EFFECTS OF 22^{0}C MUSCLE TEMPERATURE ON THE RATE OF RECOVERY OF VOLUNTARY AND EVOKED CONTRACTILE PROPERTIES OF THE PLANTAR FLEXOR AFTER HIGH INTENSITY EXERCISE

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EFFECTS OF 22°C MUSCLE TEMPERATURE ON THE RATE OF RECOVERY OF VOLUNTARY AND EVOKED CONTRACTILE PROPERTIES OF THE PLANTAR FLEXOR AFTER HIGH INTENSITY EXERCISE

BY

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EFFECTS OF MUSCLE TEMPERATURE ON RECOVERY FROM EXERCISE

ABSTRACT

The primary goal of this study was to investigate the interaction effects of 22°C local muscle temperature on the recovery of specific evoked and voluntary contractile properties in intact human plantar flexors at 1-, 5-, and 10-minutes after high intensity, isometric exercise. The secondary goal of this study was to validate previous studies and add to the body of knowledge about the main effects of recovery from fatigue with homeostatic temperature and the main effects of local hypothermia on unfatigued muscle on evoked and voluntary contractile properties in intact human plantar flexors. Twelve subjects were tested for muscle voluntary and evoked contractile properties prior to fatigue (i.e. pre-fatigue), fatigued using intermittent, high-intensity, isometric contraction, and then retested at 1-, 5-, and 10-minutes post-fatigue conditions under localized hypothermic and normothermic conditions. Voluntary properties of the plantar flexor muscles were monitored by measuring the force of a maximal voluntary isometric contraction, as well as muscle activation derived from the interpolated twitch technique (ITT) and integrated electromyographic (iEMG) activity. Evoked contractile properties included the force and temporal characteristics of a maximal twitch and tetanic contraction of the plantar flexor muscles. Data were analyzed with a 2-way repeated measures ANOVA for main effects of a) hypothermic and normothermic conditions, b) prefatigue and at post-fatigue intervals, and c) interactions between hypothermic and normothermic pre- and post-fatigue intervals. During recovery from high intensity-intermittent fatigue there was a general augmentation of evoked properties of the plantar

flexors with a deceleration and decrease of the force of voluntary properties. Hypothermia had little effect on all but the temporal characteristics of the plantar flexor muscles, which were slowed by cold. The effect of cold on the rate of recovery of the plantar flexor muscles was generally not significant. Thus it may be concluded that local muscle hypothermia does not impair recovery of voluntary and evoked contractile properties of the plantar flexors from high intensity exercise.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS AND SYMBOLS

½ RT_{te} – half relaxation time of tetanus

½ RT_{tw} – half relaxation time of twitch

°C – degrees Celsius

ATP -- adenosine 5' -triphosphate

CA – cold arm

Ca^{+2} – calcium

CNS – central nervous system

CP – creatine phosphate

EMG -- electromyographic

FT – fast twitch muscle fiber

GAST – gastrocnemius

H-reflex – Hoffman reflex

Hz – Hertz (second $^{-1}$)

iEMG – integrated electromyographic

IT – interpolated twitch

ITT – interpolated twitch technique

kcal – kilocalorie

kph – kilometers per hour

LF – low frequency

M-wave – compound muscle action potential

min – minute

MFCV – muscle fiber conduction velocity

PF – plantar flexors

Pi – inorganic phosphates

PSA – power spectrum analysis

QUAD – quadriceps

Q_{10} –sensitivity of rate processes to temperature

R_{10} –sensitivity of force generating properties to temperature

RFD – rate of force development

RMS – root mean squared

$RTDT_w$ – rate of twitch torque development

$RTDT_e$ – rate of titanic torque development

RVTD – rate of voluntary torque development

s -- seconds

SR – Sarcoplasmic reticulum

ST – slow twitch muscle fiber

TA – tibialis anterior

TPT – time to peak twitch

T_{sk} – skin temperature

t-tubule – transverse tubule

LIST OF APPENDICIES

DEFINITION OF TERMS

Core temperature:

The temperature of the body core is a measure of the core compartment of the body. It can be measured by esophageal or rectal temperature probes.

Evoked Contractile Properties:

Properties of a muscle generated by electrical stimulation. Specific definitions of evoked contractile properties can be found in Appendix C (Table of Dependent Variables).

Excitation-contraction coupling:

The sequence of events by which an action potential in the plasma membrane of a muscle fiber leads to cross bridging activity by increasing cytosolic calcium concentration.

Interpolated Twitch Technique (ITT):

ITT is a method of estimating muscle activation by stimulating the muscle from an external electrical source. This will superimpose a maximal twitch during a muscle contraction and dividing the torque generated to a twitch delivered immediately after the voluntary contraction.

Peripheral temperature:

The temperature of a target limb is measured on the skin or intramuscularly.

Voluntary Contractile Properties:

Properties of a muscle generated by a subject's deliberate muscle activation. Specific definitions of voluntary contractile properties can be found in Appendix C (Table of Dependent Variables).

EFFECTS OF 22°C MUSCLE TEMPERATURE ON THE RATE OF RECOVERY OF VOLUNTARY AND EVOKED CONTRACTILE PROPERTIES OF THE PLANTAR FLEXOR AFTER HIGH INTENSITY EXERCISE

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INTRODUCTION

Early studies to elucidate precise causes of the effects of hypothermia on man's ability to perform work in the cold centered around the systemic effects of cold on military personnel acutely immersed in ocean waters, often relating to the Second World War (Glaser, 1950; Keatinge, 1965; Molar, 1946). More recent studies are exploring effects of peripheral cooling in order to isolate if certain effects are centrally or peripherally mediated (Giesbrechet et al., 1995). As manual labor, athletics, and recreation fields expand into cold environments, it is also relevant to begin studying how humans recover from fatigue in the cold. Military personnel, commercial SCUBA divers, runners, and hikers all perform work in cold environments. Often the insulation of their torsos is adequate to prevent central hypothermia, but they still experience peripheral cooling of the muscles (Giesbrechet et al., 1995). While studies have been conducted on the rate of recovery from fatigue under normothermic conditions (Behm and St.Pierre, 1997; Petrofsky et al., 1980) none have studied recovery while hypothermic. Therefore this study explored the effects of local hypothermia on recovering from local muscular fatigue following high intensity, isometric exercise.

The effects of ambient temperature on voluntary and evoked muscle contractile properties have been previously reviewed (Bennett, 1984). Also, both decreased ambient and muscle temperatures have been demonstrated to alter in different ways properties such as voluntary force (Bennett, 1985; Bergh and Ekblom, 1979; Faulkner, 1990; Ranatunga et al., 1987; Ranatunga and Wylie, 1982), EMG (Kossler et al., 1987), EMG power spectrum (Petrofsky and Lind, 1980) twitch and tetanic stimulation (Bell and Lehmann, 1987; Close, 1972; Kossler and Kuchler, 1987; Kossler et al., 1987; Ranatunga and Wylie, 1989), rate processes (Bennett, 1984;

Close and Hoh, 1968; Cornwall, 1994; Davies et al., 1982; Davies and Young, 1983; Faulkner, 1990; Kossler and Kuchler, 1987; Kossler et al., 1987; Ranatunga, 1982; Ranatunga, 1984; Ranatunga et al., 1987; Ranatunga and Wylie 1982; Ranatunga and Wylie, 1983; Rome, 1990; Segal et al., 1986) and endurance (Bennett, 1984; Edwards et al., 1972; Ranatunga, 1977; Rome, 1990) depending on the property studied, muscle fiber type, and the extent of cooling. However it is not known to what extent these alterations are caused by central (i.e. from the brain to the neuromuscular junction) or peripheral (i.e. after the neuromuscular junction) factors.

Similarly, muscle fatigue has been extensively reviewed (Sale, 1988) and studied with the interpolated twitch technique (Allen et al., 1995, Behm et al., 1996). Properties studied after fatigue (i.e. during recovery) include twitch torque (Behm and St.Pierre, 1997; Petrofsky et al., 1980), tetanic tension (Petrofsky et al., 1980), temporal characteristics (Behm and St.Pierre, 1997; Petrofsky et al., 1980), compound muscle action potential (Behm and St. Pierre, 1997; Bellemare and Garzanti, 1988; Bigland-Ritchie et al., 1979; Fuglevand et al., 1993; Petrofsky et al., 1980), MVC (Behm and St. Pierre, 1997; Pääsuke et al., 1997; Stull and Clarke, 1971), endurance (Behm and St. Pierre, 1997; Petrofsky et al., 1980), EMG power spectrum (Le Bozek and Rougier, 1991; Stulen and de Luca, 1982), and power (de Hann et al., 1989; James et al., 1995; Nilsson et al., 1977). Recovery of voluntary and evoked contractile properties after both high and low intensity fatigue protocols in normothermic conditions has also been studied (Behm and St.Pierre, 1997). Each factor previously studied is altered in different ways by fatigue depending on the property studied, the muscle group, and the intensity and duration of the fatigue protocol. Since the effects of cold, fatigue, and recovery from fatigue have been studied independently, interaction of the two effects should be studied next. While the effects of cold environments on muscle activation are

reasonably well documented, a distinct lack of mammalian experiments in recovery from fatigue while cold indicates that further research in the area of recovery from fatigue of mammalian muscle while cold is needed.

Therefore the goals of this study were two-fold. The primary goal of this study was to investigate the interaction effects of 22°C local muscle temperature on the recovery of specific evoked and voluntary contractile properties in intact human plantar flexors at one-, five-, and 10-minutes after high intensity fatigue. The secondary goal of this study was to validate previous studies and add to the body of knowledge about the main effects of recovery from fatigue with homeostatic temperature and the main effects of local hypothermia on unfatigued muscle on evoked and voluntary contractile properties in intact human plantar flexors.

REVIEW OF LITERATURE

2.1 Introduction

Military personnel, outdoor workers, and many individuals involved in outdoor pursuit activities and sports perform work in a cold environment. In the Second World War, pilots and mariners often reported having difficulty staying afloat or holding flotation devices for extended periods of time in cold ocean water (Glaser, 1950; Keatinge, 1965; Molar, 1946). In modern society hypothermia continues to be a concern. In 1997, 39 Canadian water-related fatalities cite hypothermia as a contributing cause of death (Barss, 1999), as immersed boaters find that trying to swim to shore or hold on to a flotation device becomes more rapidly fatiguing in cold water. Canoe paddlers find it increasingly difficult to hold a paddle once the hands get cold. SCUBA divers have increasing difficulty with fine motor tasks once cold, and military personnel find their movements much slower when cold (Giesbrecht et al., 1995).

The effects of cold on muscle voluntary and evoked contractile properties have been previously reviewed (Bennett, 1984), and numerous experiments have been conducted on voluntary force (Bennett, 1985; Bergh and Ekblom, 1979; Faulkner et al., 1990; Ranatunga et al., 1987; Ranatunga and Wylie, 1982), EMG (Kossler et al., 1987), and twitch and tetanic stimulation (Bell and Lehmann, 1987; Close, 1972; Kossler and Kuchler, 1987; Kossler et al., 1987; Ranatunga and Wylie, 1989). Rate processes like time to twitch or voluntary contraction or relax a muscle contraction (Bennett, 1984; Close and Hoh, 1968; Cornwall, 1994; Davies et al., 1982; Davies and Young, 1983; Faulkner, 1990; Kossler and Kuchler, 1987; Kossler et al., 1987; Ranatunga, 1982; Ranatunga, 1984; Ranatunga et al., 1987; Ranatunga and Wylie 1982; Ranatunga and Wylie 1983; Rome, 1990; Segal et al., 1986) and rate of onset of fatigue

(Bennett, 1984; Edwards et al., 1972; Ranatunga, 1977; Rome, 1990) have also been studied. All of these listed properties have been shown to either increase or decrease by changes in temperature depending on the property being studied.

Inconsistent findings regarding the effects of cold on muscle may often be accounted for by variations in the level of temperature, the length of cold exposure, the method of cooling, method of temperature monitoring, and even the amount of body fat of the subject (Petrofsky and Lind, 1975). Similar inconsistent findings regarding the effects of fatigue may be explained by studying different muscle fiber types, training state of the subject, and duration and intensity of contractions studied (Behm and St. Pierre, 1997). Also important when studying fatigue is knowing if contractions are sustained or intermittent, evoked or voluntary, and static or dynamic (Behm and St. Pierre, 1997). This review will approach these details of cold and fatigue research in a way that describes the effects of cold on peripheral muscle and then the effects of fatigue on peripheral muscle so that inferences may be drawn to the interaction effects of 22°C local muscle temperature on the recovery of specific evoked and voluntary contractile properties in intact human plantar flexors at one-, five-, and 10-minutes after high intensity fatigue. The goal will be to investigate the main effects of recovery from fatigue with normal homeostatic temperature and the main effects of recovery from fatigue with local hypothermia on evoked and voluntary contractile properties in intact human plantar flexors.

2.2 Measuring Thermal Dependence of Muscle

Commonly used abbreviations used in research relating to how temperature affects a muscle's properties are Q_{10} and R_{10}. These abbreviations represent a ratio of normothermic and

hypothermic responses. Temperature dependence of rate processes of muscle is referred to as a "Q_{10}" score (Rome and Bennett, 1990) while an "R_{10}" score relates to force of the muscle (Rome and Bennett, 1990). In both scales a rating of 1.0 conveys no change or dependence, less than 1.0 is a negative dependence, and greater than 1.0 is a positive dependence. A score of greater than 2.0 or less than 0.5 conveys a strong thermal dependence. Q_{10} can be calculated in the following formula:

$$Q_{10} = (R_2/R_1)^{[10/(T_2-T_1)]}$$

in which R_2 and R_1 are rate processes at temperatures T_2 and T_1 (Rome and Bennett, 1990). R_{10} can be calculated by a similar formula:

$$R_{10} = (F_2/F_1)^{[10/(T_2-T_1)]}$$

in which F_2 and F_1 are forces at temperatures T_2 and T_1 (Rome and Bennett, 1990).

2.3 Functional Consequences of Cold and Fatigue on Muscle Function

2.3.1 Maximal Voluntary Contractions

2.3.1.1 Hypothermic

At a muscle temperature of 25°C isometric force production has low temperature dependence in humans (Bennett, 1985; Bergh and Ekblom, 1979; Ranatunga et al., 1987), mice (Faulkner et al., 1990) and other animals (Edwards et al., 1972; Ranatunga and Wylie, 1983). Maximal force in humans and other animals tends not to be altered by peripheral muscle temperatures when those temperatures are between 25 and 35°C and is represented by a R_{10} of 1-1.2. (Clarke et al., 1958a; Rome, 1990). Voluntary force remains unchanged with peripheral muscle temperatures of 35°C to 25°C but decreases 30% in the 12-15°C range, and at 10°C has

decreased 40% in both humans (Ranatunga et al., 1987) and in rats (Ranatunga and Wylie, 1983). Other studies demonstrate similar impairment but at slightly higher temperatures. Human muscles below 27°C have been shown to decrease isometric grip strength by 14.8% in males and 30.5% decrease in females though the authors did not offer an explanation of why the sex difference existed (Cornwall, 1994). Other studies indicate maximal force declines by 3-5% /1°C drop in muscle temperature (Bergh and Ekblom, 1979; Sargeant, 1987), though this kind of linear relationship is not supported by most existing research (Bennett, 1985; Bergh and Ekblom, 1979; Faulkner et al., 1990; Ranatunga et al., 1987; Ranatunga and Wylie, 1982). While the exact mechanism of changes during peripheral cooling has not been identified, some research (Giesbrechet et al., 1995, Ranatunga and Wylie, 1982) focused on the depression of voluntary tension due to a direct effect of cold on muscle and not from central mechanisms, while others (Heier et al., 1994) focused more on central effects.

Rather than report muscle temperature, several studies report water, skin, or air temperature, a factor that may account for conflicting results. Holewijn and Heus (1992) found that immersion in 15°C water for 30 minutes resulted in significant 21.8% decline in maximal force of grip strength. However, Bundschuh and Clarke (1982) found no change in initial or final grip strength with 10 minutes of immersion of the forearm in 10°C water immersion, results that also conflict with Petrofsky and Lind (1980). Clarke and Wojciechowics (1978) demonstrated that as temperature was lowered, final strength and total work increased, rate of fatigue was slower (i.e. endurance increased), and initial force output was not significantly lowered with cold. Unfortunately, muscle or body core temperatures were not reported in these studies.

Therefore it is generally well accepted that thermal dependence of maximal voluntary force output is generally low at a muscle temperature above 25°C.

2.3.1.2 Fatigue

The effects of fatigue on MVC has been extensively studied (Behm and St. Pierre, 1997; Pääsuke et al., 1997; Stull and Clarke, 1971) and reviewed (Enoka and Stuart, 1992; Fitts, 1996). Fatigue may be defined as "an acute impairment of performance that includes both an increase in the perceived effort necessary to exert a desired force and an eventual inability to produce this force (Enoka and Stuart, 1992, p. 1631). The effects of fatigue and recovery from fatigue are dependent upon the duration of the exercise period, the type of muscle contraction, the rest period between contractions, the intensity of the contraction, and the length of time per contraction (Behm and St. Pierre, 1997).

