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Voluntary and Evoked Contractile Properties of Trained, Untrained, and Previously Immobilized Subjects Before and Following Fatigue

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A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfilment of the requirements for the degree of Doctorate in Rehabilitation Science

Preface

Candidates have the option of including, as part of the thesis, the text of one or more papers submitted or to be submitted for publication, or the clearly-duplicated text of one or more published papers. These texts must be bound as an integral part of the thesis.

If this option is chosen, connecting texts that provide logical bridges between the different papers are mandatory. The thesis must be written in such a way that it is more than a mere collection of manuscripts; in other words; results of a series of papers must be integrated. Title and reference styles in the manuscripts reflect the requirements of the journal of publication.

The thesis must still conform to all other requirements of the "Guidelines for Thesis Preparation". The thesis must include a: Table of Contents, an abstract in English and French, an introduction which clearly states the rationale and objectives of the study, a comprehensive review of the literature, a final conclusion and summary, and a thorough bibliography or reference list.

Additional material must be provided where appropriate and in sufficient detail to allow a clear and precise judgement to be made of the importance of the originality of the research reported in the thesis.

In the case of manuscripts co-authored by the candidate and others, the candidate is required to make an explicit statement in the thesis as to who contributed to such work and to what extent. Supervisors must attest to the accuracy of such statements at the doctoral oral defence. Since the task of the examiners is made more difficult in these cases, it is in the candidates interest to make perfectly clear the responsibilities of all the authors of the co-authored papers.

Original Contributions

This thesis attempts to provide original contributions to the areas of rehabilitation, exercise and muscle physiology. New data and interpretations are provided in the following areas:

a) an examination of the validity and reliability of the interpolated twitch technique (ITT) for determining muscle activation (interpolated twitch ratio and second order polynomial) with the quadriceps and plantarflexors at different joint angles, contraction intensities and modes of evoked stimulation (single, doublet, triplet),

b) an examination of the validity of comparing the interpolated twitch ratio and second order polynomials before and following fatigue,

c) similarities in the muscle activation: voluntary force relationship between healthy and previously immobilized individuals,

d) the relationship between the effects of swelling and pain on muscle activation (as measured with the interpolated twitch technique) in previously immobilized individuals (regression equations),

e) differences in the voluntary and evoked contractile properties of previously immobilized ankle fracture patients before and following fatigue,

f) differences in the voluntary and evoked contractile properties of internally-fixated and non-fixated ankle fracture patients before and following fatigue,

g) the effect of fatigue duration upon voluntary and evoked contractile properties,

h) an examination of trained state differences in muscle activation (ITT) and antagonist electromyographic activity following fatigue

Acknowledgements

Although this thesis attempts to provide original knowledge to the field of science, it would not have been possible without the rigorous and intriguing research of others. The investigation of muscle activation differences between different populations using the interpolated twitch technique would not have been conceived without the pioneering work of researchers such as Merton, Belanger, McComas and others. Fatigue differences in neural and contractile properties have been extensively investigated by Brenda Bigland-Ritchie whose work has provided much of the foundation of fatigue-related knowledge. The application of neuromuscular testing techniques to previously immobilized patient populations has been well established by the laboratory of Karl Hainaut and Jacques Duchateau. Similarly, Digby Sale's extensive research on neural and muscular adaptations with strength training provided the theoretical basis for much of the trained state research found in this thesis. Philip Gardiner provided important feedback and suggestions for all the papers. Finally Diane St-Pierre as my advisor, provided invaluable help and guidance in the conception and construction of this thesis.

Diane St-Pierre appears as co-author of all papers in the thesis. As the advisor she provided assistance in developing the studies, validating laboratory techniques, editing the manuscripts and acting as a volunteer subject. A portion of Diana Perez's data on the pre-fatigue reliability of quadriceps muscle activation was integrated into the methodology paper (Muscle Inactivation: An Assessment of the Interpolated Twitch Technique) and thus she appears as a co-author.

Other faculty members at McGill University who have provided assistance in the completion of this thesis by lending equipment and conjecture are Patricia McKinley, Charles Rice and Riccardo Torres-Moreno. Technical assistance has been provided by Michael Dispirito,

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Finally without the emotional support of family members (my father George, my late mother Mary, and my sister Barbara), the drive to finish this thesis may not have been possible. I would like to dedicate this thesis to my father: George John Behm, who dedicated his life to his family.

Table of Contents

Abstract

A series of studies were conducted to investigate differences in voluntary and evoked contractile properties of trained, untrained and previously immobilized ankle fracture subjects before and following fatigue. Measurements included twitch contractile properties, maximal voluntary contractions (MVC), electromyography (EMG) and muscle activation (interpolated twitch (IT) ratio and second order polynomials). Although second order polynomials were found to be a more precise measurement of muscle activation (~ 6% error) than the IT ratio (superimposed evoked torque / potentiated resting evoked torque), both techniques provided a general estimate of muscle activation before and following fatigue. Prior to fatigue, trained plantarflexor subjects exerted greater force (17.5%) and had significantly less antagonist activity (41.3%) than untrained subjects. Following fatigue, untrained subjects experienced greater deficits in excitation-contraction coupling and increased antagonist activity (58%) contributing to their tendency for increased fatiguability. The lower force output (45.8%) and muscle activation (20.4%) of previously immobilized non-fixated ankle fractures did not result in greater fatiguability than controls. Disuse adaptations such as potentiation of the muscle action potentials and twitch torque helped to maintain the fatiguing contractions. However the more severely fractured ankles of the internally-fixated subjects experienced increased agonist (13%) and antagonist (8.5%) EMG activity and similar changes in muscle activation and evoked contractile properties while performing fewer contractions. This would suggest that injuries of greater severity may result in an intrinsically more fatiguable muscle. To determine whether the findings could be generalized to other muscles and fatigue protocols, various contraction intensities were used to elicit short and long duration fatiguing contractions in the plantarflexors and quadriceps.

Muscle activation, and twitch half relaxation time were duration dependent with the greatest decreases occurring with long duration fatigue protocols. Twitch torque and time to peak twitch torque were muscle dependent with greatest changes occurring with the quadriceps. Muscle action potentials were affected by both duration and type of muscle.

Résumé

Une série d'études, pré et post-fatigue, a été entreprise pour élucider les différences dans les propriétés contractiles volontaires et évoquées, entre sujets entraînés,sédentaires et immobilisés pour une fracture de la cheville. Les mesures comprenaient les propriétés contractiles de la secousse, la force volontaire maximale (MVC), l'électromyographie (EMG) et l'activation musculaire, telle que mesurée par la technique de l'interpolation de la secousse. Bien que les polynômes du deuxième ordre ont été établis comme une mesure plus précise de l'activation musculaire (approximativement 6% erreur) que le rapport entre la torque evoquée interpolée et la torque évoquée au repos (torque potentialisée), les deux techniques ont fourni une estimation générale de l'activation musculaire avant et après la fatigue. Avant la fatigue, les sujets entraînés étaient caractérisés par des fléchisseurs plantaires plus forts (17.5%) et une moins grande activité des antagonistes (41.3%) que les sujets sédentaires. Suite au protocole de fatigue, les sujets sédentaires ont subi des déficits plus grands dans le couplage excitation-contraction et une augmentation plus importante de l'EMG des antagonistes (58%) que les sujets entraînés. Ces deux facteurs ont contribué à la tendance des sujets sédentaires de se fatiguer plus aisément. Pour les sujets immobilisés pour une fracture simple, même si la force (45.8%) et l'activation musculaire (20.4%) étaient inférieures à celles des sujets contrôles, le temps de fatigue était similaire. Des adaptations dans le potentiel d'action de la membrane musculaire ainsi que dans le couplage excitation-contraction ont aidé a maintenir l'endurance du muscle. Cependant, les sujets, dont la cheville avait subi une frature plus sévère nécessitant une fixation interne, démontraient une plus grande fatigue musculaire. Ceci malgré un EMG plus élevé des agonistes (13%) et antagonistes (8.5%) et des changements similaires dans l'activation musculaire et les propriétés contractiles evoquées, suggérant que les blessures plus sévères ont résulté en un muscle intrinsèquement plus fatiguable. Pour déterminer si ces découvertes pouvaient être généralisées à d'autres muscles et protocoles de fatigue, des contractions de diverses intensités ont été utilisées pour produire des protocoles de fatigue de courtes et longues durées avec les fléchisseurs plantaires et le quadriceps. L'ampleur des changements post-fatigue dans l'activation musculaire, et le temps de demi-relaxation de la secousse étaient dépendants de la durée du protocole de fatigue. Les changements post-fatigue de la torque produite lors d'une secousse et de son temps de contraction dépendaient sur le muscle, les changements étant plus marqués avec le quadriceps. Par contre, les changements post-fatigue dans le potentiel d'action de la membrane musculaire étaient influencés par la durée du protocol et le muscle étudié.

Introduction

Strength training can result in dramatic increases in force output, electromyographic (EMG) activity, reflex potentiation and synchronization of motor units (see reviews: Behm 1995, Jones et al. 1989, Komi 1986, Sale 1988). Although increases in these components have been attributed to an increased central or neural drive (Moritani and deVries 1979, Sale 1988), some studies have indicated that full muscle activation was possible even in untrained individuals (Belanger and McComas 1981, Bellemare et al. 1983, Gandevia and McKenzie 1988, Rutherford et al. 1986). However, other studies have reported differing results, showing that not all or even none of their untrained subjects could fully activate (Dowling et al. 1994, Lloyd et al. 1991, Norregard et al. 1994, Strojnik 1995). Contradictory results could be attributed to methodological differences which must be addressed. Muscle activation may differ dependent upon the muscle tested, joint angle, type of stimulation, and testing protocol.

Athletes are often able to maintain a high calibre of performance after a considerable duration of activity in comparison to the dwindling or impaired performance of the untrained. Insignificant differences in muscle activation (with the interpolated twitch technique {ITT}) and evoked contractile properties under resting conditions may result in dramatic differences under fatigued conditions.The similarities in muscle activation and twitch contractile properties between trained and untrained individuals pre-fatigue have not been investigated following fatigue. However differences in pre- and post-fatigue activation can only be investigated if the mechanisms underlying the ITT are reliable. Therefore an investigation of pre-and post-fatigue trained state differences must include an inquiry into the pre- and post-fatigue reliability and validity of the ITT.

Contrary to the trained state, disused muscle experiences a wide range of decrements including decreased force output, muscle activation, enzymatic activity and atrophy (see reviews; Behm 1993, St-Pierre and Gardiner 1987). Paradoxically, the variety of decrements associated with disuse have been reported to not significantly affect the relative rate of force loss (Duchateau and Hainaut 1987, Fuglevand et al. 1995, Halkjaer-Kristensen and Ingemman-Hansen 1985, Ingemman-Hansen and Halkjaer-Kristensen 1983, White et al. 1984). Similar fatigue indexes between control and previously immobilized individuals might be attributed to the disuse-induced decreases in muscle activation. Fatigue protocols examining control and disuse subjects may have compared maximal and submaximal contractions. Thus it would be important to ensure that all subjects were working at similar intensities. The disuse mechanisms which regulate both an impaired absolute force output and a relatively unperturbed rate of force loss with fatigue have not been fully investigated.

The objective of this thesis is to investigate changes in voluntary and evoked contractile properties in trained, untrained and previously immobilized subjects before and following fatigue, in order to elucidate some of the mechanisms underlying the similarities and differences between the groups.

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The Effects of Strength Training and Disuse on the Mechanisms of Fatigue: A Review

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Summary

Increases in force, EMG, reflex potentiation, muscle action potential amplitude and protein synthesis occur with strength training. Training-induced increases in the efficiency of the neuromuscular system and capacity of the muscle to generate force result in an improved ability to cope with a submaximal load. There is also some evidence of improved fatigue resistance with maximal contractions which could be attributed to a prolongation of membrane excitation or decreased antagonist activity with training.

On the other hand, although a variety of factors including strength are diminished with disuse, a number of studies have demonstrated no significant difference in the rate of fatigue with maximal contractions (fatigue index) between trained, untrained and disused muscle. Equivalent control and disuse fatigue indexes in some studies might be attributed to decreased muscle activation resulting in a comparison of maximal (control) and submaximal efforts (disuse). Furthermore, increases in the duration of muscle membrane electrical propagation with disuse may increase the quantity of Ca^{++} released, augmenting force production. In addition, the smaller volume of disused muscle may allow a more efficient diffusion of oxygen and energy substrates in comparison to a hypertrophied muscle.

Introduction

From a functional perspective, strength and fatigue resistance are important as they are necessary for both athletic performance and habitual daily activities such as walking and stair climbing. Fatigue definitions may differ depending on the task. Fatigue associated with repeated high intensity or maximal contractions could be defined as a transient decrease in working capacity (Asmussen 1979), loss of force output leading to reduced performance (Fitts and Metzger 1993) or a decline in the force-generating capacity of the muscle (Degens and Veerkamp 1994). Fatigue also may be experienced during prolonged submaximal intensity contractions without an apparent decrement in the targeted force. This type of fatigue may be defined as an acute impairment of performance that includes an increase in the perceived effort necessary to exert a desired force and an eventual inability to produce this force (Enoka and Stuart 1992). The changes associated with maximal and submaximal fatigue protocols can differ in their response to training or disuse.

Strength training can result in dramatic increases in muscle force output, cross-sectional area, electromyographic (EMG) activity, reflex potentiation, synchronization of motor unit impulses as well as changes in motor control such as alterations in the contractions of agonist, antagonist, stabilizer and synergistic muscles (Behm 1995, Jones et al. 1989, Komi 1986, Sale 1988). Conversely, disused muscle experiences atrophy, decreases in strength, range of motion, enzymatic activity and capacity to activate the muscle (Appell 1990, Behm 1993, Booth 1982, St-Pierre and Gardiner 1987). Fatigue adaptations do not parallel the myriad of improvements and decrements associated with strength training and disuse. Surprisingly, differences in the fatigue index (rate of force loss with maximal contractions) between trained, untrained and disused

muscles have been shown to be negligible in some studies. Conversley, training has been shown to enhance the fatigue resistance to submaximal contractions (Duchateau and Hainaut 1984b, Shaver 1964), while immobilization has been reported to have no significant detrimental effect with fatigue-related submaximal contractions (Fuglevand et al. 1995, Snyder-Mackler et al. 1993).

The objective of this paper is to review neural, muscular and motor control strength training and disuse adaptations and compare these adaptations to the mechanisms underlying fatigue. This exercise will highlight alterations in the fatigue index and submaximal fatigue protocols following strength training and disuse. The interaction of overload training and disuse on fatigue-related metabolic alterations has been reviewed previously by Degens and Veerkemp (1994). Since other review papers have also explored biochemical changes with fatigue (Edwards 1986, Fitts and Metzger 1993, Newsholme et al. 1992), this paper will concentrate on neural and muscle contractile properties.

Fatigue Mechanisms in Healthy Controls

Mechanisms underlying fatigue can be broadly defined as central (neural) or peripheral (muscle) factors. Neural factors affecting the degree of fatigue can be associated with any input that affects motoneurone functioning and their activation of corresponding muscle fibres. Fatigue also can adversely affect peripheral factors such as muscle fibre excitation, excitation-contraction coupling (E-C coupling) and cross-bridge cycling. Particular fatigue protocols (submaximal vs. maximal, intermittent vs. sustained) can have a predominantly greater effect upon specific neural or muscular components (Tables 1 and 2).

Central Factors:

Muscle Activation: Studies utilizing the interpolated twitch technique (ITT) to examine

muscle activation following fatigue have reported either no or some evidence of fatigue-related muscle inactivation. McKenzie and Gandevia (1991) indicated that their subjects on average failed to activate 4-10% of their elbow flexors after intermittent MVCs. A similar study by the same laboratory (McKenzie et al. 1992), reported decrements in muscle activation for the elbow flexors and diaphragm of approximately 17% and 8% respectively. Research from our laboratory using a submaximal, intermittent, isometric, fatigue protocol revealed similar fatigue-related decreases (10%) in plantarflexor activation (Behm and St-Pierre, under review). Although Bigland-Ritchie et al. (1986b) could not conclusively distinguish superimposed twitches following a submaximal intermittent fatigue protocol of the soleus, the 33% disparity in force between 50 Hz stimulation and MVC indicated a reduced capacity to fully activate. Newham et al (1991) utilized superimposed tetanic contractions following isometric and isokinetic (20^0/s) fatigue and reported decreases in activation of 36.4% and 28.8% respectively. Vollestad et al. (1988) could not find evidence of quadriceps or adductor pollicis inactivation during their intermittent submaximal fatigue protocol. Their research corresponds to Merton's (1954) classic study, who reported no superimposed torque following a sustained maximal fatigue protocol of the adductor pollicis and thus proposed the site of fatigue to be peripheral. Differences in the fatigue tasks (MVC versus submaximal) and/or analysis may explain the divergent muscle activation findings. The analysis of the ITT in some studies had a low signal to noise ratio since only single twitches were superimposed over a MVC without further amplification of the superimposed signal. This may have obscured possible superimposed torques which could have indicated muscle inactivation during fatigue. Fatigue-related decreases in the activation of muscle fibres would contribute to the inability to sustain a maximal or prescribed force.

Muscle activation not only represents the recruitment of motor units but their firing frequency as well (Sale 1988). In order to maintain the prescribed force of a submaximal fatigue protocol, additional motor units are recruited, and in the early stages of fatigue, firing rates increase (Dorfman et al. 1990). However, motor units decrease their firing frequency with fatiguing MVCs (Bigland-Ritchie et al. 1983, Grimby et al. 1981), prolonged submaximal contractions held to maximal intensity (Petrofsky and Philips 1985) as well as ramp and hold (Enoka et al. 1989) contractions. This decrease occurs even though EMG activity increases with submaximal fatiguing contractions, suggesting a muscle afferent reflex inhibition of motor unit firing rates (Garland et al. 1994, Woods et al. 1987). A number of studies have illustrated greater decreases in force output with evoked rather than voluntary stimulation (Bigland-Ritchie et al. 1979, Moritani et al. 1985). The non-modulating evoked signal does not allow for any physiological adaptations (reflex inhibition) to the fatigue-related impaired functioning of the muscle. Rather than acting as an impairment, the reduction in motor unit firing frequency has been suggested as a strategy for sustaining force output during fatiguing contractions (Marsden et al. 1983). Studies have reported an improved maintenance of force output when stimulation frequency was progressively decreased during evoked fatiguing contractions (Binder-MacLeod and Guerin 1990, Jones et al. 1979, Bigland-Ritchie et al. 1979). It would seem more effective to decrease firing rates to preserve the functioning of a motor unit, since a decrease of 1 impulse/s results in a force decrement 10 times less than the derecruitment of a single motor unit (Person and Kudina 1972). "Muscle wisdom" or the decrease in motor unit firing frequency with fatigue may be a defence mechanism to prevent impairments in membrane excitation which would subsequently affect the activation and release of Ca^{++} from the sarcoplasmic reticulum

(SR)(Marsden et al. 1983, Woods et al. 1987). The inability of the muscle to cope with high firing frequencies is further substantiated by the depression in force of non-fatigued muscle subjected to high frequencies of evoked stimulation (Bigland and Lippold 1954). Thus the muscle slows neural input to compensate for its impaired functioning and provide a better match between the rate at which Ca^{++} can be released and the rate of stimulation.

Peripheral Factors:

Electromyography The EMG signal is a diverse asynchronous signal representing changes in factors such as motor unit recruitment, firing frequency, synchronization, conduction velocity and membrane excitation (deLuca 1979, deVries 1968, Perry and Bekey 1981). Increases in EMG activity are reported with submaximal fatiguing protocols (Bigland-Ritchie et al. 1986a, Krogh-Lund 1993, Maton and Gamet 1989, Moritani et al. 1982, Vitissalo and Komi 1977). Loscher et al. (1994) discovered that the gastrocnemius had greater increases in EMG activity than the soleus in the first half of their 60 s submaximal fatigue protocol indicating muscle specific differences. Subsequently EMG activity decreases if submaximal fatiguing contractions approach maximal intensity (Bigland-Ritchie et al. 1986a, Dolmage and Cafarelli 1991, Duchateau and Hainaut 1993, Petrofsky and Philips 1985).

Maximal fatigue protocols result in decreases in EMG shortly following the commencement of both isometric (Bigland-Ritchie et al. 1983, Krogh-Lund and Jorgensen 1991, Moritani et al. 1986, Stephens and Taylor 1972) and isokinetic exercise (Nilsson et al. 1977). The drop in EMG occurs even after fatiguing electrical stimulation, indicating decreases are due to an afferent reflex inhibition of motoneurones (Garland et al. 1988). Bigland-Ritchie et al. (1986b) using intermittent fatigue did not find a significant decline in the quadriceps EMG following

maximal fatigue, but did report a drop (40%) in the EMG of the soleus supporting muscle specific differences. Minor changes in quadriceps EMG activity were also reported by Nilsson et al. (1977) following 100 isokinetic contractions. They attributed the minimal change to the quadriceps fibre composition. Decreases in EMG activity have been demonstrated 25 hours after fatiguing exercise that included both isometric contractions at 40% of MVC as well as submaximal concentric and eccentric contractions (Kroon and Naeije 1988). Thus fatiguing contractions may be characterized by increases in EMG with submaximal contractions, changing to decreases in EMG with maximal intensity contractions (Table 1). The extent of change may be muscle and fibre type specific.

Decreases in EMG activity with fatigue have been attributed to decreases in the excitability of the motoneurones as measured by the H-reflex (Duchateau and Hainaut 1993, Garland and McComas 1990). However, Duchateau and Hainaut (1993) reported an enhanced neural drive with fatigue since there was no significant change in the long latency reflex amplitude of fatigued abductor pollicis brevis while the non-fatigued first dorsal interosseous experienced an enhanced long latency reflex. They suggested that an enhanced supraspinal drive with fatigue is inhibited at the motoneurone level by presynaptic afferents from the fatigued muscle and/or interneurones in the oligosynaptic pathways. Since the EMG signal is more than just a representation of neuromuscular activation, decrements in EMG activity may also be attributed to alterations in muscle membrane excitation.

Neuromuscular Propagation Disturbances of neuromuscular propagation (including muscle membrane excitation) with fatigue can be examined through alterations of the compound muscle action potential wave (M-wave). Many studies utilizing repetitive MVC, (Bellemare and

Garzanti 1988, Miller et al. 1987, Milner-Brown and Miller 1986, Pagala et al. 1984), prolonged submaximal isometric contractions (Fuglevand et al. 1993) and E-stim (Moritani et al. 1985) to induce fatigue have reported decreases in M-wave amplitudes. Bigland-Ritchie et al. (1979) using a 60 s MVC fatigue protocol of the adductor pollicis did not find any change in M-wave amplitude suggesting muscle membrane transmission failure was not a component of high intensity short term fatigue. Other studies using short duration voluntary (Bellemare and Garzanti 1988, Bigland-Ritchie et al. 1979) or evoked (Garland et al. 1988) fatigue protocols have not shown decreases in M-wave amplitude.

The impairment of membrane propagation may depend both on the duration and degree of fatigue as well as the intrinsic properties of the individual muscle (Milner-Brown and Miller 1986). Pagala et al. (1984) reported greater decreases in the surface action potential of the extensor digitorum longus and diaphragm than with the soleus, while Moritani et al. (1985) found similar results when comparing the gastrocnemius and soleus. Table 2 illustrates that neuromuscular propagation can be both impaired or facilitated with sustained contractions depending upon the duration, intensity and muscle used.

Neuromuscular propagation disturbances could occur at the neuromuscular junction or with muscle membrane transmission. Early research using high frequency stimulation of rat muscle resulted in pre-synaptic failure; blocking activation of the muscle fibre (Krnjevic and Miledi 1959). Several hours of low frequency (2 Hz) stimulation of rat muscle has shown significant decreases in the quanta of acetylcholine (ACh) released per impulse (Hinz and Wernig 1988). Since ACh storage has been reported to be 100 times greater than its quantal release (Daube 1983) and significant disruptions in pre-synaptic function have been shown only under evoked stimulating

conditions in animals, it would seem likely that neuromuscular junction impairment is not a major factor with short to moderate duration voluntary activities.There still exists the possibility for neuromuscular junction impairment with very long duration activities (ie marathons, triathlons).

Muscle membrane impairments can affect E-C coupling by disrupting the capacity to depolarize, rate of depolarization, conduction velocity and the spread of depolarization down the transverse tubules to the SR affecting the release of Ca^{++} (Duchateau and Hainaut 1985, Milner-Brown and Miller 1986, Pagala et al. 1984). Fatigue-induced decreases in M-wave amplitude could be attributed to changes in the muscle membrane ionic equilibrium. The prolongation of the M-wave duration with both voluntary (Bigland-Ritchie et al. 1982, Miller et al. 1987, Milner-Brown and Miller 1986) and E-stim (Juel 1988) fatigue has been attributed to changes in extracellular potassium and intracellular pH (Juel 1988). Although M-wave depression could be potentially detrimental, a prolonged surface action potential would increase Ca^{++} deposition on the myofilaments providing more contractile force during fatigue.

A number of researchers have reported increases in M-wave amplitude during the initial portion of the fatigue protocol, followed by decreases in amplitude as the fatigue progressed (Pagala et al. 1984, Duchateau and Hainaut 1985). Fitch and McComas (1985) reported potentiation of the M-wave amplitude during the first 30 s of tetanic fatiguing contractions suggesting an increased excitability of the muscle fibers. Potentiation of the M-wave amplitude may signify that pre-synaptic and/or end-plate potentials are facilitated possibly by a reduction in the dispersion of fibre action potentials (Duchateau and Hainaut 1985).

Twitch Contractile Properties: Alterations in twitch contractile properties with fatigue are duration, and intensity dependent (Table 2). Studies have demonstrated potentiation of twitch

amplitudes with submaximal (Dolmage and Cafarelli 1991), short term (10 s) maximal (Grange and Houston 1991, Houston and Grange 1990) and electrically-induced contractions with frequencies below 50 Hz (Small and Stokes 1992). One mechanism underlying potentiation has been attributed to P-light chain phosphorylation (Grange and Houston 1991, Houston and Grange 1990), which increases the rate constant for cross-bridge attachment under moderate Ca^{++} concentrations (Metzger et al. 1989). Other potentiation mechanisms proposed include increased deposition of Ca^{++} on the myofilaments (Blinks et al. 1978) as well as increases in muscle stiffness (Sinkjaer et al. 1992).

Depression of twitch torque is evident with sustained (Grange and Houston 1991, Houston and Grange 1990, McKenzie and Gandevia 1991) and intermittent maximal (Bigland-Ritchie et al. 1983) as well as intermittent submaximal contractions (Vollestad et al. 1988). Duchateau and Hainaut (1985) reported no difference in the extent of twitch torque depression when comparing maximal intermittent and sustained fatiguing contractions. Decreases in force production and diminished rates of force development and relaxation were attributed to a depression in the SR ATPase Ca^{++} pump with exhausting exercise in rat FT muscle (Belcastro et al. 1981). Caffeine has been used to release Ca^{++} from intracellular stores following fatigue-induced force decrements. The reversal of force loss with caffeine following fatigue has been demonstrated in both humans (Lopes et al. 1983) and animals stimulated at frequencies below 50 Hz (Nassar-Gentina et al. 1981). This would suggest that a component of prolonged fatigue is related to E-C coupling impairments. A review by Williams and Klug (1995), indicated that reductions in Ca^{++} release could account for much of the tension reduction and slowing of relaxation during fatigue. Possible mechanisms of fatigue-induced SR dysfunction included

structural disruption of the SR, a decrease in the number of Ca^{++} release channels, as well as an uncoupling of Ca^{++} ATPase activity with increased temperature (Williams and Klug 1995).

Twitch torque depression could signify impairments of E-C coupling if a MVC or a tetanus could restore normal force output. Both McKenzie et al. (1992) using intermittent MVCs as well as Bigland-Ritchie et al. (1986b) using intermittent submaximal contractions reported that twitch amplitudes declined more rapidly than MVC. Similar results were found by McKenzie and Gandevia (1991) with intermittent (10 s contraction) MVCs with variable duty cycles. E-C coupling was proposed as the failure mechanism by Edwards et al. (1977) who found the decline in twitch amplitude could be overcome with high frequency tetanic stimulation. Reid et al. (1993) suggest that E-C coupling is associated with high frequency fatigue. Alway et al. (1987) reported a lack of change in twitch torque following intermittent (5 s / 5 s) MVCs despite a 35% drop in MVC following fatigue, suggesting twitch contractile properties did not affect the loss of force in their study. Whether evoked twitches will increase, decrease or remain the same with fatigue is a complex matter. The duration, intensity and frequency of activation will have differing effects upon the extent of potentiation and E-C coupling impairment. In general it might be surmised that submaximal contractions which do not reach maximum intensity and very short duration (ie 10 s) maximal contractions may result in twitch potentiation. Longer duration maximal (ie 60 s) and submaximal contractions should initially experience potentiation followed by twitch torque depression as the contractions reach maximum relative intensity.

Evoked Temporal Characteristics Some researchers have reported a prolongation of time to peak twitch (TPT) following fatigue in humans (Duchateau and Hainaut 1985) and cats (Petrofsky et al. 1980). Favero et al. (1993) subjected rats to prolonged submaximal running and

reported a reduced rate of Ca^{++} release from actively-loaded SR vesicles. Conversely TPT has also been reported to decrease with submaximal contractions (Dolmage and Cafarelli 1991). McKenzie and Gandevia (1991) found differing effects upon twitch contraction time dependent on the work-rest relationship. They reported prolongation of twitch contraction time with duty cycles of 20 and 50% while a reduction in twitch contraction time was detected with duty cycles of 5 and 10%. They surmised that there must be a crucial aerobic rest interval which determines prolongation or shortening of the twitch time.

Similarly, conflicting results have been found with twitch relaxation time. Prolongation of relaxation time has been reported with fatiguing MVCs (Duchateau and Hainaut 1985) and E-stim (Hultman and Sjoholm 1983). In accordance with prolonged relaxation, decreases in Ca^{++} uptake by the SR has been documented in both animals (Byrd et al. 1989) and humans (Gollnick et al. 1991) following exhaustive exercise. Conversely, decreases in relaxation time have been found following intermittent fatiguing MVCs (Alway et al. 1987), submaximal voluntary contractions (Dolmage and Cafarelli 1991) and E-stim (Rutherford and Jones 1988). In addition, these findings are further complicated since the durations of force production and relaxation do not always coincide. Bigland-Ritchie et al. (1983) demonstrated a prolongation in relaxation time contrasted with a lack of change in TPT. This divergence has also been reported by Viitasalo and Komi (1981), who found less slowing of relaxation with lower intensity contractions. Impairment in E-C coupling affecting Ca^{++} release (TPT) may not automatically coincide with a hindrance of Ca^{++} sequestering (relaxation time). In contrast to the release of Ca^{++} for force development, the sequestration of Ca^{++} is an active process involving ATP (Brooks and Fahey 1985) and thus would be affected by impairments to energy production. Cady et al. (1989) suggested that the

fatigue-induced increase in relaxation could be attributed to two major metabolic factors; H^+ accumulation and increases in ADP concentration. Similarly, Duchateau et al. (1987) found a correlation between the recovery of half relaxation time and reductions in venous lactate concentration. The difference in reliance upon energy production for Ca^{++} release and sequestration may explain the lack of correlation between changes in TPT and relaxation time. Prolongation of force production and relaxation would logically be an effective mechanism to combat the effects of fatigue by increasing the time for Ca^{++} release and crossbridge kinetics. However changes in muscle stiffness with fatigue (Sinkjaer et al. 1992) could result in decreased durations of force production and relaxation.

Crossbridge Kinetics Myofibrillar impairment with fatigue can been difficult to substantiate due to the number of other systems and structures (ie. motoneurone activation, membrane propagation, SR release of Ca^{++}) whose functions precede actin-myosin crossbridge kinetics. The possibility of changes occuring beyond the sarcolemnal membrane was hypothesized by Stokes et al. (1989) with decreased force output following low frequency fatigue. However high frequency stimulation restored the force output suggesting that if myofibrillar impairment was present that it may be related to Ca^{++} insensitivity and not myofibrillar protein breakdown. Hultman and Sjoholm (1983) also suggested that contractile filament Ca^{++} sensitivity can be affected by changes in electrolyte composition, pH and energy metabolism. Since myofilament crossbridge attachment and movement is the final link in the temporal chain of muscle force production, the impairment of this component may be affected by different mechanisms.

Crossbridge cycling is not only dependent upon the liberation of the troponin-tropomyosin complex by Ca^{++}, but factors affecting myosin ATPase activity as well (Brooks and Fahey 1985).

Earlier studies emphasized the inhibitory effect of lactate concentration on contractile proteins (Donaldson and Hermansen 1985, Fabiato and Fabiato 1985). Other fatigue-related factors such as a lower ratio of ATP/ADP (Chasiotis et al. 1987) or increased inorganic phosphates (P_i)(Chasiotis et al. 1987, Baker et al. 1994) could result in decreased crossbridge turnover. Cady et al. (1989) compared normal subjects with those with myophosphorylase deficiency (lacks glycogenolysis) and discovered that neither H^+ or P_i accumulation could be the sole explanation for loss of force. Newham and Cady (1990) concurred by stating, there was no evidence of a unique relationship between any one metabolite and force. True myofibrillar fatigue was proposed by Edman and Lou (1992) as a consequence of the altered metabolic state associated with a 1 s tetanus every 15 s. A second fatigue protocol eliciting a twitch every 1-2 s produced activation rather than contractile failure. Fatigue solely due to myofibrillar impairment would seem to involve changes in the intracellular environment, most likely occuring with a high intensity, short duration, more metabolically disruptive fatigue protocol.

A second scenario providing possible evidence of contraction-related myofibrillar impairment is provided by delayed onset muscle soreness (DOMS). DOMS is most evident with repeated high intensity eccentric contractions (Evans and Cannon 1991). Eccentric contractions with their capability for withstanding higher loads than concentric contractions could expose the myofilaments to excessive shearing stress (Evans and Cannon 1991). Friden et al. (1983) examined ultrastructural damage in human muscle following high tension eccentric exercise and found that disorganized myofibrillar material constituted one third of the analysed tissue one hour after exercise. The duration of the pain and damage associated with DOMS suggests there may be continued active myofibrillar protein degradation which may be related to immunological and

oxidative (free radicals) factors (Evans and Cannon 1991). Thus in addition to metabolic disruption of cross-bridge cycling, mechanical impairments arising from a high intensity overload stress may reduce myofibrillar efficiency.

Motor Control:

Co-contractions The extent of antagonist co-contraction is related to a number of factors including load (Lestienne 1979, Mustard and Lee 1987, Wierzbicka et al. 1986), velocity of movement (Karst and Hazan 1987, Marsden et al. 1983), range (Hannaford and Stark 1985, Karst and Hazan 1987), precision of motion (Gordon and Ghez 1984) and type of contraction (Osternig et al. 1984). Co-activation of antagonists also function to provide joint protection and movement control during an agonist contraction (Barrata et al. 1988, Cooke and Brown 1990, Solomonow et al. 1988, Tyler and Hutton 1986). Since antagonist muscles work in opposition to the agonists, the timing and extent of their activation could have significant effects on agonist function. Psek and Cafarelli (1993) reported a drop in hamstring EMG activity following a high-intensity isometric leg extension fatigue protocol but reported no decrease in antagonist EMG activity with a low intensity fatigue protocol. A study from our laboratory showed a 31% decrease in antagonist EMG activity following submaximal intermittent isometric fatigue of the plantarflexors (Behm and St-Pierre under review). A decrease in antagonist activity would presumably facilitate net agonist forces contributing to fatigue resistance. A motor control perspective might suggest that antagonist activity is decreased in proportion to decreases in agonist activity to maintain muscle stiffness dynamics and thus maintain efficient motor control. Alternatively, the reflex-induced inhibition of motoneurones affecting agonist EMG, muscle activation and rate coding could have an effect on antagonist muscles as well.

In summary, the effects of fatigue cannot easily be categorized (Tables 1 and 2). Dependent upon the intensity and duration of the fatigue protocol and the muscle utilized fatigue can result in decreases or no change in muscle activation, and motor unit firing frequency. Conversely EMG activity, M-wave amplitude, evoked twitch force, TPT and 1/2 RT can increase, decrease or remain the same. Not all fatigue-related adaptations impair force output. Decreases in motor unit firing frequency and increases in twitch temporal characteristics can actually contribute to force output. Changes in these variables are not only intensity, duration and muscle specific but will also be affected by whether the muscle is in a trained or disused state.

