

**REDUCED STRENGTH FOLLOWING PASSIVE STRETCH
OF THE HUMAN PLANTARFLEXORS**

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This thesis is dedicated to my dad,

whose leadership by example, always guided me without being guided;

and to my mom,

whose support for my efforts is always limitless and unending;

and finally to my brothers, thank you for your perspective,

it has helped me to keep my own.

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CHAPTER I

STRETCHING TO 'ENHANCE' ATHLETIC PERFORMANCE

1.0 INTRODUCTION

Flexibility training by stretching is advocated to enhance performance and reduce injury risk when done directly before activity (Shellock & Prentice, 1985; Smith, 1994) and as part of an athlete training program (Fleck & Kraemer, 1987; Fox *et al.* 1989; Bloomfield *et al.* 1994). Despite the fact that flexibility is considered one of five major components of physical fitness and although most authorities advocate and promote its use in athletics, there have been very few scientific studies of the influence of flexibility training or stretching on performance. The objective of this literature review will be to examine current thought on flexibility in a conditioning program and how stretching may influence athlete performance.

2.0 DEFINITIONS

Flexibility is defined as the range-of-motion (ROM) around a joint or series of joints (Cureton, 1941) and can be further defined into the components of static and dynamic flexibility (deVries, 1980). Static flexibility is referred to as the end range of movement possible when the limbs are passively stretched whereas dynamic flexibility is referred to as the extent of voluntary movement about a joint as limited by the resistance of a joint to motion. Dynamic flexibility is often implied rather than measured (Hedrick, 1993), but is contextual to sport activity (Shellock & Prentice, 1985) because it indicates the ease of practical movement and the resultant speed at which that movement can be performed as opposed to just the range itself (Fox *et al.* 1989).

Flexibility is determined by structural and architectural qualities of the muscle-tendon unit, joint articular structures, and connective tissues and skin (Fox *et al.* 1989; Hutton, 1992); all are specific to a particular joint motion (Harris, 1969). For the purposes of this review and study, flexibility will refer to the quality indicating the ROM at a joint, and stretching will refer to the acute activity intending to increase that ROM in a single bout. Flexibility training will be the common term for indicating repeated stretching bouts at regular frequency to produce chronic changes in flexibility.

Stretching can be performed ballistically, statically, or using a variety of contraction and hold techniques referred to as proprioceptive neuromuscular facilitation (PNF). Static stretching involves passively stretching a given antagonist muscle by placing

it in a position of maximal stretch and holding it there for an extended period (Shellock & Prentice, 1985). Ballistic and PNF stretching involve active phases during the stretch procedure, either caused by reflex activity from muscles spindles detecting high velocity stretch in ballistic actions, or by voluntary activation in the precontraction phase of PNF stretching. All three stretching types are thought to take advantage of the stretch reflexes in the body to produce an acute increase in ROM; however, it is not the purpose of this review to outline these processes or to evaluate the relative effectiveness of the various procedures. Refer to reviews by Hutton (1992) and Shellock & Prentice (1985) for appropriate descriptions of the neurophysiological basis of stretching. This review will be evaluating the effectiveness of stretching and flexibility training as an intervention to affect a defined performance variable. Specific delimitations of stretching technique or practice employed will be highlighted only when necessary to describe the relation of that intervention to the performance variable in question.

Common terms used to describe the parameters of flexibility are muscle stiffness and elasticity. Stiffness represents the relation of the amount of force causing a deformation in a material relative to the amount of deformation occurring, or the ratio of stress to strain. The deforming stress is measured by the force per unit of cross-sectional area of the material resisting the stress and the strain is represented by the change in length relative to the original length of the material. Stiffness is similar to elasticity in that both qualities represent a counterforce or resistance to deformation. Stiffness represents the amount of counterforce, whereas elasticity classically describes the extent to which a

material returns to its original size and shape. Stiffness and elasticity are interrelated and sometimes referred to as 'elastic stiffness'. With muscle tissue, these properties are usually represented as passive tension (or passive torque) for a given muscle length (or joint angle indicating muscle length). Elasticity is conceptually the inverse of stiffness, where more 'elasticity' is represented by smaller increments in passive tension for a given increment in muscle length as compared to a more 'stiff' tissue.

3.0 FLEXIBILITY AND 'ENHANCED' PERFORMANCE

3.1 CURRENT VIEWS

Flexibility is considered a component of physical fitness (Cureton, 1941) and important to high levels of muscular performance (Bloomfield *et al.* 1994; Fox *et al.* 1989; Hedrick, 1993). Despite this, there is a dearth of reliable and valid research on the effects of stretching and flexibility training on performance. In a roundtable discussion on flexibility published in the National Strength and Conditioning Association Journal (NSCA Journal 6(4): 10-22, 71-73; 1984), experts in the field of flexibility discussed pertinent issues in flexibility research, and the specific question of the relationship of flexibility to reducing injuries and improving athletic performance was addressed (Anderson, 1984; Beaulieu, 1984; Cornelius, 1984; Prentice, 1984; Wallace, 1984). The collective view of the panel was that data are limited and contradictory, evidence is largely empirical rather than scientific, and that more research is needed. Almost a decade later, there is still little experimental evidence to support the commonly held belief that improved flexibility enhances performance (Hedrick, 1993).

What may have propagated a seemingly unfounded theory is misinterpretation of findings in the literature. Prentice published interpretations of the literature (Shellock & Prentice, 1985; Prentice, 1984) that could be construed to advocate stretching to increase performance. Prentice synopsis the work of Awad and Kotke (1964) examining the effect of the myotatic reflex on increasing maximum muscular tension during a program of brief

isometric exercise by the quadriceps. Prentice (1985) interpreted the findings as “an improvement in strength after the muscle has been subjected to stretching”. It is true that, by the definition of strength being the maximum amount of force generated at a defined velocity, the observation is accurate but the interpretation is greatly out of context. In stretch activation experiments (Galler *et al.* 1994) and stretches during tetanic contractions (Lieber & Friden, 1993), it has been indicated that a length increment imposed during a stretch results in an increased measurement of force. The incremental force decays with time when held (Bagni *et al.* 1995), indicating that the force is likely due to the increased passive tension inherent to muscle-tendon as observed with incremental passive stretch (Magid & Law, 1985) alone. The muscle reflex could act to potentiate force output, though the interpreted ‘stretching’ by Prentice is merely an eccentric contraction where the muscle is lengthening while developing force. The force-velocity relation of muscle also predicts that forces are higher for lengthening than isometric or shortening contractions (Edman, 1988). In this example, ‘stretching’ can not be interpreted as a separate intervention that produced an increase in the intrinsic capability of the muscle to produce force, i.e. strength.

Further interpretations by Prentice of Preo’s (1967) work and Holt *et al.* (1970) and Partridge (1954) [his references] were presented under the guise that stretching improves performance. These articles explained the effects of antagonist contractions on agonist muscle strength and are probably more appropriately applied to studies on the

stretch shortening cycle of muscle (Komi, 1992), or co-contraction, or reflex potentiation (Sale, 1992).

Cornelius (1984) may also be guilty of 'stretching the truth', stating the literature supported that "flexibility enhances the performance of other particular skills". The 'other particular skills' to which Cornelius are referring is sprinting speed and cycling as evaluated in work by Dintiman (1964) and Angle (1963) [his reference]. Dintiman found that when a sprint running program was supplemented with both weight and flexibility training, running speed was significantly better than a program which was unsupplemented by weight training or flexibility alone. The fact that flexibility training alone did not improve running speed raises concern about flexibility training's ergogenic effects. His conclusions about the master's thesis by Angle (1963) are also suspect, when the title for the thesis work is "The effect of progressive program of exercise, using the exercycle, on the flexibility of college women", implying that some resistance-type exercise was the intervention and flexibility was the outcome measure and not vice-versa.

Again, despite the recommendations that more conclusive scientific research be completed to determine if, or how, stretching prior to activity or flexibility training enhances performance (Hedrick, 1993; Shellock & Prentice, 1985), very few studies at present have addressed these research questions.

3.2 'ENHANCED' PERFORMANCE

Bloomfield *et al.* (1994) highlights three main areas where improvements in performance can be made as a result of greater static ROM: 1) increased range of movement in sport activity; 2) greater contractile force in re-utilization of stored elastic energy; and 3) greater force, velocity or [impulse] resulting from increased range to develop force. In the context that stretching may decrease the incidence of injury either acutely or chronically, this could also be interpreted as 'enhanced' performance although it will not be referred to as such or specifically addressed in this review.

Some authors have indicated that more flexible athletes are better performers than inflexible athletes (Beaulieu, 1981). This rather subjective interpretation is mirrored by Bloomfield *et al.* (1994) who states that greater ROM "places athletes into more aesthetic positions... [which are] accompanied by more technically sound performance.... [that are] pleasing to watch". For sports requiring artistic expression, this implication to performance may be beneficial, but for the athlete concerned with increasing strength, speed, and agility, it has little importance. Shellock & Prentice (1985) highlight that athletes with restricted ranges of motion may be limited in speed capabilities. The authors take the example of a sprinter with inflexible hamstrings, who would have a limited stride length and therefore less distance with each step caused by the tight hamstrings. Cornelius (1989) notes that ROM exercise can be effective for improving motor performance because of the increased ability to move freely through the joints' ROM; i.e., dynamic flexibility. Hortobagyi *et al.* (1985) reported an increased stride frequency, isometric force

development and speed of contractions in young sprinters after seven weeks of flexibility training. Dynamic flexibility is indirectly related to static flexibility although greater ROM's may not be required to execute the skill effectively. For example, speed and strength coaches highlight the importance of an 'optimal stride length' for maximal sprinting speed, that overstriding can actually be detrimental to performance. DeVries (1963) reports that acute increases in flexibility have little or no effect on economy of exercise or energy expenditure for running a 100 m sprint. The dynamic flexibility requirements raised by Shellock and Prentice may have more relevance to either injury prevention or running efficiency and consequent energy expenditure in longer duration events.

The second area for improvement identified by Bloomfield *et al.* was in increasing contractile force in a rebound movement or stretch-shortening cycle (SSC). Without undertaking an elaborate review of the SSC literature, it is important to identify that Bloomfield *et al.* (1994) refers to a study by Wilson *et al.* (1992), who examined the effect of flexibility training on increasing 'the elastic contribution' to a powerful concentric bench press action when following a previous eccentric contraction in a group of experienced powerlifters. The study has a number of potential problems: 1) *the study is based on a weak relation of static flexibility to maximal musculo-tendinous stiffness* ($r = -0.544$; $P < 0.05$) (Wilson *et al.* 1991). The stiffness measured was iso-dynamic (active isometric contraction), where tension is related to the number of active cross-bridges (Gordon *et al.* 1966; Ford *et al.* 1981) and therefore, may not adequately reflect series-

elastic compliance; 2) *the Pre-Post measures for stiffness are unreliable*. Stiffness was heterogeneous between the experimental (Exp) and control (Con) groups, one stiffness value was not measured but calculated, and the statistics performed on the stiffness measures violated the assumptions of a one-tailed test; and, 3) *the flexibility exercises for Exp involved resisted exercise which were not controlled in Con for extra training*. Exp performed declined push-ups between benches and wide-range dumbbell flies, accompanied by a chest and a shoulder stretch which produced significantly increased bench press strength by 5.4 % ($P < 0.05$) and concentric only bench press by 4.5% (N.S. $P = 0.10$) in Exp only.

The major finding in the study was that work performed in the first 0.37 s of the concentric phase of a rebound bench press exercise (SSC movement) increased by 20.1% ($P < 0.05$) in the 'flexibility trained' or 'lowered stiffness' group only. Wilson and colleagues concluded greater utilization of stored energy because of lowered system stiffness contributing to increased mechanical work. In a subsequent study (Wilson *et al.* 1994), a seemingly contradictory conclusion was made that a stiffer musculotendinous unit was optimal for maximum concentric and isometric and no relationship was found between stiffness and eccentric performance. The 1994 study also had its drawbacks; extremely different resistances were used in each maximum voluntary contraction for eccentric (ECC), isometric (ISO) and concentric (CON) testing, and different arm angles were used in ECC, ISO, and CON movements which can produce extremely different performance scores in the bench press (Murphy *et al.* 1995). The augmentations of CON

and ISO performance associated with a stiff musculotendinous unit in the 1994 study were most readily seen early in the movements (over the first 100 ms), the same time-frame that resulted in increased work with a less stiff system in the 1992 study. Wilson and colleagues added the caveat that “the results obtained from such investigations may be highly specific to the individual movement analyzed”.

To elaborate on the findings of Wilson *et al.* (1992), Worrell *et al.* (1994) tested the effect of hamstring flexibility training on ECC/CON strength performance. Worrell and colleagues showed significantly increased ECC torque at 60°/s and 120°/s but only increased CON torque following the 120°/s ECC contraction after 3 wks of 5 session/wk flexibility training. There were no significant increases in flexibility in the study; however, the authors proposed that flexibility training increased utilization of stored elastic potential energy with ECC actions to increase torque at the higher velocity CON contraction. They add that at slower CON velocities the instantaneous moment may be lost from the previous ECC action. There was no control group for the study.

Wilson *et al.* (1994) described that a stiffer musculotendinous unit is more beneficial for a quick movements so that less force is wasted taking up slack in series. In sprinting, this translates to less force required to stiffen the system before force can be applied to accelerate the body. To the contrary, Hortobagyi *et al.* (1985) hypothesized that greater compliance would increase SSC performance in sprinting. DeVries (1963) reports greater running efficiency in tighter runners at a wide range of running speeds. In distance runners, a less flexible i.e. ‘stiff’ system, results in greater running efficiency

(oxygen cost at a given running velocity) because less energy is expended by the muscles to regulate proper postural positioning with each stride (Craib *et al.* 1996).

Nelson *et al.* (1996) compared jump performance in squat and countermovement (SSC) jumps before and after stretching and found that maximal jump height, maximal vertical force, and kinetic energy were significantly reduced after stretching in both jumps. The study was designed to determine if stretching would alter the ability to store and reutilize elastic energy under the premise that the SSC movements would benefit from a greater elastic potential created with stretching. The fact that both movements were affected equally introduces the possibility that other factors may affect maximal force production after stretching that are unrelated to the elastic potential.

The third mechanism proposed by Bloomfield and colleagues by which stretching might enhance performance was that it might result in greater applied impulse in ballistic actions. Their theory is that, if a greater ROM exists for an action, then more time will be available to generate force. For ballistic actions, the time to reach peak velocity is important. A greater range to accelerate a limb before contact with an external object can result in a greater impulse imparted to the object, and thus more distance on the homerun ball, or greater velocity on a drive shot in squash. For a thrower, the greater time to accelerate the arm before release, can result in a greater velocity at release. However, it is commonly observed that athletes in ballistic sports have more stiffness in the limb performing the action than the contralateral arm (Alter, 1996). Increasing stiffness may be an adaptation which enhances performance apart from the range with which to develop

force. While the increased time-for-force development ‘theory’ seems logical, once again it has not been scrutinized scientifically, and the only evidence to support the theory is empirical. Empirical evidence is not invalid, though the lack of scientific support highlights the difficulty in identifying flexibility as the only characteristic that resulted in greater performance outside of other variables such as architecture, biomechanics, strength, and skill, and different from the potential benefit in reducing injury risk.

The perceived outcome that stretching and flexibility training produces, is reduced muscle stiffness to allow greater ROM and improve dynamic flexibility. The areas for performance enhancement previously presented can be addressed from this perspective. However, there is a need to justify empirical evidence from coaches and athletes with hard scientific proof. The remaining sections of the literature review will present information related to stretching and flexibility training, that has to date, been scientifically supported.

3.3 EFFECT OF STRETCHING ON MUSCLE STIFFNESS

Stiffness of resting muscle is determined by elements within the sarcomere, connective tissue elements surrounding individual fibres, bundles of fibers and whole muscle, by the cytoskeletal network, and by components of non-muscle origin, such as the joint capsule and the skin (Bobet *et al.* 1990; Fox *et al.* 1989; Granzier & Wang, 1993b; Granzier & Wang, 1993a; Hill, 1968; Howell *et al.* 1993; Hufschmidt & Schwaller, 1987; Huijing, 1992; Hutton, 1992; Purslow, 1989; Rack & Westbury, 1974; Wang *et al.* 1991; Wang & Ramirez-Mitchell, 1983). Stiffness results from intrinsic properties of the muscle and is not a function of reflex activation by the nervous system as static measurements of muscle stiffness reflect almost exclusively the purely elastic behaviors of the system (Howell *et al.* 1993) and can occur outside of the influence of reflex EMG activity (Condon & Hutton, 1987; Magnusson *et al.* 1995; Magnusson *et al.* 1996a; McHugh *et al.* 1992; Moore & Hutton, 1980; Taylor *et al.* 1990).

Magid and Law (1985) observed that most of the resting tension in whole skeletal muscle originated from the resting elastic tension of the myofibrils. Hill (1968) had provided evidence that in normal resting cells, a small degree of cross-bridge interaction occurs which allows muscle stiffness to rise faster and decay longer than contractile element tension in twitch, tetani and partially fused contractions in whole mammalian muscle (Bobet *et al.* 1990; Stein & Gordon, 1986), or in a stretch-release cycle of human calf muscle (Hufschmidt & Schwaller, 1987). These weak-binding cross-bridges are

thought to contribute to high frequency stiffness [resist displacement], but not to force [no active cycling] (Granzier & Wang, 1993b), which varies with both filament overlap and the magnitude of passive tension (Granzier & Wang, 1993b; Granzier & Wang, 1993a). However, recent research challenges the existence of weakly binding cross-bridges in muscle (Bagni *et al.* 1995), which instead emphasizes the role of titin as the primary source of passive tension in the sarcomere length relation (Horowitz *et al.* 1986; Horowitz & Podolsky, 1987; Wang *et al.* 1991; Granzier & Wang, 1993a; Granzier *et al.* 1996). Titin is a 'giant' protein which maintains the positional stability of myosin within the sarcomere during force production (Horowitz *et al.* 1986). Titin's segmental-extension organization (Wang *et al.* 1991) as a bi-directional spring, resists extension as well as over-shortening, to elastically restore sarcomeres to the optimal resting length (Granzier *et al.* 1996).

Within the physiological range of muscle length change, myofibrillar structures are the major source of elasticity and the sarcolemma and extracellular connective tissues begin to contribute significantly only in highly extended muscles. The extent of sarcomere elasticity has been related to the titin isoform present in the muscle (Wang *et al.* 1991; Granzier & Wang, 1993a). In overextended sarcomeres, a second tension rise at the end of the exponential passive tension curve is likely to result from the intermediate filament system (Wang *et al.* 1991) (i.e. cytoskeleton). In whole muscle, the endomysium, perimysium and epimysium collagen network prevents over-stretching of muscle fiber bundles (Purslow, 1989) producing a steep rise in passive tension near maximum muscle

extension. For an entire muscle-tendon unit around a joint, Alter (1996) identifies the soft tissue structures of joint capsule, muscle (fascia), tendon, and skin relatively contribute 47%, 41%, 10% and 2% respectively, to joint resistance.

Stretching muscle results in a phenomenon called 'stress relaxation', whereby passive resistive force to extension decays with time (Taylor *et al.* 1990). As previously presented, this is not due to the reduced activation of muscle as commonly believed, because stress relaxation occurs outside of EMG activity. Rather, stress relaxation seems to be purely mechanical in nature, further evidence being that the extent of stress relaxation is not different between people with varying degrees of flexibility (McHugh *et al.* 1992; Toft *et al.* 1989a), and is repeatable for the same subjects on the same day or different days (Halbertsma, 1994; Magnusson *et al.* 1996b; Toft *et al.* 1989b) and does not change following maximal concentric or eccentric contractions (Magnusson *et al.* 1996a). Stress relaxation has been termed visco-elastic, where both elastic (linear extension 'spring') and viscous (hydraulic 'piston') like elements contribute to tension and stiffness. The viscoelastic elements' resistance to passive extension decays with time in a single stretch (Magnusson *et al.* 1995; Magnusson *et al.* 1996a; McHugh *et al.* 1992; Toft *et al.* 1989a), or with repeated stretches. Stress relaxation is realised when the muscle is stretched to the same length and peak force at stretch onset, decays with time, or when the muscle is repeatedly stretched to the same peak force, and length increments are possible with each successive stretch (Taylor *et al.* 1990; Magnusson *et al.* 1996b). Most of the viscoelastic 'give' occurs within the first four stretches of a 10 stretch protocol (30 s per

stretch) in isolated rabbit muscle (Taylor *et al.* 1990). In human soleus, it has been shown that there are a minimum of three tissue components exhibiting viscoelastic properties, all but one element totally decays within 100 s of passive stretch. Beyond 100 s of stretch, it would be believed that ‘creep’ produces further tension decay, where the reorientation of organic tissue to more ordered arrays over time reduces resistive tension to strain (Purslow, 1989). It is interesting to note that the segmental extension of titin has recently been shown to exhibit “stress relaxation” (Kellermayer & Granzier, 1996), as was originally observed for single skinned fibers by Magid and Law (1985).

3.3.1 Acute Effects of Stretching on Muscle Stiffness

It is difficult to compare the specific effect that an acute bout of stretching has on muscle stiffness because of the differences in the literature regarding target muscles, type of stretching technique, duration of stretching, and methods used to determine stiffness. In general, an acute stretching bout can significantly increase joint ROM and significantly reduce muscle stiffness (force at a given muscle length) when measured directly after stretching (Magnusson *et al.* 1995; Magnusson *et al.* 1996a; McHugh *et al.* 1992; Toft *et al.* 1989a). Toft *et al.* (1989) reported that the relative decrease in passive tension [at each joint angle] after stretching was constant from the neutral position to the maximal extension position. Conversely, Halbertsma *et al.* (1996) report that the course of the passive stiffness curve does not change due to ten minutes of ‘sport stretching’ (i.e. typical pre-activity stretch routine), that only an increase in ‘stretch tolerance’ occurs. Halbertsma

and colleagues' methodology may have influenced this observation and subsequent conclusion, because their determination of stiffness and ROM measures occurred on the best of four stretch trials. Taylor *et al.* (1990) have demonstrated that 80% of the stress relaxation response occurs within the first four stretches of a ten stretch protocol, and Magnusson *et al.* (1996) showed that significant stress relaxation occurs in five stretches of the hamstrings, the same muscle tested by Halbertsma (1994) and Halbertsma *et al.* (1996). After a significant 'stress relaxation' response, the viscoelastic 'give' in the muscle may be optimized and the only further change in ROM that can result is from a reorientation of the connective tissue matrix, which may not further affect the 'course' of the stiffness curve as observed by Halbertsma and colleagues. Evidence for this is that data reported by Halbertsma *et al.* (1994) showed that 'significance' values for changes in passive stiffness were similar between control ($P = 0.372$) and stretched ($P = 0.410$) subjects, whereas all other indicators of the effects of stretching were highly significant in stretched subjects.

The lasting effects on muscle stiffness from a single stretching bout have not been clearly elucidated. Magnusson *et al.* (1995; 1996b) reported that a single 90 second hamstring static stretch had no effect on muscle passive torque 45 min later, and five 30 s static stretches had no lasting effect on muscle stiffness or passive torque measured one hour after the stretching, respectively. Magnusson *et al.* (1995) referred to some of their other work in which they postulated that "repeated stretches are necessary to produce lasting viscoelastic changes (i.e. that last for one hour), but the threshold number [and

duration] of stretches to produce the effect remains unknown”. In contrast, Toft *et al.* (1989a) have shown acute changes with lasting effects in passive tension in the ankle plantarflexors due to contract-relax stretching. Ninety minutes following the five stretch procedure, passive tension was significantly reduced ($P < 0.01$), and in one subject still reduced by 18%. The differing results may be related to methodology. In the studies by Magnusson *et al.* subjects resumed ‘normal daily activities, excluding exercise’. Toft *et al.* did not report the activity level of the subjects between tests, but recent studies have shown that passive tension may be elevated if subjects are able to resume ‘normal daily activities, excluding exercise’ but may remain depressed if subjects are not allowed to be active and stay positioned in the testing apparatus (Fowles, unpublished observations).

3.3.2 Chronic Effects of Flexibility Training on Muscle Stiffness

With regular stretching or ‘flexibility training’, chronic changes in flexibility are possible as indicated by increased ROM (Etnyre & Lee, 1988; Halbertsma, 1994) and reduced muscle passive tension (Toft *et al.* 1989a; Toft *et al.* 1989b). Halbertsma and colleagues also completed a training study (Halbertsma, 1994) and concluded that muscle stiffness does not change due to flexibility training, that only a greater ROM is possible because of increased ‘stretch tolerance’ (i.e. greater comfort in stretching the muscle). This conclusion may be affected by the methodology as previously noted.

It has been proposed that in athletic settings, reduced passive tension can promote greater dynamic flexibility and ‘ease of movement’, and increased ROM can allow more

time to develop force. Aside from the effects on muscle stiffness, stretching and flexibility training may also influence the muscle's ability to generate force. Granzier *et al.* (1993b) postulated that the contractile protein actin may express feedback inhibition to the production of passive tension (i.e. that contraction reduces inherent passive tension due to the passive tension-length relation). This raises speculation that there may be more factors than muscle stiffness to consider when concerned with the effects of stretching and flexibility training on athletic performance.

3.4 STRETCHING TO 'ENHANCE' MUSCLE STRENGTH?

There has been only a handful of studies that directly examined the effects of an acute stretching bout on muscle strength. DeVries (1980) refers to investigations in which stretching combined with warm-up and massage significantly improved muscle strength. Wiktorsson-Moller *et al.* (1983) combined stretching with a warm-up and evaluated maximal isometric and isokinetic (30°/s, 180°/s) concentric contractions of the quadriceps and hamstrings before and after the intervention. Warm-up and stretching significantly increased ROM by 3, 9 and 5% for hip extension, hip flexion, and knee flexion respectively, but had no significant effects on muscle strength. Ankle dorsiflexion significantly increased by 31% from the contract-relax stretching, but was not tested for strength. Specific muscle activity designed to elevate muscle temperature and focus the athlete for sport (i.e. warm-up) is also believed to increase contractile performance which could confound the results of the prior studies.

A recent abstract by Kokkonen *et al.* (1996) showed that twenty minutes of static stretching of the hip, thigh, and calf muscles significantly decreased 1 RM performance measured ten minutes following the stretching. Nelson *et al.* (1996) evaluated maximal vertical force and kinetic energy (ground reaction force) in squat jump and countermovement jumps after stretching. Nelson and colleagues observed that jumping performance was significantly reduced, caused by an alteration of the body's net force production. The comparison of the two recent studies to the earlier ones would seem to

indicate that intense stretching without warm-up may decrease maximal force production whereas stretching with warm-up may have no effect or possibly even enhance force production.

Two studies have shown that flexibility training combined with resistance training increases sprint running performance (Dintiman, 1964) and rebound bench press performance (Wilson *et al.* 1992). Hortobagyi *et al.* (1985) combined slow static stretching with ROM exercise in young runners and observed increased speed characteristics but did not observe an improvement in MVC. Worrell *et al.* (1994) showed significantly increased peak torque at selective isokinetic eccentric and concentric velocities after flexibility training. However, each of these studies can be questioned as to the specific effect of flexibility training alone on strength performance, because of methodological concerns such as reliability of measures, lack of control groups and confounding effects of other training.

3.4.1 Acute effects of Stretching on Contractile Performance

Many factors may influence muscle strength directly following stretching. It has already been presented that stretching can reduce muscle stiffness and increase ROM. Reduced muscle stiffness may affect evoked muscle twitch amplitude and shape because of greater time needed to 'take-up slack' in compliant structures (Caldwell, 1995). Greater 'slack' is unlikely to affect measured peak voluntary contraction torque, because stiffness reaches maximum values in maximum contractions. Recent work with isolated myosin

molecules indicates the frequency of binding and force of the power stroke in active cross-bridge cycling may be influenced by the compliance of titin (Granzier, 1996; personal communication). This finding may be relevant to force generation in evoked twitch or maximum contractions.

It has been postulated in fatigue research that there are a number of steps from activation to cross-bridge cycling that can influence force production. Some of these steps may be influenced by stretching, although passive stretching occurs without 'fatigue' in the classic sense (i.e. without active cross-bridge cycling). There is very little research specifically on factors that may influence measured force in twitch or maximum contractions as affected by stretching. Most research employs stretching as an intervention in physical therapy and rehabilitation or as a control for research into the mechanisms of muscle damage, and has not been the focus of research into contractile performance in an athletic context.

Previous reports indicate that the evoked resting twitch can either be potentiated (Snowdowne, 1986) or attenuated (Armstrong *et al.* 1993) following passive stretch (PS). Snowdowne (1986) elicited a twitch directly after a brief single stretch in isolated muscle fibers. Armstrong *et al.* (1993) observed a 61% decrease in twitch force after a 2h stretch of rat soleus. Differing results are probably due to the different protocols of the two studies; however, both studies observed alteration of Ca^{+2} homeostasis. Passive stretch of muscle is known to increase intracellular Ca^{+2} concentration progressively with the degree of stretch (Snowdowne, 1986). Ca^{+2} influx can originate from the extracellular space

(Armstrong *et al.* 1993) or more likely by release from the sarcoplasmic reticulum. Strain may disrupt the mechanical link of the dihydropyridine-ryanodine complex involved in excitation-contraction coupling in skeletal muscle (McComas, 1996), to open 'the plug' and allow Ca^{+2} to escape from the sarcoplasmic reticulum down its concentration gradient into the cytosol. After an acute bout of passive stretch the contractile characteristics of maximal tetanic tension, rate of tetanic force development (RFD) and peak passive force were significantly depressed one hour following PS on rat hind limb muscles (Lieber *et al.* 1991).

3.4.2 Chronic effects of Flexibility Training on Contractile Performance

Few studies have examined the effect of a single bout of stretching on contractile performance following the bout, and fewer have examined the effect of flexibility training on contractile performance. Hortobagyi *et al.* (1985) combined slow static stretching with ROM exercise in young runners and observed increased stride frequency, increased isometric rate of force development, and increased speed of contractions at low loads. The Hortobagyi study did not compare the trained group to a control group so the 'ROM exercise' combined with other running could have resulted in a neural adaptation that increased performance in speed parameters, as neural adaptations can increase isometric rate of force development with training (Sale, 1992).

The study by Wilson *et al.* (1992) described enhancement of rebound bench press velocity and work after 8 weeks of flexibility training. Wilson's study also showed increased maximum strength due to flexibility training, which may have been influenced by

the end range resistance exercise performed by the flexibility training group. The novel training stress could possibly have resulted in hypertrophy, or a chronic change in muscle length. Alway (1994) observed that chronic passive stretch (30 days of limb-weighting) in chicken anterior latissimus dorsi muscles increases muscle length and mass and maximal force. Stretch hypertrophy models have been successful in producing increases in muscle mass in animals but have been criticized as not simulating human strength training (Antonio & Gonyea, 1993). Despite drastically different training stimuli, Alway's work provides indirect support that flexibility training could result in chronic changes in muscle length, size and architecture. Architectural changes have been proposed to lower specific tension in hypertrophied muscle (Kawakami *et al.* 1995), which may also influence other contractile characteristics. Alway (1994) also reported lowered specific tension in stretch-hypertrophied muscle. The results of Wilson *et al.* (1992) may be a result of a chronic increase in muscle length which could affect the strength curve for the bench press action and greatly affect measured forces (Murphy *et al.* 1995). There may also have been a neural adaptation to high resistance training in end range motions which allowed greater activation early in the movement (i.e. in the 'stretched' phase of the lift), although muscle EMG was not measured to determine this. Altering the strength curve of a muscle by changing neural drive or muscle architecture, either acutely or chronically, may have implications to motor learning and specificity of training. These topics remain to be investigated.

4.0 SUMMARY AND CONCLUSIONS

Despite the need for well controlled research on the role of flexibility training and stretching in athletics, little research has been done. Published work indicates that both acute and chronic changes in muscle stiffness and ROM are possible with stretching and flexibility training; however, the impact on performance directly following stretching or after training remains uncertain. Recent evidence implies that a single stretching bout may actually be detrimental to performance, because of impaired contractile ability. Further research is needed to confirm this observation; however, any conclusions made about the possible detrimental effects to performance must be weighed heavily against the empirical evidence supporting stretching to prevent injury in athletic competition.

REFERENCES

- ALTER, M. J. (1996). *Science of Flexibility*. Champaign, IL: Human Kinetics.
- ALWAY, S. E. (1994). Force and contractile characteristics after stretch overload in quail anterior latissimus dorsi muscle. *Journal of Applied Physiology* **77**, 135-141.
- ANDERSON, B. (1984). Flexibility. *National Strength and Conditioning Association Journal* **6**, 10-22-71-73.
- ANTONIO, J. & GONYEA, W. J. (1993). Skeletal muscle fiber hyperplasia. *Medicine & Science in Sports and Exercise* **25**, 1333-1345.
- ARMSTRONG, R. B., DUAN, C., DELP, M. D., HAYES, D. E., GLENN, G. M. & ALLEN, G. D. (1993). Elevations in rat soleus muscle $[Ca^{2+}]$ with passive stretch. *Journal of Applied Physiology* **74**, 2990-2997.
- AWAD, A. & KOTKE, F. (1964). Effectiveness of myotatic reflex facilitation in augmenting rate of increase in muscular strength due to brief maximum exercise. *Archives of Physical Medicine & Rehabilitation* **47**, 23-29.
- BAGNI, M. A., CECCHI, G., COLOMO, F. & GARZELLA, P. (1995). Absence of mechanical evidence for attached weakly binding cross-bridges in frog relaxed muscle fibres. *Journal of Physiology* **482**, 391-400.
- BEAULIEU, J. E. (1981). Developing a stretching program. *Physician and Sportsmedicine* **9**, 59-69.
- BEAULIEU, J. E. (1984). Flexibility. *National Strength and Conditioning Association Journal* **6**, 10-22-71-73.
- BLOOMFIELD, J., ACKLAND, T. R. & ELLIOTT, B. C. (1994). *Applied Anatomy and Biomechanics in Sport*. Melbourne: Blackwell Scientific Publications.
- BOBET, J., STEIN, R. B. & OGUZTORELI, M. N. (1990). Mechanisms relating force and high-frequency stiffness in skeletal muscle. *Journal of Biomechanics* **23**, 13-21.
- CALDWELL, G. E. (1995). Tendon elasticity and relative length: effects on the Hill two-component muscle model. *Journal of Applied Biomechanics* **11**, 1-24.

CONDON, S. M. & HUTTON, R. S. (1987). Soleus muscle electromyography activity and ankle dorsiflexion range of motion during four stretching procedures. *Physical Therapy* **67**, 24-30.

CORNELIUS, W. L. (1989). Two effective flexibility methods. *National Strength and Conditioning Association Journal* **11**, 61-62.

CORNELIUS, W. L. (1984). Flexibility. *National Strength and Conditioning Association Journal* **6**, 10-22-71-73.

CRAIB, M. W., MITCHELL, V. A., FIELDS, K. B., COOPER, T. R., HOPEWELL, R. & MORGAN, D. W. (1996). The association between flexibility and running economy in sub-elite male distance runners. *Medicine & Science in Sports and Exercise* **28**, 737-743.

CURETON, T. K. (1941). Flexibility as an aspect of physical fitness. *Research Quarterly for Exercise and Sport* **12**, 381-294.

DEVRIES, H. A. (1963). The "looseness" factor in speed and O₂ consumption of an anaerobic 100 yard dash. *Research Quarterly for Exercise and Sport* **34**, 305-313.

DEVRIES, H. A. (1980). *Physiology of Exercise for Physical Education and Athletics*. Dubuque, Iowa: W.C. Brown.

DINTIMAN, G. B. (1964). Effects of various training programs on running speed. *Research Quarterly for Exercise and Sport* **35**, 456

EDMAN, K. A. P. (1988). Double-hyperbolic force-velocity relation in frog muscle fibres. *Journal of Physiology* **404**, 301-321.

ETNYRE, B. R. & LEE, E. J. (1988). Chronic and acute flexibility of men and women using three different stretching techniques. *Research Quarterly for Exercise and Sport* **59**, 222-228.

FLECK, S. J. & KRAEMER, W. J. (1987). *Designing Resistance Training Programs*. Champaign, Illinois: Human Kinetics Books.

FORD, L. E., HUXLEY, A. F. & SIMMONS, R. M. (1981). The relation between stiffness and filament overlap in stimulated frog muscle fibres. *Journal of Physiology* **311**, 219-249.

FOX, E. L., BOWERS, R. W. & FOSS, M. L. (1989). *The Physiological Basis of Physical Education and Athletics*. Dubuque, Iowa: Wm. C. Brown Publishers.

- GALLER, S., SCHMITT, T. L. & PETTE, D. (1994). Stretch activation, unloaded shortening velocity, and myosin heavy chain isoforms of rat skeletal muscle fibres. *Journal of Physiology* **478**, 513-521.
- GORDON, A. M., HUXLEY, A. F. & JULIAN, F. J. (1966). The variation in isometric tension with sarcomere length in vertebrate muscle fibers. *Journal of Physiology* **184**, 170-192.
- GRANZIER, H. L. M., HELMES, M. & TROMBITAS, K. (1996). Nonuniform elasticity of titin in cardiac myocytes: a study using immunoelectron microscopy and cellular mechanics. *Biophysical Journal* **70**, 430-442.
- GRANZIER, H. L. M. & WANG, K. (1993a). Passive tension and stiffness of vertebrate skeletal and insect flight muscles: the contribution of weak cross-bridges and elastic filaments. *Biophysical Journal* **65**, 2141-2159.
- GRANZIER, H. L. M. & WANG, K. (1993b). Interplay between passive tension and strong and weak binding cross-bridges in insect indirect flight muscle. *Journal of General Physiology* **101**, 235-270.
- HALBERTSMA, J. P. K. (1994). Stretching exercises: effect on passive extensibility and stiffness in short hamstrings of healthy subjects. *Archives of Physical Medicine & Rehabilitation* **75**, 976-981.
- HALBERTSMA, J. P. K., VAN BOLHUIS, A. I. & GOEKEN, L. N. H. (1996). Sport stretching: Effect on passive muscle stiffness of short hamstrings. *Archives of Physical Medicine & Rehabilitation* **77**, 688-692.
- HARRIS, M. L. (1969). Flexibility. *Physical Therapy* **49**, 591-601.
- HEDRICK, A. (1993). Flexibility and the conditioning program. *National Strength and Conditioning Association Journal* **15**, 62-66.
- HILL, D. K. (1968). Tension due to interaction between the sliding filaments in resting striated muscle. The effect of stimulation. *Journal of Physiology* **199**, 637-684.
- HOLT, L. E., TRAVIS, T. M. & OKITA, T. (1970). Comparative study of three stretching techniques. *Perceptual and Motor Skills* **31**, 611-616.
- HOROWITS, R., KEMPNER, E. S., BISHER, M. E. & PODOLSKY, R. J. (1986). A physiological role for titin and nebulin in skeletal muscle. *Science* **323**, 160-164.

- HOROWITS, R. & PODOLSKY, R. J. (1987). The positional stability of thick filaments in activated skeletal muscle depends on sarcomere length: evidence for the role of titin filaments. *The Journal of Cell Biology* **105**, 2217-2223.
- HORTOBAGYI, T., FALUDI, J., TIHANYI, J. & MERKELY, B. (1985). Effects of intense "stretching"-flexibility training on the mechanical profile of the knee extensors and on the range of motion of the hip joint. *International Journal of Sports Medicine* **6**, 317-321.
- HOWELL, J. N., CHLEBOUN, G. & CONATSER, R. (1993). Muscle stiffness, strength loss, swelling and soreness following exercise-induced injury in humans. *Journal of Physiology* **464**, 183-196.
- HUFSCHMIDT, A. & SCHWALLER, I. (1987). Short-range elasticity and resting tension of relaxed human lower leg muscles. *Journal of Physiology* **391**, 451-465.
- HUIJING, P. A. (1992). Mechanical muscle models. In *Strength and Power in Sport*, ed. KOMI, P. V. pp. 130-150. Oxford: Blackwell Scientific Publications.
- HUTTON, R. S. (1992). Neuromuscular basis of stretching exercises. In *Strength and Power in Sport*, ed. KOMI, P. V. pp. 29-38. Oxford: Blackwell Scientific Publications.
- KAWAKAMI, Y., ABE, T., KUNO, S. Y. & FUKUNAGA, T. (1995). Training-induced changes in muscle architecture and specific tension. *European Journal of Applied Physiology* **72**, 37-43.
- KELLERMAYER, M. S. & GRANZIER, H. L. M. (1996). Elastic properties of single titin molecules made visible through fluorescent F-actin binding. *Biochemical & Biophysical Research Communications* **221**, 491-497.
- KOKKONEN, J. & NELSON, A. G. (1996). Acute stretching exercises inhibit maximal strength performance. *Medicine & Science in Sports and Exercise* **28**, S190(Abstract)
- KOMI, P. V. (1992). Stretch-shortening cycle. In *Strength and Power in Sport*, ed. KOMI, P. V. pp. 169-179. Oxford: Blackwell Scientific Publications.
- LIEBER, R. L., WOODBURN, T. M. & FRIDEN, J. (1991). Muscle damage induced by eccentric contractions of 25% strain. *Journal of Applied Physiology* **70**, 2498-2507.
- LIEBER, R. L. & FRIDEN, J. (1993). Muscle damage is not a function of muscle force but active muscle strain. *Journal of Applied Physiology* **74**, 520-526.

MAGID, A. & LAW, D. J. (1985). Myofibrils bear most of the resting tension in frog skeletal muscle. *Science* **230**, 1280-1282.

MAGNUSSON, S. P., SIMONSEN, E. B., AAGAARD, P., MORITZ, U. & KJAER, M. (1995). Contraction specific changes in passive torque in human skeletal muscle. *Acta Physiologica Scandinavica* **155**, 377-386.

MAGNUSSON, S. P., SIMONSEN, E. B., AAGAARD, P., DYHRE-POULSEN, P., MCHUGH, M. P. & KJAER, M. (1996a). Mechanical and physiological responses to stretching with and without preisometric contraction in human skeletal muscle. *Archives of Physical Medicine & Rehabilitation* **77**, 373-378.