Impairment of maximal contraction during high intensity, isometric, intermittent fatigue is likely caused by a build-up of inorganic phosphates and production of hydrogen ions from the glycolytic pathway that directly inhibits cross-bridging (Westerbland and Allen, 1991). Fatigue may also result from impairment of the dihydropyridine (DHP) receptors that result from a decreased depolarization gradient thereby inhibiting calcium release (Fitts, 1996). Accumulating hydrogen ions also bind with troponin-C thus inhibiting the binding of calcium. Slightly longer duration fatigue may also result from decreased creatine phosphate (CP) (Hargreaves et al., 1998). Thus, impaired MVC results from impairment of E-C coupling and decreased cross-bridge cycling. Impairment of E-C coupling is more severe from long duration exertion and is the major cause of low frequency fatigue (Jones, 1996) while metabolic disturbances may be the predominant factor

affecting the functioning of myofilament cross-bridging during short duration, high intensity fatigue (Bigland-Ritchie and Woods, 1984).

2.3.2 Sub-maximal Voluntary Contractions

2.3.2.1 Hypothermic

It has been generally well documented that maximal muscle endurance occurs with peripheral muscle temperatures of 25-32°C though there is not yet agreement on the exact temperature for optimal endurance (Bennett, 1984; Edwards et al., 1972; Faulkner et al., 1990; Segal et al., 1986). Lind and coworkers (Clarke et al., 1958a; Clarke et al., 1958b; Lind, 1959; Lind and Samueloff, 1957; Petrofsky and Lind, 1980) have found maximal endurance of sustained sub-maximal isometric muscle contractions in humans occurred in 20°C water with a similar muscle temperature of 27°C, a muscle temperature that was employed by Davies and Young (1983). Similar findings indicated the endurance of a fatiguing isometric contraction of cat soleus muscle was three times longer at 28°C than at 22 or 38°C (Kossler and Kuchler, 1987).

While muscle force generating ability remains reasonably constant with cooling to approximately 25°C, temporal characteristics, as will be discussed later in this review, steadily decline thereby reducing power. Ranatunga (1977) explained that rats attempt to generate the same power and force no matter what the temperature. They accomplish this through a neural mechanism referred to as "compression of the recruitment order" which is recruiting more muscle fibers at low temperatures than at high ones. The muscle fibers are recruited in the same order at different temperatures (i.e. slow twitch fibers are recruited before fast twitch). The lower power output of

slow twitch fibers at lower temperature causes the faster, more powerful fibers to be recruited earlier. Therefore, at a given workload, more fast twitch muscle fibers are used at lower temperatures. For example, EMG activity increased by 25% as skin temperature (T_{sk}) is decreased below 19-20°C (Rissanen et al., 1996). Note that this is total activity as measured by integrated activity, not firing frequency. This lowers endurance at a given work load at low temperatures due to a greater use of glycolysis (Faulkner et al., 1990; Rome, 1990).

At a muscle temperature range of 25-32°C, a lower firing frequency may compensate for the increased rate of glycolysis, thereby allowing increased endurance. At a stimulation rate of 28Hz, the extensor digitorum longus had single twitches at 30–40°C, unfused tetanus at 25°C, and fused tetanus at 20°C (Segal et al., 1986). As muscle temperature increases, the TPT and ½ RT_{tw} shorten and the stimulation frequency must be increased to attain similar levels of force development (Close and Hoh, 1968; Ranatunga, 1982; Segal and Faulkner, 1985; Segal et al., 1986). This effect is attributed to the accompanying increase in myosin ATPase activity and calcium sequestering by the sarcoplasmic reticulum (SR) as muscle temperature increases (Segal et al., 1986). There is also a shift of cold muscle to lower firing frequencies that will be discussed later in this review.

The reason for optimal endurance at muscle temperatures of 25-32°C could be explained by the fluctuation of muscle temperature during rest and exercise. At rest and following exhaustive exercise in an environmental temperature of 20-25°C the temperatures of leg muscles are 34°C and 41°C (Bishop et al., 1975) respectively. In an ambient temperature of 5°C and exposed to wet and wind, resting muscle temperature declined to 23°C (Segal et al., 1986). The depression of endurance may be due to low temperatures inhibiting neuromuscular transmission

along the motor nerves, at the neuromuscular junction, or along the sarcolemma and to failure of the muscle fibers due to factors such as increases in glycolytic metabolism while cold (Ranatunga et al., 1987). The attenuation of local muscular endurance in of muscles in a temperature range of 25-32°C may be due to slowing the energy release and local metabolism of the muscle cells by colder temperatures.

2.3.2.2 Fatigue

The rapid decrease in the endurance time at sustained isometric levels for contractions above 15-20% MVC has been attributed to restriction in blood flow in the muscle, producing an ischaemic effect due to the increased intramuscular pressure from the heavy load (Körner et al., 1984). Ischaemia in turn causes an inability to clear the muscle of metabolites and depletes the muscle of oxygen, thereby increasing lactate production and impairing the contraction process. Ionic imbalance (e.g. K^+), and reduced energy substrates (e.g. CP) impair a variety of contractile processes. Decrements associated with impaired oxygen delivery and metabolic removal will have direct effects upon the muscle as well as effects upon the motoneuron. Further detail regarding the mechanisms of these impairments will be presented further in this review.

As previously presented, metabolic by-produced such as hydrogen ions (Westerbland and Allen, 1991), inorganic phosphates (Westerbland and Allen, 1991), and potassium (McKenna, 1995) are major contributors to high intensity fatigue. Blood flow serves to flush accumulated metabolites to assist recovery (Viires et al., 1983). Reactive hyperaemia is known to increase blood flow immediately after occlusion originating from intense muscle contraction (Pitcher and Miles, 1997). Many metabolites, such as H^+, lactate, and nitric oxide are also known to mediate

functional hyperaemia (Balon and Nadler, 1994; Pitcher and Miles, 1997; Poucher, 1995). Therefore increased blood flow could result if metabolites accumulated at a faster rate due to both contraction-induced and metabolic by-product induced hyperemia thereby removing metabolites faster, and thus enhancgin recovery (Badier et al., 1994; Pitcher and Miles, 1997).

2.4 Neuromuscular Mechanisms of Voluntary EMG and Power Spectrum

Electromyograph signals (EMG) represent the total electrical activity in a given muscle. EMG signal is typically rectified and then measured for the area under the curve as integrated EMG (iEMG). It is assumed that recruitment, firing frequency and/or muscle fiber conduction velocity of the muscle will cause the area of the EMG signal to increase (Petrofsky and Lind, 1980). Engineering analysis on signals may also involve power spectrum analysis (PSA) (Kwatny et al., 1970). The power spectrum represents the average distribution of the power, or area under the curve, across the frequency range of interest (Kamath and Fallen, 1993). The PSA can be used to analyze iEMG signals and isolate how a certain firing frequency contributed to the total contraction (Freeman et al., 1991). PSA provides the basic information of how power distributes as a function of frequency.

2.4.1 Hypothermia

Unfortunately, many studies do not consider the effects of cooling the body core on muscle temperature or how peripheral cooling alters central mechanisms. Several studies (Giesbrecht et al., 1995; Heier et al. 1989; Plattner et al.; 1996) have demonstrated that there is little restriction of heat flow between peripheral and core tissues. Isolating cooling to the body

core can even alter evoked contractile properties of peripheral muscles (Heier et al., 1994). Therefore if the core cools, central temperature changes will further modify peripheral activity and temperature. While these effects may be minimal (Giesbrecht et al., 1995), they are nevertheless present. Since Giesbrecht et al. (1995) did not study evoked contractile properties, not finding central effects does not necessarily contradict Heier et al. (1994) who found significant central effects on peripheral action but did not actively control limb temperature.

Signals from the central nervous system (CNS) contribute to the control of rate of force development and force output (Sale, 1988). FitzGibbon et al. (1984) concluded that impairment of the signals from the CNS would only occur at a muscle temperature of below 33° C, though mild hypothermia may cause mild impairment that would be detectable in physical performance (Clarke et al., 1958a). This may indeed be the case since Giesbrecht et al. (1995) averaged a low esophageal temperature of 35.6°C during total body cooling which was responsible for 2-15% of the decreases found in a variety of motor tasks. This temperature would not be cold enough, according to FitzGibbon et al. (1984) to evoke central changes, thus supporting Clarke et al. (1958a). Impairment of the signals of the CNS to the muscles may be a result of decreased firing frequency induced by cold.

EMG amplitude has been shown to be highest at a muscle temperature of 20°C (Mucke and Heuer, 1989; Winkel and Jorgensen, 1991), increasing by 3.5% /1°C (Zipp, 1977) though decreases of EMG begin below 20°C. Possible explanations for this increase include increased duration of the action potential, compression of the recruitment order, or loss of force due to cold necessitating increased recruitment and firing frequency (Winkel and Jorgensen, 1991). As there is more activity in the muscle, it may be expected that endurance of muscle contractions will

decrease. However while more fibers are active, as demonstrated with PSA, they are active at a lower frequency, thereby extending endurance time.

Petrofsky and Lind (1980) noted that changes in EMG PSA due to decreasing muscle temperature have been found to resemble the effects of fatigue-induced muscle wisdom, (i.e. a decrease of muscle firing frequency in order to maintain force) showing similar changes in amplitude and frequency to lower frequency components. For example, Oska et al. (1997) found during the shortening phase of maximal rebound jumps, the mean power spectrum of agonists declined from 124 Hz at 27°C to 82 Hz at 10°C ambient temperature. Slow twitch motor units tetanizing at lower frequencies due to prolonged twitch duration at cooler temperatures likely cause this effect. Thus fibers are recruited at a more economical frequency of discharge (Petrofsky and Lind, 1980). Since FT fibers are recruited earlier, thus increasing the rate of fatigue, defense mechanisms to earlier fatigue will be beneficial to maintaining contractions.

As central changes have only a mild to moderate effect on peripheral activity, as discussed by Giesbrecht et al. (1995), changes of the periphery tend also to have minimal effects on central activation. Several experiments have experimented with the effects of skin (Kregel et al., 1992) and muscle (Ray et al., 1997) cooling on autonomic nervous activity, specifically muscle sympathetic nervous activity. Their observations do not indicate that temperature-sensitive muscle afferents regulate muscle sympathetic nervous activity during peripheral cooling. Similarly, Rissanen et al. (1996) demonstrated that altering the temperature of one leg did not alter the EMG of the contralateral leg, though it did increase EMG activity of the cooled leg. This indicates that that EMG activity increase is not due to increased sympathetic outflow

since the effect of this would be systemic. Thus, mild alterations of local cooling do not seem to alter autonomic nervous activity.

2.4.2 Fatigue

Both average amplitude of EMG and total power of the EMG power spectrum have a linear relationship to increasing force (Zedka et al., 1997) in moderate (i.e. 20°C) temperatures (Petrofsky and Lind, 1980), though it becomes curvilinear at lower intensities and lower temperatures (Petrofsky and Lind, 1980). Increasing muscle tension occurs by recruiting additional motor units (i.e. increasing recruitment) or by increasing firing frequency of already firing motor unit (i.e. increasing rate coding) (Deluca, 1985). By increasing either or both of these, EMG output will increase.

Linssen et al. (1990) demonstrated that there is an initial increase in EMG during intermittent submaximal isometric contractions. The initial increase may be accounted for by increasing recruitment of motor units, and synchronization of motor unit firing (Bigland-Ritchie and Woods, 1984). During sustained contractions EMG initially increases, plateaus, and as fatigue progresses EMG begins to decrease (Linssen et al., 1990). This decrease is an effect likely caused by decreasing muscle fiber conduction velocity (MFCV) (Linssen et al., 1990), a conclusion supported by proportional declines of MFCV and surface EMG (Lateva, 1988) though may also be linked to muscle wisdom (Marsden et al., 1983).

Central fatigue from maximal or near maximal contractions results in a decline of neural discharge during sustained contraction. A feedback loop from the muscle via the Ia afferent to central activation causes a decrease of the α-motoneuron discharge rate and force generation.

often referred to as "muscle wisdom" (Marsden et al., 1983). As muscle fatigue increases, central neural drive attempts to maintain force generation by decreasing the discharge rate (Bigland-Ritchie et al., 1986b) so that the discharge rate is maintained at the minimum level to maintain force (Bellemare et al., 1983), thereby optimizing firing frequency to the desired force output in an attempt to postpone central neural fatigue (Enoka and Stuart, 1992). Reflexes from the muscle to central mechanisms via afferents are responsible for this effect (Bigland-Ritchie et al., 1986b), a theory that seems likely since this reduction of firing frequency can be prevented by blocking the muscle afferents (Hagbarth and Macefield, 1995).

During fatigue there is also increased antagonist muscle activity (i.e. lower levels of reciprocal inhibition) (Moritani, 1993). There is diminished discharge from the Ia afferent as well as increased inhibition from the Ib afferents (Häkkinen and Komi, 1986; Kraemer et al., 1988) and additional inhibition from type III and IV afferents (Badier et al., 1993). These inhibitors are speculated to be a form of protection against muscle and joint damage found in the muscle proprioceptors and connective tissue (Kraemer et al., 1988). By increasing antagonist activity, total voluntary force is reduced and energy expenditure increases as the antagonist muscle group resists force production.

Le Bozek and Rougier (1991) demonstrated a shift of the EMG PSA to lower frequencies, indicating a decline in the mean power frequency. Also, the amplitude of the low frequency peak and the power of the corresponding component in the EMG PSA increased, thereby decreasing the mean power frequency of the sample (Le Bozek and Rougier, 1991; Stulen and de Luca, 1982). While the mean frequency declines, as muscle fatigue begins, the total power of the EMG signal increases (Kwatny et al., 1970). This is produced by an elevation

of the low frequency portion of the spectrum that outweighs a simultaneous decrease in the high frequency portion, in addition to an increase in the synchronization of motor units with fatigue.

The most common explanations for the shift of PSA to lower frequencies include changes in the synchronization of motor units and decreasing MFCV (Datta and Stephens, 1990; Naeije and Zorn, 1982; Stulen and de Luca, 1982; Zwarts et al., 1987). There is a linear relationship between the median frequency and conduction velocity (Stulen and de Luca, 1981). Van der Hoven et al. (1993) showed decreases of median power frequency and MFCV during their one-minute isometric MVC fatigue protocol. Patients with McArdle's Disease, a deficiency of myophosphorylase resulting in an inability to use muscle glycogen as fuel, do not produce intramuscular lactate. Such patients also have a significant shift of the PSA to lower frequencies (Mills and Edwards, 1984). Therefore Mills and Edwards (1984) speculate that the shift to lower frequencies is due to disturbances of muscle membrane excitability such as extracellular potassium ions, not lactic acid. Such disturbances thereby slow MFCV. Also, decreased median frequency from fatiguing isometric contractions recovers faster than intramuscular pH (Vestergaard-Poulsen et al., 1995). Slowing conduction velocity may also help explain the similarity of PSA shift of cold and fatigue (Mills and Edwards, 1984).

Badier et al. (1993) demonstrated a progressive shift to lower frequencies in EMG PSA a few seconds after the muscle begins to contract isometrically. The leftward spectral shift occurs prior to actual muscle fatigue. This led to their conclusion that EMG PSA changes were likely due to changes in rate coding patterns of motoneurons during the development of fatigue (Badier et al., 1993; Bigland-Ritchie et al., 1981). This shift is suggested to correspond to reflex-induced change in central motor control. Group III and IV afferents (i.e. chemically sensitive

metabaloreceptors) exert a powerful inhibition of the spinal motoneuron (Badier et al., 1993). They are activated when muscle pH falls and extracellular potassium concentrations increase with the development of fatigue. Thus the decline of firing frequency may result from the subsequent stimulation of group III and IV afferents. Therefore, the analysis of power spectrum mean frequency seems to be more useful to recognize recruitment patterns of motor units rather than muscle failure itself, however both are closely related.

2.5 Evoked Contractile Properties

2.5.1 Twitch Tension

2.5.1.1 Hypothermic

Generally, the thermal dependence of electrically stimulated skeletal muscle force generation is low (Bell and Lehmann, 1987; Bennett, 1985; Bergh and Ekblom, 1979; Faulkner et al., 1990; Kossler and Kutchler, 1987; Kossler et al., 1987; Ranatunga et al., 1987; Ranatunga and Wylie, 1983; Ranatunga and Wylie, 1989). Human peripheral muscle may experience a large thermal range on a daily basis, reaching as high as 41° C after exhaustive exercise or as low as 23° C at rest depending on activity level and environmental temperature (Segal et al., 1986) with the exception of fast twitch fibers. Consequently it may be expected that muscle would remain unchanged with cooling (i.e. a Q_{10} of approximately 1.0) or increase with cooling low (i.e. a Q_{10} of below 1.0).

The thermal dependence of peak twitch force is both temperature and fiber type specific Bennett, 1984). The twitch tension in a fast twitch muscle type (FT) generally increases down to a muscle temperature of 22 to 24°C (Kossler and Kutchler, 1987; Ranatunga and Wylie, 1989)

whereas twitch tension in a slow twitch muscle type (ST) either decreases or remains constant to this temperature (Bennett, 1984; Kossler and Kutchler, 1987; Ranatunga and Wylie, 1989). It has been demonstrated that a FT muscle warmed from 20°C up to 37°C decreased peak twitch (R_{10} = 0.7) but ST muscle remains constant (R_{10}= 1.0) (Close, 1972; Ranatunga et al., 1987). Primarily fast twitch fibers exhibited a distinct maximum between 22 and 24°C followed by a 50% decrease between 22 and 34°C (Kossler and Kutchler, 1987; Davies et al., 1982). In the first dorsal interosseus muscle in the human hand however, a muscle which is 60% ST fibers, maximal twitch tension decreased by 50% in cooling from 35°C to 12°C with tension decrease in these fibers was significantly more pronounced below 25°C (Ranatunga et al., 1987). In other human studies, twitch output of predominantly slow twitch soleus (SOL) muscle fibers increased steadily with warming up to 36°C (Kossler and Kutchler, 1987).