Effects of Strength Training on Fatiguability

Animal studies using compensatory hypertrophy have reported decreases (Baldwin et al. 1977), and no changes in selected aerobic enzymes (Baldwin et al. 1982, Ianuzzo and Chen 1979) suggesting hypertrophy would not offer an advantage for fatigue resistance or endurance. However, Michel et al. (1989) and Roy et al. (1982) reported increased fatigue resistance with compensatory hypertrophy in rats. These authors suggested that either decreases in the fast fibre area (Michel et al. 1989) or a conversion of fast to slow twitch fibres (Roy et al. 1982) might explain the increased fatigue resistance. St-Pierre et al. (1988) strength- (grid climbing) and swim-trained rats and discovered an improvement in fatigue resistance only with swim training. Improvements in fatigue also have been reported in humans. McDonagh et al. (1983) showed an improvement in the fatigue index after 5 weeks of isometric training. Rube and Secher (1990) trained their subjects for 5 weeks with two and one leg isometric contractions and reported improvements in the fatigue index specific to the training mode. Duchateau and Hainaut (1984b) trained normal subjects' adductor pollicis muscle for three months finding an augmentation of

tetanic force during fatigue and a marked reduction in the fatigue-related changes in the surface action potential. Costill et al. (1979) used 7 weeks of isokinetic strength training to show that trained individuals exhibited greater strength in the first few seconds of a 60 s isokinetic fatigue test, but declined to the same mean power as the untrained after 30-40 s. Grimby et al. (1973) used isometric training for 6 weeks and reported increases in force output throughout the intermittent fatigue protocol but no change in the rate of fatigue. An early study by Shaver (1964) found a high correlation (0.93) between maximum strength and endurance using a submaximal load, but no significant relationship (-0.19) when working at the same relative intensity. This finding indicated that greater strength resulted in more repetitions of a similar submaximal load, but did not provide an advantage in performing repetitions of a similar percentage of the individual's maximal load. Similarly, a cross-sectional study (Huczel and Clarke 1992) indicated that strength-trained women demonstrated greater absolute fatigue resistance (total force output) than controls but no significant difference in relative endurance (fatigue index) during 6 minutes of maximal rhythmic exercise of the elbow flexors. Strength training may promote greater total force output during fatigue, and improve the maintenance of an absolute submaximal load, but the research is inconsistent regarding the maintenance of maximal contractions (fatigue index) or submaximal loads at the same relative intensity. The mechanisms regulating strength or hypertrophic adaptations may not always correspond to adaptations with fatigue. This was supported by the findings of St-Pierre et al (1992) with a study on isokinetic rehabilitation following arthroscopic meniscectomy. It was found that the indices of fatigue recovered sooner than quadriceps torque

Effect of Strength Training on Fatigue-Related Central Adaptations

Muscle Activation: Neural adaptations have been cited as the primary mechanism underlying strength gains in the early portion of a strength training program (Moritani and DeVries 1979, Sale 1988). The use of the ITT however, has demonstrated full muscle activation even in untrained individuals (Belanger and McComas 1981, Bellemare et al. 1983, Gandevia and McKenzie 1988, Rutherford et al. 1986). In contrast, there have also been reports that not all subjects could fully activate their plantarflexors (Belanger and McComas 1981), elbow flexors (Dowling et al. 1994, Lloyd et al. 1991) or quadriceps (Norregard et al. 1994, Strojnik 1995). Allen et al. (1995) indicated that although all of their subjects could fully activate their elbow flexors in some trials, they failed in 75% of their contractions. A number of training studies have reported that all or almost all of their subjects could fully activate prior to training (Garfinkel and Cafarelli 1992, Jones and Rutherford 1987, Rutherford and Jones 1986). Subjects who were not fully activated prior to training achieved full activation post-training (Rutherford and Jones 1986, Jones and Rutherford 1987). Conversely, Sale et al. (1992) reported that incomplete quadriceps activation was not altered with dynamic resistance training. Research from our laboratory has shown no significant difference in the extent of muscle activation of trained (99.5%) and untrained (98.7%) plantarflexors when attempting a MVC (Behm and St-Pierre, under review). Since training adaptations and the ability to fully activate in untrained individuals are not consistent in the literature, the effect of strength training on muscle activation is still equivocal.

It could be speculated that the lack of trained state differences in the extent of muscle activation pre-fatigue might become more apparent with the stress of fatigue. Duchateau and Hainaut (1988) hypothesized that a training-related increase in muscle activation led to an improved fatigue index of subjects trained with submaximal voluntary contractions. In contrast,

electrostimulation training which would not promote neural adaptations did not improve the fatigue index. A cross-sectional fatigue study from our laboratory found no trained state difference in the extent of muscle inactivation following a submaximal intermittent fatigue protocol (Behm and St-Pierre under review). Considering the small to negligible trained state differences in muscle activation, it seems unlikely that this factor plays an important role for improving the fatigue resistance of trained individuals.

Reflex Potentiation: Reflex potentiation reflects the degree to which motoneurone excitability has been raised during a voluntary effort by comparing the amplitudes of reflex responses to a maximum M-wave. This potentiation is considered to be mediated by reflex pathways of both short and long loops, indicating changes at either the proprioceptive or supraspinal level. Sale and colleagues found both increases (extensor digitorum brevis, brachioradialis, hypothenar: 1983b) and decreases (thenar: 1982) in reflex potentiation following strength training. In a cross-sectional study of weight-trainers and controls, Sale et al. (1983a) reported greater reflex potentiation for the weight-trainers. In another cross-sectional study, Upton and Radford (1975) reported enhanced reflex potentiation in sprinters versus controls. Conversely, Casabona et al. (1990) reported the H-reflexes of explosive jump-trained individuals (sprinters and volleyball players) were significantly less than control subjects. Casabona et al. explain their findings by suggesting that explosive trained athletes may have a lesser synaptic strength of Ia excitatory afferents on intermediate motoneurons. Secondly they may have a higher incidence of high threshold fast twitch (FT) units which are not activated to the same extent by the Ia afferent volley. Thus the role of reflex potentiation may be differentially altered dependent upon the specific type of training (strength; power; endurance) and/or muscles used. Whereas

muscle activation may be inhibited with long duration, submaximal, fatigue protocols (Bigland-Ritchie et al. 1986b, Newham et al. 1991), increased reflex potentiation with training could help to offset the fatigue-induced inhibition of motoneurones with submaximal contractions. The advantage of increased reflex potentiation may not be as evident with maximal contractions, since untrained individuals are capable of full or nearly full activation (Belanger and McComas 1981, Bellemare et al. 1983, Gandevia and McKenzie 1988, Rutherford et al. 1986). Thus reflex potentiation would not be expected to augment an already fully activated motor unit.

Rate Coding: Studies examining changes in motor unit firing frequency (rate coding) with training are not common. More consistent firing patterns have been reported following resistance training (Cracaft and Petajan 1977, Kawakami 1955). Grimby et al. (1981) in a fatigue study indicated that some subjects could only achieve motor unit firing frequencies necessary for complete fusion after repeated trials. Hannerz (1974) indicated that motor unit firing frequencies necessary to achieve full fusion (contraction) of the muscle were achieved in untrained individuals. However, the discharge rates of motor units in a hand muscle during maximum contractions have been reported to increase with short- and long-term training (Leong et al. 1995, Patten et al. 1995). Although motor unit discharge rates have been recorded at high frequencies of 60-120 Hz (Desmedt and Godaux 1977), complete fusion may be achieved at relatively low firing rates (Kukulka and Clamann 1981, Person and Kudina 1972). Since high motor unit firing frequencies are related to increases in rate of tension development (Buller and Lewis 1965, Miller et al. 1981), and the force-frequency relationship shows minimal changes in force at the highest frequencies (Thomas et al.1991), increases in rate coding with strength training should not appreciably contribute to increased force output with fatigue involving maximal contractions. With high

intensity contractions, the greater number of stimuli associated with a more consistent signal would place more stress on the muscle membrane affecting excitability and offsetting other training-related advantages. However, the more consistent firing pattern found with strength training may improve motor control, sustaining a submaximal load more efficiently.

Effect of Strength Training on Fatigue-Related Peripheral Adaptations

Electromyography: If maximal fatigue protocols cause a reduction in EMG activity, increases in EMG activity with training (Sale 1988) might be hypothesized to aid fatigue resistance. Evidence for increased neural drive has been suggested by the increase in EMG activity with isometric (Davies and McGrath 1982, Thepaut-Mathieu et al. 1988), concentric (Hakkinen et al. 1985a, Hakkinen and Komi 1986) concentric and eccentric (Hakkinen and Komi 1983), isokinetic (Narici et al. 1989) and explosive dynamic training (Hakkinen and Komi 1985, Hakkinen et al 1985b). Greater decreases in the EMG of untrained versus orienteering women following 50 isokinetic plantarflexion contractions indicated an EMG training effect (Johansson and Gerdle 1988). Similarly marathon runners had a minor decline in EMG compared to the significant decrease in sprinters' quadriceps EMG with fatiguing isokinetic MVCs (Lorentzon et al. 1988). However, there have been a number of examples of increases in force output without apparent increases in neural drive. Training studies using isometric (Cannon and Cafarelli 1987), concentric and eccentric (Komi and Buskirk 1972), dynamic resistance training (Thorstensson et al. 1976b), isometric and isokinetic training (Behm and Sale 1993) have not shown any changes in EMG over the training period. Strength gains in these studies may have been due to factors other than increases in neural drive (EMG) to the agonist, such as motor control changes (Rutherford and Jones 1986). It could be argued that during a submaximal fatigue protocol, an increased EMG

signal might provide a greater neural reserve prior to maximal EMG being reached. Increases in EMG activity with strength training in addition to possible hypertrophy would result in greater absolute forces to sustain throughout a maximal fatigue protocol. The maintenance of greater absolute forces without increases in oxidative (MacDougall et al. 1979) and glycolytic enzymes (Baldwin et al. 1977, Ianuzzo and Chen 1979) would provide more difficulty in prolonging the contractions. The rate of force loss with maximal contractions may not be altered even with increased EMG due to the greater absolute tension that must be maintained.

Neuromuscular Propagation: Animal endurance training studies have confirmed increases in the neuromuscular synaptic terminal area (Deschenes et al. 1993, Waerhaug et al. 1992) and the quantal release of neurotransmitters (Jasmin and Gisiger 1990, Dorlochter et al. 1991). However, improvements in the neuromuscular junction were independent of muscle hypertrophy (Deschenes et al. 1993, Diamand et al. 1974) and were muscle specific (Jasmin and Gisiger 1990, Waerhaug et al. 1992). Thus endurance (long duration) but not strength training may provide an additional reserve of neurotransmitters at the neuromuscular junction.

Few human studies have examined the effects of strength training on the muscle membrane action potential. Neither 3 months of isometric or dynamic training of the adductor pollicis (Duchateau and Hainaut1984a) or 8 weeks of isometric thenar training (Sale et al.1982) affected the M-wave amplitude. In another study, Duchateau and Hainaut (1984b) trained normal subjects' adductor pollicis muscle for 3 months finding reduced augmentation of muscle surface action potential duration and area during fatigue. They hypothesized that training improved synaptic facilitation during fatigue. Whether training improves muscle membrane functioning during fatigue is difficult to speculate due to the lack of consistent training study results. Teleologically, an

augmentation of synaptic potentials or membrane electrical conduction with training should help maintain muscle membrane excitation during fatigue.

Twitch Contractile Properties: Strength training studies with both humans and animals have reported increases (Duchateau and Hainaut 1984b, Freeman and Luff 1982, Gonyea and Bonde-Petersen 1978) and no change (Alway et al. 1989, 1990, Davies and McGrath 1982, McDonagh et al. 1983) in twitch tension. Duchateau and Hainaut (1984a) found increases in twitch tension with isometric but not dynamic resistance training. These discrepancies may be related to the specificity of training. Duchateau and Hainaut (1984a) reported that twitch contraction time was decreased (increased rate) more with dynamic than isometric training. Decreased twitch contraction time would result in less time available for the release of Ca^{++} from the SR. Alway et al. (1988, 1989) have reported a dissociation between the fibre volume of the SR and the twitch contractile properties of muscles in vivo. They could not establish a relationship in their submaximal isometric training study between the training-related decrease in contraction time and the lack of change of the SR-transverse tubule complex (Alway et al. 1990). Conversely, slower rates of SR Ca^{++} uptake have been demonstrated with compensatory hypertrophy in rats (Kandarian et al. 1994). Although compensatory hypertrophy may initially suggest strength training, the removal of the plantaris' synergists would involve extensive endurance training. Therefore training adaptations of twitch contractile properties seem to be affected by the training mode. Inconsistent evidence for increases in twitch force and the dissociation of SR and myofibrillar strength training adaptations would suggest that training-induced alterations in twitch force do not play a major role in fatigue resistance. However an increased twitch duration would provide greater opportunities for Ca^{++} release and subsequent cross-bridge attachment.

Myofibrillar Component: The role of muscle hypertrophy in promoting strength gains (Ikai and Fukunaga 1968, Maughan et al. 1983) has been suggested to be of increasing significance as the duration of the resistance training continues (Moritani and deVries 1979, Sale 1988). However the anabolic effects are tissue and organelle specific and related to the functional demands placed on the muscle (Houston et al. 1983, MacDougall et al. 1980, Thorstensson et al. 1976a). Myofibrillar proteins increase dramatically with resistance training while mitochondrial proteins increase with aerobic endurance training (Booth and Thomason 1991, Goldberg et al. 1975, Holloszy and Coyle 1984). Some researchers have demonstrated that endurance training interferes with hypertrophic or strength adaptations (Dudley and Djamil 1985, Moroz and Houston 1987, Ono et al. 1976) while strength training resulted in no change (Abernethy and Quigley 1993, Hennessy and Watson 1994, McCarthy et al. 1995, Nelson et al. 1990) in aerobic capacity. Dudley and Djamil (1985) reported that concurrent strength and endurance training did not affect increases in aerobic power but adversely affected increases in angle specific maximal torque at fast velocities. On the other hand, Sale et al. (1990) had subjects train one leg for strength while the other leg was trained for both strength and endurance. He reported no differences between the legs in aerobic capacity and one repetition maximum leg lifts, indicating that concurrent strength and endurance training did not interfere with strength gains. At the cellular level, MacDougall et al. (1979) reported decreases in mitochondrial volume density following heavy resistance training. It has also been suggested that hyperplasia may occur in extreme bodybuilding (MacDougall et al. 1982). MacDougall et al. (1982) hypothesized that there may be a "ceiling" or limit to the size of a muscle fibre before it is forced to split. The mechanism underlying this limit may be an inefficient metabolic gradient across an extremely hypertrophied

fiber (Gonyea et al. 1986), which could have direct consequences on the muscle's fatigue resistance. Conversely endurance training has been associated with a loss of strength (Ono et al. 1976, Moroz and Houston 1987) and decreased muscle fibre size (Klausen et al. 1981, Terrados et al. 1986). Thus, the lack of improvement in the fatigue index with strength training could be attributed to both changes in the metabolic gradient efficiency as well as specific increases in myofibrillar proteins to the detriment of mitochondrial and enzymatic proteins. The specific anabolic adaptations associated with hypertrophic adaptations would not contribute to improvements in the fatigue resistance to maximal contractions. On the other hand, the fatigue resistance associated with a submaximal load could be enhanced, since the load would represent relatively less tension to a stronger hypertrophied muscle.

Effect of Strength Training on Fatigue-Related Motor Control Adaptations

Co-contractions: Training may alter the co-activation of antagonist muscles. Carolan and Cafarelli (1992) demonstrated a decrease in co-activation associated with a resistance training program of the leg extensors. Co-activation increases have been shown in longitudinal resistance training (Baratta et al. 1988) and cross-sectional studies comparing explosively trained athletes (Baratta et al. 1988, Osternig et al. 1986) to non-explosive type athletes. In an evaluative stretch study, Osternig et al. (1990) reported endurance athletes had greater co-activation than explosively trained athletes and control subjects. However, stretching activities may differ from dynamic contractions in neural activation and inhibitory patterns. The variety of factors affecting co-contractions make it difficult to predict whether antagonist activity will increase or decrease with specific training. Although both trained and untrained individuals had similar decreases in antagonist activity post-fatigue, trained subjects experienced a 58% lower antagonist / agonist

EMG ratio (tibialis anterior / soleus) post-fatigue when performing submaximal isometric plantarflexor contractions (Behm and St-Pierre, under review). Thus a more efficient motor control system may contribute to the fatigue resistance of trained individuals.

In summary, strength training can contribute to improvements in sustaining absolute submaximal loads, but offers little benefit to maintaining maximal or relative (percentage of maximum) loads. Although neural adaptations with strength training may augment muscle activation (increased EMG activity and reflex potentiation), and provide a more consistent motor unit firing pattern, the most important factor may be that the increased muscle hypertrophy and strength results in relatively less tension associated with a similar absolute load. Conversely greater training-related force outputs without substantial increases in aerobic enzymes and SR would not improve the fatigue resistance of maximal or relative loads.

Effects of Muscle Disuse on Fatiguability

Much of the human disuse research has been conducted on patient populations where the added effect of trauma is difficult to estimate. This is exemplified in an animal study by Urbancova et al. (1993), who fractured rat metatarsals and reported a lack of atrophy in deafferented rats after 7 days. Afferents from the traumatized area therefore, can influence muscle atrophy. Immobilizing (reduction or elimination of movement by mechanical means: Webster's Dictionary) healthy subjects with various forms of casting, limb suspension (crutches and elevated heel) and bed rest eliminates the degradative effects of injury. Limb immobilization in healthy subjects results in muscle atrophy and decreases in strength (Berg et al. 1991, Veldhuizen et al. 1993). The atrophy and weakness in the Veldhuizen et al. (1993) study was accompanied by decreases in isokinetic endurance. However, White et al. (1984) casted 4 healthy subjects for 2 weeks and did

not find significant alterations in the fatigue resistance to intermittent evoked stimulation (20 Hz). Fuglevand et al. (1995) studied healthy individuals following 5 weeks of first dorsal interosseous immobilization reporting no change in the maximum voluntary contraction force (MVC), EMG, twitch amplitude, M-wave duration and the ability to sustain submaximal contractions. The very few immobilization studies with healthy subjects do not permit a definitive conclusion regarding the effects of disuse on fatigue.

Although both muscle trauma and immobilization can diminish muscle size and force output, they have a minimal effect on muscle endurance. Ingemman-Hansen and Halkjaer-Kristensen (1983) tested a patient population (collateral knee ligament injuries in soccer players) and reported no relative change in the fatigue index with disuse durations of 2-6 weeks. In a subsequent article, muscle function was tested before and after ligament repair, resulting in only minor alterations in an isometric fatigue index but a significant decrease in dynamic cycle endurance for the surgically treated group (Halkjaer-Kristensen and Ingemman-Hansen 1985). Duchateau and Hainaut (1987) found similar decreases in tension between control and disuse subjects with fatiguing isometric MVCs. Since the extent of muscle activation was not measured or controlled in these studies, the similar fatigue indexes might be attributed to differences in the intensity of muscle contractions. Immobilized-induced muscle inactivation could result in the comparison of submaximal and maximal intensity fatigue protocols. A study from our laboratory (Behm and St-Pierre, under review) had internally-fixated (surgery), non-fixated previously immobilized ankle fractures and control groups perform submaximal (50% of MVC), isometric, intermittent fatiguing contractions. Muscle activation was monitored with the interpolated twitch technique to ensure similar levels of activation and contraction intensities between groups.

Although internally-fixated subjects were more fatiguable than the other groups, there was no significant difference in muscle endurance between non-fixated immobilized patients and controls. The trauma associated with complicated fractures and surgery (internally-fixated) may have contributed to an intrinsically more fatiguable muscle. However, since the extent of activation was calculated to ensure similar submaximal contraction intensities, differences in muscle activation could not explain the similar fatigue durations of non-fixated and control subjects. Fatigue studies utilizing evoked stimulation can also ensure that contractions are at similar intensities. Duchateau and Hainaut (1987) showed that greater decreases occurred in disused than control hand muscles with fatiguing evoked stimulation (E-stim). Snyder-Mackler et al. (1993) utilized E-stim fatigue of the quadriceps in patients following anterior cruciate ligament reconstruction. Stimulation intensity was established to elicit contractions of 20% MVC of the unaffected limb. They reported that the healthy limb was more fatiguable than the affected limb. They hypothesized that there was either a selective disuse atrophy of type IIb fibres in the affected limbs or the greater stimulation intensity used in the affected versus the unaffected limb would involve more fatigue resistant muscle fibres located deep within the quadriceps.

A number of interventions including immobilization, drugs, hindlimb and whole body suspension, have been used to study the effects of disuse in animals. Rat studies using tetrotodoxin to eliminate motoneurone activity while maintaining the flow of neurotrophic substances have either reported no significant change (Gardiner et al. 1991, St-Pierre and Gardiner 1985) or an increase (St-Pierre et al. 1988) in muscle fatiguability. Animal studies using four (cat: Jokl and Konstadt 1983) and 6 weeks (rat: Witzmann et al. 1983) of hindlimb immobilization have indicated either a lack of change in fatiguability or that motor units were less

fatiguable (cat: Robinson et al. 1991). Robinson postulated that the increased fatigue resistance in their cat hindlimb muscles following 3 weeks of immobilization may be related to the fatigue protocol which challenged excitation-contraction coupling more than the oxidative capacity of the muscle. Muscle specific differences were illustrated by Fell et al. (1985) who used a suspension model with rats. They showed that the lack of difference in the fatiguability of suspension and control soleus, contrasted with the greater fatiguability of the suspended gastrocnemius. Thus the effects of disuse on muscle fatiguability still need to be defined.

Effect of Disuse on Fatigue-Related Central Adaptations

Muscle Activation: Disuse can have dramatic consequences upon muscle activation. The extent of muscle inactivation with knee joint pathologies and surgery has been reported to be 35% and 45% respectively (Hurley et al. 1994, Rutherford et al. 1986). Similarly, Sale et al. (1982) discovered 37% decreases in reflex potentiation in subjects immobilized for 5 weeks. In a study from our laboratory, previously immobilized ankle fracture patients had a 20.4% decrease in their plantarflexor activation which was significantly greater than the 1.3% inactivation of healthy controls. Following comparable fatigue durations, relative decreases in muscle activation were similar (10%) between non-fixated ankle fracture and control groups. Surprisingly, internally-fixated ankle fracture subjects also experienced similar levels of inactivation with significantly fewer fatiguing contractions suggesting that the extent of muscle activation was not the distinguishing factor in the internally-fixated group's greater fatiguability (Behm and St-Pierre under review). Fatigue protocols using evoked stimulation of disused animal (Jokl and Konstadt 1983, Robinson et al. 1991, Witzmann et al. 1983) and human (Snyder-Mackler et al. 1993, White et al. 1984) muscle resulting in unchanged or improved fatigue resistance further illustrates

the minor role of muscle activation.

Rate Coding: Disuse adaptations of rate coding are similar to fatigue. Duchateau and Hainaut (1990) reported a decrease in the maximal firing rate of motor units following 6-8 weeks of immobilization and the appearance of silent periods (Duchateau and Hainaut 1987). The slowing of the twitch time course with immobilization (Duchateau and Hainaut 1990) could be perceived as an adaptation of the motoneurone to the slower contractile speed of the disused muscle. Changes in the rate coding patterns of cats were also reported by Fudema et al. (1961) who found an inconsistent firing pattern of the motor unit with sustained contractions. Decreases in motor unit firing frequency with disuse may contribute to the better than expected relative fatigue performance of disused muscle. Duchateau and Hainaut (1987) used 100 Hz evoked stimulation and found similar decreases in tension between control and previously fractured subjects within the first 10 seconds with greater decreases in the disused subjects after 10 seconds. Since evoked stimulation cannot be modulated, the slower contractile speed of the disused muscle may have difficulty adapting to the high frequency stimulation. Similar to "muscle wisdom" (Marsden et al. 1983, Woods et al. 1987), lower motor unit firing frequencies would result in fewer stimuli and subsequently less stress on the muscle membrane and SR decreasing chances of muscle membrane impairment.

Effect of Disuse on Fatigue-Related Peripheral Adaptations

Electromyography: Disuse studies have demonstrated decreases in maximum EMG activity following 10 days to 6 months of immobilization in both animals (Edgerton et al. 1975, Fishback and Robbins 1969, Hnik et al. 1985) and humans (Duchateau and Hainaut 1987, Fuglsang-Frederiksen and Scheel 1978). Even following 3 months of recovery of post-

menisectomy patients, Santavirta (1979) found EMG activity was still less than the control side. Since the EMG signal is a broad representation of the neural drive (deLuca 1979, deVries 1968, Perry and Bekey 1981), decreased EMG activity with disused muscle could result from atrophied muscle fibres, reduced muscle action potentials, a smaller percentage of muscle recruited or some of the muscle fibres being submaximally stimulated. The maintenance or improvement of fatigue resistance with disused animal (Jokl and Konstadt 1983, Robinson et al. 1991, Witzmann et al. 1983) and human (Snyder-Mackler et al. 1993, White et al. 1984) muscle fatigue protocols using evoked stimulation cannot be attributed to activation changes. Nevertheless, decreased EMG activity may represent changes in neuromuscular propagation or muscle size which might prove beneficial for fatigue resistance.

Neuromuscular Propagation: The duration of the surface action potential is prolonged following disuse (Duchateau and Hainaut 1987, 1990). Duchateau et al. (1987) studied eight subjects following immobilization due to forearm fractures and reported an increased duration of the surface action potential with both sustained and intermittent contractions of the adductor pollicis, possibly due to slower membrane conduction velocity, or disruption of endplate function. Similar to the effects of fatigue, a prolonged surface action potential would contribute to greater Ca^{++} deposition and saturation of the myofilaments. Duchateau and Hainaut (1987) contend that glycolysis and the H^{+} buffering capacity are less efficient in disused muscle and thus the reduced intracellular pH would inhibit the pumping activity of the SR, increasing the duration of the surface action potential. Therefore, one of the components contributing to the similar fatigue indexes between control and disuse subjects with voluntary contractions may be a lower rate of ATP utilization associated with the decreased activities of the membrane Na^{++} / K^{+} and SR Ca^{++}

ATPases.

Twitch Contractile Properties: Immobilization studies reveal decreases in twitch tension with humans (White et al. 1984), chick embryonic skeletal muscle (Reiser et al. 1988), and the rat ST soleus muscle (Fitts and Brimmer 1985, Simard et al. 1982, Witzmann et al 1982a,b). No changes were observed in twitch tension in rat FT extensor digitorum longus muscle (Fitts and Brimmer 1985, Simard et al. 1982, Witzmann et al 1982a,b), tetrodotoxin-paralysed rat gastrocnemius muscle (St-Pierre and Gardiner 1985,1987) or 2 week immobilized human legs (White et al. 1984). Furthermore, increases were reported with human subjects following 21 days of leg immobilization (Davies et al. 1987). Virtually without exception in these studies, the change in twitch tension parallels the shortening (decreased twitch tension) or prolongation (increased twitch tension) of the twitch time to peak tension.

Changes in twitch contractile properties reflect alterations in one or more of the components associated with E-C coupling. Detraining or atrophic effects on the myofibrillar component may not be accompanied by the same relative decreases in SR volume (Alway et al. 1988, 1989) or activity (Kim et al. 1982, Schulte et al.1993). A lower absolute tension during fatigue may be maintained by a relatively more expansive SR decreasing the contribution of E-C coupling impairments to decrements in force, thus helping to maintain tension during both submaximal and maximal fatigue protocols.

Myofibrillar Component: The lack of muscle stretch and activity with disuse results in muscle atrophy (Booth 1982, St-Pierre and Gardiner 1987). Disuse atrophy is primarily due to decreases in protein synthesis with contributions from increased protein degradation (Booth et al. 1992). An increase in the number of glucocorticoid receptors with immobilization (Dubois and

Almon 1980) as well as increases in lysosomal enzyme concentrations (Max et al. 1971, Witzmann et al., 1982c) would contribute to the catabolic effect of immobilization. Rats subjected to space flight have demonstrated decreases in actin mRNA of the gastrocnemius and vastus intermedius resulting in a down-regulation of the pre-translational contractile protein expression (Thomason et al. 1992). Since force is related to cross-sectional area (Ikai and Fukunaga 1968, Maughan et al. 1983), force output is adversely affected with disuse.

Furthermore, the proportional tension of myofibrillar protein has been reported to either decrease (Gardiner and Lapointe 1982, Simard et al. 1982, Jokl and Konstadt 1983, Fitts and Brimmer 1985) or increase (Summers and Hines 1951, Fishback and Robbins 1969, Maier et al. 1972) with immobilization. Baker and Matsumoto (1988) reported degenerative changes in the myofibres of rat soleus and gastrocnemius muscle after immobilization which would contribute to the decreased tension. According to St-Pierre and Gardiner (1987) the decreased tension could reflect contractile protein dysfunction while increases may represent changes in the angle of pennation with atrophy optimizing the angle of pull on the muscle.

Although muscle atrophy provides a disadvantage for force output, the muscle's smaller volume may provide an advantage for oxygen diffusion and substrate transport within the sarcoplasm (St-Pierre and Gardiner 1987). Researchers have reported both decreases (Booth 1977, Haggmark et al. 1981, Rifenberick et al. 1974, Stump et al. 1990) and no significant change (Graham et al. 1989, Musacchia et al. 1992, Ohira et al. 1992, Virtanen et al. 1991) in the concentration of aerobic enzymes and myoglobin (Booth 1977, Virtanen et al. 1991) with disuse. Krieger et al. (1980) indicated that although subsarcolemnal mitochondria were reduced with disuse, there was no change in intermyofibrillar mitochondria. Glycolytic enzymes are primarily

unaffected (Appell 1990) or increased (Chi et al. 1992) in immobilized muscle. A lack of change in the fatiguability of disused muscle might then be attributed to the maintenance of a more efficient metabolic gradient associated with a smaller muscle.

Effect of Disuse on Fatigue-Related Motor Control Adaptations

Co-Contractions: Very few studies have examined the effects of disuse on motor control. In a study from our laboratory (Behm and St-Pierre, under review), internally-fixated ankle fracture subjects experienced increased co-activation of antagonists, while non-fixated patients had decreased antagonist activity following a submaximal, intermittent isometric fatigue protocol. Although non-fixated patients experienced a decrease in co-activation, their level of antagonist activity was still significantly greater than controls. The greater antagonist activity of immobilized subjects would suggest that the co-contractions played a protective function for the previously injured muscle. Increased antagonist activity would serve to reduce the efficiency of force output hindering fatigue resistance.

In summary, although disused muscle can experience losses in strength and size, the relative rate of force loss with fatiguing contractions is not significantly affected. Adaptations associated with disuse may contribute to the maintenance of the fatigue index. Decreases in motor unit firing frequency with disuse could reduce the stress placed on the muscle membrane, while a prolonged membrane action potential would allow a greater release of Ca^{++} upon the myofilaments. Furthermore the atrophic myofilaments are supplied Ca^{++} by a relatively greater volume and activity of SR. An attempt to maintain energy effiency with disuse is evidenced by a lack of change in the number of intermyofibrillar mitochondria and myoglobin. Finally while muscle atrophy adversely affects force output, the smaller volume of muscle may improve oxygen

diffusion and substrate transport.

Conclusions

Strength training can improve the maintenance of an absolute submaximal load, but research has found both increases and no change in the fatigue resistance with maximal contractions or submaximal loads at the same relative intensity. Absolute submaximal loads would be better maintained with a strength trained muscle since the load represents a smaller percentage of the original load. In addition, increased EMG activity may provide a greater neural reserve while a more consistent motor unit firing frequency would ameliorate motor control. Incidences of improved fatigue resistance with a maximal load may be attributed to a prolongation of membrane excitation allowing more Ca^{++} to be deposited on the myofibrils. Furthermore, alterations in motor control could result in relatively less antagonist activity to oppose the intended movement.

However the apparent advantages of strength training for maintaining submaximal contractions may not always be advantageous for the maintenance of maximal contractions. The greater force output of the hypertrophic muscle increases energetic demands which are not equally compensated by increases in mitochondria and SR. Furthermore, the muscle hypertrophy which contributes to the greater force output may increase the difficulty in transporting oxygen and nutrients to the entire volume of the muscle.

Fatigue resistance has been reported to be unchanged, increased, or decreased following disuse in animals and humans. Alterations in muscle activation are not a major factor in the unchanged fatigue resistance since animal studies using tetanic stimulation have also demonstrated a maintenance of fatiguability. However, some human immobilization studies using high frequency

stimulation have shown increased fatiguability. Decreases in motor unit firing frequency with voluntary contractions in conjuction with an increased duration of the surface action potential may contribute to the viability of the membrane propagation. The increased duration of disused E-C coupling (prolonged Ca^{++} release), and smaller diffusion distances for atrophied disuse muscle could also contribute to the maintenance of the fatigue index. Finally a relatively greater ratio of SR to myofibrillar volume and a maintenance of intermyofibrillar mitochondria and myoglobin allow disused muscle to sustain prolonged muscular activity. In conclusion, an outcome of strength training adaptations is greater energetic demands on the muscle, while disuse adaptations conserve energy efficiency resulting in minimal changes in relative endurance.

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Table: 1 Fatigue-related changes of neural properties are related to the intensity of the contractions.

	Submaximal Contractions	Submaximal Contractions held until Maximal Exertion	Maximal Contractions
EMG	Increases[a]	Initial increase followed by decrease[b]	Decreases[c]
Muscle Activation	Increases[d]	Initial increase followed by decrease[e]	Decreases[f]
Rate Coding	Increases[g]	Initial increase followed by decrease[h]	Decreases[i]
Co-contractions	No change[j]	Decrease in tibialis anterior[k]	Decrease[l]

a: Bigland-Ritchie et al. 1986a, Krogh-Lund 1993, Loscher et al. 1994, Maton and Gamet 1989, Moritani et al. 1982, Vitissalo and Komi 1977
b: Bigland-Ritchie et al. 1986a, Dolmage and Cafarelli 1991,Duchateau and Hainaut 1993, Petrofsky and Philips 1985,
c: Bigland-Ritchie et al. 1983, Krogh-Lund and Jorgensen 1991, Moritani et al. 1986 Nilsson et al. 1977, Stephens and Taylor 1972
d: Dorfman et al. 1990
e: Sale 1988
f: Bigland-Ritchie et al. 1986b, McKenzie and Gandevia 1991, McKenzie et al. 1992, Newham et al 1991
g: Dorfman et al. 1990
h: Petrofsky and Philips 1985
i: Bigland-Ritchie et al. 1983, Grimby et al. 1981, Marsden et al. 1983
j: Psek and Cafarelli 1993
k: Behm and St-Pierre under review
l: Psek and Cafarelli 1993

Table 2: Fatigue-related changes of membrane and twitch contractile properties are related to the duration of the contractions.

	Short Duration Submaximal Contractions (~ 3 min)	Short Duration Maximal Contractions (~10 s)	Long Duration Submaximal Contractions (> 5 min)	Long Duration Maximal Contractions (~60 s)
M-Wave Amplitude	Potentiation[a]	No change or Potentiation[b]	Decreases[c]	Decreases[d]
Twitch Amplitude	Potentiation[e]	Potentiation[f]	Decreases[g]	Decreases[h]

a: Behm and St-Pierre, under review
b: Bellemare and Garzanti 1988, Bigland-Ritchie et al. 1979, Duchateau and Hainaut 1985, Pagala et al. 1984
c: Fuglevand et al. 1993
d: Bellemare and Garzanti 1988, Miller et al. 1987, Milner-Brown and Miller 1986, Pagala et al. 1984
e: Dolmage and Cafarelli 1991
f: Grange and Houston 1991, Houston and Grange 1990
g: Behm and St-Pierre under review
h: Grange and Houston 1991, Houston and Grange 1990, McKenzie and Gandevia 1991,

Table 3: Strength training adaptations of neural and contractile properties.

EMG	Studies have reported both increases[a] and no change[b] (could involve motor learning)
Muscle Activation[c]	No significant change in interpolated twitch ratio
Reflex Potentiation[d]	Increases
Rate Coding[e]	No change for force production but increases may be possibly related to rate of force production
Co-contractions	Studies have reported both increases[f] and decreases[g].
M-Wave[h]	No change
Twitch Amplitude	Studies have reported both increases[i], and no change[j]
Myofilaments[k]	Hypertrophy

a: Hakkinen and Komi, 1983, 1985, 1986, Hakkinen et al. 1985a, Sale 1988,
b: Cannon and Cafarelli 1987
c: Behm and St-Pierre, under review
d: Sale et al. 1983b
e: Behm 1995
f: Barrata et al. 1988, Osternig et al. 1986,
g: Carolan and Cafarelli 1992,
h: Duchateau and Hainaut 1984a, Sale et al. 1982
i: Duchateau and Hainaut 1984a, 1984b, Freeman and Luff 1982, Gonyea and Bonde-Petersen 1978 j: Alway 1989, 1990, Davies and McGrath 1982, McDonagh et al. 1983
k: Ikai and Fukunaga 1968, Maughan et al. 1983

Table 4: Disuse adaptations of neural and contractile properties

EMG[a]	Decreases
Muscle Activation[b]	Decreases
Reflex Potentiation[c]	Decreases
Rate Coding[d]	Less consistent
Co-contractions[e]	Increases (single study)
M-wave[f]	Increased duration
Twitch Amplitude	Increases[g], decreases[h] and no change[i] (amplitude parallels changes in duration)
Myofilaments[j]	Atrophy

a: Duchateau and Hainaut 1987, Edgerton et al. 1975, Fishback and Robbins 1969, Fuglsang-Frederiksen and Scheel 1978, Hnik et al. 1985, Santavirta 1979
b: Hurley et al. 1994, Rutherford et al. 1986
c: Sale et al. 1982
d: Duchateau and Hainaut 1987 Fudema et al. 1961
e: Behm and St-Pierre, under review
f: Duchateau and Hainaut 1987, 1990
g: Fitts and Brimmer 1985, Simard et al. 1982, St-Pierre and Gardiner 1985,1987, Witzmann et al 1982a,b, White et al. 1984
h: Fitts and Brimmer 1985, Reiser et al. 1988, Simard et al. 1982, Witzmann et al 1982a,b, White et al. 1984
i: Davies et al. 1987
j: Berg et al. 1991, Veldhuizen et al. 1993

Introduction to Muscle Inactivation: An Assessment of the Interpolated Twitch Technique

The review of literature illustrates the myriad of possible factors contributing to the similar fatigue indexes of healthy and disused muscles. One of the factors discussed was the possibility that decreases in muscle activation with disuse might result in the comparison of submaximal (disused muscle) and maximal (controls) contractions. A solution to this quandry would be to use either evoked stimulation or monitor voluntary activation levels to ensure all participants are contracting at the same relative intensity. Although muscle activation can be generally assessed by the EMG activity, it does not provide a precise measurement of activation levels. The interpolated twitch technique (ITT) has been utilized in an attempt to quantify the extent of muscle activation. However the precision of the technique has yet to be fully determined. Prior to conducting an investigation of muscle activation, it should be necessary to ensure the techniques for calculating muscle activation were reliable and valid.