MAGNUSSON, S. P., SIMONSEN, E. B., AAGAARD, P. & KJAER, M. (1996b). Biomechanical responses to repeated stretches in human hamstring muscle in vivo. *The American Journal of Sports Medicine* **24**, 622-628.

MCHUGH, M. P., MAGNUSSON, S. P., GLEIM, G. W. & NICHOLAS, J. A. (1992). Viscoelastic stress relaxation in human skeletal muscle. *Medicine & Science in Sports and Exercise* **24**, 1375-1382.

MOORE, M. A. & HUTTON, R. S. (1980). Electromyographic investigation of muscle stretching techniques. *Medicine & Science in Sports and Exercise* **12**, 322-329.

MURPHY, A. J., WILSON, G. J., PRYOR, J. F. & NEWTON, R. U. (1995). Isometric assessment of muscular function: the effect of joint angle. *Journal of Applied Biomechanics* **11**, 205-215.

NELSON, A. G., CORNWELL, A. & HEISE, G. D. (1996). Acute stretching exercises and vertical jump stored elastic energy. *Medicine & Science in Sports and Exercise* **28**, S156(Abstract)

PARTRIDGE, M. (1954). Electromyographic demonstration of facilitation. *Physical Therapy Review* **5**, 227

PRENTICE, W. E. (1984). Flexibility. *National Strength and Conditioning Association Journal* **6**, 10-22-71-73.

PURSLOW, P. P. (1989). Strain-induced reorientation of an intramuscular connective tissue network: implication for passive muscle elasticity. *Journal of Biomechanics* **22**, 21-31.

RACK, P. M. H. & WESTBURY, D. R. (1974). The short range stiffness of active mammalian muscle and its effects on mechanical properties. *Journal of Physiology* **240**, 331-350.

SALE, D. G. (1992). Neural adaptation to strength training. In *Strength and Power in Sport*, ed. KOMI, P. V. pp. 249-265. Oxford: Blackwell Scientific Publications.

SHELLOCK, F. G. & PRENTICE, W. E. (1985). Warming-up and stretching for improved physical performance and prevention of sports-related injuries. *Sports Medicine* **2**, 267-278.

SMITH, C. A. (1994). The warm-up procedure: to stretch or not to stretch. A brief review. *Journal of Orthopaedic & Sports Physical Therapy* **19**, 12-17.

SNOWDOWNE, K. W. (1986). The effect of stretch on sarcoplasmic free calcium of frog skeletal muscle at rest. *Biochimica et Biophysica Acta* **862**, 441-444.

STEIN, R. B. & GORDON, T. (1986). Nonlinear stiffness-force relationships in whole mammalian skeletal muscles. *Canadian Journal of Physiology and Pharmacology* **64**, 1236-1244.

TAYLOR, D. C., DALTON, J. D., SEABER, A. V. & GARRETT, W. E. J. (1990). Viscoelastic properties of muscle-tendon units. The biomechanical effects of stretching. *The American Journal of Sports Medicine* **18**, 300-309.

TOFT, E., ESPERSEN, G. T., KALUND, S., SINKJAER, T. & HORNEMANN, B. C. (1989a). Passive tension of the ankle before and after stretching. *The American Journal of Sports Medicine* **17**, 489-494.

TOFT, E., SINKJAER, T., KALUND, S. & ESPERSEN, G. T. (1989b). Biomechanical properties of the human ankle in relation to passive stretch. *Journal of Biomechanics* **22**, 1129-1132.

WALLACE, L. (1984). Flexibility. *National Strength and Conditioning Association Journal* **6**, 10-22-71-73.

WANG, K., MCCARTER, R., WRIGHT, J., BEVERLY, J. & RAMIREZ-MITCHELL, R. (1991). Regulation of skeletal muscle stiffness and elasticity by titin isoforms: A test of the segmental extension model of resting tension. *Proceedings of the National Academy of Science USA* **88**, 7101-7105.

WANG, K. & RAMIREZ-MITCHELL, R. (1983). A network of transverse and longitudinal intermediate filaments is associated with sarcomeres of adult vertebrate skeletal muscle. *The Journal of Cell Biology* **96**, 562-570.

WIKTORSSON-MOLLER, M., OBERG, B., EKSTRAND, J. & GILLQUIST, J. (1983). Effects of warming up, massage, and stretching on range of motion and muscle strength in the lower extremity. *The American Journal of Sports Medicine* **11**, 249-252.

WILSON, G. J., WOOD, G. A. & ELLIOTT, B. C. (1991). The relationship between stiffness of the musculature and static flexibility: an alternative explanation for the occurrence of muscular injury. *International Journal of Sports Medicine* **12**, 403-407.

WILSON, G. J., ELLIOTT, B. C. & WOOD, G. A. (1992). Stretch shorten cycle performance enhancement through flexibility training. *Medicine & Science in Sports and Exercise* **24**, 116-123.

WILSON, G. J., MURPHY, A. J. & PRYOR, J. F. (1994). Musculotendinous stiffness: its relationship to eccentric, isometric, and concentric performance. *Journal of Applied Physiology* **76**, 2714-2719.

WORRELL, T. W., SMITH, T. L. & WINEGARDNER, J. (1994). Effect of hamstring stretching on hamstring muscle performance. *Journal of Orthopaedic & Sports Physical Therapy* **20**, 154-159.

CHAPTER II

REDUCED STRENGTH FOLLOWING PASSIVE STRETCH OF THE HUMAN PLANTARFLEXORS

ABSTRACT

The purpose of this study was to assess strength performance following an acute bout of maximally tolerable passive stretch (PSmax). The ankle plantarflexors of ten university students (6 men, 4 women) underwent 30 min of cyclical PSmax (13 stretches over 33 min) and a similar control period (Con) of no stretch. Isometric maximum voluntary contraction torque (MVC), interpolated twitch torque (ITT) [to assess motor unit activation] (MUA), peak twitch torque (PTT) and twitch contractile properties were assessed at 10° of dorsiflexion (D) pre (PRE), immediately post (POST) and at 5, 15, 30, 45, and 60 min after PSmax or Con. EMG was measured for MVC's (AEMG) and twitch (M-wave amplitude) contractions. Muscle stiffness, as indicated by mean passive torque of three joint angles (0°, 10°, and 20°D), was measured at each time point.

Compared to PRE, MVC was decreased POST (28%), and at 5 (21%), 15 (13%), 30 (12%), 45 (10%), and 60 (9%) min after PSmax ($P < 0.005$). MUA decreased from 97% at PRE to 81% at POST and 84% at 5 min after PSmax ($P < 0.001$), but had recovered (NS) to 93% at 15, 96% at 30, 95% at 45, and 95% at 60 min. PTT decreased

POST (18%) and only recovered to 84% of PRE at 60 min ($P < 0.0005$). M-wave amplitude decreased POST (9.1%, $P < 0.005$), was not different at 15 min, but was greater than PRE at 30 (7%), 45 (10%, $P < 0.05$) and 60 (12%, $P < 0.005$) min after PSmax. MVC AEMG showed a similar pattern to the M-wave. The only significant changes in the Con condition was a reduced PTT (9%) in post time points ($P < 0.005$). Muscle stiffness was significantly reduced POST (27%) and 15 min (14%) ($P < 0.0005$) but was restored to non-significantly different values by 30 min (8%, $P = 0.08$). An additional PSmax trial confirmed that the twitch torque-joint angle relation was temporarily altered at POST only. MVC measurements made at 30 min in the second trial, failed to exhibit any alteration of the torque-joint angle relation.

These data indicate that PSmax decreases voluntary strength for up to one hour after passive stretch, as a result of both impaired MUA and impaired contractile force in the early phase of deficit, and by impaired contractile force throughout the entire period of deficit. Contractile performance may be affected by reduced muscle stiffness in periods following PSmax.

1.0 INTRODUCTION

Stretching of various skeletal muscle groups before activity or as part of an athlete training program is commonly believed to enhance muscular performance (Bloomfield *et al.* 1994; Fox *et al.* 1989; Hedrick, 1993; Smith, 1994). However, there have been very few well controlled studies of the influence of stretching or flexibility training on performance. Some earlier studies have shown that pre-activity stretching, when combined with adequate warm-up, increases muscular strength (deVries, 1980) or has no effect on strength (Wiktorsson-Moller *et al.* 1983).

Two studies have shown that flexibility training combined with resistance training increases sprint running performance (Dintiman, 1964) and rebound bench press performance (Wilson *et al.* 1992). Hortobagyi *et al.* (1985) combined slow static stretching with ROM exercise in young runners and observed increased stride frequency, increased isometric rate of force development, and increased speed of contractions at low loads, but did not observe an improvement in MVC. Worrell *et al.* (1994) showed significantly increased peak torque at two eccentric velocities (60 °/s & 180°/s) and one of two concentric velocities (180°/s) following hamstring stretching, even though no significant increase in hamstring flexibility was achieved.

Pre-activity warm-up is also advocated to increase performance (Noonan *et al.* 1993; Shellock & Prentice, 1985). Warm-up is a routine designed to increase muscle

blood flow, muscle temperature, and focus the athlete to the activity to be performed. Therefore, the specific effects of stretching are difficult to identify when earlier studies combine pre-activity stretching with warm-up, or combined flexibility training with other training. Two recent reports indicate that just stretching without warm-up prior to activity compromises maximum voluntary force (Kokkonen & Nelson, 1996; Nelson *et al.* 1996). This observation was confirmed in our laboratory; maximal passive stretch of the ankle plantarflexors significantly impaired contractile ability directly following the stretch procedure, was nearly recovered by 1 h post, and fully recovered by 24 h (Fowles, unpublished observations - appendix 1). A reduction in maximum voluntary force with stretching may actually be detrimental to strength performance in sports requiring maximal strength and thus, should be considered when designing pre-competition routines.

The purpose of the present research was to assess the effects of one hour of maximally tolerable passive stretch on voluntary strength and contractile performance in human subjects. To determine the possible contributors to contractile effects, two studies were designed to show the time course of response within 1 h following the bout, and to control for changes in muscle length due to the stretch procedure. The hypothesis was that an acute bout of maximal passive stretch compromises maximum isometric contractile force directly following the bout and has a rapid time-course for recovery. Whereas earlier studies can be questioned as to the specific role of stretching or flexibility training as a separate intervention to affect maximal strength performance, the present study was

controlled so that stretching was identified as the variable causing a decrease in contractile performance following the bout.

2.0 METHODS

2.1 SUBJECTS

Eight men (means (SD; age, 22.3 (2.2 years; mass, 71.4 (9.3 kg; height, 175.9 (3.6 cm) and four women (age, 20.3 (0.2 years; mass, 55.0 (3.5 kg; height, 166.8 (1.9 cm) with a background of physical activity and no history of injury or abnormality affecting the ankle joint, were recruited for the study. All subjects completed two trials in one experiment and one trial in a second experiment. The order of trials for experiment 1 (Exp 1) was randomized, and all subjects completed the single trial in experiment 2 (Exp 2) at least three weeks after completion of Exp 1. The leg tested was the same for all three trials for a single subject. Informed, written consent was obtained from each subject before participating in the experiment. The study carried the approval of McMaster University's Human Ethics Committee.

Two male subjects completed all trials of the experiment but were removed from analysis because EMG above the criterion threshold was detected during the passive stretch protocols. Therefore, data were collected on 12 subjects and analyzed for only 10 subjects (6 males, 4 Females). There was no intention to compare gender differences in response to the passive stretch protocol.

2.2 EXPERIMENTAL DESIGN

The objective of the study was to determine the effects of maximal passive stretch (PSmax) on contractile performance of the human plantarflexors. To determine the possible contributors to contractile effects following PSmax, two experiments were designed. In experiment one (Exp 1) the time course for alterations in contractile response following PSmax was examined and compared to a no-stretch control condition. Exp 1 involved measuring passive stiffness, evoked twitch and isometric maximum voluntary contractions (MVC) at a number of time points within one hour following the intervention. A second experiment (Exp 2) involving PSmax was completed with the same subjects to assess contractile performance at different muscle lengths following the stretch procedure. Contractile performance was assessed at three joint angles in Exp 2 as opposed to the single testing angle of Exp 1. Testing at three angles was designed to control for contractile performance alterations due to any changes in the muscle force-length relation following PSmax. Because the time course for maximum voluntary force recovery from PSmax was mapped in Exp 1, and it was determined that POST MVC measures were affected by reduced activation in Exp 1, MVC measures were only performed at 30 min and at 60 min following the stretch in Exp 2. This design kept the total number of MVC's approximately the same for Exp 1 and Exp 2.

2.2.1 Experiment 1 - Time Course for Neuromuscular Response

Following three days rest from strenuous activity with the lower legs, subjects underwent either a maximal passive stretch (PSmax) or a neutral ankle angle control (Con) protocol. Protocol order and experimental leg were randomly assigned. The general experimental procedure was as follows: pre-exercise measures (PRE), 10 min rest period, the PSmax or Con protocol, and contractile measures at immediately post (POST), and post + 5 mins, +10 min, +15 min, +30 min, +45 min, and +60 min. Resting twitches were omitted at the 5 min time point, because of the confounding effects of post-activation potentiation (Vandervoort *et al.* 1983). A minimum of three days after the first trial, subjects returned to the lab to perform the remaining protocol, using the same leg that performed the previous trial.

The testing protocol for the Con trial in Exp 1 was performed identically to the PSmax protocol (see above) with the exception that no stretch of the ankle plantarflexors occurred, but rather the ankle was kept in a resting joint angle position selected by the subject ($\sim 10^\circ\text{P}$). During the Con trial the subject was secured into the apparatus with the same tension on the supports and velcro straps and for the same total duration as during the PSmax trial. The testing protocol is outlined in Table 1.

Table 1. Testing Protocol for Experiments 1 and 2

Subjects completed PRE and post testing on the same experimental day

Experiment 1 (Exp 1) was performed with two trials separated by a minimum of three days rest;

Subjects randomly completed either maximal passive stretch (PSmax) or neutral ankle angle control (Con)

Experiment 2 (Exp 2) was performed with a single PSmax intervention.

Exp 1

	PRE			POST	5 min	15 min	30 min	45 min	60 min
(practice two days prior to testing*)	PasTor	10 min rest		Twitch	MVC	PasTor	PasTor	PasTor	PasTor
	Twitch		30 min of	PasTor	PasTor	Twitch	Twitch	Twitch	Twitch
	MVC		PSmax	MVC		MVC	MVC	MVC	MVC
	PasTor		or Con			PasTor	PasTor	PasTor	PasTor
	MVC								

Exp 2

	PRE			POST		15 min	30 min	45 min	60 min
**30 min rest	PasTor	10 min rest		3 Twitches		PasTor	PasTor	PasTor	PasTor
	Twitch			PasTor	(no 5 min testing)	3 Twitches	3 Twitches	3 Twitches	3 Twitches
	3 Twitches		PSmax			Pas Tor	3 MVC's	PasTor	3 MVC's
	3 MVCs						PasTor		PasTor
	Pas Tor								

PasTor, Passive Torque assessment in order from 0°, 10°, to 20°D; Twitch, Evoked twitch at 10°D; MVC, Maximum voluntary contraction at 10°D; 3 Twitches, Evoked twitches at 0°, 10°, and 20°D; 3 MVCs, Maximum voluntary contractions at 0°, 10°, 20 °D.

* Subjects came into the lab two days prior to testing day only for Exp 1, to practice MVCs and to be accommodated to the stimulations.

** Subjects entered the lab and rested in the sitting position for 30 min prior to any testing to limit any potentiation effects.

2.2.2 Experiment 2 - Relative Joint Angle Assessment

Approximately three weeks following completion of Exp 1, subjects returned to the lab to complete the relative joint angle trial on the same leg that performed Exp 1. The results of Exp 1 revealed that contractile and stiffness measures are very stable in the Con condition over the post measures of the experiment. Therefore, a control trial was not included in Exp 2. The testing protocol was similar to the PSmax trial of Exp 1 with a few modifications.

1) A 30 min rest interval was placed at the beginning of the testing protocol for Exp 2, prior to PRE. The Con twitch results of Exp 1 indicated that subjects may have exhibited lingering potentiation in the PRE measures, following a walk to the lab for testing.

2) Evoked twitch and voluntary MVC's were collected at three joint angles (0°D , 10°D , 20°D). Joint angle order was randomized between subjects using a Latin Square. Two subjects performed each of the six possible combinations of test order for the total of twelve subjects tested. The joint angle testing order was the same for twitch and MVC contractions in a single subject. Two subjects were disqualified from analysis so that in the analysis, two of the test order combinations had only single subjects while the remaining four combinations had two subjects each.

3) MVC measurements were made at 30 min post and 60 min post only, to avoid confounding effects of decreased motor unit activation on the voluntary MVC's in brief

time frames following PSmax (see results - Exp 1). Two minutes of recovery were given between each MVC. An extra five minutes was added to post testing to avoid the confounding effects of potentiation on twitch contractile properties following the MVC measurements at 30 min. Therefore, the 45 min and 60 min measures were actually at 50 min and 65 min post PSmax. However, no 'resting recovery' from PSmax was assumed to occur while successive MVC's were being performed, so for ease of presentation, the 45 min and 60 min time points remain to indicate those post testing time points. The total number of MVC's performed during Exp 2 was one more than the total number for either protocol of Exp 1 (one extra MVC at PRE in Exp 2).

Twitches were recorded at POST, 15 min, 30 min, 45 min, and 60 min, and prior to the MVC's at 30 min and 60 min. Approximately 20 s of recovery was given between successive twitch measurements.

2.3 APPARATUS

Experiments were performed on the triceps surae muscle group which includes the soleus and gastrocnemius. By having the knee joint set at 90° , the gastrocnemius, which crosses the knee joint and therefore was at a more shortened length, bears less of the load imposed in the stretch and contributes less force during active plantarflexion than soleus (Fugl-Meyer *et al.* 1979; Herman & Bragin, 1967; Sale *et al.* 1982). A leg holder device described by Marsh *et al.* (1981) and employed by Sale *et al.* (1982) was used for all testing and P_{Smax} measures. When in the apparatus the subject was positioned so that the knee and hip angles were at 90° . Ankle movement is limited to 48° of either dorsiflexion (D) or plantarflexion (P) from the midposition of a 90° ankle angle to the tibia (or 0° D). Subjects did not wear shoes, and were firmly secured with forefoot velcro straps and anterior tibial and femur compression supports. It was not appropriate to leave the subject bound in the apparatus for the entire duration of the trial (~2.5 hrs). Therefore, subjects were freed from the apparatus constraints after the 5 min time point, and between each successive time point for the subjects' comfort. The compression supports were marked during pre-testing so that the apparatus was set to the same compression for each test.

The axis shaft was aligned with the axis of rotation of the ankle through the medial malleolus. Strain gauges at the axis shaft of the translated pressure to the metal foot plates into a torque signal. The torque signal from the boot apparatus was amplified (Honeywell

Accudata 143 bridge amplifier), converted to a digital signal, and fed into a 12 bit A/D converter (Dataq Electronics) and then into an IBM computer for on-line analysis. Coda data acquisition software (Dataq Electronics) was used to process the data.

2.4 MAXIMAL PASSIVE STRETCH (PSmax)

The PSmax protocol was as follows: without prior warm-up or stretching, the subject's leg was secured in the device and pre-tested. Following a ten minute rest interval, the plantarflexors were passively stretched by the experimenter to the maximum possible dorsiflexed position achievable, without pain. The joint angle was then locked into place and every 2 min and 15 s, the ankle joint was released to 10° for 5 s, then manually passively stretched over 5-10 s at $\sim 2^\circ/\text{s}$ to a new maximal joint angle as limited by the tolerance of the subject. Torque was zeroed between each stretch to eliminate the effects of drift from the torque transducer. A total of 13 maximal stretches was imposed in 33 min. (i.e., 30 min of time under stretch). Subjects were given visual feedback of torque and EMG activity during the stretch protocol. Maximum joint angle achieved with each stretch during the protocol was visually read by the experimenter from the apparatus (angle (0.25°)). Post-testing began directly after cessation of PSmax and for time points up to one hour.

2.5 TESTING AND MEASUREMENTS

Two days prior to testing, subjects were familiarized with performing MVCs and became accommodated to the stimulation protocol. It has been reported that 100% voluntary activation is difficult with the plantarflexors (Belanger & McComas, 1981) but with practice, full activation is achievable (Bigland-Ritchie *et al.* 1986b). All subjects performed a minimum of five MVC's in the orientation session, and received electrical stimulation a minimum of ten times. One brief submaximal stretch during the orientation was performed to demonstrate the passive stretch procedure.

Testing was conducted on only the leg used for the trials. All testing measures in a single trial were recorded on the same day, without modification or adjustment to the electrode arrangement. Each subject completed the three trials at approximately the same time of day, to account for possible variance in strength and muscle stiffness throughout a day. Testing measures included: evoked twitch contractile properties, isometric MVC with interpolated twitch, and muscle stiffness measures. EMG was recorded for twitch and MVC measures. Passive torque and EMG were monitored during the PSmax protocols.

Isometric MVC

For MVC measurements, subjects sat in the testing apparatus with hands folded at their waist. Subjects performed an isometric MVC as forcefully as possible. The MVC was held for three to five seconds; an interpolated stimulus was delivered after approximately

two seconds when a plateau in the torque trace was clearly visible to the tester. Because of the number of MVC's performed in the experimental protocols, only single MVC's were performed for each time point except in the PRE measurements for Exp 1, where the best of two MVC's was taken. Motor unit activation (MUA) was calculated from the interpolated twitch torque values using the methods of Belanger & McComas (1981).

Evoked Isometric Twitch Contractile Properties

Twitch contractions were evoked by percutaneous electrical stimulation. The stimulating electrodes were lead plates, wrapped in gauze and impregnated with conducting gel. The cathode (2 cm x 3 cm) was positioned in the popliteal fossa overlying the posterior tibial nerve. The anode (1.5 cm x 1.5 cm) was positioned at the motor point for the soleus; along the medial line directly below the belly of the medial and lateral gastrocnemius muscles. Skin over the stimulation sites was abraded and cleaned with isopropyl alcohol pads.

Contractile measures of resting twitch and MVC with interpolated twitch were assessed at 10 degrees of dorsiflexion (10°D) for Exp 1. Ten degrees of dorsiflexion is optimal for eliciting twitch responses in the plantarflexors and is on the plateau of the joint angle/torque curve (Sale *et al.* 1982). Contractile measures were performed at 0°D, 10°D, and 20°D for Exp 2.

Stimuli were delivered from a high voltage Grass S88 stimulator through a Grass SiU5 stimulus isolation unit with single rectangular voltage pulses of 150 μ s

(microseconds). The intensity (voltage) was adjusted to elicit a maximal twitch peak torque for an individual subject trial. A single pulse of identical parameters to that eliciting the single twitch, was employed for the interpolated twitch. Stimulating voltage remained constant during a single testing session for all twitch and interpolated twitch measures. Maximal twitch responses were analyzed on a computer software program specially designed in our lab to evaluate the following contractile parameters: peak twitch torque (PTT), time to peak torque (TPT), maximum rate of torque development (MRTD), maximum rate of torque relaxation (MRTR), torque-time integral (TTI), TTI to 1/2 relaxation time (TTIHRT), and 1/2 relaxation time (HRT). The software program also evaluated peak MVC torque and interpolated twitch torque (ITT) for the MVC's.

Electromyography (EMG)

EMG recordings were made with 10 mm diameter Ag/AgCl (Meditrace 60) surface electrodes. Electrodes were placed over the soleus (approximately 15 cm proximal from the lateral malleolus and 1.5 cm lateral from the medial line, and at the lateral insertion into the Achilles tendon - interelectrode distance was ~12 cm). One ground electrode was positioned on the tibia. The skin was shaved, abraded with high grit sandpaper and cleaned with alcohol. This electrode placement was aimed to record, as selectively as possible, the muscle compound action potentials (M-wave) produced by the soleus. Electrode positioning was not altered during a single testing trial.

The EMG signal was passed through an AC amplifier (Honeywell Accudata 135A). The gain was calibrated to optimize signal amplitude for A/D conversion. Because the sampling frequency was 6 kHz total for the system, sampling was divided by two for twitch recordings (3.0 kHz per channel for twitch torque and EMG) and divided by three for MVC recordings (2.0 kHz per channel for MVC torque, EMG, and interpolated twitch torque). EMG and torque were only sampled at 50 Hz during PSmax to provide a visual signal of passive torque and EMG activity to the subject and experimenter during PSmax.

Codas software was used to acquire and analyze EMG records. Raw EMG signals were full wave rectified, and the resulting signal was integrated over the duration of the contractions. Integrated EMG in two one-half second windows, prior to and following the interpolated stimulus in each maximum voluntary contraction (MVC), was divided by time

(1 s) to achieve average integrated EMG (AEMG, mV). Four PSmax trials were selected at random and sampled at 1.0 kHz/channel to estimate AEMG at time intervals during PSmax.

The peak-to-peak amplitudes of M-waves associated with twitch responses were measured for each muscle twitch elicited. M-wave areas were calculated on four subjects to confirm the consistency of M-wave area to peak-to-peak M-wave changes following PSmax. MVC AEMG and M-wave values can be affected by electrode positioning, so AEMG:M-wave ratios (AEMG/M-wave) were calculated to control for this variation as it is assumed that differences in electrode positioning would affect both variables equally. Variability in EMG recordings between trials within the same subject is more caused by differences in electrode placement than by inherent differences in activity of the muscle (Viitasalo & Komi, 1975).

Muscle Stiffness (Passive tension)

Passive tension was measured as the passive torque at ankle angles of 0°D, 10°D, and 20°D after torque was zeroed at 10°P. Therefore, the passive torque measure is the increment in torque from 10°P, where it is observed that passive torque is negligible for the plantarflexors (Kawakami, unpublished observations; Fowles, unpublished observations). Passive torque measures were always performed in successive order from 10°P, 0°D, 10°D, to 20°D. Passive torque was measured before and after contractile measures at each time point, and before and after maximal passive stretch (PSmax).

2.6 STATISTICAL ANALYSIS

Statistical analysis was performed on Statistica for Windows R.4.5 software (Statsoft Inc., 1993). Descriptive statistics included means, standard deviation (SD) and standard error (SE). Data in the text are presented as means (SE unless otherwise indicated). Multi-factor analysis of variance (ANOVA) with repeated measures was used to analyze performance measures. Post hoc analysis of mean values was performed using Tukey's HSD method. The probability level for statistical significance was accepted at $P \leq 0.05$.

2.6.1 Experiment 1

Two factor (condition, time) ANOVA's with repeated measures on the time factor were used to analyze contractile parameters. Twitch measures design was 2 x 6 (two condition, six time point). MVC measures design was 2 x 7 (two conditions, seven time points).

Stiffness measures were analyzed using a four factor ANOVA. As passive torque was measured at three joint angles, both before and after the contractile measurement in a single time point, for seven time points in two conditions, there were 84 variables in the stiffness design (two conditions, seven time points, three joint angles, two measures before and after contraction). Only the mean passive torque (stiffness), measured as the passive torque average of three joint angles, pre and post contraction, was compared in the

results. In effect, the 84 variable analysis is presented in the results as the mean passive torque measured for two conditions over time, 2 x 7 (two conditions, seven time points). A Pearson product moment correlation was performed on the relationship between average peak twitch torque and mean passive torque for ten subjects.

2.6.2 Experiment 2

Exp 2 did not involve a Con trial. Subsequently, two factor (time points, joint angle) within subject ANOVA's were used to analyze contractile parameters. Differences were evaluated relative to the PRE values. Twitch measures design was 6 x 3 (six time points, three joint angles). MVC measures design was 3 x 3 (three time points, three joint angles).

Stiffness measures from Exp 2 were combined in an analysis with the stiffness measures from Exp 1. Stiffness measured before contractile parameters was compared using a 2 x 6 x 3 ANOVA (two PSmax trials, six time points, three joint angles). Only the 2 x 6 interaction was evaluated so effectively, the mean stiffness (mean of three joint angles) was compared between experimental trials.

Two ANOVA's compared the PSmax parameters of Exp 1 and Exp 2. A two factor ANOVA (two PSmax trials, twelve stretches) compared the relative increases in joint angle. A three factor ANOVA 2 x 12 x 2 (two PSmax trials, twelve stretches, two torque measurements at initiation and end of stretch) was used to compare passive torque achieved between the two experiments.

3.0 RESULTS

The results are presented separately for Exp 1 and Exp 2. To avoid duplication, only pertinent results from Exp 2 will be presented; that is, those unique from Exp 1. The remaining results from Exp 2 will be referred to and tabulated in the Appendix.

3.1 EXPERIMENT 1

Maximal Passive Stretch (PSmax)

The maximal passive stretch (PSmax) protocol simulated an intense stretch of the ankle plantarflexors. To the subjects, the sensation was similar to that produced by standing on one leg with the ball of the foot on a stair, and allowing the extended heel to drop and passively stretch the calf muscles. The experimental set-up allowed measurement of ankle joint angle (JA), passive torque, and soleus EMG activity with each stretch (Figure 1).

Angular Displacement. PSmax caused an increase in maximum JA of 31.3 (1.5 (D to 37.8 (1.7 (D during the course of 13 repeated stretches (relative increase, 20.8%, $P < 0.0005$). A significant increase in JA was achieved after the first stretch (31% of total JA increase $P < 0.0005$). Over half of the JA increase (57.0%) attained in thirteen stretches was achieved by the fourth stretch (Figure 2). One female subject reached the end-range of the apparatus at 48°D by the seventh stretch.

Passive Torque. Passive torque traces during PSmax indicated stress relaxation. Passive torque decayed rapidly after initiation of a single stretch then decayed gradually ~30 s after stretch onset (Figure 1). Average peak passive torque at initiation of a stretch interval increased from 38.2 (2.3 Nm in the first to 41.2 (2.0 Nm on the third stretch (relative increase 7.8%, $P < 0.0005$) then did not change for any stretches thereafter, indicating a 'set point' for stretch tolerance within the subjects (Figure 2).

PSmax EMG. EMG recorded during the stretch protocol indicated that two subjects were not totally 'passive' during PSmax and were therefore disqualified from the analysis for both Exp 1 and Exp 2. The AEMG for the subject presented in Figure 3A, and the other subject removed from the analysis, indicated EMG activity approaching 10-12% of MVC AEMG. The disqualified subject(s) reported no 'intent' to voluntarily contract, so the activity was likely reflex in origin. All other subjects' AEMG was at, or below the lower detectable limit of the collection equipment (~ 1-3% of MVC AEMG, similar in amplitude to the noise of the recording system) as shown in Figure 3B.

Figure 1. Measurements of angular displacement, passive torque, and soleus EMG during PSmax for stretches 1, 2, 3, and 12 in a female subject. Angular displacement was facilitated by the experimenter passively dorsiflexing the ankle to the stretch limit or 'stretch tolerance' indicated by the subject, then the joint was locked in place for the duration of the stretch interval. There were 13 total stretches of 2 min 15 s each in the PSmax protocol. The passive torque trace exhibits stress relaxation, the incremental increase in angular displacement is indicative of tissue 'creep'.

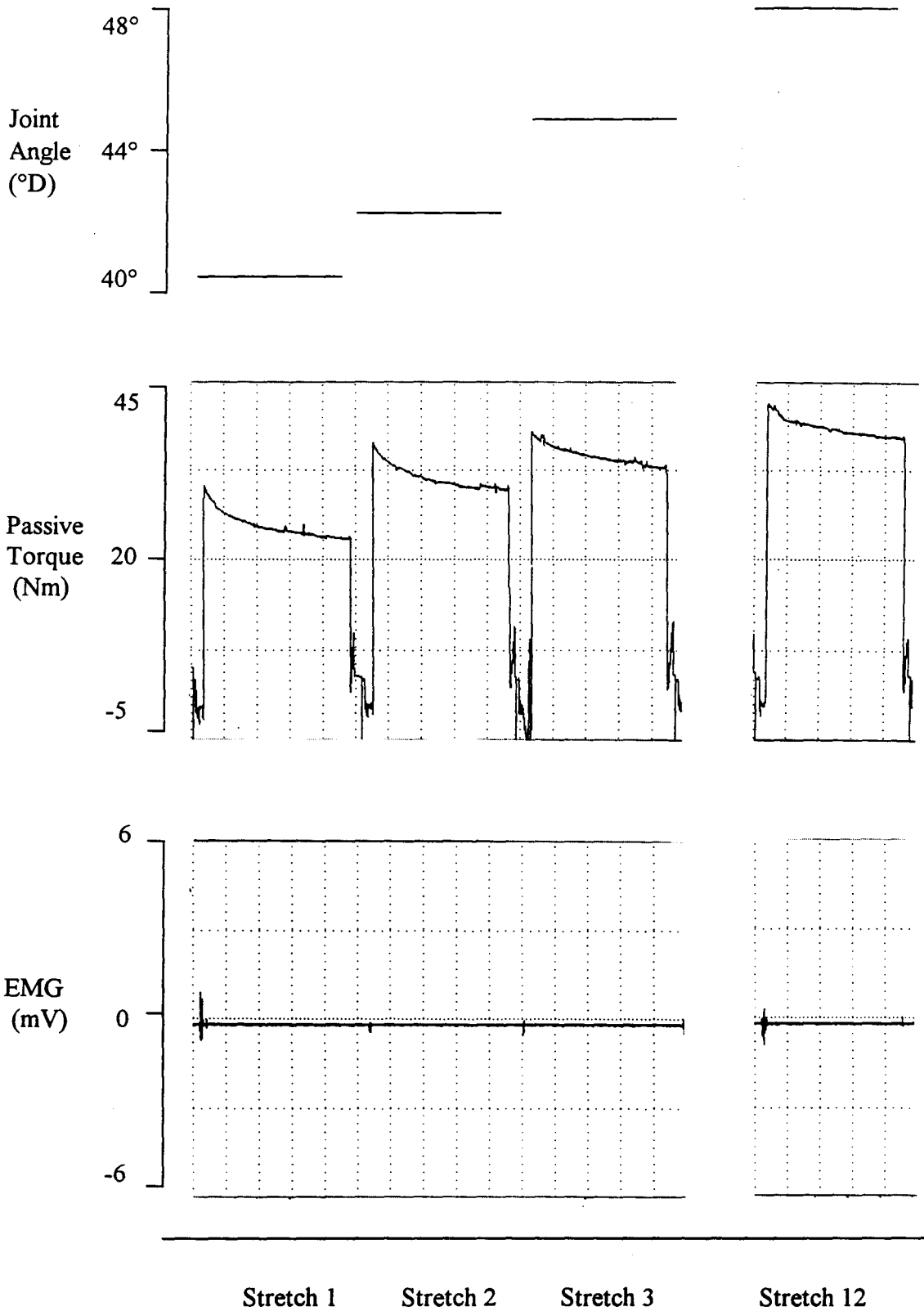
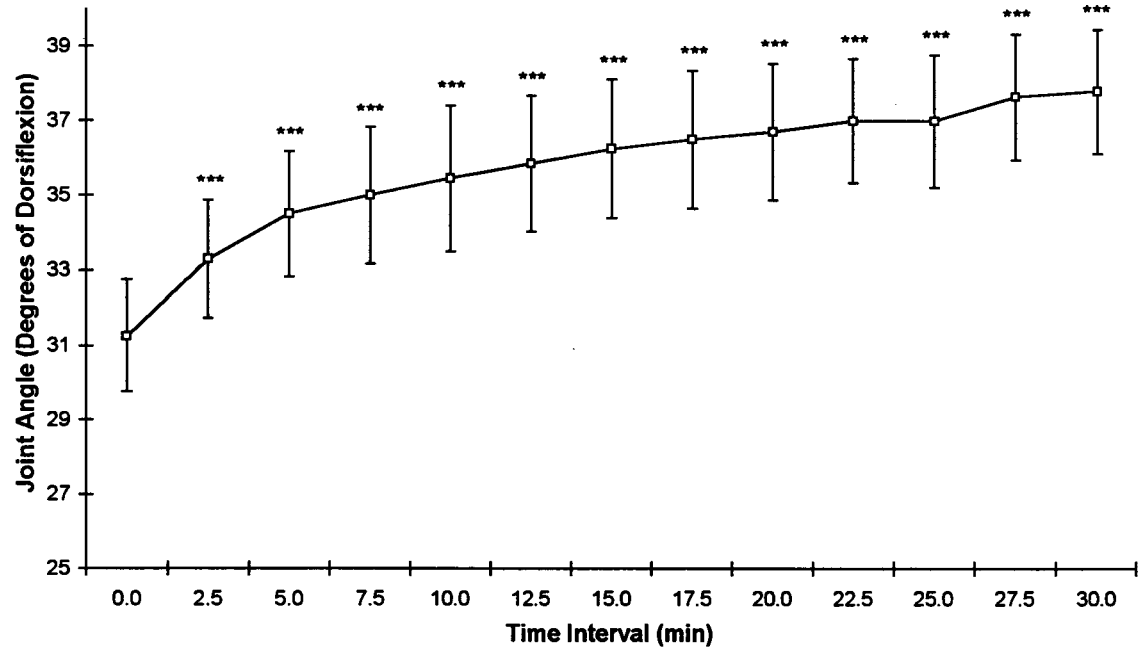


Figure 2. Top: Change in maximum dorsiflexion joint angle during the passive stretch protocol (PSmax). There was significant main effect of time ($P < 0.000001$). *** significantly different than stretch one ($P < 0.0005$). The duration of stretch was 30 min, but there were 15 s pauses at 2 min 15 s intervals. Bottom: Passive torque from initiation to end of a stretch interval. *** significantly greater passive torque at initiation from stretch one. ### significantly lower end stretch torque than all other end stretch torques ($P < 0.0005$). Values are mean (SE).

Angular Displacement



Passive Torque

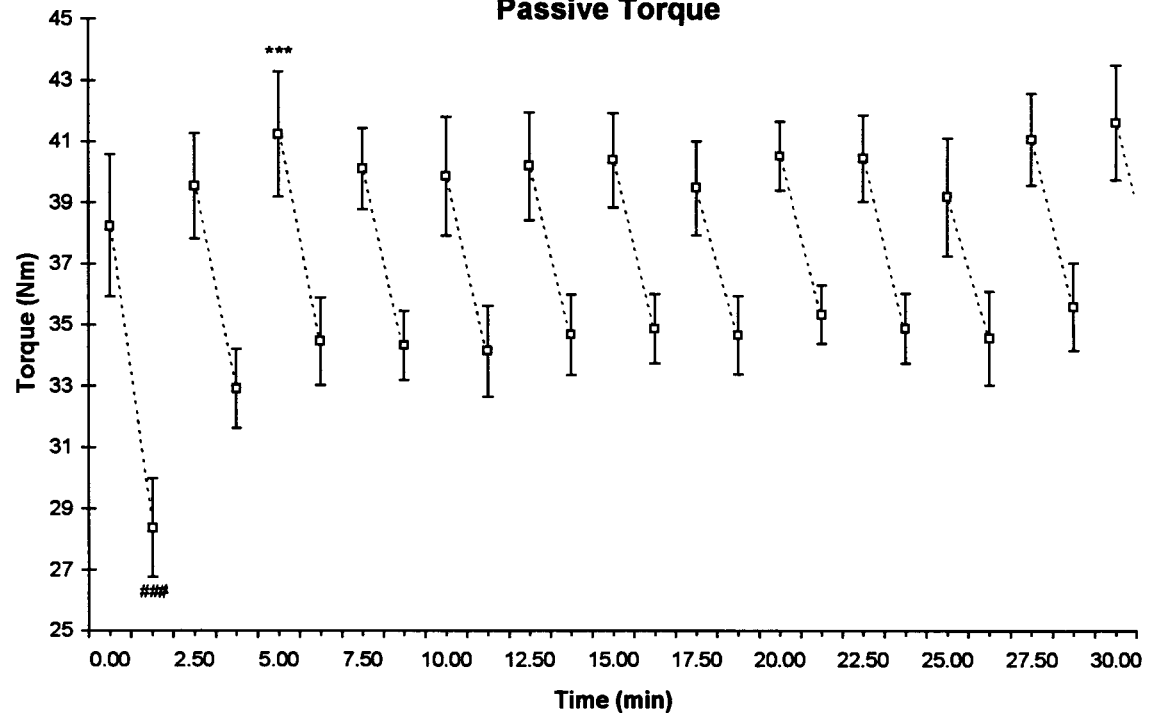


Figure 3A. Passive torque trace of P_{Smax} for a male subject exhibiting reflex EMG activity. Peak passive torque reached ~ 25% of MVC torque and AEMG approximated 10-12% of MVC AEMG. The subject was disqualified from analysis.