Varying twitch tensions in the fiber types can be explained by the difference in the development of the SR. The sarcoplasmic reticulum (SR) of FT fibers is more extensive than ST fibers since FT fibers are estimated to have 2–4 times higher density of Ca^{+2} release channels (Damiani and Margreth, 1994) and Ca^{2+} ATPase was found to be 10 times greater for the EDL than the SOL (Kossler and Kutchler, 1987). In a warm state, the higher rate of calcium re-uptake of the FT fiber has the calcium sequestering process started before full tension can be produced. The FT muscle therefore produces lower tension in a warm state since, in this context, the calcium is sequestered too rapidly for maximum tension to develop. In a cool FT fiber or in a warm ST fiber, higher twitch tension is achieved because the calcium sequestering process is slower. Accordingly, the rate of decay of the active state is faster in FT muscle fibers (Kossler and Kutchler, 1987). It is

this difference between the two fiber types that suggest that cooling slows calcium sequestering, since cooling allows the longer utilization of the active state.

Furthermore, calcium sensitivity of myofibrillar (thin filament) activation has been shown to increase with cooling (Ranatunga and Wylie, 1989; Stephenson and Williams, 1981; Stephenson and Williams, 1985). This calcium sensitivity potentiates tension between 20-37°C. Below approximately 20°C temperature in the fast twitch, and below 37°C in the slow twitch, the cooling depression of tension production may be dominating over the tension potentiating effect of increased calcium sensitivity.

2.5.1.2 Fatigue

There is some variation of the response of twitch tension depending upon fiber type and method of fatiguing. For example, Petrofsky, et al. (1980) found that after isometric fatigue protocols of 40 or 70% of maximum stimulated force, twitch tension was reduced in both the medial gastrocnemius and the soleus in intact cat fibers. Behm and St.Pierre (1997) investigated the difference of evoked contractile property differences of quadriceps and plantar flexors in long term (25 or 50% MVC respectively) and short-term fatigue (50 or 75% respectively) in intact human fibers with mean endurance times of approximately 20 minutes and approximately 4 minutes respectively. Behm and St. Pierre (1997) found that while quadriceps twitch torque did not decrease, the plantar flexors were significantly increased by 16.1% while fatigued. Differences likely exist between the findings of Behm and St. Pierre (1997) and Petrofsky et al. (1980) because different muscles were used, namely medial gastrocneminus of the cat and the quadriceps femoris of humans, at different intensities of 25 or 40% and 50 and 70% of maximal

force. Petrofsky et al. (1980) also used maximal stimulation while Behm and St. Pierre (1997) used voluntary contraction. In vivo human muscles are known to be heterogeneous, often being predominantly, but not exclusively, FT or ST (Behm and St. Pierre, 1997).

Pääsuke et al. (1997) demonstrated that while there is potentiation of twitch by 155.1% immediately after a 5 second MVC in plantar flexors immediately after fatigue, tension declines during recovery from sustained sub-maximal contractions. The demonstrated decrease in force and twitch torque indicate both peripheral and central fatigue, similar to the results of Behm and St. Pierre (1997). Twitch tension recovered in 10- minutes in the medial gastrocnemius but required only 3-minute recovery in the soleus (Petrofsky et al., 1980). The variety of results is summarized in Table 1 while Tables 2 summarizes differences of methodology between Behm and St.Pierre (1997), Petrofsky et al. (1980) and Pääsuke et al. (1997). These tables summarize according to the dominance of fiber types in the specific muscle; no human muscle is exclusively one fiber type.

Depression of twitch torque has been attributed to a depression of SR release of calcium (Lopes et al., 1983) or by impairment of E-C coupling (Bigland-Ritchie et al., 1986a). Furthermore, Edwards et al. (1977) demonstrated that declining twitch amplitude could be overcome with high frequency stimulation. The impairment of these mechanisms may be induced by accumulation of metabolic by-products in the more anaerobic FT fibers. Greater twitch potentiation in ST fibers has been ascribed to fewer impairing by-products being produced by ST fibers and an increasing accumulation of calcium in the cytoplasm in addition to increased sensitivity of calcium binding sites to calcium (Lindinger et al., 1995).

Table 1: Comparing results of Behm and St. Pierre (1997) and Petrofsky et al. (1980)

	LD ST	LD FT	SD ST	SD FT
Inactivation	Increase [1]	Increase [1]	Increase [1]	Increase [1]
Force of peak twitch	Increase [1] decrease [2]	Decrease [1,2]	Increase [1] decrease [2]	Decrease [1,2]

Table 2: Comparing Intensities and Fiber Types Used By Behm and St. Pierre (1997) and Petrofsky et al. (1980)

	FT	ST
LD	25% of MVC quad [1] 40% of maximal stimulation medial gastrocnemius [2]	50% MVC plantar flexors [1] 40% of maximal stimulation soleus [2]
SD	50% of MVC quad [1] 70% medial gastrocnemius [2]	75% of MVC plantar flexors [1] 70% of maximal stimulation soleus [2] 60% plantar flexors [3]

Tables 1 and 2 compare studies of Behm and St. Pierre (1997), Petrofsky et al. (1980), and Pääsuke et al. (1997). SD – short duration (approximately 4 minutes), LD – long duration (approximately 20 minutes), ST – slow twitch, FT – fast twitch: 1- Behm and St. Pierre (1997) 2- Petrofsky et al. (1980) 3- Pääsuke et al. (1997)

2.5.2 Tetanic Tension

2.5.2.1 Hypothermic

There is a sharp difference between hypothermic twitch tension and hypothermic tetanic tension. The different muscle fiber types have almost identical increases in maximal tetanic tension (Bennett, 1984) with regards to thermal dependence. Maximum tetanic tension will steadily decline with cooling from 30°C to 6°C (Kossler and Kutchler, 1987) dropping only 10-20% in cooling to 25°C (Buller et al., 1984; Close and Hoh, 1968; Ranatunga, 1980; Ranatunga and Wylie, 1989) but 40-50% when cooled to 10°C (Bressler, 1981; Kossler and Kutchler, 1987; Ranatunga, 1982; Segal et al., 1986; Stephenson and Williams, 1985), so that it is clear that there is increased temperature sensitivity at lower temperatures. Kossler and Kutchler (1987) demonstrated an R_{10} of 2.3 (EDL) and 2.7 (SOL) for temperatures between 12 and 22°C. Segal et al. (1986) attributed this decrease to impairment of contractile protein binding.

Since active muscle stiffness, a measure of the number of attached cross bridges, is similar regardless of temperature (Kossler and Kutchler, 1987; Kutchler and Patzak, 1989), decreased force by lower temperatures may be the result of decreased force generation by each attached cross bridge (Bressler, 1981; Kossler and Kuchler, 1987; Kossler et al., 1987; Kutchler and Patzak, 1989; Stein et al., 1982), possibly by a lower rate of cross bridge cycling (Kossler and Kuchler, 1987, Kutchler and Patzak, 1989). As a result of the decline of these mechanisms with cooling, as muscle temperature drops below 25°C (Bennett, 1984) or 20°C (Ranatunga et al., 1987) there is a steep decline in maximal tetanic tension. Cold may alter any or all of the membrane potential, ionic pumps, the propagation of the action potential via the t-tubules, or gating properties of ionic channels, though the extent of alteration remains unknown (Kossler and Kuchler, 1987).

Maximal tetanic tension has been reported to increase, decrease, or remain constant at temperatures greater than 30°C depending upon stimulation rate (Segal et al., 1986). Therefore, care should be taken when comparing studies, especially in those using low stimulation frequencies. New stimulation rates should be re-determined at each temperature if attempting to use minimal stimulation rates. During tetanic stimulation, the calcium is not rapidly sequestered as it was during the twitch due to slowing of SR ATPase. Therefore, there is not as large a decrease in tension in the fast twitch fibers during tetanic stimulation in the higher temperature ranges as was seen with twitches.

There is an expected difference between peak twitch tension and peak tetanic tension since voluntary muscle contraction is tetanized in all physiological conditions (Bennett, 1984). In an organism, muscle fibers do not regularly experience twitches, so muscles would not evolve a functional response to twitch, but would evolve to tetanic stimulation (Fig. 2-1). For this reason tetanic stimulation has more consistent results with voluntary contractions.

2.5.2.2 Fatigue

The response of tetanic tension development while the muscle is fatigued is similar to the response to cold. Isometric fatigue at either 40 or 70% of maximal stimulated force led to a decline of tetanic tension for both soleus and the medial gastrocnemius in cats (Petrofsky et al., 1980). Tetanic tensions recovered in 10- minutes for gastrocnemius and in 3- minutes in soleus (Petrofsky et al., 1980). A decline of tetanic force is expected since this represents impairment of the myofilaments associated with impaired cross bridge cycling. As previously discussed with

MVC. a build-up of inorganic phosphates and production of hydrogen ions directly inhibits cross-bridging by impairing the bonding on calcium with troponin C.

2.5.3 Muscle Fiber Excitation

2.5.3.1 Hypothermic

As previously mentioned, rate processes deal with the time dependent features. They are useful in studying the speed at which a particular process is accomplished and may be used to explain such characteristics as power development or speed of a response. The thermal dependence of the skeletal muscle rate processes is very strong (Bennett, 1984; Close and Hoh, 1968; Cornwall, 1994; Davies et al., 1982; Davies and Young, 1983; Ranatunga, 1982; Ranatunga, 1984; Ranatunga et al., 1987; Ranatunga and Wylie, 1982; Ranatunga and Wylie, 1983; Rome, 1990). As temperature increases, the speed of rate processes continue to increase up to a point that may damage the muscle structure (Bennett, 1984). Time to peak twitch (TPT), time to half relaxation of twitch (½ RT_{hw}) and shortening velocity, all rate processes, have average Q_{10} scores of 2-2.5.

Dynamic performance involving factors that are rate process dependent, such as maximal power output, improve as temperature increases (Bennett, 1984; Bennett, 1985; Binkhorst et al., 1977) thus reflecting the positive relationship of increasing temperature on rate processes. Within a muscle temperature range of 25-35° C (Faulkner et al., 1990; Ranatunga et al., 1986) or 22-38°C (Binkhorst et al., 1977) time to peak twitch increases with decreasing temperature. Therefore, there is a clear relationship between thermal dependence (Q_{10} = 1.6-3) of rate processes and temperature over physiological temperature ranges (Rome, 1990), though exactly what that temperature range is not clear. Q_{10} scores for rate processes as a whole (time-to-peak twitch, time to half relaxation of

twitch, and rate of rise of tetanic tension) determined for temperatures below 20°C were considerably higher than those obtained for temperatures above 25°C, being 2.75 and 1.48 respectively (Elmubarak and Ranatunga, 1984).

Time to peak twitch (TPT) specifically increases as temperature decreases (Bennett, 1984; Cornwall, 1994; Davies et al., 1982; Davies and Young, 1983) with a TPT Q_{10}= 2.2 - 2.4 (Kossler and Kuchler, 1987; Kossler et al., 1987; Rome, 1990). Between 25-35°C the rate of force development is also impaired, being slowed by 22% (Faulkner et al., 1990). This slowing of muscle contraction results in a shift of the force-velocity curve to the left (Holewijn and Heus, 1992) resulting in lower forces at low velocities.

There is some disagreement on the thermal dependence of TPT though the variance is slight. TPT has been shown to have a similar thermal dependence in both muscle types (Q_{10}= 2 in fast and 2.2 in slow) (Bennett, 1984; Ranatunga, 1984). There has also been shown a greater thermal dependence in FT (Q_{10} of 2.1-2.2) than in ST (1.4-1.6) for shortening velocity (Bennett, 1984). It has also been shown that maximum shortening velocity of the rat SOL was more sensitive (Q_{10}=2 and 3.5 at 25°C and 20°C respectively) than the EDL (Q_{10} =1.8 and 2.4 at 25°C and 20°C respectively) (Ranatunga, 1984). It should be noted that while variation exists between studies, variance is not great and likely results from different methodologies or individual variations among subjects.

In both muscle fiber types of the rat, another characteristic effect of temperature is the prolongation of ½ RT_{tw} in response to cooling (Q_{10}= 2.7) (Kossler and Kuchler, 1987; Kossler et al., 1987). There is a pronounced thermal dependence of ½ RT_{tw}, particularly below 20°C. Q_{10} values of 2.5-2.8 for ½ RT_{tw} in rat EDL and SOL (Rome, 1990; Segal et al., 1986) while a Q_{10} of

2.7 has been shown in frog muscle (Kossler et al., 1987). In cooler muscles, the prolongation of relaxation may be caused by slower detachment of the cross-bridges (Faulkner et al., 1990) or slowed calcium sequestering.

The thermal dependence of muscle fiber excitation different muscle types may also be explained by a change in the net attachment rate of cross bridges due to cooling (Elmubarak and Ranatunga, 1984), though these exact changes remain unknown (Ranatunga, 1984). These changes are unlikely to be caused by a change in muscle stiffness since viscous resistance to sliding movement of filaments is negligible (Ranatunga, 1984). Changes may be a consequence of temperature-dependent decrease of the maximal rate of adenosine 5'-triphosphate (ATP) hydrolysis (Bergh and Ekblom, 1979), or impaired neuromuscular transmission (Edwards et al., 1972). The direct result of increasing temperature is accelerated metabolic rate (Bergh and Ekblom, 1979; Faulkner et al., 1990), and accelerated kinetics of the muscle fiber action potentials (Segal and Faulkner, 1985; Ward and Thesleff, 1974). The impairment of maximal velocity of shortening during cold might also be a function of the actomyosin ATPase activity (Faulkner et al., 1990). Rome and Bennett (1990) described that temperature's effect on maximal performance and maximal sustainable performance are from cold's effect on shortening velocity. Decreases in contractile kinetics can be explained by decreased cycling frequency. In addition, it has been shown that cooling the muscle may interfere with neuromuscular transmission of the superficial muscle fibers and conduction velocity within the muscles (Bigland-Ritchie et al. 1992, Franssen and Wieneke, 1994) and hence maximum speed thereby setting limits on power and speed (Ranatunga, 1977). The limited existing body of evidence indicates that the explanation may lie in any of these hypotheses though future research in this area is needed.

2.5.3.2 Fatigue

Evoked temporal characteristics vary by muscle group, time to fatigue, and intensity of contractions. As demonstrated by Behm and St. Pierre (1997), quadriceps TPT was prolonged 15.3% while PF TPT was not significantly changed. This study also indicated that subjects in a short duration, more intense (quadriceps at 50% MVC and PF at 75% MVC) fatigue protocol experienced a greater prolongation of TPT. The authors speculated that the decline of the quadriceps twitch torque and prolongation of the TPT might imply impairment of E-C coupling, likely the Ca^{+2} release from the SR and/or cross-bridge cycling (Behm and St. Pierre, 1997). E-C coupling involves the activation of the surface membrane and the propagation of the signal down the t-tubules. In addition to depression of twitch torque, diminished rates of force development and diminished rates of relaxation have been attributed to a depression of SR ATPase Ca^{+2} pump as demonstrated with caffeine studies (Lopes et al., 1983). Time to peak twitch has been demonstrated to either increase or decrease depending on the work intensity. McKenzie and Gandevia (1991) found that higher intensities (20-50% MVC) seem to prolong TPT but low intensities (5-10% MVC) shorten TPT in elbow flexors. They surmised that there must be a crucial aerobic rest interval. Discrepancy between this study and Behm and St. Pierre (1997) may be due to different fatigue protocols or because Behm and St. Pierre (1997) used greater than 50% MVC for all protocols except the long duration fatigue of the quadriceps.

In the long duration fatigue protocol, Behm and St. Pierre (1997) found 16.8% faster twitch half-relaxation times ($½ RT_{tw}$) but the short duration fatigue protocol experienced a 9.7% slowing, and also found a difference in the $½ RT_{tw}$ and the TPT depending on the length of the protocol. It may be expected that if one of the temporal characteristics is impaired so should the

others, though impaired calcium release, a passive process, does not necessarily mean there will be impaired calcium reuptake, an active process (Behm and St. Pierre, 1997).

Petrofsky et al. (1980) demonstrated that contraction velocity in cats of both fiber types slowed with fatigue though the greatest decrements of the unloaded muscle occurred during longer fatigue at 40% of maximum stimulated force rather than shorter fatigue protocols at 70% of maximum stimulated force. Orizio et al. (1999) found that TPT of the human tibialis anterior recovered within 1 minute post-fatigue while ½ RT_{tw} was still significantly slower at 6 minutes post-fatigue when stimulated at 35 Hz. Contraction time (CT) and ½ RT_{tw} also indicated that recovery of the diaphragm had not occurred after 30 minutes of rest. CT was still 10% slower and ½ RT_{tw} was 30% slower (Metzger and Fitts, 1987) after stimulated fatigue of the rat diaphragm at high (75 Hz) and low (5 Hz) frequencies. While the time course may not agree, both studies agree that rate of contraction recovers faster than relaxation time. CT has also been implicated to get faster, as with Pääsuke et al. (1997). Twitch was delivered immediately after a 5 second MVC. CT was faster by 21.9% while ½ RT_{tw} was not altered. Holding 60% of MVC of the elbow flexors to exhaustion 3 times with 2 minutes rest between trials was then used to induce fatigue. Post-fatigue, CT got faster though ½ RT_{tw} was slower post-fatigue, recovering 5 minutes after fatigue (Pääsuke et al., 1997). Petrofsky et al. (1980) demonstrated that isometric fatigue at either 40 or 70% of maximum stimulated force in rats led to declines in contraction velocity while twitch duration increased for both soleus (ST) and the medial gastrocnemius (FT). Contraction velocity recovered in less than 1 minute (Petrofsky et al., 1980).