Muscle Inactivation: An Assessment of the Interpolated Twitch Technique

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Abstract

The validity, reliability, and protocol for the interpolated twitch technique (ITT) were investigated with isometric plantarflexor and leg extension contractions. Estimates of muscle inactivation were attempted by comparing a variety of superimposed to potentiated evoked torques with submaximal and maximal voluntary contraction torques or forces (MVC). The use of nerve and surface stimulation to elicit the ITT were reliable, except for problems maintaining maximal stimulation with nerve stimulation at 20° plantarflexion and during leg extension. The interpolated twitch (IT) ratio-force relationship was best described by a shallow hyperbolic curve resulting in insignificant MVC prediction errors with second order polynomials (1.1%-6.9%). The prediction error under 40% MVC was approximately double that over 60% MVC, contributing to poor estimations of MVC in non-weight bearing post-immobilized ankle fracture patients. There was no significant difference in the ITT sensitivity using twitches, doublets or quintuplets. The ITT was valid and reliable when high intensity contractions were analysed with a second order polynomial.

Index Terms: Interpolated twitch, electrical stimulation, muscle activation, reliability, validity,

Introduction

The interpolated twitch technique (ITT) was first utilized by Merton (13) to observe possible muscle inactivation with a fatiguing protocol of the adductor pollicis. He superimposed an evoked stimulation on a voluntary contraction to detect the presence of muscle fibers not activated by the voluntary contraction. A number of researchers have used the ITT to demonstrate that full activation of the dorsiflexors (3) and quadriceps (Quads)(4, 6, 8) is possible in untrained individuals. Others have reported that not all their subjects could fully activate their plantarflexors (PF)(3) or elbow flexors (1, 11). Dowling et al. (9) reported that none of his subjects could fully activate their elbow flexors. Could the effective detection of muscle inactivation be related to the sensitivity of their techniques? Some studies have superimposed twitches (3, 6, 8) upon a voluntary contraction and visually inspected the contraction for evoked increases in force. The noise of a high intensity voluntary contraction may obscure the superimposed evoked torque diminishing the sensitivity of the measure. This may be particularly true in large muscles such as the Quads. Gandevia and McKenzie (10) utilized multiple stimuli (2-4) to increase the signal to noise ratio and inspected the superimposed torque after it was further amplified (10 X) and offset by a DC clamp amplifier. Dowling et al. (9) achieved a very high superimposed signal to noise ratio using a triggered averaging technique with an amplified (10 X) offset system. There have been very few studies (7, 12, 16) which have extensively investigated the ITT methodology. Do attempts to increase the signal to noise ratio with the ITT make a significant difference in the ability to detect muscle inactivation?

Rather than just detecting muscle inactivation, is the measurement of superimposed torque a valid tool for quantifying the extent of muscle activation? Although some studies have reported

a linear relation between the interpolated twitch (IT) ratio (superimposed torque / potentiated torque) and voluntary force (8, 15), others have shown the relation to be non-linear (3, 9, 11, 14, 16). Some researchers have assumed a linear relation and attempted to estimate the extent of muscle activation using a single data point (1, 5, 6, 15). A non-linear relation would not allow a simple extrapolation of muscle activation from a single IT ratio. Bulow et al. (7) reported that the curvilinear relationship between superimposed twitch torque size and Quads voluntary force was more linear when force levels greater than 25% of the maximum voluntary contraction (MVC) were used. Loring and Hershenson (12) studied the adductor pollicis and found the curvilinear relationship became more linear with a non-compliant loading device. Does the IT ratio-force relationship allow for a quantitative and sensitive measure of muscle inactivation?

Many of the previously cited studies have indicated that the subjects needed a number of trials to achieve full activation. Once the subject is accustomed to the experimental set-up can full activation be achieved repeatedly and reliably? Since there have not been any studies to systematically measure the reliability of the ITT with the PF or Quads, another objective of this study was to investigate the reliability of the ITT with different modes of stimulation, and ankle joint angles.

Methodology

Subjects. Table 1 summarizes the series of experiments and number of subjects. Subjects were recruited from McGill University students and staff. All subjects were fully informed of the procedures and signed a consent form prior to experimentation. The study was approved by McGill University's Ethics committee.

Experimental Set-up. For all voluntary and evoked contractile properties, the subjects

were seated in a straight back chair with their hips and knees flexed at 90°. For measurements taken at the knee, ankles were secured in a padded strap attached to a high tension wire clamped to a strain gauge perpendicular to the line of pull of the lower limb. PF subjects had their leg secured in a modified boot apparatus (3). Testing was done with the ankle in either a neutral position of 90°, or 20° of plantar flexion or dorsiflexion. Tibial nerve surface stimulating electrodes were placed on the popliteal space and distal portion of the triceps surae. Femoral nerve stimulating electrodes were placed on the inguinal triangle and buttocks. Electrodes were shifted during initial stimulation to determine the optimal position for the greatest peak torque. During bipolar stimulation, electrodes were secured to the superior and distal aspects of the triceps surae or Quads muscle groups. Polarity was reversed to determine the best arrangement for twitch torque.

Compound muscle action potentials (M-waves) were monitored with surface electromyographic (EMG) electrodes (Medi-Trace) placed 3-5 cm apart on the soleus or vastus lateralis distal to the stimulating electrodes A ground electrode was secured superficially to the head of the tibia. Thorough skin preparation for all electrodes included sanding of the skin around the designated areas followed by cleansing with an isopropyl alcohol swab. To ensure that the intensity of stimulation remained constant throughout the experiment, M-waves were monitored during data collection for all knee measurements (nerve and bipolar stimulation) and with the ankle at 90^0. M-waves were amplified (IHS 830 Isolation amplifier, BMA 830 EMG amplifier CWA Ardmore Pa.), filtered (10-1000 Hz) and monitored on oscilloscope (Tektronix Model 2220). The EMG signal was recorded at a sampling rate of 2000 Hz.

Evoked and Voluntary Torque. Stimulating electrodes were connected to a high voltage

stimulator (Digitimer Stimulator; Model DS7). The amperage (10-100 mA) and duration (500-2000 μs) of a 400-volt rectangular pulse was progressively increased in an attempt to obtain a plateau in the twitch torque. This was achieved in all PF subjects but only 9 of 16 Quads subjects. Doublets were elicited by two twitches with an interval of 10 ms (100 Hz). Supramaximum PF tetanic stimulation was evoked through the tibial nerve in a separate group of 8 subjects for 1.5 s at 100 Hz. All evoked and voluntary torque were detected by a force transducer (PF: custom design, Quads: BLH Electronics 3SB), amplified (recording amplifier and AC-DC differential amplifiers from Neurolog Systems-Model NL900A) and monitored on oscilloscope. All data were stored on computer (Seanix ASI 9000 486 DX) after being directed through an analog-digital board (Lab Master) (2000 Hz). Data were recorded and analysed with a custom designed software program (Actran; Distributions Physiomonitor Ltee.).

Interpolated Twitch Technique (ITT). Three doublets interspersed at 900 ms intervals were evoked and superimposed on a series of 3 second duration, submaximal (20, 40, 60, 80% of MVC) and three maximal voluntary contractions to estimate an average superimposed signal. Superimposed doublets rather than twitches were utilized to increase the signal to noise ratio. In addition doublets were recorded at 1 second intervals following the voluntary contractions. Superimposed and potentiated twitches and quintuplets (5 stimulations at 10 ms intervals: 100 Hz) were also used with ITT to determine possible changes in sensitivity. Torque signals were sent through both a low and high gain amplifier. The resident software program offset the high gained superimposed signal, 100 ms before each stimulation for improved resolution (Figure 1). A ratio was calculated comparing the amplitudes of the superimposed stimulation with the potentiated stimulation (IT ratio) to estimate the extent of inactivation during a voluntary

contraction. Since the potentiated evoked stimulation represents full muscle activation, the superimposed torque using the same intensity of stimulation would activate those fibres left inactivated by the voluntary contraction. All maximal and submaximal (100%, 80%, 60%, 40%, 20% of MVC) forces were correlated with their respective IT ratios in order to generate linear or second order polynomial equation for all subjects.

Statistical Analyses. Linear and second order regression equations were used to determine the line of best fit and validity of the data in predicting the MVC. Differences between the actual and predicted MVC, mode of stimulation (twitch vs doublet vs quintuplet) and tetanus torque versus MVC were analyzed separately using 1-way ANOVAs with repeated measures. A randomized 1 way ANOVA was used to compare group differences of the y intercept (a), slope (bx) and curvature of the slope (cx^2) as derived from the polynomial equations ($a + bx + cx^2$). Test-retest reliability was determined with the intra-class correlation (ICC) coefficient applied to the repeated measures ANOVA analysis for different ankle angles and forms of stimulation (17). F ratios were considered significant at $p<0.05$. If significant interactions were present, a Tukey post hoc test was conducted. Descriptive statistics include mean ± standard deviation (SD). Data in the figures are presented as mean ± standard error (SE).

Results

Description of the IT Ratio-Force Relationship. Increases in contraction intensity were correlated with decreases in the IT ratio. There were no significant changes in the M-wave amplitude at any contraction intensity with tibial nerve stimulation. The plotting of the 5 ratios (20, 40, 60, 80, 100% MVC) produced high linear regression values (r^2) for all angles: neutral: 0.90, 20° dorsiflexion 0.94, 20° plantarflexion 0.90. Higher values were found when the data were

fitted to a second order polynomial. R^2 values for the various angles were: neutral 0.99, 20° dorsiflexion 0.98, 20° plantarflexion 0.92. There was no significant difference in the IT ratio-force relationship of tibial nerve and bipolar PF stimulation (Figure 2). Thus with data collapsed across all PF groups, the IT ratio-force relationship was best fit by a shallow hyperbolic curve (Figures 3). Visual inspection of the ratios shows greater linearity at the lower contraction intensities with a tendency for the ratios to plateau at approximately 60-80% of the MVC.

The IT ratio-force relationship plateau could represent synergists not activated with evoked stimulation. To verify this hypothesis, maximal PF tetanic and voluntary PF torque were compared. Tetanic PF torque (65.6 Nm ± 19.1) was 18.7% less ($p<0.01$) than PF MVC (80.7 Nm ± 27.6).

The Quads IT ratios presented a similar relationship. The linear regression coefficients for bipolar ($r^2= 0.96$) and femoral nerve ($r^2= 0.95$) stimulated Quads IT ratios were slightly higher than PF IT ratios. The fit of a second order polynomial to the Quads was similar to the PF with r^2 values of 0.99 (Figure 4) and 0.96 respectively. Difficulties were encountered with maintaining the intensity of femoral nerve stimulation. M-wave amplitudes elicited during rest (7 mv ± 3.1) were significantly ($p> 0.001$) larger than M-waves elicited during a maximal voluntary contraction (6.2 mv ± 2.9). There was no significant difference in bipolar stimulated M-wave amplitudes between rest and voluntary contractions. The plateau of the PF IT ratios at higher contraction intensities was not as evident with the Quads.

A comparison of the values representing the y intercept (a), slope of the line (bx) and curvature of the line (cx^2) from the second order polynomial equation ($a + bx + cx^2$) demonstrated no significant difference between PF and Quads.

Validity. If the IT ratios can be used to measure the extent of muscle inactivation, then this information should allow a prediction of an individual's true MVC when not fully activated. Table 2 illustrates the poor ability of the PF and Quads IT ratio to predict the MVC of fully activated individuals when a single IT ratio is utilized. The significant errors in predicting the MVC with single IT ratios and linear equations were eliminated with second order polynomial equations (Table 2).

Further analysis of the IT ratios indicated the prediction of MVC to be less accurate when low contraction intensities were utilized with the second order polynomial. Table 3 demonstrates the improved prediction of MVC when contraction intensities above 40% MVC were included (5.8%-16.3% of MVC). The exclusion of IT ratios above 40% of MVC resulted in more than double the prediction error (33.3% of MVC). A number of subjects when tested at 20% of MVC exhibited IT ratios greater than 1 suggesting over 100% inactivation. Anomolous MVC predictions were found with two non-weight bearing previously immobilized ankle fracture subjects The prediction of one patient overestimated the contralateral MVC by 71.1%. Even when considering bilateral limb differences, this MVC prediction would be improbable due to the effects of disuse atrophy.

Sensitivity. In order to determine if the frequency of the superimposed signal influences the sensitivity of the ITT to detect the lack of full activation, single, doublet and quintuplet stimulation were compared at the neutral angle. The larger summated torques of the doublet and quintuplet should increase the signal to noise ratio and possibly improve the sensitivity and thus the predictability of the ITT. Using the best fit equation (second order polynomial), quintuplet stimulation showed a slightly better (3.3% ± 2.3) but statistically insignificant improvement in

MVC prediction than with either doublet (5.5% ± 2.9) or single stimulation (5.4% ± 3.2).

All the protocols in this study compared the amplitude of the superimposed torque to the potentiated evoked torque (single, doublet, quintuplet) immediately following the voluntary contraction. To further determine the best exponential fit, further analysis examined the effectiveness of comparing the superimposed doublet to an unpotentiated doublet. There was no significant difference in the prediction of the observed MVC with a potentiated doublet or an unpotentiated doublet.

Reliability. Table 4 illustrates the high to very high reliability of all variables at all ankle angles with tibial nerve and bipolar stimulation except for the IT ratio with tibial nerve stimulation at 20° plantarflexion. Quads' also had high to very high reliability of all variables with femoral nerve and bipolar stimulated ITT, except for the bipolar stimulated twitch (Table 4). In addition 8 healthy subjects had both limbs subjected to the ITT. In all cases, MVC force (92.7 vs 98.5 Nm), the extent of muscle activation (97.6 vs 98.7%), and the regression values for the IT ratio-force relationship (0.99 vs 0.95), were similar for both limbs.

Discussion

ITT Validity. One of the most important findings of this study was the inability of the ITT to predict the MVC from a single submaximal IT ratio. A perfectly linear relation between the IT ratio and force would allow a single IT ratio to accurately predict an individual's MVC. For example in a perfectly linear relationship, a superimposed twitch / potentiated twitch ratio of 0.25 would result in a muscle force equal to 75% of the MVC. Linear relationships between superimposed twitch and force levels have been reported with the adductor pollicis (12) and Quads (8). This study however found a better fit with a shallow hyperbolic curve for both the PF

(Figure 3) and Quads (Figure 4). Other researchers have reported that the decrease in superimposed twitch force with increasing voluntary force results in a shallow hyperbolic relationship with the PF and dorsiflexors (3), Quads (7, 14, 16) and elbow flexors (9). A hyperbolic curve best predicts the X intercept (MVC) with a second order polynomial equation which is an exponential rather than linear function. The non-linearity of the slope, however does not permit an accurate prediction of the MVC from a single IT ratio. Although the latter could be used as a general indication of muscle inactivation. Researchers however must be cautious in extrapolating quantitative measures from a single data point.

Similar caution should be exercised when the ITT depends on submaximal contractions to predict the MVC. This study has demonstrated that using contraction intensities under 40% of the MVC resulted in an unacceptable 33.3% error. The use of submaximal contractions under 80% of MVC still resulted in a 13-16% error (Table 3). This is similar to the findings of Bulow et al. (7) who stated that an intensity of 75% of muscle force is necessary to achieve a sufficiently accurate prediction. Maximal or near maximal contractions should be utilized to obtain the most accurate prediction of the MVC.

Another interesting finding was the disproportionately large superimposed doublets at low contraction intensities (20% of MVC). In many subjects contracting at 20% of MVC, the amplitude of the superimposed doublet was equal to or greater than the potentiated doublet. In order to produce a contraction, a portion of the muscle must have been activated and thus a smaller superimposed torque than the potentiated evoked torque would be expected. The disproportionately large doublets would create a gross overestimation of the muscle inactivation at that contraction intensity as well as contributing to the non-linearity of the IT ratio-force

relationship. Bulow et al. (7) reported a non-linear twitch-voluntary force relationship at contraction intensities under 25% of MVC, resulting in resting twitches as well as superimposed twitches at 10% of MVC to be smaller than the superimposed twitches on 20-25% MVCs. They attributed the smaller resting and low intensity twitches to visco-elastic force loss, suggesting force is dissipated when it is transferred from the stimulated to the non-stimulated portion of the muscle. In addition there would be additional force loss in the attempt to transfer force through subcutaneous fat and connective tissue. Belanger and McComas (3) hypothesized that the reduction in the slack of the series elastic component with weak contractions would contribute to a larger superimposed than resting twitch. The inability to produce maximal or near maximal contractions would limit the applicability of the ITT to predict MVC in some patients who are unable to produce strong contractions because of pain, swelling or apprehension.

In a study investigating the effects of muscle fatigue following ankle fractures, we attempted to use the ITT to predict the MVC in order to compare patients working at the same intensity (relative to predicted MVC). This was not possible in patients tested within 1-2 weeks of cast removal because subjects were either unable or unwilling to generate high intensity contractions. The predicted MVC of one of the subjects exceeded the contralateral MVC by 71.1%, which given the presence of atrophy, is highly unlikely. An inspection of the non-weight bearing IT ratio-force curve (Figure 6) illustrates a severe hyperbola with little distribution of the data points. The inability to accurately predict MVC may be related to the large error experienced by normal subjects when only low intensity contractions are utilized. Therefore the ITT may only be useful in estimating muscle inactivation in patients that can generate relatively strong contractions.

Mechanisms of the IT Ratio-Force Relationship. The plateau of the IT ratio-force relationship at high contraction intensities has been found by other researchers (3, 9, 12, 14, 16). The high intensity plateau may be related to the contribution of synergists to the total force output. Although the majority of PF torque is produced by the gastrocnemius and soleus innervated by the tibial nerve, there is a contribution by the peroneus longus and brevis innervated by the peroneal branch of the lateral popliteal nerve (2). The plateau of the IT ratio-force relationship at high contraction intensities may signify nearly full activation of the triceps surae while extra force is contributed by the peronei whose activation levels remain undetected by the ITT (3). In order to verify this assumption, we compared the maximum PF tetanic force to the MVC. Tibial nerve stimulated tetanic torque was 18.7% less than the MVC. This disparity is very similar to the contraction intensities at which the plateau effect commences in the PF of this study. This could also be an adequate explanation for the plateau in Dowling et al.'s (9) data examining the ITT of the bicep brachii. Their plateau may reflect the extra contribution of the brachioradialis to elbow flexion. The Quads however do not have any synergists contributing to leg extension torque. The Quads IT ratio-force relationship is also more linear (0.96) than the PF (0.9-0.94). Thus synergistic activity may have a significant contribution to the non-linearity of the PF IT ratio-force relationship.

Another possibility was offered by Loring and Hershenson (12) who investigated the effect of series compliance on the superimposed twitch of the adductor pollicis. The relationship of superimposed twitch forces when attached to a compliant link resembled the shallow hyperbolic curve illustrated in this study and others. A non-compliant link however resulted in a more linear superimposed twitch-voluntary force relationship. The compliance associated with the

relatively long achilles and patellar tendons may contribute to the shallow hyperbolas in this study. The placement of the ankle at a neutral testing angle as well as 20° PF and dorsiflexor would be expected to have some effect on the compliance of the achilles tendon. Although placing the achilles tendon under greater stretch with dorsiflexion did result in a slightly more linear relation (0.98) than with plantarflexion (0.92), there was no significant difference in the prediction of MVC. Even though not significantly different, the greater linearity of the stretched dorsiflexor position does provide some corroborating evidence to the Loring and Hershenson proposal.

Protocol Sensitivity. It may also be hypothesized that the high intensity plateau effect may be related to a low signal to noise ratio. Difficulty in ascertaining the true amplitude of the superimposed doublet amongst the noise and fluctuations of a high intensity contraction may contribute to the plateau effect. A larger signal to noise ratio may be achieved by increasing the amplitude of the superimposed stimulation. Single, doublet and quintuplet stimulation were compared to determine if the greater summated torques of doublets and quintuplets would improve the signal to noise ratio. Although the quintuplet showed a slightly more linear relation (r^2= 0.96) than the single (r^2= 0.94) or doublet (r^2= 0.92) stimulation, the data was still best fit by a secord order polynomial (quintuplet: r^2= 0.99, doublet: r^2= 0.97, single: r^2= 0.98). There was no significant differences in the prediction of MVC with the three forms of stimulation.

A similar lack of difference was found when investigating the effectiveness of comparing the superimposed doublet to either a potentiated doublet or unpotentiated doublet. The most important factor seems to be the resolution of the superimposed torque. Gandevia and McKenzie (10) improved their resolution with an offset DC clamp amplifier. Dowling et al. (9) achieved high resolution of their superimposed doublet by utilizing triggered averaging with an amplified offset

to enhance the signal to noise ratio. Dual amplification (low and high gain amplifiers, computer software) of the signal as well as precise temporal measurement of the offset signal allowed for high resolution in this study.

Reliability. A technique must not only be valid but reliable as well. Test-retest reliability measures were very highly correlated for both nerve and bipolar stimulation of the PF (Table 4). A moderate level of reliability was found with tibial nerve stimulation of the ankle plantarflexed at 20° (Table 4). The lower level of reliability at 20° plantarflexion could be attributed to a movement of the stimulating electrode away from the optimal position on the popliteal surface. Subjects had a tendency to involve the hamstrings when contracting at 20° plantarflexion, pushing the electrode from its original position, decreasing the intensity of the stimulation.

The very high reliability of the Quads IT ratio with bipolar stimulation (0.96) contrasted with the less stringent but still highly correlated reliability with femoral nerve stimulation (0.78)(Table 4). This may also be related to electrode displacement. Quads M-wave amplitudes elicited during rest were significantly ($p < 0.0001$) larger than M-waves elicited during a maximum voluntary contraction. The contraction of the Quads during a maximum voluntary contraction probably displaced the stimulating electrode from its optimal position over the femoral nerve in the inguinal space. Although Rutherford et al. (16) did not find a significant difference in the Quads IT ratio-force relationship with femoral nerve or percutaneous stimulation, they surmised that the effectiveness of the ITT was not dependent upon full evoked activation. To improve reliability, bipolar muscle stimulation or improved placement of nerve stimulating electrodes is necessary to ensure the same proportion of muscle is stimulated with all contractions.

The moderate reliability of bipolar stimulation twitch did not affect the very high reliability

of the bipolar PF and Quads IT ratio (Table 4). Lower twitch reliability may be attributed to inaccurate placement of electrodes from test to test. Smaller inter-electrode distance could result in less muscle mass stimulated, affecting the amplitude of the stimulated torque. This would not severely affect the ITT since the IT ratio is derived from the superimposed and potentiated torque stimulated from the same proportion of muscle. Although the absolute amount of muscle stimulated from test to test may differ the ratio should not be affected.

Summary. Estimating MVCs by using second order polynomial equations derived from IT ratio-force relationships, were not statistically different from the observed MVCs of subjects able to maximally activate, thus providing an acceptable estimation of muscle activation. The exclusion of maximal or near maximal voluntary contractions in the polynomial equations resulted in significant MVC prediction errors. The use of single submaximal IT ratios or linear equations resulted in MVC prediction errors of 21.5% and 10.9% respectively. The shallow hyperbolic curve of the IT ratio-voluntary force relationship was not significantly altered when using either single, doublet or quintuplet stimulation for the superimposed and potentiated torque. The non-linearity of the relationship may be attributed to synergistic muscle contribution and/or compliance of the system. The technique was shown to be reliable in both the PF and Quads.

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Table 1: Experimental Protocol and Subject Characteristics

Experiment	# of Subjects	Age (± SD)
1. PF ITT at 3 angles (neutral, plantarflexion, dorsiflexion) using tibial nerve stimulation	8	26 ± 5.4
2. PF ITT at 3 angles (neutral, PF, dorsiflexion) using bipolar muscle stimulation	6	23 ± 3.5
3. Quads ITT using femoral nerve stimulation	10	24 ± 4.1
4. Quads ITT using bipolar muscle stimulation	6	26 ± 6.4
5. Comparison of the sensitivity of superimposed single, doublet, and quintuplet stimulation	6	25 ± 3.8
6. Tetanus torque versus PF MVC	8	26 ± 5.7
7. Non-weight bearing post-immobilized ankle fracture ITT	2	28 ± 4

Plantarflexors (PF), quadriceps (Quads), interpolated twitch technique (ITT)

Table 2: Percent difference between the predicted and true MVC of PF and Quads from IT ratios.

	20° Dorsiflexion	Neutral Position	20° Plantarflexion	Quads
Individual Data Points	15.2% ± 6.1*	23.3% ± 4.1*	25.7% ± 4.5*	18.3% ± 5.2*
Linear Equation	8.4% ± 2.3*	12.1% ± 3.1*	12.2% ± 1.9*	7.8% ± 4.9*
Second Order Polynomial	6.9% ± 1.8	5.5% ± 2.3	1.1% ± 0.9	4.6% ± 2.5

Data includes means ± SD. Asterisks (*) indicate significant difference from the observed MVC ($p<0.05$).

Table 3: Error and predicted force (Nm) obtained, using IT ratios derived from different contraction intensities.

Contraction Intensities	0-100%	0-80%	0-60%	0-40%
Predicted Force (Nm)	87.2 ± 16.8	77.5 ± 17.8	79.9 ± 23.2	61.7 ± 25.6
Difference between observed and predicted force (%)	5.8%	16.3%	13.7%	33.3%

Data includes means ± SD.

Table 4: Reliability (ICC) of PF and Quads voluntary and evoked contractile characteristics with tibial, femoral nerve and bipolar stimulation.

	20° Dorsiflexion		Neutral Position		20° Plantarflexion		Quadriceps	
	Nerve	Muscle	Nerve	Muscle	Nerve	Muscle	Nerve	Muscle
IT Ratio	0.93	0.99	0.99	0.96	0.67	0.84	0.78	0.96
MVC	0.93	0.91	0.97	0.95	0.82	0.99	0.99	0.99
Doublet	0.97	0.91	0.89	0.90	0.98	0.75	0.90	0.67
Twitch	0.81	0.65	0.86	0.74	0.84	0.78	0.83	0.54
TPT	0.99	0.99	0.95	0.95	0.99	0.86	0.92	0.90
1/2 RT	0.92	0.90	0.87	0.92	0.90	0.90	0.89	0.91

Intraclass correlation coefficient (ICC), superimposed doublet / potentiated doublet (IT ratio), torques or forces of maximum voluntary contractions (MVC), potentiated doublets, and twitches, as well as time to peak twitch (TPT) and half relaxation time (1/2 RT).

Figure Legends

Figure 1: Both diagrams illustrate the voluntary force output of an individual with 3 superimposed doublets followed by 2 potentiated doublets. In the top diagram, the strain gauge torque signals were passed through a low gain amplifier (amplification 1000X). The bottom diagram shows the same force output and series of stimulations with the strain gauge signal passed through both low and high gain amplifiers (amplification 10000X). The high gain signal is offset to baseline by the computer software program.

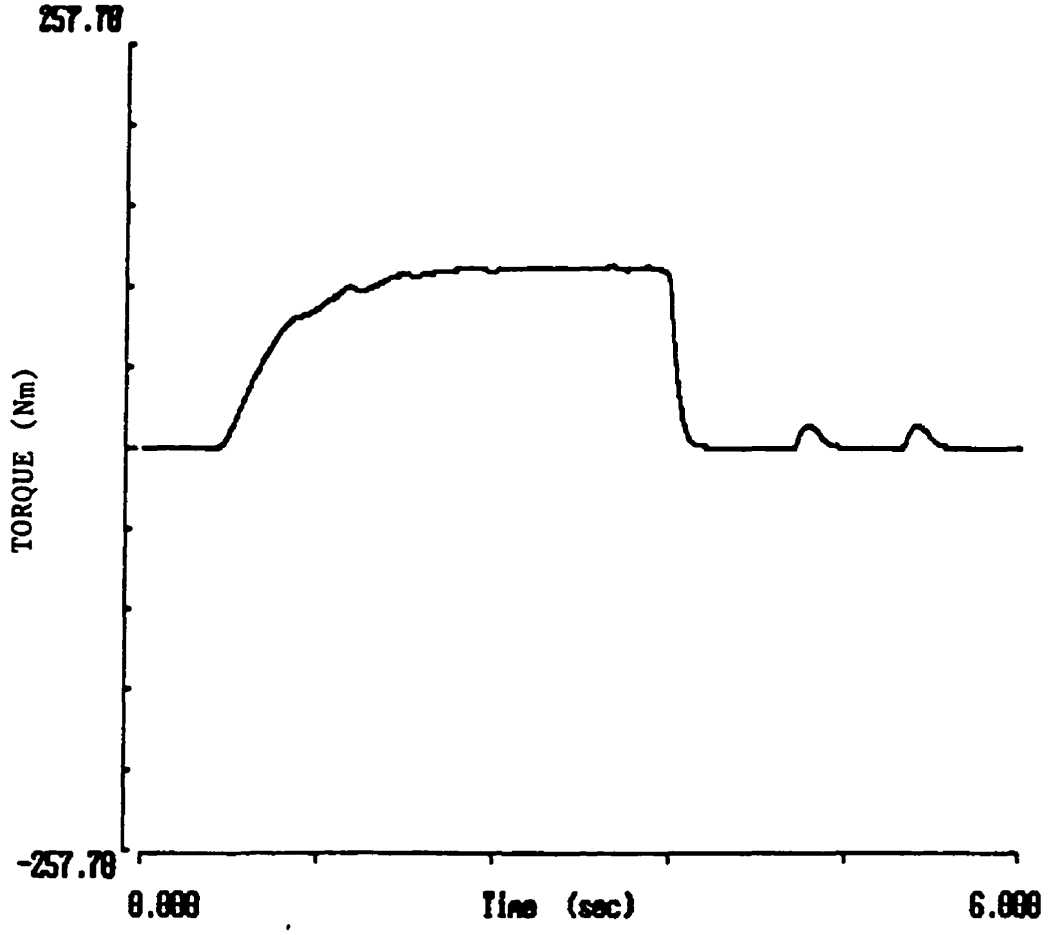
Figure 2: PF Interpolated twitch (IT) ratio-voluntary force relationship (neutral angle): raw data (n = 6). Superimposed doublet / potentiated doublet (PotD) = IT ratio. Each symbol represents a different subject.

Figure 3: Combined PF IT ratio-voluntary force relationship. Horizontal and vertical bars represent ± SE. Superimposed doublet / potentiated doublet (PotD) = IT ratio

Figure 4: Quads IT ratio-voluntary force relationship. Horizontal and vertical bars represent ± SE. Superimposed doublet / potentiated doublet (PotD) = IT ratio

Figure 5: Non-weight bearing post-immobilized IT ratio-voluntary force relationship of a single subject. Circles represent affected limb. Squares represent contralateral limb. Superimposed doublet / potentiated doublet (PotD) = IT ratio

Figure 1


257.70
TORQUE (Nm)
-257.70
0.000
Time (sec)
6.000

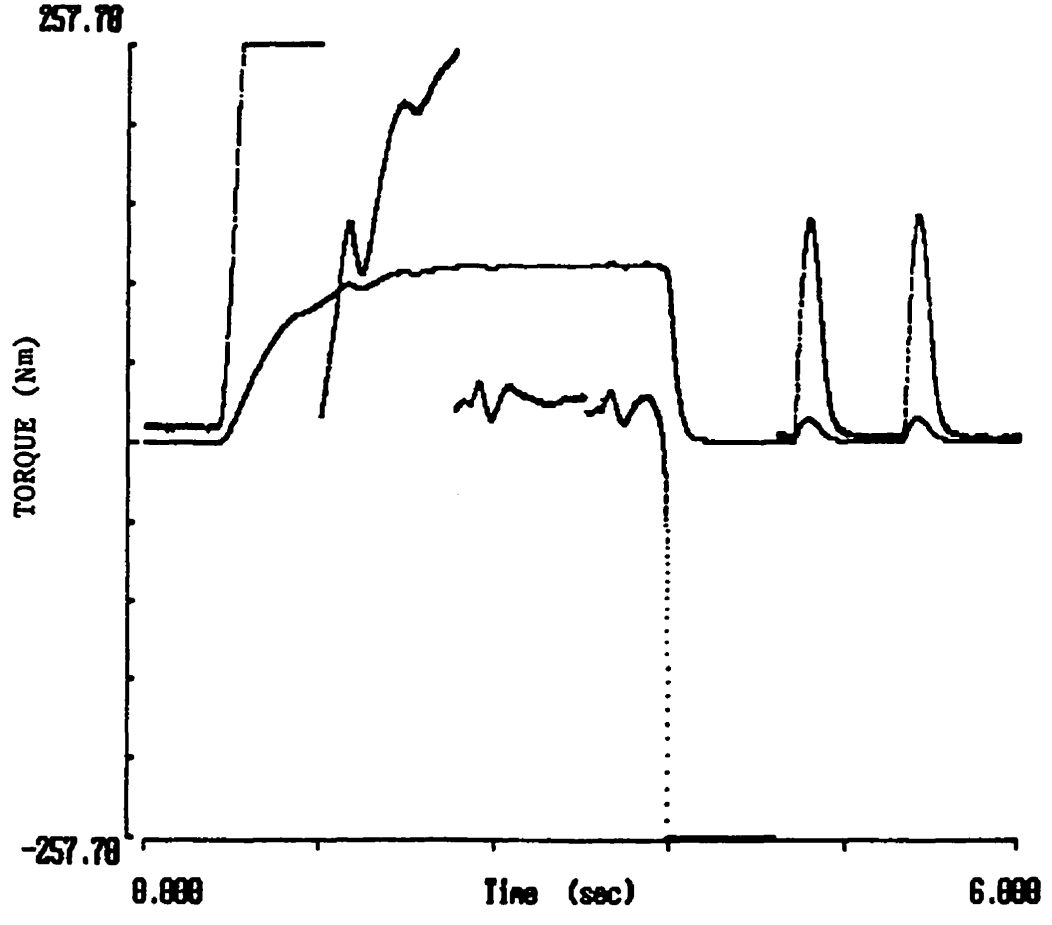

257.70
TORQUE (Nm)
-257.70
0.000
Time (sec)
6.000

Figure 2

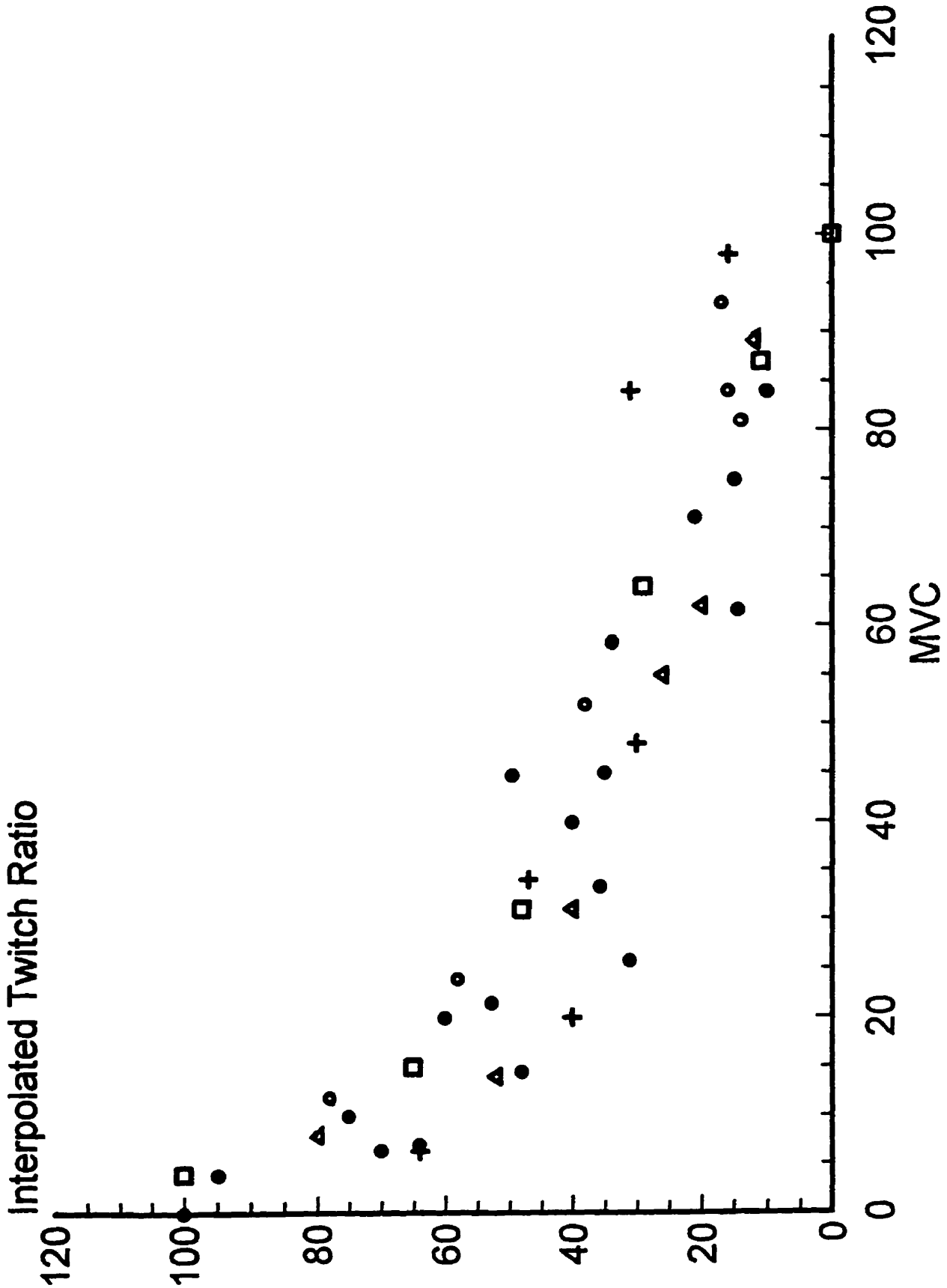

Interpolated Twitch Ratio
120
100
80
60
40
20
0
0
20
40
60
80
100
120
MVC

Figure 3

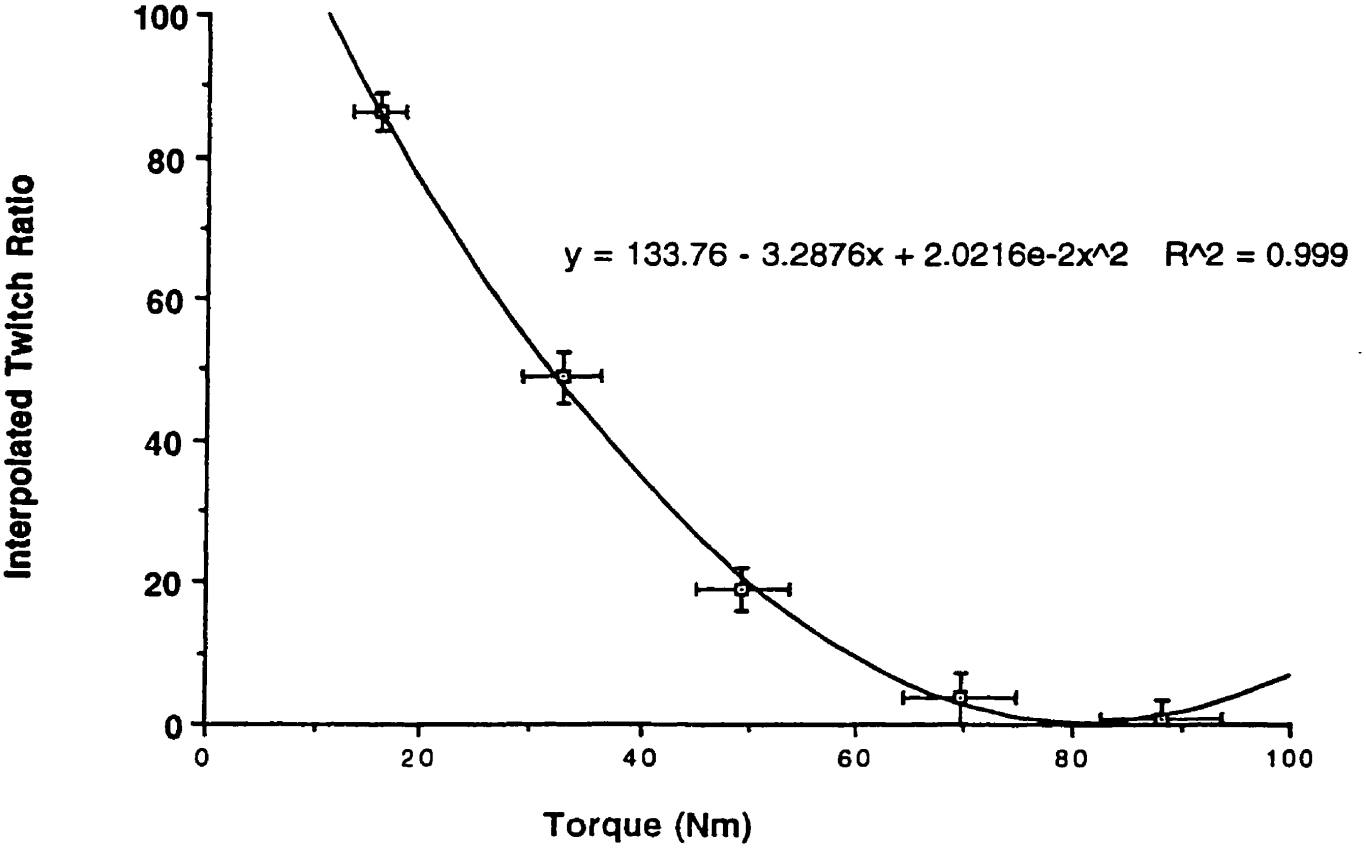

Interpolated Twitch Ratio
100
80
60
40
20
0
0
20
40
60
80
100
Torque (Nm)
y = 133.76 - 3.2876x + 2.0216e-2x^2 R^2 = 0.999

