biodex at a 100 Hz resolution were converted by a 12-bit A/D converter (CED Model 1401 Plus; Science Park, Cambridge, UK). Spike2 computer software (CED, Science Park, Cambridge, UK) permitted real-time display and off-line analysis of torque on a computer monitor. The interpolated doublet

torque, MVC torque, and post-MVC doublet torque values were measured from the MVC with the highest level of maximal voluntary activation. At the submaximal contraction intensities, mean interpolated doublet torque and mean voluntary torque values were obtained by averaging the two contractions that most closely targeted the appropriate torque level. At each contraction intensity, interpolated doublet torque amplitude was calculated from the first pulse of the doublet stimuli to peak doublet torque, and voluntary torque was recorded as torque output coinciding with the first pulse of the doublet. As the interpolated and post-MVC doublet torque amplitudes would depend on the torque-length relationship of the muscle, both interpolated doublet and post-MVC doublet torque were normalized to peak MVC torque (28). Percentage voluntary activation was quantified using the formula: (1-interpolated doublet torque / post-MVC doublet torque) x 100. For each subject, linear and non-linear (second-order polynomial) regression equations were formed from extrapolation of actual voluntary/ interpolated torque values to more accurately identify the predicted post-MVC doublet amplitude. As performed previously by others (4), post-MVC doublet amplitudes, as well as voluntary activation, were estimated by linear extrapolation of values from 40-100% of MVC to account for the potential reduction in the post-MVC doublet amplitude with changes in muscle length. Additionally, I performed both linear and non-linear extrapolation of actual voluntary/interpolated torque values between 40-100% of MVC to compare the y-intercept value (i.e., predicted post-MVC doublet torque amplitude), and therefore the ability of each method of extrapolation to account for the potential reduction in post-MVC doublet amplitude at short lengths in the elbow extensors. Although the interpolated torques obtained at 5, 10, and 20% of MVC were not used for extrapolation,

these interpolation values were used to calculate actual and predicted submaximal voluntary activation values across a broad range of contraction intensities between 5-80% of MVC.

2.1.5 Data and Statistical Analyses

The statistical significance of differences between muscle lengths across all contraction intensities (5-100% of MVC) in normalized evoked doublet torque, and voluntary activation, were determined using a Friedman's test as the data were not normally distributed. Friedman's tests were used to perform pairwise comparisons of voluntary activation calculated with the actual post-MVC doublet versus voluntary activation predicted from linear or non-linear extrapolation from contraction intensities between 5 and 100% of MVC. Statistical significance of differences between normalized evoked doublet torque and voluntary activation at each contraction intensity, were determined using Mann-Whitney U tests. Paired t-tests were performed to compare MVC torque, the actual versus predicted post-MVC doublet torque amplitudes, and also to compare the correlations obtained from linear and non-linear regression equations. Statistical significance was defined as $p < 0.05$. All tabulated data are presented as Mean (SD) and for graphical clarity, all figures are presented as Mean (SEM).

2.2 RESULTS

2.2.1 Torque, Voluntary Activation Calculated Using the Actual Post-MVC Doublet, and Correlations for Regression Analyses

MVC torque was not significantly different at short [56.9 (12.5) N·m and long [51.8 (9.6) N·m] muscle lengths, but normalized post-MVC doublet torque amplitudes (i.e., 0% of

MVC, Fig. 2.1a) were significantly reduced at the short [19.5 (9.1)%] versus long length [28.6 (7.2)%]. Subjects were able to match the submaximal (5%, 10%, 20%, 40%, 60%, 80%) target values well. At the short length the percentage values were: 5.0 (0.9)%, 10.1 (1.0)%, 20.4 (1.0)%, 41.2 (2.1)%, 60.8 (2.5)%, 80.5 (4.4)%, respectively, and at the long length, they were 5.5 (0.6)%, 10.8 (0.7)%, 21.0 (0.9)%, 41.2 (1.0)%, 60.5 (3.0)%, 81.1 (1.5)%, respectively. In addition, there were no length-dependent differences in normalized interpolated doublet torque across most contraction intensities, except for the significantly lower normalized interpolated doublet torque at 5% and 20% of MVC at the short compared to long length (Fig. 2.1a). Voluntary activation calculated using the actual post-MVC doublet amplitude was significantly lower at the short length at 10% and 20% of MVC, but there were no significant length-dependent differences at the other contraction intensities (Fig. 2.1b). Furthermore, voluntary activation was mostly incomplete at 100% MVC at both the short [93.7 (5.3)%] and long muscle length [94.6 (3.5)%], with the elbow extensors only capable of maximal activation in ~ 4% of all trials (i.e., 2 of 36 trials at the short length and 1 of 37 trials at the long length).

Second-order polynomial regression equations provided the best fit of the data compared to linear regression equations performed for each subject. Mean correlation values ranged from $R^2 = 0.99$ (0.01) at the short length to $R^2 = 0.99$ (0.01) at the long length, which were significantly better than correlations for linear extrapolation of $R^2 = 0.92$ (0.04) at the short length and $R^2 = 0.95$ (0.04) at the long length.

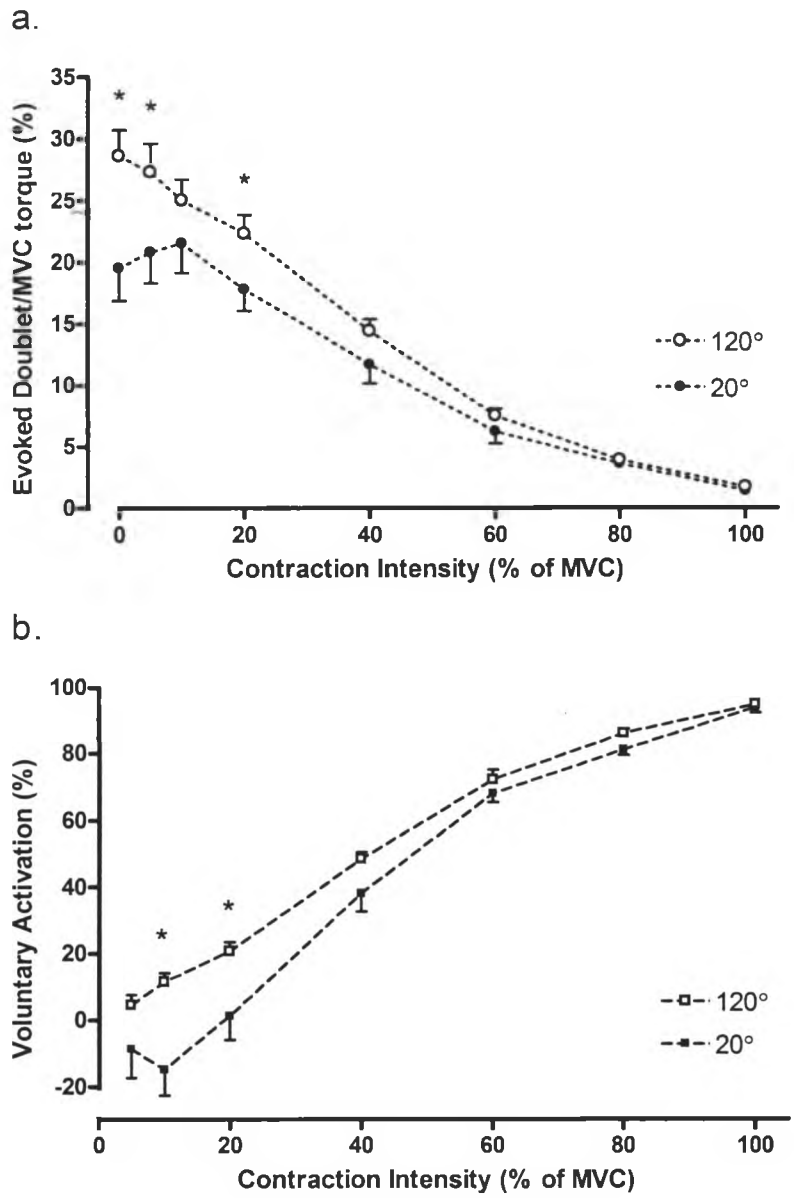


Figure 2.1 **a.** Normalized voluntary torque and normalized evoked doublet torque relationship at short (20˚) and long (120˚) muscle lengths. **b.** Voluntary activation calculated using the actual post-MVC doublet at the short (20˚) and long (120˚) muscle length. Figures are displayed as mean (SEM). * indicates significant difference between muscle lengths in values at the same contraction intensity.

2.2.2 Linear Extrapolation

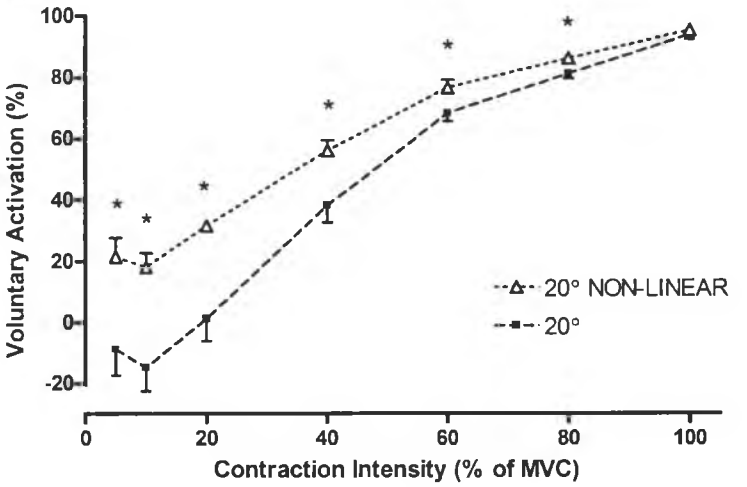
At the short length, the normalized post-MVC doublet amplitude predicted from linear extrapolation [20.7 (7.4)%] was significantly different to the actual post-MVC doublet amplitude [19.5 (9.1)%]. At the long length, normalized post-MVC doublet amplitude predicted from linear extrapolation [25.8 (5.8)%] was significantly lower than the actual post-MVC doublet amplitude at the same length [28.6 (7.2)%]. Thus, voluntary activation values estimated from linear extrapolation at the short length at all contraction intensities were not significantly different to voluntary activation calculated using the actual post-MVC doublet. Voluntary activation values at the long length were not significantly different at most contraction intensities to voluntary activation calculated using the actual post-MVC doublet, except a [7.8 (11.9)%] smaller voluntary activation value predicted at 10% of MVC.

2.2.3 Non-Linear Extrapolation

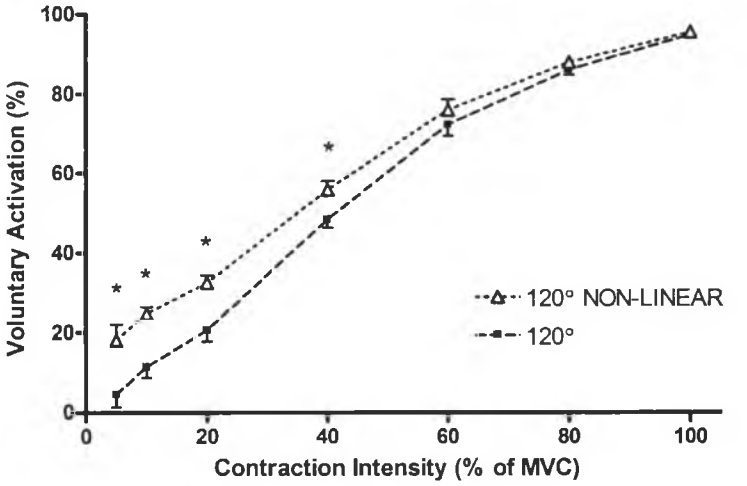
From non-linear extrapolation, the normalized post-MVC doublet amplitudes predicted at the short length [26.2 (9.3)%] and long length [33.6 (8.8)%] were significantly greater than the actual post-MVC doublet amplitude at both muscle lengths. In comparison to voluntary activation calculated using the actual post-MVC doublet, the second-order polynomial regression equations gave significantly higher estimated voluntary activation values between 5 and 80% of MVC at the short length, ranging from an increase in estimated voluntary activation by 32.9 (34.7)% (at 10% of MVC) up to 5.1 (5.8)% (at 80% of MVC) (Fig. 2.2a). At the long length (Fig. 2.2b), second-order polynomial regression equations gave significantly higher estimated voluntary activation values but only at contraction intensities between 5% and 40% of MVC, from an increase in

Figure 2.2 **a.** Voluntary activation at the short muscle length (20°) using actual versus predicted values from non-linear extrapolation. **b.** Voluntary activation at the long muscle length (120°) using actual versus predicted values from non-linear extrapolation. **c.** Relative to MVC torque, percentage voluntary activation values are similar at the short (20°) and long (120°) muscle length calculated using predicted post-MVC doublet torque values obtained from non-linear extrapolation. Figures are displayed as mean (SEM). * indicates significant difference between muscle lengths in values at the same contraction intensity, $p < 0.05$.

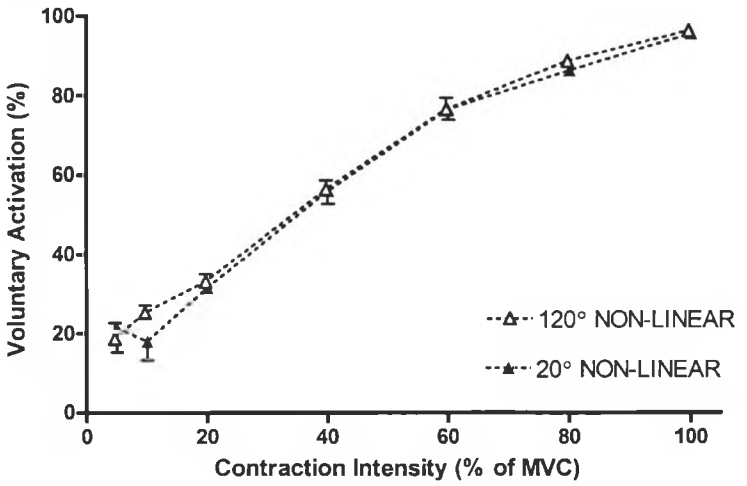
a.


Voluntary Activation (%)
100
80
60
40
20
0
-20
0
20
40
60
80
100
Contraction Intensity (% of MVC)
20° NON-LINEAR
20°

b.


Voluntary Activation (%)
100
80
60
40
20
0
0
20
40
60
80
100
Contraction Intensity (% of MVC)
120° NON-LINEAR
120°

c.


Voluntary Activation (%)
100
80
60
40
20
0
0
20
40
60
80
100
Contraction Intensity (% of MVC)
120° NON-LINEAR
20° NON-LINEAR

estimated voluntary activation values by 13.6 (10.8)% (at 5% of MVC) to 7.4 (5.5)% (at 40% of MVC). Voluntary activation values predicted using non-linear extrapolations at both lengths were not significantly different at all contraction intensities from 5% to 100% of MVC (Fig. 2.2c). Thus, after accounting for the reduction in post-MVC doublet torque at short lengths, voluntary activation was clearly not muscle length-dependent in the elbow extensors.

2.3 DISCUSSION

The purpose of this study was to evaluate comprehensively the voluntary activation of the elbow extensors at short (20°) and long (120°) muscle lengths. To address the reduction in post-MVC doublet amplitude at the short muscle length, the post-MVC doublet was predicted using either linear or non-linear extrapolation of voluntary and interpolated torque values. In comparison to voluntary activation values calculated using the actual post-MVC doublet, the predicted post-MVC doublet torque obtained from non-linear extrapolation at the short length led to significantly higher estimated values of voluntary drive at all submaximal contraction intensities (Fig. 2.2a). In contrast, the predicted post-MVC doublet amplitudes obtained from linear extrapolation were similar at the short length, but were smaller at the long length than the actual post-MVC doublet amplitudes; the linear extrapolation did not adequately account for changes in post-MVC doublet amplitude with alterations in muscle length. Thus, the smaller (actual) post-MVC doublet amplitude at short lengths played a role in underestimating the extent of voluntary activation at the short length, and

non-linear extrapolation at the short length was useful in accounting for the reduction in the actual post-MVC doublet amplitude.

2.3.1 Torque and Voluntary Activation During Maximal Efforts

The average elbow extensor MVC torques at 20° and 120° in my study were similar to those reported previously at a 90° joint angle (20), which is in line with the finding that the elbow extensors operate on the plateau region of its torque-length relationship (13, 15, 29) (see Appendix A). Also, the mean voluntary activation of ~ 94% during MVCs in my study, were similar to other studies at the 90° joint angle in this muscle group (9, 24), which suggests that activation is high but incomplete at all lengths in the elbow extensors. The high but incomplete voluntary activation during maximal efforts is not likely explained by the timing of the evoked interpolated doublet because the actual MVC torque immediately preceding the delivery of the interpolated doublet was similar to peak MVC torque at 20° [56.9 (12.5) N·m vs. 58.3 (12.9) N·m, respectively] and 120° [51.8 (9.6) N·m vs. 53.6 (10.1) N·m, respectively].

No length-dependent change in maximal voluntary activation was observed in the one previous study in the elbow extensors with four subjects (13). In my study, I confirm and extend these findings in twelve subjects that maximal voluntary drive was not length-dependent in the elbow extensors. These results also support previous findings from other muscles (25, 28) that during maximal efforts, if near maximal voluntary activation can be achieved (> 94%), any reduction in post-MVC doublet amplitude at shortened lengths does not affect the ITT calculation of voluntary activation due to the diminutive or non-existent superimposed twitch amplitude evoked during the maximal contractions. Near maximal voluntary activation at both short and long lengths have also been reported

for the quadriceps, abductor digiti minimi, tibalis anterior, and triceps surae (2, 3, 10, 18, 25) but activation in most of these studies was tested within a narrow range of muscle length changes (2, 3, 10, 18). In contrast, others have shown variable results in activation deficits at more extreme lengths (both short and long) that seem to be muscle and age-dependent (4, 25, 26, 28, 31, 35). The discrepancy among these studies was likely due to methodological differences in calculating or estimating the level of voluntary activation, or due to potential physiological factors such as greater difficulty in maximally activating the muscle when it is slack (31) or from influences of spinal reflexes (4, 26, 28, 35) when testing voluntary activation at very short or very long lengths.