Increases in the ½ RT_{tw} are typically accounted for by decreases in calcium re-uptake (Gollnick et al., 1991). Note that the active sequestering process is not necessarily related to the

passive calcium release process. Therefore energy availability and use may play a part in slowing sequestering but not release. Again, the development levels of the fiber's SR can account for the difference between FT and ST. Alterations of CT may be related to similar processes as increasing or decreasing availability of residual calcium in the sarcoplasm.

Table 3: Effects of Fatigue on Temporal Characteristics

	LD ST	LD FT	SD ST	SD FT	Very LD	Recovery (TA)
½ RT_{tw}	Speed-up [1] Slowed [2]	Speed-up [1] Slowed [2]	Slowed [1,2]	Slowed [1,2]	Slowed [3]	> 6 min [3]
TPT	No change [1] slowed [2]	Slowed [1,2]	no change [1] slowed [2]	Slowed [1,2]	Speed up [3]	< 1 min [3]

Table 3 compares studies of Behm and St. Pierre (1997), Petrofsky et al. (1980), and Pääsuke et al. (1997). SD – short duration, LD – long duration, ST – slow twitch, FT – fast twitch, TA – tibialis anterior; 1- Behm (1997) 2- Petrofsky et al. (1980) 3- Pääsuke et al. (1997)

2.5.4 M-wave

2.5.4.1 Hypothermic

The muscle compound action potential (M-wave) represents the propagation of the action potential along the sarcolemma. It is usually measured through the skin during a twitch and is collected in the same fashion as EMG. The limited research in this area has demonstrated that cooling alters M-waves. In an experiment by Bell and Lehmann (1987) on the triceps surae, a muscle cooled to 12°C resulted in a decrease in the amplitude of the M-wave. It would be expected that the impairment of much of the neural functioning and propagation along the

sarcolemma would decrease the amplitude of the H-reflex (i.e. neural propagation) and M-wave (i.e. sarcolemmal) respectively. Slowing of the rate of ATP hydrolysis may lengthen the latency between stimulation and the beginning of both the M-wave and H-reflex.

In summary, progressively cooling muscle to a range of 22-24°C causes potentiation of twitch tension in FT fibers (Bell and Lehmann, 1987; Kossler et al., 1987) but causes either no change or depression of ST fibers (Ranatunga et al., 1987). Most research agrees that fatigue of FT fibers decreases twitch torque (Petrofsky, et al., 1980) though this is often difficult to demonstrate in intact muscle fibers. There is some disagreement regarding the effect of prolonged contractions of ST fibers on evoked contractile properties (see Tables 1.3. and 4). There seems to be agreement regarding the decline of tetanic tension with cold. Agreement is also undivided on the slowing effects of cold on ½ RT_{tw} and TPT.

2.5.4.2 Fatigue

The M-wave is much better studied in fatigued states rather than in cold. Behm and St. Pierre (1997) found a 14.7% depression of M-wave amplitude after long duration fatigue but a 15.7% potentiation after short duration fatigue. Similarly, it has been shown that using short term high intensity contractions did not alter M-wave amplitude (Bigland-Ritchie et al., 1979), while prolonged submaximal (Fuglevand et al., 1993) and repetitive MVC (Bellemare and Garzanti, 1988) have demonstrated decreases of M-waves. Petrofsky et al. (1980) found that after fatigue protocols of 40 or 70% of maximum stimulated force, duration of the action potential was extended in both fiber types though amplitude of the action potential was not significantly

altered. The duration and amplitude of the compound muscle action potential recovered in less than 1 minute (Petrofsky et al., 1980).

The ionic balance of the muscle membrane can affect E-C coupling by hindering the sum of depolarization, the rate of depolarization, conduction velocity, and the transmission down the t-tubule to the SR that ultimately affects the release of calcium. Although M-wave depression could be potentially detrimental, prolonging the surface action potential could increase the amount and length of time the myofilaments are exposed to calcium thereby providing more contractile force. Declining M-wave amplitude implicates failing membrane propagation or muscle membrane excitability. Therefore muscle membrane propagation failure is not the primary cause of fatigue, at least not in the short term.

Table 4: Effects of fatigue duration of different fiber types on M-wave amplitude found by Behm and St. Pierre (1997) and Petrofsky et al. (1980)

	LD ST	LD FT	SD ST	SD FT
M-wave amplitude	Decrease [1] No change [2]	Decrease [1] No change [2]	Increase [1] No change [2]	Increase [1] No change [2]
M-wave duration	Longer [2]	Longer [2]	Longer [2]	Longer [2]

Table 4 compares studies of Behm and St. Pierre (1997) and Petrofsky et al. (1980). SD – short duration, LD – long duration, ST – slow twitch, FT – fast twitch; 1- Behm (1997) 2- Petrofsky et al. (1980).

2.6 Conclusion

How cold and fatigue alters voluntary and evoked contractile properties of muscle tends to be dependent upon factors including fiber type, intensity, and duration of exposure to the stress. Thermal dependence of twitch torque is both temperature and fiber type specific. FT twitch torque tends to increase up to a muscle temperature of 22-24°C and declines in temperatures below this while ST twitch torque decreases or remains constant in this muscle temperature range and declines below this. While there is agreement that FT twitch torque decreases, disagreement exists regarding ST twitch torque though duration of fatigue and the muscle group is a possible explanation for this. Hypothermic tetanic tension steadily declines with cooling in either fiber type as is the case with fatigue as well. Cooling drastically slows rate processes such as maximal tetanic tension, TPT, ½ RT_{tw}, time to peak tetanic tension, and contraction velocity, with higher sensitivity as temperature declines though disagreement exists regarding which fiber type is more sensitive. Duration of the fatiguing protocol seems to be the determining factor regarding rate processes. While Petrofsky et al. (1980) indicates all rate processes slow at either duration in either fiber. Behm (1997) demonstrates a great deal of variety. As cold slows the rate processes the amplitude of the H-reflex and M-wave diminish and the waveform and time to onset extends, though there is very limited research in this area. Usually the M-wave becomes longer and either does not change in amplitude or is dependent upon duration of fatigue. Muscle temperature ranging from 25-32°C has been demonstrated to extend isometric endurance while fatigue undisputedly shortens it. Cold and fatigue are known to have similar effects of decreasing iEMG and lowering the firing frequency range.

Therefore it is relevant to study the interaction effects of 22°C local muscle temperature on the recovery of specific evoked and voluntary contractile properties in intact human plantar

flexors at one-, five-, and 10-minutes after high intensity fatigue since hypothermic recovery has not been studied. It is also relevant to validate previous studies and add to the body of knowledge about the main effects of recovery from fatigue with homeostatic temperature and the main effects of local hypothermia on unfatigued muscle on evoked and voluntary contractile properties in intact human plantar flexors. The following research will contribute the first known study to the area of hypothermic recovery by investigating the interaction effects of local muscle hypothermia and high intensity intermittent local fatigue.

METHODOLOGY

3.1 Subjects

Ten male volunteer subjects of a mean age of 23 years (+/- 6.1), with a mean weight of 82.8 kg (SD +/- 8.5 kg) and a mean height of 177.5 cm (SD +/- 6.3 cm), were recruited from the university community (Appendix A). They read and signed a consent form (Appendix B) approved by the Human Ethics Committee prior to experimentation. Subjects were informed that they could withdraw from the experiment at any time without prejudice.

3.2 Instrumentation

All voluntary and evoked torque signals were detected by a strain gauge, sent through a high gain amplifier (Biopac Systems Inc. DA100 and analog to digital converter MP100WSW), and monitored on computer (Sona Phoenix PC). The sampling rate was set at 1000 Hz and all data were stored on computer. Data were recorded and analyzed with a commercially designed software program (AcqKnowledge III, Biopac Systems Inc.). EMG activity was amplified (Biopac Systems Inc. EMG 100 and analog to digital converter MP100WSW), filtered (10-1000 Hz), monitored and stored on computer. The computer software program rectified and integrated the EMG signal. Measurements were monitored over a 500 ms period during a maximal voluntary contraction (MVC).

Fine abrasive (sand) paper and 70% isopropyl alcohol was used to clean the anesthetic injection site and EMG electrode placement sites. The temperature probe site was anesthetized

with 2% injectable xylocaine (Astra Pharma Inc.) that was injected from a 1 ml syringe through a 26G 3/8" intradermal bevel needle to a depth of approximately 2 cm.

The refrigerating pump circulated liquid glycol through 17 meters of plastic (Tygon) tubing of 3/8" diameter with a 1/16" wall (R-3603).

3.3 Overview of Testing

In this study, subjects performed isometric maximum voluntary contractions (MVC) of the plantar flexors. Subjects were tested on the rate of recovery of a variety of voluntary and evoked contractile properties of the plantar flexors while either normothermic or hypothermic (see appendix C). Subjects served as their own controls and it was randomly determined if hypothermic or normothermic session would be tested first. During plantar flexion, subjects were seated with hips and knees at 90°, with their foot in a modified boot apparatus outfitted with a custom designed strain gauge.

Subjects performed each testing session, separated by at least three days, at a similar time of day since Martin et al. (1999) indicate that force produced during a maximal voluntary contraction was 8.9% higher in the evening than in the morning and CT and ½ RT_{rw} were also reduced during the evening. The time of day that the subject was tested was chosen on the basis of the subject's and experimenter's availability, but remained consistent for both testing sessions. While all subjects did participated in regular physical activity, including progressive resistance exercises, they had not performed any strenuous activity within 24 hours of the testing session.

3.4 Protocol Setup and Initial Testing

Surface EMG recording electrodes were placed over the belly of the tibialis anterior, belly of the lateral gastroneminus, and distal portion of the soleus. Ground electrodes were secured to bony landmarks on the patella and lateral malleolus. Thorough skin preparation for all electrode sites included shaving the area and removal of dead epithelial cells with an abrasive (sand) paper around the designated area followed by cleansing with an isopropyl alcohol swab.

Bipolar surface stimulating electrodes were secured over the peroneal nerve in the poplitial fossa behind the knee and the gastrocnemius-soleus intersection. Stimulating electrodes, 4-5 centimeters in length, were constructed in the laboratory from aluminum foil, and paper coated with conduction cream (Signa Creme) and immersed in water. The electrode length was sufficient to wrap the width of the posterior portion of the limb. The electrodes were placed in approximately the same position for each subject.

Prior to insertion of a myocardial temperature probe (thermister) into the lateral gastroneminus, the subject's leg was swabbed with 70% isopropyl alcohol and anesthetized with a xylocaine injection. While it was the temperature of the gastroneminus that was measured, a myocardial temperature probe was ideal in design for our purpose. The xylocaine was injected by a physician, who then inserted the myocardial temperature probe into the belly of the lateral gastroneminus to a depth of 2 cm. The myocardial temperature probe was then secured with tape. Teenier and associates (1991) demonstrated that EMG and voluntary force of the four muscles of mastication were not impaired by local lidocaine anesthetic. Nydahl and associates (1990) also demonstrated that intravenous local anesthetic (mepivacaine and etidocaine) did not affect isometric muscle force or EMG of the knee extenders. There was therefore no concern that the

small area of the gastroneminus anaesthetized would affect the results. The temperature probe was carefully place to avoid vascular and neurally dense areas.

Once the subject was prepared and secured into the modified boot apparatus, the voltage and amperage required to evoke maximal twitch amplitude was determined. Maximal twitch amplitude was determined by gradually increasing voltage and amplitude of the electrical stimulation until maximal twitch torque was obtained. Stimulation did not exceed 150 volts at 1 amp.

Once a maximal twitch was established, sub-maximal tetanic torque was measured. Sub-maximal tetanus was evoked with 100 volts at the amperage that was used for maximal twitch. A stimulating frequency of 100 Hz for 300 milliseconds was used. The short duration was chosen to reduce the extent of discomfort for the subject.

The interpolated twitch technique (ITT) was then administered during an MVC first and then 75, 50, and 25% voluntary contraction intensities that were performed in random order to an unfatigued muscle. A doublet (2 twitches delivered at a frequency of 100 Hz) rather than a single twitch was utilized for the interpolated evoked stimulation since it provides a higher signal to noise ratio. An interpolated twitch (IT) ratio would later be calculated comparing the amplitudes of the superimposed stimulation with the post-contraction stimulation to estimate the extent of inactivation during a voluntary contraction. Since the post-contraction stimulation represents full muscle activation, the superimposed torque using the same intensity of stimulation would activate those fibers left inactivated by the voluntary contraction. All maximal and submaximal (100%, 75%, 50%, 25% of MVC) forces were correlated with their respective IT ratios in order to generate a second order polynomial equation for all subjects. Second order polynomials using

both maximal and submaximal contractions (IT ratios) have been shown to be valid and reliable providing a more accurate estimation of muscle activation than a single IT ratio (Behm and St. Pierre, 1997). Once these unfatigued, normothermic (baseline) measures were taken the protocol branched. In the cold protocol the limb was cooled prior to fatigue and further testing. In the normothermic protocol the cooling protocol was omitted.

3.5 Cooling Protocol

To lower muscle temperature during the hypothermic testing session, active cooling methods were used involving a refrigerating pump circulating cold (-3°C) liquid glycol (anti-freeze) through Tygon tubing. The subject's lower leg was wrapped with the tubing in a coiling fashion to cover the entire leg. The baseline testing procedures were repeated as soon as possible after the subject's muscle temperature reached 22°C.

3.6 Fatigue Protocol

In both normothermic and hypothermic protocols, fatigue of the plantar flexors was induced within 5 minutes after baseline voluntary and evoked contractile properties were measured for that condition (i.e. normothermic baseline measures for the normothermic testing session, and normothermic baseline measures and unfatigued hypothermic measures for the hypothermic session). The fatigue protocol consisted of isometric contractions of the plantar flexors at 75% of the subject's MVC. Subjects took 3 seconds to rise to this level, held 75% of MVC for 14 seconds, and took 3 seconds to relax. This intermittent protocol repeated for as long

as the subject could maintain 75% of MVC. Verbal encouragement was provided throughout though no music was played. The recovery process was passive.

3.7 Post Fatigue Protocol

Many of the dependent variables could be assessed in the same test procedure. For example from a single twitch each of time to peak twitch, rate of twitch torque development, twitch torque, half-relaxation time of twitch, M-wave amplitude, and M-wave duration could be assessed. The testing protocol of peak twitch, sub-maximal tetanus, and ITT at 25, 50, 75 and 100% of MVC was repeated at 1, 5, and 10 minutes post fatigue.

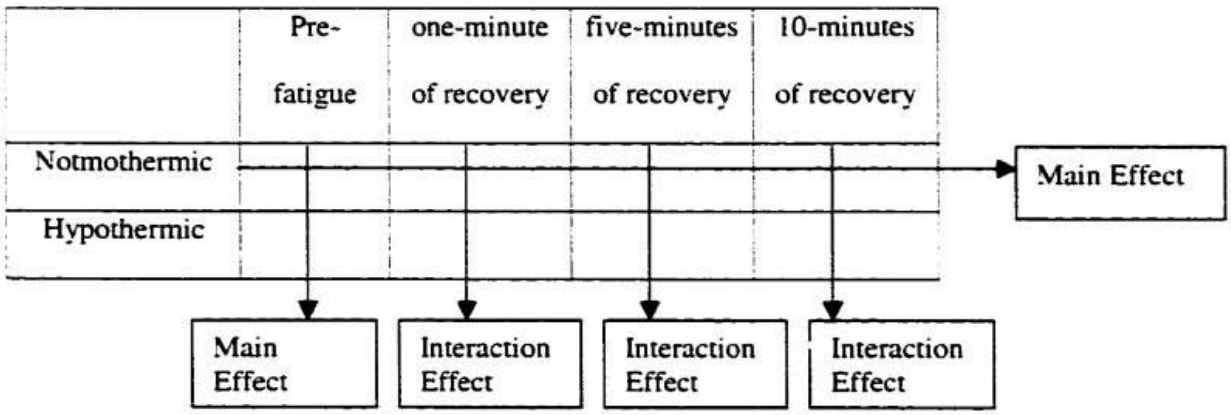
3.8 Analyses

Data were initially converted to percent change from the previous measurement (i.e. pre-fatigue, pre-fatigue to one-minute post-fatigue, one-minute post-fatigue to five-minutes post-fatigue, and five-minutes to 10 minutes post-fatigue). Percent changes were analyzed with a two-way ANOVA with repeated measures. The two factors (2x4) were temperature (normothermic and hypothermic levels) and testing (pre-fatigue, one-, five-, and 10-minutes of recovery levels). F ratios were considered significant at $p<0.05$. A Bonferroni – Dunn's post-hoc test was used to assess significant differences between variables. Results in the text include means +/- standard deviation.

Assessed on each dependant variable was the main effect of rate of recovery from fatigue on a normothermic muscle, the main effect of hypothermia on the unfatigued muscle, and then of most importance to this study was the interaction effect of hypothermia on the dependant

variable's rate of recovery. Each dependent variable was analyzed in an ANOVA format (Table 5).

Table 5: ANOVA format to analyze both the main effects of muscle temperature and the interaction of the muscle temperatures at different points of recovery



Therefore the goals of this study were two-fold. The primary goal of this study was to investigate the interaction effects of 22°C local muscle temperature on the recovery of specific evoked and voluntary contractile properties in intact human plantar flexors at one-, five-, and 10-minutes after high intensity fatigue. The secondary goal of this study was to validate previous studies and add to the body of knowledge about the main effects of recovery from fatigue with homeostatic temperature and the main effects of local hypothermia on unfatigued muscle on evoked and voluntary contractile properties in intact human plantar flexors.