Figure 4

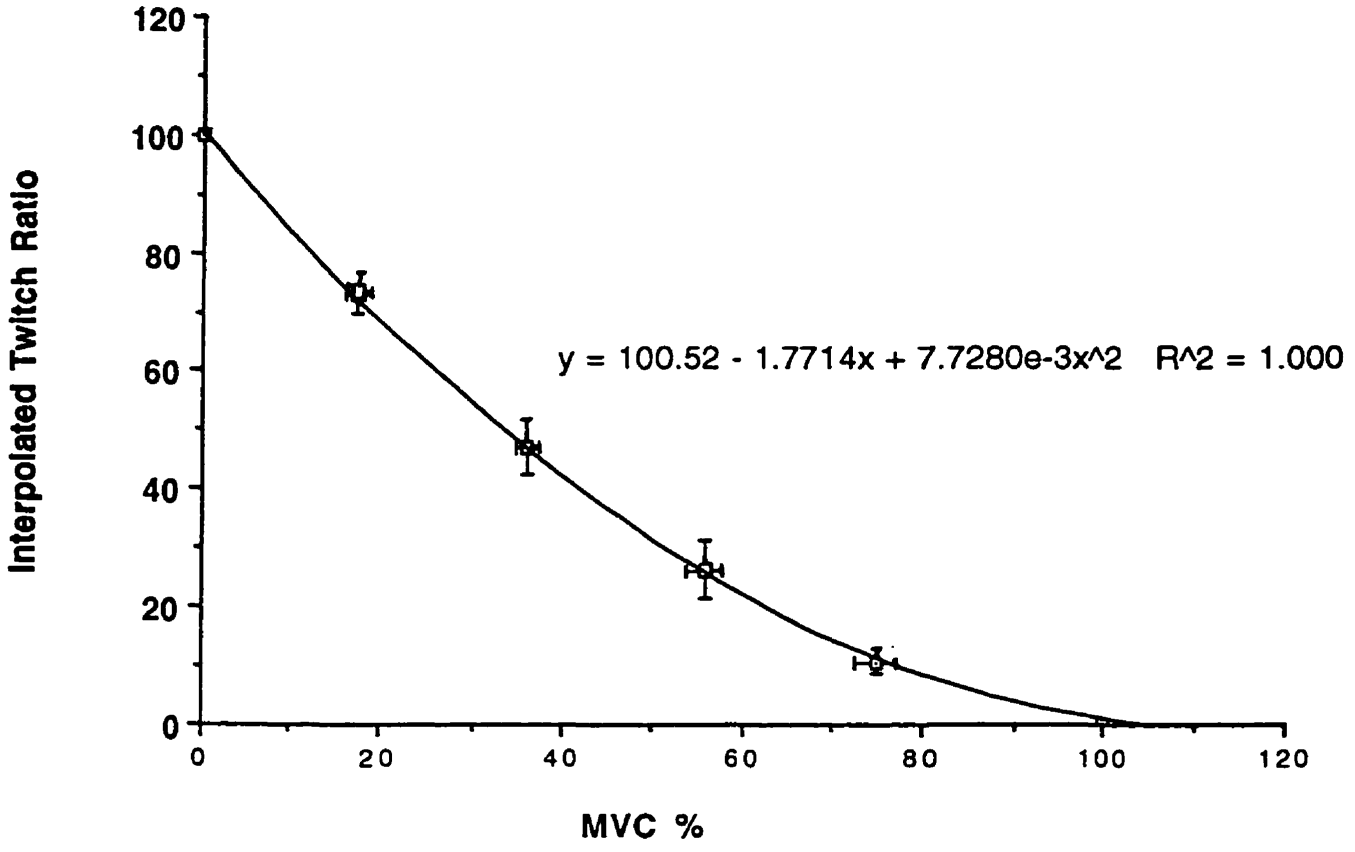
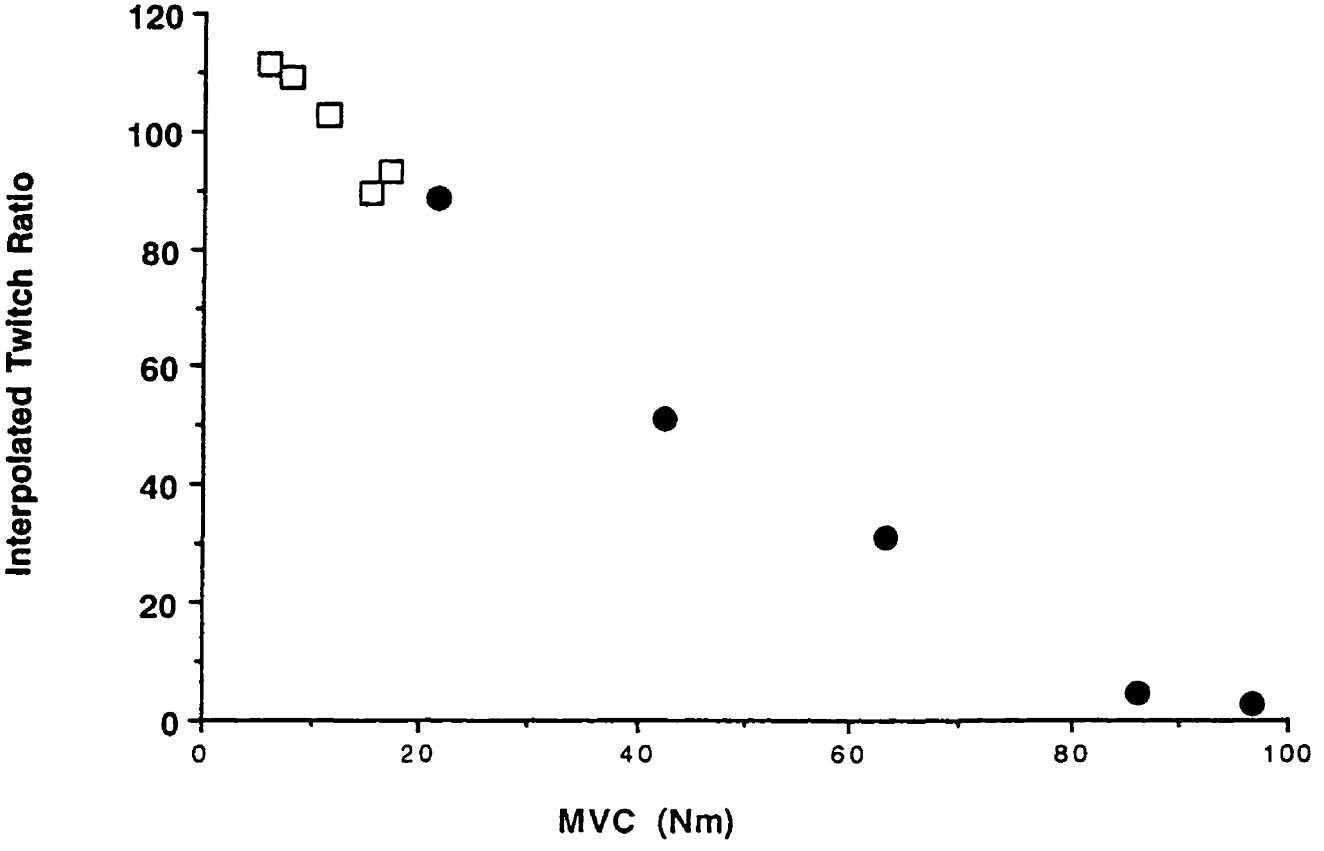

Interpolated Twitch Ratio
120
100
80
60
40
20
0
0
20
40
60
80
100
120
MVC %
y = 100.52 - 1.7714x + 7.7280e-3x^2 R^2 = 1.000

Figure 5


Interpolated Twitch Ratio
120
100
80
60
40
20
0
0
20
40
60
80
100
MVC (Nm)

Introduction: The Muscle Activation-Force Relationship Is Unaffected By Ischaemic Recovery

The previous paper indicated that the muscle activation-force relationship is best described by a shallow hyperbolic curve. Although the interpolated twitch (IT) ratio may be used as a general estimate of muscle activation with contractions over 40% MVC, the use of a second order polynomial equation provides more precision. A number of studies have used a single IT ratio from a MVC to investigate the effects of fatigue upon muscle activation.

Fatigue can have dramatic results upon muscle performance including disruption of muscle strength, activation, compound muscle action potentials, and excitation-contraction coupling. Whereas the IT ratio is based on the ratio of superimposed and resting evoked torques, fatigue-induced changes in muscle kinetics may affect the ITT. An alteration in the ratio of superimposed and resting evoked torques could then be attributed to either changes in muscle activation or muscle kinetics leaving the validity of the technique in doubt. Therefore it would be important to ascertain whether the ITT is as valid following fatigue as it is in the resting condition.

Furthermore, the recovery time course of muscle characteristics are not equivalent. While some contractile properties may recover quickly, others may be prolonged. Thus ischaemic conditions must be utilized to ensure that deficits accrued during fatigue are maintained during the testing period.

The Muscle Activation-Force Relationship Is Unaffected by Ischaemic Recovery

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Submitted to the Canadian Journal of Applied Physiology

Key Words: fatigue, interpolated twitch technique, second order polynomial, ischaemia

Abstract

The effects of fatigue and recovery on the plantarflexors' activation-force relationship were investigated with an isometric, intermittent, submaximal, fatigue protocol. Voluntary and evoked force and muscle activation were tested pre-, and post-fatigue with ischaemic (recovery inhibited) and non-ischaemic recovery. The muscle activation-force relationship of ischaemic and non-ischaemic groups was best described by a second order polynomial equation with similar y intercepts, slopes, and curvature of the slopes. A significantly increased muscle activation-force slope during recovery may be attributed to decreased muscle activation and not impaired muscle kinetics. The index of muscle activation immediately post-fatigue was not significantly different between ischaemic and non-ischaemic groups (88.5% vs 92.7%). However the significantly larger difference in the predicted and observed MVC of the ischaemic and non-ischaemic groups over the entire recovery period may reflect the disruptive effects of ischaemia on twitch kinetics affecting the estimation of muscle activation. No significant difference in the estimate of muscle activation post-fatigue with polynomials and interpolated twitch (IT) ratios (superimposed / potentiated doublets) suggested that IT ratios can be used as a general estimate of muscle inactivation following fatigue.

Key Words: muscle activation, interpolated twitch technique, fatigue, ischaemia

Introduction

The interpolated twitch technique (ITT) has been used as an estimate of muscle activation in a variety of fatigued (Bigland-Ritchie et al. 1978, 1983, 1986, Merton 1954, Newham et al. 1991, Vollestad et al. 1988) and non-fatigued young (Belanger and McComas 1981, Bellemare et al. 1983, Dowling et al. 1994, Gandevia and McKenzie 1988, Rutherford et al. 1986) and aged (Vandervoort and McComas 1986) individuals as well as with clinical populations (Allen et al. 1994, Lloyd et al. 1991, Minotti et al. 1992, Norregard et al. 1994, Rice et al. 1992, Rutherford et al. 1986, 1990). The technique involves the detection of superimposed evoked torques upon a voluntary contraction. Many studies have calculated a ratio between the amplitude of the superimposed torques and resting torques (interpolated twitch ratio) to quantify the extent of muscle inactivation (Allen et al. 1994, Bigland-Ritchie et al. 1978,1986, Rice et al. 1992).

A number of fatigue studies have used a single interpolated twitch (IT) ratio to estimate the extent of fatigue-induced muscle inactivation (Bigland-Ritchie et al. 1978,1986, McKenzie and Gandevia 1991), based on the assumption of a linear relationship between muscle activation and force output which is not affected by fatigue. However, the IT ratio-force relationship is best described by a second order polynomial (Bulow et al. 1993, Dowling et al. 1994), so that a single IT ratio cannot be used as an accurate estimate of muscle activation. Furthermore, the IT ratio-force relationship may be affected by fatigue, making comparisons of pre- and post-fatigue activation levels invalid. Reports that fatigue does not affect (Bigland-Ritchie et al. 1978, 1983, Merton 1954, Vollestad et al. 1988) or results in decreases in muscle activation as measured by the IT ratio (Ikai et al. 1967, McKenzie and Gandevia 1991) could be affected by fatigue-induced changes in the kinetics of the superimposed and resting torques (IT ratio). Thus one of the

objectives of this study was to determine if changes occured in the muscle activation (IT ratio)-force relationship following fatigue in order to determine if a single IT ratio could be used to estimate changes in activation levels post-fatigue.

The response of voluntary and evoked contractile properties changes over time with recovery from fatigue (Alway et al. 1987, Kroon and Naeije 1988, Garland et al.1988, McKenzie and Gandevia 1991). Ischaemic conditions have been utilized in a variety of studies to prolong the effects of fatigue during the recovery period (Woods et al. 1987, Garland and McComas 1990). Possible alterations in the IT ratio-force relationship may be more dramatic under ischaemic recovery conditions. Therefore, ischaemic and non-ischaemic recovery conditions were investigated to compare the effects of normal and prolonged recovery on the IT ratio force-relationship.

Experimental Design and Methodology

SUBJECTS

Table 1 summarizes subjects' characteristics. Subjects were recruited from McGill University students and staff. All subjects were fully informed of the procedures and signed a consent form prior to experimentation. The study was approved by McGill University's Ethics committee.

EXPERIMENTAL SET-UP

Subjects were seated in a straight back chair with their hips, knees, and ankles flexed at 90^0. Subjects had their leg secured in a modified boot apparatus (Belanger and McComas 1981). Tibial nerve surface stimulating electrodes were placed on the popliteal space and distal portion of the triceps surae. Electrodes were shifted and polarity reversed during initial stimulation to

determine the optimal arrangement for the greatest peak torque. Thorough skin preparation for all electrodes included sanding of the skin around the designated areas followed by cleansing with an isopropyl alcohol swab.

EVOKED AND VOLUNTARY TORQUE

Stimulating electrodes were connected to a high voltage stimulator (Digitimer Stimulator; Model DS7). The amperage (10 mA-1A) and duration (50-100 μs) of a 100-volt rectangular pulse was progressively increased until a plateau was achieved in the peak twitch torque indicating maximum stimulation. Doublets were elicited by two twitches with an interval of 10 ms (100 Hz). All evoked and voluntary torque were detected by a force transducer (PF: custom design, Quads: BLH Electronics 3SB), amplified (recording amplifier and AC-DC differential amplifiers from Neurolog Systems-Model NL900A) and monitored on oscilloscope (Tektronix Model 2220). All data were stored on computer (Seanix ASI 9000 486 DX) after being directed through an analog-digital board (Lab Master) (2000 Hz). Data were recorded and analysed with a custom designed software program (Actran; Distributions Physiomonitor Ltee.).

INTERPOLATED TWITCH TECHNIQUE (ITT)

Three maximal doublets interspersed at 900 ms intervals were evoked and superimposed on a series of 3 s duration, submaximal (20, 40, 60, 80%) voluntary contractions to estimate an average superimposed signal. Only the smallest or occluded superimposed signal was recorded with MVCs (3 trials). In addition to eliciting a doublet from a previously relaxed muscle, two potentiated doublets were recorded at 1 s intervals following the voluntary contractions. Superimposed doublets rather than twitches were utilized to increase the signal to noise ratio. Torque signals were sent through both a low and high gain amplifier. The resident software

program offset the high gain superimposed signal, 100 ms before each stimulation for improved resolution (Figure 1). A ratio comparing the amplitudes of the superimposed doublets with the potentiated doublet (IT ratio), represented muscle force which was not voluntarily activated. The percentage of recruited force was estimated from a single IT ratio by subtracting the ratio from a value of 1 and multiplying by 100 to represent an index of muscle activation during a voluntary contraction pre- and post-fatigue. All maximal and submaximal contraction (100%, 80%, 60%, 40%, 20% of MVC) forces were correlated with their respective IT ratios in order to generate second order polynomial equations for all subjects.

FATIGUE

After testing, the subject proceeded with the fatigue test. The fatigue protocol had the subject gradually increase the intensity of their contraction for 3 s until 50% of the MVC was attained. This intensity was maintained for 10 s, followed by a 3 s gradual decrease to a resting state. The sequence was resumed after a 4 s rest period. The contraction cycles (work:rest ratio of 16 s: 4 s) continued until the effects of fatigue disrupted the subject's ability to maintain the 50% MVC for the 10 s period.

RECOVERY

Groups were tested during a 2 min recovery period either under ischaemic or non-ischaemic conditions. Ischaemia was initiated immediately post-fatigue, in an attempt to ensure that fatigue-related muscle deficits were maintained during recovery testing. A deflated air pressure cuff was placed over the proximal segment of the calf prior to the fatigue test, and was inflated and maintained at over 200 mm Hg during the recovery period. Both groups performed a MVC with the IT immediately post-fatigue followed by a series of randomly selected submaximal

contractions (20, 40, 60, 80% of post-fatigue MVC) with IT every 30 s. The ischaemic group had the cuff deflated and removed 2 min into the recovery period.

STATISTICAL ANALYSES

Second order polynomial regression equations were used to determine the line of best fit for the IT ratio-force relationship. Calculations from the second order polynomial equations were used to compare observed and predicted MVCs pre- and post-fatigue. Differences between the observed and predicted MVCs were analyzed using 1-way ANOVAs with repeated measures. A randomized 1 way ANOVA was used to compare group differences for the y intercept (a), slope (bx), and curvature of the slope (cx^2) as derived from the second order polynomial equations ($a + bx + cx^2$). F ratios were considered significant at $p<0.05$. If significant interactions were present, a Tukey post hoc test was conducted. Descriptive statistics include mean ± standard deviation (SD). Data in the figures are presented as mean ± standard error (SE).

Results

PRE-FATIGUE

There were no significant differences between ischaemic and non-ischaemic groups for MVC, potentiated doublet, or index of muscle activation during an MVC. Similar values for the y intercept (a), slope (bx) and curvature of the slope (cx^2) with a second order polynomial indicated comparable IT ratio-force relationships for ischaemic ($100.6 + 1.65x + 6.56x^2$: $r = 0.99$) and non-ischaemic ($104.9 + 1.86x + 7.92x^2$: $r = 0.98$) groups. Ten of the 12 subjects could fully activate their plantarflexors during a MVC.

Although no significant difference between observed and predicted MVC from the IT ratio-force relationship was apparent, the estimation of MVC with a second order polynomial had

a prediction error of 5.8% in the fully activated subjects.

FATIGUE

There were no significant differences between the groups in the number of contractions to fatigue or percentage decrease in MVC following fatigue (Table 3).

RECOVERY: INDEX OF MUSCLE ACTIVATION

Both groups demonstrated significant muscle inactivation following fatigue. The 11.5% (± 6.4) decrease in the ischaemic recovery group's index of muscle activation as derived from a single MVC IT ratio immediately post-fatigue was not significantly different from the non-ischaemic group (7.3% ± 6.5). Neither were there significant differences in ischaemic and non-ischaemic groups in the prediction of the MVC using IT ratios and second order polynomials (Table 3). Although, there were no significant differences in the y intercepts, slopes and curvature of the lines (second order polynomial) between the groups during recovery, both groups experienced a significant ($p<0.0003$) upward shift of the IT ratio-force relationship slope post-fatigue (Figures 2 and 3). Moreover, there was a significant difference between the groups in the disparity between predicted and observed post-fatigue MVC (Table 3). The ischaemic group's prediction of recovery MVC had an 11.7% (± 1.3) error in estimating the observed MVC while the non-ischaemic group had a 1.3% (± 1.1) error.

Discussion

One of the major findings of this paper was the similarity of the curvilinear muscle activation (IT ratio)-force relationship pre- and post-fatigue. Although there were no significant differences in the y intercepts, and curvature of the second order polynomials pre- and post-fatigue, the slope of the polynomial shifted significantly higher following fatigue in both groups.

The change in the slope of the polynomial would be expected with fatigue-induced decreases in muscle activation. Subjects were requested to perform specific submaximal and maximal loads (20, 40, 60, 80, 100%) pre- and post-fatigue. However, the loads used post-fatigue were calculated as a percentage of a fatigued (non-ischaemic and ischaemic MVC decreased 26.9% and 31.5% respectively), partially inactivated (7-11%) muscle. The shifting of the slope without a difference in the slope curvature would indicate that all the percentage loads of the fatigued muscle would be expected to have greater inactivation as well.

Changes in the slope of the IT ratio-force relationship are derived from changes in the superimposed doublet to potentiated doublet ratios. Alterations in the polynomial slope might not be due to differences in muscle activation but due to modifications in muscle kinetics which affect the response to evoked stimulation. A lack of significant difference in the potentiated doublet amplitude pre- (21.1 ± 7.0 Nm) and immediately post-fatigue (20.5 ± 1.8 Nm) would suggest that the evoked doublet surmounted possible impairments in excitation-contraction coupling. Since the same stimuli were utilized with the superimposed torque, changes in the IT ratio are more likely to be derived from alterations in muscle activation than impairments in muscle function. The similarity in the muscle activation-force relationship pre- and post-fatigue (y intercepts and slope curvature) would indicate that the use of a second order polynomial equation should provide a valid estimate of muscle activation following fatigue.

Although, the accuracy of the post-fatigue prediction may be similar to the pre-fatigue prediction with a second order polynomial, is the estimate of muscle inactivation from a single IT ratio similar to the polynomial? Some fatigue studies have used a single IT ratio to estimate the extent of muscle inactivation with fatigue (Bigland-Ritchie et al. 1978, 1986, McKenzie and

Gandevia 1991). Considering the lack of significant difference between post-fatigue single IT ratios and polynomial equations for both ischaemic, and non-ischaemic recovery groups (Table 3), the use of single IT ratios should provide a general estimate of muscle inactivation following fatigue.

The inhibition of recovery by ischaemia preserved the post-fatigue muscle activation-force relationship. During the two minute recovery period, the ischaemic predicted MVCs deviated from observed MVCs by 11.7%, while non-ischaemic groups showed only 1.3% differences. Fatigue-induced muscle inactivation would be expected, based on the IT ratio (index of muscle inactivation) in this study (7.3-11.5%) and other research findings (McKenzie and Gandevia 1991). The lack of inactivation predicted by the non-ischaemic polynomial equation could be attributed to the recovery-induced changes in muscle kinetics. Research from our laboratory (Behm and St-Pierre, under review) has shown little change in the fatigue-related deficits in muscle activation and MVC in the first two minutes of recovery following an isometric, intermittent, submaximal fatigue protocol. However, the same research indicated that the evoked peak twitch torque recovered to pre-fatigue levels within 30 s of recovery under non-ischaemic conditions. Differences in the prediction of the MVC during recovery between ischaemic and non-ischaemic groups may be related to the maintenance of fatigue conditions throughout the recovery period with ischaemia. Considering the IT ratios are calculated from superimposed and potentiated evoked twitches, an alteration in twitch kinetics with non-ischaemic recovery conditions would affect the ratios used in the polynomial estimate of muscle activation. The disruption of blood flow with the accompanying anoxic conditions, decreases the ability to deliver substrates, electrolytes and remove waste material resulting in an adverse effect upon recovery,

maintaining the muscle activation-force relationship derived from fatigue.

Summary

With the exception of an increased slope during recovery, IT ratio-force relationships were similar pre- and post-fatigue in ischaemic and non-ischaemic groups. The increased slope during recovery for both groups could be attributed to decreases in muscle activation or changes in muscle kinetics. The lack of change in the amplitude of the potentiated doublet pre- and post-fatigue would suggest that alterations in the slope were not due to changes in muscle kinetics. Greater muscle inactivation post-fatigue with the ischaemic group was supported by greater IT ratios and greater differences in the prediction of MVC with a second order polynomial. A lack of significant difference between the second order polynomial predictions and estimates of muscle inactivation with a single IT ratio post-fatigue suggest that a single IT ratio may be used as a general estimate of muscle inactivation post-fatigue.

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Table 1: Subject characteristics

Group	Age (yrs)	Height (cm)	Weight (kg)	Male / Female
Ischaemic (n = 6)	26.0 ± 5.4	161.5 ± 28.0	68.9 ± 16.7	3 / 3
Non-Ischaemic (n =6)	29.5 ± 6.7	166.3 ± 28.8	74.2 ± 14.5	3 / 3

Data (± SD) indicates mean values.

Table 2: Pre-fatigue ischaemic and non-ischaemic voluntary and evoked force.

Groups	Observed MVC (Nm)	PotD (Nm)	Index of Muscle Activation (%)	Predicted MVC (Nm) (IT ratio)	Predicted MVC (Nm) (polynomials)
Ischaemic	101.9 ± 47.2	21.1 ± 7.1	98.3 ± 2.9	103.6 ± 31.5	95.9 ± 49.2
Non-Ischaemic	114.6 ± 47.8	21.1 ± 6.5	100 ± 0	114.6 ± 27.6	108.3 ± 48.5

Data (± SD) indicates mean values. PotD = potentiated doublet, MVC = maximum voluntary contraction. Index of muscle activation = 1 - IT ratio X 100. Second order polynomials derived from the IT-ratio force relationship.

Table 3: Post-fatigue voluntary and evoked force with ischaemic and non-ischaemic subjects.

Groups	Number of contractions	Observed MVC (Nm)	Index of Muscle Activation (%) (IT ratio)	Predicted MVC (Nm) (IT ratio)	Predicted MVC (Nm) (polynomials)
Ischaemic	56.9 ± 14.1	69.7 ± 16.3*	88.5 ± 6.4*	79.0 ± 17.1	78.9 ± 22.7
Non-ischaemic	63.4 ± 11.8	83.7 ± 37.0*	92.7 ± 6.5*	91.1 ± 38.6	82.6 ± 41.5

Data (± SD) indicates mean values. Asterisks (*) indicate significant difference from pre-fatigue values ($p<0.001$). MVC = maximum voluntary contraction. Index of muscle activation = 1 - IT ratio X 100. Predicted MVC values derived from second order polynomial equations (IT-ratio force relationship).

Figure Legends

Figure 1: Both diagrams illustrate the voluntary force output of an individual with three superimposed doublets followed by two potentiated doublets. In the top diagram, the strain gauge torque signals were passed through a low gain amplifier (amplification 1000X). The bottom diagram shows the same force output and series of stimulations with the strain gauge signal passed through both low and high gain amplifiers (amplification 10000X). The high gain signal is offset to baseline by the computer software program.

Figure 2: Muscle activation (IT ratio)-force relationship pre- and post-fatigue in the non-ischaemic recovery group.

Figure 3: Muscle activation (IT ratio)-force relationship pre- and post-fatigue in the ischaemic recovery group. In both figures 2 and 3, second order polynomial equations with respective R^2 values illustrate the similarity of the relationship pre- and post-fatigue. Data points are mean values. Horizontal and vertical bars represent standard error of the mean.

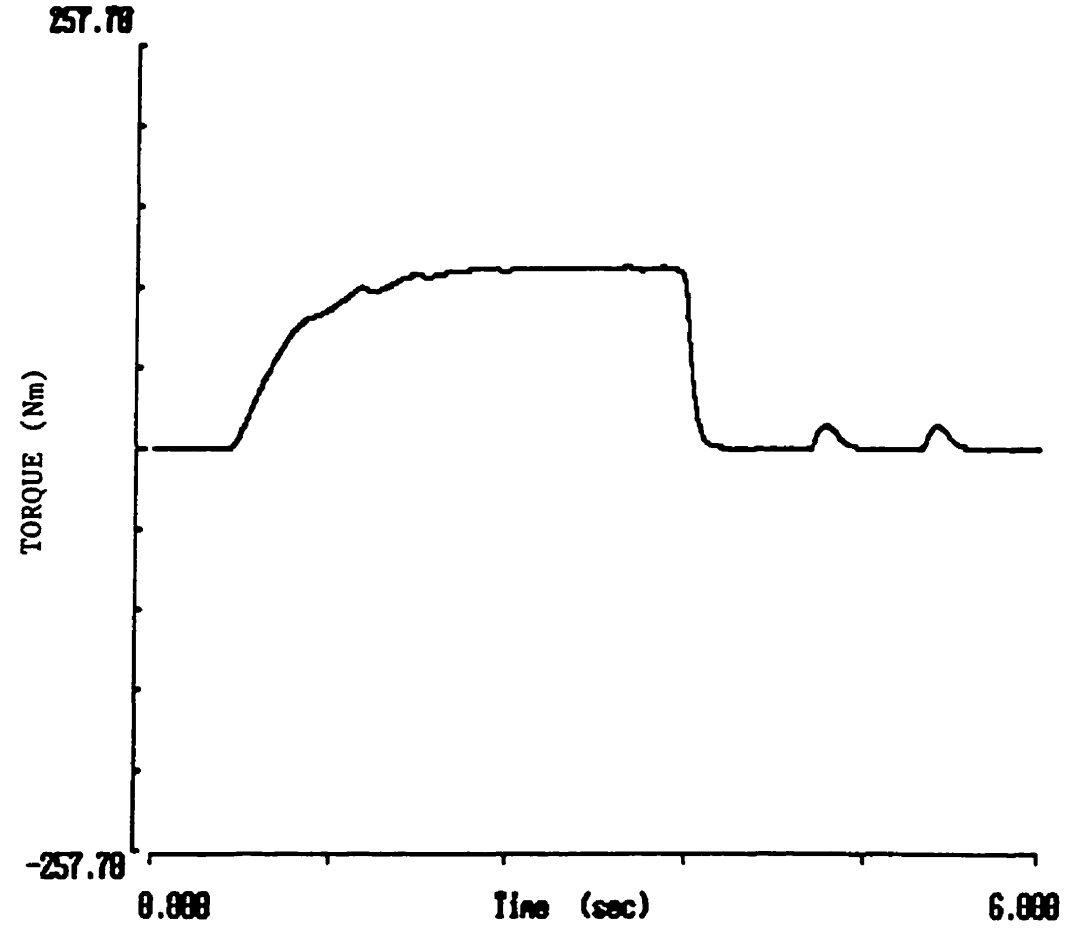

257.70
TORQUE (Nm)
-257.70
0.000
Time (sec)
6.000

Figure 1

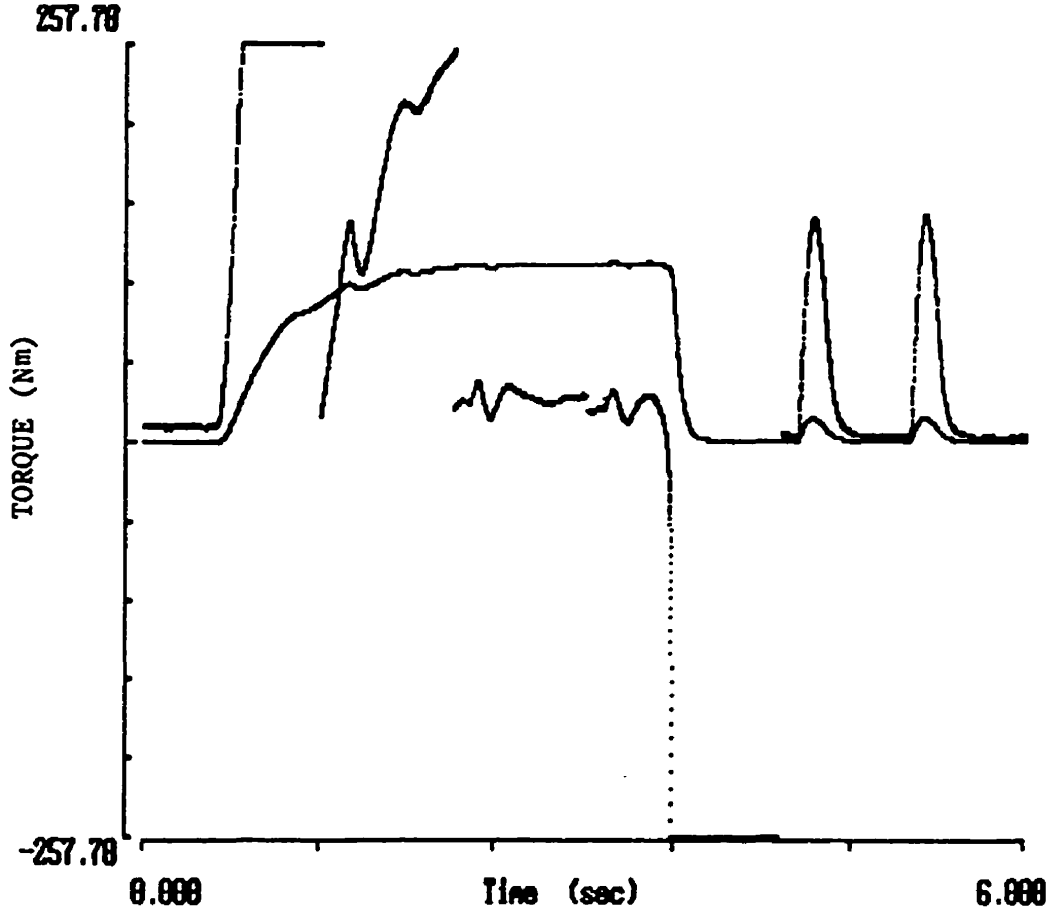

257.70
TORQUE (Nm)
-257.70
0.000
Time (sec)
6.000

Figure 2

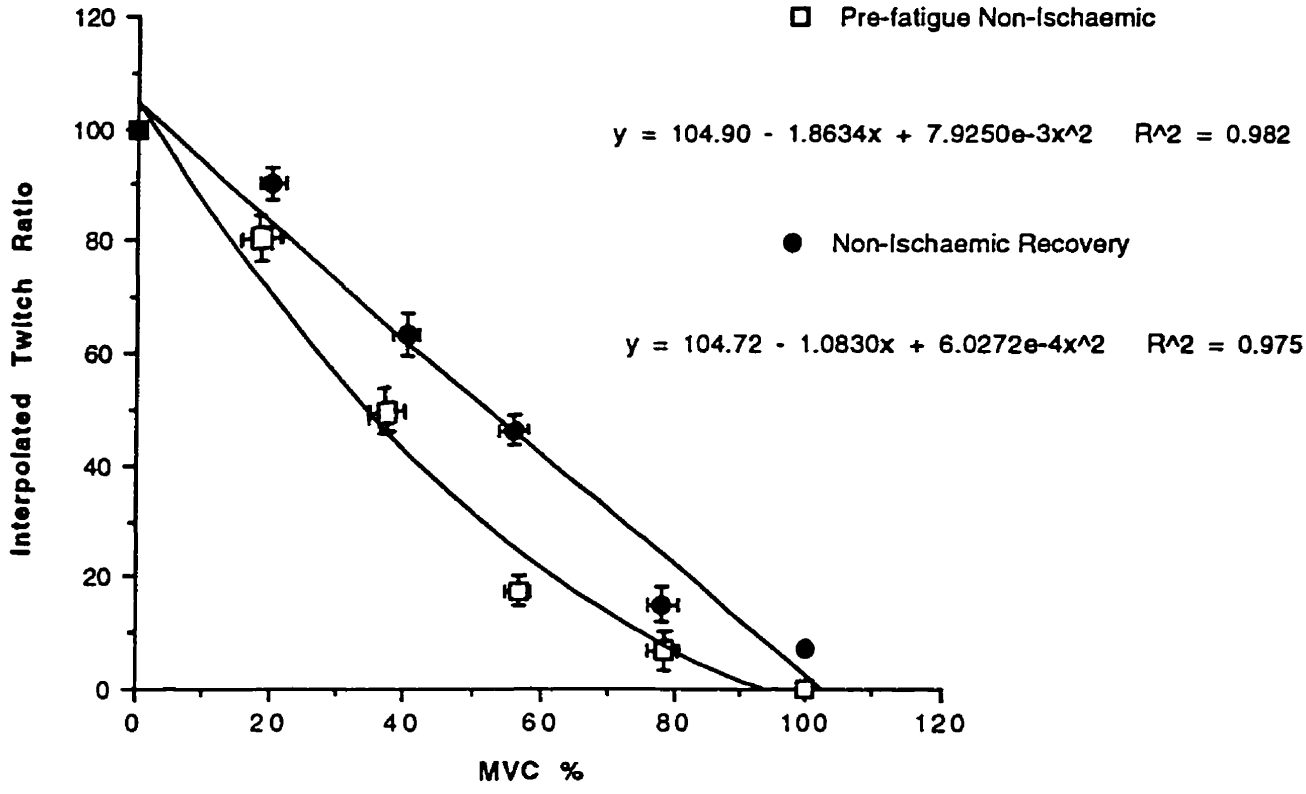
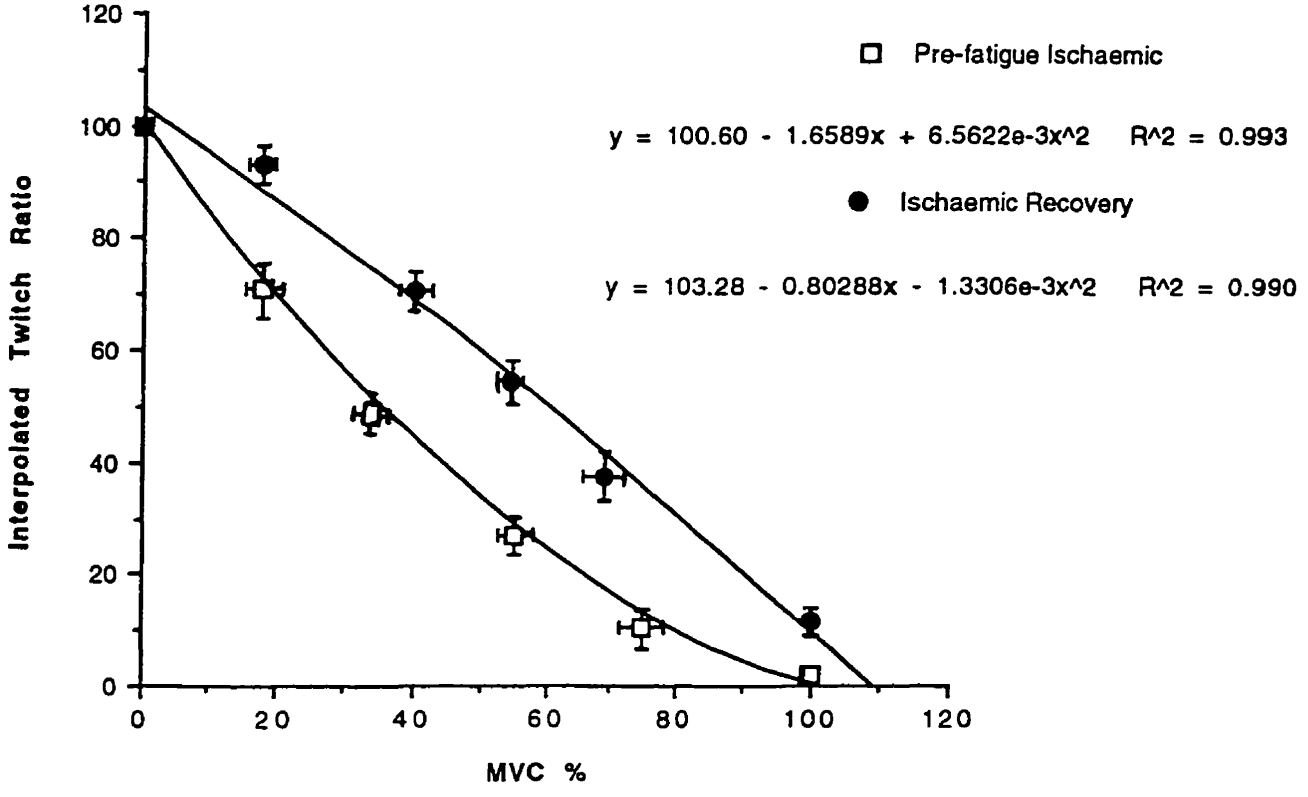