2.3.2 Voluntary Activation During Submaximal Contractions

Studies on voluntary activation have not tested systematically whether "muscle slackness" observed at short muscle lengths leads to an underestimation of voluntary activation calculated using the ITT. Even small reductions in voluntary activation actually may represent large deficits in excitation of a motoneuron pool (22) and thus I assessed the methodological error in calculating voluntary activation at short lengths. In addition, I evaluated the utility of using linear extrapolation as well as non-linear extrapolation to account for the reduction in post-MVC twitch amplitude at shortened lengths. I showed that using either the actual post-MVC doublet or the predicted doublet values obtained from linear extrapolation led to invalid voluntary activation values. This is revealed by the non-physiological negative voluntary activation values calculated for contraction intensities of less than 20% of MVC (Fig. 2.1b and 2.2a). Instead, my findings show that non-linear extrapolation of voluntary and interpolated torque values was effective because 1) the smaller post-MVC doublet at short lengths, and thus

"slackness", resulted in an underestimation of voluntary activation when contraction levels were 80% of MVC or lower (Fig. 2.2a), and 2) using the predicted post-MVC doublets from non-linear extrapolation at both the short and long lengths resulted in similar voluntary activation-torque relationships (Fig. 2.2c). These findings illustrate that predictions from non-linear extrapolation accounted for changes in post-MVC doublet that could otherwise lead to an erroneous underestimation of voluntary activation (principally at the short length). Using non-linear extrapolation at the long length (120°) also resulted in a slightly larger predicted versus actual post-MVC doublet amplitude. Some subjects were capable of achieving comfortably greater than 120° of elbow flexion whereas in others the biceps brachii muscle mass or joint stiffness restricted range of motion to 120°. Choosing 120° allowed all subjects to be tested but it should be recognized that for some subjects 120° of flexion was not maximal and thus musculotendinous slack might have remained (Fig. 2.2b).

Although it is unclear whether linear (1, 17, 27, 30) or non-linear extrapolation (5-7, 10, 16, 37) better represents the true voluntary/interpolated torque relationship of any muscle group, it is clear that each provides different results for the elbow extensors. Non-linear regression more closely matched the curvilinear voluntary/interpolated torque relationship of the elbow extensors than linear regression, and was more useful in accounting for the decrease in post-MVC doublet torque at the short compared to long length. Activation of muscle synergists may contribute to a small extent to the non-linearity of the voluntary/interpolated torque relationship at submaximal contraction intensities (1, 37), as there are no major synergists other than the anconeus muscle, which contributes at most ~15% of elbow extension torque (38). Furthermore, it should be

noted that a limitation of any study testing voluntary activation of the elbow extensors is the difficulty in fixing completely the shoulder joint, which might allow for small muscle length changes (1) to create some of the non-linearity of the voluntary and interpolated torque relationship of the elbow extensors at both short and long lengths. In comparison, for the quadriceps others found that linear regression accurately represented the voluntary and interpolated torque relationship above 25% of MVC (30). Thus, the method of extrapolation should consider the muscle group tested and factors that could influence the shape of its voluntary torque and interpolated torque relationship.

In healthy subjects who are capable of near maximal activation, the ITT using the actual post-MVC doublet was sufficient to test maximal voluntary activation in the elbow extensors at any length. However, the muscle length at which voluntary activation is assessed may be more important when testing voluntary activation in a population who have diminished, or submaximal voluntary drive to their muscles (23, 32) or in other muscle groups in which voluntary drive in healthy individuals is found to be less than ~ 80% at the short length (4, 26). From a clinical or rehabilitation perspective, a consequence of underestimating voluntary activation is that potentially useful interventions to enhance voluntary drive to muscles might be masked if changes in musculotendinous stiffness are not recognized. As recently suggested by Taylor and colleagues (36), I also emphasize that the ITT measure is best used as a general indicator of voluntary drive, and should be used cautiously to quantify an exact level of voluntary activation. This is because the relationship between voluntary activation and contraction intensity is not necessarily linear, leading to differences between calculated voluntary activation values and voluntary contraction intensity (Fig. 2.2c). In addition, fatiguing

contractions may reduce voluntary drive and at the same time diminish the amplitude of an evoked twitch, each potentially minimizing the utility of the post-MVC response in the calculation of voluntary activation. In general, my findings in this study highlight that interpretations must be made carefully as voluntary activation values calculated using the ITT could be affected by changes in the post-MVC stimuli amplitude.

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CHAPTER 3

ISOMETRIC TORQUE AND SHORTENING VELOCITY FOLLOWING FATIGUE AND RECOVERY OF DIFFERENT VOLUNTARY TASKS IN THE DORSIFLEXORS

3.0 INTRODUCTION

Fatigue is often defined as a decrease in maximum voluntary contraction (MVC) force (or torque) after an exhaustive contraction task (3, 16, 28, 37). Multiple factors have been implicated for the decrease in MVC torque (9, 45), including decreased voluntary drive to the muscle from spinal and supraspinal centres (i.e., central fatigue), failure at the neuromuscular junction and from impairments in excitation-contraction coupling within the muscle (i.e., peripheral fatigue). Many studies have used the reduction in MVC torque to quantify the overall extent of fatigue of the neuromuscular system, but it is not clear whether torque per se is the most appropriate parameter to assess or compare fatigue during various tasks. For instance, fatigue of dynamic contractions is due to decreases in both torque and velocity (12, 26, 27, 34, 38, 42), which together lead to a greater loss in power compared with torque alone (12, 25-27). The different time courses for the fatigue-induced decrease in maximum isometric torque and maximum shortening velocity from stimulated contractions suggest there are different underlying mechanisms (2, 27, 47). Based on these findings, power loss is only partially explained by reductions in torque, and thus, assessments of shortening velocity in

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addition to torque-generating capacity may be useful in identifying the relative contributions of factors contributing exclusively to fatigue induced under dynamic rather than isometric tasks, but this has not been tested previously.

Only a few studies have directly compared isometric and dynamic (shortening) contractions in humans, and the findings of either similar (22, 28, 37), greater (33) or lesser fatigue following isometric compared to dynamic contractions (22, 37) are inconsistent. Differences among studies may be due to variations in the fatigue task (i.e., differences in contraction load, speed, isokinetic or isotonic-like contractions, electrically evoked or voluntary contractions) or the primary outcome measures used to quantify fatigue (i.e., peak torque, electrically evoked torque, surface electromyography (EMG) amplitude or voluntary activation assessed from the interpolated twitch technique). One of these studies compared maximal isometric sustained contractions to isokinetic maximal effort knee extensions at 90°/s, and observed a greater and earlier presence of peripheral fatigue (i.e., the decrease in electrically evoked doublet amplitude) during the dynamic task (3). One inherent limitation in this study and others (28, 33) was that they compared sustained isometric contractions, in which voluntary activation failure (8) and blood flow occlusion (23, 41) are known to exacerbate fatigue, with intermittent contractions which by their nature allow blood to flow during rest periods between repetitions. Despite its limitations, the study by Babault and colleagues (3) provided an interesting model for comparison of isometric and dynamic contractions and suggested that fatigue processes were different, even after matching for reductions in MVC torque.

Considering the known greater metabolic cost of shortening contractions compared with isometric contractions (18, 24, 31, 37), and bearing in mind the greater

influence of fatigue on decrements in power than isometric MVC torque (12, 25-27), it might be expected that even after matching for MVC torque loss, velocity would decrease to a greater extent following dynamic (velocity-dependent) contractions as compared with intermittent isometric (static) contractions. Limitations of previous studies that compared isometric to dynamic contractions (3, 22, 28), include using the constant velocity (isokinetic) technique that does not allow for the assessment of fatigue-induced changes in shortening velocity, not matching duty cycles for intermittent contractions, nor controlling for blood perfusion.

Thus, the present study was designed to compare the relative influence of various fatigue-related factors involved in isometric and dynamic task failure following an equivalent decrease in isometric MVC torque. I employed surface EMG and electrical stimulation to comprehensively assess possible changes in voluntary activation, motor unit activation, neuromuscular transmission, and contractile function during voluntary contractions. Under these conditions, it was hypothesized that despite a similar decrease in isometric MVC torque, a greater decrease in shortening velocity and greater slowing in electrically evoked contractile properties would be observed following dynamic compared to intermittent isometric contractions.

3.1 METHODS

3.1.1. Subjects

Nine recreationally-active young men [26.1 (2.4) y, 174.6 (6.4) cm, 79.7 (6.7) kg] were recruited for this study. Subjects were instructed to refrain from caffeine consumption and physical activity for at least 24 h prior to testing. Informed written

consent was received from all subjects and the local Research Ethics Board for the Review of Health Sciences Research Involving Human Subjects approved the study. The study was performed according to the Declaration of Helsinki.

3.1.2 Testing Overview

Subjects performed one familiarization session and two testing sessions with each session separated by at least 48 h. The familiarization session included electrically evoked twitches and tetani as well as sequences of voluntary isometric and dynamic practice contractions. The first testing session began with the assessment of baseline (PRE) electrical and voluntary contractile measurements and was succeeded by a dynamic fatiguing task. The second testing session also began with the assessment of PRE electrical and voluntary contractile measures but was followed by an isometric fatiguing task. The dynamic task session was always performed before the isometric task session in order to establish the same relative decrease in torque among all subjects during the subsequent isometric task (refer to section 3.1.4 below).

3.1.3 Experimental Setup

A Biodex System 3 dynamometer (Biodex Medical Systems, Shirley, NY, USA) was used to test dorsiflexion torque and velocity. The isometric mode of the Biodex was utilized during the assessment of MVC, percent voluntary activation (VA), electrically evoked peak twitch torque, and peak 50 Hz torque as well as maximum relaxation rate (MRR) of the 50 Hz contractions. The isotonic mode was used to perform the fatiguing dynamic contractions and allowed for the assessment of changes in shortening velocity throughout the experiment. Subjects sat in a reclined position with the hip at ~ 70°, knee at ~ 20° (relative to full knee extension) with the lower leg

aligned parallel to the ground. A velcro strap was placed across the right thigh, and adjustable belts across the waist and chest minimized hip as well as upper body movement. Velcro straps across the toes and dorsum of the foot secured the limb to the dynamometer footplate. Only the right leg was tested because this was the dominant limb for all subjects. Isometric dorsiflexion contractions were performed at a plantar flexed position of 25° because this angle in previous studies was close to peak isometric torque in young men (32, 44). Also, when stimulating the common peroneal nerve, the influence of the peroneal muscles on the evoked contractions of the dorsiflexors is reduced by using a more plantar flexed position (32, 44). The dynamic dorsiflexion contractions began at 25° of plantar flexion and ended at a neutral ankle position (0°), such that the total joint range of motion was 25°. This range of motion represents the mid-range of the length-tension relationship of the dorsiflexors (32, 44).

To examine changes in neuromuscular transmission, M-wave amplitude was assessed using surface EMG. Monopolar EMG was recorded using self-adhering electrodes (3 x 2 cm; Kendall-LTP, Chicopee, Massachusetts) after abrading the skin and cleaning with alcohol. The active electrode was placed over the motor point of the tibialis anterior, approximately 7 cm distal to the tibial tuberosity and 2 cm lateral to the anterior border of the tibia. The motor point was determined by moving the active electrode more proximal or distal until M-wave amplitude was maximal. The reference electrode was positioned over the distal tendon of the tibialis anterior. The ground electrode was positioned over the patella. Using a Digitimer stimulator (Model DS7AH, Digitimer Ltd., Welwyn Garden City, Hertfordshire, UK), all electrical stimulation was delivered using a bar electrode positioned over the peroneal nerve distal to the fibular head. Surface EMG

signals were band-pass filtered between 10 Hz and 5 kHz and then sampled at 2500 Hz.

3.1.4 Experimental Procedures

Each testing session began with the Biodex in the isometric mode and the ankle at 25° of plantar flexion. Twitch and M-wave amplitude were determined by increasing the stimulation intensity in a stepwise method to achieve maximum twitch and M-wave amplitude, and then stimulation intensity was adjusted 10-20% beyond that needed to achieve maximum twitch and M-wave amplitude. This intensity was used for doublet stimulation (10 ms inter-pulse interval) to assess voluntary activation and twitch stimulation to determine post-activation potentiation. To assess maximum relaxation rate (MRR), 50 Hz stimulation bursts of 1.5 s duration were gradually increased in intensity via a second computer-controlled Digitimer stimulator until the stimulation intensity was 10-20% above the intensity needed for 50 Hz torque to reach a plateau. A second stimulator was used because the stimulation intensity used to evoke twitches (100 μs pulse width, 50-75 mA) was higher than needed to evoke maximum 50 Hz amplitude (50 μs pulse width, 40-60 mA).

Following ten minutes of rest, PRE measurements were obtained from the performance of three MVCs and evoked twitches and tetani, with 2 min of rest between each MVC (Fig. 3.1). Each sequence, in order, of a 5 s MVC with an interpolated doublet followed by a post-MVC twitch, a doublet, and a 50 Hz train (with ~ 1 s duration between evoked contractions) (Fig. 3.1). In addition to the real-time display of torque feedback on an oscilloscope, strong verbal encouragement was provided during the MVC portion of each trial. The peak torque value of the three attempts was recorded as the subject's PRE MVC. PRE measurements for the evoked contractions were taken from

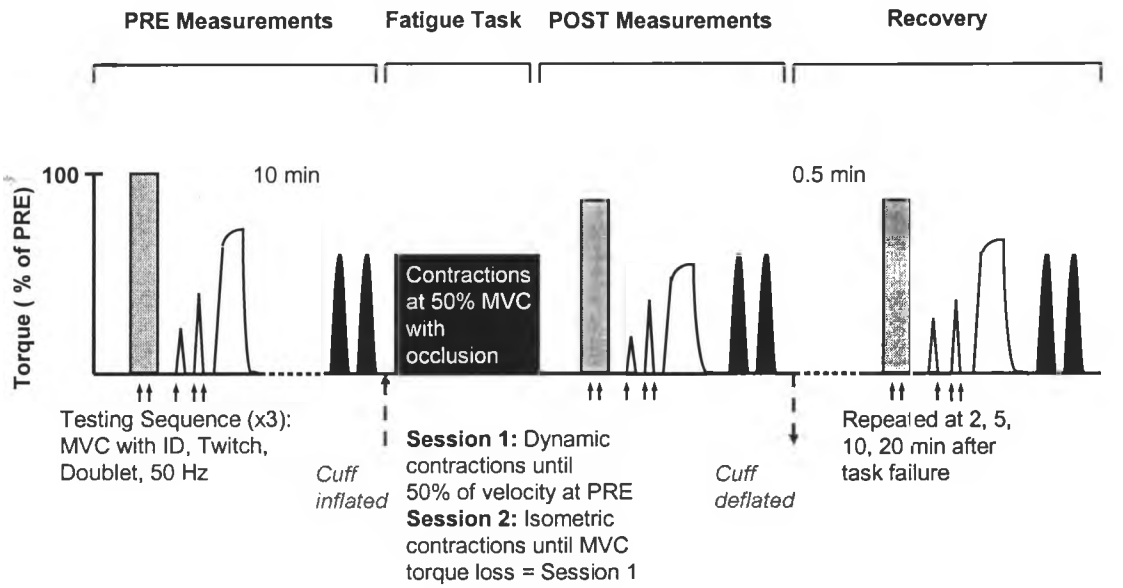


Figure 3.1 Schematic diagram of experimental protocol. Grey bars are isometric maximum voluntary contractions (MVC); open torque profiles are electrically evoked contractions (twitches, doublet, 50 Hz); black-filled profiles are dynamic contractions at 50% MVC; single arrows are electrically evoked twitches; double arrows are electrically evoked doublets; and ID is interpolated doublet.

the testing sequence with the highest MVC torque. The dynamic contraction torque was then set at 50% of MVC, corresponding to the load at which peak power is achieved for the dorsiflexors (35). This resistance load was used for all of the dynamic contractions in the testing session. Velocity at PRE was determined by performing two dynamic contractions as fast as possible (~ 1 s rest between contractions) subsequent to the ten minutes of rest provided after the MVCs (Fig. 3.1). The ankle passively returned to the start position after performing each shortening contraction.

After the two dynamic contractions assessing velocity at PRE, a blood pressure cuff, positioned over the distal thigh, was inflated rapidly to above 220 mmHg to occlude blood flow to the leg. Doppler ultrasound (GE/Vingmed, System 5 Horten, Norway) over the anterior tibial artery was used during pilot testing to determine that blood flow to the dorsiflexors was occluded at 220 mmHg throughout the fatigue tasks.

For the fatiguing protocol of the first testing session, the subject performed at 50% of MVC load, maximal effort dynamic contractions at the fastest velocity he could achieve. To induce a similar relative decrease in velocity among the subjects, the criterion for task failure was determined as the point at which the velocity for two consecutive contractions nearing the end of the fatiguing task declined to < 50% of velocity at PRE. The subject was provided with visual feedback for contractile velocity via an oscilloscope. The contract:relax pace of the fatiguing protocol was ~ 1:1 s in duration. Following the shortening phase of each dynamic contraction of the fatiguing protocol, the subject was told to relax and to allow his ankle to return passively to the starting position. Approximately 5 s after task failure from the dynamic contractions, the subject performed the isometric testing sequence followed by two dynamic contractions (Fig. 3.1) with ~1 s

rest between each contraction. This time-point after task failure is referred to as POST. After the two dynamic contractions, the blood pressure cuff was deflated rapidly. The subject repeated the isometric testing sequence followed by two consecutive dynamic dorsiflexions at 0.5, 2, 5, 10, and 20 minutes following task failure (i.e., the recovery period). This testing sequence allowed for the assessment of both isometric and dynamic contractile function during the recovery phase (Fig. 3.1).

The second testing session differed only by the fatiguing task, in which subjects performed isometric rather than dynamic contractions. For the fatiguing protocol of the second session, subjects performed intermittent isometric contractions using a similar torque level (50% of MVC) and contract:relax pace of 1:1 s. Subjects were asked to target the contraction load as quickly as possible and rest immediately during the relaxation phase of the isometric contractions. To determine task failure, 1 s MVCs were performed at contraction fifteen and every fifth contraction thereafter, i.e., task failure of the isometric session occurred when MVC torque decreased to the level observed immediately following task failure of the dynamic session. These 1 s MVCs were included in the repetitions to task failure for the isometric fatigue task. The periodic MVCs during the isometric task allowed the MVC torque loss at task failure to equal the MVC torque loss following dynamic task failure.