3.9 Dependent Variables

A wide variety of specific dependent variables that represent a range of physiological implications were investigated (Appendix C).

3.10 Hypothesis

1. The rate of recovery of recovery of voluntary activation will be slowed by local muscular hypothermia.
2. The rate of recovery of voluntary torque will be slowed by local muscular hypothermia.
3. The rate of recovery of twitch torque will be slowed by local muscular hypothermia.
4. The rate of recovery of tetanic torque will be slowed by local muscular hypothermia.
5. The rate of recovery of the rate of voluntary torque development will be slowed by local muscular hypothermia.
6. The rate of recovery of the rate of twitch torque development will be slowed by local muscular hypothermia.
7. The rate of recovery of the rate of tetanic torque development will be slowed by local muscular hypothermia.
8. The rate of recovery of twitch half-relaxation time will be slowed by local muscular hypothermia.

9. The rate of recovery of tetanic half-relaxation time will be slowed by local muscular hypothermia.
10. The rate of recovery of the duration of the compound muscle action potential will be slowed by local muscular hypothermia.
11. The rate of recovery of amplitude of the compound muscle action potential will be slowed by local muscular hypothermia.
12. The rate of recovery of time to peak torque of twitch contraction will be slowed by local muscular hypothermia.
13. The rate of recovery of time to peak torque of tetanus contraction will be slowed by local muscular hypothermia.
14. The rate of recovery of time to peak torque of voluntary contraction will be slowed by local muscular hypothermia.

RESULTS

4.1 Muscle Temperature

In the normothermic muscle, mean temperature was 34.6 +/- 0.7°C. The muscle was cooled to 21.3 +/- 0.5°C. Temperature steadily but insignificantly increased during recovery, reaching 22.2 +/- 1.2°C, 23.8°C +/- 1.3°C, and 24.2 +/- 1.6°C after one-, five-, and 10- minutes of recovery respectively.

4.2 Hypothermia

With all hypothermic data pooled over recovery data, the 22°C muscle temperatures did not significantly alter voluntary properties such as MVC, muscle activation as measured by the ITT and iEMG activity of the GAST, SOL, TA, or any agonist to antagonist ratios. Although there was a significant decrease in tetanic torque (figure 3-1), there was no significant change in other evoked contractile properties (i.e. twitch torque, M-wave amplitude or duration) except those related to time that were slowed (i.e. MVC rate of torque development, TPT, rate of twitch torque development, ½ RT_w, time to peak tetanus, rate of tetanic torque development, and ½ RT_e) (Figures 3-2 to 3-8 respectively). Endurance time did not demonstrate a significant change with cooling. Significant changes were predominantly associated with temporal contractile properties.

4.21 Force

The only significant alteration to force with muscle cooling was a decrease in tetanic torque of 29.1% ($p<0.01$, $F= 40.34$) (Fig. 3-1). There was no significant change in endurance

time averaging 161 +/- 61.2 seconds when normothermic versus 153 +/- 61.7 seconds when hypothermic.

4.2.2 Temporal Characteristics

Hypothermic MVC rate of torque development (Fig. 3-2) was 25.1% lower than normothermic MVC rate of torque development ($p<0.05$, $F= 6.10$). Time-to-peak twitch (Fig. 3-3) increased 30.3% ($p<0.05$, $F= 6.50$). This is further demonstrated in the present study with the slowing of the rate of twitch torque development ($p=0.01$, $F= 7.94$) (Fig 3-4) by 33.4%. Also slowed was the ½ RT_{tw} ($p<0.01$, $F= 32.66$) (Fig. 3-5) by 34.1%. The time to peak tetanus (Fig. 3-6) showed a significant increase of 6.7% ($p<0.05$, $F= 5.56$), while rate of tetanic torque development (Fig. 3-7) decreased by 33.4% ($p<0.01$, $F= 12.97$). The other temporal characteristic altered by cold was the ½ RT_{tc} (Fig. 3-8), slowing 38.1% ($p<0.01$, $F= 18.03$).

4.3 Recovery

Assessing for the main effect of fatigue, varying recovery times did not alter either M-wave duration or amplitude, or the time to peak twitch. Fatigue caused significant changes, at each recovery interval after fatigue (i.e. one-, five-, and 10-minutes), in torque generation, muscle activation, and temporal characteristics. MVC, MVC rate of force development (RFD), voluntary inactivation, twitch torque, tetanic torque, and all temporal characteristics of both twitch and tetanus, except TPT_{tw}, were all significantly altered by fatigue. All iEMG signals were also significantly altered during recovery, except the SOL:TA ratio.

4.3.1 Force Output

MVC torque of the plantar flexors (Fig. 3-9) significantly declined 13.5% between pre-fatigue and one- minute recovery ($p= 0.01$, $F= 49.92$), and continued to be significantly lower at five- ($p= 0.01$, $F= 35.43$) and 10- minutes ($p= 0.01$, $F= 26.04$) recovery, recovering to 11.3% and 9.7% respectively. There was a 22.4% potentiation of twitch torque (Fig. 3-10) between pre-fatigue and one-minute ($p= 0.01$, $F= 56.81$) post fatigue, which was still significantly higher (12.7%) at five- minutes ($p<0.01$, $F= 14.56$), but had recovered by 10- minutes of recovery. A decrease also existed between one-minute recovery and five- ($p= 0.01$, $F= 13.57$) and 10-minutes ($p= 0.01$, $F= 35.35$) of recovery compared to one minute.

There was an increase in the torque generated by the recovering plantar flexors during tetanic stimulation (Fig. 3-11). Torque increase from tetanus was not significant until five-minutes of recovery when there was an increase of 28.1% ($p<0.01$, $F= 81.60$) and by 10- minutes had reached an increase of 24.8% ($p<0.01$, $F=58.19$). Five- and 10- minute of recovery ($p= 0.01$, $F= 50.95$ and 32.87 respectively) were also greater than one-minute of recovery.

4.3.2 Muscle Activation

Voluntary inactivation increased with recovery (Fig. 3-12). While there was an increase in inactivation at one- minute of recovery, the increase was not significant. The only significant increase of inactivation was by five- minutes of recovery, by which time inactivation had risen significantly ($p<0.05$, $F= 8.99$) from 1.2% to 5.7%.

An insignificant decrease of the antagonist, TA EMG (Fig. 3-13) at one-minute recovery, had a significant ($p<0.05$, $F=10.39$) 22% increase between one- and 10- minutes recovery. While

EMG of the TA experienced little change, GAST EMG (Fig 3-14) experienced significant 32.5% decreases between pre-fatigue and one- minute ($p<0.01$, $F= 27.58$), 25.9% by five-minutes ($p<0.01$, $F= 17.59$), and 16.9% by 10- minutes ($p<0.05$, $F= 7.50$). Changes of SOL EMG (Fig. 3-15) experienced an initial significant decline of 18.6% between pre-fatigue and one- minute ($p<0.01$, $F=13.52$) and then began to increase. The increases after one- minute increased significantly by 18.8% from one- to five- minutes ($p<0.01$, $F= 13.62$) and 24.1% from one- to 10- minutes ($p<0.01$, $F= 25.89$), though the five- and 10- minute intervals did not vary significantly from pre- fatigue values.

The EMG ratio of the GAST and TA had a significant 19.8% decline by one- minute of recovery (Fig 3-16) ($p<0.01$, $F= 12.35$). This EMG ratio continued to decline, reaching 20.9% by five- minutes ($p<0.01$, $F= 13.68$) and 22.1% by 10- minutes ($p<0.01$, $F= 15.22$) of recovery.

4.3.3 Temporal Characteristics

Rate of voluntary torque development (Fig. 3-17) declined 29.3% between pre-fatigue and one-minute of recovery ($p<0.05$, $F= 9.60$). Rate of twitch torque development (Fig. 3-18) was increased at one- minute ($p<0.01$, $F= 58.59$), five- minutes ($p<0.01$, $F= 20.94$), and 10-minutes ($p<0.05$, $F= 10.31$) of recovery by 36%, 25.2%, and 18.9% respectively. One- minute of recovery was significantly higher than five- and 10-minutes ($p<0.05$, $F= 9.28$; $p<0.01$, $F= 19.58$ respectively) after fatigue. Significant alterations also occurred with several temporal characteristics of tetanus. The time to peak tetanus (Fig. 3-19) had a slight but significant 6.1% decrease by both one- and five- minutes ($p<0.01$, $F= 15.84$; $p<0.01$, $F= 15.74$ respectively) and 5.4% by 10- minutes of recovery ($p<0.01$, $F= 12.26$). The rate of the tetanic torque development

(Fig. 3-20) significantly increased with recovery, increasing 14% at one- minute ($p<0.01$, $F= 10.18$) and by five- minutes had increased 31.5% ($p<0.01$, $F= 80.64$) and was still 28% faster by 10- minutes ($p<0.01$, $F= 57.24$). Five- and 10- minutes of recovery ($p<0.01$, $F= 33.52$; $p<0.01$, $F= 19.15$ respectively) were also significantly greater than one- minute of recovery.

½ RT_{tw} (Figure 3-21) became faster as recovery continued thought did not reach significance until 10- minutes of recovery, at which time it was 25.5% faster ($p<0.01$, 13.82). The only significant ($p<0.05$, $F= 8.19$) change of ½ RT_{tc} (Fig. 3-22) was in comparing one- and 10- minutes of recovery that declined 28.6%.

4.4 Effects of Cold on Recovery

Cooling of the plantar flexors made no significant difference in the rate of recovery of MVC, rate of torque development, voluntary inactivation, EMG, or twitch or tetanic torque. Differences were found with ½ RT_{tw}, time to peak tetanus, and M-wave duration between the normothermic and hypothermic conditions.

4.4.1 Temporal Characteristics

The recovery of the ½ RT_{tw}, (Fig. 3-23) was 32.5% longer by 10- minutes of recovery ($p< 0.05$, $F= 12.78$) and time to peak tetanus (Fig. 3-24) was 12.3% longer ($p=0.01$, $F= 35.49$) at one- minute of recovery. The other difference found was in the first minute of recovery of the M-wave duration (Fig. 3-25) that was significantly 22.9% longer ($p<0.05$, $F= 11.01$).

DISCUSSION

5.1 General Comments

The main effects of each variable in the current study were not unexpected. The main effects of the rate of recovery from the high intensity fatigue protocol were generally in agreement with current literature (e.g. Behm and St. Pierre, 1997; Petrofsky et al., 1980). For example, voluntary force was decreased while evoked force was potentiated. Also, each indicator of voluntary activation decreased while rate processes were generally faster. The main effects of local hypothermia on muscle function were also consistent with the literature (e.g. Bennett, 1984; Bennett, 1985). For example, temporal characteristics of both evoked and voluntary contractile properties were impaired though force was generally not.

An effect of interaction occurs when a relation between at least two variables (i.e. rate of recovery from fatigue from 0- to one- minute of recovery, from one- to five- minutes of recovery and from five- to 10- minutes of recovery) is modified by at least one other variable (muscle temperature). Due to slowed rate processes it was expected that recovery from fatigue would be impaired due to hypothermia. This was not the case however. The primary goal of this study was to investigate hypothermic recovery of evoked and contractile properties of PF muscles. The recovery rate of most properties investigated, including both evoked and voluntary, were not impaired by the cold.

5.2 Hypothermic Effects on Recovery

5.2.1 Recovery of Many Voluntary and Evoked Contractile Properties Are Not Affected By Cold

Contrary to prior expectations, cold had minimal effects on the recovery of most voluntary and evoked contractile properties. This was unexpected since most other rate process are impaired by cooling. One possible explanation is related to blood flow following exercise. Blood flow serves to flush accumulated metabolites out of the muscle and into the vascular and lymphatic systems to be absorbed, filtered, or metabolized by other tissues and organs, thus assisting recovery of the muscle (Viires et al., 1983). It is known that there is an increase in blood flow immediately after occlusion, known as reactive hyperaemia (Pitcher and Miles, 1997). Occlusion would originate from both the intense muscle contraction and the hypothermia-induced vasoconstriction. Also occurring is functional hyperemia that also results in increased blood flow to exercised muscles immediately after exercise, especially those of higher intensity (Walloe and Wesche, 1988) and may be present beyond 20 minutes after intense exercise. Many metabolites, such as H^+ and lactate, are known to mediate functional hyperaemia while vascular occlusion stimulates reactive hyperaemia (Pitcher and Miles, 1997). Nitric oxide, a dose dependent vasodilator increasing 50 – 200% during periods of repetitive isometric contraction (Balon and Nadler, 1994), is also known to stimulate functional hyperaemia (Poucher, 1995). Increased blood flow could result if metabolites accumulated at a faster rate due to both contraction-induced and hypothermic vasoconstriction-induced occlusion. This increased blood flow, removing metabolites faster, may enhance recovery (Badier et al., 1994) enough to offset the impairing effects of cold. Pitcher and Miles (1997) demonstrated that recovery from fatigue was much faster in a muscle that was fatigued while receiving no circulation than in a muscle

that was fatigued while receiving intermittent circulation. Therefore, little change in the rate of recovery may have been seen between normothermic and hypothermic recovery since adequate blood flow was maintained throughout the recovery period. It should be noted however that blood flow during hypothermic exercise was neither directly or indirectly measured in this study and may be a direction for future research.

5.2.2 Recovery of Contractile Properties Affected by Cold

5.2.2.1 Time to Twitch Half-Relaxation Time

½ RT_{tw} (Fig. 3-23) did not increase until 10-minutes of hypothermic recovery ($p<0.05$). Since normothermic fatigue also caused a decrease of the ½ RT_{tw} (Fig. 3-21) only at 10- minutes it seems likely that the mechanisms underlying this response were similar. As previously discussed, it is possible that the reactive and functional hyperemia triggered an influx of blood to the muscle capillaries. The increase of fluids would contribute to increased muscle stiffness (Ebersole et al., 1999) by increasing intracellular fluid pressure. Increasing stiffness would lead to reduced ½ RT_{tw}. An increase in the intra-cellular fluid pressure would result in a greater osmotic pressure, forcing plasma fluid from the blood into intracellular and extracellular spaces. A delayed of this increase in the intra-cellular fluid pressure (>5- minutes) might be expected since time would be needed to build up intracapillary osmotic pressure and subsequent diffusion of fluid to the sarcoplasm of the muscle fibers.

5.2.2.2 Time to Peak Tetanus

TPT_e was longer ($p<0.01$) (Fig. 3-24) at one- minute of hypothermic recovery. Westerblad et al. (1997) demonstrated that while shortening velocity of tetanized muscle was not altered at 30°C with a decline of 0.5 pH units, it declined 20% at 12°C thus demonstrating the temperature dependence of pH on rate of torque generation. A similar effect has been observed with inorganic phosphates (Pi) at lower temperatures (Dantzig et al., 1992). Dantzig et al. (1992) explained this decline in rate of torque generation by a heightened effect of Pi while Westerblad et al. (1997) explained a similar effect of H^+ in lower temperatures. While a similar phenomenon occurred in the current experiment, it is not clear why TPT_e was the only torque or contraction velocity characteristic affected by this relationship.

5.2.2.3 M-Wave Duration

In the present study, M-wave was prolonged only at one- minute of hypothermic recovery ($p<0.05$). This is likely related to impairment used to power the sarcolemmal cation pumps. While Green (1998) explained that Na^+-K^+ ATPase activity slowed in relation to fatigue, it has also been shown that the rate of ATP hydrolysis is impaired with cold (Bergh and Ekblom, 1979). This could lead to a slowing of the compound muscle action potential as demonstrated in the M-wave.

5.3 Hypothermic Effects Pre-Fatigue

5.3.1 Force

The decline of tetanic torque of skeletal muscle with hypothermia is strongly supported in current literature and therefore was expected though the extent of the decline was unusual. Most literature reported that tetanic force drops only 10-20% in cooling of muscle to 25°C (Buller et al., 1984; Close and Hoh, 1968; Ranatunga, 1980; Ranatunga and Wylie, 1989) though the present study found a decline of 29.1% of tetanic force at 22°C (Fig. 3-1).

It is speculated that this decline is related to a lower rate of cross bridge cycling (Kossler and Kuchler, 1987, Kutchler and Patzak, 1989). A change in the force-velocity relationship at the level of the muscle could be due to a slowing of cross bridge cycling (de Hann et al., 1989) leading to slower generation of force over a specific stimulation period. The decline of tetanic torque of the PF muscles in this study is likely the result of a decline in rate of force development. Since, in no subject did tetanic torque reach a plateau (i.e. maximum force) over the 300 ms stimulation period, the slope of the force-time interval was still increasing. A lower rate of cross-bridge cycling would adversely affect rate of force development resulting in an even lower position on the force-time integral slope. This decrease of cross-bridge cycling is evidenced by the decrease in the rate of tetanic torque development and increase of the time to peak tetanus. Therefore, the decrements in tetanic torque with hypothermia in this experiment may be related more to a decrease in rate of force development than the ability to develop maximal tetanic torque.