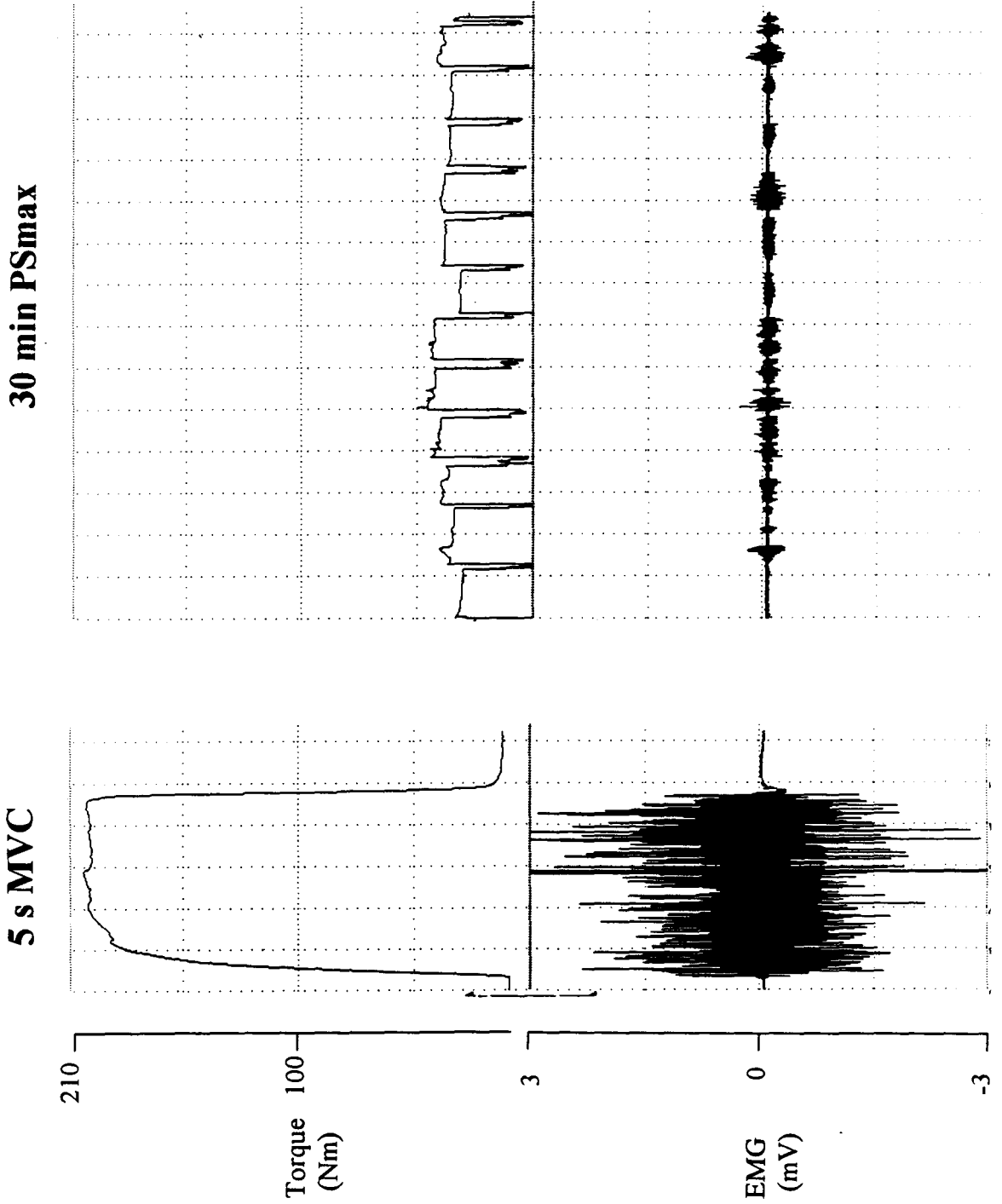
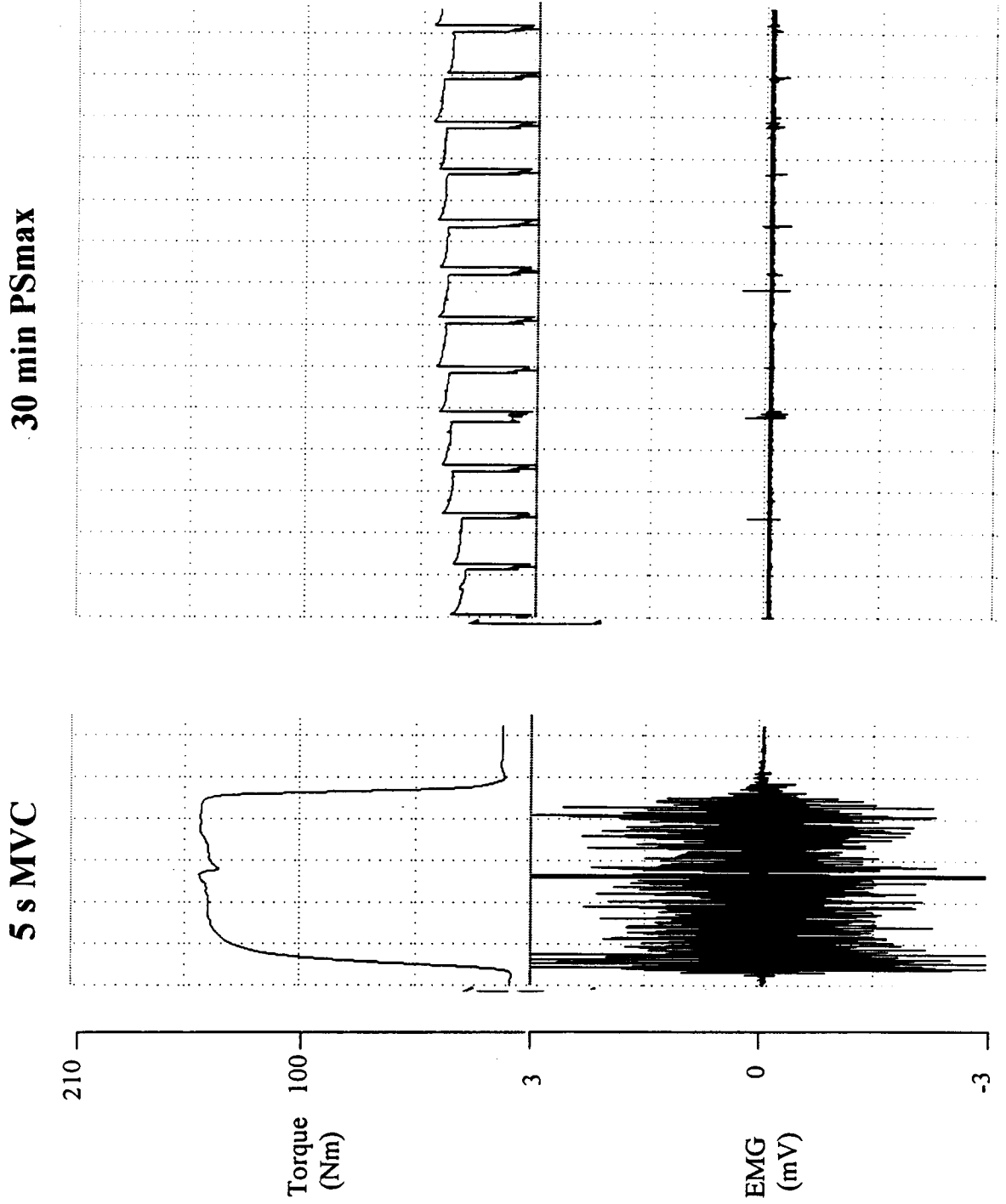


Figure 3B. A typical P_Smax trace for a male subject exhibiting no EMG activity. The lack of EMG activity represents a truly 'passive' stretch.



Isometric MVC. PSmax caused a 27.9 % decrease ($P < 0.0005$) in MVC (Figure 4). MVC had recovered to 80.0 % of the PRE value at 5 min, and to 87.2 % at 15 min. MVC was still below (8.6%) the PRE value at 60 min after the stretch (N.S.). MVC did not change significantly in the Con condition. PSmax values were significantly less than Con values at POST (22.9%) and 5 min (17.3%).

Interpolated twitch (ITT) and motor unit activation (MUA). PSmax caused a 370% increase in ITT ($P < 0.0005$) (Figure 5). ITT was still significantly elevated (280%) 5 min after PSmax. ITT did not change significantly in the Con condition. MUA calculated from ITT was significantly decreased immediately (POST, 15.8%) and 5 min (13.0%) after PSmax. MUA did not change in the Con condition.

The decrease in MVC after PSmax was partly the result of decreased MUA, but PSmax may also have decreased muscle force generating capacity. Estimates of the relative contributions to the MVC deficit, of reduced MUA and reduced muscle force generating capacity, were made using the method of Duchateau (1995). These estimates are shown in Figure 6. Immediately following PSmax (POST, 5 min), force deficit was caused by reduced MUA (~ 60%) and reduced muscle force generating capacity (~40%). Reduced MUA played a minor role in the force deficit at 15, 30, 45 and 60 min post PSmax.

Electromyography (MVC AEMG). MVC average integrated EMG (AEMG) at PRE was similar in Con (0.610 (0.096 mV) and PSmax (0.581 (0.083 mV). This observation may have implications when interpreting differences in AEMG over time between conditions. Con AEMG increased over the trials, immediately by 8.5 % at POST (N.S.) and reaching a significant elevation over the PRE value at 60 min (20.1% increase over PRE). AEMG in PSmax was reduced by 15.1% at POST (N.S.), recovered quickly at 5 min and at 15 min to PRE values, and elevated over PRE at 45 min ($P < 0.05$) and 60 min ($P < 0.005$). Because of the increase in Con AEMG also observed, only the reduced AEMG at POST and 5 min in PSmax was significantly different from Con ($P < 0.001$ and $P < 0.05$ respectively). The increased AEMG activity in recovery from PSmax is more evident when normalized to the torque produced in the MVC contraction (MVC AEMG : MVC torque ratio), although no specific effects were significant (Figure 7).

Figure 4. Effect of maximal passive stretch (PSmax) on maximal voluntary strength (MVC). There was a condition x time interaction ($P < 0.000001$). *** indicates difference from PRE value, $P < 0.0005$; ** ($P < 0.005$). ### indicates difference between PSmax (■) and Con (□) conditions, $P < 0.0005$. Values are means + SE.

Maximum Voluntary Contraction

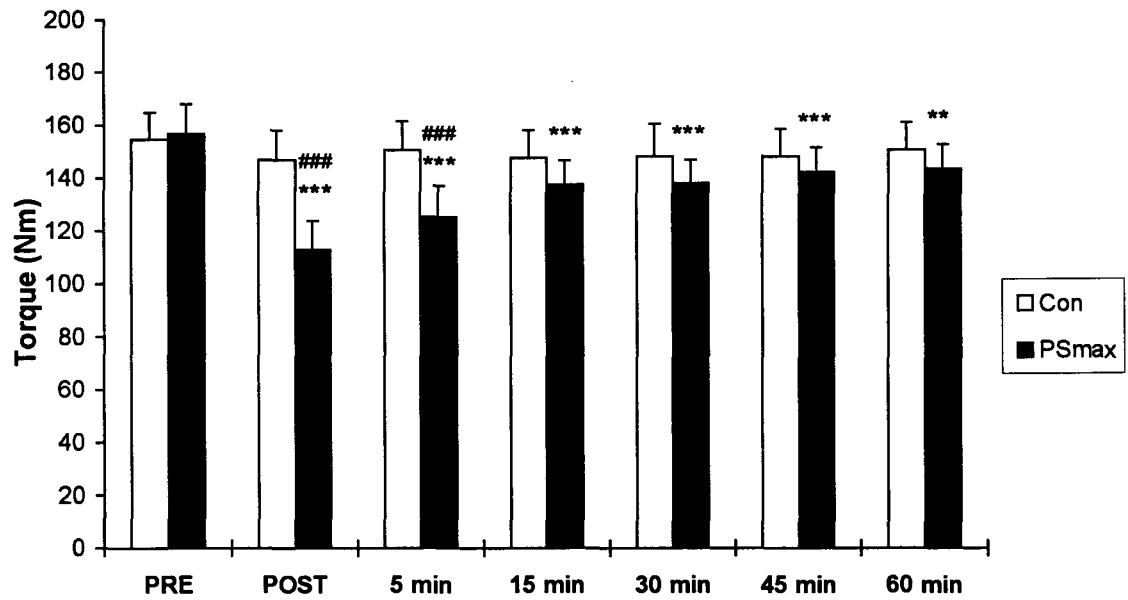
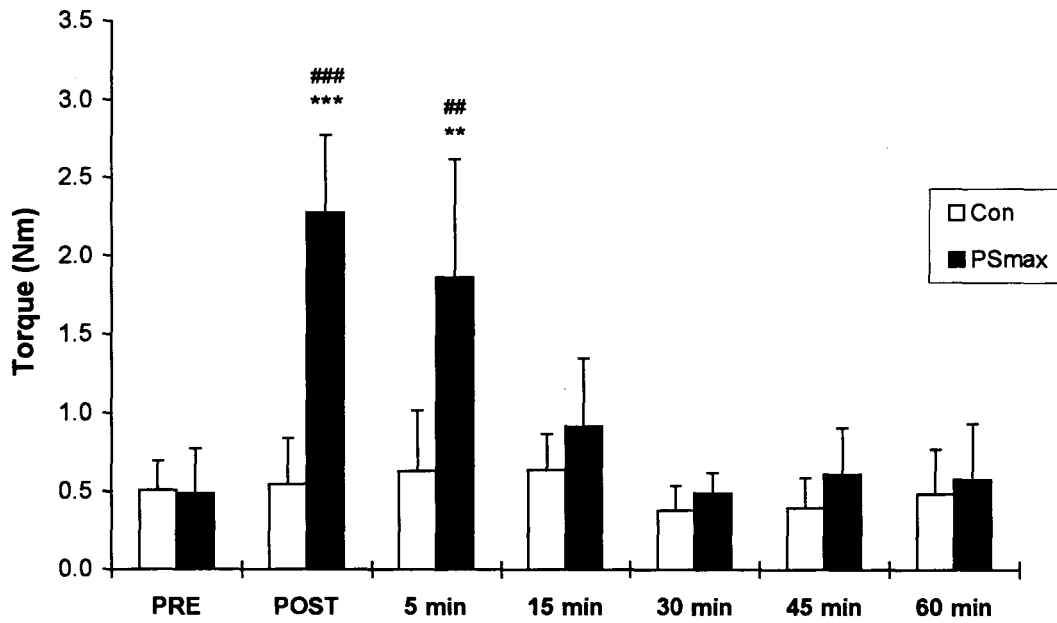


Figure 5. Interpolated Twitch Torque (ITT) (top) and Motor Unit Activation (MUA) (bottom) following 30 min of maximal Passive Stretch (PSmax ■) of the ankle dorsiflexors or neutral angle control (Con □). There were significant main effects for condition ($P < 0.05$) and highly significant interactions ($P < 0.00005$) for both ITT and MUA. *** significantly different from PRE, $P < 0.0005$; ** $P < 0.005$. ### significantly different from Con, $P < 0.0005$; ## $P < 0.005$. Values are means + SE.

Interpolated Twitch Torque



Motor Unit Activation (MUA)

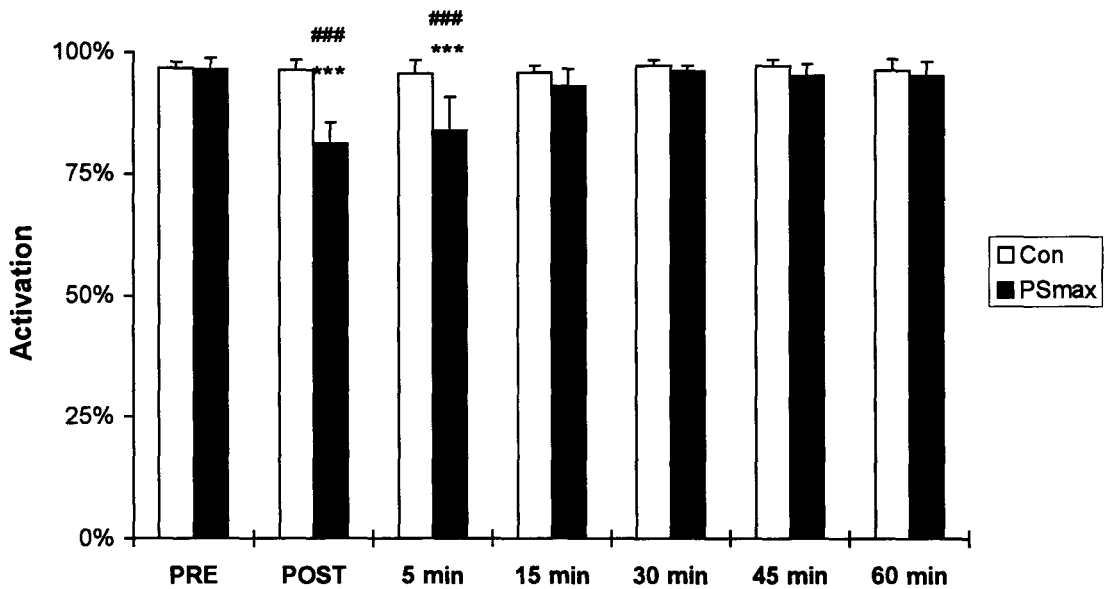
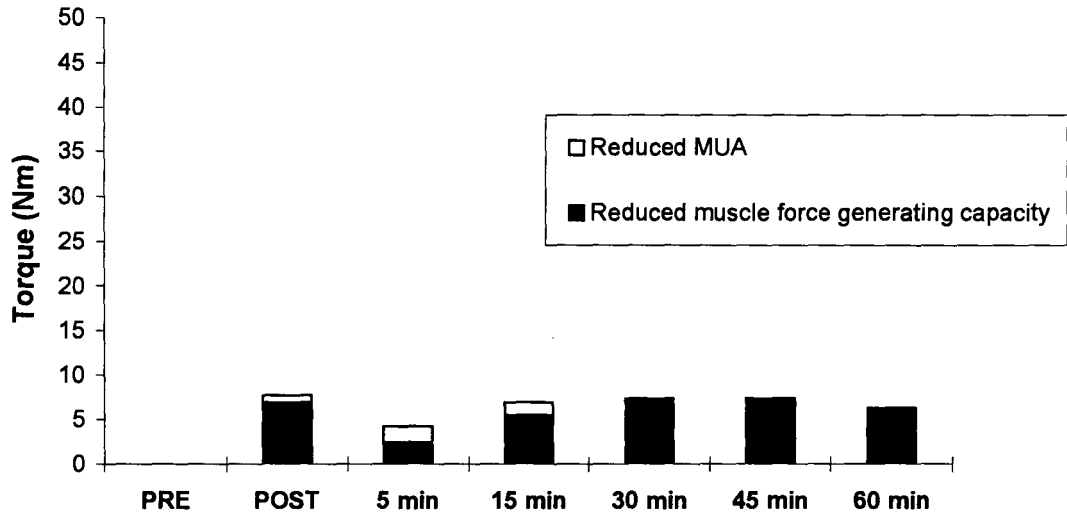


Figure 6. Estimated contributions of reduced motor unit activation (MUA □) and reduced muscle force generating capacity (■) to the MVC deficit after passive stretch. Top: In the control (Con) condition the MVC deficits were not significant. Bottom: MVC deficits were significant at all time points post PSmax ($P < 0.01$). *** significant decrement from PRE value, $P < 0.0005$; ** $P < 0.005$; * $P < 0.05$.

Absolute force decrement in Control



Absolute force decrement due to PSmax

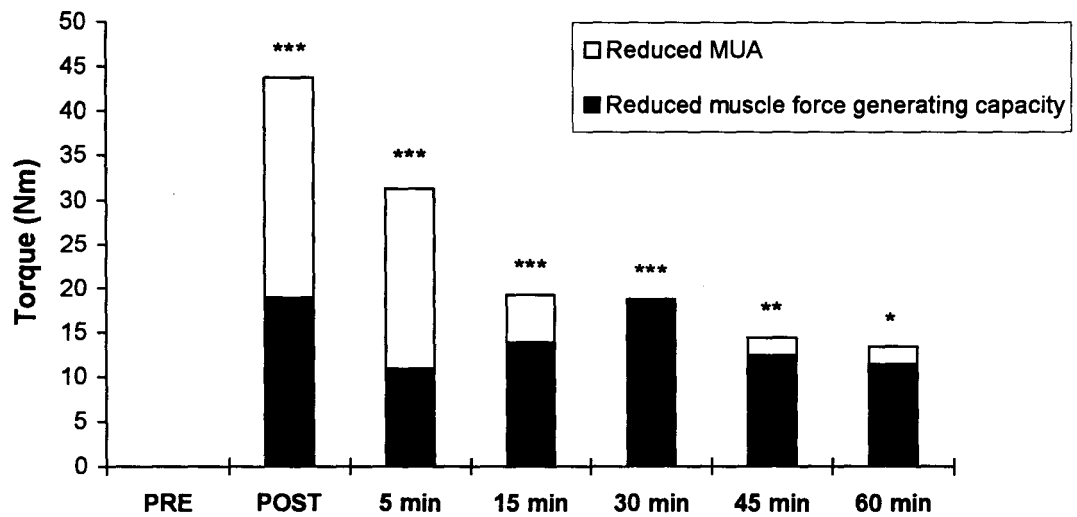
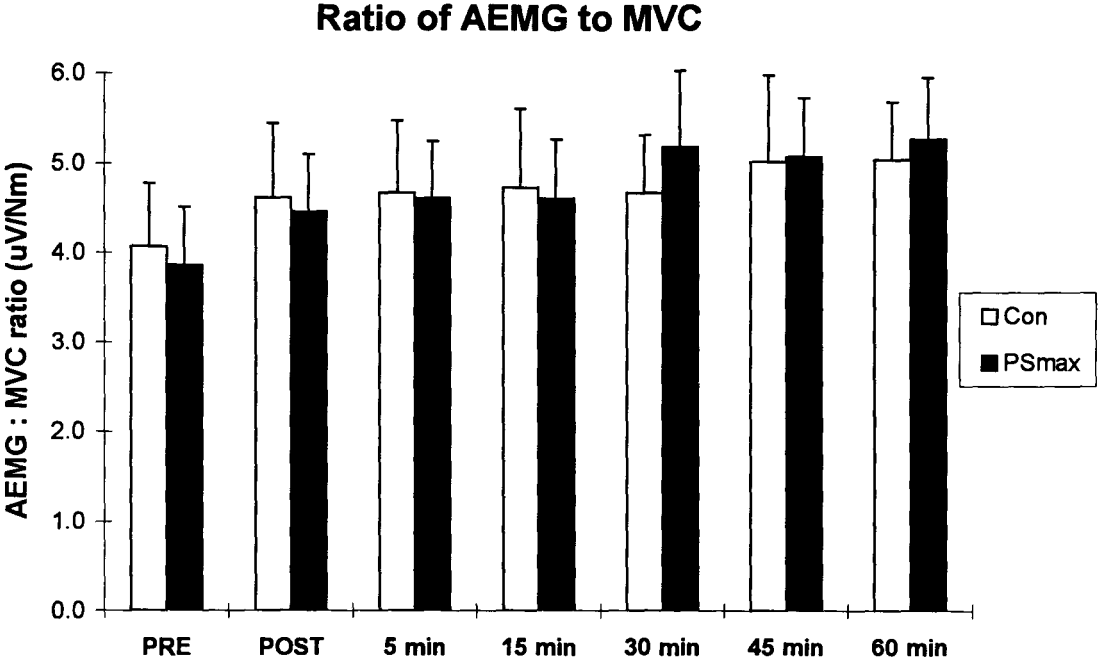
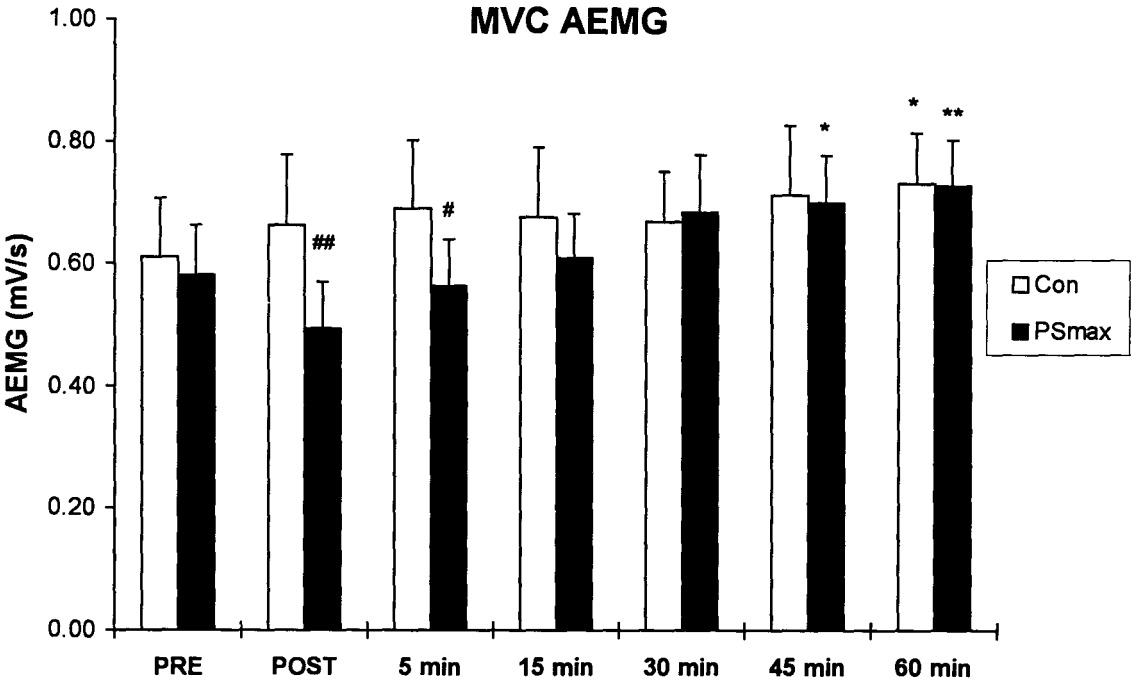


Figure 7. Isometric MVC AEMG in the maximal passive stretch (PSmax ■) and control (Con □) condition (top). There was a significant interaction of time x condition ($P < 0.005$). MVC AEMG to MVC torque ratio (bottom) only exhibited a main effect for time ($P < 0.000005$). ** significantly different from PRE, $P < 0.005$; * $P < 0.05$. ## significantly different PSmax to Con, $P < 0.005$; # $P < 0.05$. Values are means + SE.



Peak Twitch Torque (PTT). PTT significantly decreased immediately after PSmax (17.7%) and also in the Con condition (8.9%) (Figure 8). The decrease after PSmax was greater (condition x time interaction, $P < 0.05$). In the PSmax condition, PTT recovered to 91.5% of the PRE value at 15 min, then decreased to 86.6% of PRE values for the remaining time points. In the Con condition, PTT recovered to 95.9% of the PRE value at 15 min, then decreased 92.5%, 91.2% and 91.0% of PRE values at 30, 45, and 60 min. A twitch was not measured at 5 min because of the confounding effects of post-activation potentiation (Vandervoort & McComas, 1983) that may result from the POST MVC. The 15 min twitch may have exhibited some lingering potentiation effects of the MVC completed at 5 min.

Time related contractile properties. The decreased twitch size was accompanied by an increase in contractile speed following PSmax (Table 2, Figures 9-10). Half Relaxation time was significantly lower in the PSmax condition compared to Con ($P < 0.0005$). Time to peak torque (TPT) was faster in PSmax compared to Con only at POST ($P < 0.0005$) and at 45 min ($P < 0.05$). TPT and HRT increased in time points post ($P < 0.05$) in the Con condition.

Twitch to MVC ratio was relatively constant (range: 0.098 - 0.103) in all Con and between PRE and 15 min, 30 min, 45 min, and 60 min time points in PSmax condition (range: 0.096 - 0.11 excluding 5 min) (Table 2).

Muscle compound action potential (M-wave). M-Wave areas showed the same trends as peak-to-peak M-wave values in this study, so only peak-to-peak M-wave data are presented. Evoked M-wave followed a similar pattern to the MVC AEMG response following PSmax, decreasing by 9.1% at POST ($P < 0.0005$) recovering to PRE values at 15 min, then significantly increasing over the duration of the recovery (values over PRE at 30 min, 6.9%; 45 min, 9.7%; 60 min, 12.2%) (Figure 11). The Con M-wave was stable over all time points, within a range of less than 2.1 % difference from PRE (N.S.). The difference in response between the conditions was more apparent when expressed relative to PTT (Figure 11, bottom). The 60 min PSmax M-wave value was not significantly increased over Con (12.3% increase, $P = 0.074$), but when expressed relative to the twitch torque (M-wave to Twitch ratio) was significantly above Con ($P = 0.007$).

When MVC AEMG is expressed relative to the evoked M-wave (AEMG to M-wave ratio) (Figure 12) there is no difference in EMG between PSmax and Con conditions. Although the AEMG to M-wave ratio was 10.8% and 13.8% lower in PSmax at POST and 15 min likely due to reduced MUA in the MVC at POST and 15 min, and the main effect for time was significant ($P < 0.05$), there were no significant changes with time or interactions of time x condition ($P > 0.25$).

Table 2. Twitch Contractile Properties before and after 30 min of maximal passive stretch (PSmax) or neutral angle control (Con). Means for 10 subjects (SE).

Variable	PRE	POST	15 min	30 min	45 min	60 min
PTT (Nm)						
Con	15.7±0.6	***14.3±0.6	**15.1±0.7	***14.6±0.7	***14.2±0.7	***14.3±0.7
% Diff. PRE		-8.9	-4.1	-7.5	-9.8	-9.1
PSmax	***15.8±0.8	***13.0±0.8	***14.3±0.7	***13.7±0.6	***13.7±0.6	***13.6±0.7
% Diff. PRE		## -17.7	-9.4	-13.4	-13.3	-13.8
TPT (ms)						
Con	113.0±4.0	**120.6±6.0	**120.6±6.0	**120.6±6.0	**123.6±6.0	**123.7±6.0
% Diff. PRE		6.7	6.7	6.7	9.4	9.4
PSmax	115.2±3.8	###109.8±4.1	115.5±5.1	119.4±5.2	#117.6±5.4	*121.2±6.6
% Diff. PRE		-4.7	0.3	3.7	2.0	5.2
HRT (ms)						
Con	102.4±4.7	*114.2±5.5	***118.9±6.1	***120.6±5.6	***124.1±6.1	***122.8±6.3
% Diff. PRE		11.5	16.2	17.8	21.3	20.0
PSmax	102.8±5.4	###93.4±3.3	###103.2±3.8	#108.4±3.8	##111.6±4.5	*113.7±4.6
% Diff. PRE		-9.1	0.4	5.4	8.6	10.7
PTT:MVC						
Con	0.10±0.00	0.10±0.01	0.10±0.01	0.10±0.00	0.10±0.00	0.10±0.00
% Diff. PRE		-3.1	1.0	-1.7	-5.6	-6.4
PSmax	0.10±0.00	0.12±0.01	0.11±0.00	0.10±0.00	0.10±0.00	0.10±0.00
% Diff. PRE		17.4	3.0	-2.0	-4.8	-6.2

*** significantly different from PRE value, $P < 0.0005$; ** $P < 0.005$; * $P < 0.05$.

significant difference of PSmax to Con value, $P < 0.0005$, ## $P < 0.005$, # $P < 0.05$.

Figure 8. Peak Twitch torque (PTT) following maximal Passive Stretch (PSmax ■) or neutral angle control (Con □). All time points post were significantly decreased from PRE in the PSmax condition ($P < 0.005$). Con values were significantly decreased from PRE ($P < 0.005$) except for the 15 min time point ($P = 0.45$), evidence for the presence of potentiation in pre-testing. *** significant decrease below PRE values. ### significant decrease of PSmax below Con, $P < 0.0005$. Values are means + SE.

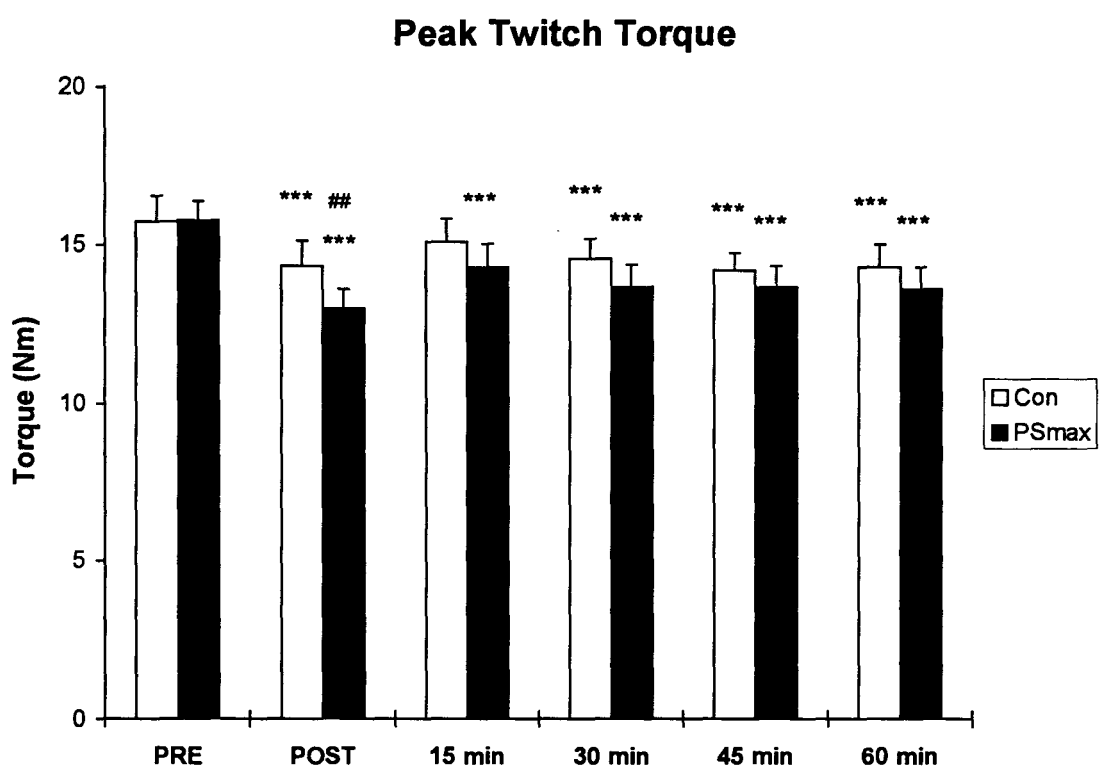


Figure 9. Typical evoked twitch traces at PRE, POST and 60 min following PSm_{max} in a male subject. Traces show a reduction in twitch size and increased contractile speed.

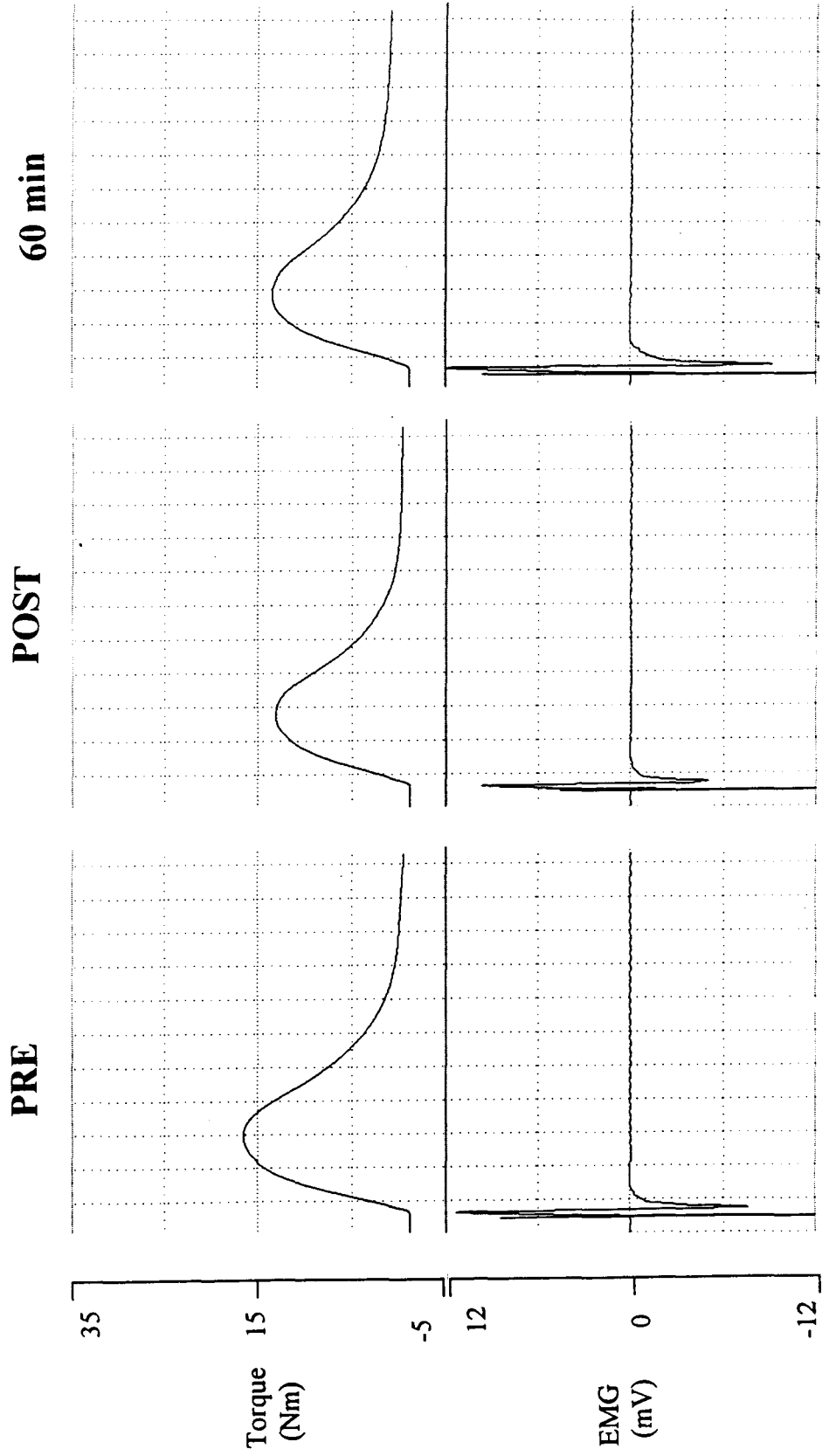
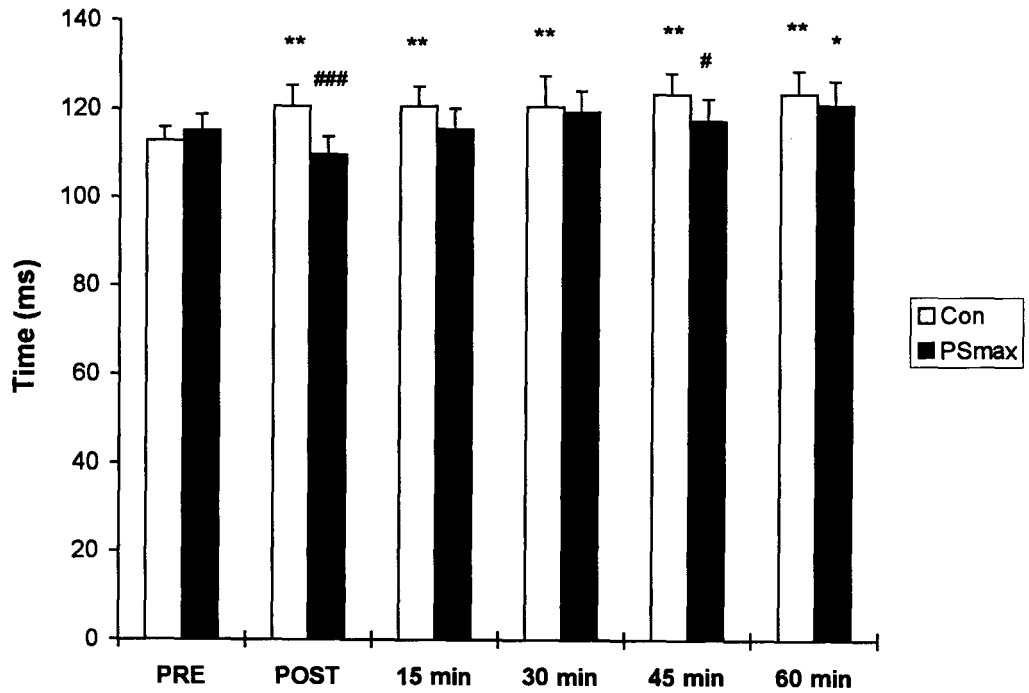


Figure 10. Time to peak torque (TPT) (top), and half relaxation time (HRT) (bottom) following maximal Passive Stretch (PSmax ■) or neutral angle control (Con □). Con measures show increased TPT ($P < 0.005$) and HRT ($P < 0.005$) in post testing. PSmax showed a reduction of twitch speed. *** significantly different from PRE values, $P < 0.0005$; ** $P < 0.005$; * $P < 0.05$. ### PSmax significantly below Con value, $P < 0.0005$; ##, $P < 0.005$; # $P < 0.05$. Values are means + SE.

