

**EFFECT OF POSTACTIVATION POTENTIATION ON
ISOTONIC KNEE EXTENSION PERFORMANCE**

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ISOTONIC KNEE EXTENSION PERFORMANCE**

By

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I dedicate this thesis to my father and to my mother...every step was made easier because of you...every mountain made a little smaller.

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Chapter 1: Force Potentiation of Skeletal Muscle

1.0 Introduction

The current review examines theories underlying force potentiation in skeletal muscle. Although phosphorylation of the myosin regulatory light chains is the best cellular correlate to isometric twitch potentiation, there still remains experimental evidence suggesting that the phosphorylation process is not obligatory to tension potentiation. Contractile characteristics associated with staircase, posttetanic and post-activation potentiation, under both isometric and dynamic conditions, will be used to introduce hypothesized mechanisms underlying intracellular skeletal muscle force augmentation and the physiological significance of these processes. Through further examination of potentiation through the context of muscle fatigue and temperature, the applicability of animal models to humans will be shown to be equivocal. Future direction for research will consequently be suggested.

1.1 Force Potentiation Defined

Skeletal muscle utilizes a variety of mechanisms to modulate contractile force throughout a variety of movement conditions. These include recruitment of additional motor units as well as a diversity of motor unit rate coding characteristics, depending on the demands of the task or environment (for reviews see Henneman and Mendell, 1981; Heckman and Sandercock, 1996). An intracellular skeletal muscle mechanism that increases force has also been reported. The degree of this type of force potentiation is dependent on prior muscle activity and consequently has been conceptualized as a form of 'muscle memory' (Levine et al., 1996). There are several ways of inducing this type of potentiation, and each form is classified in terms of the type of stimulus used to elicit the force augmentation.

The staircase or treppe effect is associated with successive increases in isometric twitch force during low or subfusion stimulation frequencies (MacIntosh et al., 1993; MacIntosh and Kupsh, 1987; Rankin et al., 1988). A second form of skeletal muscle twitch potentiation observed at low frequencies of stimulation can be induced by an added pulse approximately 10 ms after a preceding pulse, and is known as doublet potentiation (Sandercock and Heckman, 1997). A third form refers to the resulting augmentation in peak isometric twitch tension (P_t) after a brief high frequency tetanic stimulation (Close and Hoh, 1968a) known as posttetanic potentiation (PTP); a similar form of potentiation in humans can be elicited through a maximal voluntary

contraction (Vandervoort et al., 1983) and is commonly known as post activation potentiation (PAP).

Although the aforementioned forms of potentiation are central to current research, one other form of potentiation worth mentioning has not received a great deal of attention but is relevant to the field. Cold potentiation, referring to an increase in P_1 with decreasing muscle temperatures, has been observed in fast rat and mouse skeletal muscle in vitro (Close and Hoh, 1968b; Manning and Stull, 1982; Moore et al., 1990), providing yet another environmental context through which the mechanisms and effects of PTP can be studied.

This literature review will centre on current theories of skeletal muscle posttetanic and postactivation potentiation. It will begin by examining contractile properties of isometric twitches and tetany during force potentiation. Subsequent evaluation of a variety of independent measures such as movement type, stimulation frequencies and durations, muscle temperature and fibre type, in addition to addressing the applicability of animal models to humans, will be utilized to examine current postulated physiological mechanisms of twitch potentiation. This approach will illustrate the need for further studies examining intracellular mechanisms of force augmentation in human mixed skeletal muscle.