3.1.5 Data and statistical analyses

All torque and velocity data were obtained directly from the Biodex, converted by a 12-bit A/D converter (CED Model 1401 Plus; Science Park, Cambridge, UK), and then sampled at 100 Hz. Spike2 computer software (CED, Science Park, Cambridge, UK) permitted real-time display and inspection of all data channels, and off-line analysis of

torque, velocity, and EMG. MVC, loaded shortening velocity, evoked twitch and 50 Hz tetani were analyzed for peak values. Percentage voluntary activation was calculated as: (1- a/b) x 100%, where a is the torque of the interpolated response of the doublet at peak torque, and b is the torque of the post-MVC doublet at rest (20). The normalized MRR of the 50 Hz tetanus (s^{-1}) was calculated by dividing the peak rate of change of torque during the relaxation (N·m/s) by the peak torque of the 50 Hz tetanus (N·m) (27). During the isometric and dynamic contractions of the fatiguing task, root-mean-square (RMS) values of the raw surface EMG signal were calculated over successive 0.125 s segments for the duration of the isometric contraction, or the concentric portion of the dynamic contractions. The criterion for determining the PRE and POST RMS values was based on selecting, at the beginning and end of the fatigue tasks, the highest velocity for the dynamic contractions or the torque level closest to the target load for the isometric contractions. The maximum rates of torque development (N·m/s) were calculated from the contraction at the beginning of the isometric fatiguing task that most closely matched the target load. The maximum rates of torque development (N·m/s) were also calculated for the dynamic fatiguing task from the dynamic contraction with the highest velocity at the beginning of the task. To examine the extent of change in MVC, velocity, M-wave amplitude, and MRR, data for the fatigue and recovery protocol were normalized relative to values at PRE.

Using SPSS software (v.16), a two-factor (contraction task x time) within-subjects repeated measures ANOVA was used to assess the change between the measures (MVC, velocity, M-wave amplitude, peak twitch torque, MRR, 50 Hz torque) for the fatigue and recovery protocol. The statistical significance of differences in voluntary activation

between contraction tasks over time were determined using a Friedman's test as the data were not normally distributed. For each contraction task, Mann-Whitney U-tests were used to determine the statistical significance of differences in voluntary activation over time. Paired T-tests were used to compare absolute values at PRE, repetitions to task failure between the testing sessions, and PRE and POST RMS amplitude. Statistical significance was defined as $p < 0.05$. If statistical significance was achieved for the ANOVA, a Tukey's HSD post-hoc analysis was performed. All tabulated data are presented as means (SD).

3.2 RESULTS

3.2.1 Baseline Measurements

Due to technical problems, one subject was excluded from the data analysis of the M-wave amplitude. Neuromuscular measures at PRE were not significantly different between the two testing sessions (Table 3.1).

3.2.2 Fatigue and Recovery

For the fatiguing task, there were significantly fewer repetitions to task failure for the dynamic compared to the isometric task [31.0 (8.3) and 47.2 (13.4), respectively]. The greater endurance of the isometric task was not due to significant differences in contraction duration or torque with mean contraction durations of 1.0 (0.6) s for the dynamic task and 0.9 (0.2) s for the isometric task (Fig. 2.2). The mean contraction torque was 24.1 (4.0) N·m for the dynamic task and 24.3 (6.1) N·m for the isometric task. The maximal rates of torque development for the initial contractions of the isometric fatiguing task were 125.6 (28.3) N·m/s, which were significantly slower than the dynamic

Neuromuscular Measures	Session 1 (Dynamic Fatigue Task)		Session 2 (Isometric Fatigue Task)	
	PRE	POST	PRE	POST
Peak MVC torque (N·m)	50.4 (7.9)	35.4 (7.5) *	50.1 (9.1)	35.7 (7.3) *
Peak shortening velocity (°/s)	130.3 (25.6)	50.4 (23.2) *	131.5 (29.7)	43.7 (22.0) *
Voluntary activation (%)	100 (0.1)	100 (0.2)	100 (0)	99.8 (0.5)
M-wave (mV)	13.7 (2.6)	13.3 (2.4)	13.2 (2.2)	13.2 (2.2)
Peak twitch torque (N·m)	8.0 (1.9)	2.4 (1.0) *	9.2 (2.1)	3.7 (2.7) *
50 Hz torque (N·m)	34.4 (7.2)	23.7 (6.4) *	34.3 (10.0)	25.5 (8.2) *
50 Hz MRR (s^{-1})	6.6 (3.9)	2.8 (1.9) *	6.6 (4.3)	2.9 (2.1) *
Absolute RMS amplitude (mV)	1.2 (0.3) †	1.1 (0.2) †	0.5 (0.3)	0.6 (0.2)

Table 3.1 Neuromuscular properties of the dorsiflexors at baseline (PRE) and at task failure (POST). Maximum voluntary isometric contraction (MVC), normalized maximum rate of relaxation (MRR), root mean square (RMS). Data are presented as means (SD). * indicates values at POST are significantly reduced compared to PRE, $p < 0.05$. † indicates significantly larger RMS values for first and last voluntary contraction of the dynamic task compared to isometric task, $p < 0.05$.

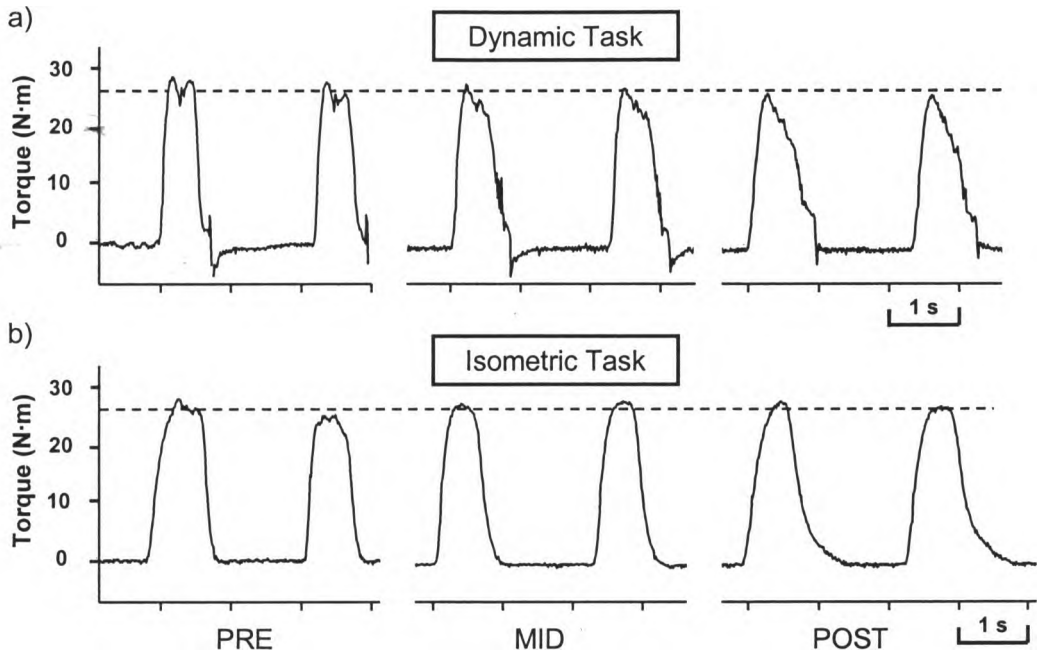
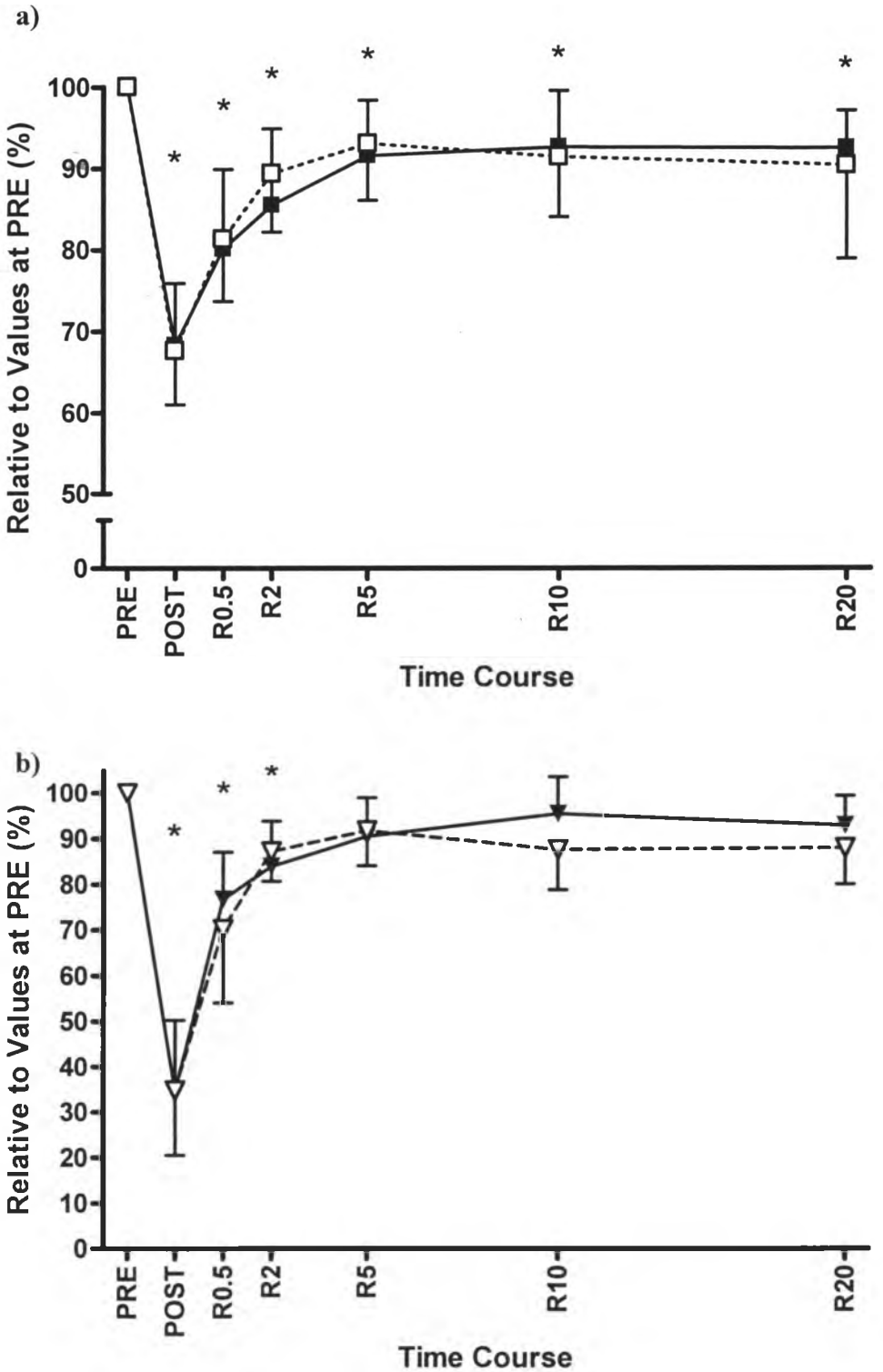


Figure 3.2 Torque output for a representative subject performing voluntary dynamic (**a**), and intermittent isometric (**b**) contractions at 50% MVC during the fatiguing tasks. Two contractions were taken from the beginning (PRE), near the midpoint (MID), and the end (POST) of each fatiguing task. The horizontal dashed lines indicate the mean torque level which was similar for each task. A spike in positive torque near the end of the dynamic contractions is a mechanical artifact of the dynamometer caused by the foot reaching the end of the range of motion during the shortening phase of the contraction. Torque below 0 N·m during the dynamic contractions is representative of relaxation.

contractions at 256.7 (48.9) N·m/s. Neural activation was likely different between the contraction tasks as indicated by the significantly greater absolute RMS values at PRE and POST for the dynamic contractions (Table 3.1). In addition, there was a trend toward increased absolute RMS following the isometric fatigue task ($p = 0.08$) whereas relative RMS was unchanged following the dynamic fatiguing task.

After matching for torque loss, no significant differences were observed between the two fatiguing tasks in neuromuscular properties (i.e., loaded shortening velocity, peak twitch torque, slowing in MRR of the 50 Hz tetanus, voluntary activation, M-wave amplitude) following task failure (Table 3.1), and during recovery. The results from both sessions are presented separately in the graphs, but due to the lack of significant differences between any measures, results from both sessions are presented together in the text to assess overall neuromuscular changes following equivalent MVC torque loss. Relative to values at PRE, the MVC torque significantly decreased to 67.8 (7.0)% at POST and recovered to only 91.5 (8.6)% at 20 min, a value still significantly below the value at PRE (Fig. 3.3). In contrast, relative to PRE, loaded shortening velocity significantly decreased to 34.7 (14.6)% at POST, returning rapidly toward its PRE value after 0.5 min of recovery [73.5 (13.5)%], but it did not recover fully until 5 min (Fig. 3.3). Percent voluntary activation remained high and unchanged throughout the duration of both testing sessions (Table 3.1). M-wave amplitudes were unchanged at POST following both sessions and during recovery, except at 10 min of recovery at which time an 8.5 (8.3)% increase was observed. Peak twitch torque at PRE significantly decreased to 35.3 (19.7)% at POST, recovered to its greatest extent at 5 min to 76.5 (14.5)% of PRE, but decreased thereafter and remained depressed to 66.5 (10.1)% of PRE at 20 min

Figure 3.3 Fatigue-induced changes in isometric MVC (a), and velocity (b) are not significantly different for the dynamic task and isometric task. Data are presented as: dynamic fatigue task (filled symbol, solid line), isometric fatigue task (open symbol, dotted line). The normalized fatigue protocol is represented as PRE and POST and the time course of the recovery protocol by absolute times as R0.5 to R20 (min). Data are presented as means (SD). * indicates significantly different from PRE, $p < 0.05$.


a)
Relative to Values at PRE (%)
100
90
80
70
60
50
0
PRE
POST
R0.5
R2
R5
R10
R20
Time Course
b)
Relative to Values at PRE (%)
100
90
80
70
60
50
40
30
20
10
0
PRE
POST
R0.5
R2
R5
R10
R20
Time Course

of recovery. 50 Hz torque significantly decreased to 71.4 (8.1)% of PRE at task failure, returning quickly toward PRE values at 0.5 min after task failure but did not significantly recover throughout the 20 min following task failure [90.4 (4.3)% of PRE at 20 min]. MRR of the 50 Hz tetanus relative to PRE significantly decreased to 52.0 (16.4)% at POST but recovered to 101.5 (17.3)% after 5 min of rest.

3.3 DISCUSSION

After achieving equal fatigue-induced torque loss (assessed by isometric MVC), dynamic contractions required fewer contractions to task failure than the isometric task, indicating a faster development of fatigue following these maximal effort contractions. Voluntary activation was equally well-maintained and did not change over the duration of either task. After matching for MVC torque loss, fatigue resulted in equal impairments in muscle contractile function at task failure and recovery for both tasks. This was shown by similar changes and time courses for both tasks in shortening velocity, MVC torque, evoked peak twitch and 50 Hz contraction torque, as well as 50 Hz MRR. Thus, measurements of both isometric MVC torque and shortening velocity did not help differentiate the relative influence of potential factors affecting fatigue for the different voluntary tasks. Instead, the ~33% greater reduction at task failure and subsequent faster recovery of shortening velocity compared to MVC torque suggests that the assessments of torque and velocity were useful in providing new insights pertaining to different sites affected by fatigue. This was unrelated to whether a submaximal intermittent isometric or maximal intermittent dynamic fatigue task was performed.

3.3.1 Endurance

Under conditions of equal muscle activation, perhaps the greater metabolic cost of

dynamic compared to the isometric contractions found during electrically stimulated contractions in animals (18, 24, 31) and humans (37) explains the reduced endurance of a dynamic task. Although in this study I equated both tasks for contraction load (50% of MVC torque), muscle activation was not equal during the voluntary tasks. This is highlighted by the two-fold greater EMG activity of the dynamic contractions at both PRE and POST task failure compared to the isometric task, (Table 3.1). It is unlikely that the greater EMG during the dynamic task was due to changes in dorsiflexion ankle position as previous findings have shown no difference in EMG during isometric MVCs performed at short and long lengths in the dorsiflexors (32). I also found at PRE that the maximum rate of torque development of the dynamic contractions was nearly twice as great as that for the isometric task supporting the EMG results. Thus, the reduced endurance of the dynamic task may be related to both greater muscle activation and the potential greater metabolic cost of dynamic contractions.

3.3.2 Central Fatigue

Complete voluntary activation is often observed for the dorsiflexors (5, 6, 21, 34) and thus as expected, voluntary drive was maintained and did not explain task failure under either condition. One limitation of the present study was not being able to assess central drive during the dynamic contractions, but additional support of maintained voluntary drive to the tibialis anterior is provided by the unchanged 50 Hz tetanus/MVC ratio at POST, compared to PRE [0.70 (0.09)]. Thus, it seems for both conditions, fatigue was related to peripheral factors.

3.3.3 Peripheral Fatigue

The unchanged M-wave amplitudes suggested that neuromuscular transmission

impairments (axonal conductance, neuromuscular junction and muscle membrane conductance) were not factors during either task. However, M-wave measures are not sensitive to changes in electrical propagation within the transverse tubules, which could affect excitation-contraction coupling (4, 40). This site has been highlighted as one significant factor in isolated preparations for the fatigue-related decrease in force during isometric contractions (10, 11, 43) although its direct role during in vivo conditions has yet to be confirmed (10). In my study however, the 36% greater reduction in peak twitch compared to 50 Hz contraction torque in both tasks signified prolonged low-frequency force depression, and provided an indication of excitation-contraction coupling failure (1, 17). In addition, I observed similar fatigue and recovery following both tasks in the 50 Hz MRR, in the decrease in peak twitch torque, and no significant difference between tasks in changes in the MVC/50 Hz torque ratio. These changes in evoked contractile measures in both tasks provide strong evidence for equal muscle contractile failure due to excitation-contraction coupling and crossbridge function impairments (7, 27, 29, 46). I also found similar reductions in shortening velocity following both fatigue tasks and comparable rates of recovery. Because I controlled for torque loss it was not surprising to find equal changes in contractile failure properties, however I had expected that dynamic fatigue would result in greater impairments in shortening velocity than the isometric task.

Several current reviews argue that there are independent mechanisms for the fatigue-related reduction in torque (or force) compared to shortening velocity (1, 13, 19). Factors for torque loss include numerous transarcolemmal ionic imbalances (10), increased ROS production (36, 39), and increased P_i (1, 15, 19) altogether impairing SR

$[Ca^{2+}]$ handling. These metabolic processes may reduce torque through several different sites within the muscle (1). These include ROS and K^+ accumulation in the transverse tubules, which prevents action potential propagation, P_i – induced inhibition of the ryanodine receptors, formation of Ca^{2+}- P_i precipitate in the SR that reduces Ca^{2+} availability for release, ROS-induced structural degradation of troponin, and P_i – induced inhibition of strongly-bound force-generating crossbridges (1). In comparison, different mechanisms appear responsible for the reduction in shortening velocity, and this is related to the direct inhibition of crossbridge cycling rates with $[ADP]_i$ (47, 48) and $[H^+]_i$ accumulation (14, 30). Thus, it appears that depending on the contraction task, the contributions of factors involved in the reduction in torque might dominate whereas in other tasks, the primary factors are those related to the reduction in shortening velocity (1). However, the relative influence of these factors following different types of fatiguing tasks is largely unknown. In contrast to my hypothesis, the contraction task used to induce fatigue was not important in differentiating the mechanisms of fatigue because the fatigue-induced changes in torque and velocity were similar following both tasks.