Time to Peak Torque



Half Relaxation Time

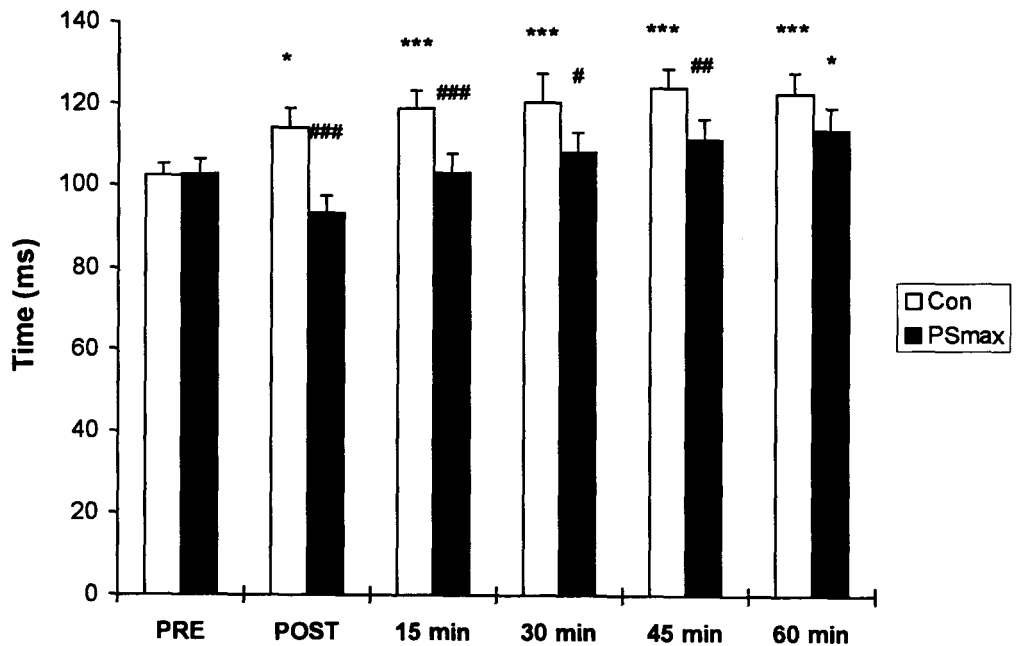
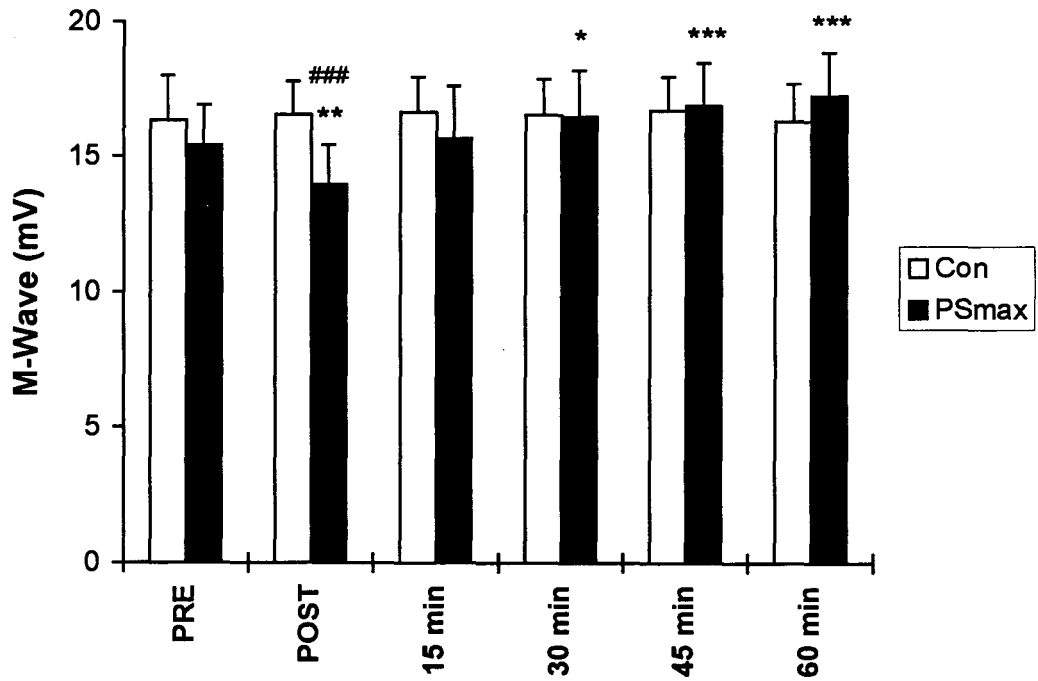


Figure 11. Top: Peak-to-peak M-wave amplitude in Passive Stretch (PSmax ■) and the control (Con □) condition. There was a significant condition x time interaction ($P < 0.000001$). There was no significant difference in Con values. Bottom: M-Wave to peak twitch torque (PTT) ratio also exhibited an interaction ($P < 0.005$). *** significant difference from PRE values, $P < 0.0005$; ** $P < 0.005$; * $P < 0.05$. ### significant difference of PSmax to Con condition ($P < 0.0005$); # $P < 0.05$. Values are means + SE.

Evoked twitch M-Wave



M-Wave to Twitch Ratio

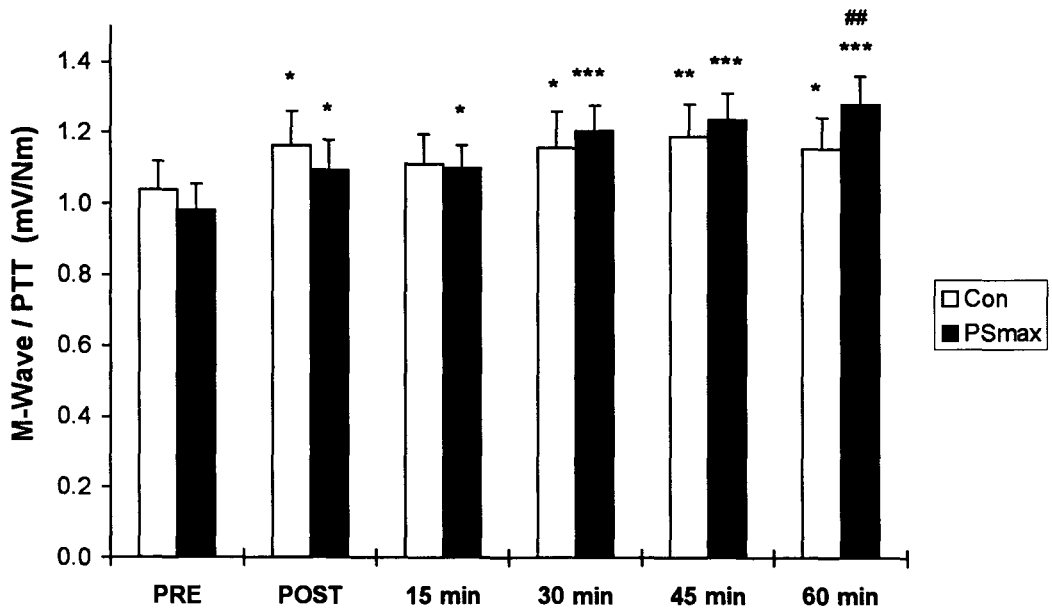
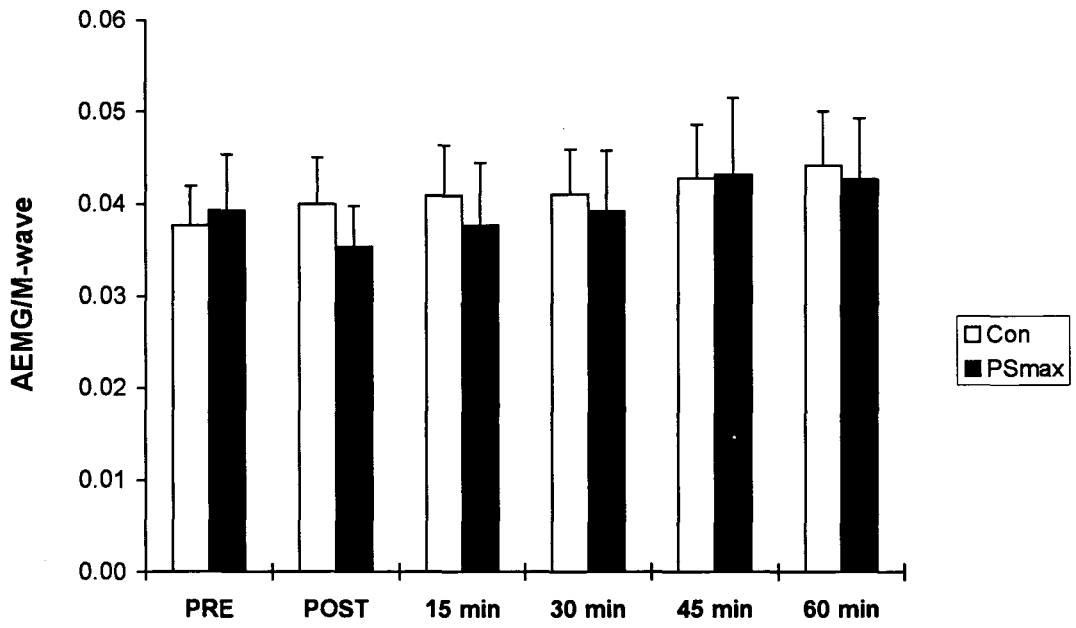


Figure 12. MVC AEMG to M-wave ratio in maximal Passive Stretch (PSmax and control (Con □) conditions. There is no difference between conditions or interaction with time or time x condition. Values are means + SE.

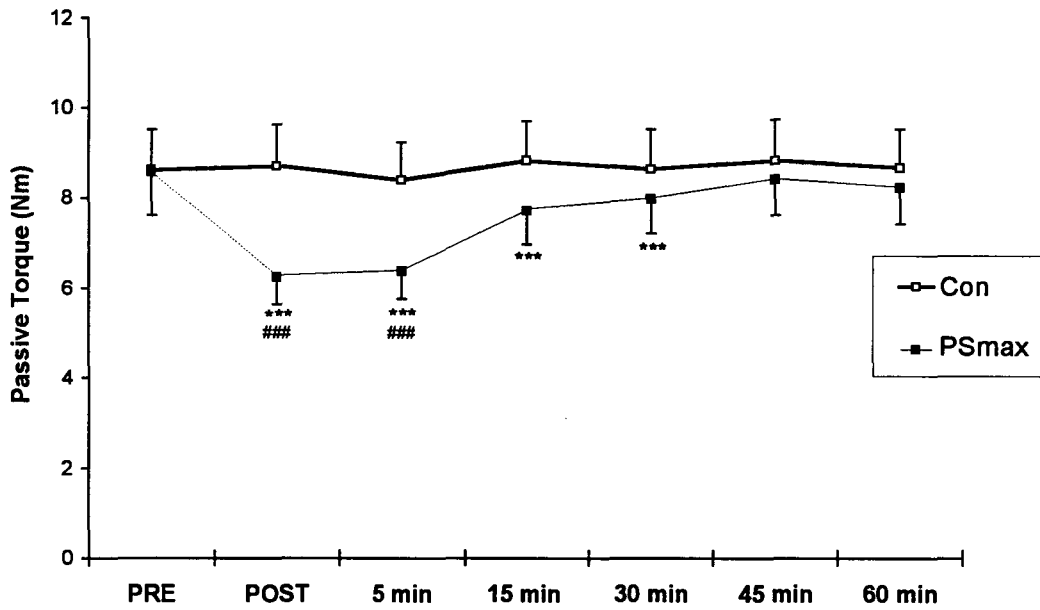
AEMG to M-wave ratio



Muscle Stiffness (Passive Torque). Mean passive torque was calculated as the average torque at a time point for three joint angle measurements both before and after an MVC. Therefore, each data point of mean passive torque contains 6 measurements for 10 subjects, or 60 data points. Mean passive torque was reduced by 27.0% directly following the PSmax protocol ($P < 0.0005$) caused by a shift in the passive tension curve 'down and to the right' (Figure 13). Muscle stiffness was quickly restored so that by 15 min, mean passive torque was no longer significantly below Con ($P = 0.0504$) although still 14.0% below PRE ($P < 0.0005$). Muscle stiffness did not fully recover within one hour, as mean passive torque was still depressed below PRE by 7.8% at 45 min (N.S.; $P = 0.078$) and 8.1% at 60 min (N.S.; $P = 0.058$).

Average PTT was calculated as the average PTT of 10 subjects for a single time point, or 10 data points. Mean passive torque was significantly correlated to average PTT ($n=12$ sample points for both Con and PSmax twitch measures; $r = 0.62$, $P < 0.05$). Figure 14 exhibits the correlation between the average PTT and mean passive torque measures.

Figure 13. Mean passive torque following maximal passive stretch (PSmax ■) or control (Con □) condition (top). The decrease in mean passive torque is caused by a shift in the passive torque-joint angle relation (passive torque curve) 'down and to the right' (bottom). There were no significant differences in Con. *** significantly different from PRE, $P < 0.0005$. ### significant decrease of PSmax below Con, $P < 0.0005$. Values are means \pm SE. Some error bars are omitted for clarity.



Mean Passive Torque (Mean of three joint angles)

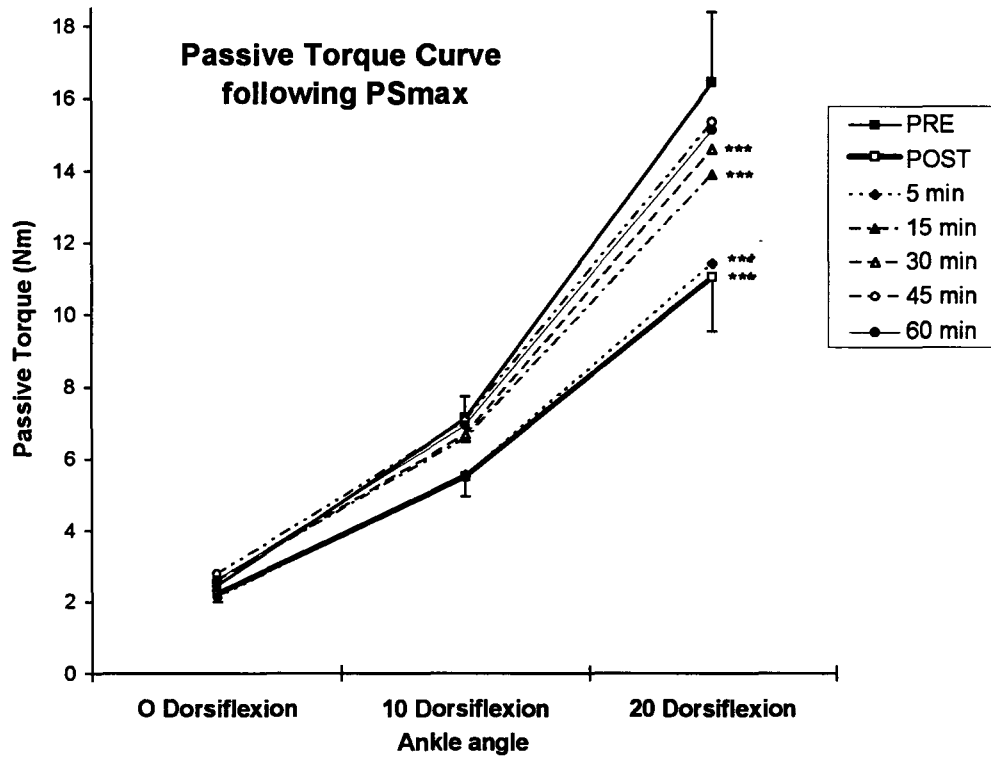
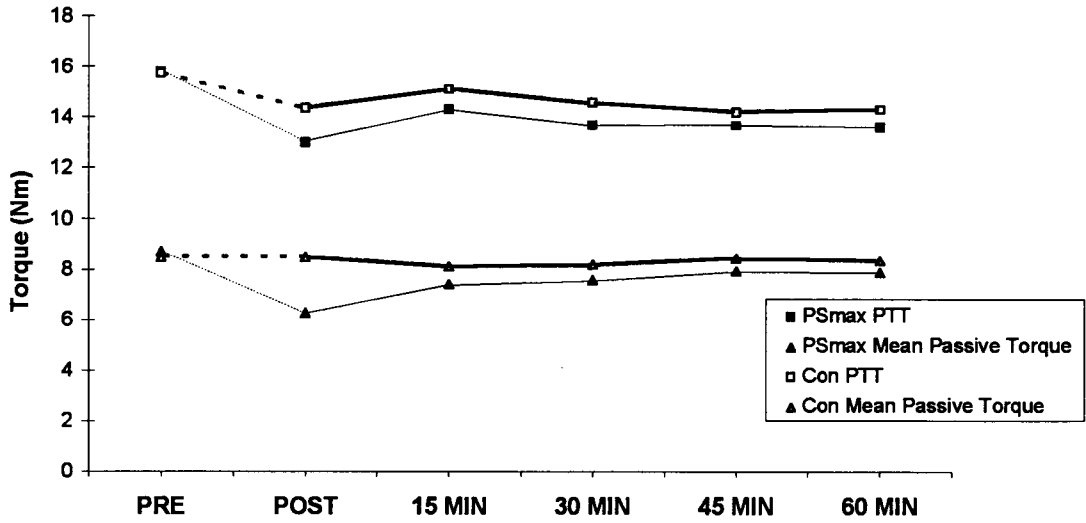
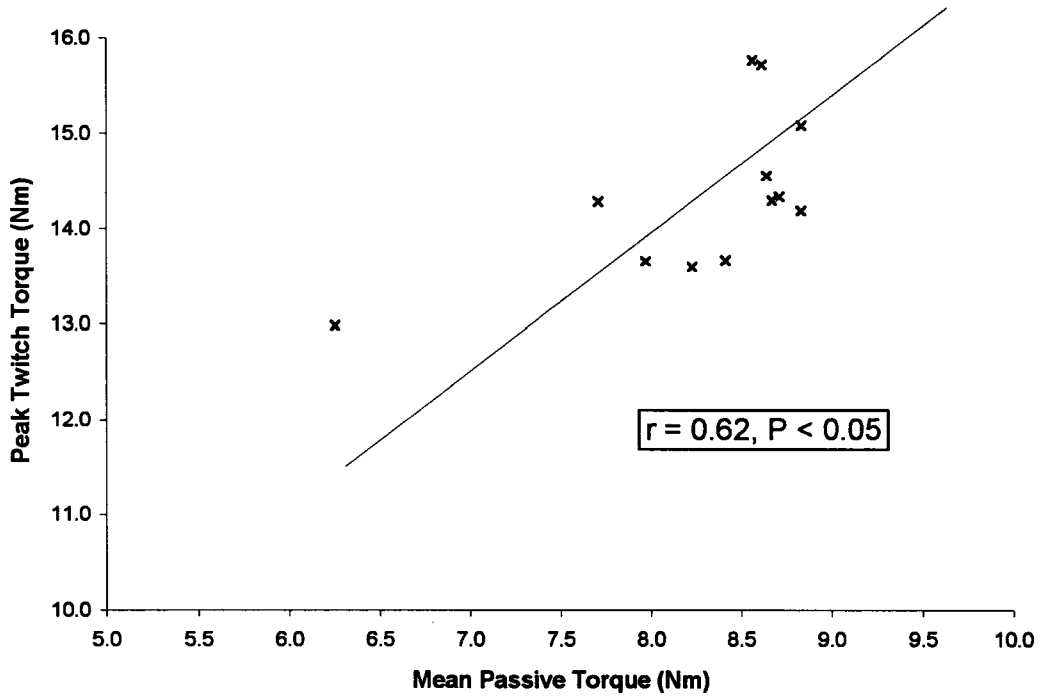


Figure 14. Relationship of average PTT (average of 10 subjects) to mean passive torque (mean passive torque over three joint angles, prior to and post contraction, averaged for 10 subjects) for 12 time points in Con (□) and PSmax (■) conditions. The correlation was significant ($r = 0.62$, $P < 0.05$).



Relation of PTT to Muscle Stiffness



Summary of Experiment 1. Thirty minutes of maximal passive stretch reduced maximum voluntary force and evoked twitch force for up to one hour following the stretch. Decreased maximum voluntary force directly following PSmax was partly due to reduced activation and partly due to reduced muscle force generating capacity. Activation quickly recovered within 15 min whereas recovery of muscle force generating capacity was more prolonged. Muscle stiffness was significantly reduced directly following PSmax. Stiffness recovery was biphasic, recovering mostly within the first 15 min after stretch and more slowly to up to 60 min. EMG activity for MVC and twitch contractions showed a similar pattern of being depressed directly after stretch, then recovering to exceed PRE values by one hour following PSmax.

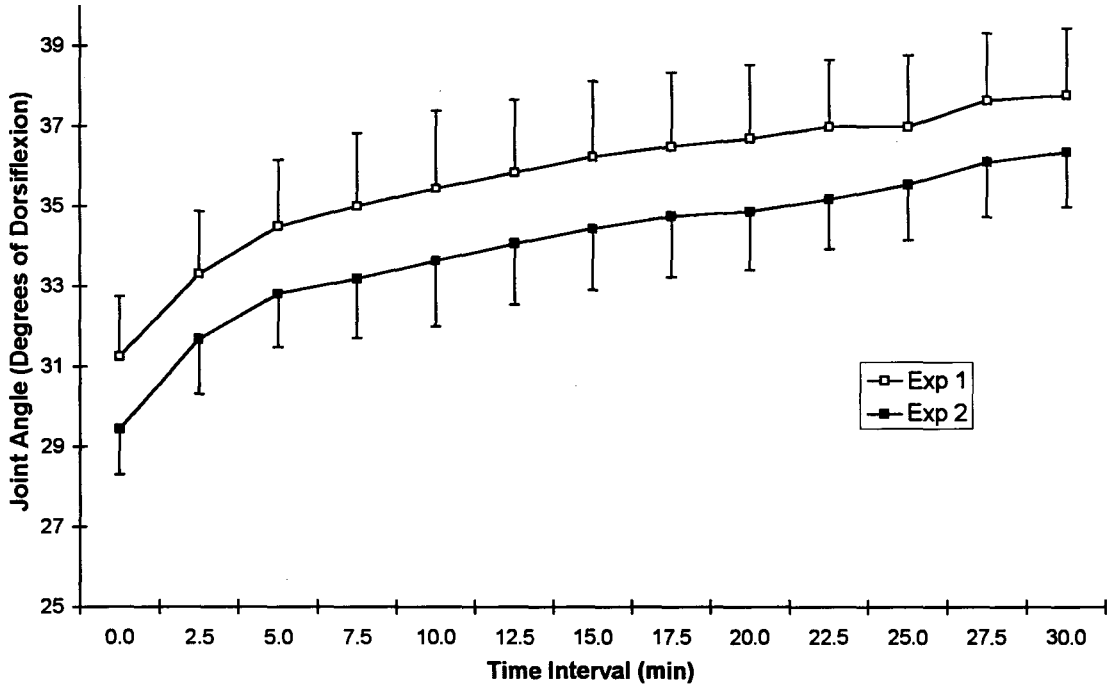
3.2 EXPERIMENT 2

Due to the similarity between reduced contractile force and reduced muscle stiffness observed in Exp 1, the second experiment was designed to control for the increase in muscle length and concomitant reduction in muscle stiffness facilitated by PSmax. Testing included two more joint angles, one 10° below and one 10° above the test angle from Exp 1, to determine if PSmax compromises force generation at a joint angle relative to the increase in muscle length. The hypothesis was that PSmax would cause a 'shift' in optimal force generating joint angle from 10°D [as identified previously by other researchers (Sale *et al.* 1982; Herman & Bragin, 1967)], to a greater joint angle.

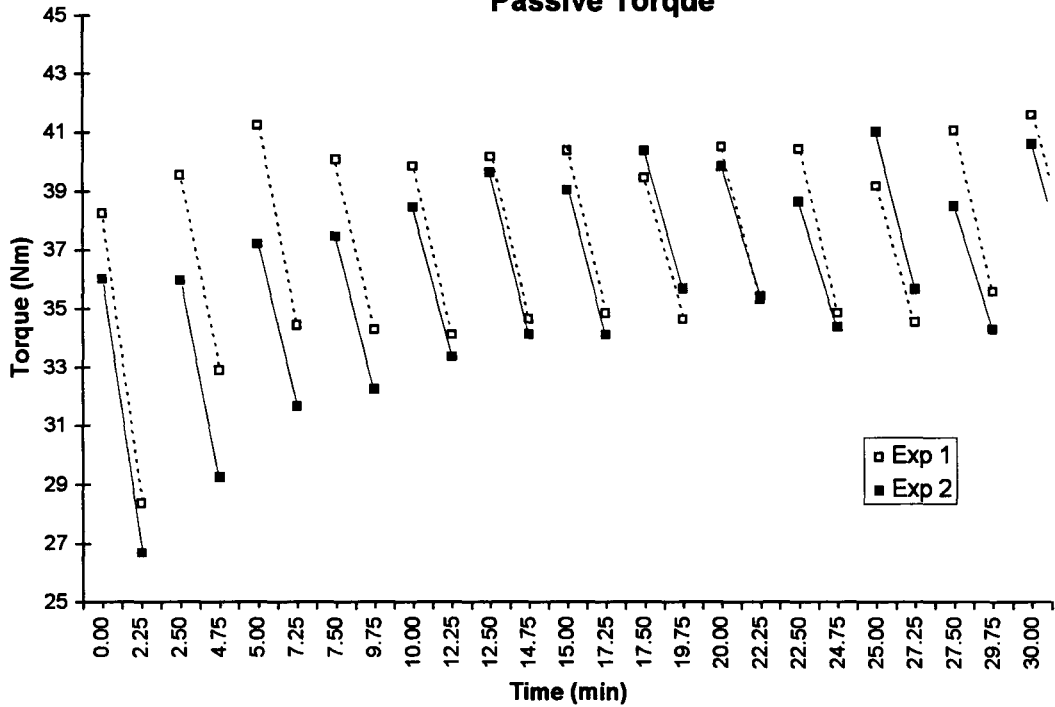
Maximal Passive Stretch (PSmax). There were no significant differences in the maximum joint angles achieved, total increase in maximum joint angle over 13 stretches, or peak passive torque at initiation or end of a stretch interval, between the PSmax protocol in Exp 1 vs Exp 2 (Figure 15). The subject directed the maximum joint angle achieved in the Exp 2 as for Exp 1, so variation in maximum joint angles and passive torques are probably more due to minor variations in apparatus setup than to differences in inherent stiffness of a subject's muscle. It has been shown in previous experiments with the human plantarflexors that stiffness is relatively stable with repeated tests on the same day or different days within subjects (Toft *et al.* 1989a; Toft *et al.* 1989b).

Figure 15. Comparison of angular displacement (top) and passive torque (bottom) between experiment 1 (Exp 1 □) and experiment 2 (Exp 2 ■). There were no significant differences for angular displacement ($P = 0.19$) or passive torque ($P = 0.46$) between Exp 1 and Exp 2. The effects for time are as shown in the results from Exp 1. Values are means and SE. Some error bars are excluded for clarity.

Angular Displacement



Passive Torque



Isometric MVC. MVC was tested at three joint angles in Exp 2 and only at three time points. MVC testing at POST, and 15 min was eliminated because of confounding effects of reduced MUA and to limit the total number of MVCs performed by the subject. The 45 min time point was also excluded to limit the total number of MVCs. When the MVC's tested at 10°D in Exp 2, were compared with the same time points of Exp 1, there was no difference in results found between Exp 1 and Exp 2 meaning that the decreased force was consistent between Exp 1 and Exp 2 at 30 min and 60 min (i.e. main effect for time conserved) (Figure 16).

Peak MVC torque occurred at 10°D at PRE (Figure 17) although there was no significant difference between MVC torque at 10°D and 20°D. The shape of the torque curve was not different at the 30 min time point after 'lengthening' PS_{max}, as there was no interaction of joint angle tested with time ($P = 0.2$). The shape of the torque curve was not different when calculated relative to an individual's 100% MVC. This would indicate that the intensive stretching performed in this protocol was not sufficient to maintain a 'lengthened' state for 30 min after PS_{max} or was not sufficient to cause a significant shift in the contractile element force-length relation, at a time when MVC was still depressed. The mean MVC torque of three joint angles was below PRE by 7.0% at 30 min ($P < 0.01$) and 5.9% at 60 min ($P < 0.05$).

Interpolated twitch (ITT) and motor unit activation (MUA). Interpolated twitch and calculated MUA indicated a main effect for testing joint angle although no effect for time ($P = 0.73$) or interaction ($P = 0.13$) was evident. The 20°D testing angle had lower MUA than either 0°D or 10°D ($P < 0.05$) (Figure 18).

Electromyography (MVC AEMG). MVC AEMG exhibited main effects for joint angle ($P < 0.005$) and time ($P < 0.0005$). Mean AEMG of tests at 20°D (0.721 (0.66)) was greater than testing at 10°D (0.656 (0.62, $P < 0.05$)) and 0°D (0.613 (0.073, $P < 0.005$)). AEMG was significantly elevated over PRE values (0.600 (0.077)) at 30 min (0.671 (0.070, $P < 0.05$)) and at 60 min (0.719 (0.078, $P < 0.0005$)), although 30 min and 60 min values were not different. There were no interactions of joint angle x time (Figure 19). AEMG expressed relative to MVC torque (AEMG:MVC ratio) showed no significant interactions.

Figure 16 Comparison of MVC torque following passive stretch (PSmax) in two experiments (Exp 1 (and Exp 2 ■). There was no significant differences in MVC torque between experiments ($P = 0.46$). *** significant difference from PRE for both experiments, $P < 0.0005$; ** $P < 0.005$. Values are means + SE.

Maximum Voluntary Contraction

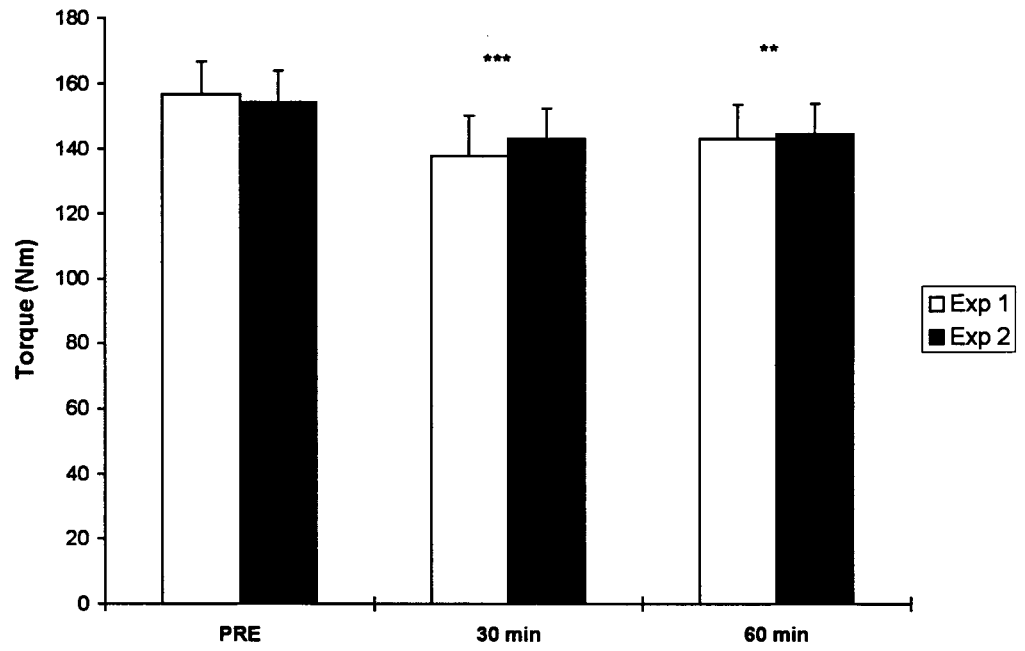


Figure 17. MVC tested at ankle angles of 0°D, 10°D and 20°D. There were no significant changes in the MVC torque curve at 30 min and 60 min following maximal passive stretch (PSmax) ($P = 0.2$). ** significantly reduced mean MVC torque compared to PRE, $P < 0.01$; * $P < 0.05$. ### significantly greater MVC torque at 10°D and 20°D than at 0°D. Values are means \pm SE.

Maximum Voluntary Contraction Torque Curve

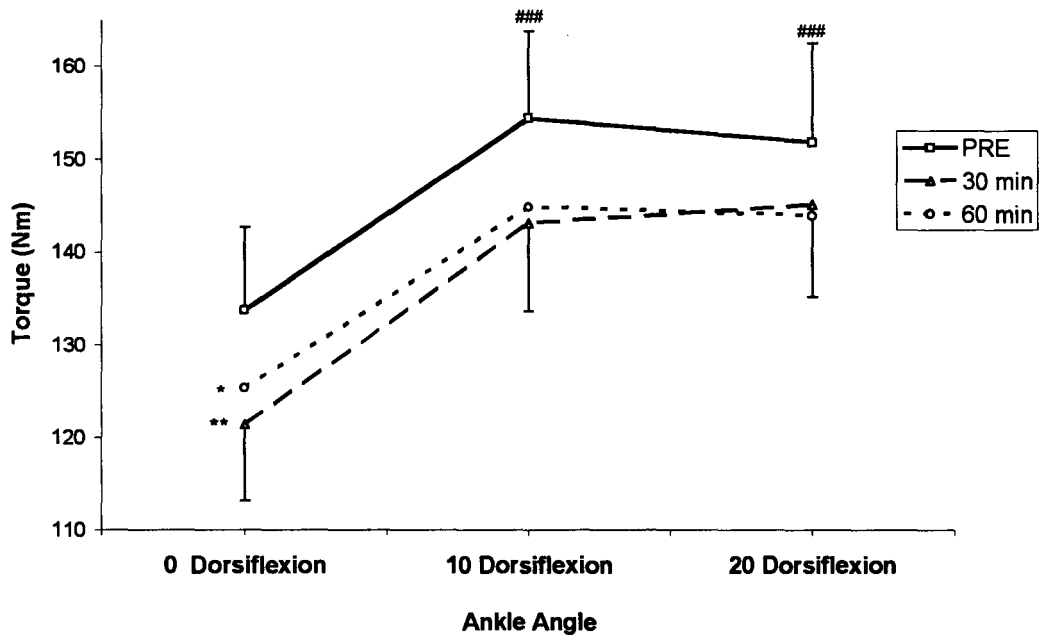


Figure 18. Motor unit activation as a function of joint angle in the passive stretch PSmax condition. # significantly lower activation at 20°D compared to 0°D and 10°D, $P < 0.05$. Values are means + SE.

Motor Unit Activation

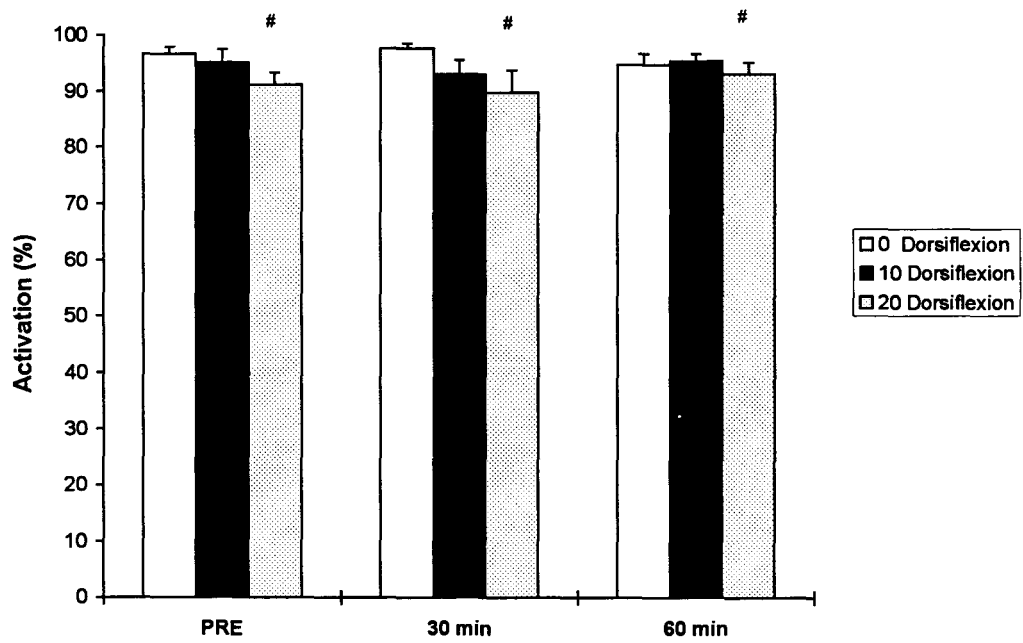
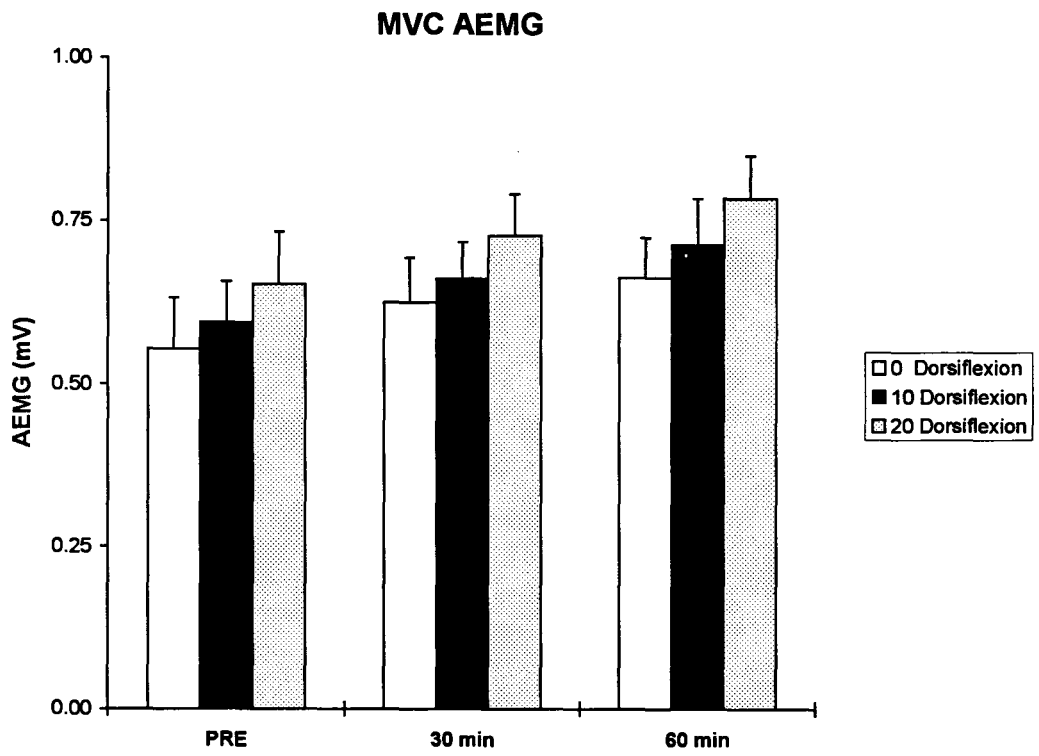


Figure 19. AEMG in MVC's at three joint angles in the PSmax condition. There is a significant effect of angle ($P < 0.005$) and time (0.0005), but no interaction of condition x time. Values are means + SE.



Peak Twitch Torque. Twitch contractile measures in Exp 1, may have indicated twitch potentiation at PRE after subjects' walk to the lab for testing. Therefore for Exp 2, a half hour 'rest' period was included in the testing protocol once the subjects arrived at the lab. This caused minimal effect on PTT (Exp 2 vs Exp 1; 15.6 (0.9 vs 15.8 (0.7) and TPT (114.7 (4.4 vs 114.1 (3.9) but allowed HRT to slow (111.8 (5.3 vs 102.6 (5.1, $P < 0.05$) to a value similar to the POST Con value from Exp 1 (114.2 (5.5, N.S.).

After PSmax, the normal PTT main effect for joint angle of $10^{\circ}\text{D} > 20^{\circ}\text{D} > 0^{\circ}\text{D}$ ($P < 0.00005$) was temporarily altered (Figure 20). At POST, PTT at 20°D was 1.6% greater than PTT at 10°D (N.S.), whereas at PRE the PTT at 10°D was 6.9% greater than PTT at 20°D ($P < 0.0005$). This 'shift' in the force curve to a more optimal testing angle of 20°D at POST had disappeared by 15 min.

The twitch to MVC ratio indicated no main effect for time, but a significant interaction revealed that the twitch to MVC relation was significantly reduced from PRE at 30 min ($P < 0.01$) and 60 min ($P < 0.005$) for the 20°D testing angle only (Figure 21).

Time related contractile properties. TPT and HRT were momentarily altered at POST following PSmax concurrent with the 'shift' of optimal testing angle from 10°D at PRE to 20°D at POST and back to 10°D by 15 min. All other responses from 15 min to 60 min were consistent to the findings of Exp 1.

Muscle compound action potential (M-wave). The significant joint angle effect for M-Wave ($20^{\circ}\text{D} < 0^{\circ}\text{D}$; $P < 0.05$) was in the opposite direction to the AEMG joint angle effect ($20^{\circ}\text{D} > 0^{\circ}\text{D}$; $P < 0.005$). Despite the minor angle effect, M-wave exhibited the same relation over time as Exp 1 by decreasing at POST ($P < 0.0005$), recovering by 15 min (N.S.) and increasing by 45 min ($P < 0.005$) and at 60 min ($P < 0.0005$) (Figure 22). When expressed relative to the PTT, the M-wave to Twitch ratio produced an interaction ($P < 0.0005$), likely because of the altered peak twitch force curve at POST.

AEMG:M-wave ratio increased 9.6% from PRE to 60 min ($P < 0.05$). AEMG:M-wave ratio showed a joint angle effect so that the ratio at 20° (was greater than at 10°D ($P < 0.05$) and at 0°D ($P < 0.001$) although there were no interactions of time x joint angle (Figure 23).

Figure 20. Peak Twitch torque (PTT) tested at ankle angles 0°D, 10°D, 20°D following maximal Passive Stretch (PSmax). The PTT curve indicates a temporary 'shift' of peak twitch optimal angle from 10°D at PRE to 20°D at POST and back to 10°D by 15 min. +++ indicates an interaction exhibited at POST where the difference between 10°D and 20°D is no longer significant ($P > 0.05$). ** significantly different from PRE, $P < 0.005$; * $P < 0.05$ (mean of three joint angles). ### significantly different from PTT at 10°D, $P < 0.0005$; # $P < 0.05$ (mean of all time points). Values are means + SE. Some error bars are excluded for clarity.

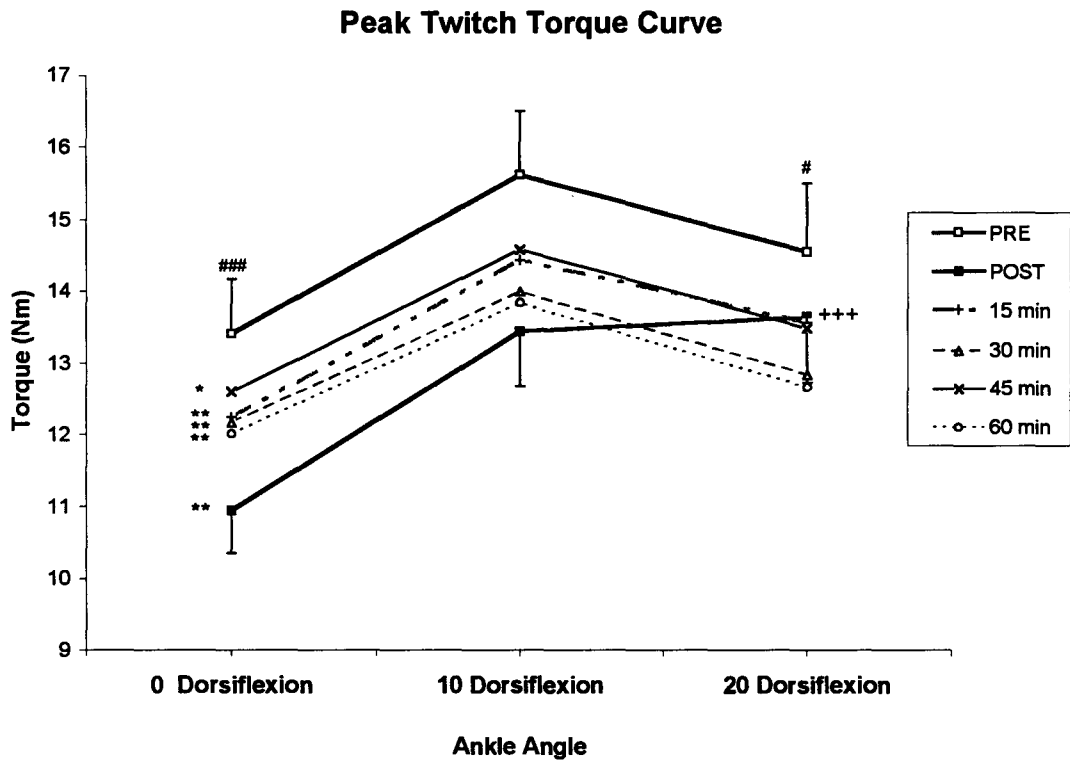


Figure 21. Twitch to MVC ratio at three joint angles following maximal passive stretch (PSmax). ** significantly different from PRE value, $P < 0.005$; * $P < 0.01$. Values are means + SE.