Pre-fatigue Non-Ischaemic
y = 104.90 - 1.8634x + 7.9250e-3x^2 R^2 = 0.982
Non-Ischaemic Recovery
y = 104.72 - 1.0830x + 6.0272e-4x^2 R^2 = 0.975
Interpolated Twitch Ratio
MVC %
0
20
40
60
80
100
120

Figure 3


Interpolated Twitch Ratio
MVC %
0
20
40
60
80
100
120
Pre-fatigue Ischaemic
y = 100.60 - 1.6589x + 6.5622e-3x^2 R^2 = 0.993
Ischaemic Recovery
y = 103.28 - 0.80288x - 1.3306e-3x^2 R^2 = 0.990

Introduction: Fatigue Mechanisms in Trained and Untrained Plantarflexors

Greater strength and muscle hypertrophy are just two of the factors which differentiate strength-trained and untrained individuals. Surprisingly, other voluntary and evoked contractile properties such as muscle activation as measured by the ITT, and twitch contractile properties do not significantly differ between trained and untrained individuals. In addition, the effect of training on the interaction of agonist and antagonist muscles is inconclusive in the literature.

Insufficient stress may be placed on these factors under resting conditions to illustrate trained state differences. Just as some cardiac abnormalities can only be detected with stress tests, some muscle characteristic differences may be better illustrated under fatiguing conditions. Thus an investigation of trained state differences in voluntary and evoked contractile properties was conducted under resting and fatigued conditions.

Fatigue Mechanisms in Trained and Untrained Plantarflexors

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Key Words: evoked contractile properties, muscle activation, co-contractions, recovery

Abstract

The effect of an isometric intermittent submaximal fatigue protocol on the voluntary and evoked contractile properties of 14 trained and 14 untrained plantarflexor muscles was investigated pre-, post-fatigue and during recovery. The greater force output of trained subjects was not attributed to differences in muscle activation or evoked contractile properties. Trained subjects had 40.3% less antagonist electromyographic (EMG) activity than untrained subjects which may have contributed to their increased strength. Trained individuals had a tendency to perform a greater number of contractions till fatigue (75.8 vs 51.4) and experience significantly ($p<0.01$) smaller decrements in maximum voluntary contraction force immediately post-fatigue (32.3% vs 45.9%). The greater fatiguability of untrained subjects was accompanied by significant decreases in M-wave amplitude (32.1%; $p<0.02$) and peak twitch torque (10.6%; $p<0.0001$) immediately post-fatigue. With only the untrained agonist EMG decreasing (25.9%), and similar decreases in antagonist EMG for both groups; the trained subjects experienced relatively less antagonist activity. These findings would suggest that trained state differences in plantarflexor fatiguability are related to peripheral (excitation-contraction coupling) and motor control changes rather than central (muscle activation) impairments.

Index Terms: fatigue, twitch interpolation, evoked contractile properties, training, muscle activation

Introduction

The response to fatigue can be modified by a variety of factors including muscle type, trained state, duration and intensity of contractions, and whether the contractions are sustained or intermittent (see reviews: Asmussen 1979, Enoka and Stuart 1992, Fitts and Metzger 1993, Kirkendall 1990). Although most activities of daily living involve intermittent submaximal contractions, a greater proportion of fatigue studies have focused on the effects of sustained maximal and to a lesser extent, sustained submaximal contractions. The few submaximal intermittent studies provide conflicting reports on the effects of fatigue. While Dolmage and Cafarelli (1991) reported no muscle inactivation in the vastus lateralis, McKenzie and Gandevia (1991) indicated a small degree of central fatigue in both the elbow flexors and diaphragm following a submaximal intermittent fatigue protocol. Bigland-Ritchie et al. (1986) reported no central fatigue in the quadriceps and adductor pollicis muscles but some central fatigue (maximum voluntary contraction {MVC} less than tetanic force) in the soleus muscle following submaximal intermittent fatigue. In contrast to the potentiation of the adductor pollicis compound muscle action potential (M-wave) amplitude (Duchateau and Hainaut 1985), Bigland-Ritchie et al. (1986) reported no change in adductor pollicis membrane electrical properties with an intermittent submaximal fatigue protocol. Using intermittent submaximal fatigue protocols, an initial potentiation, followed by a depression in twitch amplitude reported by Dolmage and Cafarelli (1991), contrasted with the lack of change in twitch amplitude found by Bigland-Ritchie et al. (1986). With the lack of conclusive information in the literature, more research is needed to clarify the fatigue mechanisms associated with intermittent submaximal contractions.

Strength training has been shown to influence the muscle's resistance to fatigue (Huczel

and Clarke 1992, Rube and Secher 1990). Increases in the time to reach fatigue, smaller losses in force, electromyography (EMG), motor unit discharge frequency, rates of tension development and relaxation as well as increases in M-wave amplitude have been reported following a variety of strength training protocols (Duchateau and Hainut 1984, Grimby et al. 1981, Vatine et al. 1990). Many of these studies however, have based their results on short-term training programs of 5 weeks to 3 months (Duchateau and Hainaut 1984, Rube and Secher 1990) with the impact of longer training studies not investigated. Given that strength training may lead to a relative decrease in oxidative potential of the muscle (MacDougall et al. 1980) resulting in a diminished resistance to fatigue (Burke 1975), the impact of extended training on fatigue resistance and mechanisms needs to be investigated.

Experimental Design and Methodology

Subjects. The study had 14 trained and 14 untrained subjects (Table 1). Trained subjects were intercollegiate varsity athletes who had been regularly resistance training for at least two years and represented a long-term training group. All subjects had been participating in their off-season training programs for 3 months, which included maximal strength training techniques (3-5 sets of 3-10 repetitions at 75-90% of one repetition maximum, 3 days per week) for the plantarflexors as well as some cardiovascular activity. Subjects were recruited from the McGill University staff and student population, were fully informed of the procedures and signed a consent form prior to experimentation. The study was approved by McGill University's Ethics committee.

Experimental Set-up. Subjects were seated in a straight back chair with hips and knees at 90°. Subjects had their leg secured in a modified boot apparatus with their ankles at 90° (Belanger

and McComas 1981). All voluntary and evoked torques were detected by a force transducer (custom design), amplified (recording amplifier and AC-DC differential amplifiers from Neurolog Systems-Model NL900A) and monitored on an oscilloscope (Tektronix Model 2220). All data were stored on computer (Seanix ASI 9000 486 DX) at a sampling rate of 2000 Hz after being directed through an analog-digital board (Lab Master). Data were recorded and analyzed with a commercially designed software program (Actran; Distributions Physiomonitor Ltee. Montreal).

Bipolar surface stimulating electrodes were secured to the superior and distal aspects of the triceps surae muscle group. Stimulating electrodes were constructed in the laboratory from tin foil, cheesecloth and paper coated with conduction gel (Aquasonic) and immersed in a saline solution. The electrode length was sufficient to wrap the width of the muscle belly with an electrode width of approximately 4-5 centimeters. The electrodes were placed in approximately the same positions for each subject. Surface EMG recording electrodes were placed 3-5 cm apart over the distal segment of the tibialis anterior (TA) and soleus. A ground electrode was secured superficially to the head of the tibia. Thorough skin preparation for all electrodes included sanding of the skin around the designated areas followed by cleansing with an isopropyl alcohol swab. Agonist and antagonist EMG activity were analyzed during MVCs. EMG activity was amplified (IHS 830 Isolation amplifier, BMA 830 amplifier CWE Ardmore Pa.), filtered (10-1000 Hz), monitored on oscilloscope and stored on computer. The computer software program rectified and integrated the EMG signal (IEMG) over a 500 ms period during a MVC. M-wave amplitudes elicited by the twitch were measured under the same conditions prior to MVCs, pre- and post-fatigue.

Pre- and Post-Fatigue Measurements. Peak twitch torques were evoked with electrodes

connected to a high-voltage stimulator (Digitimer Stimulator; Model DS7H+). The amperage (10 mA-1A) and duration (50-100 μs) of a 100 volt rectangular pulse was progressively increased until a maximum twitch torque was achieved. Pre-fatigue, the average of 3 trials was used to measure twitch amplitude, time to peak twitch torque (TPT) and half relaxation time (1/2 RT).

The interpolated twitch technique (ITT) was administered with a series of 3 s duration submaximal (80%, 60%, 40%, 20% of MVC) and maximal contractions. Three doublets (2 twitches with a 10 ms interval) interspersed at 900 ms intervals were evoked and superimposed on the voluntary contractions to obtain an average response. Only the smallest or occluded superimposed signal was recorded with MVCs (3 trials). Superimposed doublets were utilized in an attempt to ensure a large signal to noise ratio. Two potentiated doublets were also recorded at 1 s intervals following the voluntary contractions. Torque signals were sent through both a low and high gain amplifier. The resident software program offset and amplified by a magnitude (10X) the high gain signal for improved resolution. An interpolated twitch (IT) ratio was calculated comparing the amplitudes of the superimposed doublets with the potentiated doublets to estimate the extent of inactivation during a voluntary contraction. Since the potentiated evoked doublet represents full muscle activation, the superimposed torque using the same intensity of stimulation would activate those fibres left inactivated by the voluntary contraction. The percentage of muscle fibres activated from a single IT ratio, can be calculated by subtracting the ratio from a value of 1 and multiplying by 100 to represent an index of muscle activation during a voluntary contraction. In addition, muscle activation was also estimated pre-fatigue by correlating all maximal and submaximal forces with their respective IT ratios to generate a second order polynomial equation.

Fatigue. After voluntary and evoked testing, the subjects proceeded with the fatigue test.

Contraction intensity was gradually increased for 3 s until 50% of the predicted MVC (calculated from the index of muscle activation) was attained. This intensity was maintained for 10 s, followed by a 3 s gradual decrease to a resting state. The sequence was resumed after a 4 s rest period. Contraction cycles (work:rest ratio of 16 s: 4 s) continued until the effects of fatigue disrupted the subject's ability to maintain the desired force for the 10 s period. Voluntary and evoked properties were monitored immediately post-fatigue, and after 30 s, 1, 2, 5 and 10 min of recovery.

Statistical Analyses. Data were analyzed using a 2-way ANOVA with repeated measures on the second factor. The two factors (2 X 7) included trained state and testing period (pre-, post-fatigue and recovery periods of 30 s, 1, 2, 5, 10 min). F ratios were considered significant at $p<0.05$. If significant interactions were present, a Tukey post hoc test was conducted. Second order regression equations were used to determine the y intercept (a), slope (bx), curvature of the slope (cx^2) and prediction of muscle activation. A randomized 1 way ANOVA was used to compare group differences derived from the polynomial equations ($a + bx + cx^2$). Descriptive statistics in the text include means and ± standard deviations (SD). Data in the figures include means and ± standard errors (SE).

Results

Pre-fatigue: Trained subjects (107.1 Nm ± 39.7) exerted significantly ($p<0.001$) more torque than untrained subjects (91.2 Nm ± 21.5). The greater torque output of trained individuals was not accompanied by significant differences between the groups in the index of muscle activation, occurence of full muscle activation, peak twitch torque, and 1/2 RT. However, TPT was 11.5% longer in trained subjects. In addition, trained subjects exhibited significantly less

antagonist activity than untrained as evidenced by their 41.3% lower antagonist/agonist IEMG ratio (Table 2).

Since previous findings from our laboratory demonstrated that the use of a single submaximal IT ratio to estimate muscle inactivation could have an error of approximately 20% (Behm and St-Pierre, in press), muscle inactivation was estimated with a second order polynomial equation. Data points for the equation were derived from submaximal and maximal voluntary contraction forces (20, 40, 60, 80, 100% of MVC). There were no trained state differences in the y intercept (a), slope of the line (bx), curvature of the slope (cx^2) or prediction of muscle inactivation from the IT ratio-force relationship (Table 2).

Post-fatigue: There was a tendency ($p=0.07$) for trained subjects to perform more contractions than untrained subjects (75.8 ± 24.9 vs 51.4 ± 15.8). Furthermore, the untrained subjects had a significantly ($p<0.01$) greater percentage decrease in MVC immediately following fatigue ($45.9\% \pm 8.5$ vs $32.3\% \pm 10.0$)(Figure 1). Although the decrement in MVC continued throughout the recovery period, no significant differences between groups were observed after 30 s of recovery (Table 3). Fatigue-related differences in the number of contractions and torque immediately following fatigue were not associated with differences in the index of muscle activation, antagonist IEMG, TPT or 1/2 RT. However, both groups experienced an average 9.4% decrease in the index of muscle activation throughout the recovery period (Table 3).

In contrast, trained IEMG was relatively unchanged from pre-fatigue values while untrained IEMG dropped 25.9% (Figure 2). To ensure that changes in soleus IEMG represented the activity of the triceps surae, gastrocnemius IEMG activity was calculated in five untrained subjects. Medial and lateral gastrocnemius IEMG activity had an average decrease over the entire

recovery period of 22.9% (± 8.1) and 20.7% (± 11.6) respectively.

Although there were no group differences in the 31.3% decrease in antagonist IEMG activity, trained subjects experienced a 58% lower antagonist / agonist IEMG ratio ($p<0.001$), indicating relatively less antagonist activity in trained subjects throughout the recovery period.

Similar to IEMG findings, untrained subjects experienced an average 31.2% decrease in M-wave amplitude during the recovery period whereas no change in the trained M-wave was observed (Figure 3)($p<0.02$).

Significant differences between trained and untrained (IEMG, M-wave) subjects continued ($p<0.0001$) with trained subjects experiencing a twitch potentiation of 14.7% in the testing procedure immediately post-fatigue, while untrained subjects had a decrease of 10.6% (Figure 4). There were no significant differences between the groups during the remainder of the recovery period. In addition there were no differences during the recovery period in TPT or 1/2 RT (Table 3).

In an attempt to further specify the locale of fatigue in the plantarflexors, five untrained subjects were subjected to maximum tetanic stimulation pre- and post-fatigue. The 5.4% (± 3.9) decrease in tetanic torque post-fatigue was less than the decrements in twitch torque (10.6%) or MVC (45.9%).

Discussion

Pre-fatigue: The greater torque of trained subjects pre-fatigue was not accompanied by significant differences in muscle activation (IT ratio or second order polynomial), peak twitch torque or 1/2 RT. Sale et al. (1992) reported no difference in knee extensor motor unit activation with the ITT after 19 weeks of weight training. Some short term training studies (5-8 weeks) have

failed to demonstrate increases in neural drive as evidenced by a lack of change in EMG with isometric (Cannon and Cafarelli 1987, Garfinkel and Cafarelli 1992), eccentric and concentric resistance training (Komi and Buskirk 1972). Rutherford and Jones (1986) suggested that the improvement in strength in the early part of a training program was in large part due to alterations in the activity of antagonist muscle groups and an increased ability to co-ordinate other synergistic and stabilizing muscle groups. Although the greater strength of trained subjects could not be attributed to a greater neural drive or improvements in evoked contractile properties, trained subjects exhibited 41.3% less antagonist activity.

Antagonist co-contractions function to provide both protection from the inertial forces of the agonist (Barrata et al. 1988, Solomonow et al. 1988, Tyler and Hutton 1986) and improve the ability to target forces (Gordon and Ghez 1984). Decreased antagonist activity would result in less resistance to the intended agonist forces. Trained individuals may have a greater ability to inhibit excessive antagonist contractions contributing to the net force output of the agonist muscle. Training-induced decreases in antagonist activity have been confirmed in both cross-sectional studies involving dynamic contractions (Patton and Mortenson 1971) and longitudinal isometric training studies (Carolan and Cafarelli 1992). Although muscle hypertrophy probably exerts a more significant influence, changes in motor control by decreasing antagonist activity could contribute to the greater agonist forces of trained individuals.

Post-fatigue: Although trained subjects demonstrated a trend towards superior fatigue resistance as evidenced by the greater number of contractions to fatigue and a significantly smaller decrease in MVC immediately post-fatigue, there was no significant difference between groups in the index of muscle activation. Both groups experienced significant muscle inactivation following

fatigue (9.4%). Fatigue-induced muscle inactivation is not a consistent finding in the literature. Studies that have examined fatigue-related neural adaptations with the IT ratio following fatigue have reported either no (Merton 1954, Vollestad et al. 1988) or some evidence of muscle inactivation (McKenzie and Gandevia 1991, Newham et al. 1991). Reflex studies have shown decreases in neural drive with fatigue. Kukulka et al. (1986a) and Garland and McComas (1990) studied H-reflex responses following sustained maximum isometric contractions and 15 Hz stimulation respectively and reported a reduction in H-reflex after fatigue. The decrease in motoneurone excitability could not be attributed to supraspinal fatigue since, in the Garland and McComas (1990) study, voluntary contractions were not utilized. This would suggest that inhibitory afferents arising from the muscle are involved in down regulating motoneurone excitability with fatigue. Evidence for fatigue-induced reflex inhibition are substantiated by the decline and impaired recovery of motoneurone firing rates with ischaemic rest (Woods et al. 1987). The full recovery of motoneurone firing rates with non-ischaemic recovery indicates that muscle inactivation is affected by fatigue processes in the muscle. Duchateau and Hainaut (1993) demonstated decreases in the H-reflex which were concommitant with increases in long latency reflexes to neighboring muscles. This suggested that an increase in the central drive with fatigue was accompanied by possible presynaptic inhibition of Ia afferents or inhibition of interneurones in the oligosynaptic pathways.

Surprisingly, although both groups experienced declines in muscle activation, only the untrained group had significant decreases (25.1%) in agonist IEMG activity. More significant decreases in the IEMG of untrained subjects is similar to the findings of Rube and Secher (1990), who reported smaller decreases in EMG activity following isometric MVC leg extension training.

Other researchers have reported unaltered IEMG activity and smaller changes in frequency and duration of motor unit spikes following fatigue in endurance trained individuals (Grimby et al. 1981, Lorentzon et al. 1988, Vatine et al. 1990). The decrease in untrained IEMG activity may be related to changes in the M-wave amplitude.

Indeed decreases in the M-wave amplitude (32.1%) were observed in the untrained group whereas no significant change occured in the trained subjects. A reduction in M-wave amplitude or area may signify impairment in neuromuscular propagation and/or muscle membrane excitability (Bigland-Ritchie and Woods 1984). Many studies utilizing repetitive MVC to induce fatigue have reported decreases in M-wave amplitudes (Borg et al. 1983, Kukulka et al. 1986b, Miller et al. 1987, Milner-Brown and Miller 1986). Bigland-Ritchie et al. (1986) did not find any significant change in the M-wave amplitudes of the adductor pollicis in their intermittent submaximal fatigue study. Similar lack of decreases were reported by Garland et al. (1988) with 15 Hz tetanic fatigue of the dorsiflexors. Duchateau and Hainaut (1985) examined continuous and intermittent 30 Hz tetanic fatiguing contractions of the adductor pollicis, reporting an increase in the amplitude of the surface action potentials. Potentiation of the M-wave may signify that pre-synaptic and/or end-plate potentials are facilitated possibly by a reduction in the dispersion of fibre action potentials (Duchateau and Hainaut 1985). Thus one factor contributing to fatigue-related differences would be the greater impairment in neuromuscular propagation in untrained subjects. Since both trained and untrained subjects experienced similar muscle inactivation, trained state differences in IEMG activity would more likely be related to the reduction of the untrained M-wave than decreases in neural drive.

Another factor contributing to fatigue-related trained state differences would be

alterations in evoked contractile properties. The peak twitch torque of untrained subjects decreased 10.6% immediately following fatigue while trained subjects increased 14.7%. In order to ascertain whether excitation-contraction (E-C) coupling was actually impaired, five untrained subjects were subjected to 100 Hz maximal tetanic stimulation prior to and following the fatigue protocol. A smaller deficit in tetanic (5.4%) versus twitch torque in addition to decrements in M-wave amplitude following the submaximal fatigue protocol would suggest that the trend for greater muscular fatigue in untrained subjects could be related to impairments in E-C coupling.

There were no trained state differences in the relative drop in antagonist IEMG activity following fatigue. With data collapsed, tibialis anterior IEMG activity significantly decreased 31.3% following the fatigue protocol. However, the ratio of antagonist / agonist IEMG activity was 58% lower in trained individuals. Whereas there was a similar drop in antagonist IEMG activity between groups, relatively less antagonist activity in the trained would arise from the finding that trained agonist IEMG activity did not decrease. Similar to pre-fatigue findings, motor control changes in the form of relatively lower antagonist activity could contribute to the greater fatigue resistance of the trained subjects.

Thus trained subjects experienced fatigue (MVC decreased 32%) with only a small decrement in muscle activation (9.4%) and a lack of impairment in EMG, neuromuscular propagation or E-C coupling. Although the trained post-fatigue drop in MVC was less than the untrained, the energy demands associated with sustaining greater absolute forces would still place considerable stress on the metabolic flux of the trained muscle.

Summary: A lower ratio of antagonist to agonist IEMG activity may be a contributing factor to the increased torque output of trained subjects pre-fatigue. The increased strength of

trained subjects was not related to differences in muscle activation or evoked contractile properties. Trained subjects were more fatigue resistant following a submaximal intermittent fatigue protocol. Peripheral factors such as impairments in E-C coupling and neuromuscular propagation affected the fatiguability of the untrained but not trained subjects. Although there were no trained state differences in muscle activation following fatigue, a lower ratio of antagonist to agonist IEMG activity could again have contributed to the greater endurance of trained subjects.

Table 1: Subject Population

Group (# of subjects)	Age (yrs)	Height (cm)	Weight (kg)	Years of Training
Male Trained PF (7)	22 ± 4.2	176.1 ± 7.4	79.0 ± 6.3	4.3 ± 2.4
Female Trained PF (7)	21.1± 3.8	162.4 ± 5.3	51.9 ± 4.1	3.2 ± 2.1
Male Untrained PF (6)	26.8 ± 5.7	175.8 ± 9.4	80.6 ± 7.5	N/A
Female Untrained PF (8)	21.9 ± 4.3	160.1 ± 3.2	55.6 ± 6.8	N/A

Values indicated are group means ± SD. Plantar flexors = PF.

Table 2: Pre-fatigue trained and untrained plantarflexor voluntary and evoked contractile properties.

	Trained	Untrained
MVC (Nm)	107.1 ± 39.7*	91.1 ± 21.4 p< 0.001
Index of Muscle Activation (IT ratio)	99.5% ± 1.1	98.7% ± 2.1
Occurence of full activation	12 / 14	11 / 14
Antagonist / Agonist IEMG Ratio	0.17 ± 0.01*	0.29 ± 0.02 p<0.001
Twitch torque (Nm)	9.6 ± 3.7	7.7 ± 1.7
TPT (ms)	141.9 ± 16.7*	125.4 ± 19.3 p< 0.001
1/2 RT (ms)	93.1 ± 16.9	92.7 ± 12.9
Second Order Polynomial Equations:		
Prediction of Muscle Activation	91.3% ± 5.2	95.4% ± 1.7
Y intercept of polynomial	133.8 ± 52.1	145.3 ± 46.2
Slope of polynomial	2.7 ± 0.31	3.0 ± 0.18
Curvature of the polynomial line	2.7 ± 1.08	1.6 ± 0.14

Data includes means ± standard deviation. Asterisks (*) indicate significant differences. MVC= maximum voluntary contraction, Index of muscle activation = IT ratio - 1 X 100, Prediction of muscle activation derived from the equation: $y = a + bx + cx^2$, TPT = time to peak twitch torque, 1/2 RT = half relaxation time

Table 3: Percent difference between pre-fatigue and recovery values in trained and untrained plantarflexor voluntary and evoked contractile properties.

	Trained	Untrained	
MVC	73.1 ± 27.8	58.8 ± 14.5	
Index of Muscle Activation	90.9 ± 8.1	89.3 ± 10.0	
Agonist IEMG	93.5 ± 16.2*	74.1 ± 8.5	$p<0.01$
Antagonist IEMG	68.9 ± 16.9	68.5 ± 15.1	
Antagonist / Agonist IEMG Ratio^{+}	0.21 ± 0.10*	0.50 ± 0.21	$p<0.001$
M-wave amplitude	101.1 ± 24.4*	67.9 ± 23.8	$p< 0.04$
Twitch torque	95.9 ± 17.8	98.4 ± 25.7	
TPT	99.8 ± 19.1	102.6 ± 18.2	
1/2 RT	97.2 ± 14.3	91.1 ± 13.9	

Data includes means ± standard deviation. Mean values averaged over entire recovery period (30 seconds to 10 min). Asterisks (*) indicate significant differences. MVC = maximum voluntary contraction, Index of muscle activation = IT ratio - 1 X 100, TPT = time to peak twitch torque, 1/2 RT = half relaxation time. Plus sign ($^{+}$) indicates agonist / antagonist values represent a ratio and not a percentage of pre-fatigue values.

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Figure Legends

Figure 1: Figure illustrates mean percentage changes in MVC of trained (squares) and untrained (triangles) subjects pre-post fatigue and during 10 min. of recovery. The vertical arrow represents a significant difference between trained and untrained groups. Vertical bars represent ± SE.

Figure 2: Figure illustrates mean percentage changes in agonist integrated electromyographic (IEMG) activity of trained (squares) and untrained (triangles) subjects pre-post fatigue and during 10 min of recovery. The vertical arrows represent a significant difference between trained and untrained groups. Vertical bars represent ± SE.

Figure 3: Figure illustrates mean percentage changes in compound muscle action potentials (M-wave amplitude) of trained (squares) and untrained (triangles) subjects pre-post fatigue and during 10 min. of recovery. The vertical arrows represent a significant difference between trained and untrained groups. Vertical bars represent ± SE.

Figure 4: Figure illustrates mean percentage changes in peak twitch torque of trained (squares) and untrained (triangles) subjects pre-post fatigue and during 10 min. of recovery. The vertical arrows represent a significant difference between trained and untrained groups. Vertical bars represent ± SE.