5.3.2 Temporal Characteristics

Many of the temporal characteristics in 22°C PF muscles including decreases in the rate of voluntary torque development (RVTD) (Fig 3-2), rate of twitch torque development ($RTDT_w$) (Fig. 3-4), rate of tetanic torque development ($RTDT_c$) (Fig. 3-7), and increases in time to peak twitch (TPT_w) (Fig. 3-3) and time to peak tetanus (TPT_c) (Fig. 3-6) each can be explained by two factors. Changes may be a consequence of temperature-dependent decrease of the maximal rate of ATP hydrolysis (Bergh and Ekblom, 1979) due to impaired actomyosin ATPase activity (Faulkner et al., 1990) causing a decreased rate of cross bridge cycling. Decreases of RFD could also be due to impaired neuromuscular transmission (Edwards et al., 1972) as seen in impaired conduction velocity within hypothermic muscles (Bigland-Ritchie et al. 1992, Franssen and Wieneke, 1994). Cold is known to impair both of these processes (Bergh and Ekblom, 1979; Franssen and Wieneke, 1994).

While RFD is directly affected by cross bridge attachment, Ca^{+2} release, ATP hydrolysis, and neural factors such as rate coding can also affect rate of force development and can be measured with iEMG (Miller et al., 1981). However neural factors did not seem to be a factor in the present study since there was no alteration of iEMG activity.

The other temporal characteristics affected were increases of ½ RT_{tc} and ½ RT_{tw}. Impairment of relaxation properties may be explained by a hypothermia-induced decrease of calcium re-uptake (Kossler and Kutchler, 1987), possibly from decreased ATP hydrolysis leading to an impairment of the mechanisms responsible for the re-uptake of Ca^{+2} from the sarcoplasm into the SR (i.e. SR Ca^{+2} ATPase).

Another possibility for increased ½ RT_{tw} and ½ RT_{te} could be an increase of viscosity. As hypothesized by Lakie et al. (1986). Price and Lehmann (1990) demonstrated that viscous stiffness increases as a direct relation to cold in the forearm and ankle respectively. Furthermore. Edwards et al. (1972) demonstrated that bonding of actin and myosin becomes greater at low temperatures thereby causing muscle stiffness. By increasing viscosity and parallel elastic components ½ RT_{tw} and ½ RT_{te} could increase.

5.4 Overall Fatigue Effects

5.4.1 Force

MVC remained significantly lower throughout the 10- minutes of recovery from fatigue ($p<0.01$). Voluntary force may be affected either positively or negatively by either or both central and peripheral factors. Myosin ATPase activity has been demonstrated to reduce with the accumulation of metabolites in addition to reducing the energy released from ATP hydrolysis (Cook and Pate. 1990). Peripherally. the accumulation of hydrogen ions. inorganic phosphates (Pi). and lactate can all impair force generation by slowing cross bridge cycling (Cook and Pate. 1990: de Hann et al.. 1989: Westerblad et al.. 1998). Slower cross-bridge cycling may be a result of reduced ATP hydrolysis (de Hann et al.. 1989) and reducing force per attached cross bridge. though the major determinant seems to be Pi since acidification alone caused only minor impairment of force generation (Westerblad et al.. 1998).

Also found in this study was potentiation of the twitch torque at one- and five-minutes of recovery from fatigue ($p<0.01$) (Fig. 3-10). Short-term fatigue may also cause a potentiation of evoked force. Calcium will act as a second messenger to activate myosin light chain kinase

(MLCK), a protein kinase. MLCK will cause phosphorylation of phosphorylatable light chain (P-LC) on the myosin molecule. Phosphorylated myosin light chain (PLC) regulates force generation by increasing actomyosin ATPase activity (Grange et al., 1993). This can result in a potentiation of force following short-term fatigue. Moore et al. (1990) suggest that this phosphorylation also makes contractile proteins more sensitive to available Ca^{+2}. Zhu and Nosek (1991) demonstrated that accumulating inorganic phosphates impair SR Ca^{+2} ATPase causing slow Ca^{+2} reuptake. With the slower removal of Ca^{+2}, the PLCs remain phosphorylated longer thereby maintaining tension (Grange et al., 1993), becoming dephosphorylated with a time constant of ~5 minutes (Moore et al., 1990). Therefore, a given level of stimulation will generate a larger twitch due to the accumulation of Ca^{+2} in the cytoplasm that will eventually subside as the Ca^{+2} is sequestered back into the SR . While the potentiated twitch is directly linked to PLC (Moore and Stull, 1984), the exact nature is still not well understood (Grange et al., 1993). The study of Ca^{+2} sequestering is difficult to isolate due to the variety of compensatory mechanisms to generate and maintain force.

Tetanic torque was potentiated in the PF at five- and 10-minutes recovery (Fig. 3-11). The explanation is likely related to an increase of the rate of tetanic torque development. As mentioned previously, the short 300 ms stimulation period resulted in a force output on the ascending slope of the force-time integral. Increases in rate of tetanic torque development would allow greater forces to be achieved in the set period of stimulation. This effect was not seen at one-minute of recovery from fatigue ($p<0.01$) (Fig. 3-11) possibly due to a decrease of force per attached cross bridge that occurs with fatigue (de Hann et al., 1989) that occurs for the same reason that decreases occur in voluntary contractions.

5.4.2 Muscle Activation

Another finding of this study was the increase of voluntary inactivation (i.e. decrease in voluntary activation) throughout recovery from fatigue, though this was only significant at five-minutes of recovery ($p<0.05$) (Fig. 3-12). Also decreased by fatigue was iEMG of the GAST ($p<0.01$) (Fig. 3-14) and GAST:TA ($p<0.01$) (Fig 3-16) ratio throughout recovery, and decreased iEMG of SOL at one-minute of recovery ($p<0.01$) (Fig. 3-15). While fatigue-induced decrements in MVC force can be attributed to metabolic disturbances in the periphery, there is also evidence for decreases in muscle activation (i.e. decreased ITT and agonist EMG). It is unlikely that supraspinal controls were depressed by the fatigue protocol, however peripheral processes may reflexively inhibit motoneuron excitability since inhibition may be derived from III and IV afferents (Badier et al., 1993). These inhibitors are found in the muscle proprioceptors and connective tissue and are speculated to be a form of protection against muscle and joint damage (Kraemer et al., 1988). Group III and IV afferents are metaboreceptors that are sensitive to, among other stimului, ischemia and pH (Kaufman et al., 1984) and exert a powerful inhibition to decrease motocortical discharge. They are activated when muscle pH falls and extracellular potassium concentrations increase with the development of fatigue. Ischemia also results from any muscle contraction over 50% (Sjogaard, 1987). Thus, the inhibition of motoneuron excitability from the subsequent stimulation of group III and IV afferents may result in a decline of firing frequency to a muscle (Badier et al., 1993; Bigland-Ritchie et al., 1986b). Further evidence of afferent inhibition is provided by findings that show blocking the afferent pathways results in no decline of firing frequency (Hagbarth and Macefield, 1995).

Since increasing muscle tension occurs by recruiting additional motor units (increasing recruitment) or by increasing firing frequency of already firing motor unit (increasing rate coding) (Deluca, 1985) decreased muscle activation may result by decreasing either or both of these. This study, and others also demonstrate that fatigue impairs activation (Behm and St. Pierre,1997; Petrofsky et al., 1980). Fatigue can impair voluntary activation due to failure of regeneration of the action potential along the axon, across the sarcolemma, and into the t-tubule to the terminal cisternae (see review by Green, 1997). Also implicated in failing activation are the regulatory and contractile proteins, and impaired ATP generation (see review by Green, 1997).

5.4.3 Co-contractions

Lower levels of reciprocal inhibition could explain the increase of TA iEMG at 10- minutes of recovery as compared to one-minute of recovery from fatigue ($p<0.05$) (Fig. 3-13). It has been demonstrated that during fatigue there is increased antagonist muscle activity (i.e. lower levels of reciprocal inhibition) (Moritani, 1993) likely to stabilize and protect the joint (Katz et al., 1991), though in athletes it is not uncommon to observe an initial decrease of antagonist activity (Katz et al., 1991). Similarly, with recovery from fatigue this experiment demonstrated an initial decrease of TA iEMG, though not significant, followed by slowly increasing antagonist activity (Fig. 3-13). The relatively small but significant changes in both the EMG of the SOL and TA explains the lack of significance being found in the SOL – TA ratio.

5.4.4 Temporal Characteristics

The present study found an increased rate of twitch (Fig. 3-18) and tetanic torque development (Fig. 3-20) in the plantar flexors and a decreased time to peak tetanus (TPT_e) (Fig. 3-19). in addition, ½ RT_{tw} (Fig. 3-21) declined for both twitch and tetanic torque. While some research indicates that there is a decrease of contraction velocities with fatigue (Metzger and Fitts, 1987; Orizio, 1999; Petrofsky et al., 1980), others indicate an increase (Pääsuke et al., 1997). While rate of voluntary torque development decreased, the rate of twitch and tetanic torque development increased. As mentioned previously, central activation decreased, possibly resulting in decreased firing frequency leading to a lower rate of voluntary force development. Central activation is not a factor in evoked contractile properties since the source of stimulation is external (Behm and St. Pierre, 1997). Therefore a reduced rate of Ca^{+2} reuptake would maintain PLC phosphorylation allowing for force to be generated more quickly. An additional possibility to explain the increased rate of evoked torque is a post-activation increase of the sensitivity of calcium as initially described by Metzger and associates (1989), and further supported by studies from Grange and associates (Grange et al., 1995; Vandenboom et al., 1995).

The ½ RT_{te} experienced a slight, insignificant increase at one- minute of recovery from fatigue followed by a decrease by 10- minutes ($p<0.05$) (Fig. 3-22) compared to one- minute of recovery. The ½ RT_{tw} also only decreased at 10- minutes of recovery from fatigue ($p<0.01$) (Fig. 3-21) compared to pre-fatigue values. It may be likely that the ½ RT_{tw} was decreased throughout the recovery period, though the potentiation of twitch torque at one- and five- minutes of recovery overshadowed the more rapid ½ RT_{tw}. It was only when the twitch torque returned to baseline values that the decrease of relaxation time was demonstrated (Fig. 3-10 and 3-21).

Behm and St. Pierre (1997) also found a decrease of ½ RT_{tw} from high intensity fatigue. As previously discussed, it is possible that the reactive and functional hyperemia triggered an influx of blood to the muscle. The increase of blood would contribute to increased stiffness (Ebersole et al., 1999) by the elastic components thereby leading to reduced ½ RT_{tw}. While other properties showed rapid change to hyperemia due to clearing of metabolic byproducts, osmotic pressure would require more time to build and cross into the muscle tissue.

5.5 Summary

The goals of this study were two-fold. The primary goal of this study was to investigate the interaction effects of 22°C local muscle temperature on the recovery of specific evoked and voluntary contractile properties in intact human plantar flexors at one-, five-, and 10-minutes after high intensity fatigue. This was accomplished but found little interaction effect of cold on the rate of recovery. Since most rate processes are impaired by cold in muscle it was hypothesized that rate of recovery would also be slowed. This was not the case for the most part. One possible explanation, though not measured, is a reflex vasodilation of the blood supply to and from the PF muscles allowing for unimpaired recovery thereby negating any inhibition effects of cold. The vasodilation can be caused by metabolic byproducts that occur as a result of high intensity contractions (i.e. functional hyperemia). Occlusion will also occur due to cold: occlusion that will in turn stimulate further reactive hyperemia. The vasodilation may continue to allow for recovery despite the low temperature.

The secondary goal of this study was to validate previous studies and add to the body of knowledge about the main effects of recovery from fatigue with homeostatic temperature and the

main effects of local hypothermia on unfatigued muscle on evoked and voluntary contractile properties in intact human plantar flexors. The results found for both cold and fatigue were generally well supported by the current literature.

SUMMARY AND CONCLUSION

The primary goal of this study was to investigate the interaction effects of 22°C local muscle temperature on the recovery of specific evoked and voluntary contractile properties in intact human plantar flexors at one-, five-, and 10-minutes after high intensity fatigue. The secondary goal of this study was to validate previous studies and add to the body of knowledge about the main effects of recovery from fatigue with homeostatic temperature and the main effects of local hypothermia on unfatigued muscle on evoked and voluntary contractile properties in intact human plantar flexors. The effects of both local hypothermia and local muscle fatigue on voluntary and evoked contractile properties have previously been well documented. In the present study, analyzing each variable independently generally confirmed the results that have been previously demonstrated. The current study has demonstrated that while the majority of rate dependent processes in the PF muscle group are impaired by local hypothermia, recovery from fatigue is generally not. While these results were unexpected, reactive and functional hyperemia seems a logical explanation for maintaining the rate of recovery between normothermic and hypothermic conditions. Studying this theory more directly should be a direction for future research.

This study began with the following statements of hypothesis:

1. The rate of recovery of recovery of voluntary activation will be slowed by local muscular hypothermia.
2. The rate of recovery of voluntary torque will be slowed by local muscular hypothermia.

3. The rate of recovery of twitch torque will be slowed by local muscular hypothermia.
4. The rate of recovery of tetanic torque will be slowed by local muscular hypothermia.
5. The rate of recovery of the rate of voluntary torque development will be slowed by local muscular hypothermia.
6. The rate of recovery of the rate of twitch torque development will be slowed by local muscular hypothermia.
7. The rate of recovery of the rate of tetanic torque development will be slowed by local muscular hypothermia.
8. The rate of recovery of twitch half-relaxation time will be slowed by local muscular hypothermia.
9. The rate of recovery of tetanic half-relaxation time will be slowed by local muscular hypothermia.
10. The rate of recovery of the duration of the compound muscle action potential will be slowed by local muscular hypothermia.
11. The rate of recovery of amplitude of the compound muscle action potential will be slowed by local muscular hypothermia.
12. The rate of recovery of time to peak torque of twitch contraction will be slowed by local muscular hypothermia.
13. The rate of recovery of time to peak torque of tetanus contraction will be slowed by local muscular hypothermia.

14. The rate of recovery of time to peak torque of voluntary contraction will be slowed by local muscular hypothermia.

The results of this study can conclude that the null hypothesis of all but hypothesis 8, 10, and 13 cannot be rejected since local hypothermia had no statistically significant effect on any other dependent variable.

There are several variables that can be altered or manipulated in order to study from another perspective the question of how hypothermia affects recovery from fatigue. This study investigated high intensity, isometric exercise and recovery from the subsequent fatigue in plantar flexors at 22°C. There are several future directions that this research could take. The theory put forward here that reactive and functional hyperemia kept recovery at a normothermic rate could be investigate by measuring blood flow. The duration of the fatigue protocol has been demonstrated to have different mechanisms of fatigue as has the type of contraction used so that using lower intensity dynamic contractions would be a logical progression to the current study. Evoked and voluntary contractile properties have also been shown to display different results at varying levels of cold or heat so experimenting with alternative temperatures would be another possible research topic. It may also be interesting to study core hypothermia on local muscle fatigue and aerobic recovery.

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Appendix A

THE EFFECTS OF LOCAL HYPOTHERMIA ON PLANTAR FLEXOR VOLUNTARY AND EVOKED CONTRACTILE PROPERTIES

Volunteers Needed for Physiology Research (18-30 years old)

Research is being conducted to investigate the effects of cold water on the voluntarily and evoked activation of muscles.

This study will involve two 1½ hour test sessions in which subject's leg will be partially wrapped in cold tubing during which muscles will be electrically stimulated. Invasive temperature monitoring will be conducted during testing.

For more information in test procedures, please contact
Dr. David Behm
School of Physical Education, Recreation, and Athletics
Office: PE 2006
Phone: 737-3408

Appendix B

SCHOOL OF PHYSICAL EDUCATION. RECREATION AND ATHLETICS
MEMORIAL UNIVERSITY OF NEWFOUNDLAND

Consent To Participate In Bio-medical Research

THE EFFECTS OF LOCAL HYPOTHERMIA ON PLANTAR AND DORSIFLEXORS
VOLUNTARY AND EVOKED CONTRACTILE PROPERTIES

INVESTIGATORS: Eric Drinkwater. BPE: David Behm. PhD: Matthew White. PhD: Neil Rodgers. MD

You have been asked to participate in a research study. Participation in this study is entirely voluntary. You may decide not to participate or may withdraw from the study at any time without penalty.

Information obtained from you or about you during this study. which could identify you. will be kept confidential by the investigators. The investigator will be available during the study at all times should you have any problems or questions about the study.

1. Purpose of study

This study will demonstrate the effects of localized cooling on the recovery from fatigue of muscles and nerves of the lower leg. While a great deal of research exists regarding muscle activation and recovery under normal temperatures. research is limited in the area regarding how cooling changes recovery. This will be important to many people involved in outdoor labor and recreation.

2. Description of procedures and tests

The subject's skin of the lower dominant leg will be shaved and cleaned. A physician will numb a small section of the leg and an intramuscular thermometer will be inserted 20 millimeters into the calf muscle. A total of nine surface monitoring electrodes will be placed on the subject's skin. Two stimulating electrodes will also be placed above and below the calf muscle on the skin surface.

In the first experimental day all procedures will be conducted in a warm room with the subject clothed in shorts and a t-shirt. During the second experimental session in which the limb cooling procedure is. the subject's leg will be wrapped in tubing circulating -3°C anti-freeze and then wrapped in insulation.

Once all apparatus are in place, the subject will be secured into a modified boot apparatus. During initial testing, subjects will be asked to maintain varying intensities of muscle contractions by pressing the toes down (plantar flexion), much like pressing the gas pedal of a car. Stimulation of these muscles will occur during resting and contracting conditions.

After this initial testing, plantar flexion fatigue testing will proceed. Subjects will be given 3 seconds to contract the calf muscle to 75% of their maximum, hold that contraction for 14 seconds, and then slowly release it over the final 3 seconds. This will proceed for as long as the subject can continue to do so. The initial testing will then be repeated immediately after fatigue, five minutes after fatigue, and ten minutes after fatigue.