The assessment of torque and velocity were useful in elucidating the different sites affected by fatigue, regardless of the contraction task used to induce fatigue. For example, following both tasks I found a 33% greater reduction in shortening velocity than isometric MVC torque; this suggests that irrespective of the task, dynamic muscle function as assessed by shortening velocity is reduced to a greater extent by fatigue than static muscle function. Previous studies in humans have not assessed torque and velocity after isometric fatiguing tasks, but following dynamic fatiguing tasks, results are consistent with my findings of a greater decrease in power output than isometric MVC

torque at task failure (12, 25-27), suggesting reduced velocity at peak power is a key component in power loss. Although these findings are different than those in animals, which have showed a smaller decrease in maximum unloaded shortening velocity (i.e., at 0% of MVC) than peak isometric force following fatigue (47), I used a loaded shortening velocity (50% of MVC load) because this is likely more functionally representative of muscles in vivo that always shorten against some resistance (e.g., limb weight).

Furthermore, following both the isometric and dynamic fatiguing tasks in my study, the more rapid recovery of shortening velocity within 5 min after task failure compared to the incomplete recovery of isometric MVC torque after 20 min following task failure suggests that factors affecting velocity such as $[ADP]_i$, and $[H^+]_i$ are more quickly restored to unfatigued levels, and thus are not the same as those affecting MVC torque. These findings during recovery are supported by observations from a prior study in the quadriceps, in which there was incomplete recovery in isometric knee extension MVC torque by 10 min after dynamic task failure but the full recovery of velocity-dependent power by 5 min following task failure (12).

Although many studies have used a fatigue-related reduction in isometric MVC torque to indicate the overall extent of fatigue of the neuromuscular system, my results in conjunction with previous supportive findings show that measurements of isometric MVC torque are inappropriate to reflect entirely fatigue-induced changes in the muscle, especially fatigue-induced changes in muscle power output. Thus, by controlling for the potentially confounding factors of muscle perfusion and blood flow, the indicators of peripheral fatigue were the same following different tasks. The greater reduction at task failure and faster recovery of shortening velocity than isometric MVC torque following

both tasks suggest these two measurements reflect different sites affected by fatigue.

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CHAPTER 4

THE FATIGUE RESPONSE OF TWO DIFFERENT MUSCLES DURING VOLUNTARY DYNAMIC CONTRACTIONS

4.0 INTRODUCTION

Generalizations about fatigue-related processes are often made from the fatigue response of one muscle or functional group (e.g., knee extensors) following voluntary dynamic contraction tasks in humans. Due to the unique intrinsic properties and anatomic features of individual muscles, it seems unlikely that these generalizations are warranted and indeed could be misleading (8). Unlike isometric fatiguing contraction tasks, dynamic contraction tasks are affected by both reductions in torque (or force) as well as shortening velocity, which when combined contribute to the fatigue-related decline in muscle power. Few studies have examined fatigue in different muscles following either isometric or dynamic contractions, and methodological differences among these studies have obscured an improved understanding of fatigue-related processes under different contraction tasks.

Fatigue-induced contractile slowing may be beneficial during fatiguing isometric contractions because it may allow for reduced motor unit discharge rates to ensure fusion of torque output (7). In contrast, during dynamic contractions, contractile slowing leads to a decrease in shortening velocity and hence, decline in power output (2). The reduction in shortening velocity with fatigue seems to be largely caused by the accumulation of metabolites that slow crossbridge cycling (12, 30, 39). Thus, compared

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to long-duration endurance-type fatigue tasks, shortening velocity is likely a greater factor during short-duration, metabolically-challenging fatiguing tasks, in which there is a greater reliance on anaerobic energy provision and an impaired removal of metabolites (2). Consequently, muscles with a high aerobic capacity should have a greater endurance against fatigue-induced reductions in shortening velocity than muscles with a lower aerobic capacity. A competing influence on reductions in shortening velocity is post-activation potentiation, which is known to enhance shortening velocity in unfatigued conditions (3, 31). Therefore, during fatigue, post-activation potentiation may mitigate some of the impairment in shortening velocity. This is beneficial in muscles with greater post-activation potentiation ability, such as those with a greater preponderance of fast-twitch fibres. However, during voluntary contractions the increase in shortening velocity due to potentiation is only ~14% (3), and thus may compensate only to a modest extent for any fatigue-induced reductions in shortening velocity.

A comparison of the triceps brachii and soleus muscles offers a potentially useful model to study fatigue-induced changes in shortening velocity in vivo. From many perspectives these two muscle groups represent opposite ends of the functional and morphological spectrum of various muscles in humans. The soleus is a distal lower-limb antigravity muscle from the plantar flexor group composed predominantly of slow-twitch fibres (i.e., ~ 78-89% type I) (19, 22), whereas the triceps brachii is a proximal upper-limb gravity assisted muscle of the elbow extensor group with the lowest fibre composition of slow-twitch fibres (i.e., ~ 33-37% type I) amongst limb muscles (19, 22). The different composition of myosin heavy-chain as well as light-chain content between these two muscles determines several physiological disparities related to faster contractile

speeds (19), greater post-activation potentiation (27, 28, 34), and greater fatigability of the triceps brachii compared to the soleus following repetitive electrically evoked isometric tetani (19). Because the soleus is a chronically-trained antigravity muscle, the different functional role of muscles in vivo might also explain the fatigue resistance of the soleus (17). These physiological and functional differences can potentially be exploited to determine their contribution to fatigue during dynamic velocity-dependent contraction tasks.

The purpose of this study was to evaluate the fatigue response of the triceps brachii and soleus muscles using velocity-dependent contraction tasks. Both muscles performed dynamic contraction tasks consisting of repetitive shortening contractions at a load of 50% of maximum voluntary isometric contraction (MVC) torque, with the criterion for task failure being an equivalent relative reduction in shortening velocity. It was hypothesized that there would be a longer time to task failure in the soleus compared to the triceps brachii, because in spite of a small possible compensatory influence of post-activation potentiation in offsetting fatigue, the soleus has been shown previously during isometric tasks to be highly fatigue resistant when compared to the triceps brachii (19).

4.1 METHODS

4.1.1 Subjects

Seven recreationally-active young men [23.1 (1.3) y, 176.9 (7.5) cm, 79.7 (8.2) kg] were recruited for this study. All subjects were asked to refrain from exercising at least 48 h before the testing session, and to not consume caffeine on the day of testing. Informed written consent was obtained from all subjects. This experiment was approved

by The University of Western Ontario Human Research Ethics Board and was performed in accordance with the Declaration of Helsinki.

4.1.2 Testing Overview

Testing on the triceps brachii and soleus were performed on two separate sessions, no more than two weeks apart, and the testing order of the muscle groups was randomized. On each testing session, subjects performed a dynamic fatiguing task until task failure (POST), which was defined as a relative reduction in shortening velocity to 50% of baseline (PRE) values. For each testing session, measures were taken at PRE, POST, and during the recovery period at 1.5, 3, and 5min after task failure. Measurements included peak isometric MVC torque and electrically evoked 50 Hz tetani torque, shortening velocity, voluntary activation, post-activation potentiation, and normalized maximum rates of relaxation (MRR) of the isometric 50 Hz tetani.

4.1.3 Experimental Setup

All testing was performed on a Biodex System 3 multi-joint dynamometer (Shirley, New York, USA). The isometric mode of the Biodex was utilized during the assessment of MVC, voluntary activation, electrically evoked peak twitch torque, and peak 50 Hz torque as well as MRR of the 50 Hz contractions. The isotonic mode was used to perform the fatiguing dynamic contractions and allowed for the assessment of changes in shortening velocity throughout the experiment. Only the right limbs were tested as these were the dominant limbs for all seven subjects. For elbow extension, subjects were secured in an upright chair with their hips at a 100° angle. In order to prevent extraneous movement of the shoulder and body, two inelastic straps fixed the right shoulder, another strap was placed tightly around mid-torso, an inelastic strap

secured the thighs, and the arm was secured to an elbow rest. One inelastic strap secured the wrist to a custom designed aluminum plate built onto the arm attachment of the dynamometer. The right arm was rotated externally 40° from the sagittal plane and was abducted approximately 20° from the torso, with the subjects forearm placed in a semi-prone position. The axis of rotation of the dynamometer was aligned with the lateral epicondyle of the humerus. A goniometer was used to determine joint range of motion (ROM), which was set from 0° (i.e., anatomical reference with the elbow extended) to 120° of elbow flexion. 120° ROM was determined in the present experiment as the maximum ROM that most subjects could attain.

For plantar flexion, subjects were secured in an upright chair with their hip angle at ~ 60°. Because the soleus has the largest physiological cross-sectional area of any muscle in the leg, it contributes ~ 70% of maximum isometric torque output of the triceps surae when the knee is fully extended (15). To diminish the influence of the gastrocnemius in contributing to plantar flexion torque, the subjects knee angle was bent to 90° to slacken the gastrocnemius muscle (26). Two inelastic straps fixed the upper body and one strap was placed around the waist. One strap secured the thigh to a rigid attachment to the dynamometer, preventing involvement of the thigh muscles. Inelastic straps across the toes and dorsum of the foot secured the limb to the dynamometer footplate. The axis of rotation of the dynamometer was aligned with the lateral malleolus of the tibia and a goniometer was used to determine plantar flexion ROM, which was set from 30° of dorsiflexion, near the angle of optimal plantar flexion peak torque (38), to 20° of plantar flexion. This 50° of plantar flexion ROM represented the maximum ROM of the plantar flexors in this experiment.

Two aluminum electrode pads (4.5 x 4 to 4.5 x 8cm in size) were customized to each subject to stimulate the triceps brachii and soleus. These stimulation electrodes were placed transversely over the belly of the triceps brachii or soleus muscle to electrically stimulate the muscles using a Digitimer stimulator (DS7AH, Digitimer Ltd, Welwyn Garden City, Hertfordshire, UK). For the triceps brachii, the cathode was centered over the proximal section of the triceps brachii 1 cm distal to the posterior deltoid muscle and the anode was centered midway between the distal border of the deltoid muscle to the olecranon; the placement which gave the greatest twitch amplitude evoked in a relaxed muscle. A ground electrode was placed on the acromion of the right shoulder when testing the elbow extensors. As performed previously for the soleus (6), the electrodes were positioned over its muscle belly with the cathode placed one cm distal to the posterior border of the gastrocnemius and the anode placed at the superior border of the Achilles tendon. A ground electrode was placed on the right patella when testing the soleus muscle.

4.1.4 Experimental Procedures

At the beginning of each testing session, and at the longest muscle length (i.e., 120° elbow flexion angle for the elbow extensors and 30° dorsiflexion angle for the plantar flexors), electrically evoked twitches (100μs pulse width) were incrementally increased in stimulation intensity until twitches reached a plateau in peak torque. If twitch torque decreased with an increase in stimulation intensity, the electrode pads were repositioned or a different electrode size was used until a plateau in twitch torque was achieved with increasing stimulation intensity. Stimulation intensity was then set at ~10% above that needed to achieve peak twitch torque. Next, subjects performed three 5

s duration MVCs followed by 2 min rest after each MVC. In the time period of each MVC, a twitch was evoked to test voluntary activation using the interpolated twitch technique (16), and post-activation potentiation was assessed by delivering a twitch before the MVC, and in a relaxed and potentiated muscle ~ 3 s after each MVC. Subjects were verbally encouraged during all voluntary contractions and were also provided with visual feedback of their torque on a computer monitor. Because the stimulation intensity used to evoke twitches was much higher than could be tolerated for the 50 Hz stimulation, muscle contractile function was tested with a 50 Hz (50μs pulse width, 1.5s duration) isometric tetanus delivered from a second Digitimer stimulator. Previous findings (6) confirmed my observations from pilot testing that for high-frequency tetani, stimulation intensities using transcutaneous stimulation much beyond that needed to elicit 25% of soleus MVC torque is not well-tolerated. Thus, the stimulation intensity of the 50 Hz in the soleus was set to achieve 25% of MVC torque, which is likely sufficient to assess contractile function in this highly homogenous slow-twitch muscle. To stimulate both muscles comparably, the stimulation intensity of 25% of MVC torque was also used for the predominantly fast-twitch triceps brachii. After 2 min of rest, subjects performed one 5 s duration MVC with a 50 Hz evoked tetanus following the MVC. This was to ensure that the 50 Hz contractile properties measured at PRE were compared under similar circumstances to measurements made at task failure and during the recovery period when the 50 Hz was evoked following the MVC.

After 2 min of rest, subjects were then familiarized with the dynamic fatigue protocol by performing eight voluntary shortening contractions, in which the subjects attempted to achieve the fastest velocity they could during each of these contractions.

The dynamic contraction torque was set at 50% of MVC, and this resistance load was used for all of the dynamic contractions in the testing session. Although the resistance was 50% of the MVC load, this is indeed a substantial load during a shortening contraction. Also, during these contractions, subjects were focused on making maximal efforts by attempting to move as fast as possible throughout the ROM. Ten minutes of rest were provided after familiarization before the start of the fatigue protocol. During this rest period, peak shortening velocity at PRE was obtained off-line from the familiarization contractions in order to set the criterion for task failure (i.e., 50% reduction in shortening velocity relative to the value at PRE) on an LCD display providing visual feedback of velocity (as well as torque) to the subjects prior to the start of the fatiguing task.

The fatigue protocol consisted of repeated dynamic contractions at the fastest velocity they could achieve until task failure, which was defined as a 50% reduction in loaded shortening velocity relative to the value at PRE. The dynamic contractions of the fatigue task were shortening contractions only, in which the experimenter returned the dynamometer as rapidly as possible to the start position (i.e., < 1s between repetitions) after the subject performed each shortening contraction. To induce rapid fatigue, these contractions continued successively until task failure. Subjects performed a MVC testing sequence immediately after task failure (POST), and then during the recovery period at 1.5, 3, and 5 min following task failure. Each MVC testing sequence consisted in order of a MVC with an interpolated twitch, a post-MVC twitch, and a 50 Hz tetanus. Then, each MVC testing sequence was followed immediately by two maximal effort dynamic contractions to assess shortening velocity at task failure and during the recovery period.

4.1.5 Data and Statistical Analyses

All torque, shortening velocity, and joint position data were obtained directly from the Biodex, converted by a 12-bit analog-digital converter (CED Model 1401 Plus; Science Park, Cambridge, UK) and then sampled at 100 Hz. Spike2 computer software (CED, Science Park, Cambridge, UK) permitted real-time display, inspection, and analysis of all data channels. PRE values for MVC torque, voluntary activation, and potentiated twitch were gathered from the MVC with the greatest peak torque amplitude. The unpotentiated twitch amplitude was obtained from the twitch that preceded the first MVC. Peak MVC values were recorded at POST and throughout recovery. Peak values were tabulated for shortening velocity at 20%, 40%, 60%, 80% time to task failure, POST and throughout recovery. Power (W) was calculated as the product of torque at peak shortening velocity (N.m) and peak shortening velocity (rad/s). Work (J) for each contraction was calculated as the product of mean torque [from the onset of shortening to the end of shortening (in N.m)] and joint range of motion [from the start position to the end position (in rad)]. Total work was calculated as the sum of work performed by all contractions of the fatiguing task. Voluntary activation was quantified using the formula 1-(interpolated twitch torque/ post-MVC potentiated twitch torque) x 100% (16). Post-activation potentiation was calculated from each MVC at PRE as post-MVC/ pre-MVC evoked twitch torque x 100%. Peak values of the post-MVC evoked twitch torque were used to evaluate twitch amplitude changes following fatigue and during the recovery period. The maximum rate of relaxation of the 50 Hz tetani (s^{-1}) were normalized by dividing the peak rate of change of torque (N·m/s) by the peak torque of the 50 Hz tetanus (N·m). To examine the extent of fatigue-induced changes in MVC, velocity,

twitch peak torque, 50 Hz peak torque, and 50 Hz maximum rate of relaxation, data at POST and during recovery were expressed relative to 100% of values at PRE.

Using SPSS software (v.16), two-factor (muscle x time) repeated measures ANOVAs were used to assess the statistical significance of relative change between measures over time (MVC, velocity, twitch torque, 50 Hz peak torque, 50 Hz normalized maximum rate of relaxation), and to assess the statistical significance of differences in work generating capacity between muscles over time. If significance at $p < 0.05$ was achieved for the ANOVA, a Tukey's HSD post-hoc analysis was performed. The statistical significance of differences in voluntary activation between muscles over time were determined using a Friedman's test as the data were not normally distributed. For each muscle, Mann-Whitney U-tests were used to determine the statistical significance of differences in voluntary activation over time. Linear regression was used to determine the association between post-activation potentiation and time to task failure. Paired T-tests were used to compare absolute values at PRE, repetitions to task failure, contraction pace, and time to task failure. All tabulated data are presented as means (SD).

4.2 RESULTS

4.2.1 Baseline Measurements

The soleus muscle had significantly greater MVC and 50 Hz torque than the triceps brachii, but the triceps brachii had a significantly faster shortening velocity than the soleus (Table 4.1). Also, power output was not significantly different in the soleus

Measures	Soleus		Triceps Brachii	
	PRE	POST	PRE	POST
MVC (N·m)	155.5 (28.4)	141.3 (27.8) *	80.4 (16.2) †	53.7 (8.8) * †
Peak shortening velocity (°/s)	309.9 (45.8)	142.0 (17.7) *	454.1 (27.0) †	194.2 (27.9) * †
Power at peak shortening velocity (W)	309.1. (58.0)	133.2 (15.3) *	299.9 (47.5)	107.5 (20.8) *
Voluntary activation (%)	96.5 (2.3)	96.3 (4.2)	94.3 (4.3)	93.7 (8.7)
Twitch torque (N·m)	26.1 (4.1)	22.1 (4.0) *	11.0 (4.0) †	2.7 (0.8) * †
50 Hz torque (N·m)	40.9 (5.8)	33.4 (6.3) *	21.6 (3.8) †	13.8 (4.4) * †
50 Hz MRR (s^{-1})	-9.0 (1.2)	-6.0 (1.1) *	-12.6 (1.4) †	-5.9 (1.8) *

Table 4.1 Neuromuscular properties of the soleus and triceps brachii at baseline (PRE) and at task failure (POST). Maximum voluntary isometric contraction torque (MVC), normalized maximum rate of relaxation (MRR). Data are presented as means (SD). * indicates significantly different from PRE, $P < 0.05$, † indicates significantly different between muscle groups at the same time point, $P < 0.05$.

compared with the triceps brachii (Table 4.1). Voluntary activation was not significantly different and nearly complete, in both muscle groups (Table 4.1). Post-activation potentiation in the triceps brachii [196.0 (42.5)%] was significantly greater than in the soleus muscle [140.2 (12.2)%]. The triceps brachii also had significantly faster 50 Hz normalized maximum rates of relaxation than the soleus (Table 4.1).