Figure 1

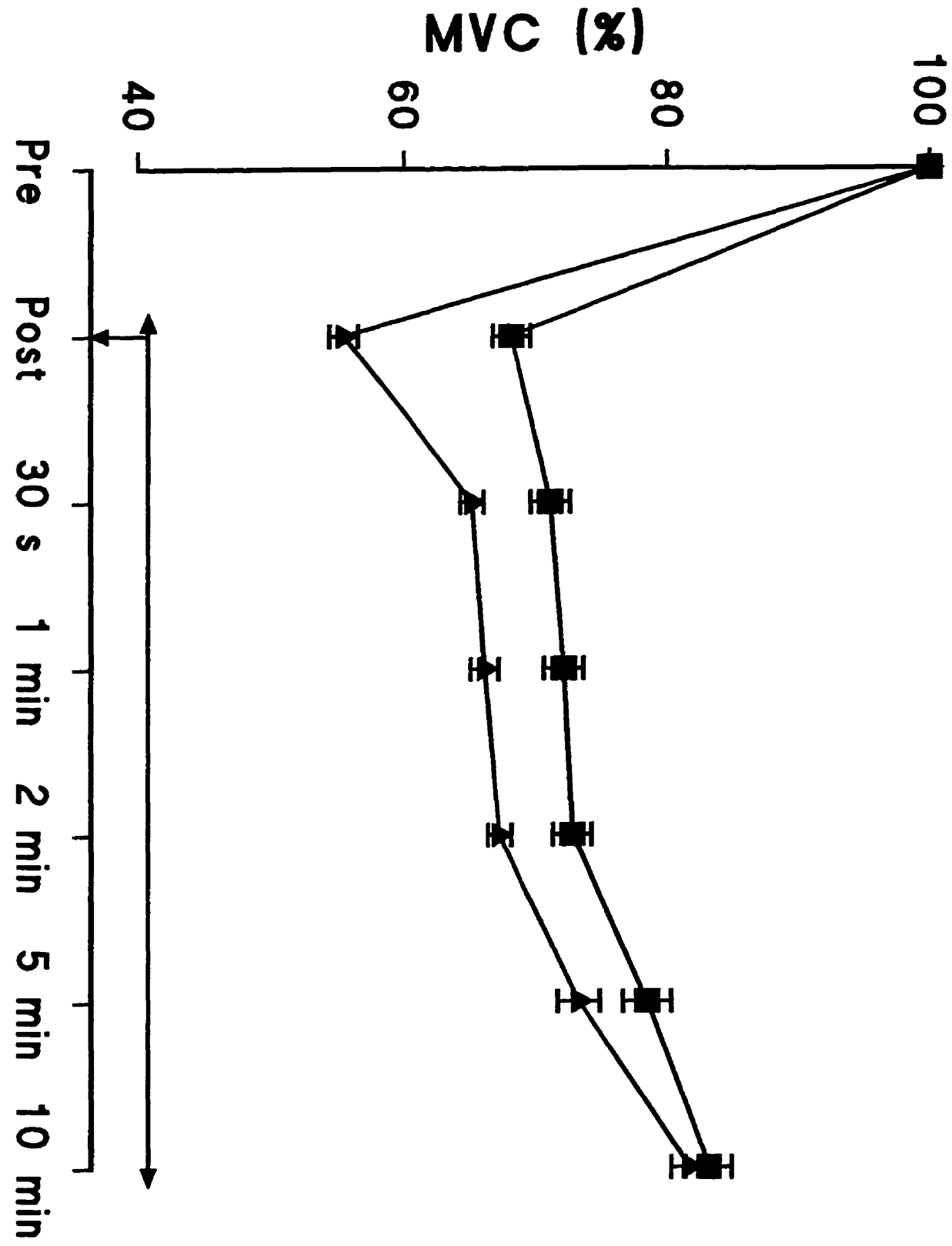


Figure 2

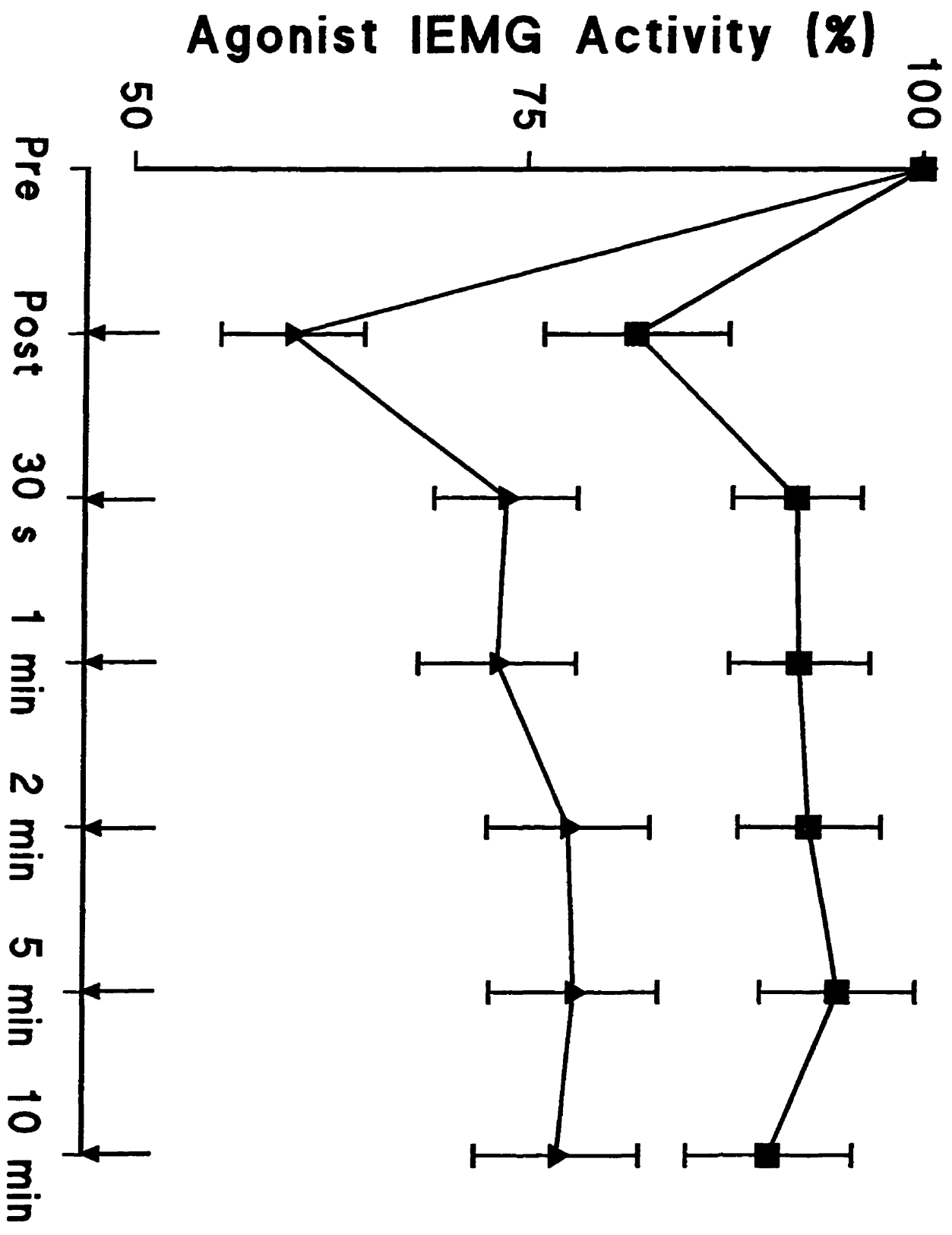

Agonist IEMG Activity (%)
50
75
100
Pre
Post
30 s
1 min
2 min
5 min
10 min

Figure 3

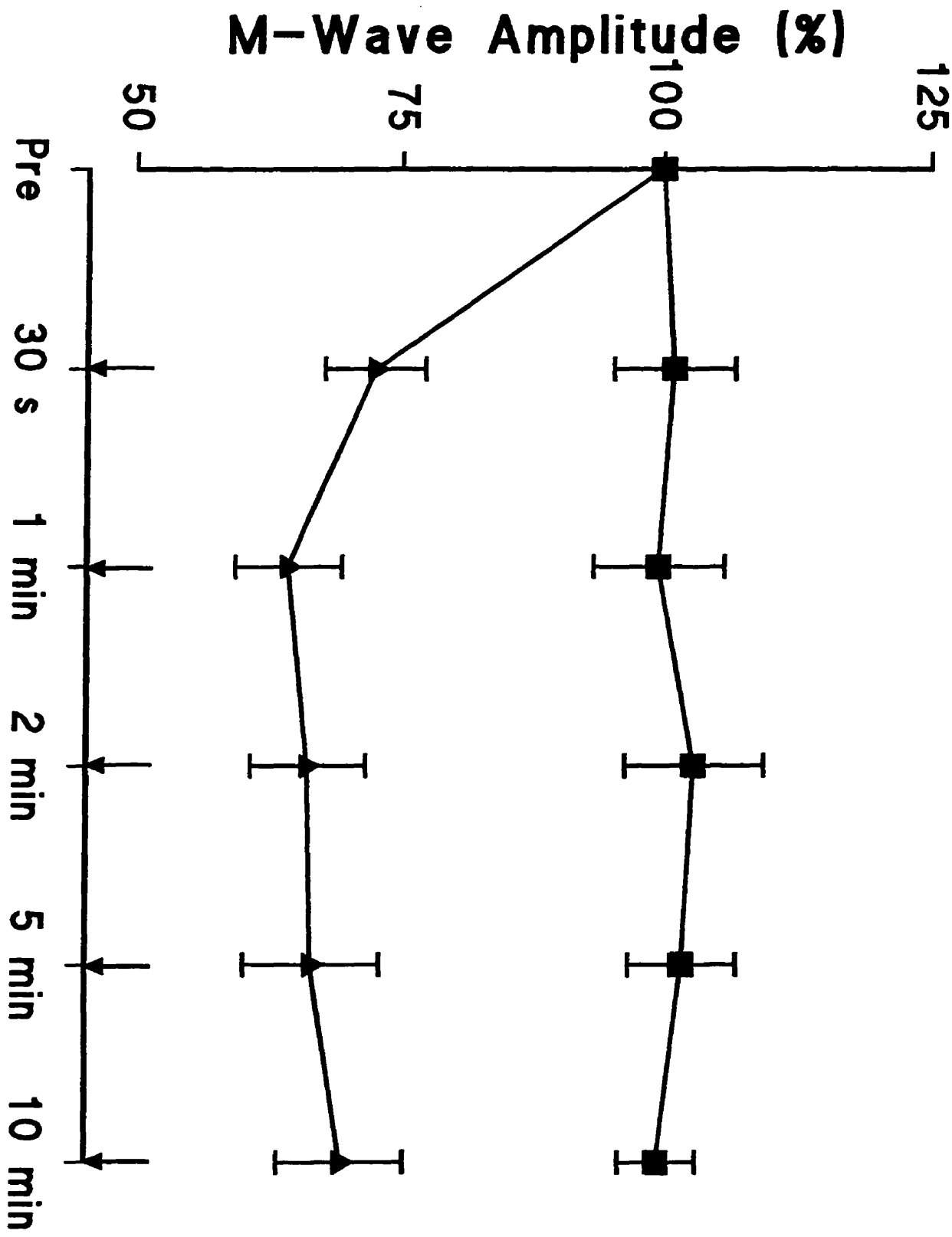
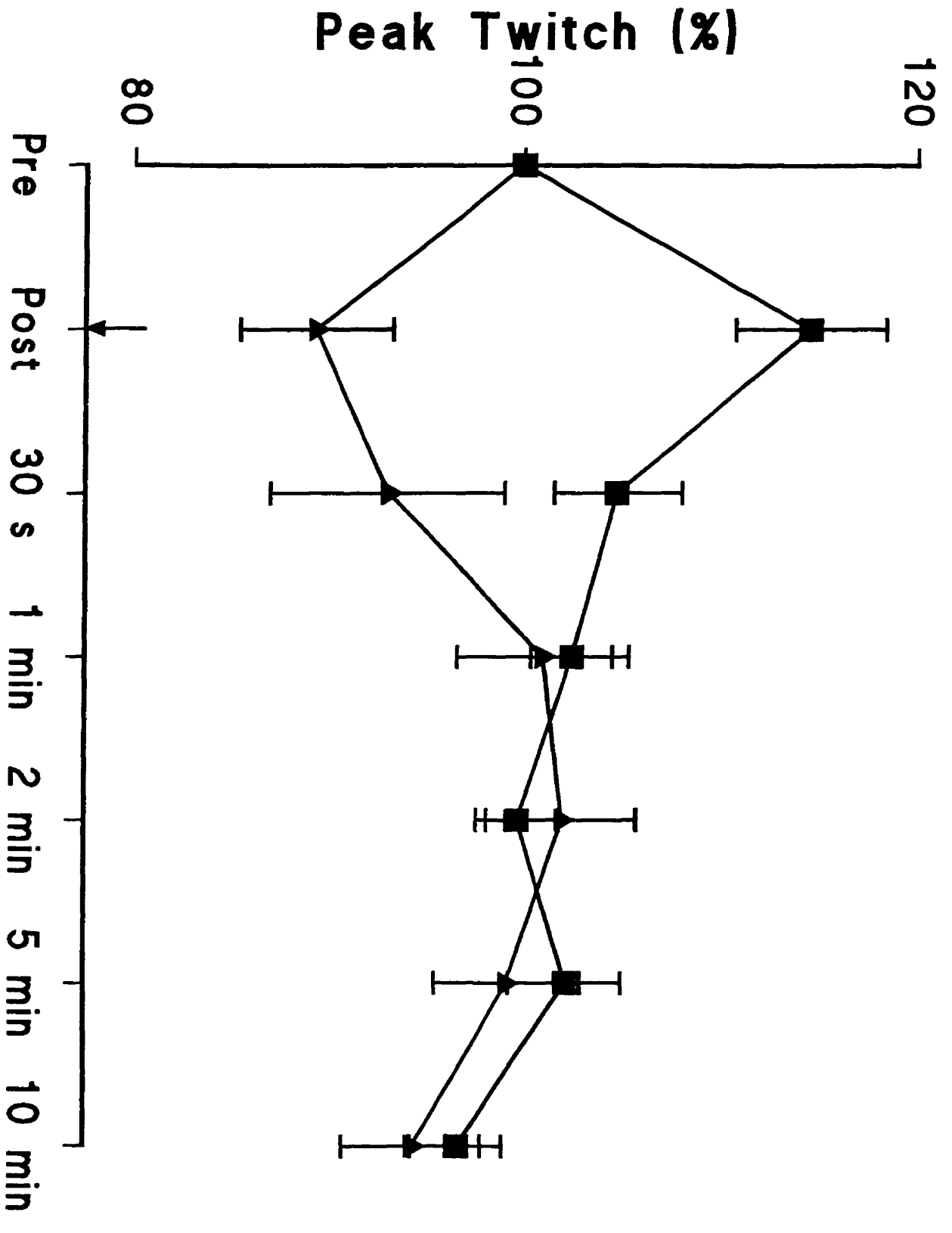


Figure 4



Introduction: Fatigue Characteristics Following Ankle Fractures

Although disused muscle experiences significant deficits in strength, hypertrophy, oxidative capacity, and activation, a number of studies have found no significant change in the rate of force loss with fatiguing maximal contractions (fatigue index) between control and disused muscle. This may be attributed to adaptive changes within the disused neuromuscular system or to the fatigue protocols used. Disuse-induced decreases in motor unit firing frequency and prolongation of membrane action potentials may help to maintain the fatigue characteristics of the muscle. On the other hand, fatigue duration may be inadvertently prolonged if maximal contractions are reduced to submaximal contractions by decreases in muscle activation. In order to accurately determine whether fatigue differences exist between control and disused muscle, studies must ensure similar contraction intensities between groups.

In addition, extensive research has not been conducted on whether the maintenance of the fatigue indexes can be applied to a diversity of patient populations. Patients experiencing more severe injuries or still in an early stage of recovery may not benefit from disuse adaptations for fatigue resistance. Thus an isometric submaximal intermittent fatigue study was conducted comparing internally-fixated, non-fixated previously immobilized ankle fractures, and controls under similar contraction intensities.

Fatigue Characteristics Following Ankle Fractures

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Abstract

Voluntary and evoked contractile properties of controls, previously immobilized (4-14 weeks post-fracture) internally-fixated and non-fixated ankles were investigated before and following an isometric, intermittent, submaximal, fatigue protocol of the plantarflexors. Prior to fatigue, fracture groups had significantly lower force output (42.7 vs. 78.8 Nm) and muscle activation (78.3% vs. 98.7%) than controls. Decreased activation may be attributed to the inhibitory effects of injured muscle and swelling. All groups had similar force and muscle activation (7-10%) decreases following fatigue, however, the internally-fixated group performed significantly fewer contractions (19) than the non-fixated (71) and controls (61). In contrast to the other groups, internally-fixated subjects experienced increased (13%) rather than decreased EMG activity (controls: 10.9%, non-fixated: 21.1%) suggesting a greater intrinsic fatiguability. M-waves and twitch torques potentiated to a similar extent in the fracture groups (4.5% and 5.7%) but decreased significantly in the control group (24.2% and 9.8%). Thus the similar fatigue durations of non-fixated subjects compared to controls may be attributed to a lack of impairment in non-fixated neuromuscular propagation and contractile kinetics.

Index Terms: immobilization, internal fixation, fatigue, twitch interpolation, contractile properties

Introduction

Immobilization for joint fractures can have dramatic consequences upon muscle function. Deficits due to immobilization in humans include muscle atrophy (15, 22), decreases in dynamic (22) and static strength (47), and increases in the duration of twitch contractile properties (10, 12, 47). Indices of muscle activation such as agonist electromyographic (EMG) activity (20), reflex potentiation (39), and maximum motor unit firing rates (13) have been reported to decrease with immobilization. Surprisingly, fatigue is not affected to the same extent as the immobilized-induced deficits in muscle strength, size, and activation. Animal immobilization studies have reported no change (38, 48), improvements (34) and increases (38) in fatiguability using maximal evoked stimulation. Insignificant changes in fatiguability have been reported in human immobilization studies (14, 22), which predominantly used voluntary contractions. However, the activation levels of the voluntary contractions were not strictly controlled in these studies. Immobilization studies of healthy (10, 47) and patient (12, 28) populations have demonstrated greater deficits with maximum voluntary contractions (MVC) than tetanic force suggesting immobilization had affected the ability to fully activate the muscle. It would be difficult to compare or measure the extent of fatigue between individuals with varying degrees of muscle activation. A preliminary assessment of muscle activation with the interpolated twitch technique (ITT) could predict an individual's true MVC ensuring that individuals were working at similar intensities. One of the purposes of the study was to investigate the effects of immobilization following ankle fractures on muscle fatigue and recovery following similar intensities of fatiguing contractions.

In addition to immobilization, surgery or the severity of the fracture may affect the extent of muscle recovery. Edwards et al. (16) demonstrated the repercussions of surgery with the significant decrements in lower extremity isometric strength and isokinetic endurance following abdominal surgery. Thus the effects on voluntary and evoked contractile properties of previously immobilized internally-fixated (surgery) and non-fixated ankle fractures were compared with healthy control plantarflexors.

Experimental Design and Methodology

Subjects. Twelve control subjects (mean age of 26.2 ± 8.2 years) were recruited from the McGill University staff and student population. Twelve previously immobilized ankle fracture patients (37.6 ± 12.4 years) from three Montreal area hospitals (Jewish General, Queen Elizabeth, Royal Victoria) were selected with the help of physiotherapy staff (Table 1). Physiotherapists identified and informed prospective patients and requested their permission to be contacted. The investigators contacted the patients by phone to inform them of the study and seek their co-operation. All previously immobilized patients were undergoing physiotherapy treatment at the time of testing. Ten of the twelve patients were fully weight bearing while two patients were partially weight bearing. Patients were not tested immediately following immobilization since our previous research with the ITT (Behm and St-Pierre, in press) had indicated that non-weight bearing patients could not exert sufficient force to provide reliable muscle activation data. Exceptionally large superimposed twitches were found with low force outputs providing inaccurate estimations of muscle activation. In addition, the availability of patients for testing determined the testing date. Previously immobilized patients met the following criteria:

1. The subjects' ankle was immobilized for an ankle fracture in either a rigid (plaster) or

removable casts (air splint, back slab). Both internally-fixated and non-fixated ankle fractures were included since clinical recovery has been reported to be similar (5). Subjects however were stratified into internally-fixated and non-fixated groups, since surgery has been reported to influence the degree of atrophy, muscle weakness (22) and isokinetic endurance (16). Depending on the surgeon, subjects were allowed to weight bear while in the cast (5 patients). Studies have not demonstrated any difference in outcome between subjects immobilized in a walking cast and those in a non-weight-bearing cast (17). The casted ankle was set at a neutral position (90° ± 5) for all subjects.

2. Presence of a sound contralateral leg as well as no other major joint problems of either limb that would interfere with the subject's ability to participate in the study.

All subjects read, signed a consent form prior to experimentation. All appropriate McGill University and hospital ethics committee approval was obtained.

Testing Protocol. Evaluations were conducted at the Sports Medicine laboratory of McGill University's School of Physical and Occupational Therapy. Patients were given a pain analog scale prior to, during and following fatigue testing to measure the possible influence of pain. In addition, both limbs were immersed separately in a specially designed cylinder filled with water. Percent differences in water displacement between limbs was measured as an indicator of lower limb swelling. Previously immobilized patients had both the affected and contralateral limbs pre-tested. Fatigue testing was conducted only on the affected limb due to possible contralateral fatigue effects in a single testing session and the subjects' time constraints. Healthy subjects served as controls for voluntary and evoked fatigue testing.

Experimental Set-up. Subjects were seated in a straight back chair with hips and knees at

90°. The leg was secured in a modified boot apparatus with the ankle at 90° (6). All voluntary and evoked torques were detected by a force transducer (custom design), amplified (recording amplifier and AC-DC differential amplifiers from Neurolog Systems-Model NL900A) and monitored on an oscilloscope (Tektronix Model 2220). All data were stored on computer (Seanix ASI 9000 486 DX) after being directed through an analog-digital board (Lab Master)(2000 Hz). Data were recorded and analyzed with a commercially designed software program (Actran; Distributions Physiomonitor Ltee.).

Bipolar surface stimulating electrodes were secured to the superior and distal aspects of the triceps surae muscle group. Surface EMG recording electrodes (Medi-Trace) were placed 3-5 cm apart over the tibialis anterior (TA) and distal portion of the soleus. A ground electrode was secured superficially to the head of the tibia. Thorough skin preparation for all electrodes included sanding of the skin around the designated areas followed by cleansing with an isopropyl alcohol swab. Agonist and antagonist EMG activity were amplified (IHS 830 Isolation amplifier, BMA 830 amplifier CWE Ardmore Pa.), filtered (10-1000 Hz), monitored on oscilloscope and stored on computer at a sampling rate of 2000 Hz. For analysis the EMG signal was rectified and integrated (IEMG) over a 500 ms period during a MVC.

Pre- and Post-Fatigue Measurements. Peak twitches were evoked with electrodes connected to a high voltage stimulator (Digitimer Stimulator; Model DS7H+). The amperage (10 mA-1A) of a 100 volt rectangular pulse (50 μs) was progressively increased until a maximum peak twitch torque was achieved. In two of the 12 previously immobilized patients, maximum twitch torques were not achieved due to patient discomfort. The average of 3 trials was used to measure twitch amplitude, time to peak twitch torque (TPT) and half relaxation time (1/2 RT) and

muscle action compound (M-wave) amplitude.

The ITT was administered with a series of 3 s duration submaximal (20, 40, 60, 80% of MVC) and three maximal contractions. Three doublets (2 twitches with a 10 ms interval) interspersed at 900 ms intervals were evoked and superimposed on the voluntary contractions to obtain an average response. Superimposed doublets were utilized in an attempt to ensure a large signal to noise ratio. Two potentiated doublets were also recorded at 1 s intervals following the voluntary contractions. Torque signals were sent through both a low and high gain amplifier. The resident software program offset the gained (10X) superimposed signal, 100 ms before each stimulation to improve resolution. An interpolated twitch (IT) ratio was calculated comparing the amplitudes of the superimposed doublets with the potentiated doublet to estimate the extent of inactivation during a voluntary contraction. Since the potentiated doublet represents full muscle activation, the superimposed torque using the same intensity of stimulation would activate those fibres left inactivated by the voluntary contraction. An index of muscle activation (percentage) was derived by subtracting the IT ratio from a value of 1 and multiplying by 100. Previous work from our laboratory has demonstrated that the use of a second order polynomial equation to predict a fully activated MVC was more accurate than the analysis of single IT ratios. Thus prior to fatigue, an interpolated twitch ratio-force relationship was derived from the indices of muscle activation of submaximal and maximal contractions and analysed with a second order polynomial equation. Submaximal contractions were not included during recovery in order to limit the number of possibly fatiguing contractions.

Fatigue. After voluntary and evoked testing, the subjects proceeded with the fatigue test. The fatigue protocol had the subject gradually increase the contraction intensity for 3 s until 50%

of the predicted MVC (calculated from the index of muscle activation) was attained. This intensity was maintained for 10 s, followed by a 3 s gradual decrease to a resting state. The sequence was resumed after a 4 s rest period. The contraction cycles (work:rest ratio of 16 s: 4 s) continued until the effects of fatigue disrupted the subject's ability to maintain the desired force for the 10 s period. Voluntary and evoked properties were monitored at 30 s, 1, 2, 5 and 10 min of recovery.

Statistical Analyses. This study was analyzed using a two way ANOVA with repeated measures on the second factor. The two factors (3 X 6) included subject groups (internally-fixated, non-fixated, and controls) and testing period. F ratios were considered significant at $p<0.05$. If significant interactions were present, a Tukey post hoc test was conducted. Correlation coefficients were used to compare pain analog scores and the extent of swelling with the index of muscle activation. Descriptive statistics include means ± standard deviations (SD). Data in the figures are presented as mean ± standard error (SE).

Results

Pre-fatigue. There were no significant differences pre-fatigue, between internally-fixated and non-fixated groups in MVC, muscle activation, twitch torque, TPT, 1/2 RT (Table 2), swelling or pain. The control group MVC (78.8 Nm) significantly ($p<0.0001$) exceeded the previously immobilized (40.4 Nm) groups. The mean index of muscle activation for controls (98.7%) exceeded ($p<0.01$) the previously immobilized groups (78.3%). Since the IT ratio force-relationship was best described by a shallow hyperbolic curve, a second order polynomial equation was derived. The polynomial equation also demonstrated a significantly ($p=0.04$) lower muscle activation in previously immobilized subjects (83.4% ± 15.1) than controls (94.5% ± 4.1). In addition, IT ratios derived from MVCs (index of muscle activation) were not significantly

different from the inactivation predicted from second order polynomial equations, suggesting that the index of muscle activation would be a valid indicator both pre- and post-fatigue. The contralateral limb MVC and index of muscle activation of the previously immobilized groups were not significantly different from the control group indicating control and patient groups were similar when unaffected by injury (Table 2).

In order to determine the factors contributing to the greater inactivation of the previously immobilized groups, coefficient correlations comparing the index of muscle activation with either a pain analog scale or the extent of swelling were analyzed. There was a moderate correlation ($r = 0.49$) between swelling and muscle activation compared to the poor correlation ($r = 0.1$) of pain and activation (Figure 1).

Fatigue and Recovery. A lack of difference between groups in the voluntary force decrement following fatigue (mean: 29% ± 4.5) contrasted with the significantly ($p<0.01$) fewer number of contractions to fatigue of the internally-fixated group (Table 3). There were no significant differences between the controls and non-fixated groups in the number of contractions till the onset of fatigue (Table 3).

Index of Muscle Activation. Although the number of contractions to fatigue was significantly less in the internally-fixated group (Table 3), this greater fatiguability could not be explained by differences in muscle activation between groups (Figure 2). Whereas the absolute amount of muscle activation differed between groups, the pattern of fatigue-induced muscle inactivation (7-10% decrease from pre-fatigue over entire recovery period) was similar. In addition, the index of muscle activation was not significantly different between individuals immobilized for five weeks or less compared to those immobilized for more than six weeks.

IEMG. The agonist and antagonist IEMG responses of the internally-fixated group following fatigue contrasted with the other groups. Both the control (10.9%) and non-fixated (21.1%) groups experienced declines in agonist maximal IEMG activity following fatigue. In contrast, despite the decline in voluntary force following fatigue, internally-fixated individuals had significant increases (13%) in agonist IEMG activity at 30 s, 1, 5 and 10 min of recovery (Figure 3a).

Similarly, the internally-fixated group also showed significant ($p<0.01$) overall increases (8.5%) in antagonist IEMG activity following fatigue. The control group experienced significantly ($p<0.01$) greater decreases (26.1%) in antagonist IEMG activity than the non-fixated (14.1%) group prior to the 10 min recovery testing period (Figure 3b).

M-Wave Amplitude. There were no significant differences in M-wave amplitude between internally-fixated and non-fixated groups. The overall 24.2% decline of the control M-wave amplitude contrasted with a lack of significant change in previously immobilized individuals (Figure 4a).

Evoked Twitch Contractile Properties. There were no significant differences in twitch torque between the internally-fixated and non-fixated groups. The twitch torque potentiation of previously immobilized groups conflicted with the twitch torque depression of the control group. The previously immobilized groups had significant ($p<0.01$) potentiation of the twitch torque until 5 min of recovery (Figure 4b). The control group experienced a significant ($p<0.0001$) decline in twitch torque until 2 min of recovery. After 5 min of recovery, there was again significant ($p<0.01$) differences between the control and previously immobilized groups as the patient groups lost their twitch torque potentiation and the control group began to experience a late

potentiation.

Although there was little change in the TPT during the recovery period, the internally-fixated group had a significant ($p=0.02$) prolongation of TPT at 30 s of recovery (Figure 4c). At the same time non-fixated and control groups experienced a significant ($p<0.05$) decrease in TPT. The control group also demonstrated a significantly ($p<0.01$) longer prolongation of TPT than the other groups at 5 min of recovery.

There were no significant differences between groups in 1/2 RT. Overall, 1/2 RT was significantly ($p<0.0001$) shortened 13.9% (± 13.4) over the entire recovery period.

Discussion

This is the first study to use the ITT to assess the effects of disuse on muscle activation, pre- and post-fatigue. Previously immobilized subjects could not activate to the same extent as controls prior to fatigue. Therefore, to investigate the effects of disuse on fatigue, it was important to measure muscle activation while controlling for differences in the relative intensity of the contractions.

Effects of Disuse on Muscle Activation: The significant pre-fatigue decrease in the index of muscle activation of previously immobilized subjects (78.3%) could arise from reflex inhibition of either or both the joint capsule and muscle afferents. McComas et al. (30) demonstrated greater inactivation in patients with joint pathology. Sabbahi et al. (37) desensitized healthy ankle joint receptors with xylocaine and then observed motoneurone excitability by monitoring H-reflex activity. They found no significant changes in H-reflex activity suggesting the joint receptors have minimal inhibitory effects on the excitability of the motoneurones. Fatigues studies have illustrated fatigue-induced inhibition of motoneurones may be derived from muscle afferents (21, 29). Thus

the reflex inhibition of motoneurones with immobilization might be attributed more to disruptions of the muscle than the joint capsule.

Other factors related to musculo-skeletal injury such as swelling may affect muscle activation as well. A number of studies have documented decreases in muscle force, EMG activity (11, 49) and H-reflex amplitude (43) with knee joint effusion in humans. Decreases in EMG activity and H-reflex amplitude would indicate a decline in motoneurone excitability resulting from a swelling induced reflex inhibition. In the present study we showed a correlation coefficient of 0.49 between the index of muscle activation and the extent of swelling (Figure 1). This indicates that approximately 25% of the muscle inactivation could be related to the extent of swelling.

In contrast to swelling, a statistical treatment of the pain analog data revealed a correlation coefficient (pain analog and index of muscle activation) of only 0.1, indicating that pain may only be related to about 1% of the muscle inactivation (Figure 1). Rutherford et al. (36) reported extensive quadriceps inactivation with muscle pain. However, both deAndrade et al. (11) and Wood et al. (49) reported that swelling-induced reflex inhibition of the quadriceps was independent of pain. Stokes and Young (44) infiltrated human knee joints with bupivacaine to block the pain of post-surgery meniscectomies and reported no change in the severity of inhibition. Thus the major factors affecting muscle inactivation would be inhibitory afferents arising from swelling and muscle with minor contributions from pain and possibly joint capsule afferents.

Cross-education or cross transfer effects have been observed in the untrained contralateral limb following unilateral training of the ipsilateral limb. This has been demonstrated in a number

of studies and attributed to central or neural adaptations (23, 26, 33). It might be logical to assume that if training effects are transferable then detraining or injury effects may be transferred as well. The uninjured leg of arthroscopic patients in the Hurley et al. (25) study experienced greater inactivation (18.6%) than what has been reported in the literature on quadriceps activation in healthy individuals (35, 36). Long term testing of previously immobilized patients (1-5 years) showed full activation of both the affected and contralateral limbs (36). The 1.7% inactivation of the contralateral plantarflexors was similar to the 1.3% inactivation of healthy controls in this study. Thus cross-education injury effects were not evident in the present study.

Effects of Disuse on Fatigue: Previous human studies investigating the impact of immobilization on muscle fatigue have not controlled for inter-individual differences in the ability to maximally activate (14). The similar fatigue characteristics reported between controls and previously immobilized subjects could be attributed to a comparison of maximal and submaximal voluntary efforts. The major finding in this study was that greater muscle fatiguability was observed in the previously immobilized internally-fixated subjects in comparison to controls and non-fixated subjects, when were working at the same relative intensity. One of the puzzling findings, however, was the mechanism underlying the greater fatiguability of fixated subjects in view of the fact that both fixated and non-fixated subjects were characterized by similar changes in muscle activation. M-wave and twitch contractile properties.

Mechanisms of Fatigue with Internally-Fixated Previously Immobilized Subjects: The increased fatiguability of the internally-fixated subjects could not be attributed to a greater degree of inactivation post-fatigue. Indeed, muscle inactivation increased to a similar extent in all groups following fatigue and during the recovery period (7-10%). McKenzie and Gandevia (31) indicated

that their subjects failed to activate 4-10% of their elbow flexors with intermittent maximal contractions. In a previous study with healthy subjects, we determined that the extent of muscle inactivation post-fatigue was related to the duration of the fatigue protocol (Behm and St-Pierre, under review). However in this study, internally-fixated subjects experienced similar levels of inactivation with a shorter fatigue duration than non-fixated subjects, suggesting that the time to fatigue does not influence the degree of inactivation post-fatigue in previously immobilized muscles.

The changes observed in maximum agonist EMG activity post-fatigue were also unexpected. Both control and non-fixated ankles were characterized by a decrease in maximal agonist EMG activity whereas significant increases were observed in the internally-fixated ankles. Typically in submaximal fatigue protocols, EMG activity initially increases in order to maintain the same tension output by recruiting additional motor units (9, 32). Subsequently EMG activity starts to decrease when the target torque can no longer be maintained in the muscle (9). Increases in maximal EMG post-fatigue are usually indicative of failure to reach the point of true fatigue. However the percent drop in MVC and muscle activation were the same in all groups. Therefore the efficiency of the muscle contraction (EMG / muscle torque) may actually be less in the internally-fixated group. This greater inefficiency combined with a shorter time to fatigue would suggest a greater intrinsic fatiguability in the internally-fixated subjects.

Alterations in antagonist activity must also be considered. The fact that antagonist EMG activity significantly increased by 8.5% in the internally-fixated group post-fatigue suggests an increase in co-contractions which would overestimate to some extent the fatigue-induced drop in MVC. The decrease in antagonist EMG activity of the non-fixated (14.1%) and control (26.1%)

groups would suggest, on the other hand, a decrease in co-contractions, which would tend to underestimate the observed drop in MVC post-fatigue. Whereas antagonist co-contractions provide protection from the inertial forces of the agonist (4, 42, 46), increases in the antagonist EMG activity of the internally-fixated group could represent a strategy of greater joint protection for the more severely fractured subjects. Although force output was maintained at the same relative intensity for all groups during fatigue, internally-fixated subjects may have been working at a greater intensity due to greater antagonist activity. However greater antagonist activity probably does not fully explain the significantly fewer internally-fixated contractions. Since neuromuscular propagation (M-wave amplitude) and excitation-contraction coupling (twitch torque) were not impaired post-fatigue, the greater fatiguability of internally-fixated muscle may be associated with a deficiency of the contractile proteins to maintain repeated submaximal contractions.

Mechanisms of Fatigue with Non-Fixated Previously Immobilized Subjects: The similar fatiguability of previously immobilized non-fixated subjects and controls in this and other studies while experiencing impaired force production (3, 14, 34, 48) and decreased oxidative capacity (48) may be due to a number of reasons. The similar fatiguability of non-fixated and control subjects could not be attributed to differences in the extent of fatigue-induced neural or central inhibition since decreases in muscle activation were comparable (7-10%). However the fatigue-induced impairments in neuromuscular propagation and contractile kinetics evident in controls were not present in previously immobilized groups. Control subjects experienced a 24.2% decrease in M-wave amplitude in contrast to the lack of change in internally-fixated and non-fixated groups. Decline in M-wave amplitude has been reported in the abductor pollicis of healthy

subjects, after 90-100 s of fatiguing MVCs (7). Fuglevand et al. (18) also illustrated declines in M-wave amplitude of healthy subjects with fatigue, concluding that when force is sustained at a submaximal value, impairment in muscle membrane propagation may occur. A reduction in M-wave amplitude or area may signify an impairment in neuromuscular transmission or muscle membrane excitability (8). Conversely, human paralyzed soleus muscle exhibited minimal changes in M-wave amplitude compared to significant fatigue-induced decreases in torque suggesting that the source of fatigue was within the contractile mechanism and not attributable to neuromuscular transmission compromise (40). The maintenance of previously immobilized M-wave amplitudes in the present study may be related to the lower maximum firing frequencies of motoneurones in immobilized subjects (13), resulting in a decrease in the frequency and total quantity of stimuli reaching the muscle membrane. Thus the mechanisms underlying fatigue in immobilized muscle could not be related to impairments of neuromuscular propagation or muscle membrane excitability.

The decrease in the control subjects' twitch torque (9.8%), contrasted with the potentiation of the other groups. Other studies have found both decreases (2), and no change (1) in twitch torque following prolonged intermittent maximal fatigue protocols with healthy subjects. Conversely, potentiation (24) of twitch torque has been demonstrated following a short term maximal fatigue protocol in healthy subjects. Although Fuglevand et al. (19) showed twitch potentiation in non-fatigued short term immobilized human hand muscle, they found the twitch torque to be depressed in the same subjects following a submaximal (35% MVC) fatigue protocol. Detraining or atrophic effects on the myofibrillar component with immobilization or disuse are not accompanied by the same relative decreases in sarcoplasmic reticulum (SR) activity

(27, 41). A relatively more expansive SR within an atrophied muscle fibre may augment Ca^{++} delivery providing greater concentrations of Ca^{++} to the myofibrils. Twitch torque potentiation would preclude excitation-contraction coupling as a mechanism underlying fatigue in previously immobilized muscle.

The similar fatigue characteristics of control and non-fixated subjects may be a function of alterations in energy requirements or diffusion. Witzmann et al. (48) suggested that a lower force output in immobilized muscle is due to a lower number of active cross-bridges resulting in lower energy expenditure and thus greater than expected endurance. Turcotte et al. (45) reported lower levels of myosin ATPase with disused muscle which would slow cross-bridge cycling reducing energy demand. St-Pierre and Gardiner (38) suggested that the atrophic changes in the muscle would result in a smaller surface area contributing to improved diffusion. This study suggests that even with similar contraction intensities, changes in muscle energetics may be responsible for the similar fatiguability of non-fixated previously immobilized and control subjects.

Summary. Prior to fatigue, previously immobilized subjects experienced greater muscle inactivation than controls which could be attributed to the inhibitory effects of injured muscle and swelling. Non-fixated and control subjects performed a comparable number of similar intensity contractions when subjected to a submaximal intermittent fatigue protocol. Although there were no differences in the fatigue-induced decreases in muscle activation between groups, a lack of impairment to the previously immobilized groups' M-wave amplitude (neuromuscular propagation) and twitch torque (contractile kinetics) illustrated differences between the groups in the underlying mechanisms of fatigue. The shorter duration of internally-fixated fatigue was associated with increases rather than decreases in agonist and antagonist IEMG. Since, internally-

fixated muscle did not experience greater impairments in muscle activation, membrane excitability or contractile kinetics than the other groups, the more severe ankles fractures may contribute to an intrinsically more fatiguable muscle than normal or less severely injured muscles.

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Table 1: Patient Population Characteristics

Fracture	Immobilization	Duration of Immobilization	Weight Bearing Prior to Testing	Post-Immobilization to Testing Period
Internally-Fixated				
Bimalleolar	Back Slab	10 days	2 weeks	3 weeks
Bimalleolar	Air Splint	6 weeks	7 weeks	8 weeks
Bimalleolar	Air Splint	3 weeks	1 week	1 week
Bimalleolar	Plaster Cast	6 weeks	5 weeks	5 weeks
Bimalleolar	Back Slab	3 weeks	3 weeks	6 weeks
Trimalleolar	Plaster Cast	6 weeks	4 weeks	6 weeks
Non-fixated				
Unimalleolar	Plaster Cast	5 weeks	3 weeks	2 weeks
Unimalleolar	Back Slab	5 weeks	2 weeks	6 weeks
Unimalleolar	Plaster Cast	5 weeks	1 week	1 week
Fibula	Plaster Cast	6 weeks	4 weeks	4 weeks
Fibula	Plaster Cast	7 weeks	4 weeks	4 weeks
Fibula	Plaster Cast	7 weeks	7 weeks	6 weeks

Table 2: Pre-fatigue Voluntary and Evoked Contractile Properties

Groups	MVC (Nm)	Index of Activation	Twitch (Nm)	TPT (ms)	1/2RT (ms)
Control	78.8 ± 17.3*	98.7 ± 2.3*	5.7 ± 0.7	121.2 ± 17.4	102.1 ± 13.7
Fixated	37.4 ± 16.1	70.1 ± 21.3	5.4 ± 2.3	131.9 ± 7.9	113.2 ± 29.5
Non-fixated	43.5 ± 13.8	86.5 ± 9.2	7.4 ± 2.1	138.0 ± 30.8	136.8 ± 55.4
Contralateral Limb of Fixated and Non-fixated	67.4 ± 23.5*	98.3 ± 0.9*	6.2 ± 1.8	130.3 ± 24.6	121.4 ± 37.8

Data indicate mean ± SD. Asterisks (*) illustrate significant differences between controls and fracture groups and contralateral limbs and fracture groups at the $p<0.01$ level. MVC = maximum voluntary contraction, TPT = time to peak twitch and 1/2 RT = half relaxation time.

Table 3: Fatiguability of groups.

Group	% decrease in MVC post-fatigue	Number of contractions till onset of fatigue
Control	32.8 ± 3.6	61.4 ± 19.2
Internally-Fixated	27.0 ± 21.9	19.0 ± 9.8 *
Non-fixated	27.4 ± 10.8	71.0 ± 15.2

Data indicate mean ± SD. Asterisks (*) illustrate significant differences in the number of contractions to fatigue between internally-fixated and the other groups at the $p<0.01$ level. MVC = maximum voluntary contraction.

Figure Legends

Figure 1: Figures illustrate the relationship between the index of muscle activation and percentage increase in swelling (top) and pain (bottom) of the affected ankle. The upper Y axis indicates the percentage increase in swelling (volume) of the affected ankle in comparison to the healthy contralateral ankle. The lower Y axis indicates scores on a pain analog scale from 0-1. Correlation coefficients are indicated by r values (swelling = 0.49, pain = 0.1).

Figure 2: Figure illustrates changes in the index of muscle inactivation pre-fatigue and during 10 min. of recovery. Closed squares = controls, O = non-fixated previously immobilized subjects and open semicircles = internally-fixated previously immobilized subjects. Horizontal arrow = significant differences from pre-fatigue values. Vertical arrows = significant differences between control and previously immobilized groups. Arrows represent significant differences at the $p<0.05$ level.