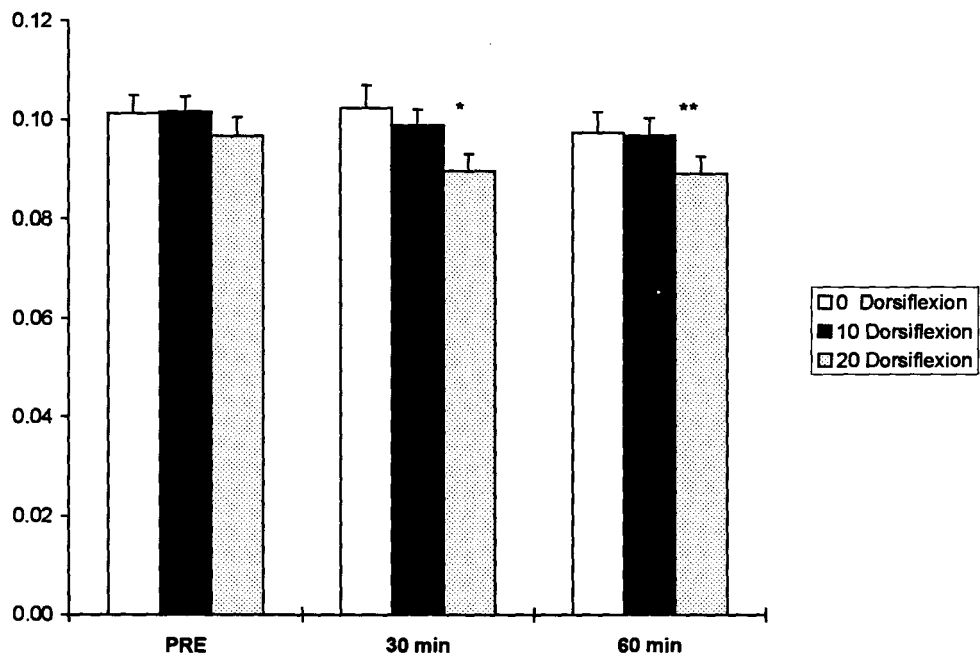
Twitch to MVC ratio

Figure 22. Twitch peak-to-peak M-Wave tested at ankle angles 0°D (□), 10°D (■) and 20°D (Δ) following in the maximal Passive Stretch (PSmax) condition. *** significant main difference from PRE values (mean of three joint angles) at $P < 0.0005$, ** $P < 0.005$. # significantly lower M-wave than 0°D. Values are means and SE. Some error bars have been omitted for clarity.

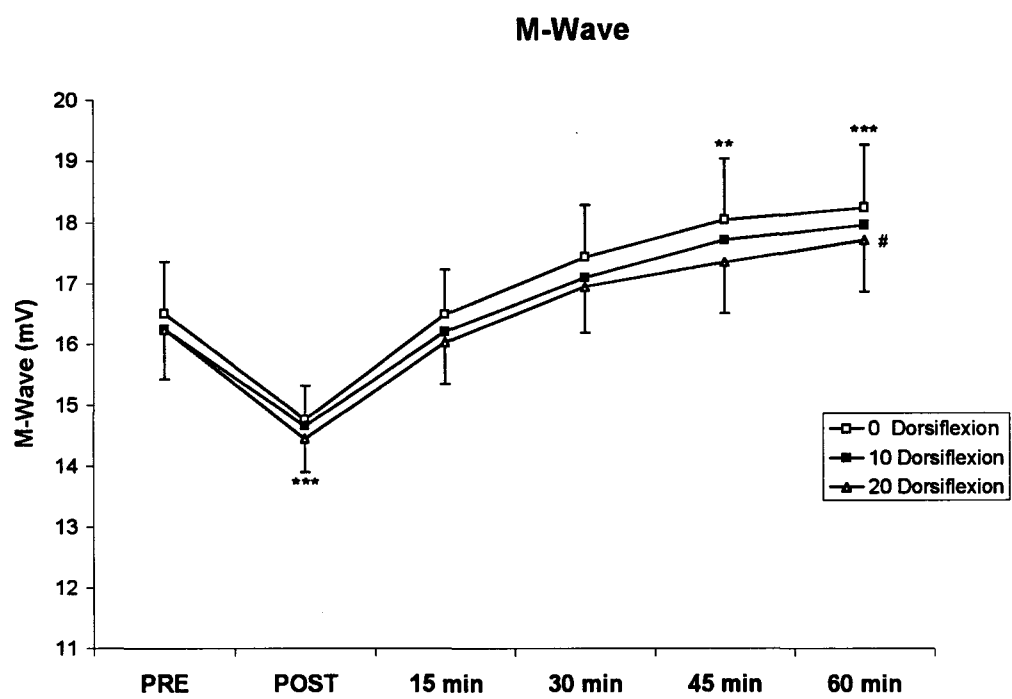
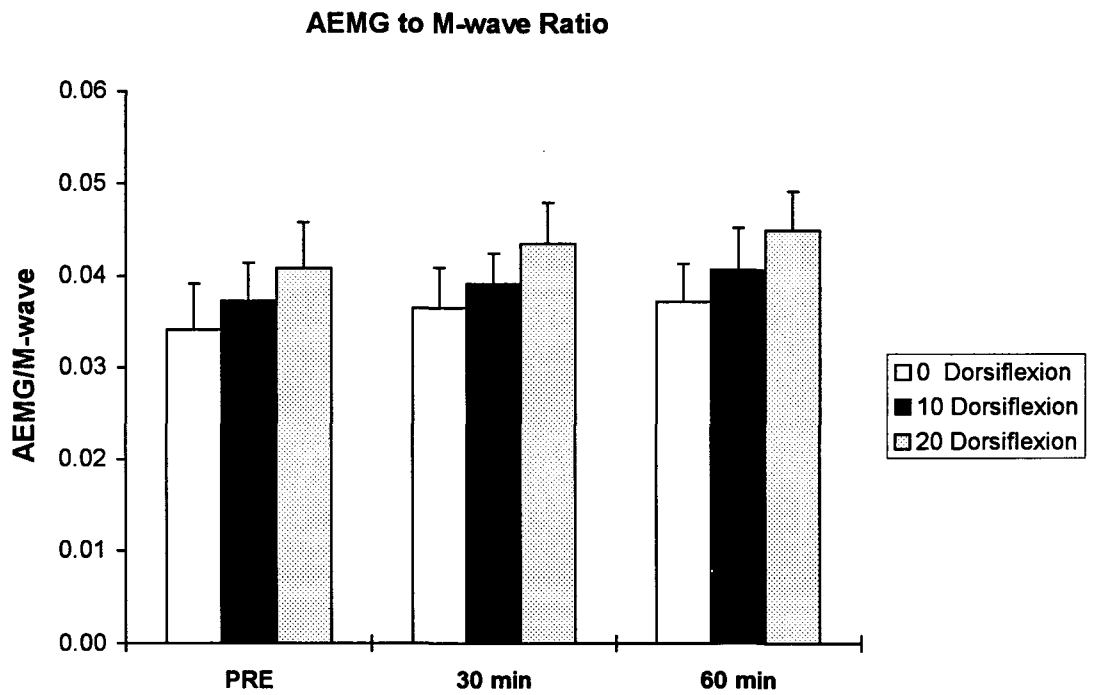
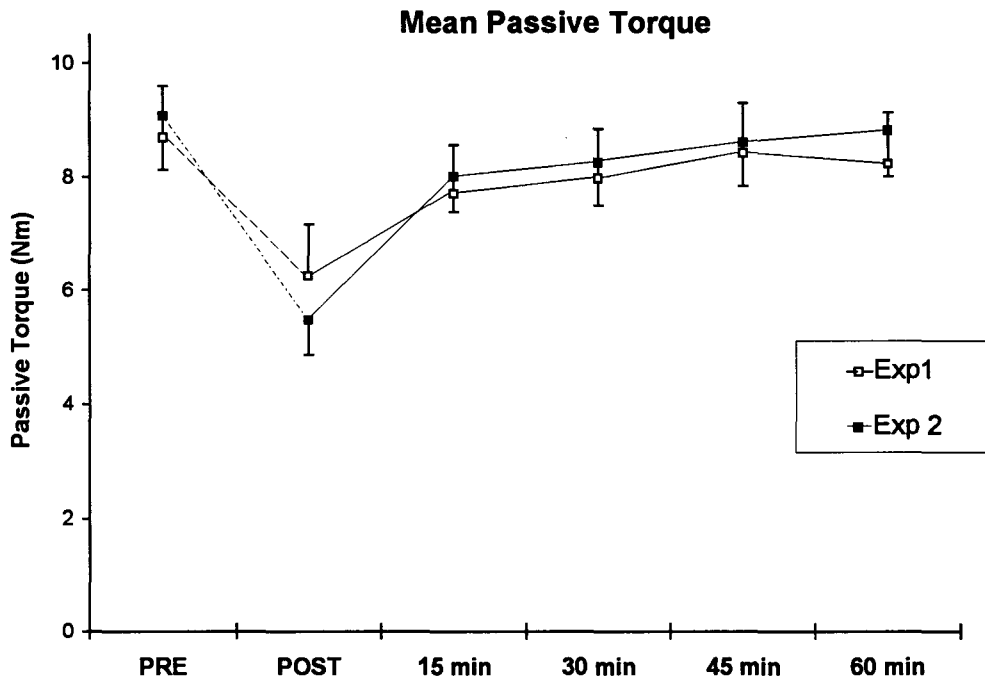


Figure 23. AEMG to M-wave ratio for three joint angles in the passive stretch (PSmax) condition. Main effects are discussed in the text. There was no interaction of time x condition. Values are means + SE.



Muscle Stiffness (Passive Torque). Mean passive torque changes due to PSmax were not significantly different between Exp 1 and Exp 2 ($P = 0.62$), indicating that the elastic response to PSmax was similar for the repeated trials (Figure 24).

Figure 24. Comparison of mean passive torque responses from maximal passive stretch (PSmax) between experiment 1 (Exp 1) and experiment 2 (Exp 2). Effects for time are as shown in the results of Exp 1. Values are means (SE. Some error bars are omitted for clarity.



Summary of Experiment 2. In the relative joint angle trial, PSmax caused a temporary alteration fo the twitch torque-joint angle relation directly POST to peak at a joint angle with a longer muscle length. This temporary alteration was not exhibited in MVC testing measured 30 min following the stretch bout. Alteration of the MVC torque-joint angle relation may have occurred at time points prior to 30 min, although this effect was not tested in this experiment because of the confounding effects of reduced activation in MVC's observed after PSmax in Exp 1. The second PSmax trial confirmed the M-wave alterations due to PSmax, as well as stiffness changes with time. Although some differences were observed with activation and EMG for the three joint angles tested, there were no significant interactions of joint-angle with time due to PSmax, other than the brief alteration of PTT.

4.0 DISCUSSION

Thirty minutes of maximal passive stretch of the human plantarflexor muscles resulted in a 25% loss in maximum voluntary force. The immediate loss in force was partly due to reduced activation and partly due to compromised muscle force generating capacity. By 15 min of recovery when full activation of the plantarflexors has been restored, muscle force generating capacity in an MVC was still compromised so that it remained 8% - 12% below PRE values up to one hour following the stretch. Any significant reduction in maximal force generating capacity following PSmax is a relevant finding. The results of the present study corroborate two recent reports that stretching compromises maximum voluntary force in the muscles participating in the pre-activity stretching routine (Kokkonen & Nelson, 1996; Nelson *et al.* 1996).

Depression of plantarflexor MVC directly following PSmax was associated with a significant increase in interpolated twitch torque (ITT), an indication of reduced muscle activation (Belanger & McComas, 1981). Although the relation of ITT to activation may be non-linear and exponential with declining extra torque (Dowling *et al.* 1994), the indication is that activation decreased following PSmax. Using a formula by Duchateau (1995) to account for force decrements as either neural or muscular in origin, we estimated that 60% of the 25% reduction in MVC directly following PSmax was neural mediated and 40% of the reduction originated in muscle. By 30 min, reduced activation

accounted for only ~1% of the 10% decrement in MVC. It is important to note that the 'Duchateau' formula relates activation to an assumed 100% activation, which is an extrapolated maximum muscle force larger than that achieved by voluntary effort. This assumption has been challenged (Dowling *et al.* 1994). Nevertheless, application of the 'Duchateau' formula indicates that the immediate PSmax - induced force decrement is both neural and contractile in origin.

A response that could contribute to activation failure following PSmax is the Golgi tendon reflex. This autogenic inhibition occurs when the Golgi tendon organs (GTOs) located at myotendon junctions, detect high force combined with muscle lengthening. The GTOs' feedback inhibit agonist activation to lower force production and reduce potentially injurious strain on the muscle. Kokkonen and Nelson (1996) postulate that the strength decrease following acute static stretching in humans could be related to the GTO inhibitory action. An extremely intense stretch is necessary to activate GTOs (Houk *et al.* 1971), GTO discharge rarely persists during maintained muscle stretch, and the inhibitory effects are momentary (Alter, 1996). The fact that peak passive torques in PSmax averaged ~ 28% of MVC (range: 21-45 % of MVC), and the drop in activation occurred at time points after cessation of the stretch, make the possibility of high GTO discharge unlikely in this experiment. Therefore, mechanisms other than GTO feedback could also serve to reduce voluntary activation in this experiment.

Mechanoreceptor (Type III afferent) and nociceptor pain feedback (Type IV afferent) can reduce central drive (Mense & Meyer, 1985; Bigland-Ritchie *et al.* 1986a).

The sensation of stretch and discomfort associated with the stretch protocol could cause temporary activation failure, however any perceptions of discomfort or pain were not present during the post-stretch MVC's. Some subjects commented that their muscle 'just didn't want to contract' despite maximal voluntary effort. The impaired activation directly POST was only temporary, whereas the decline in MVC persisted. Thus, reduced muscle force generating capacity played the dominant role in the later stages following PSmax.

Simpson *et al.* (1996) proposed that fatigue can cause central activation failure and local metabolic effects to reduce force. The initial activation failure and the force decrements that persisted following in this experiment could then be thought to result from fatigue. The normal response to muscle elongation is the stretch reflex which is a feedback loop from muscle spindles that cause agonist contraction resisting the stretch. This reflex can be adjusted through gamma activation, to modulate the amount of lengthening sensed by the muscle spindles. During slow velocity stretches this modulation can limit afferent feedback and thus limit agonist contraction. Silent EMG activity during passive stretching indicates a lack of stretch reflex response. We confirmed that the slow stretching procedure performed in this experiment was truly 'passive' with no EMG activity, as has been achieved by other researchers using stretch protocols in human subjects (Condon & Hutton, 1987; Magnusson *et al.* 1995; Magnusson *et al.* 1996a; McHugh *et al.* 1992; Moore & Hutton, 1980). Our two subjects that exhibited EMG activity during PSmax were not included in the analysis. Therefore, fatigue could not play a major role in the responses to PSmax.

PSmax produced a marked decrease in muscle passive torque (force) measured at the same absolute joint angles after PSmax, confirming previous observations (Toft *et al.* 1989a; Toft *et al.* 1989b). The reduction in passive torque is a result of the muscle lengthening during the stretch, so that when returned to the same absolute joint angle after the stretch, the muscle is effectively at a shorter muscle length at the same absolute test angle than before the stretch. This would place the muscle in a different point in the passive torque curve and would exhibit as lowered passive torque following stretch. Slow passive stretch of the human plantarflexors has been observed to directly lengthen the muscle belly and not the tendon (Halar *et al.* 1978). This effect was confirmed for our experiment by B-mode ultrasound which measures muscle fascicle lengths (Kawakami *et al.* 1995). Using a similar PSmax protocol in a single subject, PSmax was observed to facilitate muscle fascicle elongation of 8 mm, 8 mm and 2 mm for the soleus, lateral gastrocnemius, and medial gastrocnemius muscles respectively. The hypothesis in the present study was that the lengthened muscle fascicles would be in a less optimal portion of the length-tension curve when returned to the same absolute testing angle (as in Exp 1) after PSmax, but may not be compromised at a joint angle relative to the increase in muscle length (as in Exp 2). We observed that PSmax altered the twitch torque-joint angle relation, such that the greatest torque occurred at greater ankle dorsiflexion directly POST. The alteration was short-lived; the PRE torque-joint angle relation was restored by 15 min, even though passive torque was still significantly decreased. The MVC torque curve measured at 30 min, was not influenced by the temporarily reduced passive torque,

since the trend to shift the peak in the MVC torque curve was not significant ($P = 0.20$). Passive torque was below PRE at 30 and 60 min but this observation did not reach significance ($P = 0.057$).

We measured evoked twitch contractile properties in this experiment to provide a picture of contractile performance following PSmax. Twitch contractile properties were probably influenced by changes in muscle stiffness as previously discussed, because mean passive torque (muscle stiffness) significantly correlated with PTT. Caldwell (1995) has determined through computer modeling that twitch force becomes smaller and contraction time slower due to increased series elasticity. PTT did decrease due to PSmax, although contractile speed generally increased. Sale *et al.* (1982) showed that shortening the plantarflexors caused PTT to decline and contraction and relaxation times to become shorter. Elongating the muscle during PSmax may effectively result in testing a shorter muscle than at PRE when the muscle is returned to the same absolute joint position after the stretch. This temporary effect was confirmed in Exp 2, where twitch contractile properties tested at 10°D POST were more representative of the PRE 0°D values. PSmax produced a temporary 'shift' in the passive torque curve down and to the right which results in a temporary 'shift' in the PTT length-tension relation at POST. There were no significant observations of twitch measures that would be inconsistent with this explanation.

Elongation of the muscle-tendon unit in this experiment was facilitated by stress relaxation and tissue 'creep'. Stress relaxation indicates a decay in passive torque over

time for a given stretched muscle length. Stress relaxation is viscoelastic and purely mechanical in nature since the torque decay occurs outside of the influence of EMG activity and has also been observed to not differ between people with varying degrees of flexibility (McHugh *et al.* 1992; Toft *et al.* 1989a), is repeatable for the same subjects on the same day or different days (Halbertsma, 1994; Magnusson *et al.* 1996b; Toft *et al.* 1989b), and does not change following maximal concentric or eccentric contractions (Magnusson *et al.* 1996a). Viscous or hydraulic 'piston-like' elements and elastic 'spring-like' elements within the muscle are taken up early in a single stretch (Toft *et al.* 1989b) or in a stretch routine (Taylor *et al.* 1990). Creep results from maintained tissue strain which causes a reorientation of the supporting connective and soft tissue supporting structures of the muscle to more ordered (i.e. parallel) arrays (Purslow, 1989) which allows muscle lengthening over time. Fifty-seven percent of the joint angle increase (i.e. muscle elongation) occurred in the first four stretches of this 13 stretch protocol, or 71% of lengthening occurred in the first 4 out of 10 stretches. This implies that stress relaxation occurred early in PSmax and that creep may have allowed angular displacement in the later stages of PSmax. If recovery from PSmax was similar to the lengthening response during PSmax, the rapid and prolonged phases of stiffness recovery may be related to the muscle elastic recoil. Rapid and slow stages of stiffness recovery following PSmax would then be recovery from stress relaxation and tissue creep respectively. This would explain why a temporary shift in the twitch-torque curve was observed directly POST, but was not observed in twitch or MVC testing at time points after 15 min.

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Relative joint angle testing was done in 10° increments between 0°D and 20°D. It is possible then that the average 'shift' in the length-tension curve due to an average seven degree increase in maximal dorsiflexion may have fallen between the MVC testing angles. It was inappropriate in this study to test at more joint angles due to the time course for recovery and possible confounding effects of additional contractions. The shift 'down' in MVC strength was significantly below PRE values in Exp 2 until 60 min ($P < 0.05$) which points to causes other than a possible shift in the length-tension relation, for compromised force following PSmax.

One other possible relation between muscle stiffness and contractile performance, it is indicated in a study by Garfin *et al.* (1981) which evaluated the effects of fascia and compartment pressure on force production in contractions of dog hindlimb. Garfin *et al.* found that using a surgical fascial release to apply a small slit in the epimysium resulted in a 15% reduction in force produced and a 50% lower compartment pressure during the contraction. The extended creep observed with PSmax may have lowered fascia stiffness to a point which reduced force production in the stages of recovery. The fascia could serve as the 'weightlifting belt' to the muscle, to facilitate external translation of maximal force. A 'weightlifting belt' supports stabilizing muscles of the trunk during weightlifting serving to increase maximum force exerted at the extremities in an weight training exercise. If the 'weightlifting belt' is loose, maximal force production may be compromised. This situation could occur in a single muscle if the fascia is 'loose' from passive stretching and reduced muscle stiffness, as observed in this experiment.

Aside from the acute changes to muscle stiffness, alteration of the connective tissue by muscle damage has been previously proposed to contribute to force production loss with passive stretch protocols in animals (Armstrong *et al.* 1993; Lieber *et al.* 1991). Creatine kinase enzyme activity is used as a marker of exercise-induced muscle damage (EIMD) and was observed to increase by 62% after seventeen minutes of passive hamstring/low back stretching in humans (Smith *et al.* 1993) and by 250% after acute stretching in chickens (Ashmore *et al.* 1988). It is unlikely however, that contractile element damage contributed to the force decrement in the present experiment because maximal force production is restored to 100% at twenty-four hours post PSmax (Fowles, unpublished observations - appendix 1). Force decrements due to eccentrically-induced myofiber injury take between 5 and 14 days to recover to normal values (Ebbeling & Clarkson, 1989).

An interesting finding in this study was the effect of PSmax on EMG in recovery. Evoked M-wave and voluntary EMG both followed a similar pattern. PSmax caused a drop in EMG at POST which recovered over time and elevated significantly above PRE values by 60 min. These effects were more apparent when elevated EMG was presented relative to compromised force production (i.e., M-wave:twitch ratio, AEMG:MVC ratio). MVC EMG can be affected by central drive, motorneuron excitability (not measured in this experiment) and muscle fiber potential. We observed reduced activation directly following PSmax and it is possible that heightened activation with successive MVC's may be responsible for the increased EMG in recovery. It is unlikely that motorneuron

excitability was affected since other researchers have uncovered that motorneuron excitability (as indicated by the Hoffman reflex or H-reflex) reduces by 80% directly after the onset of a ramp or static passive stretch, recovers moderately during the stretch (Ballegaard *et al.* 1991; Crone & Nielsen, 1989; Guissard *et al.* 1988), then is quickly restored to normal values within 10-15 s after cessation of the stretch (Crone & Nielsen, 1989; Nielsen *et al.* 1993; Guissard *et al.* 1988). Motorneuron excitability would have little effect on the contractile measures made minutes after stretching. The evoked M-wave should not be affected by central factors so PSmax may have affected muscle membrane excitability. M-wave area changes (data not presented) were identical to changes in peak to peak M-wave so the M-wave alteration was not due to a change in shape. M-wave amplitude can differ with changes in joint position in the ankle plantarflexors (Sale *et al.* 1982). M-wave measurements at three joint angles in Exp 2 did show a small but significant difference, although M-waves at all three joint angles showed the same trend. It is possible that muscle length changes due to PSmax may have changed the geometric orientation of the electrodes to the muscle to affect the M-wave (Merletti *et al.* 1992), although representing EMG as the AEMG to M-wave ratio should account for differences in electrode positioning. There were no significant changes in AEMG to M-wave ratio when presented relative to the control condition so the trends indicate that EMG was affected in some way, as opposed to just electrode positioning changes. When represented relative to joint angle as in Exp 2, the AEMG to M-wave ratio did not differ between angles over time ($P = 0.98$).

It is possible that local ion imbalances may have resulted from the intensive stretching which would affect the muscle membrane potential directly post PSmax and in time points of recovery. Diminished force, reduced M-wave size, and reduced voluntary EMG can result from fatiguing contractions under ischemic conditions (McComas, 1996). As well, M-wave can potentiate during repeated MVC's (Hicks *et al.* 1989) and following tetanic fatiguing contractions (Fitch & McComas, 1985; Hicks & McComas, 1989). M-wave size reflects the muscle membrane potential and size of the muscle action potential. Reduced M-wave amplitude results from an increase in intracellular Na^+ and increased extracellular K^+ which reduces membrane potential, whereas M-wave potentiation is due to increased electrogenic Na^+/K^+ pump activity (Hicks & McComas, 1989) to prevents the muscle fibers from depolarizing and becoming inexcitable with fatigue (McComas *et al.* 1994). Increased intracellular Ca^{2+} has been considered to contribute to a fatigued state (McComas, 1996) and has been observed after brief (Snowdowne, 1986) or prolonged passive stretch in animals (Armstrong *et al.* 1993). The increase in intracellular Ca^{2+} with stretch may have the same effect on membrane potential as the increased intracellular Ca^{2+} and increased extracellular K^+ in the fatigued state, to originally reduce the membrane potential and subsequently cause a response to increase membrane potential.

The mechanism to alter the membrane potential can not be determined with the results of this experiment, but can be inferred from known dynamic events within the cell, as presented by McComas (1996). The proposed mechanism for the alteration of membrane potential with passive stretch is as follows: 1) Shear force from stretch disrupts

the mechanical link of the dyhydropyridine-ryanodine (DHP-RYR) complex at the T-tubule/sarcoplasmic reticulum (SR) interface; 2) separation of the DHP 'plug' from RYR allows the escape of Ca^{2+} from the SR down its concentration gradient and into the cytosol; 3) increased intracellular Ca^{2+} stimulates restoration of normal intracellular Ca^{2+} concentration, and Ca^{2+} is exchanged with Na^+ on a three-to-one ratio in the non-ATP dependent $\text{Ca}^{2+} / \text{Na}^+$ antiporter at the sarcolemma; 4) the exchange of one Ca^{2+} out for every three Na^+ in results in a net influx of one positive charge into the cell which decreases membrane potential; 5) increased intracellular Na^+ concentration stimulates the Na^+ / K^+ pump to extrude two Na^+ ions out for three K^+ ions into the cell; 6) the increased intracellular K^+ concentration increases membrane potential and restores muscle membrane excitability.

The justification for the proposed mechanisms rely on two assumptions: 1) Intracellular Ca^{2+} concentration increases with the degree of stretch (Snowdowne, 1986) and therefore, the resultant increase in Na^+ concentration would continue throughout the passive stretch and would recover quickly after relief of the stretch and removal of the stimulus; 2) the SR Ca^{2+} ATPase pump which normally reduces the concentration of intracellular Ca^{2+} following contraction, is not as active during a rested or 'passive' state, and therefore, restoration of intracellular Ca^{2+} due to passive stretch would be accomplished by mechanisms other than the SR Ca^{2+} ATPase pump.

The mechanisms affecting M-wave and EMG can not be explained with the results of this experiment. The similarity to the fatigued state can be highlighted, as well as the

fact that 'passive stretch' should not produce classic fatigue. Precautions were taken to avoid EMG activity during PSmax and to limit any ischemia potentially caused by the stretch or the testing apparatus itself. The fact that the time course for M-wave changes in this experiment (~1 h) are much different than the 3-12 min recovery times from intense fatiguing contractions (Hicks *et al.* 1989; Hicks & McComas, 1989; McComas *et al.* 1994) also raises the question that other mechanisms such as excitation-contraction uncoupling may have contributed to force loss following PSmax. The proposed ionic alterations within the cell may relate to mechanisms involved in stimulating muscle protein synthesis in muscle cells undergoing chronic stretch, where stretch acts as a potent stimulator to increase sarcomeres in series and increase muscle cross-sectional area.

SUMMARY AND CONCLUSIONS

It is concluded that an intense prolonged stretch of the ankle plantarflexors reduces maximum voluntary force for up to one hour following the stretch. Decreased maximum voluntary force directly after stretch was partly due to reduced activation and partly due to reduced muscle force generating capacity. Voluntary activation is quickly recovered, as is any shift in the muscle torque-joint angle relation encouraged by the lengthening stretch. Complete recovery of force generating capacity is more prolonged, similar to the recovery in muscle stiffness. Stiffness recovery may represent the elastic mechanical recoil from the stretching activity. Elements contributing to muscle stiffness may 'stabilize' the muscle to generate force and any alteration of those elements may compromise force production.

It must be noted that this experiment simulated an intense maximal stretch far beyond what an athlete may attempt either pre-activity or as part of a flexibility training program. It has been identified in the literature that the intensity and duration of stretching required to produce lasting stiffness changes in muscle is unknown (Magnusson *et al.* 1996a), although the upper conceivable limit of stretching performed here was not sufficient to produce significant muscle stiffness changes lasting one hour. By its viscoelastic nature, muscle has a strong tendency to return to its resting or genetically and biomechanically determined length. It may be questionable to oppose this tendency with the use of stretching to enhance performance, when performance can be compromised by

altering the fine dynamic balance of neural, architectural, and electrophysiological factors that exists in muscle to create force.

The results of this study indicate that intense stretching within one hour of activity requiring maximum strength, may be detrimental to performance in activities requiring maximal force production. Generalizations made from the results of this study to aid in design of a normal pre-activity stretching routines are difficult because of the intense nature of the stretch performed here. The PSmax routine performed would be the absolute upper limit of what a stretching routine may involve and further study into the effects of stretching on strength performance may involve a less intense stretch protocol. Additionally, future study may combine stretching with a light to moderate warm-up, to determine if the potentially detrimental effects of stretching are negated by the positive effects of warm-up.

REFERENCES

- ALTER, M. J. (1996). *Science of Flexibility*. Champaign, IL: Human Kinetics.
- ARMSTRONG, R. B., DUAN, C., DELP, M. D., HAYES, D. E., GLENN, G. M. & ALLEN, G. D. (1993). Elevations in rat soleus muscle [Ca^{2+}] with passive stretch. *Journal of Applied Physiology* **74**, 2990-2997.
- ASHMORE, R. C., HITCHCOCK, L. & LEE, Y. B. (1988). Passive stretch of adult chicken muscle produces myopathy remarkably similar to hereditary muscular dystrophy. *Experimental Neurology* **100**, 341-353.
- BALLEGAARD, M., HULTBORN, H., ILLERT, M., NIELSEN, J. & PAUL, A. (1991). Slow passive stretches of a muscle depresses transmission of its monosynaptic reflex. *European Journal of Neuroscience [Suppl 4]*, 298
- BELANGER, A. Y. & MCCOMAS, A. J. (1981). Extent of motor unit activation during effort. *Journal of Applied Physiology* **51**, 1131-1135.
- BIGLAND-RITCHIE, B. R., DAWSON, N. J., JOHANSSON, R. S. & LIPPOLD, O. C. J. (1986a). Reflex origin for slowing of motorneurone firing in fatigue of human voluntary contractions. *Journal of Physiology* **376**, 451-459.
- BIGLAND-RITCHIE, B., FURBUSH, F. & WOODS, J. J. (1986b). Fatigue of intermittent submaximal voluntary contractions: central and peripheral factors. *Journal of Applied Physiology* **61**, 421-429.
- BLOOMFIELD, J., ACKLAND, T. R. & ELLIOTT, B. C. (1994). *Applied Anatomy and Biomechanics in Sport*. Melbourne: Blackwell Scientific Publications.
- CALDWELL, G. E. (1995). Tendon elasticity and relative length: effects on the Hill two-component muscle model. *Journal of Applied Biomechanics* **11**, 1-24.
- CONDON, S. M. & HUTTON, R. S. (1987). Soleus muscle electromyography activity and ankle dorsiflexion range of motion during four stretching procedures. *Physical Therapy* **67**, 24-30.
- CRONE, C. & NIELSEN, J. (1989). Methodological implications of the post activation depression of the soleus H-reflex in man. *Exp. Brain Res.* **78**, 28-32.

DEVRIES, H. A. (1980). *Physiology of Exercise for Physical Education and Athletics*. Dubuque, Iowa: W.C. Brown.

DINTIMAN, G. B. (1964). Effects of various training programs on running speed. *Research Quarterly for Exercise and Sport* **35**, 456

DOWLING, J. J., KONERT, E., LJUCOVIC, P. & ANDREWS, D. M. (1994). Are humans able to voluntarily elicit maximum muscle force? *Neuroscience Letters* **179**, 25-28.

DUCHATEAU, J. (1995). Bed rest induces neural and contractile adaptations in triceps surae: a clinically relevant case study. *Medicine & Science in Sports and Exercise* **27**, 1581-1589.

EBBELING, C. B. & CLARKSON, P. M. (1989). Exercise-induced muscle damage and adaptation. *Sports Medicine* **7**, 207-234.

FITCH, S. & MCCOMAS, A. J. (1985). Influence of human muscle length on fatigue. *Journal of Physiology* **362**, 205-315.

FOX, E. L., BOWERS, R. W. & FOSS, M. L. (1989). *The Physiological Basis of Physical Education and Athletics*. Dubuque, Iowa: Wm. C. Brown Publishers.

FUGL-MEYER, A. R., SJOSTROM, M. & WAHLBY, L. (1979). Human plantarflexion strength and structure. *Acta Physiol. Scand.* **107**, 47-56.

GARFIN, S. R., TIPTON, C. M., MUBARAK, S. J., WOO, S. L. Y., HARGENS, A. R. & AKESON, W. H. (1981). Role of fascia in maintenance of muscle tension and pressure. *Journal of Applied Physiology* **51**, 317-320.

GUISSARD, N., DUCHATEAU, J. & HAINAUT, K. (1988). Muscle stretching and motoneuron excitability. *European Journal of Applied Physiology* **58**, 47-52.

HALAR, E. M., STOLOV, W. C., VENKATESH, B., BROZOVICH, F. V. & HARLEY, J. D. (1978). Gastrocnemius muscle belly and tendon length in stroke patients and able-bodied persons. *Archives of Physical Medicine & Rehabilitation* **59**, 476-484.

HALBERTSMA, J. P. K. (1994). Stretching exercises: effect on passive extensibility and stiffness in short hamstrings of healthy subjects. *Archives of Physical Medicine & Rehabilitation* **75**, 976-981.

HEDRICK, A. (1993). Flexibility and the conditioning program. *National Strength and Conditioning Association Journal* **15**, 62-66.

HERMAN, R. & BRAGIN, S. J. (1967). Function of the Gastrocnemius and Soleus muscles. *Physical Therapy* **47**, 105-113.

HICKS, A., FENTON, J., GARNER, S. & MCCOMAS, A. J. (1989). M wave potentiation during and after muscle activity. *Journal of Applied Physiology* **66**, 2606-2610.

HICKS, A. & MCCOMAS, A. J. (1989). Increased sodium pump activity following repetitive stimulation of rat soleus muscles. *Journal of Physiology* **414**, 337-349.

HORTOBAGYI, T., FALUDI, J., TIHANYI, J. & MERKELY, B. (1985). Effects of intense "stretching"-flexibility training on the mechanical profile for the knee extensors and on the range of motion of the hip joint. *International Journal of Sports Medicine* **6**, 317-321.

HOUK, J. C., SINGER, J. J. & GOLDMAN, M. R. (1971). Adequate stimulus for tendon organs with observation on mechanics of the ankle joint. *Journal of Neurophysiology* **34**, 1051-1065.

KAWAKAMI, Y., ABE, T., KUNO, S. Y. & FUKUNAGA, T. (1995). Training-induced changes in muscle architecture and specific tension. *European Journal of Applied Physiology* **72**, 37-43.

KOKKONEN, J. & NELSON, A. G. (1996). Acute stretching exercises inhibit maximal strength performance. *Medicine & Science in Sports and Exercise* **28**, S190(Abstract)

LIEBER, R. L., WOODBURN, T. M. & FRIDEN, J. (1991). Muscle damage induced by eccentric contractions of 25% strain. *Journal of Applied Physiology* **70**, 2498-2507.

MAGNUSSON, S. P., SIMONSEN, E. B., AAGAARD, P., MORITZ, U. & KJAER, M. (1995). Contraction specific changes in passive torque in human skeletal muscle. *Acta Physiologica Scandinavica* **155**, 377-386.

MAGNUSSON, S. P., SIMONSEN, E. B., AAGAARD, P., DYHRE-POULSEN, P., MCHUGH, M. P. & KJAER, M. (1996a). Mechanical and physiological responses to stretching with and without preisometric contraction in human skeletal muscle. *Archives of Physical Medicine & Rehabilitation* **77**, 373-378.

MAGNUSSON, S. P., SIMONSEN, E. B., AAGAARD, P. & KJAER, M. (1996b). Biomechanical responses to repeated stretches in human hamstring muscle in vivo. *The American Journal of Sports Medicine* **24**, 622-628.

- MARSH, E., SALE, D. G., MCCOMAS, A. J. & QUINLAN, J. (1981). Influence of joint position on ankle dorsiflexion in humans. *Journal of Applied Physiology* **51**, 160-167.
- MCCOMAS, A. J., GALEA, V. & EINHORN, R. W. (1994). Pseudofacilitation: a misleading term. [Review]. *Muscle & Nerve* **17**, 599-607.
- MCCOMAS, A. J. (1996). *Skeletal Muscle*. Champaign, Illinois: Human Kinetics.
- MCHUGH, M. P., MAGNUSSON, S. P., GLEIM, G. W. & NICHOLAS, J. A. (1992). Viscoelastic stress relaxation in human skeletal muscle. *Medicine & Science in Sports and Exercise* **24**, 1375-1382.
- MENSE, S. & MEYER, H. (1985). Different types of slowly conducting afferent units in the cat skeletal muscle and tendon. *Journal of Physiology* **363**, 403-417.
- MERLETTI, R., KNAFLITZ, M. & DELUCA, C. J. (1992). Electrically evoked myoelectric signals. *Critical Reviews in Biomedical Engineering* **19**, 293-340.
- MOORE, M. A. & HUTTON, R. S. (1980). Electromyographic investigation of muscle stretching techniques. *Medicine & Science in Sports and Exercise* **12**, 322-329.
- NELSON, A. G., CORNWELL, A. & HEISE, G. D. (1996). Acute stretching exercises and vertical jump stored elastic energy. *Medicine & Science in Sports and Exercise* **28**, S156(Abstract)
- NIELSEN, J., PETERSEN, N., BALLEGAARD, M., BIERING-SORENSEN, F. & KIEHN, O. (1993). H-reflexes are less depressed following muscle stretch in spastic spinal cord injured patients than in healthy subjects. *Exp. Brain Res.* **97**, 173-176.
- NOONAN, T. J., BEST, T. M., SEABER, A. V. & GARRETT, W. E. J. (1993). Thermal effects on skeletal muscle tensile behavior. *The American Journal of Sports Medicine* **21**, 517-522.
- PURSLOW, P. P. (1989). Strain-induced reorientation of an intramuscular connective tissue network: implication for passive muscle elasticity. *Journal of Biomechanics* **22**, 21-31.
- SALE, D., QUINLAN, J., MARSH, E., MCCOMAS, A. J. & BELANGER, A. Y. (1982). Influence of joint position on ankle plantarflexion in humans. *Journal of Applied Physiology* **52**, 1636-1642.

- SHELLOCK, F. G. & PRENTICE, W. E. (1985). Warming-up and stretching for improved physical performance and prevention of sports-related injuries. *Sports Medicine* **2**, 267-278.
- SIMPSON, M. R., BURKE, J. R., DAVIS, J. M. & JACKSON, D. A. (1996). Relationship between peripheral fatigue and the inhibition of neural activation. *Medicine & Science in Sports and Exercise* **28**, S141(Abstract)
- SMITH, C. A. (1994). The warm-up procedure: to stretch or not to stretch. A brief review. *Journal of Orthopaedic & Sports Physical Therapy* **19**, 12-17.
- SMITH, L. L., BRUNETZ, M. H., CHENIER, T. C., MCCAMMON, M. R., HOUMARD, J. A., FRANKLIN, M. E. & ISRAEL, R. G. (1993). The effects of static and ballistic stretching on delayed onset muscle soreness and creatine kinase. *Research Quarterly for Exercise and Sport* **64**, 103-107.
- SNOWDOWNE, K. W. (1986). The effect of stretch on sarcoplasmic free calcium of frog skeletal muscle at rest. *Biochimica et Biophysica Acta* **862**, 441-444.
- TAYLOR, D. C., DALTON, J. D., SEABER, A. V. & GARRETT, W. E. J. (1990). Viscoelastic properties of muscle-tendon units. The biomechanical effects of stretching. *The American Journal of Sports Medicine* **18**, 300-309.
- TOFT, E., ESPERSEN, G. T., KALUND, S., SINKJAER, T. & HORNEMANN, B. C. (1989a). Passive tension of the ankle before and after stretching. *The American Journal of Sports Medicine* **17**, 489-494.
- TOFT, E., SINKJAER, T., KALUND, S. & ESPERSEN, G. T. (1989b). Biomechanical properties of the human ankle in relation to passive stretch. *Journal of Biomechanics* **22**, 1129-1132.
- VANDERVOORT, A. A., QUINLAN, J. & MCCOMAS, A. J. (1983). Twitch potentiation after voluntary contraction. *Experimental Neurology* **81**, 141-152.
- VANDERVOORT, A. A. & MCCOMAS, A. J. (1983). A comparison of the contractile properties of the human gastrocnemius and soleus muscles. *Eur. J. Appl. Physiol.* **51**, 435-440.
- VIITASALO, J. T. & KOMI, P. V. (1975). Signal characteristics of EMG with special reference to reproducibility measurements. *Acta Physiologica Scandinavica* **93**, 531-539.