4.2.2 Fatigue and Recovery

Although there were greater repetitions to task failure in the soleus than triceps brachii muscle [62.9 (12.6) vs. 39.3 (5.7), respectively], this was possibly due to the smaller ROM during plantar flexion compared to elbow extension ROM. When comparing repetitions to task failure relative to time to task failure, the smaller plantar flexion ROM resulted in a significantly faster contraction pace in the soleus of ~ 0.8 repetitions/s than in the triceps brachii at ~ 0.6 repetitions/s. In both muscles, rest was kept at a minimum (< 1 s) between contractions. The times to task failure between the soleus and triceps brachii were not significantly different [79.3 (15.5) s and 69.3 (12.2) s, respectively] although total work during the fatiguing task was significantly lower for the soleus compared with the triceps brachii [1938.8 (459.6) J vs. 3298.5 (931.0) J, respectively] (Fig. 4.1).

During the fatiguing tasks performed in the triceps brachii and soleus, the relative time course changes in shortening velocity were nearly identical between muscle groups (Fig. 4.2a). At POST, the soleus and triceps brachii shortening velocity significantly decreased to a similar relative extent [50.6 (3.0)% and 45.3 (5.8)% of PRE, respectively] and were not significantly recovered in both muscles by 5min [89.9 (10.2)% and 83.2 (5.4)%, respectively] (Fig. 4.2a). There was a significantly slower recovery of shortening

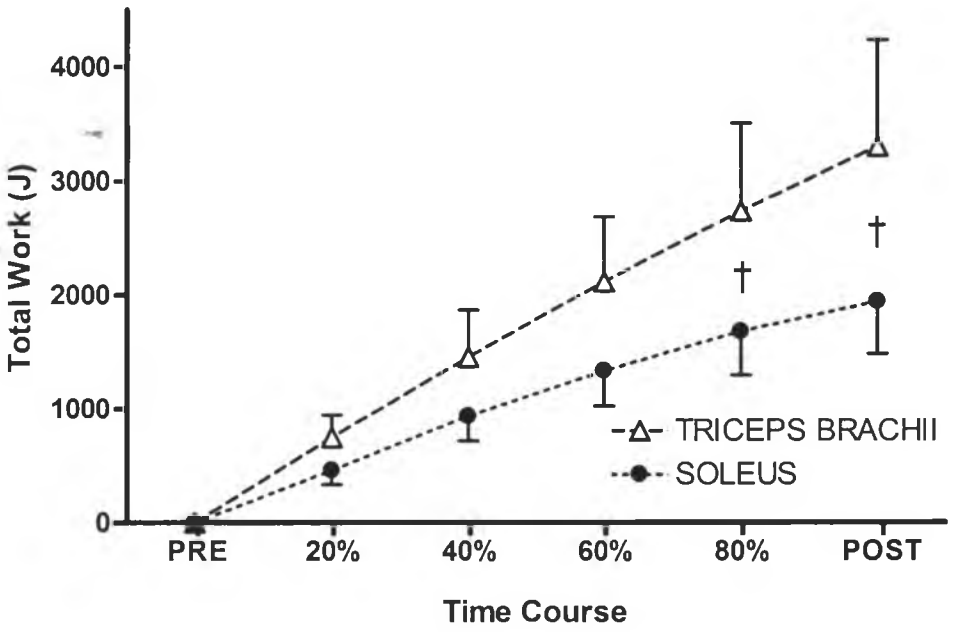
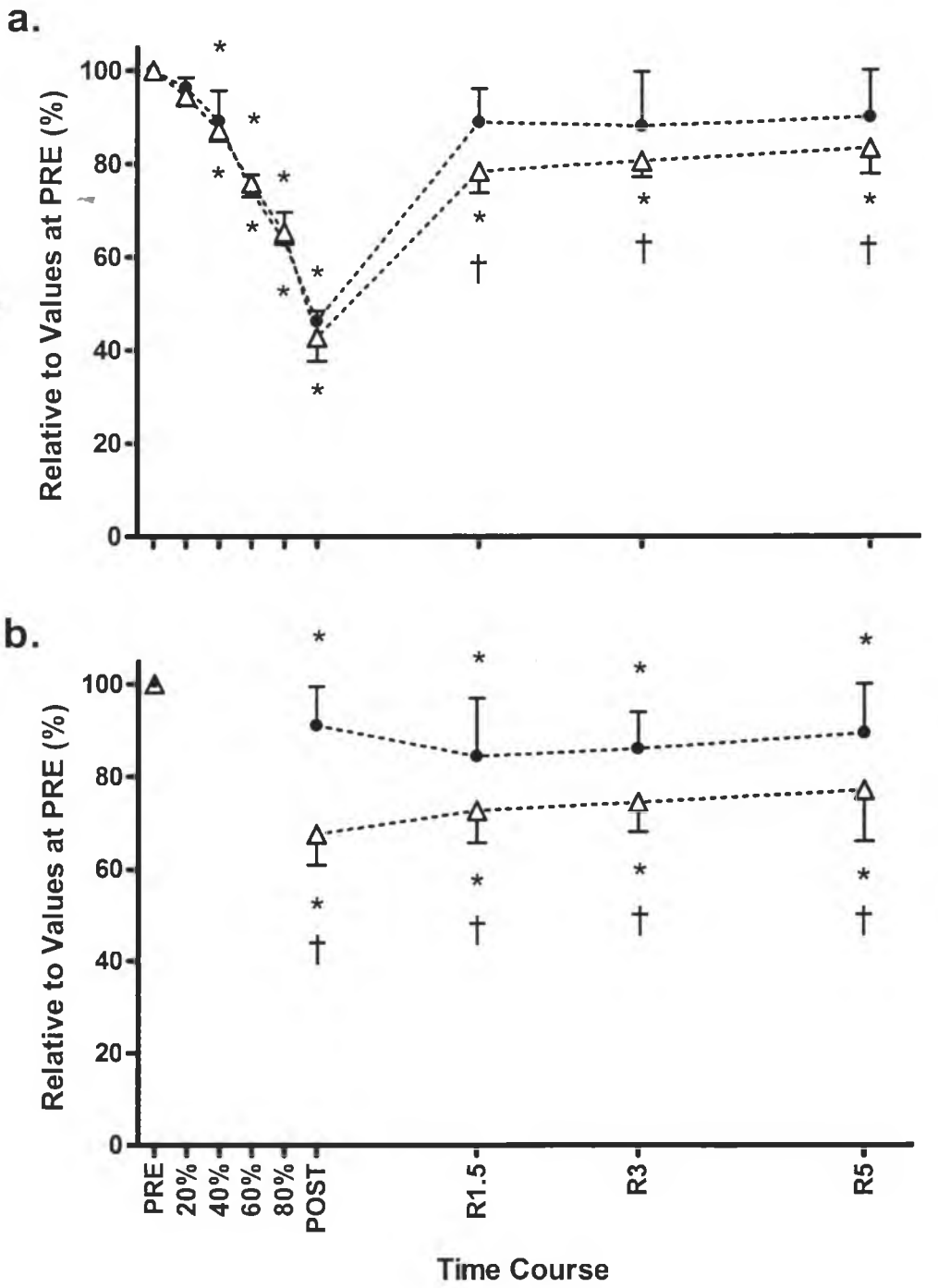


Figure 4.1 A comparison of total work performed during the fatiguing task for the soleus and triceps brachii muscles. The normalized fatigue protocol is represented as PRE, 20-80% time to task failure, and POST. Data are presented as means (SD). † indicates significantly different between muscles, $p < 0.05$.

Figure 4.2 a. Fatigue-induced changes in loaded shortening velocity, and **b.** maximum voluntary isometric (MVC) torque for the soleus and triceps brachii muscles. Data are presented as: soleus (filled circle), triceps brachii (open triangle). The normalized fatigue protocol is represented as PRE, 20-80% time to task failure, POST, and the time course of the recovery protocol by absolute times as R1.5 to R5 (min). MVC was not tested during the fatiguing task per se, but was measured at PRE, POST, R1.5 to R5. Data are presented as means (SD). * indicates significantly different from PRE, $p < 0.05$. † indicates significantly different between muscles, $p < 0.05$.


a.
Relative to Values at PRE (%)
100
80
60
40
20
0
b.
Relative to Values at PRE (%)
100
80
60
40
20
0
PRE
20%
40%
60%
80%
POST
R1.5
R3
R5
Time Course

velocity in the triceps brachii, as shown by the relative 10.6% greater depression in shortening velocity in the triceps brachii than soleus at 1.5 min. Relative to values at PRE, MVC torque at task failure significantly decreased to 91.1 (10.0)% in the soleus, but significantly decreased to a greater extent in the triceps brachii to 67.6 (6.8)% (Fig. 4.2b). The smaller depression in MVC torque of the soleus than triceps brachii remained throughout recovery, and neither soleus or triceps brachii MVC torque were significantly recovered by 5 min after task failure [89.4 (10.6)% and 77.0 (11.0)% of PRE values, respectively] (Fig. 4.2b).

Voluntary activation was nearly complete in both the soleus and triceps brachii at task failure (Table 4.1) and at 5 min of recovery [92.6 (5.9% and 93.9 (8.2)%, respectively]. However, at intermediate time points during recovery, deficits in voluntary activation were observed in both the soleus and triceps brachii. These time points were 1.5 min after task failure in the soleus, in which voluntary activation was 87.6 (9.6)%, and in the triceps brachii, voluntary activation was significantly lower than PRE at 3 min after task failure [79.7 (8.3)%]. In the soleus, twitch torque was significantly reduced at task failure to 81.5 (8.0)% of PRE but was recovered fully by 1.5 min after task failure. In the triceps brachii, twitch torque relative to PRE was significantly reduced at task failure to 29.5 (19.2)% and was only 67.1 (31.6)% by 5 min after task failure. Relative to values at PRE, 50 Hz torque significantly decreased at task failure in both the triceps brachii to 64.0 (16.6)% and in the soleus to 81.5 (8.1)%, but with significantly greater relative reductions in triceps brachii 50 Hz torque. 50 Hz torque was significantly restored to PRE values in the soleus at 1.5 min after task failure but 50 Hz torque in the triceps brachii did not significantly recover up to the 5 min time point after task failure

[72.2 (19.8)% of PRE]. Normalized 50 Hz maximum rate of relaxation was not significantly different ($p = 0.25$) in both muscle groups at task failure, but recovered significantly faster in the soleus by 3 min after task failure [89.1 (19.4)% of PRE], whereas recovery in the triceps brachii was delayed until 5 min [91.2 (22.2)% of PRE]. Finally, a moderate and negative association was found between post-activation potentiation and time to task failure in the triceps brachii ($R^2 = -0.41, p < 0.05$) but no association in the soleus ($R^2 = -0.08, p > 0.05$) (Fig. 4.3).

4.3 DISCUSSION

These findings indicate that the soleus and triceps brachii muscles express different functional properties in the unfatigued condition. This is expressed by a ~ 1.9 fold greater MVC torque, but ~ 1.5 times slower shortening velocity and normalized evoked 50 Hz maximum rate of relaxation, and ~ 56% lower post-activation potentiation in the soleus compared to the triceps brachii. Using the same criterion for task failure, endurance times were similar in both muscles but significantly less total work was performed in the soleus (~1939 J) compared with the triceps brachii (~3299 J). This was unexpected, and suggests that endurance capacity was greater in the triceps brachii than the soleus during the repetitive shortening contraction task. Differences in post-activation potentiation capability between muscles did not appear to result in different time courses for the reduction in shortening velocity during the fatiguing dynamic contraction task. The soleus having a smaller fatigue-related reduction in torque (voluntary and evoked) than the triceps brachii is consistent with a previous finding (19). Furthermore, in both muscles, changes in MVC torque followed a different time course

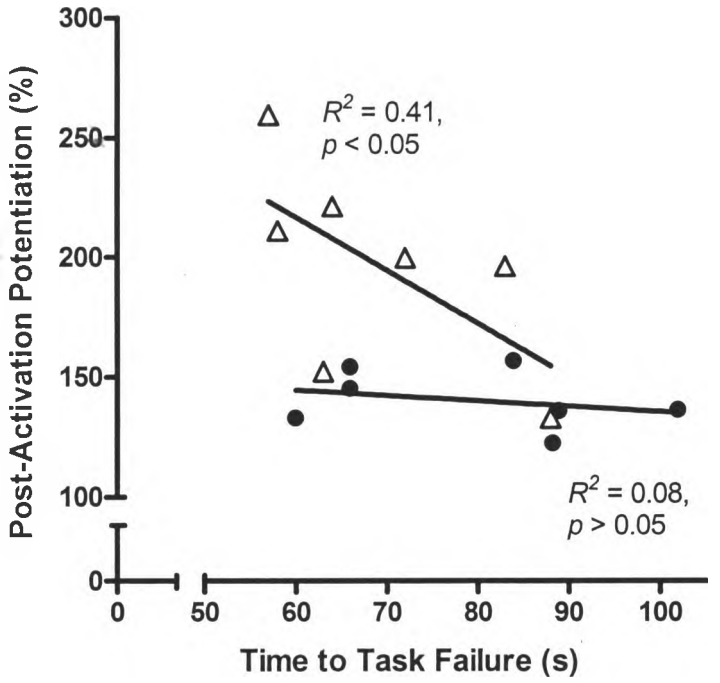


Figure 4.3 Baseline post-activation potentiation for each subject compared to time to task failure. Data are presented as: soleus (filled circle), triceps brachii (open triangle).

compared to shortening velocity during fatigue and recovery. These findings indicate dissimilar fatigue mechanisms for the reduction in MVC torque compared with shortening velocity.

4.3.1 Baseline Measurements

The soleus and triceps brachii muscles indeed provided different functional abilities. In the present study, isometric MVC torque was approximately two-fold greater in the soleus compared to the triceps brachii, which is similar to the MVC torque reported previously in the soleus (5, 18, 19) and triceps brachii (18, 19). This is likely explained by the larger physiological muscle cross-sectional area of the soleus (15) compared to the triceps brachii (29). Also, in the present study, the soleus had ~ 1.5 times slower shortening velocities than the triceps brachii, which compares similarly with the ~ 1.5 times slower evoked 50 Hz maximum rate of relaxation in the soleus than the triceps brachii. The slower contractile speed in the soleus may be associated with its predominance of type I fibres and that it has no type II X fibres, whereas the triceps brachii consists predominantly of type II A and II X fibres (19). Interestingly, power output (at peak shortening velocity) was not significantly different between the soleus compared with the triceps brachii because of the greater absolute torque generating capacity of the soleus, but the faster contractile speed in the triceps brachii.

Consistent with the known greater myosin regulatory light-chain phosphorylation of fast- compared to slow-twitch fibres (36), ~ 56% lower post-activation potentiation was shown in the soleus compared with the triceps brachii. Other studies using similar short 5 s conditioning contractions to induce post-activation potentiation also showed minimal potentiation in the soleus after an isometric MVC (~ 15%, compared to the

unpotentiated state) (27), and greater potentiation ability (ranging from 130-240%) in the triceps brachii (28, 34). Thus, the inherent properties of the soleus and triceps brachii are different, with a greater absolute torque output, slower contractile properties and lower post-activation potentiation capability in the soleus compared to the triceps brachii.

4.3.2 Work

Longer endurance times were expected for the soleus than the triceps brachii because of its previously reported greater fatigue resistance during isometric tasks (4-6, 19). Furthermore, if endurance capacity is greater in the soleus than the triceps brachii during dynamic contraction tasks, then it might be expected that greater total work would be performed by the soleus. On the contrary, similar endurance times were shown for both muscles, but this parameter was confounded by the significantly greater number of repetitions performed to task failure for the soleus compared to the triceps brachii. Because plantar flexion ROM (50°) was smaller than elbow extension ROM (120°), this suggested that less work was performed per plantar flexion contraction. Indeed, when total work performed was calculated for each muscle, approximately half as much total work was performed by the soleus than the triceps brachii using the same criterion for task failure (reduction in shortening velocity by 50%). Because less total work was required to reach task failure in the soleus, this suggests that the soleus had reduced endurance compared with the triceps brachii during the dynamic contraction tasks. Somewhat consistent with previous studies performing isometric contraction tasks that found fibre-type differences do not completely explain differences in fatigability between muscles (17), the present findings of differences in dynamic endurance capacity also appear inadequately explained by fibre-type differences. Perhaps the soleus is largely fatigue-resistant during isometric contractions because the soleus is a chronically-trained antigravity (postural) muscle (17), but shortening contractions

are more energetically costly than isometric contractions (35, 37), placing a high metabolic demand on the muscle. It is possible that fast shortening contractions may be relatively more metabolically costly to perform in the soleus than the triceps brachii, and this could be related to a biomechanical disadvantage of the soleus when performing high-velocity plantar flexion contractions.

From a mechanical perspective, the soleus functions as a second-class lever in plantar flexion, which is optimized for high torque production but not fast shortening velocities, whereas the triceps brachii muscle operates as a first-class lever in elbow extension, with a small lever arm close to the axis of rotation that is optimized for both large joint displacements and fast shortening velocities (21, 32). Thus, the soleus seems to be at a biomechanical disadvantage during high-velocity shortening contractions compared with the triceps, and this is probably related to their respective unique roles in human function. In the present study study, less total work was performed by the soleus compared with the triceps brachii, and this may be related to differences in both muscle morphology and joint biomechanics that might explain the greater endurance of the soleus during isometric, but not during fast dynamic contraction tasks.

4.3.3 Torque and Shortening Velocity

For both muscles, voluntary activation measured during the isometric MVCs remained high at task failure and at 5 min of recovery, and thus the fatigue-related reductions in torque were most likely due to peripheral fatigue processes. Impaired Ca^{2+} release has been argued recently as the primary mechanism responsible for fatigue-induced reductions in torque (1). Therefore, compared with the soleus, the greater reduction at task failure and greater depression during recovery in torque (voluntary and evoked) in the triceps brachii likely indicates a greater impairment in saturated Ca^{2+}

release in the triceps brachii. Indeed, these findings are similar to previous investigations in humans that found better preservation of isometric torque (or force) in the soleus using comparable isometric contraction tasks, when compared to the triceps brachii (19), quadriceps (4, 6), or tibialis anterior (5).