Figure 3a: Figure illustrates mean percentage changes in the agonist EMG activity pre-fatigue and during 10 min. of recovery.

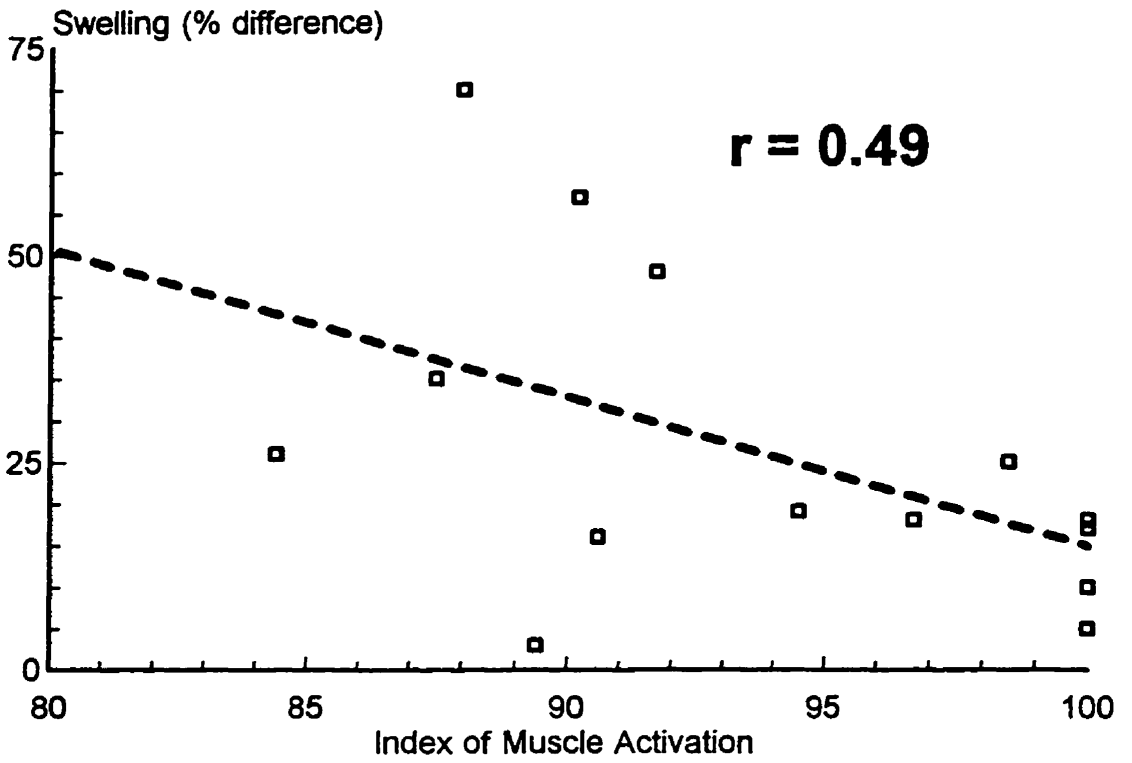
Figure 3b: Figure illustrates mean percentage changes in the antagonist EMG activity pre-fatigue and during 10 min. of recovery. Closed squares = controls, O = non-fixated previously immobilized subjects and open semicircles = internally-fixated previously immobilized subjects. # = significant ($p<0.05$) differences between previously immobilized internally-fixated and all other groups. Vertical arrows = significant differences ($p<0.05$) between control and all other groups.

Figure 4: Figures illustrate mean percentage changes in M-wave amplitude (top), twitch amplitude (middle), and TPT (bottom), pre-fatigue and during 10 min. of recovery. Closed squares = controls, O = non-fixated previously immobilized subjects and open semicircles = internally-

fixated previously immobilized subjects. Vertical arrows = significant ($p<0.05$) differences between control and all other groups. Horizontal arrow = significant differences from pre-fatigue values for the control group.

** = significant ($p<0.01$) differences between previously immobilized internally-fixated and all other groups.

Figure 1


Swelling (% difference)
75
50
25
0
r = 0.49
80
85
90
95
100
Index of Muscle Activation

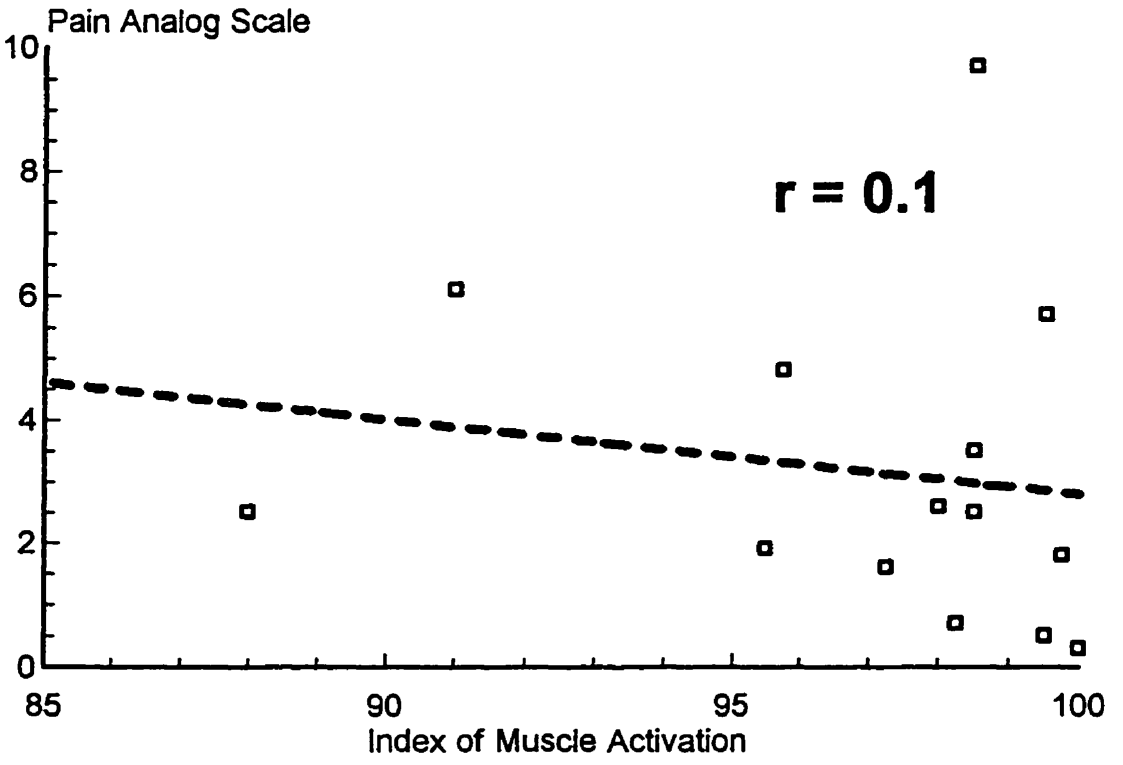
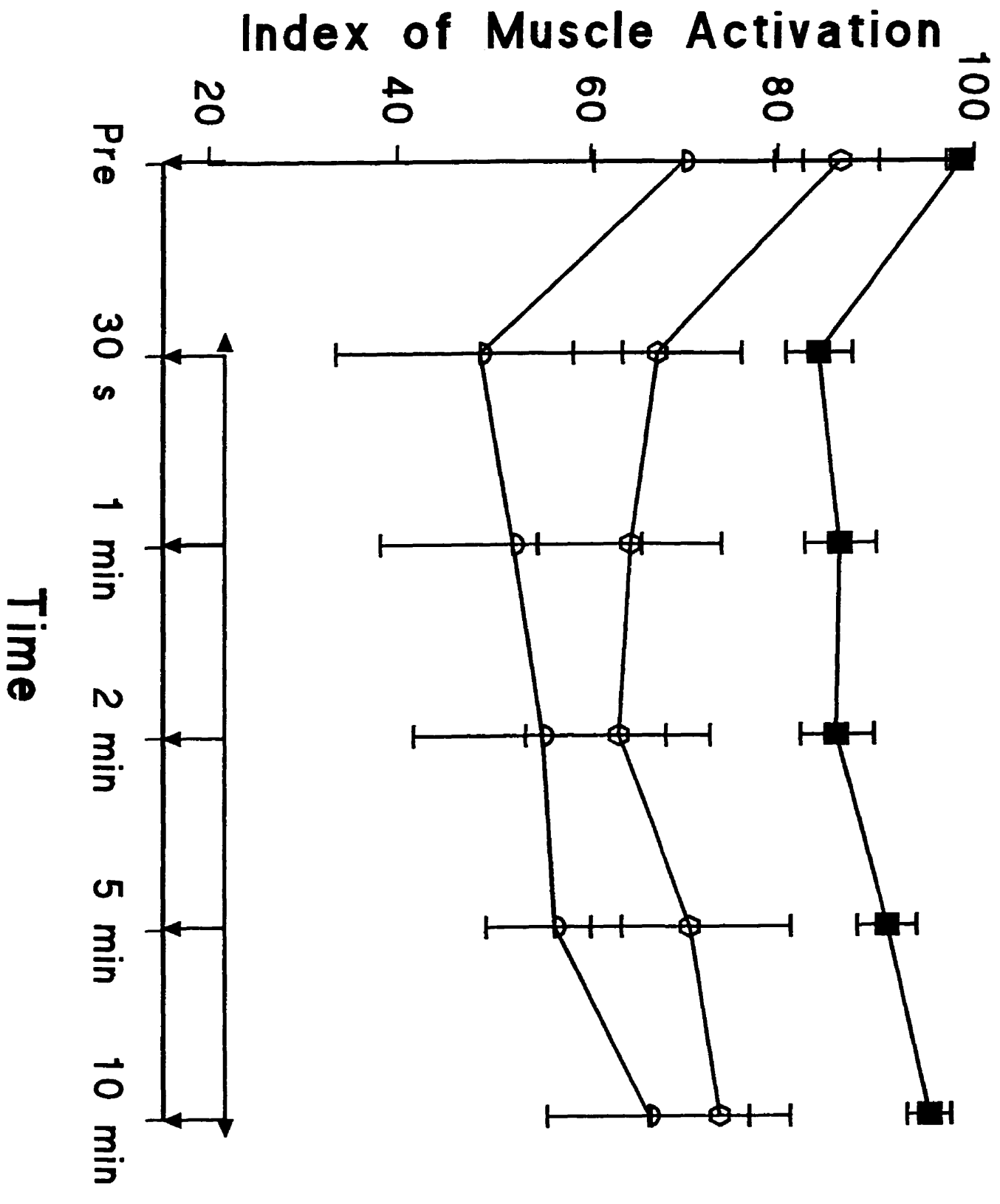

Pain Analog Scale
10
8
6
4
2
0
r = 0.1
85
90
95
100
Index of Muscle Activation

Figure 2


Index of Muscle Activation
20
40
60
80
100
Pre
30 s
1 min
2 min
5 min
10 min
Time

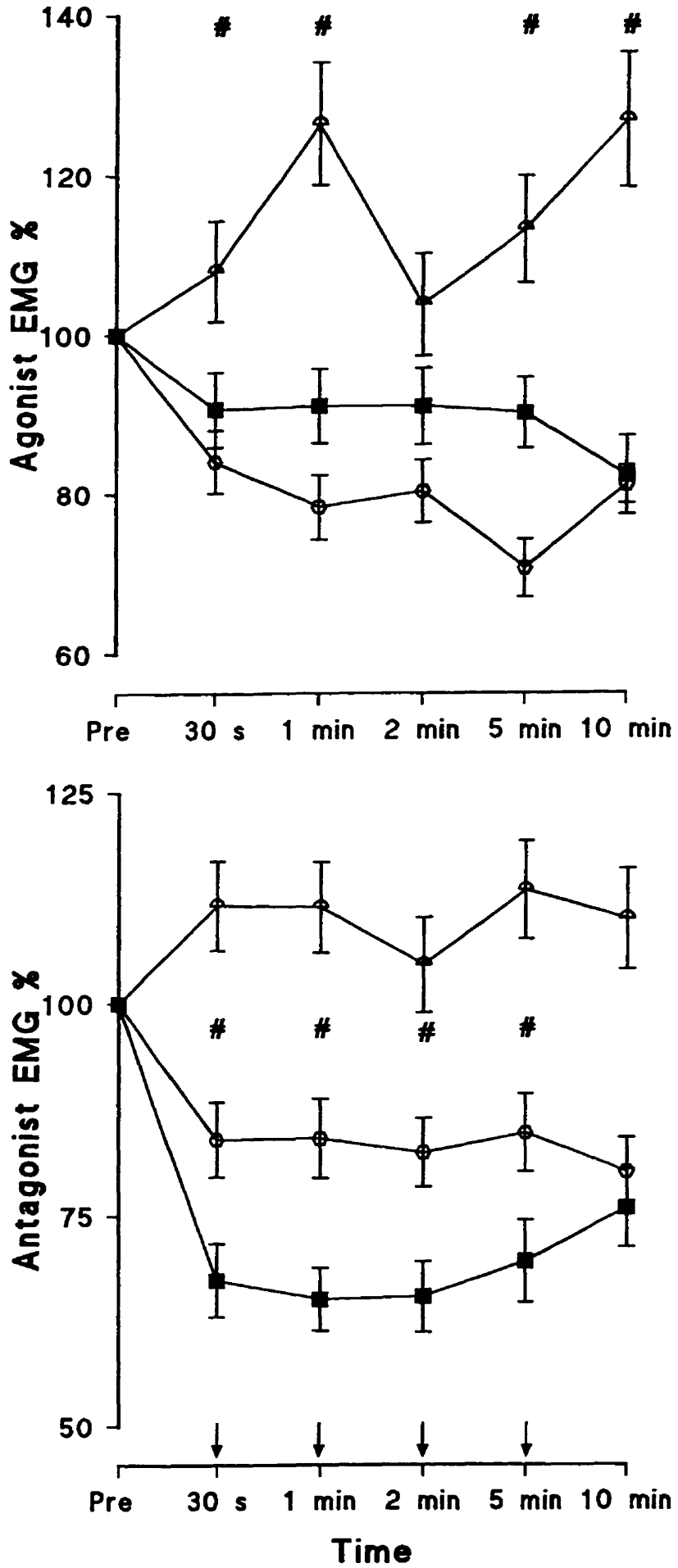
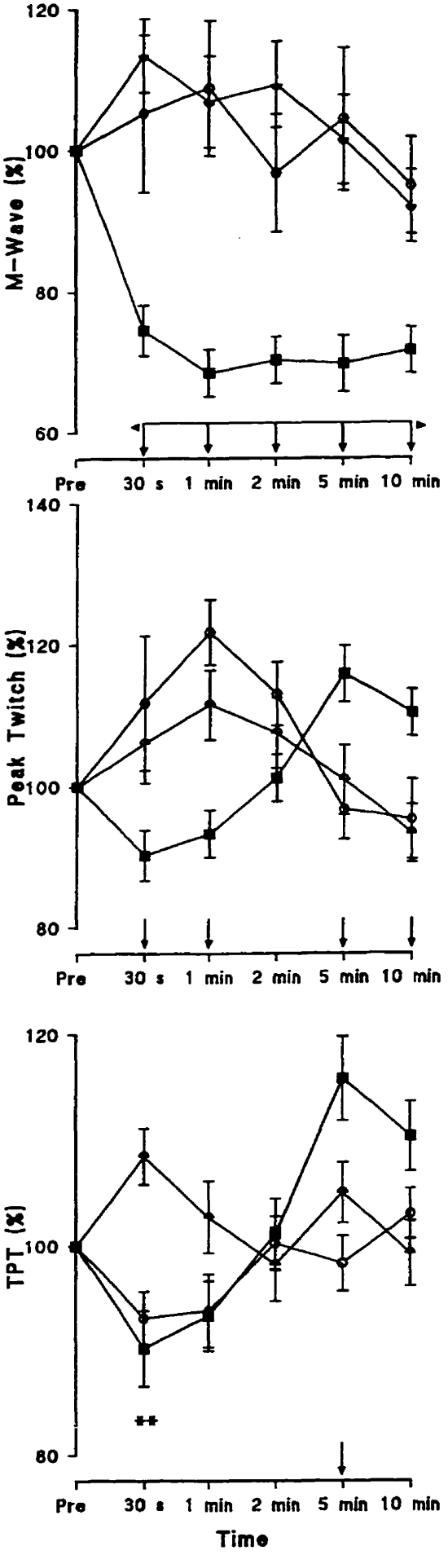

140
120
100
80
60
Agonist EMG %
#
#
#
#
Pre
30 s
1 min
2 min
5 min
10 min
125
100
75
50
Antagonist EMG %
#
#
#
#
Pre
30 s
1 min
2 min
5 min
10 min
Time

Figure 3

Figure 4


120
100
80
60
M-Wave (%)
Pre
30 s
1 min
2 min
5 min
10 min
140
120
100
80
Peak Twitch (%)
Pre
30 s
1 min
2 min
5 min
10 min
120
100
80
TPT (%)
**
Pre
30 s
1 min
2 min
5 min
10 min
Time

Introduction: Effects of Fatigue Duration and Muscle Type Upon Voluntary and Evoked Contractile Properties

The effects of fatigue are very diverse and specific to the muscle and fatigue protocol. Fatigue-induced changes in the present studies cannot be generalized to all muscles and types of fatigue. All subjects except the internally-fixated ankle fracture patients experienced prolonged plantarflexor contractions. The shorter fatigue duration of the internally-fixated also resulted in increased agonist and antagonist EMG activity. However it is unknown whether the aberrant EMG response was due to the population utilized or the duration of the fatiguing contractions.

In order to examine whether the changes in voluntary and evoked contractile properties were specific to the muscle used (plantarflexors) or fatigue protocol, both short and long duration fatiguing contractions were performed on two different muscles (plantarflexors and quadriceps).

Effects of Fatigue Duration and Muscle Type Upon Voluntary and Evoked Contractile Properties

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Running Title: Effects of Duration and Muscle Type on Fatigue

Abstract

The effects of fatigue duration and muscle type on voluntary and evoked contractile properties were investigated with an isometric, intermittent, submaximal fatigue protocol. Four groups performed contractions of the plantarflexors (PF) and quadriceps at various intensities to produce long (LDF: 19:30 min) and short (SDF: 4:17 min) duration fatigue. The LDF had a significantly greater decrease in muscle activation than the SDF (12 vs.5.8%) during recovery, although there was no difference in the impairment of maximum voluntary contraction force (MVC) beyond 30 s of recovery. The significant decrease in LDF compound muscle action potential (M-wave amplitude; 14.7%) contrasted with SDF M-wave potentiation (15.7%), suggesting changes in membrane excitation may affect LDF. The quadriceps performing contractions at 50% MVC experienced a smaller decrease in agonist electromyograph (EMG) activity than other groups, indicating both muscle and fatigue duration specificity. Impairments in excitation-contraction coupling were indicated by changes in quadriceps peak twitch and time to peak twitch, while decreases in PF M-wave amplitudes suggested a disruption of membrane potentials. Results suggest that fatigue mechanisms may be duration (activation, ½ RT) or muscle specific (EMG, twitch torque) or a combination of both (M-wave, TPT).

Index Terms: fatigue, twitch interpolation, contractile properties, muscle type

Introduction

Fatigue studies have demonstrated a diversity of mechanisms underlying fatigue-associated decrements in force (see reviews 1, 14, 16, 21). These mechanisms are commonly subdivided in central (alpha motoneurone pool or above) as well as distal sites (motoneurone endplate and below) and may be task dependent (14). Indeed, changes induced by fatigue may well be influenced by whether or not the muscle is contracting voluntarily or is being induced to contract (27), whether or not the contraction is static (4, 5, 6, 22) or dynamic (11), sustained versus intermittent (12), maximal (4, 5, 6) or submaximal (17, 24), or dependent upon the characteristics of the specific muscle.

Muscles with higher percentages of fast-twitch fibers have been shown to fatigue more rapidly than muscles with a greater percentage of slow twitch fibers (7, 11, 23). Furthermore similar fatigue protocols in a variety of muscles have resulted in dissimilar changes in force (10, 25), muscle activation (7), as measured with the interpolated twitch technique (ITT), EMG activity (24, 27), and M-wave (26). This suggests that mechanisms underlying fatigue may differ depending on the muscle (4) or its fibre composition (11, 23). However because the time to fatigue differs in different muscles, it is difficult to determine whether the differences in underlying fatigue mechanisms are muscle or duration dependent. More specifically, it is not known if the mechanisms underlying fatigue would be the same in two muscles of different fiber type composition (20) if the time to fatigue was similar. In order to compare the influence of similar fatigue durations on two different muscles of different fiber type composition (quadriceps and PF), different contraction intensities were utilized to alter the duration of an intermittent, submaximal, isometric, fatigue protocol.

Experimental Design and Methodology

Subjects. The study had four groups of eight moderately active to active subjects (Table 1). Subjects were recruited from the McGill University staff and student population, were fully informed of the procedures and signed a consent form prior to experimentation. The study was approved by McGill University's Ethics Committee.

Experimental Set-up. Subjects were seated in a straight back chair with hips and knees at 90°. PF subjects had their leg secured in a modified boot apparatus with their ankles at 90° (2). Quadriceps subjects were seated in a Cybex chair (Cybex Inc., Lumex N.Y.) with their foot in a padded strap attached to a strain gauge, perpendicular to the lower limb. All voluntary and evoked torques were detected by a force transducer (PF: custom design, quadriceps: BLH Electronics 3SB), amplified (recording amplifier and AC-DC differential amplifiers from Neurolog Systems, Model NL900A) and monitored on an oscilloscope (Tektronix, Model 2220). All data were stored on computer (Seanix ASI 9000, 486 DX) at a sampling rate of 2000 Hz after being directed through an analog-digital board (Lab Master). Data were recorded and analysed with a commercially designed software program (Actran, Distributions Physiomonitor Ltee., Montreal).

Bipolar surface stimulating electrodes were secured to the superior and distal aspects of the triceps surae or quadriceps muscle group. Stimulating electrodes were constructed in the laboratory from tin foil coated with a conduction gel, wrapped in cheesecloth and paper, and immersed in a saline solution. The electrode length was sufficient to wrap the width of the muscle belly with an electrode width of approximately 4-5 centimeters. The electrodes were placed in approximately the same positions for each subject. Surface EMG recording electrodes were placed 3-5 cm apart over the distal segment of the tibialis anterior (TA) and soleus (PF group) or

vastus lateralis and biceps femoris (quadriceps group). A ground electrode was secured superficially to the head of the tibia. Thorough skin preparation for all electrodes included sanding of the skin around the designated areas followed by cleansing with an isopropyl alcohol swab. Agonist and antagonist EMG activity were analysed during MVCs. EMG activity was amplified (Isolation Head Stage 830 amplifier, Biomedical 830 amplifier CWA, Ardmore Pa.), filtered (10-1000 Hz), monitored on oscilloscope and stored on computer. The computer software program rectified and integrated the EMG signal (IEMG) over a 500 ms period during a MVC. IEMG activity was normalized to pre-fatigue values for analysis. M-wave amplitudes elicited by the twitch were measured under the same conditions prior to MVCs, pre- and post-fatigue.

Pre- and Post-Fatigue Measurements. Peak twitch torques were evoked with electrodes connected to a high-voltage stimulator (Digitimer Stimulator, Model DS7H+). The amperage (10 mA-1A) and duration (50-100 μs) of a 400 volt rectangular pulse were progressively increased until a maximum twitch torque was achieved in the PF. Dependent upon the mass of an individual's quadriceps, voltage was increased until either a plateau in the twitch torque was obtained or the stimulator reached maximum output. Nine of 16 quadriceps subjects achieved maximal twitch torque. Pre-fatigue, the average of 3 trials was used to measure twitch amplitude, time to peak twitch torque (TPT) and half relaxation time (1/2 RT).

The ITT was administered with a series of 3 s duration MVCs (3 trials). Three doublets (2 twitches with a 10 ms interval) interspersed at 900 ms intervals were evoked and superimposed on the voluntary contractions to obtain an average response (Figure 1). Superimposed doublets were utilized in an attempt to ensure a large signal to noise ratio. Two potentiated doublets were also recorded at 1 s intervals following the voluntary contractions. Torque signals were sent through

both a low and high gain amplifier. The resident software program offset the gained superimposed signal, 100 ms before each stimulation for improved resolution. A ratio was calculated comparing the amplitudes of the superimposed doublets with the potentiated doublet, representing muscle fibers which were not voluntarily activated. The percentage of muscle fibers activated, was estimated by subtracting the ratio from a value of 1 and multiplying by 100 to represent an index of muscle activation during a voluntary contraction.

Fatigue. After voluntary and evoked testing, the subjects proceeded with the fatigue test. Each of the four groups were subjected to a different contraction intensity. The two quadriceps groups performed voluntary contractions at 50% or 25% MVC while PF groups performed voluntary contractions at 50% or 75% MVC. Preliminary work indicated that performing quadriceps contractions at 25% of MVC led to a similar number of contractions as when performing work with the PF at 50% MVC. In addition, the time to fatigue for the quadriceps at 50% MVC was similar to the fatigue duration of the PF at 75% MVC. However, the number of contractions to fatigue was less in the latter (50% quadriceps, 75% PF) than in the former (25% quadriceps, 50% PF), leading to what will be referred to as short (SDF) and long duration fatigue (LDF) protocols. In all protocols, the subject's contraction intensity was gradually increased for 3 s until the desired force was attained. This intensity was maintained for 10 s, followed by a 3 s gradual decrease to a resting state. The sequence was resumed after a 4 s rest period. Contraction cycles (work : rest ratio of 16 s: 4 s) continued until the effects of fatigue disrupted the subject's ability to maintain the desired force for the 10 s period. Voluntary and evoked properties were monitored immediately post-fatigue, and after 30 s, 1, 2, 5 and 10 min of recovery.

Statistical Analyses. The effect of fatigue duration and muscle type on voluntary and

evoked twitch properties were analysed using a 3-way ANOVA with repeated measures on the third factor. The three factors (2 X 2 X 7) included muscle type (quadriceps and PF), fatigue duration (long and short) and testing period (pre-, post-fatigue and recovery periods of 30 s, 1, 2, 5, 10 min). F ratios were considered significant at $p<0.05$. If significant interactions were present, a Tukey post hoc test was conducted. Descriptive statistics include means and ± standard deviations (SD). Figures include means and ± standard errors (SE).

Results

Fatigue. When contracting at the same intensity (50% MVC) the quadriceps (4:04 min ± 1:02) fatigued more rapidly than the PF (19:33 min ± 8:00). However, there were no significant differences in the time to fatigue between the 50% PF (19:33 min) and 25% quadriceps (19:27 min ± 3:08) groups (long duration fatigue) or between the 75% PF (4:30 min ± 1:44) and 50% quadriceps (4:04 min) groups (short duration fatigue). The LDF (50% PF and 25% quadriceps) group had a significantly ($p<0.0001$) greater number of contractions to fatigue than the SDF (75% PF and 50% quadriceps). Immediately following the fatigue protocol, the LDF group had a significantly ($p=0.03$) greater drop in MVC force than the SDF group (LDF 40% vs SDF 30.9%). MVC recovered to the same extent in the subsequent 10 min of recovery in both the LDF and SDF, although significant differences from pre-fatigue were still observed (Figure 2).

Muscle Activation. Prior to the fatigue test, full muscle activation was achieved in 11 of 16 PF subjects and 5 of 16 quadriceps subjects. Changes in activation with fatigue were influenced by the duration of the fatigue protocol and not by muscle type. When averaged over the entire recovery period, the index of muscle activation decreased significantly ($p=0.02$) more in the LDF (12% ± 7.5) than the SDF protocol (5.8% ± 4.5) (Figure 3).

In contrast, changes in M-wave following fatigue were influenced by both fatigue duration (Figure 4)($p=0.003$) and muscle type ($p=0.002$). LDF protocols diminished M-wave amplitudes by 14.7% (± 15.5) contrasting with the 15.7% (± 25.6) potentiation with SDF (Figure 5). Muscle type differences were demonstrated by the average 16.7% (± 15.5) potentiation of the quadriceps M-waves contrasting with the 15.7% (± 25.5) reduction in PF M-waves throughout the recovery period (Figure 6).

The most important factor underlying the changes in maximum IEMG following fatigue was not as clear cut. Irrespective of fatigue duration, all PF IEMG activity significantly decreased following fatigue to a similar extent (50% PF: 19.3% ± 25.2 and 75% PF: 26.3% ± 8.7). Although the long duration quadriceps (25% MVC: 30.4% ± 17) group experienced a corresponding IEMG decrease, the short duration quadriceps (50% MVC: 3.7% ± 1.9) exhibited no significant change in IEMG following fatigue.

In order to ensure that changes in soleus IEMG represented the activity of the triceps surae, gastrocnemius IEMG activity was calculated following a 50% MVC fatiguing protocol of the PF in five subjects. Medial and lateral gastrocnemius IEMG activity had an average decrease over the entire recovery period of 22.9% (± 8.1) and 20.7% (± 11.6) respectively.

Evoked Twitch Contractile Properties. Changes in peak twitch torque with fatigue were dependent on muscle type and not fatigue duration (Figure 7). Quadriceps twitch torque had an insignificant ($p=0.34$) average decrease of 14.1% (± 2.3) contrasting with the 16.1% (± 2.6) potentiation of the PF ($p=0.004$) during the recovery period (Figure 6). TPT was affected by both muscle type and fatigue duration. The lack of change in PF TPT contrasted with the significant prolongation ($p=0.02$) of the quadriceps (15.3% ± 3.2) over the entire recovery period (Figure 6).

Fatigue duration effects were exhibited by the SDF protocol which experienced significantly ($p=0.008$) longer TPT post-fatigue and at 30 s of recovery than LDF (Figure 5).

In contrast, fatigue duration was the only significant ($p=0.0007$) factor affecting ½ RT. The LDF ½ RT was shortened 16.8% (± 12.2) compared to the 9.7% (± 2.5) increase in the SDF ½ RT during the recovery period (Figure 5).

Reliability: Intraclass correlation coefficients were used to determine the test-retest reliability of the variables. Very high correlation coefficients (over 0.9) were established for the index of muscle activation, MVC, potentiated doublet, TPT and ½ RT. Moderate to high correlation coefficients were found with PF twitch torque (0.74) and quadriceps twitch torque (0.54).

Discussion

One of the most important findings of this paper was that fatigue-related changes of specific voluntary and evoked contractile properties were influenced by different factors. Fatigue duration exerted its greatest effect upon muscle activation and ½ RT. However, the effect of fatigue on twitch torque was primarily determined by muscle type. Fatigue related changes in M-wave amplitude and TPT were affected by both duration and muscle type (Table 2).

Muscle Activation. Increased time to fatigue resulted in greater decreases in muscle activation. The 12% decrease in the LDF index of muscle activation following fatigue was significantly greater than the 5.8% decrease of the SDF group. The soleus has been reported to be more susceptible to central (neural) fatigue than the quadriceps (7). This could suggest that fatigue-induced muscle inactivation was related to the specific muscle. It was shown in this study however that the muscle type was incidental to the amount of muscle inactivation. Alterations in

contraction intensity allowed the 25% quadriceps and 50% PF groups to contract longer than the 50% quadriceps and 75% PF groups resulting in greater inactivation in the LDF groups independent of the muscle utilized.

M-wave amplitudes in this study decreased with LDF but increased with SDF. Bigland-Ritchie et al. (5) using a 60 s MVC fatigue protocol of the adductor pollicis did not find any change in M-wave amplitude suggesting muscle membrane transmission failure was not a component of high intensity, short term fatigue. Other studies using short duration voluntary (7, 22) or evoked (18) fatigue protocols have not shown decreases in M-wave amplitude. Decline in M-wave amplitude has been reported in the abductor pollicis, after 90-100 s of fatiguing MVCs (3). Fuglevand et al. (17) illustrated greater declines in M-wave amplitude with lower intensity (ie. longer duration) contractions, concluding that when force is sustained at a submaximal value, impairment in muscle membrane propagation may occur. Decline in M-wave amplitude with LDF was evident in our study as well indicating an impairment of the muscle membrane excitability or neuromuscular propagation. The 14.7% depression of LDF M-wave amplitude contrasted with the 15.7% SDF potentiation.

The fact that the M-wave potentiated during SDF would suggest that muscle membrane propagation failure is not the primary determinant of muscle fatigue with SDF. Potentiation of the M-wave during the first 30 s of tetanic fatiguing contractions has also been observed, suggesting an increased excitability of the muscle fibers (15). M-wave potentiation may signify that pre-synaptic and/or end-plate potentials are facilitated possibly by a reduction in the dispersion of fiber action potentials (12). The increase in M-wave amplitude could support Buchthal and Madsen's (9) report of increased synchronization with fatiguing contractions.

Alterations in M-wave amplitude were not only dependent on fatigue duration but muscle type as well. Overall, quadriceps M-waves were potentiated 16.7% while PF M-waves were depressed 15.7%. Milner-Brown and Miller (26) concluded that the impairment of membrane propagation depends both on the duration and degree of fatigue as well as the intrinsic properties of the individual muscle. Pagala et al. (28) reported greater decreases in the action potentials of the extensor digitorum longus and diaphragm than with the soleus while Moritani et al. (27) found similar results when comparing the gastrocnemius and soleus.

The unchanged IEMG activity of the 50% quadriceps (3.7%) contrasted with significant decreases for both PF and 25% quadriceps groups. Since the extent of muscle activation and compound muscle action potential (M-wave) both contribute to the EMG signal, the differing response of the 50% quadriceps might suggest that the EMG is also affected by both duration and muscle type.

Evoked Twitch Contractile Properties. Changes in twitch torque were muscle dependent. Quadriceps twitch torque had an insignificant 14.1% decrease in contrast to the 16.1% potentiation of PF. Conversely, alterations in TPT were both muscle and fatigue duration dependent. Quadriceps TPT was prolonged 15.3% while PF did not experience a significant change. The prolongation of quadriceps TPT may help to explain why the decreased quadriceps twitch torque did not achieve significance. Muscle dependence was also reported by Hatcher and Luff (19), who found contrasting results when comparing the heterogenous flexor digitorum longus (FDL) and slow twitch soleus muscles of the cat. In contrast to the significant changes in the FDL, the soleus had smaller decreases in tetanic tension and no change in maximum shortening velocity. The decline in quadriceps twitch torque and prolongation of TPT in the

present study may imply an impairment in excitation-contraction (E-C) coupling (13, 30). The quadriceps M-wave potentiation would argue against failure in membrane propagation, suggesting the impairment was related more to the sarcoplasmic reticulum's release of Ca^{++} and/or crossbridge kinetics. Potentiation of PF twitch torque and a lack of change in PF TPT in conjunction with decreases in M-wave amplitude would reinforce the hypothesis that impairments in PF evoked properties can be mainly attributed to impairments of membrane potentials.

Fatigue duration was the major factor affecting ½ RT with LDF experiencing a 16.8% decrease contrasting with a 9.7% increase in SDF ½ RT. Alterations in twitch torque or TPT did not correspond to changes in ½ RT. The divergence of TPT and ½ RT has also been reported by Viitasalo and Komi (30), who found differing recovery profiles. Bigland-Ritchie et al. (9) demonstrated a prolongation in ½ RT contrasted with a lack of change in TPT. Impairment in E-C coupling affecting Ca^{++} release (TPT) may not automatically coincide with a hindrance of Ca^{++} sequestering (½ RT). The sequestration of Ca^{++} is an active process involving ATP (8) and thus would be affected by alterations in the muscle metabolic milieu. Resynthesis of ATP in glycolysis has been suggested to be inhibited by low intramuscular pH (29) and thus may affect ½ RT more with the higher intensity contractions of the SDF. The release of Ca^{++} however is not an active process (8). The differing effects of metabolism may explain the lack of correlation between changes in ½ RT and the other evoked contractile properties of twitch torque and TPT.

Summary. In summary, this study found duration-induced impairments in muscle activation and ½ RT, muscle-dependent decreases in peak twitch torque and IEMG, while M-wave amplitude and TPT were affected by both factors. The decrease in LDF muscle activation and M-wave amplitude indicated that impairment in muscle activation and membrane action

potentials contributed to fatigue with long duration contractions. The potentiation of SDF M-waves decreased the possibility of muscle membrane impairments. Potentiation of PF twitch torque and a lack of change in PF TPT in conjunction with decreases in M-wave amplitude would suggest impairments of PF membrane potentials. Fatigue-related decreases in quadriceps twitch torque and prolongation of TPT may be associated with disruptions in E-C coupling. Impairments in ½ RT were directly affected by fatigue duration and thus possibly related to muscle metabolic changes.

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Table 1: Subject Characteristics

Group	Height	Weight	Age	Gender (Male : Female)
75% PF	163.4 ± 7.1	71.7 ± 13.0	25.3 ± 3.4	5 : 3 (n = 8)
50% PF	160.9 ± 10.2	70.1 ± 12.1	21.7 ± 9.6	4 : 4 (n = 8)
25% quadriceps	166.8 ± 6.9	76.8 ± 17.4	24.6 ± 5.2	5 : 3 (n = 8)
50% quadriceps	162.8 ± 8.9	69.4 ± 21.6	22.4 ± 7.4	4 : 4 (n = 8)

Means and standard deviations (± value).

Table 2: Factors (muscle type or fatigue duration) affecting selected voluntary and evoked contractile properties.

	Muscle	Fatigue Duration
IEMG	yes	yes (?)
Twitch torque	yes	no
TPT	yes	yes
M-wave amplitude	yes	yes
1/2 RT	no	yes
Muscle activation	no	yes

IEMG: integrated electromyographic activity, TPT: time to peak twitch torque, M-wave: compound muscle action potential, 1/2 RT: half relaxation time

Figure Legends

Figure 1: Top two figures represent pre- (left) and post-fatigue (right) of the long duration fatigue protocol (25% MVC of the quadriceps), bottom figures show the pre- (left) and post-fatigue (right) of the short duration fatigue protocol (50% MVC of the quadriceps). The increase in the IT ratio post-fatigue was greater in the subject after the long duration fatigue protocol than after the short duration fatigue protocol. Also illustrated is the greater drop in MVC immediately after fatigue observed in the long duration fatigue protocol. The twitch preceding the ITT post-fatigue was compared to the unpotentiated evoked resting twitch (twitches generated at rest prior to any voluntary muscle contractions, data not shown) Y axis is in volts. Pre- and post-fatigue gains are the same. X axis represents time.