WIKTORSSON-MOLLER, M., OBERG, B., EKSTRAND, J. & GILLQUIST, J. (1983). Effects of warming up, massage, and stretching on range of motion and muscle strength in the lower extremity. *The American Journal of Sports Medicine* 11, 249-252.

WILSON, G. J., ELLIOTT, B. C. & WOOD, G. A. (1992). Stretch shorten cycle performance enhancement through flexibility training. *Medicine & Science in Sports and Exercise* 24, 116-123.

WORRELL, T. W., SMITH, T. L. & WINEGARDNER, J. (1994). Effect of hamstring stretching on hamstring muscle performance. *Journal of Orthopaedic & Sports Physical Therapy* 20, 154-159.

APPENDIX 1 - Pilot study

**Contractile deficits following an acute bout of maximal passive stretch
in human subjects.**

completed June 1996

ABSTRACT

The purpose of this investigation was to determine if an acute bout of maximal passive stretch (PSmax) elicits contractile deficits and symptoms indicative of the delayed-onset-muscle-soreness (DOMS) condition normally associated with eccentrically biased exercise (ECC). Seven active males underwent an acute bout of 30 min of PSmax of the ankle plantarflexors. Contractile measures [maximum voluntary contraction torque (MVC) and interpolated twitch torque (ITT)] were assessed at 10° of dorsiflexion (D) pre-stretch (PRE), post-stretch (POST), 1 hour post-stretch (1 h) and 24 hours post-stretch (24 h). Indirect indicators of muscle damage; mean passive torque of 0°, 10°, and 20°D (PT_⊗) were measured PRE, POST, and 24 h, or at PRE and 24 h; perceived DOMS (1=normal, 10= very, very sore), and creatine kinase activity (CK)]. Means for each time point are compared to PRE. Immediately POST, MVC decreased to 161.5 ± 28.0 N·m from 202.4 ± 25.6 N·m at PRE (P<0.005). ITT indicated that 30% and 70% of the MVC force decrement was due to decreased motor unit activation (MUA) and reduced muscle force-generating capacity, respectively. By 1H, MVC was at 189.8 ± 34.1 N·m and by 24 H, MVC was fully recovered, while MUA had recovered to 98.7% and 100% of PRE, respectively. PT_⊗ is reduced by 16.2% POST (p<0.005) but is quickly elevated above PRE by 1H (PT_⊗ +31.5% above PRE). Minor perceptions of DOMS are observed at 24H (2.2 ± 0.4), concurrent with still elevated PT_⊗ (+21.4%). Two subjects exhibited elevated CK at 24H (N.S.). The data indicate that PSmax results in a short-term reduction in voluntary strength, caused primarily by contractile deficit and partly by impaired neural

activation. Contractile properties may be affected by reduced muscle stiffness. PSmax may damage non-contractile elements of the muscle to elicit some typical DOMS symptoms and minor perceptions of pain.

INTRODUCTION

Researchers speculate that the contractile element alteration normally associated with eccentric exercise-induced muscle damage (EIMD) can be affected by cytoskeletal and connective tissue disruption (see reviews: Armstrong *et al.* 1991; Ebbeling and Clarkson, 1989; Fridén and Lieber, 1992; Smith, 1991; Waterman-Storer, 1991). Cytoskeletal damage is also implicated in contributing to symptoms of delayed onset muscle soreness (DOMS) (Friden *et al.*, 1984; Jones *et al.*, 1987; Stauber *et al.*, 1990). DOMS is a marker of EIMD and has been observed to increase 3-fold in 24 hours after 17 min. of static stretching in humans (Smith *et al.*, 1993). Other common markers of EIMD are elevations in serum creatine kinase (CK) and force production decrements (Ebbeling and Clarkson, 1989), also observed to occur after stretching. A 2.5 fold increase in plasma CK was observed after acute stretch in chickens (Ashmore *et al.*, 1988), and a 62% increase in creatine kinase (CK) enzyme activity was found by Smith *et al.*, (1993) after passive hamstring/low back stretching. Lieber *et al.* (1991) observed a 13% decrement in tetanus force one hour following sham-operated PS on rat hind limb muscles, speculating that damage to the myotendinous junction or breakage of myosin cross-bridges explained

the force production loss. Nelson *et al.* (1996) reported that stretching has a significant negative impact on vertical jump performance in humans without affecting elastic contribution to the jump. Kokkonen and Nelson (1996) report that acute static stretching significantly impairs isometric maximal voluntary contraction (MVC) force in humans, suggesting that the strength decrease could be related to the Golgi tendon organ stimuli causing an inhibitory action of spinal cord neurons of some of the muscle groups involved in the heavy stretching.

It is possible that the force decrement observed following ECC may be contributed by damage to non-contractile components, as directly stressed during PS, or by some other mechanism not related to muscle damage. The purpose of this study was to assess if PS can elicit EIMD and symptoms of DOMS, and to determine the nature of the force decrement following PS as being muscular or neuromuscular in origin.

METHODS

Subjects: Seven male university students [2 experienced weight trainers, 3 active controls (recreational WT, running, and/or sports), 1 triathlete, 1 gymnast] (means; age: 23.4, ht: 176 cm, wt: 77.8 kg) volunteered for the pilot study.

Apparatus: The twitch and boot apparatus used by Sale *et al.* (1982) was employed. Testing was performed with a 90° knee angle, and contractile tests were performed at an

ankle angle of 10°D. Stimulating electrodes were placed to isolate contractile properties of the soleus muscle. Stimulating voltage was individually adjusted prior to testing to maximize muscle twitch amplitude.

Experimental Procedure: Subjects were initially assessed for DOMS (1-10 scale); with movement and palpation (DOMSM, DOMSP), limb girth with (GGas) and without gastrocnemius (GSol), resting ankle joint angle (RJA), range-of-motion (ROM), passive torque (mean torque of 0°, 10°D, and 20°D: PT \bar{x}), and a resting blood sample for creatine kinase activity (CK) followed by pre-stretch contractile measurements in the following order: resting twitch (TW), maximum isometric voluntary contraction (MVC) with twitch interpolation (IT), post activation potentiated twitch (PAP-TW; 10 s MVC, 5 s post MVC), passive torque (PT \bar{x}), and a second MVC with interpolated twitch.

Approximately 10 min after pre-testing, subjects completed the stretching exercise protocol. The contractile testing protocol was repeated immediately post-stretch, and again at 1 h and 24 h. The initial measures were repeated at 1 h and 24 h with the exception of DOMS and CK measures only repeated at 24 h.

10° D was standardized as the “comparative joint angle” for repeated measures testing of contractile characteristics because it is optimal for eliciting twitch responses and is in the plateau of the JA/force twitch curve (Sale *et al.*, 1982) to minimize possible shifts in the length-tension relation induced with PS. Two subjects repeated the protocol with

the modification that post stretch contractile measurements were made at joint angle relative to the increase in ROM and decrease in passive tension (testing at $\sim 17^\circ\text{D}$).

Stretch Protocol (PSmax): Without prior warm-up or stretching, subjects were secured in the apparatus and were passively stretched by the experimenter to a maximum possible dorsiflexed position achievable without pain. The joint angle was then locked into place, and every 2:15 (min:s), the subjects' ankle joint was released for 10 s to a neutral angle, then passively stretched over 5-10 s to a new maximal joint angle for 30 min of maximal passive stretch (PSmax) (10:1 stretch:rest ratio for 33 min of total stretching time).

Performance Measures:

PSmax:	Maximum Joint Angle each interval (Dmax)
	Passive Torque - trace throughout PSmax
Contractile:	Twitch, Interpolated twitch, MVC, PAP twitch @ 10°D
	Twitch contractile measures
Muscle Damage:	DOMS (perceived rating: 1-10 scale; 1 normal - 10 very, very sore)
	CK (serum creatine kinase activity - CK kit Sigma diagnostics)
	Passive Torque (PTx; mean passive torque of 0° , 10°D , 20°D)
	Relaxed joint angle (RANG), range-of-motion (ROM)
	Swelling (circumference by tape measure at muscle belly)

Analysis: Only four subjects completed the blood sample protocol for assessing CK activity. Only 5 subjects completed girth measurements. ANOVA's were used to analyze all parameters except DOMS and CK. DOMS was analyzed using a single sample t-test, CK analyzed using a paired t-test. Post hoc analysis was done with a Tukey HSD. Significance was accepted at $P \leq 0.05$. Mean values are given with standard deviation (SD) unless otherwise indicated.

RESULTS

Stretching Protocol (PSmax): Maximum dorsiflexion joint angle (Dmax) at time zero was $34.7 \pm 3.6^\circ$ of ankle Dorsiflexion (D) and increased 26.2% to $43.8 \pm 4.1^\circ$ D ($p < 0.0005$) by the end of the 30 min of PSmax. Torque transducers were calibrated to zero at 10° plantarflexion (P) where ankle passive tension in plantarflexors is observed to be negligible (Kawakami *et al.*, 1995), so that all measured passive torque is the increase in passive torque from 10° P. Passive torque at the maximum joint angle achieved was phasic, peaking at an average of 52.9 ± 8.5 N·m and decaying by an average of 6.3 ± 2.1 N·m, per 2:15 (min:s) stretch interval.

Contractile Measures: Immediately POST, MVC decreased 20.2% to 161.5 ± 28.0 N·m from 202.4 ± 25.6 N·m at PRE ($P < 0.005$) (Figure 1). By 1 h, MVC was at 93.8% of PRE values and by 24 h, MVC had fully recovered to PRE values. Resting twitch (TW) decreased by 5.9% (N.S.) directly POST, significantly increased to 4.5% above PRE at 1 h ($P < 0.05$) then returned to PRE values at 24 h. Post activation potentiated twitch (PAP-TW) was reduced significantly at POST and at 1 h ($P < 0.005$) and returned to PRE values at 24 h. Interpolated twitch (ITT) was significantly elevated and activation (MUA) was significantly decreased POST compared to all other time points ($P < 0.05$).

Secondary damage indicators: Four of the seven subjects reported mild delayed onset muscle soreness at 24 h, the mean (2.1 ± 1.2) for seven subjects was not significant ($P = 0.067$). There was no difference between perceptions of soreness with movement or palpation. Two subjects' CK values decreased and two subjects' CK values increased due to stretch, for a resultant 19% increase in CK at 24 h (N.S.). Only 4 subjects were assessed for CK activity PRE - 24 h.

Maximum plantarflexion ROM was reduced at 1 h, compensated by a concurrent increase in maximum dorsiflexion ROM, so that total range of motion remained relatively constant over post time points. RANG changed non-significantly to a more dorsiflexed position from 11.5 ± 4.0 °P at PRE to 8.6 ± 4.6 °P at 1 h. At 24 h, RANG was returning to PRE values, although all changes in RANG and ROM were non-significant. Swelling was marginal and non-significant (GSol +1.7%, GSG +1.3%), although only 5 subjects were assessed for swelling.

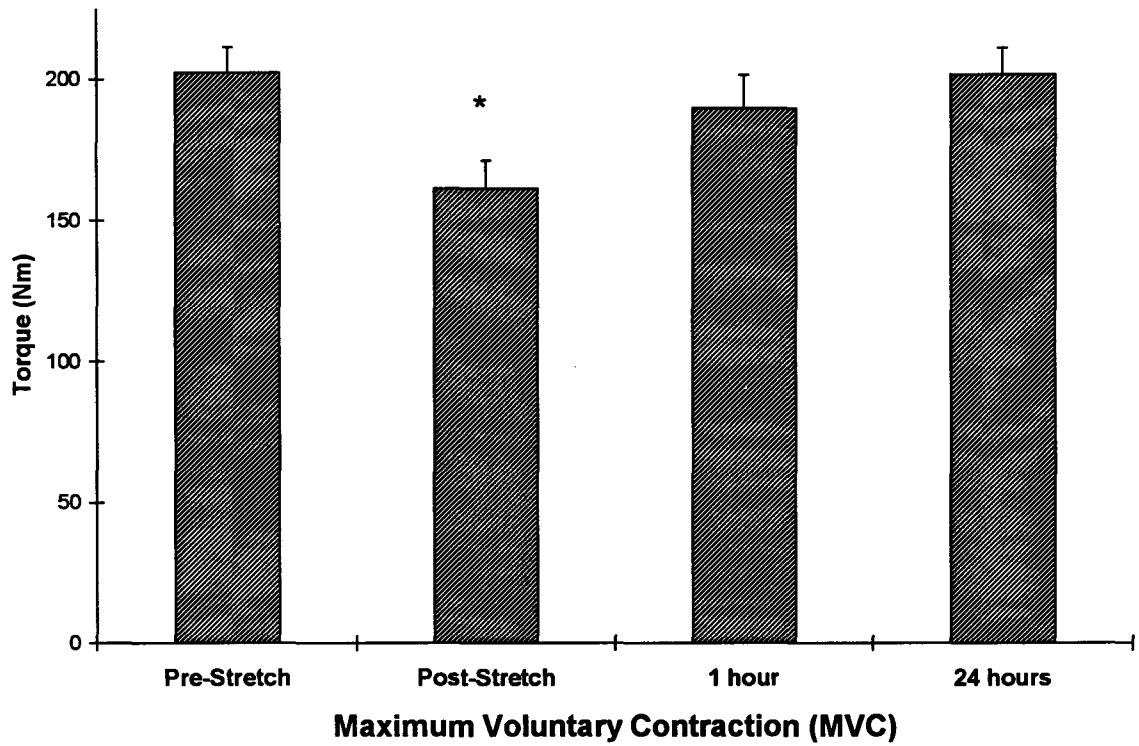


Figure 1. Maximum Voluntary Contraction Pre and Post 30 min of PSmax in human plantarflexors. * indicates significant decrease from Pre-stretch at $P < 0.005$)

DISCUSSION

The results of this study imply that PSmax can affect non-contractile element components of the muscle/tendon unit to elicit some symptoms of DOMS following PSmax. Contractile deficits and DOMS indicators were present 24 h following PSmax as was observed previously in human subjects (Smith *et al.*, 1993). Although MVC force was significantly decreased after PSmax, DOMS indicators were not significantly altered. Damage to the tendon has previously been posed to account for the decrement in force following PS (Lieber *et al.*, 1991). Although damage to the tendon is possible, it is unlikely, however, that tendon damage significantly compromised contractile element performance as maximal force production of the plantarflexors was restored in 24 h. Previous reports of the time course for exercise-induced muscle damage (EIMD) indicate that significant contractile element damage depresses force-generating capacity for up to and greater than one week following the insult (Ebbeling and Clarkson, 1989). McCully and Faulkner (1986) observed a 25% force decrement three days after sham-operated stretch on rat extensor digitorum longus muscles and attributed the decrement to tendon trauma incurred during the mounting procedure. Previous reports of EIMD have not partitioned what amount of force decrement following EIMD may be attributable to connective tissue disruption. Connective tissue disruption is thought to contribute to symptoms of pain with EIMD, i.e. DOMS (Jones *et al.*, 1987; Smith, 1991; Stauber *et al.*, 1990).

Depression of maximal force generating capacity of the plantarflexors directly following PSmax was associated with a significant increase in interpolated twitch torque (ITT), an indication of decreased central activation (Belanger and McComas, 1981). Using the formula of Duchateau. (1995), ITT indicated that most of the MVC force decrement was due to reduced muscle force-generating capacity and some to decreased motor unit activation (MUA). By one hour, a muscular cause can only account for approximately 1.3% of the decrement in MVC. The Golgi tendon reflex may contribute to force loss following stretching as posed by Kokkonen and Nelson (1996), although other factors may contribute to reduced muscle force generating capacity directly following PSmax.

Jones *et al.* (1989) found that passive lengthening of the elbow flexors had no effect on force generating capacity or perceptions of pain. We did not use the elbow flexors because we believed that the elbow joint structure limits full muscle elongation resulting from stretch relaxation. Using an intense stretch of the ankle plantarflexors, we observed mild perceptions of DOMS. One hour following PSmax, passive stiffness was also significantly elevated over POST and ROM was reduced despite a greater ankle dorsiflexion angle induced by overelongation of the muscle/tendon unit with PSmax. By 24 h, passive stiffness was still elevated, swelling in the muscle was marginal but apparent, and some subjects exhibited elevated CK. Those who experienced muscle pain, felt pain mainly at the distal and proximal myotendinous junctions. Myotendinous junctions are the sites most susceptible to strain injury (Tidball and Chan, 1989). Disruption at the

myotendinous junction is definitely plausible in this maximal stretch protocol; however, it is interesting to note that the symptoms of DOMS occur at a time point when MVC is at 100% of pre-stretch values. This fact is inconsistent with the postulation by Lieber *et al.* (1991) that damage to the myotendinous junction or breakage of myosin cross-bridges explained the force production loss observed by them after PS_{max} . However, the observation is consistent with the explanation that DOMS follows a different time course with different mechanisms than indicators of muscle damage (Newham, 1988; Stauber *et al.*, 1990). A poor correlation also exists between the time courses for decreases in force generation and for increases in the sensation of DOMS (Ebbeling and Clarkson, 1989), perhaps because not myofiber injury but connective tissue disruption mediates pain (Stauber *et al.*, 1990).

It is again interesting to note that McCully and Faulkner (1986) reported a significant decrease in tetanic force (P_o) at three days after a treatment of slow lengthening (ECC) contractions with little change in the histological appearance of the fibers, where otherwise a high correlation of -0.70 ($P < 0.001$) was observed between histological appearance of injury and the decrease in P_o . The authors proposed that other factors involved in lengthening contractions than peak force may contribute to both the decrease in force and injury. This observation may be related to some passive stretch conditions not exhibiting fiber damage at the subscopic level while still suffering some type of perturbation at the ultrastructural (sarcomere) level which might promote elevated muscle protein synthesis. The perturbation to stimulate muscle protein synthesis may be in the

form of activating a ground substance or secondary messenger cascade, possibly using Ca^{2+} as a mediator (Armstrong, 1990; Byrd, 1991) or prostoglandins (McComas, 1994).

Armstrong *et al.* (1993) observed a 61% decrease in twitch force after a 2 h stretch of rat soleus. Armstrong and colleagues tested the mounted muscles at the same absolute muscle length and explained their results with a damage mechanism; to quote "Static stretch causes elevation in muscle $[\text{Ca}^{2+}]$ via influx from extracellular space.....Whether the attenuation in force was caused by the elevated $[\text{Ca}^{2+}]$ is not known, but it is a reasonable hypothesis that increased $[\text{Ca}^{2+}]$ stimulated degradative pathways that caused proteolysis of myofibrillar proteins (Jackson *et al.*, 1984 - his reference) and, hence, loss of force-producing capability..... in the present study, we have no direct evidence that the elevations in $[\text{Ca}^{2+}]$ activated any of these degradative pathways, although marked reductions in the ability of the muscles to produce force may have resulted from disrupted myofibrillar structure in the affected fibers." Duncan and Jackson (1987) found that $[\text{Ca}^{2+}]$ caused rapid dissolution of myofibrillar proteins, although Armstrong *et al.* (1993) did not observe any significant elevations in CK loss from the isolated stretch muscles.

It is possible that contractile element damage may lower PTT (as postulated above) with PS_{max} , but a more likely cause to reduce twitch size is a greater series-elastic-component (SEC) slack after PS_{max} . Testing at the same absolute JA of 10°D showed a slight decrease in PTT; however, testing angle relative to the increase in JA and passive tension in this experiment showed PTT unchanged or slightly 'potentiated'.

Stretch-induced potentiation was not to the extent resulting from post-activation potentiation (PAP) following an MVC. The slight 'potentiation' observed here at the relative JA may be due to a shift in the length-tension relation to a new JA. Armstrong and colleagues' results probably were affected by the SEC compliance change and shift in the length-tension relation, thereby making their postulation that "stretch induced Ca^{2+} -activated degradation of myofibrillar proteins maybe being mechanistically involved in loss of the ability to produce force", unlikely.

Newham *et al.* (1987) claimed that subjects can maximally drive their muscles while suffering from DOMS. Pain and damage does not decrease central drive at times post eccentric damaging exercise when suffering from the symptoms of DOMS (i.e. at 24 h or 48 h), as indicated by the same interpolated twitch technique (Gibala *et al.*, 1995; Newham *et al.*, 1987). Gibala *et al.* (1995) did observe that motor unit activation (MUA; i.e., central drive) was significantly depressed immediately following a weight training bout. This is what may be expected, that high force eccentric exercise may activate the same neural response that passive lengthening does in inhibiting force after stretch. A recent report has also indicated that peripheral fatigue can lower central drive directly following exercise, possibly by reduced motivation or by some peripheral feedback mechanism (Simpson *et al.*, 1996). Lieber *et al.* (1991) postulated that the force decrement following ECC has components of damage, fatigue and some mechanism involved with passive stretch. Further investigation to evaluate the influence of neural

inhibition following eccentric exercise induced muscle damage and passive stretch is recommended as it may have implications for strength performance in sport.

REFERENCES

1. Armstrong, R. B. Initial events in exercise-induced muscular injury. *Med. Sci. Sports Exerc.* 22: 429-435, 1990.
2. Armstrong, R. B., C. Duan, M. D. Delp, D. E. Hayes, G. M. Glenn, and G. D. Allen. Elevations in rat soleus muscle $[Ca^{2+}]$ with passive stretch. *J. Appl. Physiology* 74: 2990-2997, 1993.
3. Armstrong, R. B., G. L. Warren, and J. A. Warren. Mechanisms of exercise-induced muscle fiber injury. *Sports Medicine* 12: 184-207, 1991.
4. Ashmore, R. C., L. Hitchcock, and Y. B. Lee. Passive stretch of adult chicken muscle produces myopathy remarkably similar to hereditary muscular dystrophy. *Experimental Neurology* 100: 341-353, 1988.
5. Belanger, A. Y. and A. J. McComas. Extent of motor unit activation during effort. *J. Appl. Physiology* 51: 1131-1135, 1981.
6. Byrd, S. K. Alterations in the sarcoplasmic reticulum: a possible link to exercise-induced muscle damage. *Med. Sci. Sports Exerc.* 24: 531-536, 1992.
7. Dowling, J. J., E. Konert, P. Ljucovic, and D. M. Andrews. Are humans able to voluntarily elicit maximum muscle force? *Neuroscience Letters* 179: 25-28, 1994.
8. Duchateau, J. Bed rest induces neural and contractile adaptations in triceps surae: a clinically relevant case study. *Med. Sci. Sports Exerc.* 27: 1581-1589, 1995.
9. Duncan, C. J. and M. J. Jackson. Different mechanisms mediate structural changes and intracellular enzyme efflux following damage to skeletal muscle. *J. Cell Sci.* 87: 183-188, 1987.
10. Ebbeling, C. B. and P. M. Clarkson. Exercise-induced muscle damage and adaptation. *Sports Medicine* 7: 207-234, 1989.
11. Friden, J., U. Kjorell, and L.-E. Thornell. Delayed muscle soreness and cytoskeletal alterations: an immunocytological study in man. *Int. J. Sports Med.* 5: 15-18, 1984.
12. Friden, J. and R. L. Lieber. Structural and mechanical basis for exercise-induced muscle injury. *Med. Sci. Sports Exerc.* 24: 521-530, 1992.

13. Gibala, M. J., J. D. MacDougall, M. A. Tarnopolsky, W. T. Stauber, and A. Elorriaga. Changes in human skeletal muscle ultrastructure and force production after acute resistance exercise. *J. Appl. Physiology* 78: 702-708, 1995.
14. Jackson, M. J., D. A. Jones, and R. H. T. Edwards. Experimental skeletal muscle damage: the nature of the calcium-activated degenerative process. *Eur. J. Clin. Invest.* 14: 369-374, 1984.
15. Jones, D. A., D. J. Newham, and P. M. Clarkson. Skeletal muscle stiffness and pain following eccentric exercise of the elbow flexors. *Pain* 30: 233-242, 1987.
16. Jones, D. A., D. J. Newham, and C. Torgan. Mechanical influences on long-lasting human muscle fatigue and delayed-onset pain. *J. Physiol.* 412: 415-427, 1989.
17. Kokkonen, J. and A. G. Nelson. Acute stretching exercises inhibit maximal strength performance. *Med. Sci. Sports Exerc.* 28: S1901996.(Abstract)
18. Lieber, R. L., T. M. Woodburn, and J. Friden. Muscle damage induced by eccentric contractions of 25% strain. *J. Appl. Physiology* 70: 2498-2507, 1991.
19. McComas, A. J. Human neuromuscular adaptations that accompany changes in activity. *Med. Sci. Sports Exerc.* 26: 1498-1509, 1994.
20. McCully, K. K. and J. A. Faulkner. Characteristics of lengthening contractions associated with injury to skeletal muscle fibers. *J. Appl. Physiology* 61: 293-299, 1986.
21. Nelson, A. G., A. Cornwell, and G. D. Heise. Acute stretching exercises and vertical jump stored elastic energy. *Med. Sci. Sports Exerc.* 28: S1561996.(Abstract)
22. Newham, D. J. The consequences of eccentric contractions and their relationship to delayed onset muscle pain. *European Journal of Applied Physiology* 57: 353-359, 1988.
23. Newham, D. J., D. A. Jones, and P. M. Clarkson. Repeated high force eccentric exercise: Effects on muscle pain and damage. *J. Appl. Physiology* 63: 1381-1386, 1987.
24. Sale, D., J. Quinlan, E. Marsh, A. J. McComas, and A. Y. Belanger. Influence of joint position on ankle plantarflexion in humans. *J. Appl. Physiology* 52: 1636-1642, 1982.

25. Simpson, M. R., J. R. Burke, J. M. Davis, and D. A. Jackson. Relationship between peripheral fatigue and the inhibition of neural activation. *Med. Sci. Sports Exerc.* 28: S141-1996.(Abstract)
26. Smith, L. L. Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Med. Sci. Sports Exerc.* 23: 542-551, 1991.
27. Smith, L. L., M. H. Brunetz, T. C. Chenier, M. R. McCammon, J. A. Houmard, M. E. Franklin, and R. G. Israel. The effects of static and ballistic stretching on delayed onset muscle soreness and creatine kinase. *Research Quarterly for Exercise and Sport* 64: 103-107, 1993.
28. Stauber, W. T., P. M. Clarkson, V. K. Fritz, and W. J. Evans. Extracellular matrix disruption and pain after eccentric muscle action. *J. Appl. Physiology* 69: 868-874, 1990.
29. Tidball, J. G. and M. Chan. Adhesive strength of single muscle cells to basement membrane at myotendinous junctions. *J. Appl. Physiology* 67: 1063-1069, 1989.
30. Waterman-Storer, C. M. The cytoskeleton of skeletal muscle: is it affected by exercise? A brief review. *Med. Sci. Sports Exerc.* 23: 1240-1249, 1991.

APPENDIX 2 - Raw Data Experiment 1

**Raw Data Experiment 1: PSmax Parameters;
Joint Angle (deg Dorsiflexion)**

Subject	PSmax Stretch Interval (min)												
	0.0	2.5	5.0	7.5	10.0	12.5	15.0	17.5	20.0	22.5	25.0	27.5	30.0
CB	26.0	26.5	27.5	26.5	26.5	28.0	27.5	28.5	29.0	30.0	30.0	30.0	30.5
IE	24.5	26.5	28.0	28.0	28.0	29.0	30.0	30.0	30.0	31.0	31.0	32.0	32.0
JA	32.5	35.0	35.0	36.0	36.5	37.0	38.0	37.5	37.5	37.5	37.5	38.0	38.5
TC	32.0	36.0	37.5	37.0	39.0	39.0	39.0	40.0	39.0	39.0	40.5	41.0	40.5
TH	31.0	32.5	33.0	34.0	34.5	34.0	34.5	35.0	35.5	36.0	36.0	36.0	36.5
TS	27.5	29.0	30.5	31.0	31.0	31.0	31.5	31.5	31.5	32.5	32.5	34.0	34.0
CZ	33.0	35.0	36.0	37.5	38.0	38.5	39.0	39.0	40.0	39.0	40.0	40.5	41.0
JM	30.0	33.0	34.5	34.5	34.0	35.0	35.0	34.5	35.0	36.0	33.0	35.0	35.0
SKA	41.0	42.5	45.5	46.5	47.5	47.0	48.0	48.0	48.0	48.0	48.0	48.0	48.0
TB	35.0	37.0	37.5	39.0	39.5	40.0	40.0	41.0	41.5	41.0	41.5	42.0	42.0
Mean	31.3	33.3	34.5	35.0	35.5	35.9	36.3	36.5	36.7	37.0	37.0	37.7	37.8
SD	4.8	5.0	5.3	5.8	6.1	5.7	5.9	5.9	5.8	5.3	5.6	5.4	5.3
SE	1.5	1.6	1.7	1.8	1.9	1.8	1.9	1.9	1.8	1.7	1.8	1.7	1.7

**Raw Data Experiment 1: PSmax Parameters;
Passive Torque at initiation and end of a stretch interval (Nm)**

Sub	0.00	2.25	2.50	4.75	5.00	7.25	7.50	9.75	10.00	12.25	12.50	14.75	15.00
CB	54.64	38.75	44.98	38.22	52.65	39.99	44.44	36.60	43.42	32.90	46.75	37.97	43.53
IE	31.40	22.92	38.16	30.38	38.70	33.28	36.18	29.52	32.63	28.98	33.12	28.61	37.89
JA	44.44	33.49	46.59	37.46	44.44	36.17	41.44	35.53	41.22	34.99	41.76	36.28	47.23
TC	40.47	31.88	47.25	37.14	48.20	40.90	45.51	39.93	51.74	43.48	47.77	39.97	46.59
TH	32.96	25.71	35.26	31.34	37.14	31.94	35.64	32.63	36.82	33.06	34.35	32.04	35.10
TS	37.76	27.00	40.58	34.67	42.78	37.03	41.38	36.66	39.02	35.75	39.99	34.99	41.70
CZ	38.22	27.16	36.71	30.81	40.04	33.17	42.83	35.53	44.23	38.11	45.30	39.07	42.94
JM	34.46	27.05	37.57	31.24	37.03	32.42	34.24	30.06	33.17	29.31	36.28	31.77	34.89
SKA	34.89	25.23	36.39	31.56	40.25	33.49	42.08	35.64	41.11	34.78	41.11	36.39	38.11
TB	33.17	24.58	31.94	26.19	31.13	26.09	37.25	31.08	35.26	29.98	35.53	29.62	35.96
MEAN	38.24	28.38	39.54	32.90	41.24	34.45	40.10	34.32	39.85	34.13	40.20	34.67	40.39
SD	6.98	4.86	5.16	3.84	6.12	4.30	3.95	3.36	5.82	4.43	5.28	3.97	4.63
SE	2.33	1.62	1.72	1.28	2.04	1.43	1.32	1.12	1.94	1.48	1.76	1.32	1.54

	17.25	17.50	19.75	20.00	22.25	22.50	24.75	25.00	27.25	27.50	29.75	30.00
	35.10	44.33	37.03	48.09	38.22	49.81	40.42	46.37	40.20	48.52	39.92	50.50
	33.20	35.80	32.26	38.48	34.30	35.69	30.75	41.81	35.69	38.27	32.96	41.54
	38.54	41.65	38.00	40.04	37.37	41.33	37.47	39.61	36.39	40.25	35.83	41.76
	39.93	48.41	42.51	44.23	40.47	43.37	36.60	47.02	40.58	46.70	39.61	44.55
	30.97	34.89	31.45	40.79	34.51	40.85	34.61	35.80	30.38	41.09	35.60	41.44
	38.75	37.20	33.97	38.64	36.12	42.29	38.86	35.64	33.55	43.69	39.00	41.81
	36.50	40.68	35.32	40.47	34.25	39.61	33.48	42.19	35.64	42.19	36.80	45.30
	30.92	33.28	29.20	36.07	29.84	36.07	30.70	27.91	24.58	33.29	27.05	32.74
	33.60	39.83	34.99	38.97	33.81	39.29	34.78	40.47	34.14	37.03		
	31.13	38.75	31.83	39.50	34.46	36.23	31.13	35.10	34.45	39.66	33.50	34.89
	34.86	39.48	34.66	40.53	35.33	40.45	34.88	39.19	34.56	41.07	35.59	41.61
	3.43	4.57	3.85	3.37	2.90	4.24	3.45	5.76	4.62	4.51	4.07	5.31
	1.14	1.52	1.28	1.12	0.97	1.41	1.15	1.92	1.54	1.50	1.44	1.88

Raw Data Experiment 1; Isometric MVC
Peak MVC (MVC) (Nm)

Subject	Control MVC							PSmax MVC						
	PRE	POST	5 min	15 min	30 min	45 min	60 min	PRE	POST	5 min	15 min	30 min	45 min	60 min
CB	110.54	98.35	98.55	100.00	96.24	102.33	102.79	109.00	62.39	57.58	98.58	102.43	112.03	112.88
IE	178.88	154.24	164.43	162.05	184.63	169.95	176.88	185.87	150.22	155.22	168.00	157.94	169.46	176.30
JA	188.81	196.96	193.33	207.19	199.40	194.32	195.31	194.33	153.64	158.26	160.55	168.40	165.51	169.49
TC	155.95	146.40	147.12	145.57	147.14	156.36	159.25	150.00	107.91	107.00	128.14	128.07	132.93	136.22
TH	156.31	153.47	152.48	153.30	143.00	147.58	149.90	173.41	112.91	137.94	158.96	162.07	164.40	163.62
TS	204.68	207.80	210.02	183.80	201.72	188.81	191.90	206.78	156.84	174.95	173.38	173.38	179.32	180.89
CZ	144.35	141.00	141.29	133.59	130.29	125.12	135.24	142.60	112.00	122.60	123.91	127.87	139.97	131.39
JM	108.03	105.10	106.97	106.53	96.08	103.01	102.20	106.46	68.84	82.32	93.81	96.30	93.84	93.60
SKA	133.92	126.11	131.17	128.75	121.68	130.95	131.17	126.66	81.32	112.32	116.87	114.89	114.45	120.17
TB	164.49	139.06	158.67	155.51	160.92	162.70	159.99	171.81	124.00	145.87	152.21	148.49	151.13	148.23
MEAN	154.60	146.85	150.40	147.63	148.11	148.11	150.46	156.69	113.01	125.41	137.44	137.98	142.30	143.28
SD	31.65	34.87	34.49	32.78	38.65	32.49	33.00	35.32	34.35	36.47	28.96	27.89	28.57	29.34
SE	10.01	11.03	10.91	10.37	12.22	10.28	10.44	11.17	10.86	11.53	9.16	8.82	9.03	9.28
% diff from PRE		-5.01%	-2.71%	-4.51%	-4.20%	-4.19%	-2.67%		-27.88%	-19.97%	-12.29%	-11.94%	-9.18%	-8.56%
% diff PSmax to Con									-22.87%	-17.25%	-7.78%	-7.74%	-4.99%	-5.89%

Distribution of Force Decrement between muscle & MUA								
CONTROL	FULL	PRE	POST	5 min	15 min	30 min	45 min	60 min
actual	act	154.60	146.85	150.40	147.63	148.11	148.11	150.46
MUA%		100.00	0.97	0.96	0.96	0.96	0.97	0.98
ext MVC		159.75	154.60	153.78	152.83	153.17	155.51	156.77
dec from ext		5.16	12.91	9.35	12.13	11.64	11.64	9.29
muscle dec		0.00	6.93	2.42	5.55	7.40	7.37	6.31
act dec		5.16	5.98	6.93	6.58	4.25	4.27	2.99
dec from pre			7.75	4.19	6.97	6.49	6.48	4.13
muscle dec			6.93	2.42	5.55	7.40	7.37	6.31
act dec			0.82	1.77	1.42	-0.91	-0.89	-2.17
Mus dec %			89%	58%	80%	114%	114%	153%
Act dec %			11%	42%	20%	-14%	-14%	-53%
force dec %			-5.01%	-2.71%	-4.51%	-4.20%	-4.19%	-2.67%

Distribution of Force Decrement between muscle & MUA								
STRETCH	FULL	PRE	POST	5 min	15 min	30 min	45 min	60 min
actual	act	156.69	113.01	125.41	137.44	137.98	142.30	143.28
MUA%		100.00	0.96	0.81	0.84	0.93	0.96	0.95
ext MVC		162.39	156.69	131.91	136.40	151.35	156.47	154.78
dec from ext		5.70	49.38	36.98	24.95	24.41	20.09	19.11
muscle dec		0.00	18.91	10.99	13.91	18.49	12.47	11.50
act dec		5.70	30.48	25.99	11.04	5.91	7.61	7.61
dec from pre		0.00	43.69	31.29	19.25	18.71	14.39	13.41
muscle dec		0.00	18.91	10.99	13.91	18.49	12.47	11.50
act dec		5.70	24.78	20.29	5.34	0.22	1.92	1.91
musc dec %			0%	43%	35%	72%	99%	87%
act dec %			0%	57%	65%	28%	1%	13%
force dec %			-27.88%	-19.97%	-12.29%	-11.94%	-9.18%	-8.56%

**Raw Data Experiment 2 : Isometric MVC;
Interpolated Twitch Torque (Nm)**

Subject	Control MVC							PSmax MVC						
	PRE	POST	5 min	15 min	30 min	45 min	60 min	PRE	POST	5 min	15 min	30 min	45 min	60 min
CB	1.46	2.88	3.80	1.80	1.61	1.25	1.23	3.01	4.99	8.07	4.67	0.82	2.99	3.68
IE	0.79	1.20	1.64	1.84	0.00	1.61	0.52	0.43	0.34	1.05	1.25	0.75	0.45	0.22
JA	0.00	0.00	0.00	0.45	0.32	0.00	0.00	0.27	0.42	0.71	0.50	0.15	0.10	0.29
TC	0.45	0.19	0.00	1.20	0.77	0.00	0.43	0.00	2.04	2.05	0.62	1.36	0.00	0.58
TH	1.57	0.00	0.50	0.12	0.51	0.33	0.45	0.00	3.98	2.82	0.17	0.58	0.45	0.00
TS	0.20	0.39	0.13	0.56	0.15	0.80	0.13	0.54	2.84	0.47	0.89	0.60	1.38	0.00
CZ	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.30	0.00	0.00	0.00	0.00	0.00
JM	0.15	0.00	0.00	0.00	0.00	0.00	0.00	0.19	2.17	2.67	0.19	0.13	0.49	0.41
SKA	0.00	0.00	0.00	0.00	0.25	0.00	0.00	0.24	3.23	0.39	0.34	0.34	0.00	0.00
TB	0.49	0.82	0.24	0.43	0.19	0.00	0.00	0.19	2.45	0.39	0.54	0.19	0.30	0.67
MEAN	0.51	0.55	0.63	0.64	0.38	0.40	0.28	0.49	2.28	1.86	0.92	0.49	0.62	0.59
SD	0.59	0.92	1.22	0.72	0.50	0.61	0.40	0.91	1.59	2.40	1.37	0.42	0.93	1.12
SE	0.19	0.29	0.39	0.23	0.16	0.19	0.13	0.29	0.50	0.76	0.43	0.13	0.29	0.35
% diff from PRE		7.24%	23.48%	25.21%	-25.64%	-21.84%	-45.95%		367.16%	282.18%	88.22%	0.99%	26.44%	20.07%
% diff PSmax to Con									359.92%	258.70%	63.01%	26.62%	48.28%	66.02%

**Raw Data Experiment 1 : Isometric MVC;
Motor Unit Activation (MUA) (%)**

Subject	Control MVC							PSmax MVC						
	PRE	POST	5 min	15 min	30 min	45 min	60 min	PRE	POST	5 min	15 min	30 min	45 min	60 min
CB	89.73%	78.89%	72.14%	87.08%	88.27%	90.26%	90.62%	76.56%	55.37%	27.82%	62.70%	93.34%	75.41%	70.08%
IE	95.68%	92.76%	90.11%	89.41%	100.00%	90.39%	96.90%	97.37%	97.57%	92.51%	91.73%	94.82%	96.81%	98.48%
JA	100.00%	100.00%	100.00%	97.09%	97.87%	100.00%	100.00%	98.66%	97.49%	95.75%	96.92%	99.04%	99.34%	98.07%
TC	97.33%	98.67%	100.00%	92.18%	94.44%	100.00%	97.34%	100.00%	81.49%	81.40%	95.76%	89.73%	100.00%	95.28%
TH	90.14%	100.00%	96.74%	99.32%	97.08%	97.96%	97.30%	100.00%	70.69%	79.23%	99.00%	96.27%	97.16%	100.00%
TS	98.84%	97.60%	99.20%	96.35%	99.03%	94.68%	99.16%	96.85%	82.95%	97.18%	94.61%	96.21%	91.10%	100.00%
CZ	100.00%	100.00%	100.00%	100.00%	100.00%	100.00%	100.00%	100.00%	97.61%	100.00%	100.00%	100.00%	100.00%	100.00%
JM	98.80%	100.00%	100.00%	100.00%	100.00%	100.00%	100.00%	98.45%	77.74%	72.62%	98.29%	98.79%	95.41%	95.88%
SKA	100.00%	100.00%	100.00%	100.00%	97.91%	100.00%	100.00%	98.10%	68.82%	96.24%	96.71%	96.74%	100.00%	100.00%
TB	97.19%	94.66%	98.44%	97.38%	98.81%	100.00%	100.00%	98.92%	82.59%	97.23%	96.33%	98.63%	97.90%	95.33%
MEAN	96.77%	96.26%	95.66%	95.88%	97.34%	97.33%	98.13%	96.49%	81.23%	84.00%	93.20%	96.36%	95.31%	95.31%
SD	0.04	0.07	0.09	0.05	0.04	0.04	0.03	0.07	0.14	0.22	0.11	0.03	0.08	0.09
SE	0.01	0.02	0.03	0.01	0.01	0.01	0.01	0.02	0.04	0.07	0.03	0.01	0.02	0.03
% diff from PRE		-0.53%	-1.15%	-0.92%	0.59%	0.58%	1.41%		-15.81%	-12.95%	-3.41%	-0.14%	-1.22%	-1.22%
% diff PSmax to Con									-15.28%	-11.80%	-2.49%	-0.73%	-1.80%	-2.63%

Raw Data Experiment 1; Twitch Contractile Properties
Peak Twitch Torque (PTT) (Nm)