However, it is argued that torque (or force) and velocity are largely affected by different fatigue-related mechanisms (2, 10, 14). Torque loss is explained primarily by impaired SR Ca^{2+} handling, which is affected by transarcolemmal ionic imbalances (9), production of ROS (2), as well as P_i accumulation (13), whereas metabolites such as $[ADP]_i$ (39), $[H^+]_i$ (12), and $[P_i]$ (24) have been implicated in directly slowing crossbridge cycling rates, and thus reducing shortening velocity.

In the present study, a significant difference was found between the soleus and triceps brachii in the time course for the reduction in shortening velocity during the fatiguing task (Fig. 4.2a). Also, there were no significant differences between muscles in the slowing of 50 Hz maximum rate of relaxation at task failure, suggesting comparably slowed contractile kinetics in both muscles, despite differences in MVC torque loss at task failure. The uncoupling of mechanisms affecting torque and shortening velocity is further exemplified by a mismatch in fatigue-related changes in isometric torque and velocity in both muscles. For example, MVC torque was reduced to a small extent at task failure and remained depressed during the recovery period whereas shortening velocity was reduced greatly at task failure and subsequently followed a more rapid restoration profile (Fig. 4.2a,b). The greater reduction at task failure, and faster recovery of shortening velocity compared with MVC torque, support the argument that different fatigue mechanisms affect torque and shortening velocity. The results in the current

study from the triceps brachii and soleus are supported by previous investigations from other dynamic fatiguing tasks in single muscles which found that power (and shortening velocity) is more greatly reduced by fatigue than isometric torque (10, 11, 20, 23). Also, the faster recovery of shortening velocity compared to MVC torque is consistent with my previous findings in the quadriceps (11) and tibialis anterior muscle (10) following dynamic contraction tasks. The rapid recovery of shortening velocity seems to be linked to metabolic restoration because the recovery time course of shortening velocity is similar to the rapid rate of PCr resynthesis observed following maximal 25-30 s sprint cycle ergometry (25).

4.3.4 Post-activation Potentiation

Differences in post-activation potentiation in the soleus and triceps brachii clearly did not differentiate time course changes for the reduction in shortening velocity between the two muscles (Fig. 4.3). There was minimal potentiation in the soleus and no association with time to task failure in the soleus, whereas the negative association between potentiation and time to task failure in the triceps brachii (R^2 = -0.41) likely indicates the greater fatigability of the highly-potentiating fast-twitch fibres. These results do not contradict previous findings showing that potentiation increases shortening velocity by ~ 14% (compared to the unpotentiated state) during voluntary and evoked dynamic contractions in the human adductor pollicis (3). Instead, fatigue-induced reductions in shortening velocity seem to overcome this small compensatory influence of potentiation.

A larger decrease in evoked twitch torque was observed when compared with both MVC and 50 Hz torque in the triceps brachii, at task failure and during recovery.

This greater fatigue-induced reduction in torque at low compared to high frequencies suggests the presence of low-frequency torque depression, which is an indicator of impaired myofibrillar Ca^{2+} sensitivity (2). Post-activation potentiation is known to offset to some degree the impairment in excitation-contraction coupling during isometric fatiguing tasks (33), but it was evident in the current study that the greater post-activation potentiation capability of the triceps brachii was not sufficient to offset the low-frequency torque depression. Even though the soleus had minimal post-activation potentiation ability compared to the triceps brachii, there was a much smaller fatigue-induced reduction in evoked twitch torque relative to MVC or 50 Hz torque in the soleus, and twitch torque recovered rapidly within 1.5 min after task failure. This is supported by previous evidence from mice in situ, in which the greater preservation of SR Ca^{2+} handling with fatigue in the predominantly slow-twitch soleus was largely due to its greater aerobic capacity compared with the predominantly fast-twitch extensor digitorum longus muscle (40).

Therefore, results from dynamic contraction tasks show that the soleus has a greater apparent ability to preserve evoked twitch, 50 Hz and MVC torque compared to the triceps brachii. However, shortening velocity appears primarily affected by contractile slowing, likely due to metabolite accumulation, which seems independent from fatigue processes responsible for the fatigue-related reduction in torque. The present findings from fatiguing dynamic contraction tasks performed in two different muscles indicate that there is an uncoupling of fatigue-related mechanisms between those that reduce MVC torque compared with those that reduce shortening velocity.

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CHAPTER 5

THE INFLUENCE OF MUSCLE LENGTH ON THE FATIGUE-RELATED REDUCTION IN JOINT RANGE OF MOTION OF THE HUMAN DORSIFLEXORS

5.0 INTRODUCTION

Active joint range of motion (ROM) depends on the shortening capacity of muscles acting across a joint. Fatigue diminishes the ability of muscles to shorten and this has been reported to reduce joint ROM during dynamic contraction tasks (8, 17, 18, 20, 23, 39). While fatigue-related reductions in torque (or force) and contractile slowing (i.e., reduced shortening velocity and slowing in relaxation) will decrease muscle power output with fatigue (1), it is not known if these factors are important contributors to the fatigue-related reduction in ROM. Fatigue-related reductions in ROM may have practical implications in humans because it limits the effective ability of limbs to move over a distance. This could be problematic in many daily tasks that involve repetitive muscle shortening contractions.

Klass and colleagues (20) suggested that the reduction in ROM is primarily due to a fatigue-induced rightward shift in the torque-length relationship of the plantar flexors. In their study, the alteration in the torque-length relationship was due to a 10% greater relative reduction in maximum voluntary isometric contraction (MVC) torque at a shortened (20° plantar flexion) compared to a neutral (0°) ankle position following a

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Cheng AJ, Davidson AW, and Rice CL. The influence of muscle length on the fatigue-related reduction in joint range of motion of the human dorsiflexors. *European Journal of Applied Physiology.* In press.

repetitive shortening contraction task used to induce fatigue in the human plantar flexors. However, fatigue-induced changes in "isometric" MVC torque per se, may underestimate the effect of fatigue on muscle shortening capacity. Indeed, in a related study, the fatigue-related reduction in ROM, and its subsequent rapid restoration following a dynamic knee extension task, followed more closely the fatigue and recovery time course of shortening velocity (as well as slowing in relaxation of an isometric tetanus) compared with fatigue-induced changes in MVC torque (8). In another study (23), the slowing of crossbridge kinetics was thought to be a key factor contributing to the reduction in ROM during electrically stimulated dynamic contractions. In that study (23), ROM showed the greatest reduction during the highest load task (10% of MVC force), in which the time of force development was slowed by fatigue, whereas during the low and moderate load tasks (<1% of MVC and 5% of MVC force, respectively), the times for force development and also ROM were less affected by fatigue. Together these findings suggest that fatigue-induced alterations in ROM are affected by fatigue-induced contractile slowing, but the relationship between fatigue-induced changes in ROM and contractile kinetics has not been systematically evaluated in humans. In addition, others have speculated that the reduction in ROM may be related to more greatly impaired crossbridge mechanics at short compared with long lengths (20). This could be due to non-optimal crossbridge formation from increased interfilament spacing at shortened muscle lengths, which results in reduced rates of torque development at short lengths (30, 38). However, it is unclear whether fatigue will further exacerbate the already slower contractile kinetics present in unfatigued muscle at shortened lengths. Furthermore, it seems that studies have not assessed the length-dependent alterations in contractile

kinetics following fatiguing shortening contractions.

I chose to investigate the dorsiflexors because shortening contractions of a moderate displacement can be performed within the plateau region of its torque-length relationship (21, 27, 29), whereas in the few previous studies that describe fatigue-induced changes in ROM, the reduction in ROM might have been complicated in the knee extensors (8, 17, 18, 23, 39) and plantar flexors (20) because these muscles operate on a steep ascending torque-length relationship with greatly reduced torque output at the shorter muscle lengths (3, 27, 40).

The purpose of this study was to investigate the fatigue-related contributions to the reduction in dorsiflexion ROM. In this study, MVC torque was assessed at the short and long length after a 50% reduction in ROM at task failure and during recovery. By electrically stimulating the muscle at both a short and long length, and at various time points throughout the experiment, length-dependent alterations in muscle torque and contractile rate properties [i.e., maximal rate of torque development (MRTD) and maximal rate of relaxation (MRR)] could be assessed following fatiguing dynamic contractions. This design provides an improved ability to explore whether the fatigue-related reduction in ROM is associated with greater impaired torque (voluntary and evoked) or contractile kinetics at a short compared with a long muscle length.

5.1 METHODS

5.1.1 Subjects

Twelve recreationally active young men [24.1 (2.6) y, 177.4 (6.3) cm, 80.4 (11) kg] were recruited for this study. Subjects were asked to avoid caffeine and any lower

limb exercises at least 48 h before the day of testing. Informed written consent was received from all subjects. The University of Western Ontario Human Research Ethics Board approved the study and the experiment was performed according to the Declaration of Helsinki.

5.1.2 Testing Overview

All experimental data were collected from one testing session. The testing began with the establishment of baseline (PRE) measurements, followed by a dynamic fatiguing task that consisted of repetitive shortening contractions at a moderate-load (30% of MVC torque) until total ROM decreased by 50%. The isotonic mode of a Biodex System3 Multi-Joint Dynamometer (Shirley, NY, USA) was used to perform the fatiguing dynamic contractions and allowed for the assessment of ROM and loaded shortening velocity (at 30% of MVC torque) throughout the experiment. Also, at various time points throughout the experiment, the isometric mode of the Biodex was used to assess MVC torque, voluntary activation, and elicited isometric contractile properties, at either a short or a long muscle length.

5.1.3 Experimental Setup

Subjects were seated in the Biodex with the backrest set at an incline of 55° from the horizontal plane. All testing was performed using the subjects' dominant leg. The hip was extended at 130°, the knee bent at approximately 15° of knee flexion, and the leg supported and positioned parallel to the floor. Inflexible straps secured the torso, waist, thigh, and leg. Velcro straps across the toes and dorsum of the foot secured the limb to the dynamometer footplate. The axis of the dynamometer was aligned with the lateral malleolus of the fibula. The short and long muscle length of the dorsiflexors was defined

as the 5° dorsiflexion (DF) joint angle (i.e., 0° is the neutral ankle position) and the 35° plantar flexion (PF) joint angle, respectively. This 40° total ROM represented the maximum range near the plateau region of the torque-length relationship of the dorsiflexors (21, 27, 29). A computer display monitor was placed 2 m in front of the subject that provided visual feedback of ROM and torque.

5.1.4 Experimental Procedures

Transcutaneous electrical stimulation was performed with the anode and cathode electrode positioned over the tibialis anterior, which is the main dorsiflexor muscle (29). Similar to previous studies (4, 21, 29), two custom-made aluminum-foil electrode pads coated in conductive gel were placed longitudinally over the tibialis anterior muscle belly with the medial section of both pads in line with the anterior border of the tibia. At the proximal end, the cathode (4x8 cm) was positioned over the tibial tuberosity, and while the subjects performed a weak contraction at the short length, the anode (2x8 cm) was placed over the visible distal portion of the TA muscle belly. This setup was chosen instead of using a stimulation site over the peroneal nerve near the fibular head, because activation there also activates the peroneal muscles (plantar flexors) contributing to decreased evoked tibialis anterior torque production when the foot is placed in a dorsiflexed position (21, 29). A ground electrode was situated on the patella.

Testing began at the long length with stimulation intensity for the electrically evoked doublets (10 ms interpulse-interval, 50 μs pulse duration) increased incrementally with a Digitmer stimulator (DS7AH, Digitimer Ltd, Welwyn Garden city, Hertfordshire, UK) until a plateau in doublet torque was achieved. Because twitch amplitude is particularly susceptible to low-frequency fatigue (10), 100 Hz doublets were used. Then

stimulation intensity of the doublet was set at 10% above that needed to attain maximum doublet amplitude. These evoked doublets were used to assess voluntary activation using the interpolated twitch technique (12). At this same muscle length a second Digitimer stimulator was used to set the stimulation intensity of the high-frequency tetanic (HFT) train (average 52.8 Hz; two initial pulses with 10 ms interpulse-interval followed by 18 pulses with 20 ms interpulse-interval all generated with a 50 μs pulse duration). At the long (as well as short) muscle length, HFTs were used to assess muscle contractile properties rather than evoked twitches or doublets, because musculotendinous slackness can diminish evoked twitch and doublet contractile properties at the short length (7, 29, 34). To minimize this potential factor, HFT stimulation was chosen to measure evoked contractile rate properties with the expectation that high-frequency stimulation (with an initial 100 Hz doublet) would rapidly increase muscle stiffness and enhance force transmission by the muscle (16) and then reducing the frequency to 50 Hz to minimize high-frequency fatigue and for subject tolerance. The stimulation for the HFT was increased incrementally until a plateau in HFT torque was observed or until the maximum tolerable stimulation intensity was achieved. Typically, HFT torque amplitude was greater than 40% of MVC torque. This was similar to the 42% of MVC value from 100 Hz supramaximal stimulation train (100 pulses, 100 μs pulse duration) reported previously (29), although in my study this was achieved with a lower average stimulation frequency using the HFT (ie., 52.8 Hz). Pilot testing revealed that the stimulation intensities determined at the long length were sufficient at the short length to achieve peak doublet torque and greater than 40% of MVC torque for the HFT.

After five min of rest, baseline (PRE) measures were obtained for MVC torque

and evoked isometric muscle contractile properties at the two muscle lengths. Subjects performed three isometric testing sequences with 5 min of rest provided after each set. Each isometric testing sequence consisted of a 3-5 s duration MVC with an interpolated doublet, then an evoked doublet at rest post-MVC, followed by the HFT train (Fig. 5.1). This sequence was repeated immediately afterwards (~ 5 s later) at the other muscle length (Fig. 5.1). The testing order of muscle lengths was randomized. Subjects maintained the same testing sequence throughout the rest of the experiment (Fig. 5.1). Subjects then performed ~ 10 familiarization dynamic dorsiflexion contractions as fast as possible against a load of 30% of MVC torque (i.e., based on MVC torque at the long length for protocol consistency), with an emphasis on achieving full 40° ROM with each contraction. The contraction load of 30% of MVC torque was programmed into the Biodex dynamometer using the isotonic mode to provide resistance during the shortening contractions. After another five minute rest period, and just prior to the fatiguing task, two maximal-effort dynamic contractions were completed to obtain PRE values for loaded shortening velocity and ROM.

Subjects performed the dynamic fatiguing task, which consisted of repetitive shortening contractions (at 30% of MVC torque), until task failure was attained. Task failure was defined as a 50% reduction in ROM (i.e., from 40° total ROM at PRE to 20° total ROM at POST), in which subjects were verbally encouraged to attempt to reach full 40° ROM as rapidly as possible during each shortening contraction of the fatiguing task. Following the shortening phase of each contraction of the fatiguing task, subjects were

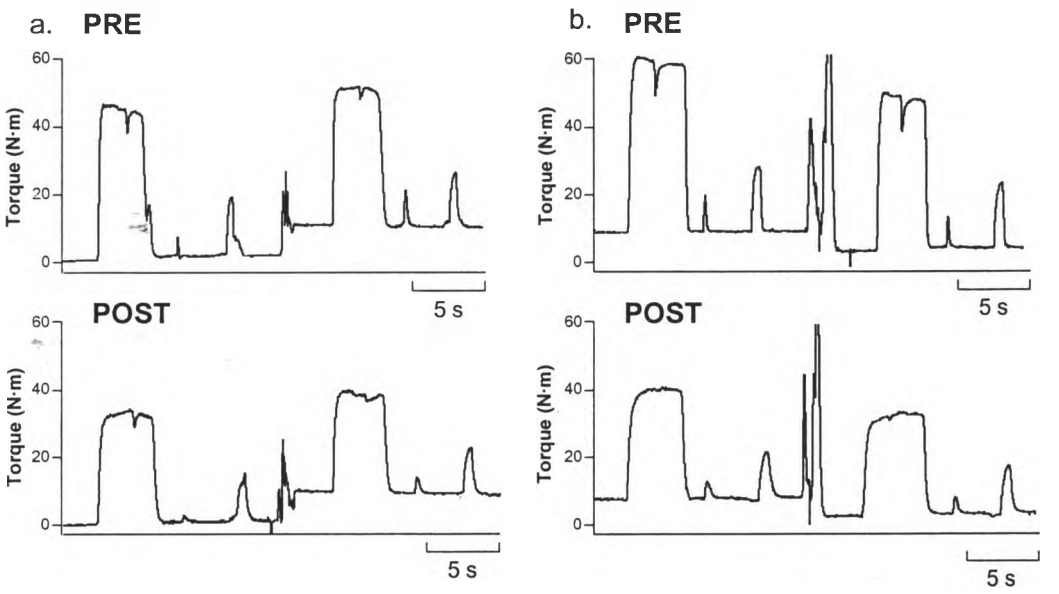


Figure 5.1 Raw data for two subjects performing MVC testing sequences at baseline (PRE) and task failure (POST). **a.** one subject performed MVC testing sequences at the short length immediately followed by tests at the long length, **b.** another subject performed MVC testing sequences at the long length followed by tests at the short length. The spike in torque between MVC testing sequences is an artifact due to the experimenter changing the subject's joint ankle position from a short to a long muscle length or vice versa. The greater baseline torque at the long length is likely attributed to a reduction in the passive stretch of the plantar flexors when the dorsiflexors are lengthened. This was accounted for by resetting the baseline torque to 0 N·m at each muscle length before recording dorsiflexion torque.

instructed to relax and to allow their foot to return passively to the starting position. The duty cycle was approximately one contraction every 1.5s. Measurements were obtained immediately after task failure, and to assess recovery at 0.5, 2, 5, and 10 min after task failure. At each of these time points, two maximum effort shortening contractions were performed, followed immediately by the isometric testing sequences at the short and long muscle length.