Figure 2: Long duration fatigue (LDF) and short duration fatigue (SDF) percentage drop in MVC following fatigue. LDF values represented by squares and SDF represented by triangles. Vertical arrow indicates a significant difference between groups while the horizontal arrow illustrates significant differences from pre-fatigue values for both groups. Vertical bars indicate ± SE.

Figure 3: Mean LDF and SDF percentage drop in the index of muscle activation over pre-, post-fatigue and recovery testing periods. LDF values represented by squares and SDF represented by triangles. Vertical arrow indicates a significant difference between groups while the horizontal arrow illustrates significant differences from pre-fatigue values for both groups. Vertical bars indicate ± SE.

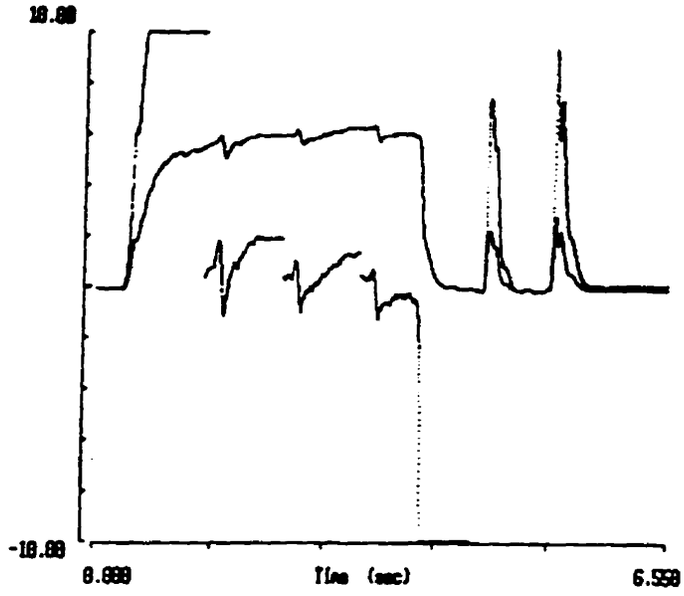
Figure 4: Quadriceps M-wave of two subjects pre- and post-fatigue. The decrease in the M-wave after the long duration fatigue protocol (25% MVC) contrasted with the increase observed after the short duration fatigue protocol (50% MVC). See legend in figure 1 for more details.

Figure 5: Mean LDF and SDF percentage change in TPT (top), 1/2 RT (middle) and M-wave amplitude (bottom) following fatigue. LDF values represented by squares and SDF represented by triangles. Vertical arrow indicates a significant difference between groups while the horizontal arrow illustrates significant differences from pre-fatigue values for both groups. Vertical bars indicate ± SE.

Figure 6: Mean Quadriceps and PF percentage change in Pt (top), TPT (middle) and M-wave amplitude (bottom) following fatigue. Quadriceps represented by hexagons and PF represented by stars. Vertical arrows indicate significant differences between muscles while the horizontal arrow illustrates significant differences from pre-fatigue values for both muscles. Vertical bars indicate ± SE.

Figure 7: Top two figures represent PF twitches before (left) and after (right) fatigue in one subject. The potentiation of PF twitches contrasted with the marked depression in quadriceps twitches. See legend in figure 1 for more details.

Figure 1



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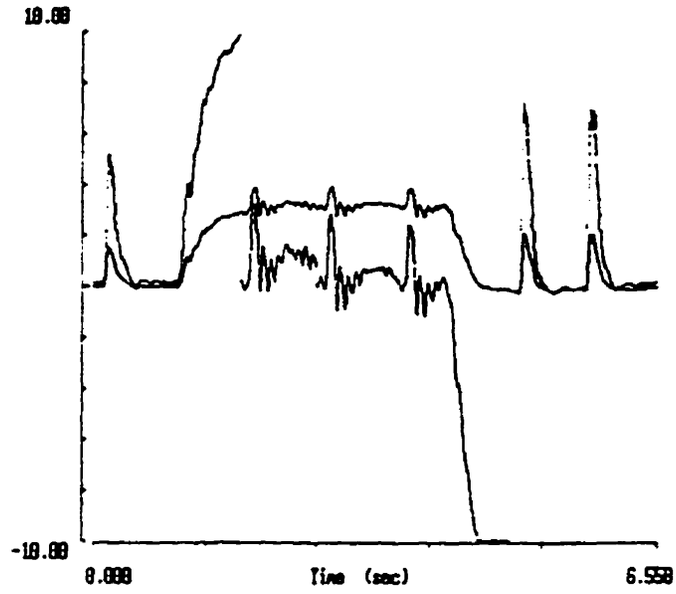

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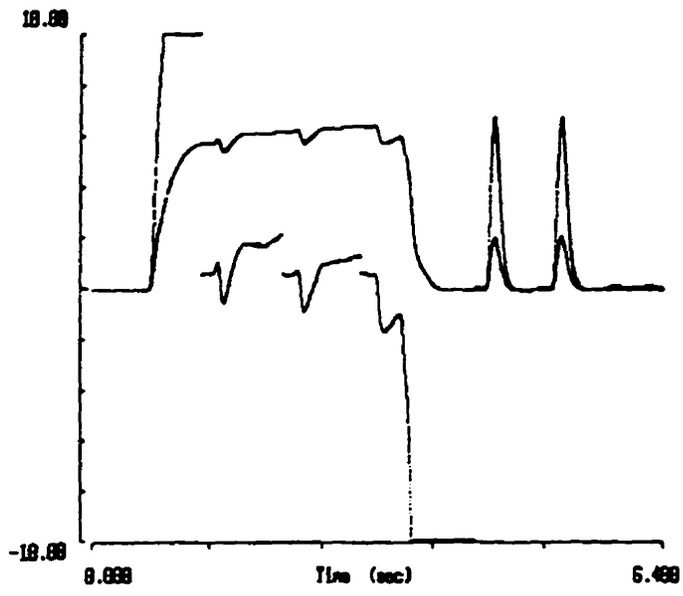

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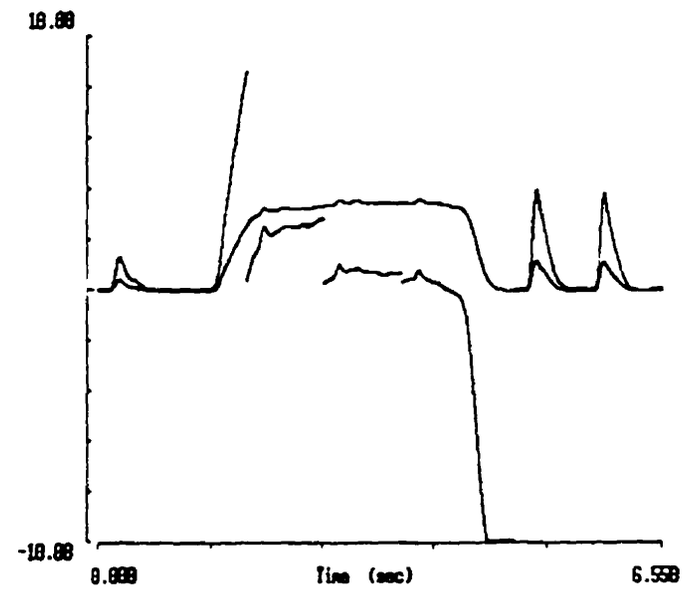

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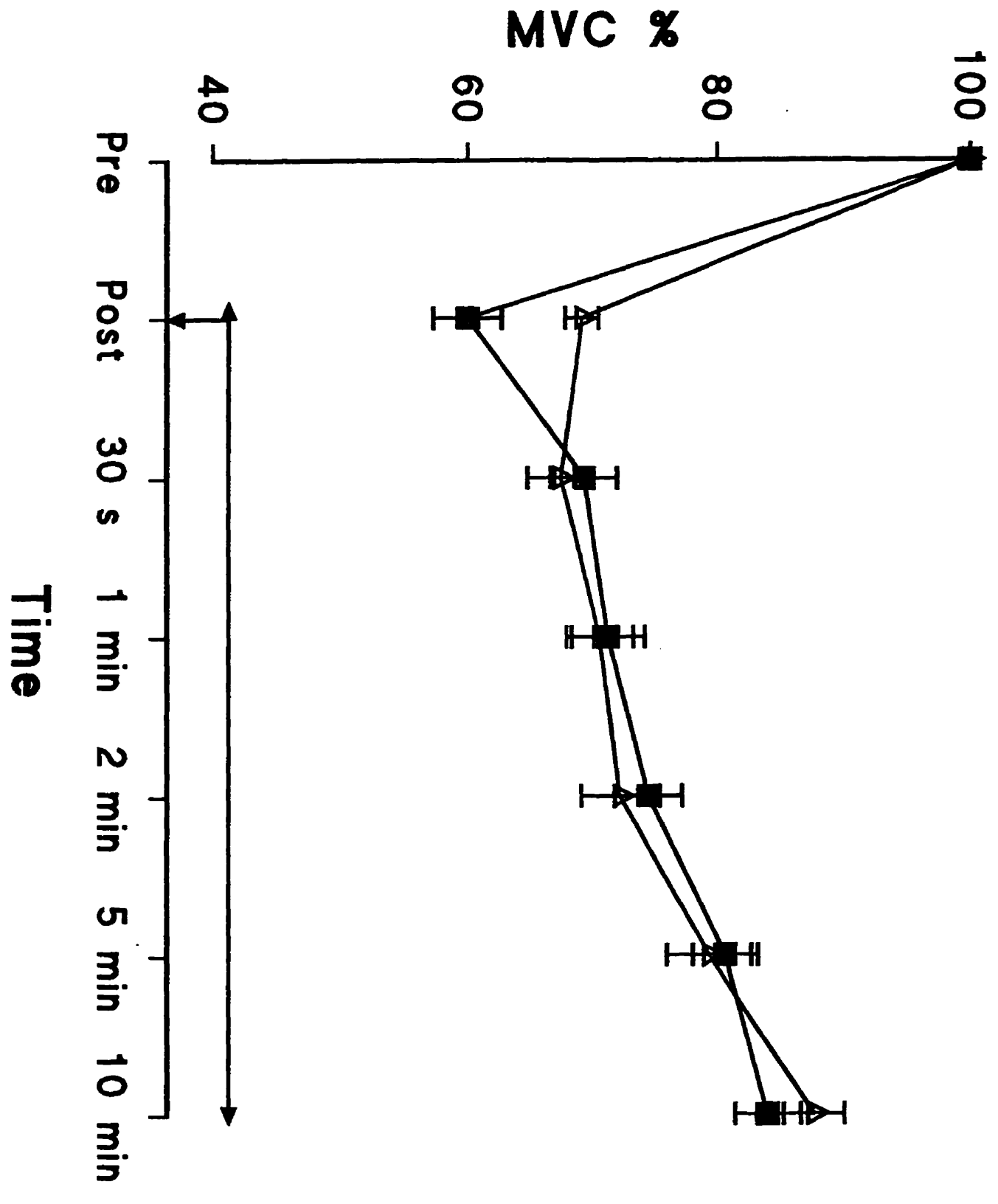

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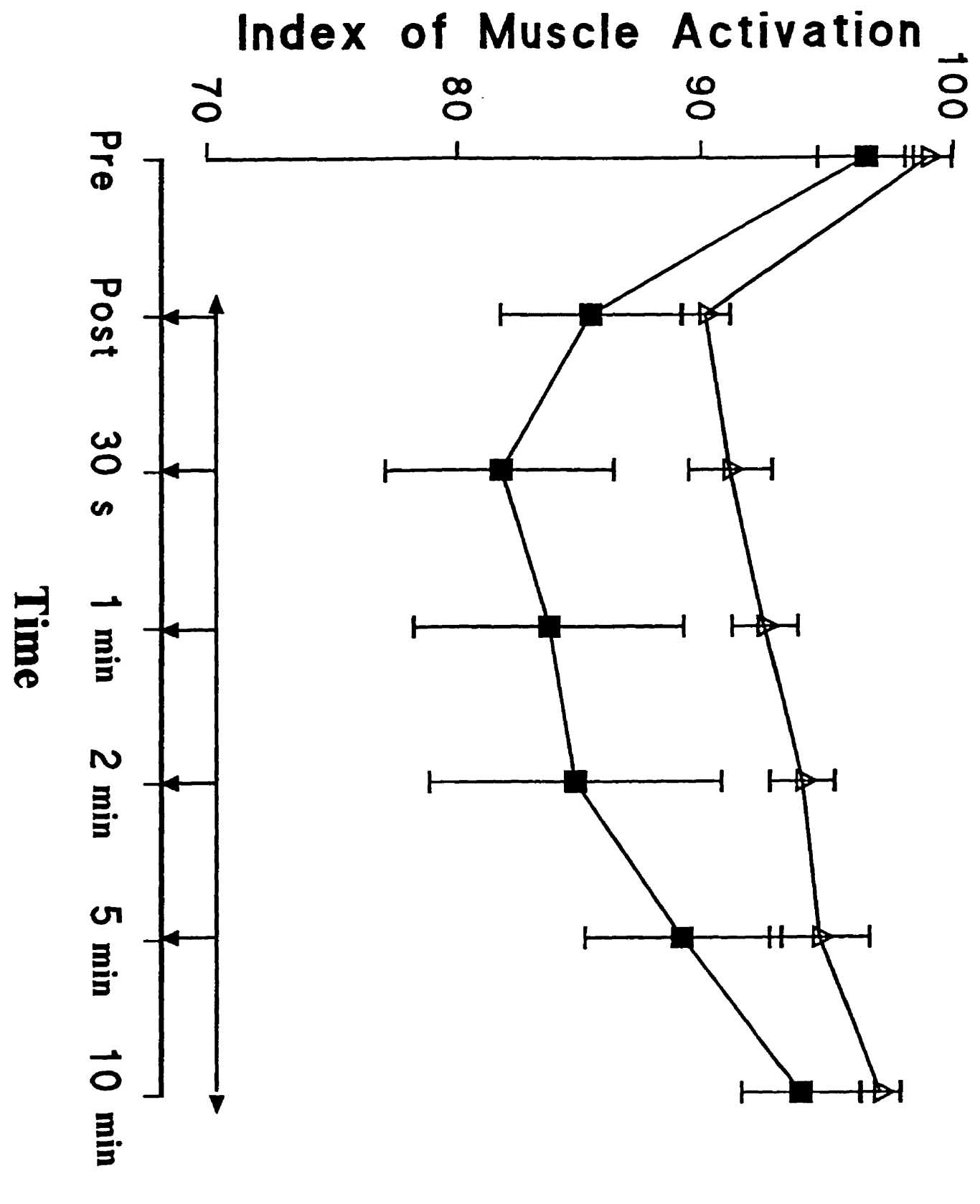
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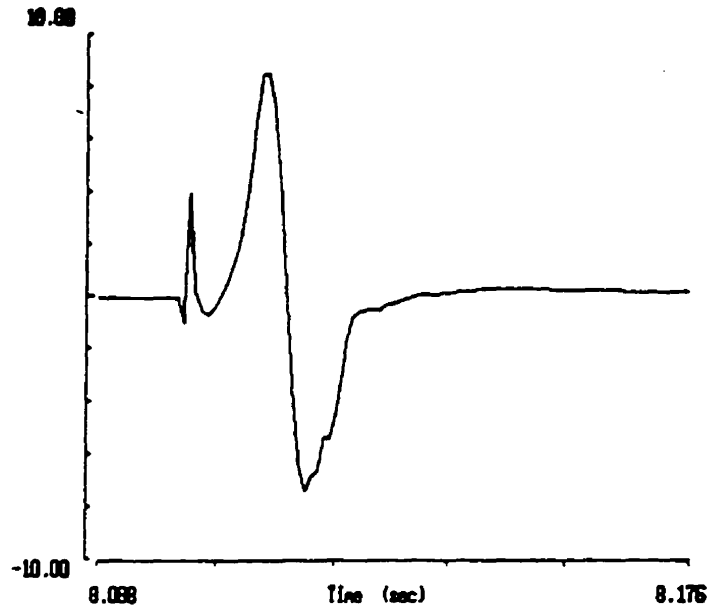
1 min
2 min
5 min
10 min
Time

Figure 4



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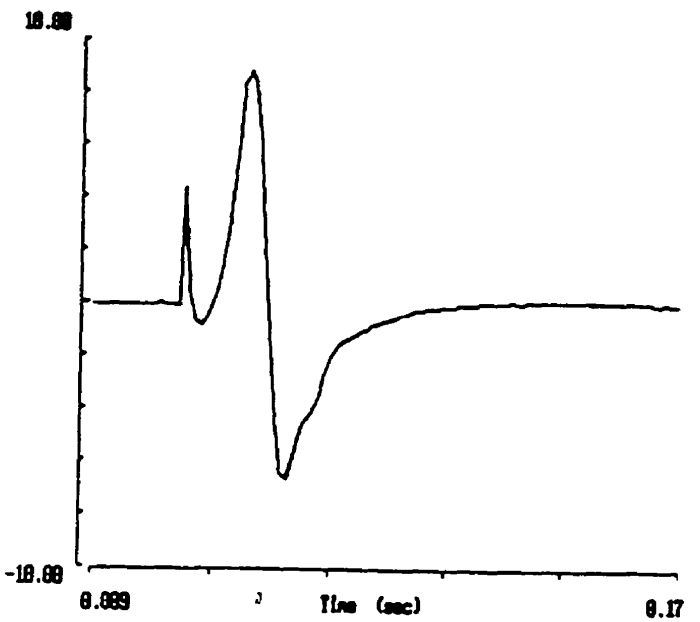

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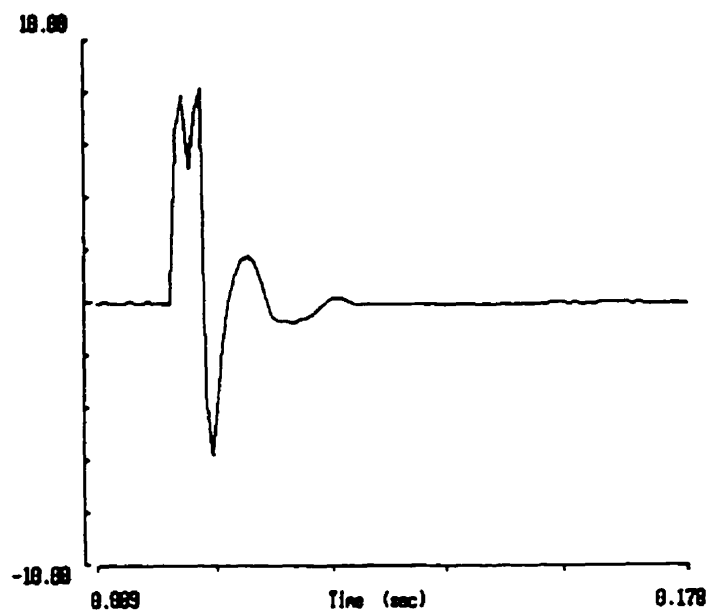

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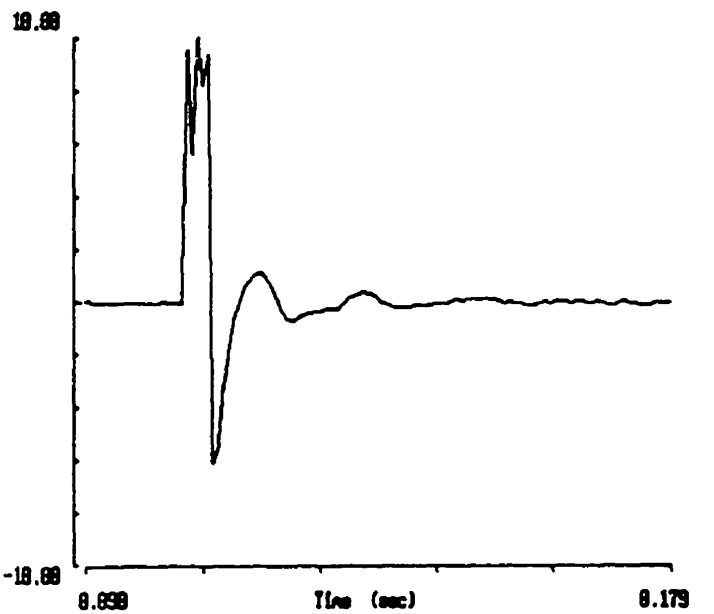

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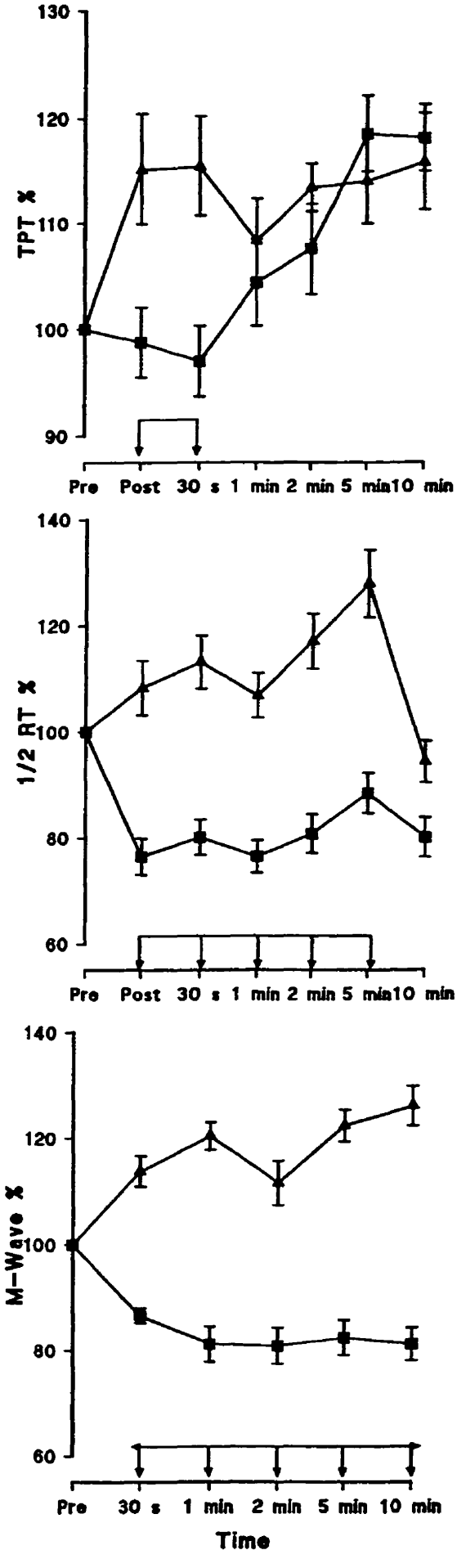
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1/2 RT %
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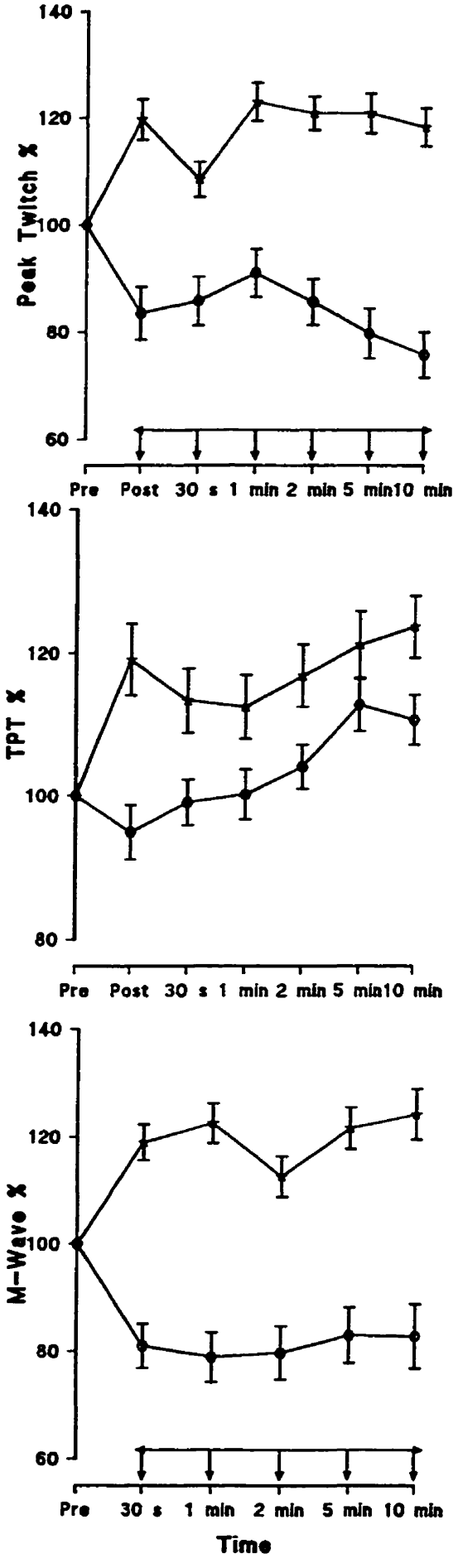
M-Wave %
Pre 30 s 1 min 2 min 5 min 10 min
Time

Figure 6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M-Wave %
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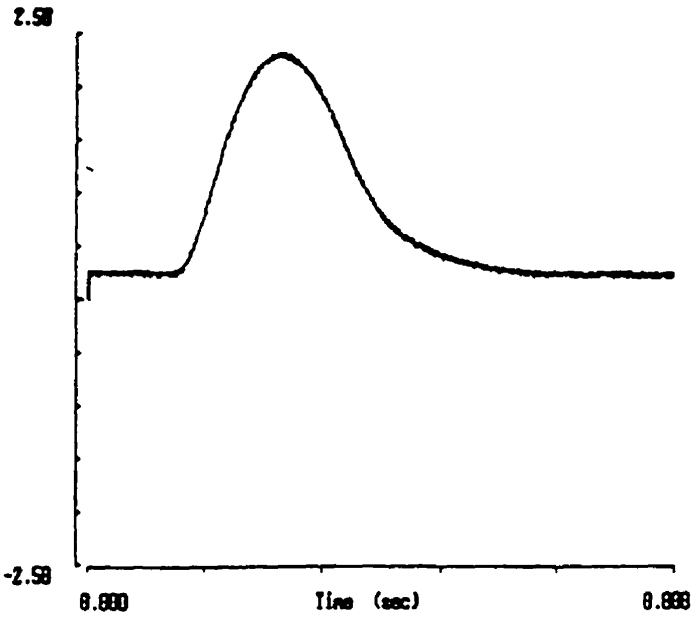
Time

Figure 7



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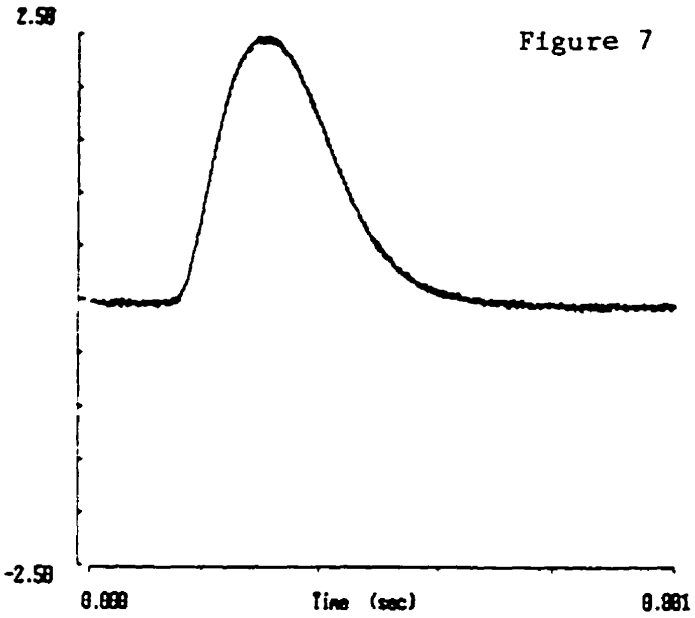

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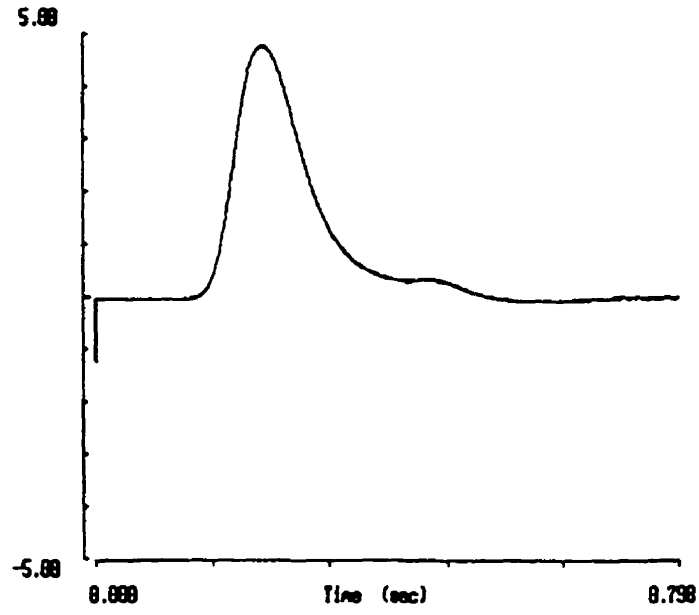

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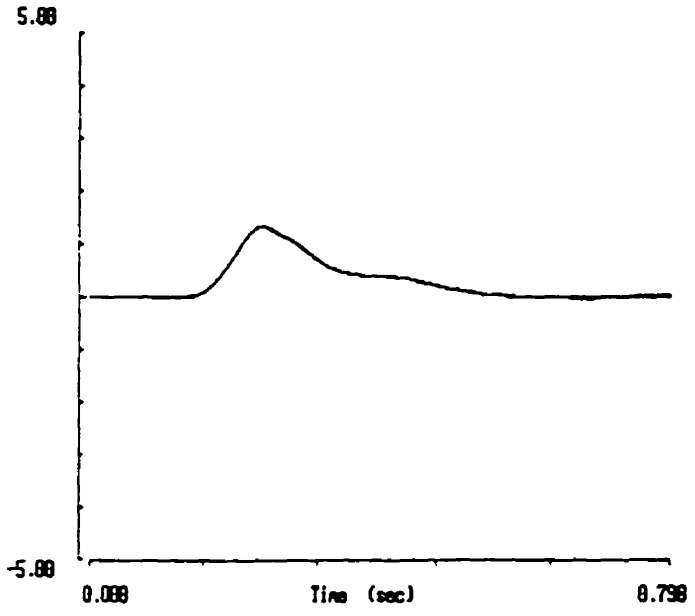

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Conclusions

The interpolated twitch (IT) ratio has been used in many studies to document changes in muscle activation. However, since the IT ratio-force relationship is best described by a shallow hyperbolic curve, second order polynomial equations provide a more accurate assessment of muscle activation. An error of 6% can be expected when maximal or near maximal voluntary contractions are utilized. Although the technique was shown to be reliable in both the plantarflexors and quadriceps, muscle force and kinetics could be altered with fatigue affecting the muscle activation-force relationship.

With the exception of an increased slope during recovery indicating decreased activation, plantarflexor IT ratio-force relationships were similar pre- and post-fatigue in ischaemic and non-ischaemic recovery groups. A lack of significant difference between second order polynomial predictions and estimates of muscle inactivation with a single IT ratio following fatigue, suggest that an IT ratio may be used as a general estimate of muscle inactivation post-fatigue. Since the muscle activation-force relationship was not significantly altered by fatigue, comparisons of pre- and post-fatigue activation levels should provide reliable and valid results.

The lack of trained state differences in evoked contractile properties under resting conditions were altered with fatigue. Whereas the decrease in muscle activation was similar following fatigue, deficits in untrained excitation-contraction coupling and significantly greater antagonist activity contributed to the tendency for trained subjects to perform a greater number of fatiguing contractions.

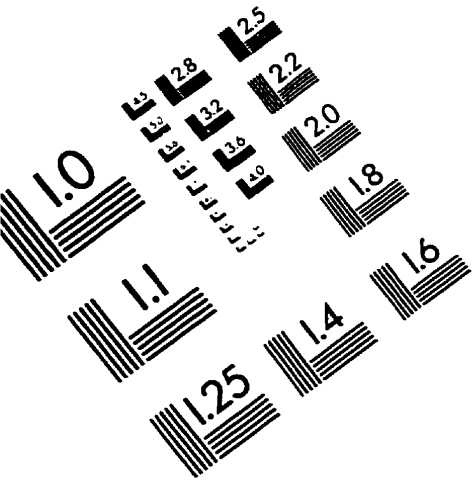
Disuse adaptations such as prolongation of membrane potentials and duration of the twitch can to a certain extent allow a significantly weaker muscle to maintain its relative fatigue

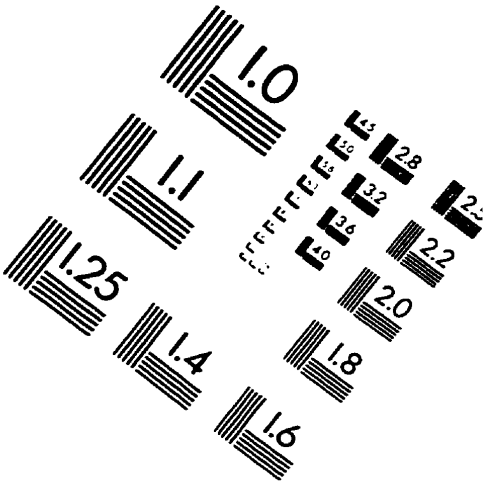
resistance. However, the severity of the fracture may adversely affect the muscle's fatiguability. The increased fatiguability of internally-fixated subjects was associated with increases rather than decreases in agonist and antagonist IEMG. Since, internally-fixated muscle did not experience more significant impairments in muscle activation, membrane excitability or contractile kinetics, the greater severity of the ankle fractures may contribute to an intrinsically more fatiguable muscle.

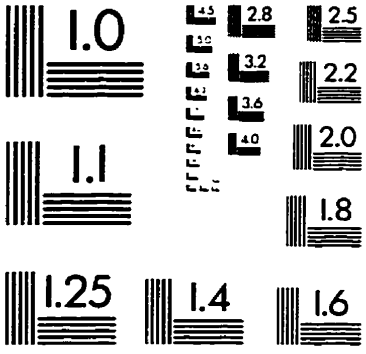
Fatigue-related muscle changes are specific to the fatigue protocol. In order to determine whether the previous findings can be generalized to other fatigue protocols and muscles, both the quadriceps and plantarflexors were subjected to different contraction durations. Changes in muscle activation and half relaxation time of the twitch were more prevalent with long duration fatigue protocols. Alterations in twitch amplitude and time to peak twitch however, were muscle specific with the greatest deficits occuring with the quadriceps. Muscle membrane action potentials were influenced by both the muscle used, and duration of the fatigue protocol.

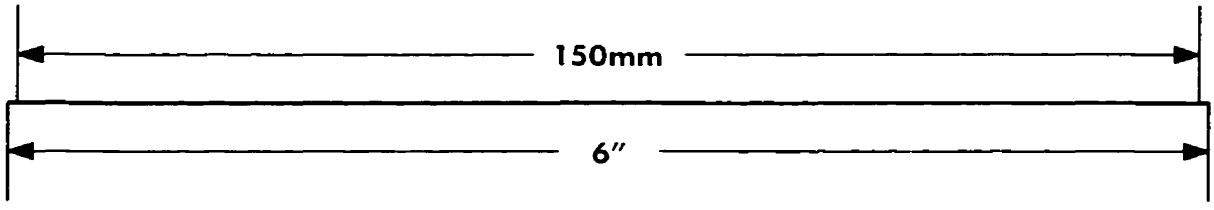
In conclusion, while training-induced adaptations of co-contractions and excitation-contraction coupling mechanisms may contribute to improved muscle endurance, specific disuse adaptations prolonging the duration of membrane excitation and twitch contractile properties help to maintain the fatigue profile of previously immobilized non-fixated muscle.

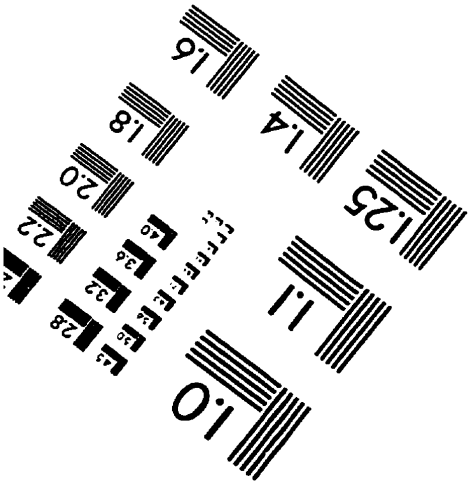
IMAGE EVALUATION
TEST TARGET (QA-3)

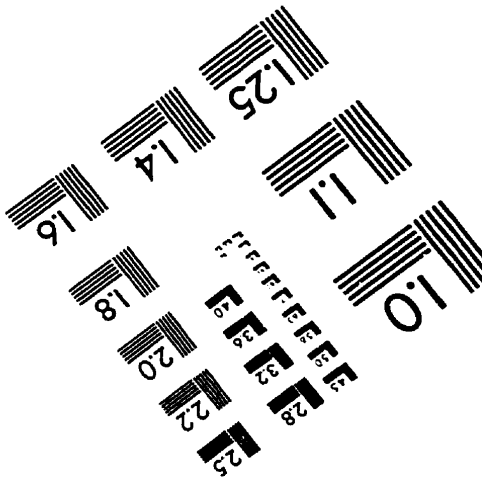












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