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	14.21	13.64	13.93	13.73	12.83	13.11	12.85	11.18	12.52	12.32	12.16	12.30
IE	18.29	16.58	17.37	17.17	16.76	16.76	16.37	14.01	15.11	14.48	14.09	14.46
JA	15.87	14.68	15.48	15.01	14.24	13.83	20.19	16.71	16.23	15.63	15.14	15.02
TC	16.88	14.30	15.34	13.85	14.56	16.15	15.80	11.02	14.62	13.24	12.79	12.30
TH	15.93	15.35	17.38	17.44	16.34	16.72	16.58	13.58	17.01	15.56	15.84	16.88
TS	17.20	16.23	15.36	15.54	15.03	15.52	17.17	16.66	16.50	15.82	15.50	15.38
CZ	16.15	14.89	16.99	14.68	14.40	13.66	16.35	12.54	14.76	14.50	14.62	14.62
JM	12.48	10.04	10.55	10.24	9.79	10.26	12.22	9.75	11.10	10.71	10.67	9.96
SKA	12.89	12.32	12.05	11.97	12.44	11.44	12.63	10.36	10.32	10.44	11.65	10.67
TB	17.43	15.36	16.42	15.93	15.46	15.50	17.60	14.07	14.70	13.91	14.26	14.36
MEAN	15.73	14.34	15.09	14.56	14.19	14.30	15.78	12.99	14.29	13.66	13.67	13.60
SD	1.95	1.95	2.29	2.23	2.05	2.23	2.52	2.46	2.27	1.96	1.76	2.20
SE	0.62	0.62	0.73	0.70	0.65	0.71	0.80	0.78	0.72	0.62	0.56	0.70
% diff PRE		-8.86%	-4.11%	-7.48%	-9.84%	-9.14%		-17.67%	-9.44%	-13.41%	-13.34%	-13.82%
% diff PSmax to Con								-8.81%	-5.33%	-5.93%	-3.50%	-4.68%

Raw Data Experiment 1; Twitch Contractile Properties
Time to Peak Torque (TPT) (msec)

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	102.35	111.96	112.29	119.24	120.57	117.59	111.96	101.36	109.64	112.29	110.96	115.27
IE	110.30	112.62	116.93	113.95	116.93	119.91	119.91	111.63	120.57	119.91	122.89	128.52
JA	124.21	134.81	139.12	140.44	138.46	140.44	121.17	112.85	115.18	115.18	120.84	124.17
TC	102.68	104.01	108.65	104.01	106.33	109.97	99.73	91.75	100.36	103.01	100.70	95.73
TH	105.66	126.81	121.32	119.82	126.81	119.82	123.88	124.88	128.52	136.47	127.86	132.82
TS	95.73	96.39	99.37	96.72	98.05	100.36	95.73	94.73	98.71	105.66	103.01	103.68
CZ	137.79	164.96	166.61	169.59	167.27	170.92	138.13	135.14	151.71	158.00	156.34	169.59
JM	116.93	118.91	121.56	111.96	121.89	120.24	111.63	108.65	107.65	114.28	112.62	105.66
SKA	110.30	116.93	109.64	113.95	121.89	122.56	114.94	112.29	121.89	119.91	121.56	124.54
TB	124.21	118.91	110.63	116.26	117.59	114.94	114.94	105.00	100.70	109.31	98.71	111.96
MEAN	113.02	120.63	120.61	120.59	123.58	123.68	115.20	109.83	115.49	119.40	117.55	121.19
SD	12.77	18.94	19.31	20.61	18.81	19.44	12.02	13.07	16.24	16.45	16.95	20.73
SE	4.04	5.99	6.11	6.52	5.95	6.15	3.80	4.13	5.14	5.20	5.36	6.56
% diff PRE		6.74%	6.72%	6.71%	9.35%	9.43%		-4.66%	0.25%	3.65%	2.04%	5.20%
% diff PSmax to Con								-11.40%	-6.47%	-3.06%	-7.31%	-4.23%

Raw Data Experiment 1; Twitch Contractile Properties
Rise Time (RT) (msec)

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	66.58	71.88	72.21	74.86	76.85	74.53	65.58	66.58	68.57	70.88	70.55	71.88
IE	68.90	65.92	70.55	113.95	73.53	73.87	73.20	70.22	72.21	73.20	74.53	75.52
JA	78.17	80.49	83.47	85.79	83.80	86.45	75.57	75.90	79.56	78.89	77.56	77.56
TC	64.92	61.61	66.91	61.28	65.25	68.27	60.62	58.30	60.62	61.84	60.28	58.96
TH	61.61	80.38	75.39	74.89	75.89	74.39	68.23	71.88	74.20	82.15	75.52	77.84
TS	57.63	59.29	60.28	60.62	61.94	61.94	58.30	60.62	61.28	61.94	62.60	63.27
CZ	84.80	102.68	102.02	104.34	105.66	110.30	88.44	88.11	97.71	101.03	100.73	106.33
JM	69.89	68.90	69.23	67.57	74.20	74.53	70.22	65.92	67.24	66.91	69.56	67.24
SKA	67.24	71.88	67.57	68.90	74.20	74.53	70.55	71.22	74.86	75.52	78.50	75.85
TB	72.54	72.21	69.56	69.23	68.23	67.90	67.57	63.60	60.95	65.25	60.62	64.59
MEAN	69.23	73.52	73.72	78.14	75.96	76.67	69.83	69.24	71.72	73.76	73.05	73.90
SD	7.86	12.37	11.61	18.00	12.14	13.41	8.40	8.54	11.21	11.80	11.88	13.16
SE	2.49	3.91	3.67	5.69	3.84	4.24	2.66	2.70	3.55	3.73	3.76	4.16
% diff from PRE		6.21%	6.49%	12.88%	9.72%	10.75%		-0.85%	2.71%	5.63%	4.61%	5.84%
% diff PSmax to Con								-7.05%	-3.78%	-7.25%	-5.11%	-4.91%

**Raw Data Experiment 1; Twitch Contractile Properties
Maximum Rate of Torque Development (MRTD) (Nm/s)**

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	226.43	197.98	192.05	193.24	182.57	189.09	216.36	173.09	183.76	180.79	188.50	174.27
IE	301.71	272.08	266.74	266.15	248.37	250.14	269.11	214.58	238.29	231.77	226.43	225.84
JA	228.81	208.65	200.95	196.20	190.87	173.09	368.28	324.95	270.89		259.96	249.13
TC	294.01	243.62	258.44	241.85	277.41	260.81	293.42	209.84	269.71	245.40	238.29	234.73
TH	286.90	224.76	260.02	277.67	251.20	264.42	275.63	222.28	262.00	224.06	247.77	245.40
TS	303.49	282.15	273.26	251.33	252.52	266.15	308.23	277.41	272.08	275.04	261.41	257.26
CZ	216.36	179.01	190.28	161.23	162.42	147.00	231.77	172.49	184.94	168.34	173.09	162.42
JM	215.76	184.94	183.86	173.68	179.61	169.53	227.62	171.31	190.87	203.32	178.42	167.16
SKA	218.73	190.28	193.24	190.28	184.35	163.60	195.61	151.75	176.64	170.12	180.79	155.90
TB	293.42	250.74	269.71	251.33	252.52	249.55	299.34	238.88	267.33	231.18	266.15	239.47
MEAN	258.56	223.42	228.86	220.30	218.18	213.34	268.54	215.66	231.65	214.45	222.08	211.16
SD	39.82	37.10	39.21	41.71	41.66	48.67	51.86	53.85	42.20	36.49	38.01	40.88
SE	12.59	11.73	12.40	13.19	13.18	15.39	16.40	17.03	13.34	12.16	12.02	12.93
% diff from PRE		-13.59%	-11.49%	-14.80%	-15.62%	-17.49%		-19.69%	-13.74%	-20.14%	-17.30%	-21.37%
% diff PSmax to Con								-6.10%	-2.25%	-5.34%	-1.68%	-3.88%

**Raw Data Experiment 1; Twitch Contractile Properties
Maximum Rate of Torque Relaxation (MRTR) (-Nm/s)**

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	329.57	340.98	597.06	535.26	514.37	415.08	63.87	163.60	341.58	280.52	530.96	518.81
IE	169.53	294.01	364.99	190.28	432.86	394.33	154.12	137.52	255.63	238.88	238.59	256.81
JA	126.85	82.39	76.47	227.47	71.28	172.05	180.53	167.89	194.07	61.38	297.87	128.18
TC	133.37	231.03	199.02	233.70	109.66	159.16	148.19	120.92	150.56	151.45	256.29	101.36
TH	140.48	141.03	130.01	134.41	121.19	123.40	138.71	115.00	136.33	118.55	117.96	125.07
TS	204.35	207.76	118.70	170.86	232.66	118.26	137.52	166.57	472.87	274.89	372.40	363.66
CZ	124.63	175.46	340.84	332.83	342.17	355.51	148.04	101.36	358.77	543.71	348.25	461.61
JM	93.06	135.89	164.64	196.65	87.28	152.49	96.62	84.17	185.24	177.09	340.10	251.03
SKA	67.24	74.09	66.39	65.80	66.98	65.80	79.43	71.72	68.17	63.43	70.54	65.80
TB	122.11	150.26	136.63	139.74	104.47	105.21	118.55	109.66	107.88	342.47	116.18	316.09
MEAN	151.12	183.29	219.48	222.70	208.29	206.13	126.56	123.84	227.11	225.24	268.91	258.84
SD	73.02	86.47	166.94	130.67	164.69	129.99	36.49	34.35	128.27	146.46	140.68	156.37
SE	23.09	27.34	52.79	41.32	52.08	41.11	11.54	10.86	40.56	46.31	44.49	49.45
% diff from PRE		21.29%	45.23%	47.37%	37.83%	36.40%		-2.15%	79.45%	77.97%	112.48%	104.52%
% diff PSmax to Con								-23.44%	34.22%	30.60%	74.65%	68.12%

**Raw Data Experiment 1; Twitch Contractile Properties
Torque-Time Integral (TTI) (Nms)**

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	3.72	4.00	4.53	4.48	4.43	4.19	3.07	2.58	3.52	3.32	3.96	4.04
IE	3.16	3.95	4.43	3.95	4.59	4.57	2.91	2.98	3.69	3.63	3.60	3.76
JA	3.12	3.53	3.74	4.01	3.51	3.75	4.09	3.93	4.10	3.98	3.43	3.97
TC	3.23	3.54	3.72	3.48	3.20	3.66	2.56	1.68	2.54	3.03	3.23	2.17
TH	3.03	3.40	3.89	3.87	3.74	3.99	3.46	2.78	3.69	3.51	3.63	3.85
TS	3.76	3.52	3.25	3.36	3.44	3.29	3.34	3.06	4.27	3.65	3.94	3.88
CZ	4.31	4.49	5.59	4.88	4.78	4.71	4.35	2.53	4.31	4.86	4.53	4.91
JM	2.77	2.57	2.82	2.82	2.99	2.73	2.73	2.04	2.76	2.73	3.13	2.85
SKA	2.63	2.59	2.65	2.67	2.84	2.60	2.65	1.97	2.10	2.11	2.59	2.30
TB	3.63	3.91	4.08	3.97	3.82	3.75	3.66	2.70	3.17	3.85	2.53	3.88
MEAN	3.34	3.55	3.87	3.75	3.73	3.72	3.28	2.63	3.42	3.47	3.46	3.56
SD	0.51	0.60	0.87	0.69	0.67	0.70	0.61	0.64	0.76	0.75	0.62	0.85
SE	0.16	0.19	0.27	0.22	0.21	0.22	0.19	0.20	0.24	0.24	0.20	0.27
% diff from PRE		6.41%	16.01%	12.38%	11.93%	11.63%		-20.02%	4.05%	5.64%	5.33%	8.50%
% diff PSmax to Con								-26.43%	-11.95%	-6.74%	-6.60%	-3.13%

**Raw Data Experiment 1; Twitch Contractile Properties
TTI to Half Relaxation Time (TTIHRT) (Nms)**

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	2.17	2.46	2.44	2.64	2.55	2.52	2.10	1.67	2.10	2.07	2.18	2.26
IE	2.60	2.53	2.80	2.73	2.79	2.84	2.44	2.04	2.36	2.31	2.31	2.41
JA	2.48	2.64	2.79	2.77	2.59	2.62	3.20	2.60	2.62	2.63	2.59	2.64
TC	2.40	2.26	2.48	2.15	2.30	2.47	2.10	1.40	1.95	1.98	1.98	1.76
TH	2.62	2.85	3.36	3.35	3.21	3.37	2.98	2.42	3.18	2.94	3.01	3.17
TS	2.33	2.13	2.10	2.16	2.12	2.20	2.17	2.12	2.31	2.23	2.26	2.23
CZ	3.18	3.34	4.03	3.43	3.36	3.26	3.23	2.09	2.81	2.99	3.03	3.21
JM	1.86	1.68	1.81	1.75	2.04	1.73	2.12	1.38	1.69	1.67	1.74	1.65
SKA	2.23	2.21	2.22	2.22	2.33	2.13	2.25	1.66	1.75	1.79	2.07	1.91
TB	2.97	2.84	2.94	2.83	2.75	2.72	2.92	2.12	2.31	2.36	1.96	2.41
MEAN	2.48	2.49	2.70	2.60	2.60	2.59	2.55	1.95	2.31	2.30	2.31	2.37
SD	0.39	0.46	0.65	0.54	0.44	0.50	0.48	0.41	0.47	0.45	0.44	0.53
SE	0.12	0.15	0.20	0.17	0.14	0.16	0.15	0.13	0.15	0.14	0.14	0.17
% diff from PRE		0.40%	8.57%	4.79%	4.83%	4.11%		-23.56%	-9.53%	-9.96%	-9.33%	-7.29%
% diff PSmax to Con								-23.96%	-18.10%	-14.75%	-14.16%	-11.40%

**Raw Data Experiment 1; Twitch Contractile Properties
Half Relaxation Time (HRT) (msec)**

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	110.30	132.83	129.84	142.10	149.06	143.76	110.63	102.02	117.26	115.93	132.16	134.81
IE	83.47	93.41	101.36	101.69	109.31	110.3	83.14	86.12	90.76	96.06	98.71	97.38
JA	90.76	108.98	106.99	112.62	109.97	117.92	93.54	99.53	105.19	112.52	110.19	112.18
TC	92.08	108.31	109.97	105.66	106.33	98.38	84.80	79.17	80.16	98.71	108.31	96.39
TH	108.65	116.33	128.31	128.31	127.81	139.79	109.97	108.31	116.26	113.95	121.89	116.26
TS	89.10	84.13	86.78	93.41	95.40	92.75	78.17	79.83	93.08	87.11	96.39	94.40
CZ	129.18	140.11	153.69	148.72	151.37	157.01	128.85	92.75	112.62	129.18	128.52	134.81
JM	94.73	108.31	110.63	119.58	143.76	120.53	108.65	84.13	99.04	98.71	110.63	120.57
SKA	120.57	124.21	134.81	133.16	128.85	127.53	123.22	105.33	108.31	112.95	120.24	117.26
TB	104.67	124.88	126.53	120.90	119.24	119.58	106.66	96.39	108.98	117.92	88.44	113.28
MEAN	102.35	114.15	118.89	120.62	124.11	122.76	102.76	93.36	103.17	108.30	111.55	113.73
SD	14.87	17.22	19.32	17.81	19.35	20.04	17.19	10.60	12.14	12.65	14.29	14.52
SE	4.70	5.45	6.11	5.63	6.12	6.34	5.44	3.35	3.84	4.00	4.52	4.59
% diff from PRE		11.53%	16.16%	17.84%	21.26%	19.94%		-9.15%	0.39%	5.39%	8.55%	10.68%
% diff PSmax to Con								-20.68%	-15.77%	-12.45%	-12.71%	-9.26%

**Raw Data Experiment 1; Twitch Contractile Properties
Twitch to MVC Ratio**

Subject	Control Twitch						PSmax Twitch					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	0.1286	0.1387	0.1393	0.1427	0.1254	0.1275	0.1179	0.1792	0.1270	0.1203	0.1085	0.1090
IE	0.1022	0.1075	0.1072	0.0930	0.0986	0.0948	0.0881	0.0933	0.0899	0.0917	0.0831	0.0820
JA	0.0841	0.0745	0.0747	0.0753	0.0733	0.0708	0.1039	0.1088	0.1011	0.0928	0.0915	0.0886
TC	0.1082	0.0977	0.1054	0.0941	0.0931	0.1014	0.1053	0.1021	0.1141	0.1034	0.0962	0.0903
TH	0.1019	0.1000	0.1134	0.1220	0.1107	0.1115	0.0956	0.1203	0.1070	0.0960	0.0964	0.1032
TS	0.0840	0.0781	0.0836	0.0770	0.0796	0.0809	0.0830	0.1062	0.0952	0.0912	0.0864	0.0850
CZ	0.1119	0.1056	0.1272	0.1127	0.1151	0.1010	0.1147	0.1120	0.1191	0.1134	0.1045	0.1113
JM	0.1155	0.0955	0.0990	0.1066	0.0950	0.1004	0.1148	0.1416	0.1183	0.1112	0.1137	0.1064
SKA	0.0963	0.0977	0.0936	0.0984	0.0950	0.0872	0.0997	0.1274	0.0883	0.0909	0.1018	0.0888
TB	0.1060	0.1105	0.1056	0.0990	0.0950	0.0969	0.1024	0.1135	0.0966	0.0937	0.0944	0.0969
MEAN	0.1039	0.1006	0.1049	0.1021	0.0981	0.0972	0.1025	0.1204	0.1057	0.1005	0.0976	0.0961
SD	0.0137	0.0178	0.0191	0.0202	0.0156	0.0158	0.0115	0.0247	0.0135	0.0109	0.0096	0.0106
SE	0.0043	0.0056	0.0060	0.0064	0.0049	0.0050	0.0036	0.0078	0.0043	0.0034	0.0030	0.0034
% diff from PRE		-3.16%	0.99%	-1.73%	-5.56%	-6.38%		17.44%	3.04%	-2.04%	-4.77%	-6.24%
% diff PSmax to Con								20.61%	2.05%	-0.30%	0.79%	0.14%

**Raw Data Experiment 1: EMG;
MVC AEMG (mvolts/s)**

Subject	Control MVC							PSmax MVC						
	PRE	POST	5 min	15 min	30 min	45 min	60 min	PRE	POST	5 min	15 min	30 min	45 min	60 min
CB	0.275	0.254	0.254	0.245	0.245	0.261	0.286	0.198	0.170	0.174	0.214	0.244	0.278	0.261
IE	0.456	0.433	0.532	0.489	0.514	0.572	0.592	0.415	0.425	0.392	0.461	0.478	0.578	0.624
JA	0.564	0.577	0.676	0.674	0.729	0.637	0.668	0.496	0.593	0.668	0.736	0.642	0.667	0.711
TC	0.631	0.800	0.724	0.695	0.844	0.786	0.726	0.636	0.591	0.578	0.639	0.749	0.752	0.784
TH	0.437	0.437	0.373	0.441	0.341	0.364	0.411	0.545	0.325	0.387	0.511	0.496	0.576	0.740
TS	0.600	0.719	0.755	0.617	0.783	0.675	0.705	0.563	0.610	0.549	0.514	0.615	0.694	0.653
CZ	1.420	1.580	1.578	1.597	1.129	1.566	1.645	1.227	1.050	1.050	1.072	1.269	1.186	1.110
JM	0.549	0.551	0.580	0.558	0.582	0.604	0.619	0.547	0.359	0.446	0.590	0.621	0.580	0.639
SKA	0.645	0.742	0.721	0.792	0.786	0.926	0.939	0.657	0.430	0.789	0.796	1.073	0.941	1.066
TB	0.525	0.528	0.708	0.660	0.742	0.745	0.734	0.525	0.381	0.596	0.568	0.670	0.761	0.699
MEAN	0.610	0.662	0.690	0.677	0.669	0.713	0.733	0.581	0.493	0.563	0.610	0.686	0.701	0.729
SD	0.305	0.362	0.353	0.358	0.258	0.357	0.368	0.261	0.239	0.242	0.227	0.294	0.241	0.237
SE	0.096	0.114	0.112	0.113	0.081	0.113	0.116	0.083	0.076	0.076	0.072	0.093	0.076	0.075
% diff from PRE		8.51%	13.07%	10.92%	9.70%	16.93%	20.07%		-15.06%	-3.11%	5.01%	18.05%	20.73%	25.45%
% diff PSmax to Con									-23.57%	-16.18%	-5.91%	8.35%	3.80%	5.38%

**Raw Data Experiment 1 : EMG;
EMG to MVC ratio (uvolts/Nm)**

Subject	Control MVC							PSmax MVC						
	PRE	POST	5 min	15 min	30 min	45 min	60 min	PRE	POST	5 min	15 min	30 min	45 min	60 min
CB	2.48	2.58	2.57	2.45	2.55	2.55	2.78	1.82	2.73	3.02	2.17	2.38	2.48	2.31
IE	2.55	2.81	3.23	3.02	2.78	3.36	3.35	2.23	2.83	2.52	2.74	3.03	3.41	3.54
JA	2.99	2.93	3.50	3.25	3.65	3.28	3.42	2.55	3.86	4.22	4.58	3.81	4.03	4.20
TC	4.05	5.46	4.92	4.77	5.74	5.02	4.56	4.24	5.47	5.40	4.99	5.85	5.66	5.76
TH	2.80	2.85	2.44	2.88	2.38	2.47	2.74	3.14	2.88	2.80	3.21	3.06	3.50	4.52
TS	2.93	3.46	3.59	3.35	3.88	3.58	3.67	2.72	3.89	3.14	2.96	3.55	3.87	3.61
CZ	9.84	11.21	11.17	11.95	8.67	12.52	12.17	8.60	9.38	8.56	8.65	9.92	8.48	8.45
JM	5.08	5.24	5.42	5.24	6.05	5.86	6.06	5.14	5.22	5.42	6.29	6.45	6.18	6.82
SKA	4.82	5.88	5.49	6.15	6.46	7.07	7.16	5.19	5.28	7.02	6.81	9.34	8.22	8.87
TB	3.19	3.80	4.46	4.24	4.61	4.58	4.59	3.06	3.08	4.09	3.73	4.51	5.04	4.72
MEAN	4.07	4.62	4.68	4.73	4.68	5.03	5.05	3.87	4.46	4.62	4.61	5.19	5.09	5.28
SD	2.22	2.62	2.53	2.80	2.03	3.01	2.88	2.03	2.03	1.99	2.08	2.66	2.04	2.16
SE	0.70	0.83	0.80	0.88	0.64	0.95	0.91	0.64	0.64	0.63	0.66	0.84	0.65	0.68
% diff from PRE		0.14	0.15	0.16	0.15	0.23	0.24		0.15	0.19	0.19	0.34	0.31	0.36
% diff PSmax to Con									0.02	0.04	0.03	0.19	0.08	0.12

**Raw Data Experiment 1 : EMG;
Twitch M-wave (mV)**

Subject	Control						PSmax					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	13.86	12.64	12.75	13.01	13.02	12.71	10.96	9.34	11.42	12.46	13.32	13.93
IE	18.72	18.80	18.82	18.56	19.07	18.57	17.82	15.24	17.69	18.25	18.67	19.01
JA	12.90	12.10	12.41	12.00	11.79	11.25	13.10	12.71	13.86	14.42	14.43	14.48
TC	21.69	21.82	21.71	22.16	21.48	21.89	15.61	13.81	17.22	17.52	17.72	18.79
TH	10.60	11.56	11.56	11.94	12.76	12.10	17.11	16.58	16.44	18.29	19.05	19.38
TS	15.29	15.95	17.50	16.50	16.75	16.42	15.26	15.64	16.83	17.82	18.30	18.71
CZ	24.08	22.88	22.53	21.89	22.00	20.22	19.26	18.90	18.91	20.35	19.38	18.62
JM	15.56	14.76	15.11	15.90	15.40	15.70	16.71	14.21	15.43	16.24	16.74	17.04
SKA	11.06	12.60	11.94	11.72	12.41	12.42	8.09	7.92	9.00	8.97	9.95	10.21
TB	19.80	22.42	22.12	21.96	22.39	22.17	20.16	15.70	20.06	20.42	21.51	22.76
MEAN	16.36	16.55	16.64	16.56	16.71	16.35	15.41	14.01	15.69	16.47	16.91	17.29
SD	4.55	4.55	4.46	4.36	4.24	4.19	3.75	3.30	3.40	3.60	3.42	3.54
SE	1.44	1.44	1.41	1.38	1.34	1.32	1.19	1.04	1.08	1.14	1.08	1.12
% diff from PRE		1.19%	1.76%	1.27%	2.14%	-0.07%		-9.10%	1.80%	6.92%	9.73%	12.22%
% diff PSmax to Con								-10.30%	0.04%	5.65%	7.59%	12.29%

**Raw Data Experiment 1 : EMG;
M-wave to Twitch Ratio (mV/Nm)**

Subject	Control						PSmax						
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min	
CB	0.98	0.93	0.92	0.95	1.01	0.97	0.85	0.84	0.91	1.01	1.10	1.13	
IE	1.02	1.13	1.08	1.08	1.14	1.11	1.09	1.09	1.17	1.26	1.33	1.31	
JA	0.81	0.82	0.80	0.80	0.83	0.81	0.65	0.76	0.85	0.92	0.95	0.96	
TC	1.28	1.53	1.42	1.60	1.48	1.36	0.99	1.25	1.18	1.32	1.39	1.53	
TH	0.67	0.75	0.67	0.68	0.78	0.72	1.03	1.22	0.97	1.18	1.20	1.15	
TS	0.89	0.98	1.14	1.06	1.11	1.06	0.89	0.94	1.02	1.13	1.18	1.22	
CZ	1.49	1.54	1.33	1.49	1.53	1.48	1.18	1.51	1.28	1.40	1.33	1.27	
JM	1.25	1.47	1.43	1.55	1.57	1.53	1.37	1.46	1.39	1.52	1.57	1.71	
SKA	0.86	1.02	0.99	0.98	1.00	1.09	0.64	0.76	0.87	0.86	0.85	0.96	
TB	1.14	1.46	1.35	1.38	1.45	1.43	1.15	1.12	1.36	1.47	1.51	1.58	
MEAN	1.04	1.16	1.11	1.16	1.19	1.16	0.98	1.09	1.10	1.21	1.24	1.28	
SD	0.25	0.31	0.27	0.33	0.29	0.28	0.23	0.27	0.20	0.23	0.23	0.26	
SE	0.08	0.10	0.08	0.10	0.09	0.09	0.07	0.09	0.06	0.07	0.07	0.08	
% diff from PRE		12.05%	7.06%	11.48%	14.59%	11.28%			11.31%	11.98%	22.75%	26.13%	30.49%
% diff PSmax to Con									-0.74%	4.93%	11.27%	11.54%	19.21%

**Raw Data Experiment 1 : EMG;
AEMG to M-Wave ratio**

Subject	Control MVC						PSmax MVC					
	PRE	POST	15 min	30 min	45 min	60 min	PRE	POST	15 min	30 min	45 min	60 min
CB	0.020	0.020	0.019	0.019	0.020	0.022	0.018	0.018	0.015	0.017	0.018	0.020
IE	0.024	0.023	0.026	0.028	0.030	0.031	0.023	0.028	0.022	0.025	0.026	0.030
JA	0.044	0.048	0.054	0.061	0.054	0.057	0.038	0.047	0.048	0.051	0.044	0.046
TC	0.029	0.037	0.032	0.038	0.037	0.034	0.041	0.043	0.034	0.036	0.042	0.040
TH	0.041	0.038	0.038	0.029	0.029	0.032	0.032	0.020	0.024	0.028	0.026	0.030
TS	0.039	0.045	0.035	0.047	0.040	0.042	0.037	0.039	0.033	0.029	0.034	0.037
CZ	0.059	0.069	0.071	0.052	0.071	0.075	0.064	0.056	0.056	0.053	0.065	0.064
JM	0.035	0.037	0.037	0.037	0.039	0.040	0.033	0.025	0.029	0.036	0.037	0.034
SKA	0.058	0.059	0.066	0.067	0.075	0.076	0.081	0.054	0.088	0.089	0.108	0.092
TB	0.027	0.024	0.030	0.034	0.033	0.033	0.026	0.024	0.030	0.028	0.031	0.033
MEAN	0.038	0.040	0.041	0.041	0.043	0.044	0.039	0.035	0.038	0.039	0.043	0.043
SD	0.013	0.016	0.017	0.015	0.018	0.019	0.019	0.014	0.021	0.021	0.026	0.021
SE	0.004	0.005	0.005	0.005	0.006	0.006	0.006	0.004	0.007	0.007	0.008	0.007
% diff from PRE		6.02%	8.60%	8.97%	13.58%	17.18%		-9.92%	-3.92%	-0.06%	10.07%	8.72%
% diff PSmax to Con								-15.94%	-12.52%	-9.02%	-3.52%	-8.46%

APPENDIX 3 - Raw Data Experiment 2

Raw Data Experiment 2: PSmax Parameters
Angular Displacement (Joint angle in Degrees dorsiflexion)

Subject	0.0	2.5	5.0	7.5	10.0	12.5	15.0	17.5	20.0	22.5	25.0	27.5	30.0
CB	26.0	26.5	27.5	26.5	26.5	28.0	27.5	28.5	29.0	30.0	30.0	30.0	30.5
IE	24.5	26.5	28.0	28.0	28.0	29.0	30.0	30.0	30.0	31.0	31.0	32.0	32.0
JA	32.5	35.0	35.0	36.0	36.5	37.0	38.0	37.5	37.5	37.5	37.5	38.0	38.5
TC	32.0	36.0	37.5	37.0	39.0	39.0	39.0	40.0	39.0	39.0	40.5	41.0	40.5
TH	31.0	32.5	33.0	34.0	34.5	34.0	34.5	35.0	35.5	36.0	36.0	36.0	36.5
TS	27.5	29.0	30.5	31.0	31.0	31.0	31.5	31.5	31.5	32.5	32.5	34.0	34.0
CZ	33.0	35.0	36.0	37.5	38.0	38.5	39.0	39.0	40.0	39.0	40.0	40.5	41.0
JM	29.0	33.0	35.0	35.5	35.5	36.0	36.0	36.5	36.5	36.5	37.0	37.5	38.0
SKA	41.0	42.5	45.5	46.5	47.5	47.0	48.0	48.0	48.0	48.0	48.0	48.0	48.0
TB	35.5	37.5	39.0	40.5	41.0	42.5	44.0	44.0	45.0	45.0	45.0	46.0	45.5
Mean	29.4	31.7	32.8	33.2	33.6	34.1	34.4	34.8	34.9	35.2	35.6	36.1	36.4
SD	3.2	3.9	3.8	4.2	4.6	4.3	4.4	4.3	4.2	3.6	4.0	3.9	3.9
SE	1.1	1.4	1.3	1.5	1.6	1.5	1.5	1.5	1.5	1.3	1.4	1.4	1.4

**Raw Data Experiment 2: PSmax Parameters;
Passive Torque at initiation and end of a stretch interval (Nm)**

Sub	0.00	2.25	2.50	4.75	5.00	7.25	7.50	9.75	10.00	12.25	12.50	14.75	15.00
CB	43.26	29.95	34.19	28.23	40.15	33.17	37.52	31.72	37.20	31.51	38.91	32.20	37.62
IE	31.94	24.42	37.89	29.25	36.55	31.56	37.84	31.56	39.40	33.92	41.97	34.84	46.21
JA	40.20	28.29	36.87	29.63	35.21	29.90	38.48	32.10	43.21	35.80	43.53	35.85	40.30
TC	30.86	23.83	29.84	25.49	35.92	29.90	35.64	31.08	37.03	32.04	38.81	33.60	37.52
TH	43.44	32.79	37.41	31.40	40.09	34.51	34.08	31.25	34.51	29.26	38.22	33.17	36.23
TS	45.68	33.81	48.47	40.09	44.60	39.66	46.64	41.06	52.65	46.11	47.66	43.35	42.56
CZ	31.16	24.31	30.75	24.21	28.55	24.80	31.18	26.57	29.90	27.53	31.29	27.32	31.99
JM	29.73	22.38	34.08	27.80	35.48	30.81	35.64	31.02	32.20	29.04	34.94	30.33	34.62
SKA	29.36	21.74	35.91	28.30	37.79	32.20	39.77	33.92	39.61	35.05	40.04	35.75	41.17
TB	34.40	25.28	34.19	27.96	37.73	30.22	37.89	32.15	39.02	33.49	41.22	35.10	42.46
MEAN	36.00	26.68	35.96	29.24	37.21	31.67	37.47	32.24	38.47	33.38	39.66	34.15	39.07
SD	6.43	4.28	5.14	4.31	4.17	3.81	4.06	3.61	6.31	5.23	4.50	4.19	4.26
SE	2.03	1.35	1.62	1.36	1.32	1.21	1.28	1.14	1.99	1.65	1.42	1.32	1.35

	17.25	17.50	19.75	20.00	22.25	22.50	24.75	25.00	27.25	27.50	29.75	30.00
	32.47	46.37	39.13	39.23	35.16	40.31	35.65	44.60	38.59	40.85	36.39	47.50
	39.77	40.15	36.66	44.01	39.28	44.55	38.85	40.85	37.30	42.72	36.41	46.48
	35.91	43.42	38.32	44.66	38.32	39.88	35.16	45.19	38.91	41.38	36.55	39.40
	32.96	37.30	33.12	38.20	34.14	37.89	34.40	40.52	35.59	34.46	31.18	40.42
	31.60	39.07	34.30	34.62	31.14	35.64	32.31	39.18	32.58	35.59	32.15	39.99
	39.13	47.66	42.99	47.00	43.87	35.96	32.27	45.89	41.06	33.71	31.29	41.65
	26.09	33.44	28.82	30.81	27.00	33.44	28.93	35.48	28.29	33.87	29.30	34.19
	30.23	33.92	30.22	33.71	30.65	33.71	30.49	34.46	31.18	36.50	33.60	34.99
	36.28	41.54	36.53	42.29	36.49	44.39	39.29	42.13	36.98	41.44	37.46	40.74
	36.82	41.00	36.77	44.01	38.16	40.68	36.60	41.97	36.30	44.44	38.75	40.85
	34.13	40.39	35.69	39.85	35.42	38.65	34.40	41.03	35.68	38.50	34.31	40.62
	4.25	4.73	4.22	5.42	4.91	4.01	3.41	3.85	3.90	4.07	3.21	4.19
	1.34	1.49	1.34	1.71	1.55	1.27	1.08	1.22	1.23	1.29	1.01	1.32

**Raw data Experiment 2; Isometric MVC;
Peak MVC (Nm)**

Subject	PRE			30 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	99.41	114.56	98.47	84.52	107.63	105.26	112.09	135.13	112.69
IE	132.61	157.76	164.60	127.73	167.40	166.93	127.53	165.77	164.35
JA	174.69	195.64	195.56	163.87	181.14	179.12	166.20	186.12	186.12
TC	130.76	151.05	143.31	110.05	138.54	144.13	113.45	129.93	126.19
TH	135.13	169.89	177.66	134.91	162.99	172.91	135.20	162.23	173.32
TS	176.26	189.25	173.19	155.87	179.49	176.44	159.36	182.06	172.69
CZ	119.84	144.90	136.40	111.70	127.61	130.73	110.93	122.37	130.69
JM	100.11	108.44	101.14	91.74	98.19	94.78	89.58	98.15	95.31
SKA	112.62	135.75	142.06	103.56	119.07	118.78	105.98	118.75	120.00
TB	157.03	177.59	186.31	130.18	149.30	161.97	133.37	148.64	158.37
MEAN	133.85	154.48	151.87	121.41	143.14	145.11	125.37	144.92	143.97
SD	27.86	29.54	33.68	25.98	29.64	31.08	23.97	28.92	30.73
SE	8.81	9.34	10.65	8.21	9.37	9.83	7.58	9.14	9.72
% Diff from PRE				-9.29%	-7.35%	-4.45%	-6.33%	-6.19%	-5.20%

**Raw data Experiment 2; Isometric MVC;
Interpolated Twitch (ITT) (Nm)**

Subject	PRE			30 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	1.64	3.10	2.80	0.80	2.22	3.27	1.94	0.13	1.44
IE	0.56	1.48	2.45	0.93	1.48	1.97	0.90	0.95	1.27
JA	0.00	0.00	0.86	0.22	0.43	0.47	0.30	0.62	0.67
TC	0.32	0.82	1.95	0.13	3.23	4.11	0.58	1.81	2.69
TH	0.24	0.00	1.01	0.00	1.20	1.89	1.16	0.86	0.99
TS	0.69	1.01	1.12	0.73	0.32	0.73	1.53	1.05	0.19
CZ	0.00	0.15	0.65	0.00	0.17	0.00	0.00	0.00	0.28
JM	0.39	0.30	1.12	0.17	0.32	0.34	0.00	0.26	0.45
SKA	0.19	0.17	0.00	0.00	0.00	0.19	0.00	0.56	0.41
TB	0.34	0.13	0.22	0.00	0.26	0.00	0.00	0.15	0.15
MEAN	0.44	0.72	1.22	0.30	0.96	1.30	0.64	0.64	0.85
SD	0.48	0.97	0.92	0.37	1.06	1.46	0.71	0.55	0.79
SE	0.15	0.31	0.29	0.12	0.34	0.46	0.23	0.17	0.25
% Diff from PRE				-31.81%	34.50%	6.49%	46.68%	-10.75%	-29.89%

**Raw data Experiment 2; Isometric MVC;
Motor Unit Activation (MUA) (%)**

Subject	PRE			30 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	85.28	76.17	76.90	92.60	81.58	71.06	81.54	98.92	87.87
IE	96.47	92.20	86.31	93.77	91.67	88.35	93.90	94.57	91.92
JA	100.00	100.00	94.83	98.20	97.17	96.70	97.42	95.60	94.66
TC	97.31	94.44	84.35	98.86	74.94	63.63	95.22	86.31	77.34
TH	98.30	100.00	94.26	100.00	93.05	88.48	91.30	94.67	94.05
TS	95.59	93.92	92.55	94.74	97.83	94.70	88.75	92.89	98.58
CZ	100.00	99.02	95.00	100.00	98.66	100.00	100.00	100.00	97.48
JM	96.02	97.02	87.34	98.16	96.84	96.02	100.00	97.40	94.76
SKA	98.18	98.77	100.00	100.00	100.00	98.43	100.00	95.54	96.55
TB	97.89	99.28	98.76	100.00	98.20	100.00	100.00	98.98	98.86
MEAN	96.50	95.08	91.03	97.63	92.99	89.74	94.81	95.49	93.21
SD	4.22	7.20	7.17	2.85	8.31	12.62	6.15	3.95	6.48
SE	1.33	2.28	2.27	0.90	2.63	3.99	1.95	1.25	2.05
% Diff from PRE				1.17%	-2.20%	-1.42%	-1.75%	0.43%	2.39%

**Raw data Experiment 2; Isometric MVC;
Twitch to MVC Ratio**

Subject	PRE			30 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	0.11	0.11	0.12	0.13	0.11	0.11	0.09	0.09	0.11
IE	0.12	0.12	0.11	0.12	0.11	0.10	0.12	0.11	0.10
JA	0.09	0.09	0.08	0.07	0.08	0.08	0.07	0.08	0.07
TC	0.09	0.10	0.09	0.10	0.09	0.08	0.11	0.10	0.09
TH	0.10	0.10	0.10	0.11	0.11	0.09	0.10	0.10	0.10
TS	0.09	0.09	0.09	0.09	0.08	0.08	0.09	0.08	0.08
CZ	0.12	0.11	0.10	0.11	0.10	0.09	0.11	0.11	0.09
JM	0.10	0.09	0.09	0.10	0.10	0.09	0.10	0.10	0.09
SKA	0.09	0.10	0.10	0.10	0.11	0.10	0.10	0.11	0.10
TB	0.10	0.10	0.10	0.09	0.10	0.08	0.09	0.10	0.08
MEAN	0.10	0.10	0.10	0.10	0.10	0.09	0.10	0.10	0.09
SD	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
SE	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
% Diff from PRE				1.05%	-2.65%	-7.42%	-3.79%	-4.72%	-8.00%

Raw data Experiment 2; EMG; MVC AEMG (mV/s)

Subject	PRE			30 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	0.285	0.329	0.316	0.386	0.429	0.433	0.487	0.518	0.435
IE	0.360	0.359	0.483	0.481	0.574	0.541	0.445	0.528	0.766
JA	0.501	0.578	0.599	0.637	0.641	0.596	0.496	0.595	0.630
TC	0.553	0.617	0.633	0.560	0.590	0.589	0.513	0.555	0.653
TH	0.392	0.484	0.614	0.443	0.517	0.788	0.459	0.759	0.842
TS	0.590	0.587	0.746	0.673	0.645	0.751	0.709	0.652	0.871
CZ	1.185	1.075	1.027	1.063	0.967	1.156	1.146	1.133	1.091
JM	0.434	0.469	0.546	0.533	0.573	0.579	0.518	0.601	0.590
SKA	0.563	0.681	0.820	0.708	0.731	0.822	0.953	0.847	0.954
TB	0.667	0.768	0.744	0.765	0.948	1.008	0.893	0.934	1.006
MEAN	0.553	0.595	0.653	0.625	0.661	0.726	0.662	0.712	0.784
SD	0.251	0.217	0.195	0.196	0.175	0.225	0.250	0.203	0.207
SE	0.079	0.069	0.062	0.062	0.055	0.071	0.079	0.064	0.065
% Diff from PRE				13.0%	11.2%	11.3%	19.7%	19.8%	20.1%

Raw data Experiment 2; EMG; AEMG to MVC Ratio

Subject	PRE			30 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	2.863	2.872	3.206	4.563	3.985	4.116	4.347	3.832	3.862
IE	2.715	2.278	2.936	3.764	3.430	3.243	3.492	3.185	4.663
JA	2.866	2.952	3.062	3.890	3.538	3.327	2.986	3.199	3.382
TC	4.228	4.081	4.420	5.087	4.257	4.089	4.525	4.273	5.175
TH	2.903	2.848	3.455	3.281	3.172	4.558	3.396	4.678	4.860
TS	3.349	3.101	4.306	4.317	3.595	4.258	4.447	3.583	5.043
CZ	9.888	7.419	7.526	9.514	7.579	8.843	10.333	9.257	8.346
JM	4.332	4.321	5.398	5.804	5.832	6.111	5.778	6.126	6.191
SKA	4.995	5.013	5.770	6.834	6.135	6.924	8.988	7.135	7.951
TB	4.249	4.325	3.992	5.873	6.347	6.222	6.697	6.285	6.352
MEAN	4.239	3.921	4.407	5.293	4.787	5.169	5.499	5.155	5.583
SD	2.139	1.503	1.456	1.846	1.545	1.800	2.477	1.997	1.627
SE	0.676	0.475	0.460	0.584	0.489	0.569	0.783	0.632	0.515
% Diff from PRE				24.9%	22.1%	17.3%	29.7%	31.5%	26.7%