5.1.5 Data and Statistical Analyses

All torque, velocity, and joint position data were obtained from the Biodex at a 100 Hz sampling rate, and were converted by a 12-bit A/D converter (CED Model 1401 Plus; Science Park, Cambridge, UK). Spike2 computer software (CED, Science Park, Cambridge, UK) permitted real-time display, inspection, and analysis of all data channels. To measure the time course changes of ROM and shortening velocity during fatigue and recovery, values were obtained at PRE, 25%, 50%, 75% of time to task failure (TTF), at task failure, and during recovery at 0.5, 2, 5, and 10 min after task failure. The ROM was calculated by subtracting the start position from the end position. Shortening velocity was determined from the peak velocities during the contraction. So that the fatiguing task was not disrupted, measurements of MVC, HFT torque, and voluntary activation were not taken during the fatiguing task (at 25%, 50%, and 75% TTF). MVC and HFT torque data were analyzed for peak values at each joint angle. Voluntary activation was calculated as: 1- (a/b) x 100%, where a is the torque of the interpolated response of the doublet at peak torque, and b is the torque of the post-MVC doublet at rest (12). MRTD and MRR of the evoked HFT trains were normalized by dividing the peak rate of change of torque (N·m/s) by peak torque of the HFT tetani

evoked at the same length (N·m). To examine the extent of change in ROM, MVC, shortening velocity, HFT torque, MRTD, and MRR, data for the fatigue and recovery protocol were normalized relative to values at PRE.

Using SPSS software (v.16), a two-factor (muscle length x time) repeated measures analysis of variance (ANOVA) was used to assess the significance of changes between the measures (ROM, MVC, shortening velocity, HFT torque, MRTD and MRR) for the fatigue and recovery protocol. If a statistically significant difference at $p < 0.05$ was achieved, then a Tukey's HSD post-hoc analysis was performed. Because the voluntary activation data was not normally distributed, a Friedman's test was used to compare voluntary activation at the short and long muscle length over time. Mann-Whitney U-tests were used to assess at each muscle length, statistically significant changes in voluntary activation over time. Linear regression analysis was used to compare the relationship between changes in ROM to MVC and shortening velocity. Paired T-tests were employed to compare absolute values at PRE and POST. All tabulated data are presented as means (SD).

5.2 RESULTS

5.2.1 Baseline Measurements

All subjects were able to attain very close to the desired 40° ROM during voluntary shortening contractions at baseline (see Table 5.1). MVC torque was significantly greater (by ~11%) at the short compared to long muscle length, and similarly, evoked HFT peak torque was significantly greater (by ~12%) at the short compared to the long muscle length (Table 5.1). For the twelve subjects, voluntary activation was nearly complete (> 98%) at both muscle lengths (Table 5.1). MRTD was

Measures	PRE	POST
ROM (°)	38.4 (0.9)	20.5 (2.3) *
Peak shortening velocity (°/s)	173.0 (28.7)	57.1 (20.2) *

Joint angle	35° PF (long length)		5° DF (short length)	
	PRE	POST	PRE	POST
MVC torque (N·m)	40.2 (9.5)	28.8 (7.7) *	45.7 (7.8) †	31.3 (5.9) *
Voluntary activation (%)	98.0 (3.1)	97.3 (5.1)	99.0 (1.8)	95.6 (8.2)
HFT torque (N·m)	18.4 (4.6)	13.8 (4.1) *	20.8 (4.3) †	15.2 (4.2) *
HFT MRTD (s^{-1})	6.3 (1.0)	4.7 (0.9) *	6.0 (0.8)	4.2 (0.9) *
HFT MRR (s^{-1})	-7.9 (1.0)	-4.8 (0.9) *	-8.8 (1.0) †	-5.1 (1.1) *

Table 5.1 Neuromuscular properties of the dorsiflexors at baseline (PRE) and at task failure (POST). Plantar flexion (PF) and dorsiflexion (DF) joint angles relative to 0° neutral ankle joint position; joint range of motion (ROM); maximum voluntary isometric contraction (MVC); high-frequency isometric tetani (HFT); normalized maximum rate of torque development (MRTD); normalized maximum rate of relaxation (MRR). Data are presented as mean (SD). * indicates significantly different from PRE, $P < 0.05$, † indicates significantly different between muscle lengths at the same time point, $P < 0.05$.

not significantly different between muscle lengths but MRR was slightly (~9%) faster at the short compared to the long muscle length (Table 5.1).

5.2.2 Fatigue and Recovery

Task failure (50% reduction in ROM) required 33.8 (12.1) repetitive shortening contractions, and time to task failure (TTF) was 48.9 (17.2) s. The decrease in ROM was non-linear with a greater loss during the final portion of the fatiguing task from 75%TTF to task failure (Fig. 5.2). Relative to values at PRE, ROM then recovered rapidly to 90.4 (6.5)% at 0.5 min after task failure. Shortening velocity decreased in a linear manner and more quickly than ROM; velocity was also slower to recover (Fig. 5.2). During the fatiguing task, and relative to values at PRE, shortening velocity significantly decreased to 71.0 (6.7)% at 50%TTF, to 55.0 (6.5)% at 75%TTF, and then eventually decreased to 33.1 (10.5)% at task failure. During the recovery period, shortening velocity rapidly returned toward PRE values at 0.5 min after task failure, achieving 73.2 (18.3)% of PRE, but did not recover fully until 5 min after task failure [to 89.2 (12.7)% of PRE]. There was a strong positive association between relative time course changes in ROM and shortening velocity during the fatiguing task ($R^2 = 0.80, p < 0.05$) although a weaker association was found between ROM and shortening velocity during the recovery period ($R^2 = 0.36, p < 0.05$), likely because ROM recovered more quickly by 0.5 min after task failure compared to shortening velocity at 5 min after task failure (Fig. 5.2).

The reduction in ROM did not appear to be related to length-dependent changes in MVC torque because at task failure the relative reduction in MVC torque at the short and long muscle lengths [to 68.4 (7.0)% and 71.8 (12.0)% of PRE, respectively] was not significantly different between muscle lengths (Fig. 5.3a). At task failure there was a

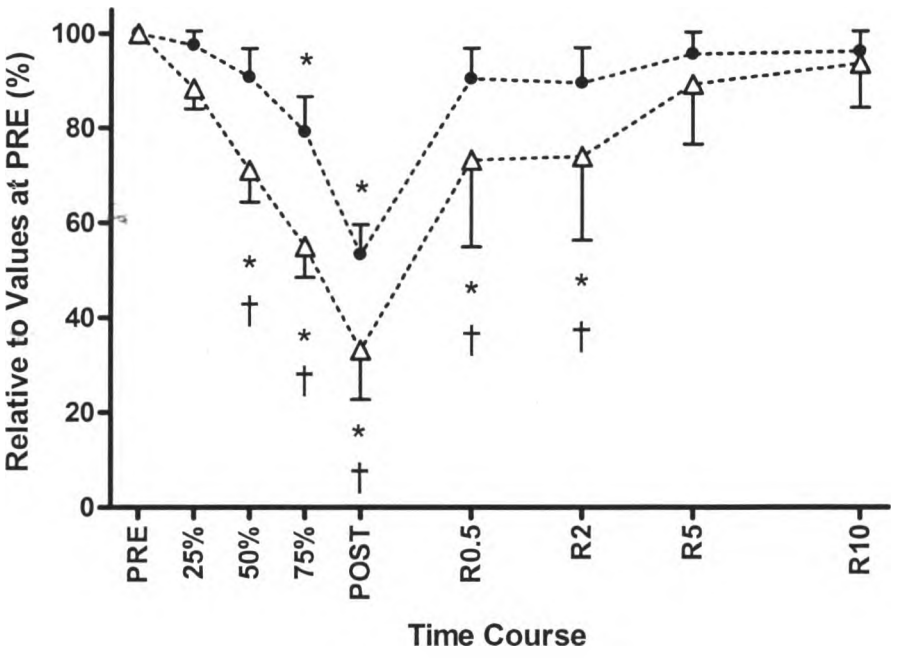
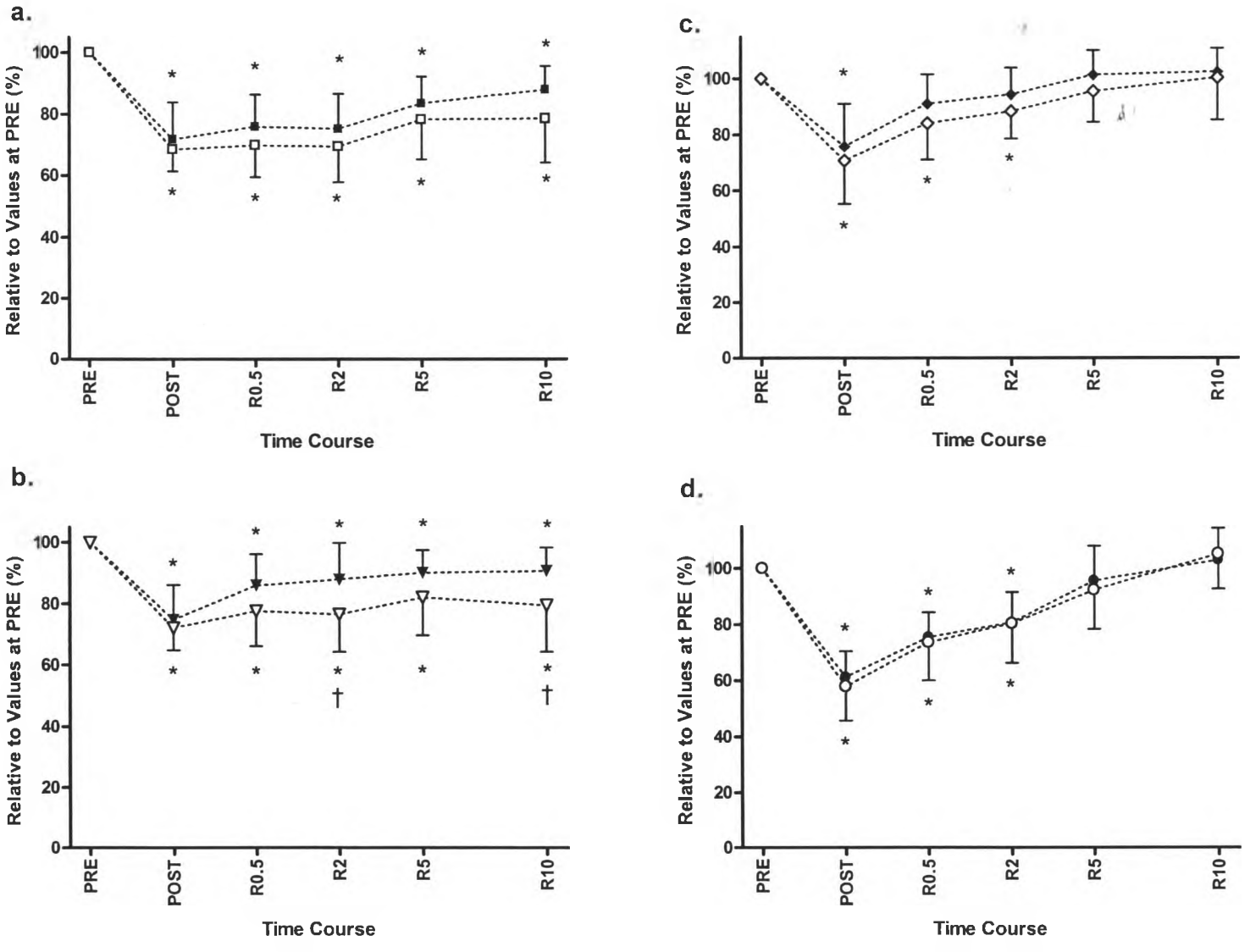


Figure 5.2 Fatigue-induced changes in ROM (filled circle) and loaded shortening velocity (open triangle). The normalized fatigue protocol is represented as PRE, 25-75% time to task failure, POST, and the time course of the recovery protocol by absolute times as R0.5 to R10 (min). Data are presented as means (SD). * indicates significantly different from PRE, $p < 0.05$. † indicates significantly different values between ROM and loaded shortening velocity at the same timepoint, $p < 0.05$.

Figure 5.3 **a.** Fatigue-induced changes in MVC torque, **b.** high-frequency tetanus (HFT) torque, **c.** normalized maximum rate of torque development (MRTD) of the HFT, and **d.** normalized maximum rate of relaxation (MRR) of the HFT at the long (filled symbols) and short muscle length (open symbols). The normalized fatigue protocol is represented as PRE, POST, and the time course of the recovery protocol by absolute times as R0.5 to R10 (min). Data are presented as means (SD). * indicates significantly different from PRE, $p < 0.05$. † indicates significantly different between muscle lengths, $p < 0.05$.


a.
b.
c.
d.
Relative to Values at PRE (%)
Time Course
PRE
POST
R0.5
R2
R5
R10
0
20
40
60
80
100
*
†

small amount of time for recovery (~5s) between tests performed at the two muscle lengths (Fig. 5.1), but post-hoc analysis revealed that the reduction in MVC torque immediately following task failure was not significantly different (~70% and 73% of PRE at the short and long lengths, respectively) in those subjects who first performed testing sequences either at the short or long length. At 10 min after task failure, MVC torque remained depressed and was only 78.5 (14.4)% of PRE at the short length and 87.7 (8.2)% of PRE at the long length. At task failure and during the recovery period, there were weak although significant relationships between time course changes in ROM compared to MVC torque at the short ($R^2 = 0.11, p < 0.05$) and long length ($R^2 = 0.17, p < 0.05$). In three subjects, VA could not be calculated at task failure or during recovery because the post-MVC doublet was not measurable at the short length. Still, for the remaining nine subjects, VA was high at both joint angles at task failure [95.6 (8.2)% at the short length and 97.3 (5.1)% at the long length] and did not change significantly over the duration of the experiment.

Compared with values at PRE, there was no significant difference in the relative reduction in HFT torque at the short and long muscle lengths [to 72.1 (7.3)% and 74.8 (11.3%), respectively] (Fig. 5.3b). However, there was a greater reduction in HFT torque at the short versus long length at 2 min after task failure [76.5 (12.1)% vs. 87.9 (11.9)%, respectively] and 10 min after task failure [79.5 (15.1)% vs. 90.7 (7.7)%, respectively] (Fig. 5.3b). Relative to values at PRE, the reduction in MRTD at task failure to 70.7 (15.4)% at the short length and 75.8 (15.3)% at the long length was not significantly different between muscle lengths (Fig. 5.3c). At the short length, MRTD was slower to significantly recover, requiring 5 min to reach 95.5 (11.0)% of PRE, whereas at the long

length, MRTD significantly recovered at 0.5 min after task failure [to 91.1 (10.4)%] (Fig. 5.3c). At task failure, the reduction in MRR [to 57.9 (12.1)% and 61.2 (9.3)% at the short and long lengths, respectively] was not significantly different between muscle lengths, and significantly recovered at both muscle lengths by 5 min after task failure to 92.2 (13.8)% and 95.5 (12.5)%, respectively (Fig. 5.3d).

5.3 DISCUSSION

Findings from previous studies have suggested that greater fatigue-induced torque loss at short compared to long muscle lengths (20) and fatigue-induced contractile slowing (8, 23) are the main factors contributing to the fatigue-related reduction in ROM. I have systematically explored these ideas, and have expanded on previous studies by investigating voluntary activation, voluntary and evoked torque, and evoked contractile rate properties (i.e., MRTD and MRR) at short and long muscle lengths in the dorsiflexors. Following task failure, defined as a 50% reduction in ROM, voluntary activation was high and stable at both muscle lengths throughout the experiment. Furthermore, I found similar fatigue-related reductions in torque (voluntary and evoked) and contractile slowing (in MRTD and MRR) at both lengths. Thus, the fatigue-induced reduction in dorsiflexion ROM could not be explained by length-dependent alterations in torque or contractile kinetics as suggested in previous papers.

5.3.1 Length-dependency of Fatigue Under Different Tasks

Numerous studies performing isometric contraction tasks have reported a leftward-shift in the torque length relationship due to fatigue, or reduced endurance for isometric contraction tasks performed at the long versus short lengths (11, 15, 22, 24, 31,

33, 35, 37). This reduced endurance at long compared with short lengths following isometric contraction tasks appears to be due to greater excitation-contraction coupling failure at the long length (11, 24). In comparison, a rightward-shift in the torque-length relationship has been reported following repetitive lengthening contractions, and this appears to be caused primarily by muscle damage (5, 6, 14, 36, 42). However, unlike isometric contractions, shortening contractions undergo larger changes in length, and contractions are not performed solely at either a fixed short or long length. Compared with lengthening contractions, extensive muscle damage does not occur following shortening contractions (41). Fatigue appears to be task-dependent, and I believe that my finding of no length-dependent alteration in torque with fatigue is specific to tasks involving fatiguing shortening contractions performed in the dorsiflexors.

In my study, fatigue was induced with repetitive shortening contractions until dorsiflexion ROM decreased by 50% at task failure, which is a similar protocol to an earlier study in the plantar flexors (20). However, in contrast to their findings, I found a similar relative reduction in MVC and HFT torque at both muscle lengths at task failure. Differences between my findings and those reported previously (20) may be due to the different muscle groups and muscle lengths evaluated. Previous literature has suggested that the plantar flexors may be more difficult to activate maximally than other muscles (12), and particularly at the short length because of spinal inhibition from muscle afferents (19, 32). In contrast, my findings agree with several previous studies showing that the dorsiflexors can be well-activated at short or long lengths (4, 13, 34). Because my data shows no length-dependent alteration in MVC torque, and considering in previous findings that at short lengths there were only slightly greater reductions in

torque compared to long lengths (20), this would discredit the idea that the fatigue-induced reduction in ROM is largely explained by greater reductions in MVC torque at the short compared with the long length.

During the recovery period, MVC and HFT torque remained depressed throughout the 10 min time period at both muscle lengths. HFT torque was slightly more depressed (~11%) at the short compared to long length during recovery, but this was only statistically significant at two time periods (i.e., 2 and 10 min after task failure). This length-dependence in HFT torque recovery may be explained by a greater impaired myofibrillar [Ca^{2+}] sensitivity at the short length (26), requiring greater stimulation frequencies than the HFT train to elicit full muscle activation at the short length. It is important to note that the fast recovery of ROM at 0.5 min after task failure could not be explained by the incomplete recovery of torque-generating capacity, in which MVC and HFT torque were blunted and remain depressed throughout the recovery period, and at both muscle lengths (Fig. 5.3a,b).