3. Duration of participant's involvement

Subjects will be needed on two separate days with two to three days between each session. Each session will last approximately one and one-half hours.

4. Possible risks, discomforts, or inconveniences

Subjects will experience moderate discomfort and have a low risk of light bleeding with the injection of the anesthetic and insertion of the thermometer into the muscle. There is an unlikely possibility of an allergic reaction to the lidocaine anaesthetic.

Cold will be experienced with the cooling of the leg for a short period of time until the leg acclimatizes to the cold.

Twitch stimulation will involve 100 to 150 volts for 50 micro-seconds. Tetanic stimulation will involve 100 volts at a high frequency for 1/3 of a second. Both procedures feel like a short muscle cramp and are not dangerous.

Subjects will be asked to perform lower leg exercise to fatigue that may cause some discomfort. Mild muscle soreness may occur one to three days after the fatiguing protocol.

Cable will be sealed onto the subject with adhesive tape. Subjects must shave the lower portion of the leg being tested. Removal of the tape will cause some discomfort.

5. Liability statement

Your signature indicates your consent and that you have understood the information regarding the research study. In no way does this waive your legal rights nor release the investigators or Memorial University from their legal and professional responsibilities

Signature Page

THE EFFECTS OF LOCAL HYPOTHERMIA ON PLANTAR FLEXOR
VOLUNTARY AND EVOKED CONTRACTILE PROPERTIES

INVESTIGATORS: David Behm, PhD; Matthew White, PhD; Eric Drinkwater; Arash Fard, MD

To be signed by participant:

I, the undersigned, agree to my participation in the research study described above.

Any questions have been answered and I understand what is involved in the study. I realize that participation is voluntary and that there is no guarantee that I will benefit from my involvement.

I acknowledge that a copy of this form has been given to me.

Signature of Participant Date

Signature of Witness Date

To be signed by investigator:

To the best of my ability I have fully explained the nature of this research study. I have invited questions and provided answers. I believe that the participant fully understands the implications and voluntary nature of the study.

Signature of Investigator Date

Appendix C

Table of Dependent Variables

Dependent Variable	**Hypothesis Question Investigated**	**Physiological Significance**
MVC torque	2	Force the subject can voluntarily generate: it is the functional output of the combination of central activation and peripheral capacity. The interpolated twitch technique evoked contractions interpolated onto voluntary contractions of 25, 50, 75, and 100% of MVC to develop a second order polynomial for the estimation of muscle activation (Behm et al, 1996).
Rate of Voluntary torque development	3	Rate at which the subject can voluntarily generate force: it is related to firing frequency and the rate of the events of excitation-contraction coupling.
Muscle Inactivation (by ITT)	1	Using evoked contractions interpolated onto voluntary contractions of 25, 50, 75, and 100% of MVC
Gastrocnemius EMG	1	Measuring electrical activity of a primary agonist during a voluntary contraction as a further measure of muscle activation.. It was collected to 1. calculate agonist to antagonist ratios, and 2. validate the results of the ITT.
Soleus EMG	1	As gastrocnemius EMG
Tibialis Anterior EMG	1	Measuring electrical activity of a primary antagonist during a voluntary contraction as an indication of the extent of co-contractions to determine possible changes in intermuscular co-ordination.
Gastrocnemius: Tibialis Anterior EMG	1	Fatigue of an agonist or synergist muscle group will often generate activity of the antagonist. Reducing this ratio indicates fatigue. This activation mechanism becomes less dominant in weight-trained individuals.
Soleus : Tibialis Anterior EMG	1	As Gastrocnemius: Tibialis Anterior
Twitch torque	2	Evoked contraction to measure the total force generated by a single maximal electrical stimulus as an indication of possible changes in excitation-contraction coupling.
Time to peak twitch torque	6	Evoked contraction to measure the rate of force development with excitation-contraction coupling. It is an assessment of the propagation of the action potential, and sarcoplasmic reticulum Ca^{2+} release and binding.

Rate of twitch torque development	3	Since a muscle may develop less force but in the same amount of time, the rate at which that force is developed is also relevant.
Twitch ½ relaxation time	4	Time to release tension from the evoked contraction to measure the rate of activity of the sarcoplasmic reticulum Ca^{2+} re-uptake and release of myosin cross-bridges.
Amplitude of compound muscle action potential (M-wave)	5	Evoked property measured from the twitch. It is a measure of the muscle electrical property measuring the voltage differential of the muscle's action potential during an evoked twitch. It is derived from the Na^{+} and K^{+} flux that generates the action potential along the sarcolemma.
Duration of M-wave	5	Muscle electrical property measuring the duration of the change in voltage differential of the muscle's action potential during an evoked twitch. It is derived from the rate of Na^{+} and K^{+} flux that generates the action potential along the sarcolemma.
Tetanic torque	2	A high frequency evoked stimulation (100 Hz) using maximal evoked twitch stimulation parameters as a measure of changes in myofilament cross-bridge kinetics.
Time to peak tetanic torque	6	Evoked stimulation at a high firing frequency used as an indication of changes in the rate of cross-bridge cycling. Again, if voluntary RFD declines, the causes are central; if tetanic RFD declines, the cause is peripheral.
Rate of tetanic torque development	3	Since a muscle may develop less force but in the same amount of time, the rate at which that force is developed is also relevant.
Tetanic ½ relaxation time	4	Time to release tension from the evoked contraction to measure the rate of activity of the sarcoplasmic reticulum Ca^{2+} re-uptake and release of myosin cross-bridges.
Duration of fatigue protocol		Voluntary local muscular endurance

FIGURES LEGEND

2-1: Graphs schematically represent the effects of fiber type on hypothermic twitch and tetanic tension

Figure 3-1: Bars represent the torque generated by the dominant plantar flexors with tetanic stimulation comparing normothermic to hypothermic conditions. Asterisks indicate that the torque generated was significantly different to the $p<0.05$ level.

Figure 3-2: Bars represent the rate of MVC torque development in the dominant plantar flexors under normothermic and hypothermic conditions. Asterisks indicate that the torque generated was significantly different to the $p<0.05$ level.

Figure 3-3: Bars represent the time to maximal twitch torque of the dominant plantar flexors torque under normothermic and hypothermic conditions. Asterisks indicate that the torque generated was significantly different to the $p<0.05$ level.

Figure 3-4: Bars represent the rate of twitch torque development of dominant plantar flexors twitch under normothermic and hypothermic conditions. Asterisks indicate that the torque generated was significantly different to the $p<0.05$ level.

Figure 3-5: Bars represent the half relaxation time of the twitch with the dominant plantar flexors under normothermic and hypothermic conditions. Asterisks indicate that the torque generated was significantly different to the $p<0.05$ level.

Figure 3-6: Bars represent the time to peak tetanus of the dominant plantar flexors under normothermic and hypothermic conditions. Asterisks indicate that the torque generated was significantly different to the $p<0.05$ level.

Figure 3-7: Bars represent the rate of tetanic torque development of the dominant plantar flexors under normothermic and hypothermic conditions. Asterisks indicate that the rate of torque development was significantly different to the $p<0.05$ level.

Figure 3-8: Bars represent the half relaxation time of the tetanus in the dominant plantar flexors under normothermic and hypothermic conditions. Asterisks indicate that the rate of torque development was significantly different to the $p<0.05$ level.

Figure 3-9: Bars represent the MVC torque generated by the dominant plantar flexors at a given recovery interval. Asterisks indicate that torque generated was significantly different from pre-fatigue values to the $p<0.05$ level.

Figure 3-10: Bars represent the peak twitch torque of the dominant plantar flexors at a given recovery interval. Asterisks indicate that the torque was significantly different from pre-fatigue values to the $p<0.05$ level.

Figure 3-11: Bars represent the peak tetanic torque of the dominant plantar flexors at a given recovery interval. Asterisks indicate that the torque generated was significantly different from pre-fatigue values to the $p<0.05$ level.

Figure 3-12: Bars represent the percent of inactive fibers as estimated by the interpolated twitch technique (ITT) during an MVC in the dominant plantar flexors at a given recovery interval. Asterisks indicate that the time was significantly different to the $p<0.05$ level.

Figure 3-13: Bars represent the integrated, rectified EMG activity during a maximal voluntary contraction from the dominant TA at a given recovery interval. Addition signs indicate that the time was significantly different from 1-minute recovery values to the $p<0.05$ level.

Figure 3-14: Bars represent the integrated, rectified EMG activity during a maximal voluntary contraction from the dominant gastrocnemius (GAST) at a given recovery interval. Asterisks indicate that the time was significantly different to the $p<0.05$ level.

Figure 3-15: Bars represent the integrated, rectified EMG activity during a MVC from the dominant soleus (SOL) at a given recovery interval. Asterisks indicate that the time was

significantly different from pre-fatigue values to the $p<0.05$ level. Addition signs indicate that the time significantly different from 1-minute recovery values to the $p<0.05$ level.

Figure 3-16: Bars represent the ratio of integrated, rectified EMG activity of the dominant gastrocnemius (GAST) and tibialis anterior (TA) during a maximal voluntary contraction at a given recovery interval. Asterisks indicate that the time was significantly different to the $p<0.05$ level.

Figure 3-17: Bars represent the rate of MVC torque development in the dominant plantar flexors at a given recovery interval with data collapsed over normothermic and hypothermic data. Asterisks indicate that the rate of torque development was significantly different from pre-fatigue values to the $p<0.05$ level.

Figure 3-18: Bars represent the rate of twitch torque development in the dominant plantar flexors at a given recovery interval. Asterisks indicate that the rate was significantly different from pre-fatigue values to at least the $p<0.05$ level. Addition signs indicate that the rate was significantly different from 1-minute recovery values to at least the $p<0.05$ level.

Figure 3-19: Bars represent the time to maximal tetanic torque in the dominant plantar flexors at a given recovery interval with data collapsed over normothermic and hypothermic data. Asterisks indicate that the time was significantly different from pre-fatigue values to the $p<0.05$ level.

Figure 3-20: Bars represent the rate of tetanic torque development in the dominant plantar flexors at a given recovery interval. Asterisks indicate that the rate of torque development was significantly different to the $p<0.05$ level. Addition signs indicate that the rate was significantly different from 1-minute recovery values to the $p<0.05$ level.

Figure 3-21: Bars represent the half relaxation time of the twitch in the dominant plantar flexors at a given recovery interval. Asterisks indicate that the time was significantly different to the $p<0.05$ level. Addition signs indicate that the time significantly different from 1-minute recovery values to at least the $p<0.05$ level.

Figure 3-22: Bars represent the half relaxation time of the tetanus in the dominant plantar flexors at a given recovery interval. Addition signs indicate that the time was significantly different from 1-minute recovery values to the $p<0.05$ level.

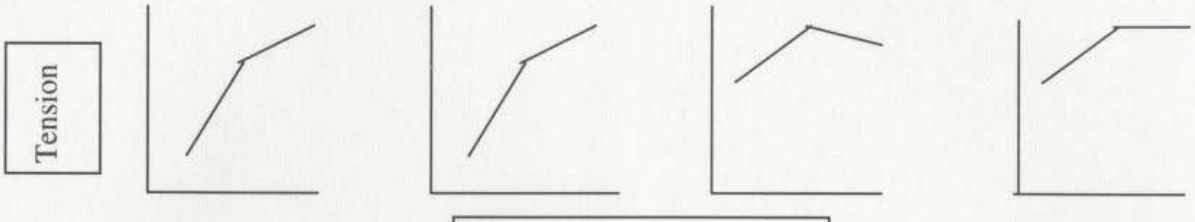
Figure 3-23: Bars represent the half relaxation time of the twitch in the dominant plantar flexors at a given recovery interval while either normothermic or hypothermic. Asterisks indicate that the time was significantly different from normothermic values to the $p<0.05$ level.

Figure 3-24: Bars represent the time to peak tetanus in the dominant plantar flexors at a given recovery interval while either normothermic or hypothermic. Asterisks indicate that the time was significantly different from normothermic values to the $p<0.05$ level.

Figure 3-25: Bars represent the duration of the M-wave in the dominant plantar flexors at a given recovery interval while either normothermic or hypothermic. Asterisks indicate that the time was significantly different from normothermic values to the $p<0.05$ level.

FIGURES

Figure 2-1: The effects of fiber type on hypothermic twitch and tetanic tension



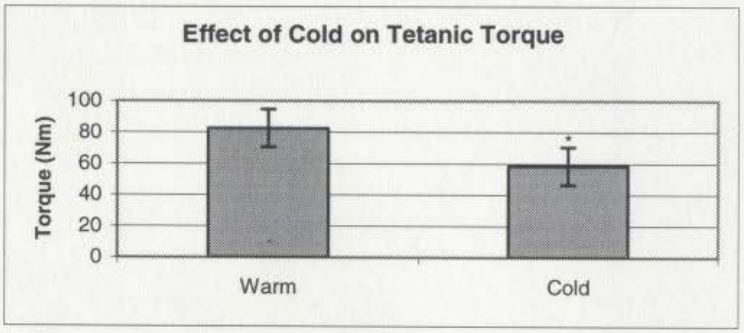
a) tetanic (fast twitch)

b) tetanic (slow twitch)

c) twitch (fast twitch)

d) twitch (slow twitch)

Figure 3-1



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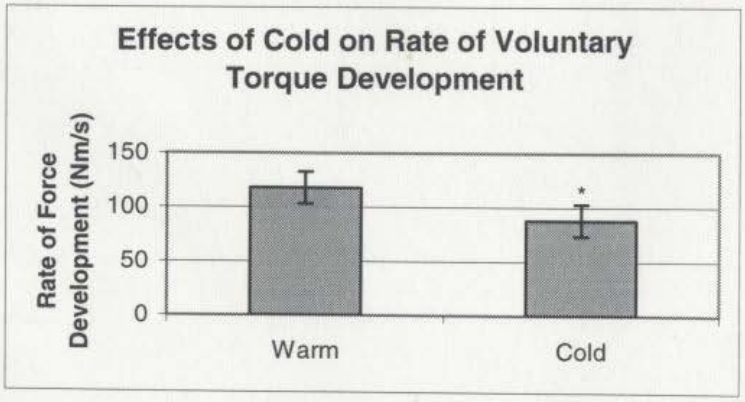

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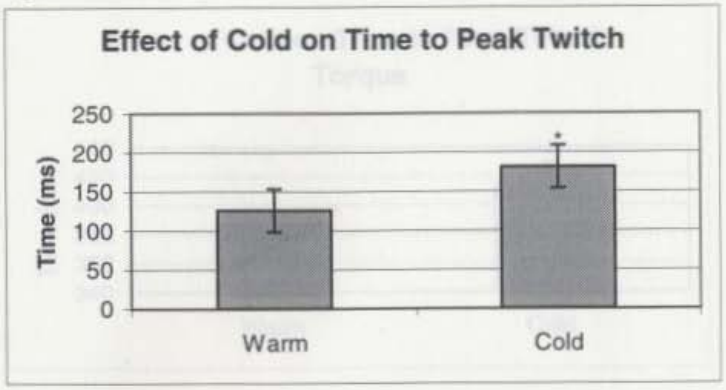

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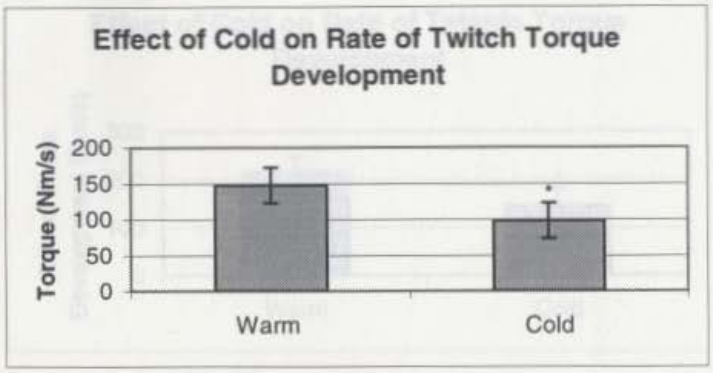
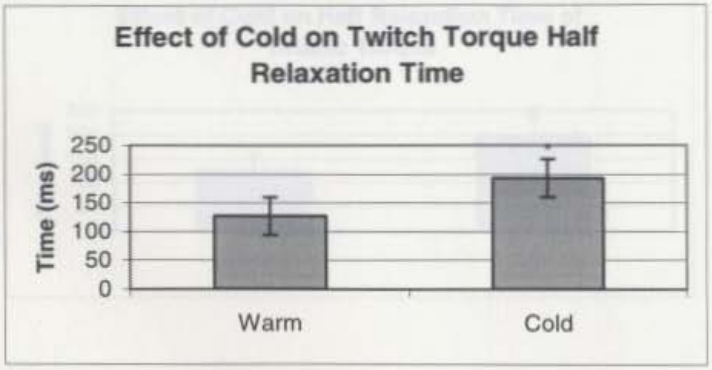


Fig. 3-5



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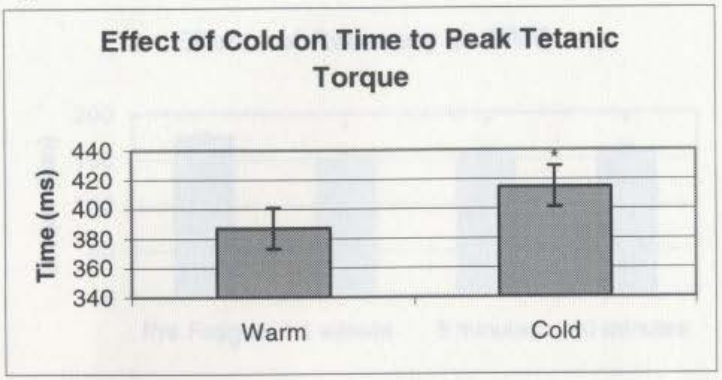