**Raw Data Experiment 2: EMG;
AEMG to M-wave Ratio**

Subject	PRE			30 min			60 min		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	0.020	0.024	0.023	0.025	0.028	0.028	0.030	0.031	0.026
IE	0.017	0.017	0.025	0.023	0.028	0.027	0.021	0.025	0.038
JA	0.031	0.037	0.037	0.041	0.040	0.039	0.032	0.037	0.042
TC	0.030	0.034	0.036	0.027	0.031	0.032	0.024	0.028	0.033
TH	0.027	0.034	0.043	0.029	0.036	0.049	0.029	0.047	0.050
TS	0.036	0.035	0.044	0.037	0.035	0.041	0.039	0.035	0.048
CZ	0.075	0.067	0.061	0.067	0.060	0.072	0.069	0.071	0.067
JM	0.029	0.034	0.040	0.033	0.037	0.038	0.031	0.038	0.036
SKA	0.043	0.053	0.063	0.047	0.051	0.061	0.061	0.057	0.066
TB	0.032	0.037	0.036	0.035	0.044	0.048	0.036	0.038	0.043
MEAN	0.034	0.037	0.041	0.036	0.039	0.043	0.037	0.041	0.045
SD	0.016	0.014	0.013	0.013	0.010	0.014	0.016	0.014	0.013
SE	0.005	0.004	0.004	0.004	0.003	0.005	0.005	0.004	0.004
% Diff from PRE				7.20%	4.97%	6.66%	9.18%	9.31%	10.18%

Raw Data Experiment 2: Twitch Contractile Properties; Peak Twitch Torque (PTT) (Nm)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	11.14	13.01	12.12	8.92	11.40	11.32	10.49	12.05	11.59	10.81	12.05	11.30	10.63	12.42	11.75	10.51	12.08	11.87
IE	15.85	18.98	17.90	13.54	17.41	17.94	14.89	17.78	17.13	14.93	17.76	16.91	15.46	18.05	16.62	14.76	17.50	15.72
JA	15.15	18.41	16.62	11.16	14.21	14.89	12.40	15.97	15.89	12.24	15.17	14.26	11.99	14.52	13.30	11.63	14.09	12.54
TC	11.89	14.74	12.46	10.30	12.40	12.36	10.99	13.44	12.16	11.40	12.89	11.30	12.24	14.26	13.73	12.14	13.22	11.87
TH	14.09	17.13	17.60	12.16	15.29	17.60	14.74	18.39	17.72	14.26	17.27	16.40	14.81	17.54	17.48	13.34	16.13	16.64
TS	15.66	16.62	15.03	13.52	15.21	14.64	14.50	15.91	14.72	13.89	14.77	13.77	14.30	15.85	14.93	13.60	14.77	13.38
CZ	13.95	15.32	12.99	10.59	13.40	12.93	12.65	13.69	11.83	12.20	12.73	11.38	12.62	13.69	11.44	12.14	13.28	11.12
JM	9.79	10.08	8.85	8.32	9.10	8.65	8.98	9.63	8.67	9.24	10.14	8.55	10.16	10.91	8.63	9.24	10.00	8.59
SKA	10.44	13.79	14.21	8.94	11.44	11.38	9.59	11.79	11.89	10.40	12.69	12.08	11.22	13.24	12.58	10.32	12.56	11.87
TB	16.15	18.07	17.72	12.02	14.58	14.77	13.16	15.62	14.11	12.30	14.42	12.50	12.60	15.27	14.48	12.44	14.74	13.13
MEAN	13.41	15.62	14.55	10.95	13.44	13.65	12.24	14.43	13.57	12.17	13.99	12.85	12.60	14.58	13.49	12.01	13.84	12.67
SD	2.40	2.79	2.99	1.87	2.41	2.90	2.15	2.79	2.84	1.80	2.36	2.54	1.77	2.20	2.59	1.67	2.12	2.28
SE	0.76	0.88	0.95	0.59	0.76	0.92	0.68	0.88	0.90	0.57	0.75	0.80	0.56	0.70	0.82	0.53	0.67	0.72
% Diff from PRE				-18.4%	-13.9%	-6.2%	-8.7%	-7.6%	-6.7%	-9.3%	-10.4%	-11.7%	-6.0%	-6.7%	-7.3%	-10.4%	-11.4%	-12.9%

Raw Data Experiment 2: Twitch Contractile Properties; Time to Peak Torque (TPT) (ms)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	128.52	112.29	102.35	121.89	114.28	106.66	128.52	119.58	101.69	125.54	112.95	105.66	121.23	115.60	106.00	129.84	111.96	109.64
IE	116.93	111.63	102.68	109.31	106.00	100.70	106.66	107.32	104.34	107.65	112.62	100.36	116.26	112.29	100.36	114.61	109.64	103.01
JA	120.57	114.94	105.00	108.65	106.00	105.66	115.60	120.90	111.96	111.97	121.89	112.29	108.98	117.92	105.00	112.29	117.26	107.32
TC	93.41	94.40	86.78	87.45	89.43	80.82	96.06	95.40	89.43	101.03	98.71	84.13	98.71	96.06	87.78	96.72	99.37	85.13
TH	123.55	126.53	113.61	117.92	126.53	110.96	135.81	135.48	120.57	134.81	136.47	124.88	132.83	138.79	113.91	133.16	133.82	117.62
TS	98.71	93.08	91.09	96.06	94.73	87.11	91.75	94.73	93.08	95.06	91.42	90.10	96.06	98.71	91.75	97.05	93.74	90.43
CZ	139.78	138.46	115.27	124.54	138.46	117.59	135.48	133.82	123.88	143.09	134.48	123.88	139.45	150.05	125.54	148.72	139.78	126.20
JM	115.60	117.59	104.01	112.62	117.92	95.73	123.88	110.96	98.38	100.03	118.58	123.22	122.23	118.58	103.35	126.53	114.94	104.01
SKA	113.28	113.95	122.23	103.97	107.65	109.31	117.92	109.97	113.61	117.92	119.91	119.91	115.60	117.59	126.53	113.28	114.61	122.23
TB	124.88	124.54	119.91	114.94	114.94	108.31	117.26	117.26	114.94	123.22	126.53	112.29	125.21	114.94	112.29	119.91	121.89	111.30
MEAN	117.52	114.74	106.29	109.74	111.59	102.29	116.89	114.54	107.19	116.03	117.36	109.67	117.66	118.05	107.25	119.21	115.70	107.69
SD	13.64	13.76	11.65	11.49	14.36	11.36	15.13	13.86	11.61	15.77	14.27	14.43	13.75	16.20	12.76	16.10	13.95	12.96
SE	4.31	4.35	3.68	3.63	4.54	3.59	4.79	4.38	3.67	4.99	4.51	4.56	4.35	5.12	4.03	5.09	4.41	4.10
% Diff from PRE				-6.6%	-2.7%	-3.8%	-0.5%	-0.2%	0.8%	-1.3%	2.3%	3.2%	0.1%	2.9%	0.9%	1.4%	0.8%	1.3%

Raw Data Experiment 2: Twitch Contractile Properties; Rise Time (RT) (ms)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	80.16	69.89	63.60	76.52	71.88	68.23	76.52	72.87	65.58	76.52	72.54	66.91	75.52	66.91	78.50	78.50	73.20	67.90
IE	70.88	68.57	61.94	63.60	64.59	62.60	66.25	65.58	62.60	66.58	68.90	63.27	69.56	67.24	62.60	70.88	66.91	63.27
JA	69.23	69.23	63.60	64.59	65.25	63.27	70.55	72.21	68.23	67.57	72.54	67.57	67.90	70.55	66.25	67.90	74.53	67.24
TC	58.96	60.62	55.32	56.97	57.97	52.34	61.28	61.61	53.33	61.28	61.28	53.33	59.95	60.65	55.65	60.62	60.62	54.32
TH	74.53	72.87	74.20	72.54	74.20	66.25	81.82	79.83	69.56	81.48	80.49	69.56	81.82	81.15	69.23	81.15	80.82	66.58
TS	60.62	57.30	58.30	59.95	56.31	56.97	59.29	58.63	59.63	59.63	57.63	57.63	60.28	59.29	58.30	59.95	58.63	58.96
CZ	84.13	84.80	75.19	80.16	81.48	76.52	83.14	85.13	83.47	90.10	86.45	80.49	89.76	91.42	82.81	91.09	88.44	80.49
JM	71.55	68.23	61.28	69.56	66.91	60.62	72.87	65.92	61.94	62.60	69.23	73.87	72.54	69.56	63.60	73.54	69.56	62.60
SKA	68.90	71.22	73.53	70.55	68.58	70.22	70.55	69.89	71.22	73.53	73.53	73.20	72.21	72.54	75.19	71.22	72.87	75.52
TB	71.88	73.20	70.55	74.53	68.90	68.90	72.87	72.54	72.54	74.53	75.19	71.22	71.88	70.55	70.55	72.87	73.53	70.88
MEAN	71.08	69.59	65.75	68.90	67.61	64.59	71.51	70.42	66.81	71.38	71.78	67.71	72.14	70.99	68.27	72.77	71.91	66.78
SD	7.67	7.40	7.09	7.47	7.40	6.98	7.84	8.02	8.27	9.71	8.38	8.00	9.00	9.43	8.74	9.32	8.81	7.67
SE	2.43	2.34	2.24	2.36	2.34	2.21	2.48	2.54	2.62	3.07	2.65	2.53	2.85	2.98	2.76	2.95	2.79	2.42
% Diff from PRE				-3.1%	-2.9%	-1.8%	0.6%	1.2%	1.6%	0.4%	3.1%	3.0%	1.5%	2.0%	3.8%	2.4%	3.3%	1.6%

Raw Data Experiment 2: Twitch Contractile Properties; (MRTD) (Nm/s)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	148.19	193.24	199.76	146.41	159.45	169.53	144.63	195.02	185.53	148.78	174.27	170.12	156.49	185.53	181.98	144.04	169.53	178.42
IE	260.22	317.13	317.72	235.92	287.49	307.64	251.92	301.71	302.31	244.81	297.57	289.86	253.70	315.35	290.45	237.10	301.71	269.11
JA	234.73	288.08	279.19	187.90	226.43	244.81	177.83	229.40	270.30	194.43	213.99	227.03	177.83	221.10	206.87	183.16	204.50	193.83
TC	209.84	273.26	238.29	194.43	223.47	246.00	193.24	236.51	240.66	199.17	226.43	222.28	222.28	249.55	261.41	217.54	238.29	225.25
TH	234.14	285.71	302.31	223.47	256.07	280.97	222.82	294.60	285.71	212.80	256.07	274.45	231.18	257.85	285.71	205.69	240.07	281.56
TS	264.96	308.23	227.03	227.03	276.23	256.07	253.11	306.46	259.04	240.66	271.48	246.00	240.07	280.38	266.15	238.29	264.96	268.52
CZ	201.54	213.39	188.50	160.64	181.38	187.90	181.98	187.31	167.75	165.38	169.53	147.00	167.16	173.09	157.08	162.42	176.05	159.45
JM	168.94	176.64	159.45	143.45	152.93	157.67	152.93	161.82	150.56	147.60	159.45	148.78	175.46	182.57	145.82	151.75	164.19	143.45
SKA	171.90	211.02	212.80	136.93	182.57	174.86	147.00	180.20	177.24	155.90	180.79	169.53	169.53	201.54	202.13	148.19	178.42	195.02
TB	254.29	285.12	283.93	183.57	223.47	228.81	215.76	239.47	214.58	201.54	213.59	191.46	206.28	240.07	228.81	194.43	214.58	200.95
MEAN	214.88	255.18	240.90	183.98	216.95	225.43	194.12	233.25	225.37	191.11	216.32	208.65	200.00	230.70	222.64	188.26	215.23	211.56
SD	41.49	51.20	52.76	36.59	47.20	50.85	40.68	53.09	53.63	35.87	46.90	51.14	34.95	46.47	52.21	36.02	45.70	48.07
SE	13.12	16.19	16.68	11.57	14.92	16.08	12.86	16.79	16.96	11.34	14.83	16.17	11.05	14.69	16.51	11.39	14.45	15.20
% Diff from PRE				-14.4%	-15.0%	-6.4%	-9.7%	-8.6%	-6.4%	-11.1%	-15.2%	-13.4%	-6.9%	-9.6%	-7.6%	-12.4%	-15.7%	-12.2%

Raw Data Experiment 2: Twitch Contractile Properties; Maximum Rate of Torque Relaxation (MRTR) (-Nm/s)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	64.02	275.34	141.82	71.13	102.84	75.28	132.93	190.72	69.95	73.21	227.77	71.72	48.31	282.30	73.50	79.43	193.39	70.54
IE	131.59	403.67	355.21	145.82	316.09	368.55	151.75	288.53	433.60	142.26	590.39	280.38	147.00	316.24	261.11	128.63	304.68	324.09
JA	120.33	128.63	150.56	106.70	117.96	234.29	111.44	122.70	341.73	106.10	93.21	191.46	106.70	180.05	257.11	104.92	218.88	191.91
TC	102.55	136.93	164.05	120.92	114.40	230.44	112.03	351.21	100.47		411.67	90.69	122.11	221.54	80.91	111.44	430.05	92.47
TH	124.48	127.44	307.49	113.81	121.52	280.23	127.44	184.94	285.71	123.29	202.28	308.98	126.85	117.37	240.81	110.85	115.85	264.22
TS	131.00	237.25	184.94	147.60	113.37	193.24	133.37	220.51	188.50	89.65	294.90	246.00	120.92	290.75	143.45	117.37	389.00	168.34
CZ	91.28	132.04	254.00	112.62	132.04	163.45	109.66	108.47	234.58	101.36	387.66	315.35	103.14	430.05	109.36	94.25	209.84	331.20
JM	70.54	169.64	137.37	81.21	68.17	56.31	65.80	219.47	150.71	62.68	319.65	63.43	77.06	287.49	306.46	68.17	247.77	87.73
SKA	63.43	70.54	82.69	75.87	73.50	68.32	78.84	80.02	156.34	77.06	80.47	80.62	82.99	81.80	89.95	71.72	81.21	71.72
TB	114.40	111.44	511.26	118.55	107.88	140.04	106.10	265.56	283.19	91.88	256.81	84.32	97.21	123.29	197.39	91.88	290.90	132.48
MEAN	101.36	179.29	228.94	109.42	126.78	181.02	112.94	203.21	224.48	96.39	286.48	173.30	103.23	233.09	176.01	97.87	248.16	173.47
SD	27.37	99.30	130.00	26.71	69.53	101.03	25.77	84.83	113.24	25.15	152.99	106.14	28.48	108.37	86.89	20.23	109.87	101.49
SE	8.66	31.40	41.11	8.45	21.99	31.95	8.15	26.82	35.81	8.38	48.38	33.56	9.01	34.27	27.48	6.40	34.74	32.09
% Diff from PRE				8.0%	-29.3%	-20.9%	11.4%	13.3%	-1.9%	-4.9%	59.8%	-24.3%	1.8%	30.0%	-23.1%	-3.4%	38.4%	-24.2%

Raw Data Experiment 2: Twitch Contractile Properties; Torque Time Integral (TTI) (Nms)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	3.76	2.63	2.94	1.81	2.79	2.37	2.78	3.23	2.39	2.72	3.34	2.53	2.57	3.45	2.56	2.42	3.30	2.71
IE	2.92	5.13	4.39	2.12	3.99	4.63	2.45	4.42	4.63	2.53	5.04	4.20	2.68	4.39	3.97	2.62	4.25	4.09
JA	2.81	3.87	3.65	1.85	2.71	3.50	2.26	3.30	4.22	2.26	3.43	3.51	2.06	3.45	3.30	2.00	3.61	3.11
TC	1.85	3.10	2.61	1.40	2.38	2.65	1.69	3.52	2.37		3.68	1.87	1.89	3.41	2.71	1.97	3.68	2.09
TH	2.88	4.08	4.80	2.25	3.02	4.38	2.95	4.50	4.75	2.90	4.33	4.38	2.99	4.11	4.50	2.75	4.02	4.53
TS	2.50	3.64	3.17	1.93	2.86	2.98	2.31	3.53	3.18	2.72	3.50	2.73	2.49	3.56	3.06	2.28	2.95	3.72
CZ	2.58	2.86	3.42	1.74	2.81	3.10	2.54	3.78	3.20	2.56	3.84	3.29	2.55	4.02	2.83	2.60	3.58	3.34
JM	1.94	2.57	2.21	1.44	1.95	1.79	1.93	2.85	2.32	1.97	3.26	2.03	2.14	3.31	2.66	1.95	3.01	2.10
SKA	2.28	3.19	3.65	1.61	2.29	2.66	1.77	2.43	3.09	2.06	2.80	3.09	2.15	2.83	3.20	2.02	2.70	2.91
TB	3.39	4.07	5.26	2.13	3.10	3.61	2.64	4.22	3.84	2.55	4.06	3.08	2.56	3.86	3.70	2.57	4.20	3.37
MEAN	2.69	3.51	3.61	1.83	2.79	3.17	2.33	3.58	3.40	2.47	3.73	3.07	2.41	3.64	3.25	2.32	3.53	3.20
SD	0.60	0.80	0.97	0.29	0.55	0.88	0.43	0.67	0.92	0.31	0.63	0.83	0.34	0.46	0.63	0.31	0.54	0.79
SE	0.19	0.25	0.31	0.09	0.17	0.28	0.13	0.21	0.29	0.10	0.20	0.26	0.11	0.14	0.20	0.10	0.17	0.25
% Diff from PRE				-32.1%	-20.6%	-12.3%	-13.3%	1.8%	-5.8%	-8.0%	6.1%	-14.9%	-10.5%	3.6%	-10.0%	-13.9%	0.5%	-11.4%

Raw Data Experiment 2: Twitch Contractile Properties; TTI to Half Relaxation (TTIHRT) (Nms)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	2.06	2.24	1.89	1.53	1.93	1.81	1.93	2.19	1.89	2.02	2.24	1.90	1.92	2.23	1.90	1.93	2.21	1.97
IE	2.53	3.14	2.69	1.87	2.61	2.68	2.15	2.76	2.62	2.20	2.89	2.58	2.31	2.86	2.54	2.24	2.78	2.49
JA	2.39	2.95	2.51	1.57	2.12	2.21	1.80	2.56	2.60	1.84	2.48	2.30	1.77	2.32	2.05	1.72	2.36	1.99
TC	1.55	2.05	1.58	1.22	1.60	1.52	1.45	1.97	1.50		1.99	1.39	1.61	2.10	1.77	1.66	1.97	1.47
TH	2.46	3.09	3.04	1.99	2.58	2.93	2.50	3.26	3.09	2.47	3.13	2.89	2.58	3.24	3.09	2.35	2.99	2.96
TS	2.50	2.15	1.96	1.59	1.85	1.81	1.87	2.12	1.93	1.95	2.06	1.80	1.93	2.14	1.95	1.83	2.07	1.78
CZ	2.58	2.86	2.20	1.53	2.86	2.08	2.09	2.14	2.06	2.10	2.38	2.01	2.17	2.56	1.97	2.20	2.46	2.03
JM	1.59	1.63	1.31	1.23	1.43	1.24	1.59	1.78	1.41	1.36	1.96	1.68	1.76	2.05	1.48	1.62	1.89	1.39
SKA	1.96	2.68	2.68	1.40	1.90	1.94	1.55	2.03	2.13	1.77	2.27	2.21	1.95	2.32	2.29	1.74	2.21	2.14
TB	2.87	3.37	3.27	1.83	2.41	2.47	2.18	2.81	2.48	2.13	2.71	2.17	2.12	3.86	2.48	2.13	2.76	2.27
MEAN	2.25	2.62	2.31	1.58	2.13	2.07	1.91	2.36	2.17	1.98	2.41	2.09	2.01	2.57	2.15	1.94	2.37	2.05
SD	0.44	0.57	0.63	0.26	0.47	0.52	0.33	0.46	0.53	0.31	0.39	0.44	0.29	0.59	0.46	0.27	0.37	0.46
SE	0.14	0.18	0.20	0.08	0.15	0.16	0.10	0.15	0.17	0.10	0.12	0.14	0.09	0.19	0.15	0.08	0.12	0.15
% Diff from PRE				-29.9%	-18.6%	-10.5%	-15.0%	-9.7%	-6.1%	-11.9%	-7.8%	-9.5%	-10.5%	-1.8%	-7.0%	-13.7%	-9.4%	-11.4%

Raw Data Experiment 2: Twitch Contractile Properties; Half Relaxation Time (HRT) (ms)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	122.89	120.90	111.63	107.32	113.95	110.96	117.26	123.88	121.56	123.88	135.14	123.88	119.58	125.54	116.59	115.27	132.49	118.25
IE	96.72	111.63	101.36	75.19	95.73	101.36	86.78	102.02	104.01	89.76	107.98	106.99	84.13	101.36	107.98	90.10	103.68	113.28
JA	90.76	101.69	99.70	81.15	95.40	96.72	83.14	96.39	110.30	90.43	99.04	105.66	90.43	99.70	106.00	87.78	110.30	108.65
TC	83.14	95.73	88.44	72.54	85.46	88.44	81.15	103.35	81.15	81.15	103.35	81.15	78.83	103.68	89.76	87.11	103.35	86.12
TH	104.67	117.59	115.27	97.38	97.71	111.30	90.76	101.69	111.63	96.39	106.00	109.31	99.37	108.31	115.93	100.70	112.62	119.58
TS	80.16	85.13	89.10	66.58	73.87	85.46	84.47	88.44	89.43	94.40	99.04	90.76	87.45	87.45	88.44	86.78	98.71	94.40
CZ	109.31	115.60	116.26	72.87	115.60	104.01	88.11	110.96	112.95	92.08	121.56	119.58	95.06	106.66	111.30	96.06	113.94	124.88
JM	102.68	102.68	99.37	83.14	93.08	101.36	107.98	134.48	123.55	115.93	140.78	115.27	106.33	132.49	132.83	104.01	137.46	116.59
SKA	134.81	145.08	131.17	99.70	116.26	121.56	99.37	120.90	128.19	110.30	121.56	127.19	106.33	117.92	119.24	111.96	122.89	121.89
TB	109.31	122.23	128.85	86.45	105.66	117.92	102.02	122.89	122.56	105.66	124.54	122.23	97.71	125.54	119.91	107.32	128.85	123.55
MEAN	103.45	111.83	108.12	84.23	99.27	103.91	94.10	110.50	110.53	100.00	115.90	110.20	96.52	110.87	110.80	98.71	116.43	112.72
SD	16.98	16.67	15.03	13.42	13.81	11.82	12.02	14.54	15.30	13.42	14.96	14.86	12.12	14.09	13.64	10.72	13.34	12.92
SE	5.37	5.27	4.75	4.24	4.37	3.74	3.80	4.60	4.84	4.24	4.73	4.70	3.83	4.46	4.31	3.39	4.22	4.09
% Diff from PRE				-18.6%	-11.2%	-3.9%	-9.0%	-1.2%	2.2%	-3.3%	3.6%	1.9%	-6.7%	-0.9%	2.5%	-4.6%	4.1%	4.3%

Raw Data Experiment 2: EMG; M-Wave (M-Wave) (mV)

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	14.27	13.69	13.88	14.88	13.83	14.01	14.48	14.48	14.59	15.21	15.47	15.71	15.70	15.14	16.00	16.45	16.55	16.74
IE	20.84	20.65	19.38	16.81	17.33	17.31	18.46	19.02	18.46	20.94	20.74	20.07	21.61	20.46	20.67	21.48	20.78	20.41
JA	16.21	15.47	16.09	14.19	16.57	13.74	14.30	15.23	14.95	15.44	15.86	15.18	16.32	17.20	15.68	15.66	16.06	15.13
TC	18.48	18.15	17.63	16.84	14.66	14.57	18.57	17.32	16.53	20.43	19.06	18.47	20.71	19.99	19.73	21.35	20.19	19.98
TH	14.43	14.31	14.32	15.52	12.35	12.57	15.25	14.00	14.50	15.13	14.20	16.04	15.69	16.33	16.19	15.74	16.32	16.81
TS	16.31	16.68	16.79	13.24	14.41	14.80	17.32	17.37	17.74	18.41	18.62	18.13	18.51	18.54	17.51	18.30	18.59	18.04
CZ	15.87	16.11	16.85	12.29	14.03	14.27	15.41	15.73	15.70	15.86	16.03	16.16	16.85	16.90	16.14	16.53	16.02	16.23
JM	14.79	13.76	13.69	13.80	13.11	13.12	15.88	14.56	14.26	16.27	15.31	15.17	15.70	15.36	15.55	16.61	15.91	16.27
SKA	13.08	12.92	13.04	12.85	13.38	12.63	14.09	14.17	13.35	14.98	14.28	13.54	15.24	14.57	13.86	15.55	14.76	14.50
TB	20.85	20.74	20.84	17.24	17.02	17.55	21.21	20.20	20.24	21.76	21.51	21.09	24.24	22.79	22.31	24.89	24.52	23.18
MEAN	16.51	16.25	16.25	14.76	14.67	14.46	16.50	16.21	16.03	17.44	17.11	16.96	18.06	17.73	17.36	18.26	17.97	17.73
SD	2.71	2.82	2.57	1.78	1.73	1.74	2.33	2.17	2.18	2.69	2.66	2.39	3.11	2.67	2.67	3.22	3.03	2.70
SE	0.86	0.89	0.81	0.56	0.55	0.55	0.74	0.69	0.69	0.85	0.84	0.76	0.98	0.84	0.84	1.02	0.96	0.85
% Diff from PRE				-10.6%	-9.7%	-11.0%	-0.1%	-0.2%	-1.4%	5.6%	5.3%	4.3%	9.3%	9.1%	6.8%	10.6%	10.6%	9.1%

Raw Data Experiment 2: EMG; M-Wave to Twitch Ratio

Subject	PRE			POST			15 MIN POST			30 MIN POST			45 MIN POST			60 MIN POST		
	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D	JA 0D	JA 10D	JA 20D
CB	1.28	1.05	1.15	1.67	1.21	1.24	1.38	1.20	1.26	1.41	1.28	1.39	1.48	1.22	1.36	1.57	1.37	1.41
IE	1.31	1.09	1.08	1.24	1.00	0.96	1.24	1.07	1.08	1.40	1.17	1.19	1.40	1.13	1.24	1.46	1.19	1.30
JA	1.07	0.84	0.97	1.27	1.17	0.92	1.15	0.95	0.94	1.26	1.05	1.06	1.36	1.18	1.18	1.35	1.14	1.21
TC	1.55	1.23	1.41	1.63	1.18	1.18	1.69	1.29	1.36	1.79	1.48	1.63	1.69	1.40	1.44	1.76	1.53	1.68
TH	1.02	0.84	0.81	1.28	0.81	0.71	1.03	0.76	0.82	1.06	0.82	0.98	1.06	0.93	0.93	1.18	1.01	1.01
TS	1.04	1.00	1.12	0.98	0.95	1.01	1.19	1.09	1.21	1.33	1.26	1.32	1.29	1.17	1.17	1.35	1.26	1.35
CZ	1.14	1.05	1.30	1.16	1.05	1.10	1.22	1.15	1.33	1.30	1.26	1.42	1.34	1.23	1.41	1.36	1.21	1.46
JM	1.51	1.37	1.55	1.66	1.44	1.52	1.77	1.51	1.64	1.76	1.51	1.77	1.55	1.41	1.80	1.80	1.59	1.89
SKA	1.25	0.94	0.92	1.44	1.17	1.11	1.47	1.20	1.12	1.44	1.13	1.12	1.36	1.10	1.10	1.51	1.17	1.22
TB	1.29	1.15	1.18	1.43	1.17	1.19	1.61	1.29	1.43	1.77	1.49	1.69	1.92	1.49	1.54	2.00	1.66	1.77
MEAN	1.25	1.06	1.15	1.38	1.11	1.09	1.38	1.15	1.22	1.45	1.24	1.36	1.44	1.23	1.32	1.53	1.31	1.43
SD	0.18	0.17	0.22	0.23	0.17	0.21	0.25	0.20	0.24	0.25	0.22	0.27	0.24	0.17	0.25	0.25	0.22	0.28
SE	0.06	0.05	0.07	0.07	0.05	0.07	0.08	0.06	0.08	0.08	0.07	0.09	0.07	0.05	0.08	0.08	0.07	0.09
% Diff from PRE				10.3%	5.5%	-4.6%	10.3%	9.2%	6.2%	16.4%	17.9%	18.2%	15.8%	16.3%	14.8%	22.8%	24.4%	24.5%

APPENDIX 4 - ANOVA summary tables**ANOVA TABLES FOR EXPERIMENTS 1 AND 2**

ANOVA SUMMARY TABLES FOR EXPERIMENT 1

Exp 1: ISOMETRIC MVC - Con vs PSmax

Summary of all Effects; MVC

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1*	5792.145*	9*	333.0513*	17.39115*	.002411*
2	6*	1255.966*	54*	47.8110*	26.26941*	.000000*
12	6*	756.484*	54*	52.9097*	14.29765*	.000000*

Summary of all Effects; ITT

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1*	10.58640*	9*	1.433666*	7.384149*	.023709*
2	6*	3.35772*	54*	.630600*	5.324640*	.000225*
12	6*	2.18024*	54*	.371378*	5.870679*	.000091*

Summary of all Effects; MUA

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1*	.089854*	9*	.012157*	7.390903*	.023661*
2	6*	.024251*	54*	.004430*	5.474396*	.000175*
12	6*	.016866*	54*	.002558*	6.594332*	.000029*

Exp 1: TWITCH CONTRACTILE PROPERTIES - Con vs PSmax

Summary of all Effects; PTT

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	14.81221	9	4.187993	3.53683	.092711
2	5*	11.82981*	45*	.650684*	18.18057*	.000000*
12	5*	1.05880*	45*	.372025*	2.84605*	.025712*

Summary of all Effects; TPT

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	457.8223	9	225.7779	2.027755	.188182
2	5*	208.3967*	45*	35.4326*	5.881499*	.000290*
12	5*	100.0602*	45*	14.9548*	6.690856*	.000098*

Summary of all Effects; RT

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1*	206.6400*	9*	34.16249*	6.048740*	.036193*
2	5*	122.5809*	45*	30.37650*	4.035386*	.004133*
12	5	16.7498	45	21.79407	.768550	.577379

Summary of all Effects; MRTD

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	2136.268	8	506.1421	4.22069	.073997
2	5*	5627.820*	40*	137.6182*	40.89444*	.000000*
12	5	209.610	40	92.8400	2.25776	.067075

Summary of all Effects; MRTR

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	1300.01	9	17905.83	.072603	.793660
2	5*	38160.59*	45*	6816.45*	5.598305*	.000429
12	5	10395.84	45	5689.35	1.827245	.126699

Summary of all Effects; TTI

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1*	3.873613*	9*	.550297*	7.039136*	.026340*
2	5*	1.063319*	45*	.112861*	9.421479*	.000003*
12	5*	.473665*	45*	.098831*	4.792685*	.001350*

Summary of all Effects; design: TTIHRT

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1*	2.363213*	9*	.151065*	15.64367*	.003328*
2	5*	.236425*	45*	.033525*	7.05230*	.000061*
12	5*	.206261*	45*	.023762*	8.68027*	.000008*

Summary of all Effects; HRT

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1*	4083.217*	9*	138.4152*	29.49978*	.000416*
2	5*	937.165*	45*	52.4576*	17.86518*	.000000*
12	5*	253.852*	45*	46.5886*	5.44881*	.000529*

Summary of all Effects; design: Twitch to MVC ratio

1-CONDITIO, 2-TIME

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	.000549	9	.000402	1.367520	.272276
2	6*	.000435*	54*	.000101*	4.321638*	.001249*
12	6*	.000354*	54*	.000067*	5.299786*	.000234*

Exp 1: PASSIVE TENSION - Con vs PSm_{ax}

Summary of all Effects; Passive Torque
 1-CONDITIO, 2-TIME, 3-MVC, 4-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	152.21	9	39.9109	3.81374	.082584
2	6*	29.65*	54*	2.2161*	13.38051*	.000000*
3	1*	79.36*	9*	2.8722*	27.62980*	.000523*
4	2*	11199.22*	18*	180.4750*	62.05417*	.000000*
12	6*	18.38*	54*	1.2080*	15.21199*	.000000*
13	1	6.28	9	1.5457	4.06056	.074713
23	6*	3.57*	54*	1.1983*	2.98094*	.013750*
14	2*	81.02*	18*	13.6536*	5.93408*	.010485*
24	12*	9.82*	108*	1.0253*	9.58042*	.000000*
34	2*	6.58*	18*	.7466*	8.81597*	.002142*
123	6	1.30	54	.5790	2.24698	.052317
124	12*	6.90*	108*	.4085*	16.89457*	.000000*
134	2*	2.33*	18*	.2856*	8.15003*	.003019*
234	12	.28	108	.2851	.98894	.464264
1234	12	.36	108	.2331	1.55378	.116373

Exp 1: EMG DATA**Summary of all Effects; MVC AEMG****1-CONDITIO, 2-TIME**

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	.109715	9	.052900	2.074030	.183689
2	6*	.064436*	54*	.007122*	9.047753*	.000001*
12	6*	.023709*	54*	.005801*	4.087183*	.001885*

Summary of all Effects; design: EMG to MVC Ratio**1-CONDITIO, 2-TIME**

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	.046705	9	2.821628	.016553	.900458
2	6*	3.189795*	54*	.353316*	9.028167*	.000001*
12	6	.328689	54	.303570	1.082747	.384429

Summary of all Effects; design: M-Wave**1-CONDITIO, 2-TIME**

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	9.605021	9	28.61473	.33567	.576553
2	5*	7.164590*	45*	.74231*	9.65175*	.000003*
12	5*	7.321523*	45*	.41466*	17.65683*	.000000*

Summary of all Effects; design: MWave-Twitch Ratio**1-CONDITIO, 2-TIME**

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	.006983	9	.086730	.08051	.783026
2	5*	.126190*	45*	.006210*	20.31905*	.000000*
12	5*	.027719*	45*	.004680*	5.92297*	.000274*

Summary of all Effects; Exp 1 AEMG to M-wave Ratio**1-CONDITIO, 2-TIME**

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	.000068	9	.000195	.348756	.569349
2	5*	.000113*	45*	.000045*	2.502885*	.044040*
12	5	.000026	45	.000019	1.373593	.252160

ANOVA SUMMARY TABLES FOR EXPERIMENT 2

Exp 2: ISOMETRIC MVC

Summary of all Effects; Exp 2 MVC

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	2*	903.984*	18*	124.7603*	7.24577*	.004915*
2	2*	4151.873*	18*	124.7507*	33.28135*	.000001*
12	4	27.145	36	18.2544	1.48701	.226540

Summary of all Effects; Exp 2 ITT

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	2	.150508	18	.424341	.354686	.706187
2	2*	3.313341*	18*	.510445*	6.491086*	.007541*
12	4	.495984	36	.230568	2.151144	.094439

Summary of all Effects; Exp 2 MUA

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	2	8.7503	18	27.47445	.318488	.731263
2	2*	191.8864*	18*	34.88376*	5.500737*	.013667*
12	4	30.0215	36	15.58256	1.926608	.127104

Exp 2: TWITCH CONTRACTILE PROPERTIES

Summary of all Effects; Exp 2 PTT

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	13.54825*	45*	1.079252*	12.55337*	.000000*
2	2*	65.91748*	18*	2.613231*	25.22451*	.000006*
12	10*	1.55490*	90*	.147779*	10.52181*	.000000*

Summary of all Effects; Exp 2 TPT

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	185.795*	45*	19.5887*	9.48484*	.000003*
2	2*	1639.142*	18*	105.9968*	15.46407*	.000123*
12	10	18.929	90	19.7922	.95639	.486743

Summary of all Effects; Exp 2 RT

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	54.4906*	45*	4.51680*	12.06397*	.000000*
2	2*	363.7395*	18*	28.90916*	12.58215*	.000381*
12	10	3.2953	90	6.52525	.50500	.882433

Summary of all Effects; Exp2 MRTD

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	4371.89*	45*	303.2032*	14.41900*	.000000*
2	2*	18206.61*	18*	928.3387*	19.61203*	.000030*
12	10*	278.25*	90*	107.5196*	2.58792*	.008364*

Summary of all Effects; Exp 2 MRTR

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5	7450.1	40	3224.65	2.31038	.061878
2	2*	181196.0*	16*	16408.71*	11.04268*	.000970*
12	10*	11590.0*	80*	3944.79*	2.93805*	.003445*

Summary of all Effects; Exp 2 TTI

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	1.51274*	40*	.090393*	16.73512*	.000000*
2	2*	19.55133*	16*	.732807*	26.68007*	.000008*
12	10*	.29122*	80*	.074721*	3.89745*	.000246*

Summary of all Effects; Exp 2 TTIHRT

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	.689926*	40*	.054409*	12.68032*	.000000*
2	2*	3.045391*	16*	.154002*	19.77503*	.000047*
12	10*	.073031*	80*	.022138*	3.29893*	.001271*

Summary of all Effects; Exp 2 HRT

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	746.099*	45*	78.2141*	9.53918*	.000003*
2	2*	3905.863*	18*	130.2525*	29.98687*	.000002*
12	10*	86.308*	90*	22.8570*	3.77602*	.000286*

Summary of all Effects; Exp 2 Twitch to MVC ratio

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	2	.000224	18	.000090	2.487845	.111182
2	2*	.000639*	18*	.000070*	9.090794*	.001867*
12	4*	.000043*	36*	.000016*	2.677066*	.047227*

Exp 2: EMG DATA

Summary of all Effects; Exp 2 MVC AEMG

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	2*	.108015*	18*	.008002*	13.49822*	.000262*
2	2*	.088408*	18*	.008996*	9.82735*	.001303*
12	4	.000402	36	.003442	.11672	.975740

Summary of all Effects; Exp 2 EMG to MVC ratio

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	2*	12.01893*	18*	.570284*	21.07533*	.000019*
2	2	1.69880	18	.531968	3.19343	.065021
12	4	.05947	36	.180998	.32854	.856946

Summary of all Effect; Exp 2 M-wave

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	45.15244*	45*	1.668913*	27.05501*	.000000*
2	2*	3.16589*	18*	.876854*	3.61051*	.048038*
12	10	.07780	90	.284983	.27300	.985600

Summary of all Effects; Exp 2 M-wave to Twitch Ratio

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	5*	.321035*	45*	.016838*	19.06579*	.000000*
2	2*	.750583*	18*	.030270*	24.79648*	.000007*
12	10*	.013574*	90*	.002889*	4.69803*	.000022*

Summary of all Effects; Exp 2 AEMG to M-wave

1-TIME, 2-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	2*	.000099*	18*	.000022*	4.43624*	.027145*
2	2*	.000384*	18*	.000036*	10.68152*	.000874*
12	4	.000001	36	.000012	.10198	.981083

ANOVA SUMMARY TABLES COMPARING EXPERIMENT 1 TO EXPERIMENT 2

PSmax PARAMETER COMPARISON Exp 1 vs Exp 2

Summary of all Effects; Joint angle

1-EXP, 2-STRETCH

	df	MS	df	MS		
Effect	Effect	Effect	Error	Error	F	p-level
1	1	10.00385	9	4.993162	2.00351	.190595
2	12*	77.09039*	108*	1.060826*	72.67014*	.000000*
12	12	.28301	108	.161218	1.75547	.064978

Summary of all Effects; Peak passive torque

1-EXP, 2-STRETCH, 3-TIME

	df	MS	df	MS		
Effect	Effect	Effect	Error	Error	F	p-level
1	1	181.881	9	301.7008	.6029	.457410
2	11*	101.372*	99*	14.5980*	6.9442*	.000000*
3	1*	3915.913*	9*	9.7246*	402.6798*	.000000*
12	11	23.789	99	12.7783	1.8617	.053679
13	1	8.938	9	5.4767	1.6319	.233401
23	11*	18.213*	99*	.9276*	19.6338*	.000000*
123	11	1.051	99	.7382	1.4239	.174112

MVC COMPARISON Exp 1 vs Exp 2

Summary of all Effects; Exp 1 vs Exp 2 MVC

1-EXP, 2-TIME

	df	MS	df	MS		
Effect	Effect	Effect	Error	Error	F	p-level
1	1	34.945	9	57.96476	.60287	.457403
2	2*	1234.559*	18*	63.99558*	19.29132*	.000033*
12	2	67.776	18	37.84732	1.79076	.195305

AEMG to M-wave RATIO COMPARISON Exp 1 vs Exp 2

Summary of all Effects; Exp 1 vs Exp 2 MWE

1-EXP, 2-TIME

	df	MS	df	MS		
Effect	Effect	Effect	Error	Error	F	p-level
1	1	.000028	9	.000259	.110015	.747720
2	2	.000064	18	.000019	3.361851	.057469
12	2	.000006	18	.000011	.516011	.605464

PASSIVE TENSION COMPARISON Exp 1 vs Exp 2

Summary of all Effects; Passive Tension

1-EXP, 2-TIME, 3-ANGLE

Effect	df	MS	df	MS	F	p-level
Effect	Effect	Effect	Error	Error		
1	1	2.196	9	8.37243	.26235	.620844
2	5*	66.231*	45*	3.08741*	21.45208*	.000000*
3	2*	4711.001*	18*	66.16523*	71.20055*	.000000*
12	5*	4.352*	45*	1.11868*	3.89008*	.005145*
13	2	4.208	18	2.24288	1.87632	.181905
23	10*	20.821*	90*	1.14851*	18.12916*	.000000*
123	10*	1.268*	90*	.48397*	2.61989*	.007643*