5.3.2 Contractile Slowing and ROM

In addition to measurements of isometric torque, evoked contractile rate properties (i.e., MRTD and MRR) were assessed at short and long lengths. In unfatigued conditions, and in contrast to previous findings, no difference was found in MRTD at the short and long length. Although previous studies found slower twitch contraction times at shortened compared to longer muscle lengths in the unfatigued dorsiflexors (4, 13, 29), dampened evoked twitch torque properties at shortened lengths could be attributed to both increased musculotendinous slackness (29, 32, 34) and reduced myofibrillar Ca2+ sensitivity at shortened lengths (2, 26). Hence, I chose HFT tetani to measure MRTD and

MRR in the dorsiflexors. Using 200 Hz HFT stimulation in a different muscle, a previous investigation found an ~30% faster normalized MRTD of 200 Hz isometric tetani at a 30° knee angle (short muscle length) compared to 60° or 90° (long) in the human knee extensors (9). However, post-activation potentiation also has a more pronounced affect at short compared to long muscle lengths (25, 34, 35) and can increase the rate of torque development in the human quadriceps muscle even at very high firing frequencies of 400 Hz (25). In my study, length-dependent influences of post-activation potentiation might have been minimized because MRTD was always recorded from an evoked HFT train that immediately proceeded a ~ 3-5s conditioning isometric MVC. I also found a 9% faster MRR at the short compared to long length, which is consistent with previous investigations in the unfatigued state (29). It is unclear why there is a slightly faster MRR at the short length, but I speculate this may be due to an increased stretch of the plantar flexors when the foot is dorsiflexed. This opposing resistance indirectly could facilitate relaxation of the dorsiflexors.

With fatigue, I found no difference in the relative reduction in MRTD and MRR at task failure between muscle lengths; suggesting that fatigue-induced slowing of contractile kinetics is not length-dependent. However, a slower recovery of MRTD at the short length was observed, which might be related to a greater impaired myofibrillar [Ca2+] sensitivity at the short length (26). In the present study, no length-dependent alterations in torque or contractile kinetics were found, but there was a substantial reduction in shortening velocity to 33% of PRE, indicating that shortening velocity was greatly affected by fatigue. Thus, it could be postulated that the reduction in shortening velocity is mainly responsible for the reduction in ROM. Indeed, a strong positive

association was found between time course changes in shortening velocity and ROM during the fatiguing task ($R^2 = 0.80$). Perhaps the fatigue-related reduction in shortening velocity decreases ROM because fatigue-induced contractile slowing may reach a critical threshold that makes it difficult to maintain sufficient limb inertia during shortening. Consequently, joint angular velocity declines to 0 °/s earlier in the ROM with fatigue, and muscle shortening ends at this timepoint when the contraction becomes isometric.

However, it may be important to also explore other possible factors involved in the fatigue-related reduction in ROM. For example, there were differences in fatigue time courses, with a linear reduction in shortening velocity, whereas the reduction in ROM was non-linear (Fig. 5.2). Also, only a moderate association was found in the recovery of ROM and shortening velocity ($R^2 = 0.36$) probably because ROM recovered more rapidly at 0.5 min after task failure compared to the full recovery of shortening velocity at 5 min after task failure. Unfortunately it is not possible to determine from my current findings, or from previous findings, which particular fatigue-related factors are responsible for the non-linear reduction in ROM during the fatiguing task and its rapid full recovery at 0.5 min after task failure.

I can speculate that perhaps, other muscles acting about the ankle joint also may be contributing in the reduction in ROM. In unfatigued conditions, increased co-activation of antagonists at the short muscle length has been suggested to reduce end ROM by counteracting agonist torque output (28). However, it remains uncertain whether co-activation of opposing ankle joint muscles is altered during fatiguing dorsiflexion shortening contractions. In conclusion, I have shown that fatigue-related reductions in ROM cannot be explained by greater fatigue-induced reductions in torque,

or a greater slowing of contractile kinetics at a shortened length. Future investigations should explore the involvement of co-contraction of muscles acting on the ankle joint, and its role on fatigue-related reductions in dorsiflexion ROM.

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CHAPTER 6

GENERAL DISCUSSION AND SUMMARY

6.0 GENERAL DISCUSSION

In the second chapter, I investigated whether the interpolated twitch technique would be affected by methodological error due to musculotendinous slackness that reduces the post-MVC twitch amplitude. In this study, the reduction in post-MVC doublet amplitude at shortened lengths underestimated voluntary activation when it was submaximal (i.e., $\leq$ 80% of MVC). These findings show that it is important to account for length-induced changes in the post-MVC doublet amplitude when calculating voluntary activation using the ITT. Thus, for the subsequent studies in this thesis I assessed voluntary activation primarily at a lengthened muscle position to diminish the influences of musculotendinous slackness, or in Chapter 5, voluntary activation was assessed at both a short and long muscle lengths in a muscle group that could be readily activated maximally at both lengths.

In addition to measurements of MVC torque, I present novel evidence that shortening velocity (Chapters 3, 4, 5) and ROM (Chapter 5) are also important parameters of dynamic contraction task performance. No previous studies have used comparable contraction tasks (i.e., intermittent contractions, 50% of MVC contraction load, blood flow occluded, equivalent reduction in MVC torque at task failure) to evaluate fatigue induced by isometric and dynamic contractions. In Chapter 3, the task involving dynamic contractions had a shorter time to task failure than the isometric contraction task, which is in line with previous findings suggesting that dynamic

contractions are more metabolically taxing to the muscle (7, 8, 10, 13). Furthermore, in the same study I found for both tasks that shortening velocity was more greatly reduced by fatigue compared with MVC torque, and appeared to be a more sensitive measure to indicate the presumed fatigue-induced impairment in crossbridge kinetics (Chapter 3). Although previous studies have compared fatigue in different muscles using isometric contraction tasks, the study reported in Chapter 4 compared fatigue in two different muscles during velocity-dependent contraction tasks. The results confirmed the finding from Chapter 3 that fatigue during velocity-dependent tasks in different muscles is most likely associated with peripheral fatigue, in which the slowing of contractile properties from metabolite accumulation was presumed largely responsible for the reduction in loaded shortening velocity in both the triceps brachii and soleus. Although in Chapter 4, there were no differences in endurance times, approximately two-fold greater total work was performed by the triceps brachii compared with the soleus during the dynamic contraction fatigue task. This suggested a greater endurance capacity during dynamic contraction tasks in the triceps brachii. Besides differences in fibre-type proportion between muscles, I speculated that joint biomechanics may also play an important role in influencing the endurance capacity of these different muscles during high-velocity contraction tasks. In line with previous findings from isometric contraction tasks, MVC torque in the soleus was better preserved compared with the triceps brachii at dynamic contraction task failure, but this did not translate into greater endurance in the soleus during the velocity-dependent contraction task. Also, post-activation potentiation did not appear to offset the fatigue-related reduction in shortening velocity in the highly-potentiating triceps brachii compared with the minimally-potentiated soleus. In Chapter

5, new findings were presented indicating that fatigue-induced reductions in torque, and fatigue-induced alterations in contractile kinetics, are not length-dependent following repetitive shortening contractions in the dorsiflexors. Consequently, the fatigue-related reduction in ROM was not related to greater torque loss or contractile slowing at the short compared with the long length. However, the fatigue-related reduction in shortening velocity largely explained the fatigue-related reduction in ROM ($R^2 = 0.80$).

6.1 LIMITATIONS

In Chapter 2, we did not make direct measurements of triceps brachii musculotendinous slackness using ultrasound imaging but the findings are consistent with musculotendinous length changes considered responsible for the reduction in evoked twitch torque at short compared with long muscle lengths (12). Also, the interpolated twitch technique is based on comparing the interpolated twitch torque response as a ratio to a twitch torque evoked in a relaxed muscle. However, an interpolated twitch will result in a change in shortening velocity that is difficult to interpret when applied to velocity-dependent contractions. Thus, although in Chapters 3, 4, and 5, voluntary activation was assessed during isometric contractions following fatigue and recovery from repetitive dynamic contractions, voluntary activation failure *during* the shortening contractions could not be ruled out completely.

There were also challenges in trying to design experimental paradigms to compare fatigue resulting from isometric and dynamic contraction tasks. Some muscle shortening will occur at the initiation of an isometric contraction to take up musculotendinous slackness. Thus, isometric contractions are not strictly isometric, although it is unlikely

that this amount of muscle shortening is equivalent to shortening during a dynamic contraction through a large joint ROM. Furthermore, potential differences in muscle perfusion were controlled between contraction-types by occluding blood flow to the tibialis anterior muscle. However, muscle energetics are altered with blood occlusion (11), and thus these findings may not be reflective of muscle metabolism during a fatiguing task with intact blood flow to the muscle. Also, in Chapter 3, the rapid restoration of MVC torque following both isometric and dynamic contraction task failure is likely exaggerated as a result of hyperemia following deflation of the blood pressure cuff following task failure. For example, in Chapters 4 and 5, and in my previous study (4), the fatigue-induced reduction in MVC torque after dynamic contraction task failure was often small and it remained blunted throughout the recovery period.

Comparing fatigue in different muscle groups is an interesting experimental approach that utilizes the different physiological properties of muscles to determine their influence on muscle fatigue. However, differences in torque-generating capacity, shortening velocity, and joint ROM between physiologically different muscles (and between biomechanically different limb joints) also present challenges when attempting to design an equivocal fatigue paradigm. As an example, few studies in the neuromuscular fatigue literature address the issue of joint and muscle mechanics which potentially can affect differences in fatigue between muscles during a dynamic contraction task. I suggested in Chapter 4 that perhaps a more effective biomechanical lever-type for the elbow joint, compared with the ankle joint may be contributing to the greater total work performed by the triceps brachii compared with the soleus during the dynamic contraction task.

In Chapter 5, the main focus of the study was to determine whether there were muscle length-dependent alterations in torque and contractile slowing in the tibialis anterior muscle following repetitive shortening contractions. However, it was evident that fatigue in the agonist muscle was not length-dependent. The fatigue-related reduction in shortening velocity largely explained the fatigue-induced reduction in dorsiflexion ROM, but perhaps the reduction in dorsiflexion ROM may also be explained by active and passive influences of the antagonistic plantar flexors. However, these were not assessed in my study.

In addition, there were several general limitations of performing experiments using high velocity shortening contractions, and some of these are due to limitations in current techniques used for assessing neuromuscular function during dynamic contractions. For example, there are known difficulties in examining motor unit activity during dynamic contractions (6). Because surface electrodes to record EMG are in a fixed location on the skin, but the muscle moves in relation to the skin, and thus surface EMG electrodes may not track motor unit activity from the same motor units during shortening (and lengthening) contractions. The potential use of indwelling needles or wires was not possible because the EMG signal is obscured by large and rapid muscle length changes, which moves the recording location of the needle or wire. The study in Chapter 3 equated both the isometric and dynamic contraction tasks for contraction pace, contraction load, task failure, and eliminated any differences in blood perfusion, but it is possible that differences in motor unit activation strategies between contraction-types, which cannot be differentiated using surface EMG measures, could have affected the outcome.

While many of the voluntary and electrically stimulated measures were used to infer fatigue-induced changes in neuromuscular transmission failure, muscle metabolites or SR Ca^{2+} handling, no direct measurements were obtained. Consequently the specific mechanisms cannot be confirmed in my experiments. This does not preclude the sensible interpretation of some of these results, but highlights that future studies should strive to utilize measures of metabolism and neural control during these tasks in humans. Furthermore, the cellular mechanisms related to some measurements remain unclear. As one example, slowing in relaxation of an isometric tetanus (e.g., MRR) has been shown in a mouse model to be dependent primarily on crossbridge kinetics, whereas in a frog model it is mainly Ca^{2+} dependent (16). Thus, it is still debated which metabolic or Ca^{2+} dependent mechanisms are responsible for the fatigue-induced changes in evoked contractile properties in humans, but likely there is a complicated interaction involving several fatigue-related factors.

6.2 FUTURE DIRECTIONS

Several questions that remain unanswered in this thesis pertain to limitations in techniques to assess neuromuscular function during dynamic contractions. Techniques have not yet been developed to evaluate motor unit recruitment and discharge rate strategies during dynamic contractions at moderate to rapid shortening velocities. Also, future investigations are needed to determine a technique to assess voluntary activation during velocity-dependent contractions at various velocities of shortening.

Future studies are also needed to understand further the peripheral fatigue-related factors involved in power loss. It has been suggested that, depending on the contraction

intensity of the fatiguing task (i.e., relative to MVC torque), the fatigue-related contributions of torque and velocity to power loss will vary (1, 2). In most of my experiments, the contraction intensities were at or below 50% of MVC torque, in which shortening velocity appeared to be the primary contributor to power loss. However, it is possible that the fatigue-related contribution of torque will play a greater role in the reduction in power output at higher contraction intensities (1).

In Chapter 4, I suggested that greater biomechanical efficiency of elbow extension movement compared with plantar flexion may be responsible for greater endurance of the triceps brachii. Future studies are needed to explore further how the triceps brachii is capable of performing greater total work during a velocity-dependent contraction task compared with the soleus. Perhaps because the soleus acts as a second-class lever (9, 14), a greater magnitude of muscle shortening is required by the soleus compared with triceps brachii to move the respective segments through the required ROM. Increased muscle shortening raises the metabolic cost of muscle contraction (7, 8, 10, 13), and this could be responsible for the smaller total work performed by the soleus. In vivo real-time metabolic measurements could help determine whether there is a lower energetic cost in performing dynamic contractions in the triceps brachii compared with the soleus.

In Chapter 5, fatigue was not length-dependent and could not explain the reduction in ROM following fatigue. I speculate that ankle muscles other than the dorsiflexors may also be playing an important role in controlling ankle joint ROM during fatiguing dynamic contractions. For example, I found in Chapter 5 that at the short length in the dorsiflexors, the plantar flexors were stretched, which could act as a counteracting resistance against the dorsiflexors. Either due to passive plantar flexion tension alone, or

active tension from co-contraction, the antagonistic plantar flexors could oppose dorsiflexion torque particularly at the short length during a shortening contraction. However, the extent to which reduced dorsiflexion ROM is related to plantar flexor muscle-tendon function is not known. Furthermore, shortening-induced torque depression following voluntary muscle shortening was shown previously to reduce torque production at short muscle lengths in the dorsiflexors (15). Shortening-induced torque depression decreases with decreased shortening magnitude (3) and is not related to fatigue (5), and so this becomes less of a factor as ROM is reduced with fatigue. However, both antagonistic torque (active or passive) in addition to shortening-induced torque depression could drastically steepen the torque-length relationship for the dorsiflexors during a shortening contraction. This could suggest that the isometric torque-length relationship overestimates the torque-generating capacity of the dorsiflexors at the short length during muscle shortening. It is apparent that continued investigation is needed to identify factors that may be involved in the fatigue-induced reduction in dorsiflexion ROM during active shortening.

6.3 SUMMARY

In addition to MVC torque loss, fatigue-related reductions in shortening velocity and joint ROM are also important factors that contribute greatly to the impairment in muscle shortening capacity during dynamic contraction tasks. Furthermore, with different time courses for the reduction in torque, shortening velocity, and ROM, it appears that each measure is affected somewhat differently by fatigue. My findings have shown that shortening velocity is greatly reduced following dynamic contraction task

failure regardless of the muscle group being tested. My findings have also shown that shortening velocity recovers rapidly after task failure, and this seems to be related to the restoration of muscle contractile kinetics. Furthermore, I have shown that the fatigue-related reduction in ROM cannot be explained by length-dependent alterations in voluntary activation, torque, or contractile kinetics. Rather, the fatigue-related reduction in ROM was most strongly related to the fatigue-induced reduction in shortening velocity.

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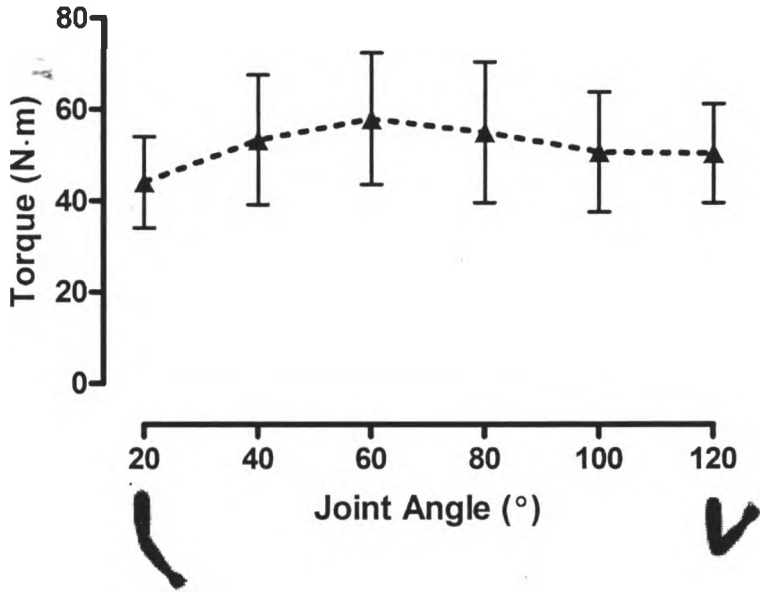
APPENDIX A



Torque-length relationship of the elbow extensors. Complete elbow extension is at 0°. These are unpublished data of Arthur J. Cheng collected from thirteen young men [26.0 (3.8) y, 178.8 (7.6) cm, 78.4 (7.6) kg]. Repeated measures ANOVA of torque x joint angle revealed no significant differences in torque across all joint angles ($p > 0.05$). Data are presented as means and standard deviations.

APPENDIX B

Ethics Approval Notice

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The University of Western Ontario
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Website www.uwo.ca/research/ethics

Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr C.L. Rice
Review Number: 10127E
Revision Number:
Protocol Title: The Effect of Velocity on Power During Isotonic Contractions in Young Men and Women
Department and Institution: Kinesiology, University of Western Ontario
Sponsor: NSERC
Approval Date: 28-Nov-03
End Date: 31-Dec-07
Documents Reviewed and Approved: UWO Protocol, Letter of Information & Consent, Advertisement
Documents Received for Information:

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Karen Kueneman, BA (Hons), Ethics Officer HSREB (Expedited)

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Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr. C.L. Rice

Review Number: 10569 **Revision Number:** 3

Review Date: December 13, 2007 **Review Level:** Expedited

Protocol Title: Age-related changes in muscular endurance and oxygenation during voluntary contractions and electrical stimulation

Department and Institution: Kinesiology, St. Joseph's Health Care London

Sponsor:

Ethics Approval Date: February 27, 2008 **Expiry Date:** March 31, 2009

Documents Reviewed and Approved: addition of co-investigators, revised study methodology, revised sample size, revised study advertisement, revised Letter of Information and Consent

Documents Received for Information:

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10. The failure of either party to enforce any term or condition of this Agreement shall not constitute a waiver of either party's right to enforce each and every term and condition of this Agreement. No breach under this agreement shall be deemed waived or excused by either party unless such waiver or consent is in writing signed by the party granting such waiver or consent. The waiver by or consent of a party to a breach of any provision of this Agreement shall not operate or be construed as a waiver of or consent to any other or subsequent breach by such other party.

11. This Agreement may not be assigned (including by operation of law or otherwise) by you without WILEY's prior written consent.

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