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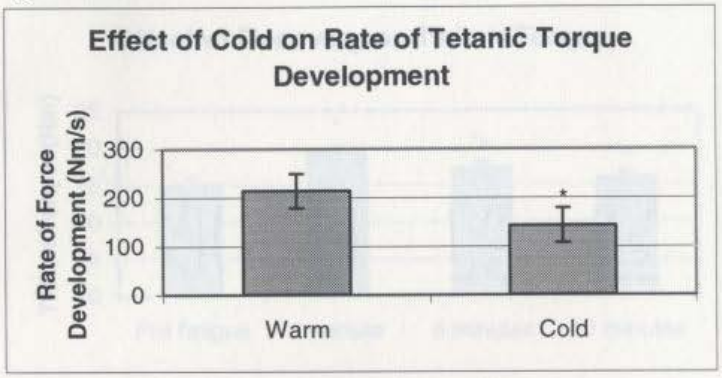

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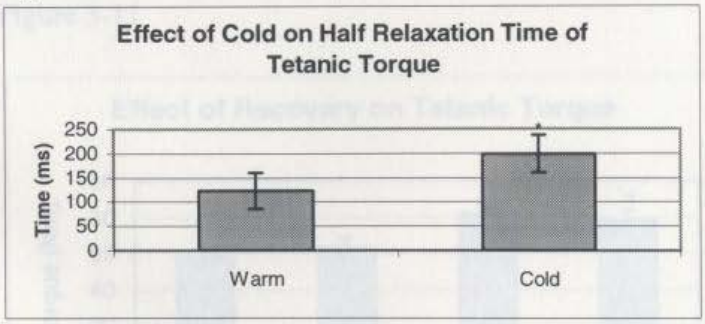

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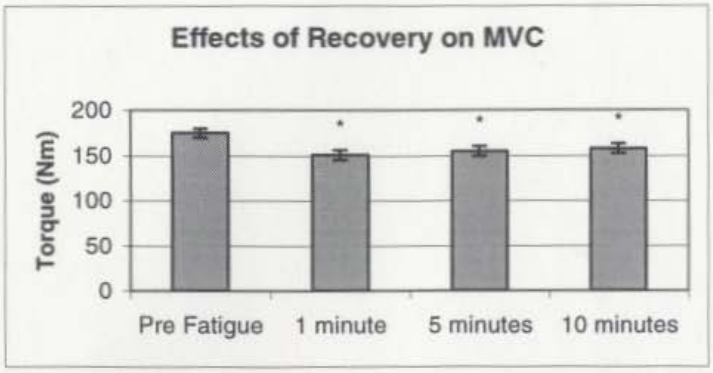

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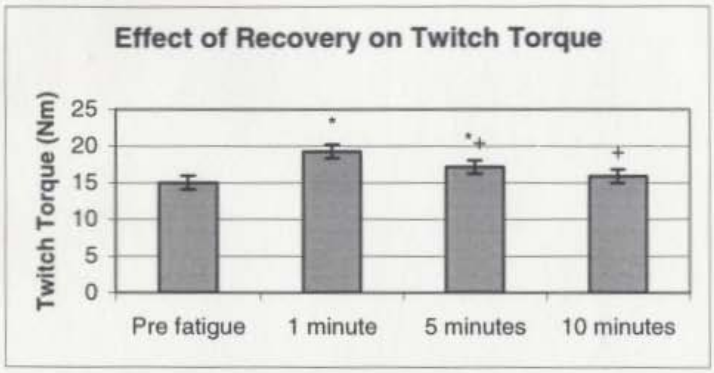

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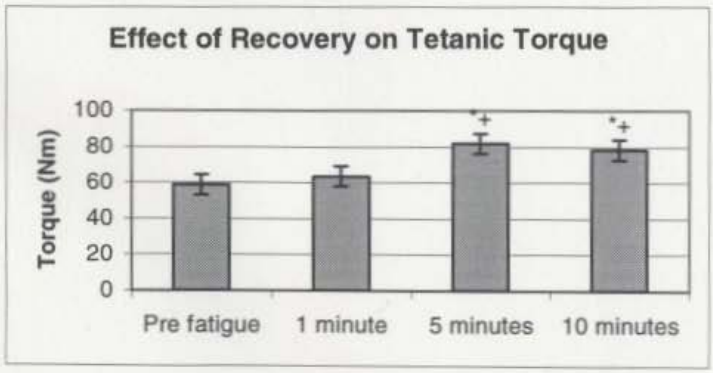

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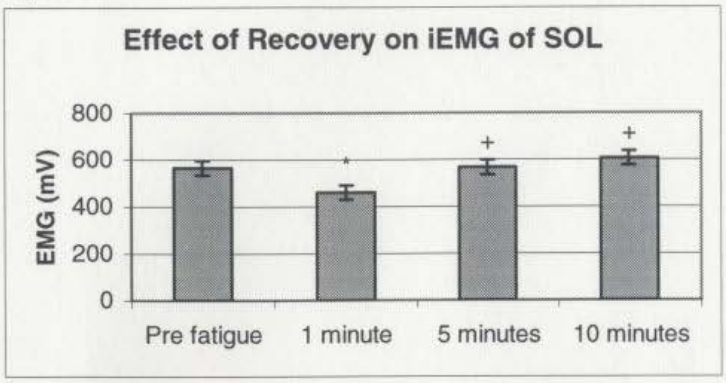
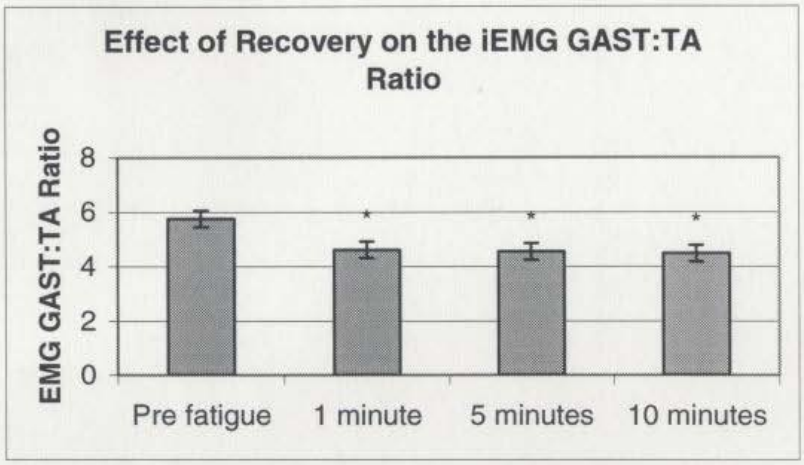


Figure 3-16



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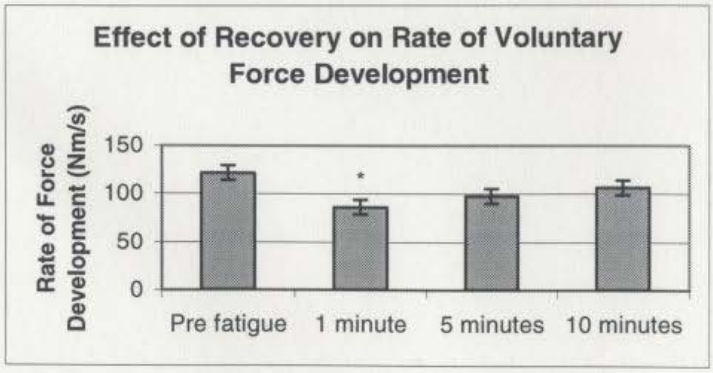

Figure 3-15

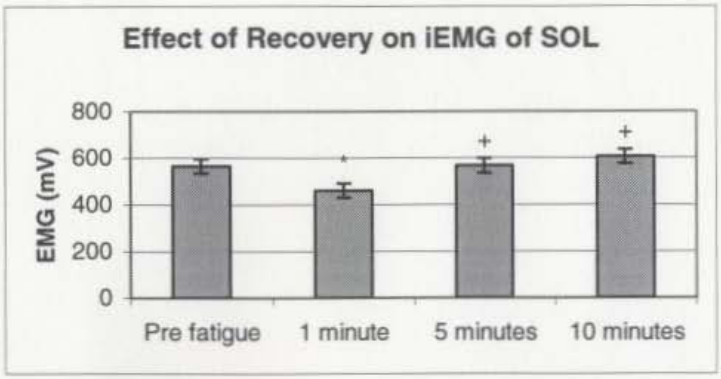

Figure 3-16

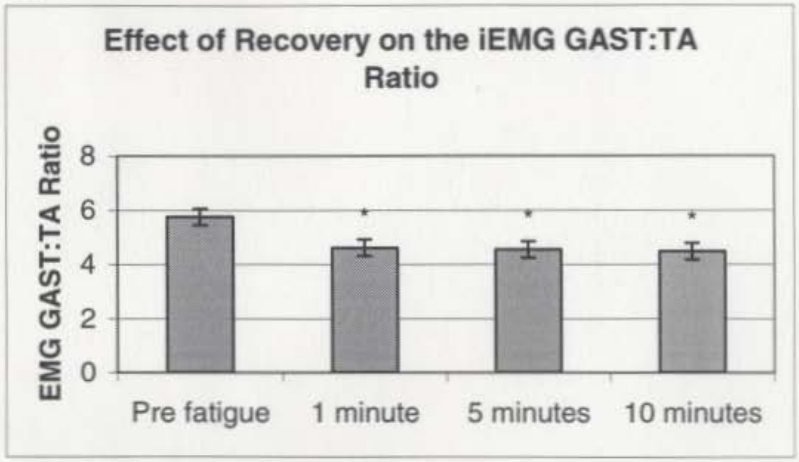

Figure 3-17

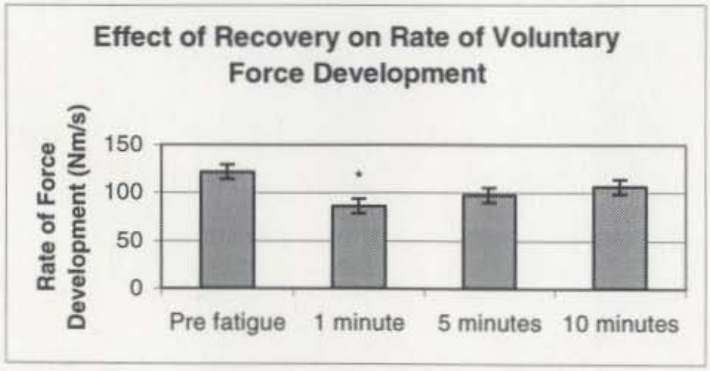

Figure 3-18

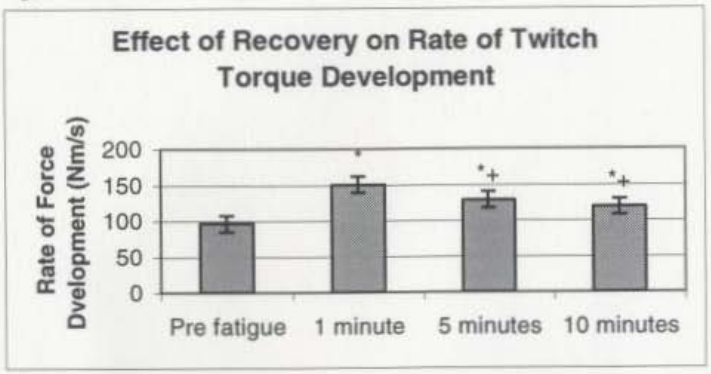

Figure 3-19

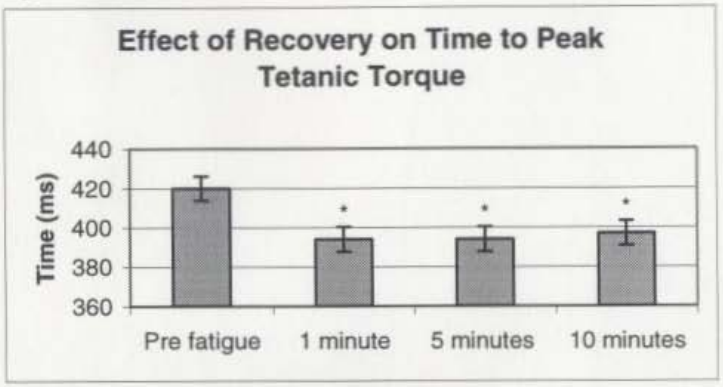

Figure 3-20

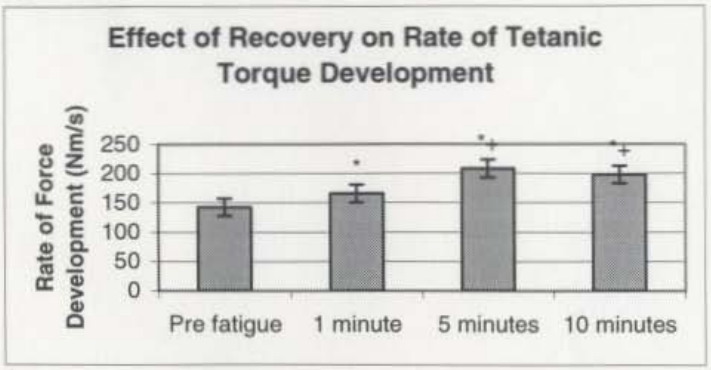

Figure 3-21

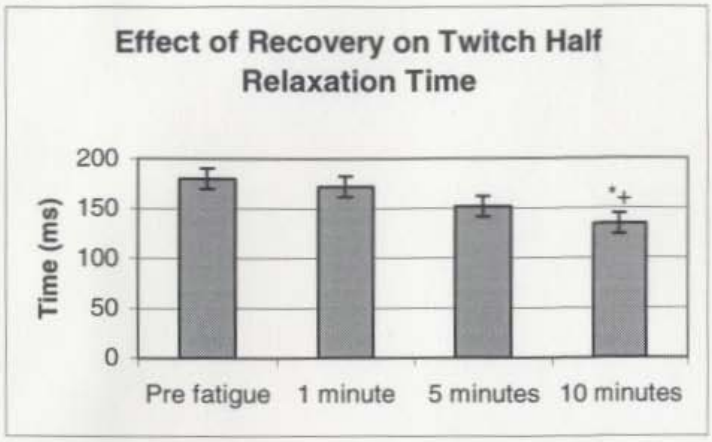

Figure 3-22

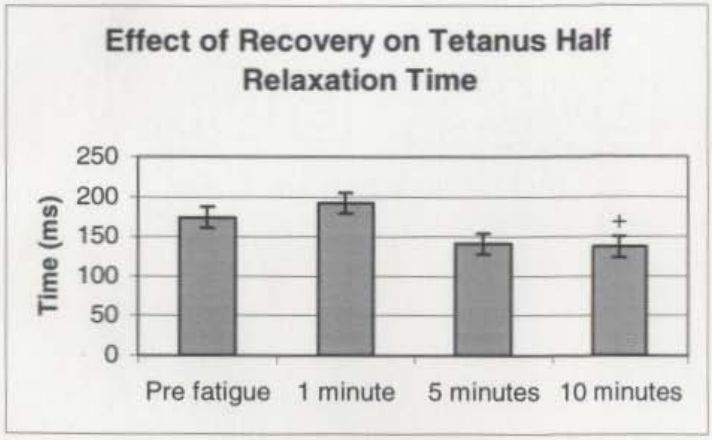

Figure 3-23

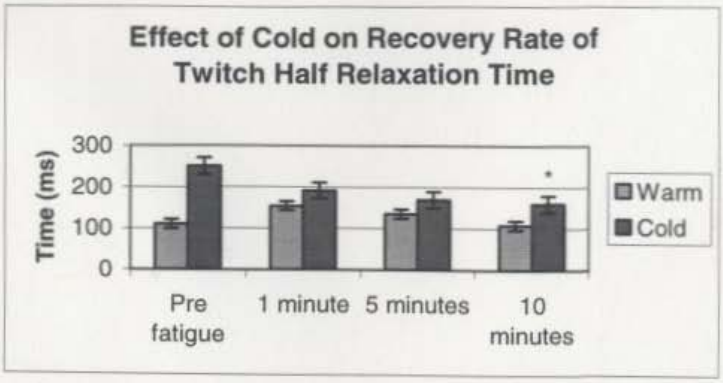

Figure 3-24

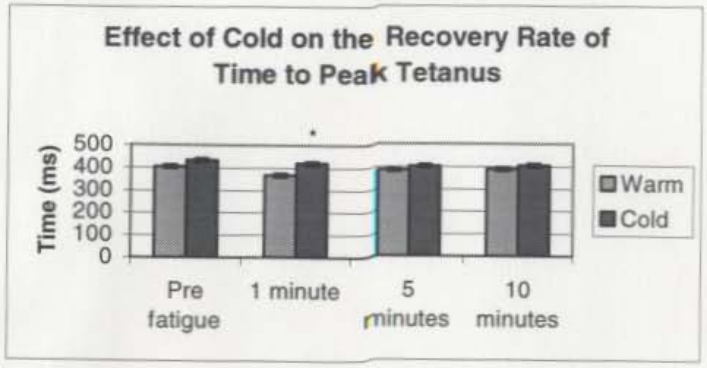

Figure 3-25