1.2 Contractile Properties of Potentiated Skeletal Muscle

1.2.1 Isometric Properties

Force potentiation of isometric twitches following a conditioning stimulus are associated with an increased P_t , which decays exponentially towards resting levels (Close and Hoh, 1968a). Observed reductions in isometric twitch durations during potentiation are a result of both decreased times to isometric peak twitch tension (TPT) and a half relaxation times (HRT) (Close and Hoh, 1968a; MacIntosh and Gardiner, 1987; O'Leary et al., 1997; Vandenoorn et al., 1995; Vandervoort et al., 1983). In addition, potentiation effects have been shown to last from 3 to 10 minutes after cessation of the conditioning stimulus (Kotsias et al., 1984; O'Leary et al., 1997) or maximal voluntary contraction (Grange and Houston, 1991; Houston and Grange, 1990) and has displayed trends of extended duration with high initial force potentiation (Close and Hoh, 1968a; O'Leary et al., 1997).

The aforementioned contractile properties of potentiated skeletal muscle have not been consistent across all studies, however. Prolonged twitch contraction times following a conditioning tetanus of 50 Hz has been observed in human (Takamori et al., 1971) and frog skeletal (Kotsias et al., 1984) muscle. Kotsias and his colleagues (1984) attributed the concurrent increase in P_t and TPT to a prolonged duration of the muscle action potential resulting from a reduced maximum rate of repolarization. Some changes in the muscle action potential after a repetitive stimulation with a similar time course to that for PTP have been previously suggested (Close and Hoh, 1968a) but not established

as causal to twitch potentiation. Indeed, changes in muscle action potential (MAP) amplitude and duration after a conditioning stimulus have been observed in humans (Cupido et al., 1996); the time course of MAP potentiation, however, is different from that of PTP and has been associated with increased Na^+/K^+ pump activity (Hicks and McComas, 1989) and is not considered a viable mechanism. Prolonged and often profound action potential alterations are observed in fatigued muscle (Bellemare and Garzaniti, 1988; Cupido et al., 1996; Hicks and McComas, 1989; Stephens and Taylor, 1972). A more in-depth investigation into mechanisms of twitch potentiation and its interaction with fatigue will be addressed later in this review.

A more physiologically relevant scheme for examining potentiated skeletal muscle comes from analyzing the changes in tetanic contractile characteristics after a conditioning tetanus. Although most studies have centred on isometric twitch characteristics, select studies examining changes in skeletal muscle tetanic contractile characteristics during periods of PTP have revealed interesting and paradoxical behaviors. Vandenoorn and his colleagues (1995) hypothesized that mechanisms responsible for PTP should be manifested through increases in rates of force development and P_t . The reasoning stemmed from models of muscle contraction that incorporated the concept of weak and strong cross-bridge binding to actin. Weakly bound cross-bridges represent a non-force generating state and strongly bound cross-bridges represent a force-generating state. A currently adopted scheme utilizes the assumption that the rate constant of force development is represented through isometric cross-bridge turnover kinetics expressed through two rate constants f_{app} and g_{app} (Brenner, 1988), where f_{app}

represents the transition from a non-force generating state to a force generating state and g_{app} represents the reverse process. The rate constant of isometric steady state force (K_{redev}) was consequently expressed as the sum of these two states (i.e., $K_{redev} = f_{app} + g_{app}$). In terms of physiological mechanisms, it was suggested that intracellular calcium concentrations affected f_{app} but not g_{app} (Brenner, 1988), denoting a Ca^{2+} controlled basis for increasing f_{app} values. This notion has been modified by the hypothesis that Ca^{2+} dependent mechanisms responsible for PTP serve to augment f_{app} above that which would normally be expected for a given concentration of intracellular calcium while leaving g_{app} unaffected (Sweeney and Stull, 1990; Vandenoorn et al., 1995). This would be analogous to an increase in Ca^{2+} sensitivity within the muscle fibre and a subsequent increase in isometric force and rate of force development due to the enhanced rate at which cross-bridges enter force-producing states from non-force-producing states (Sweeney and Stull, 1990).

When applied to tetanic contractions, Vandenoorn et al. (1995) found that despite decreases in peak tetanic torque (P_o), the maximal rate of tension development (dF/dt_{max}) was always greater during periods of PTP. Such observations of increased rates of tension development during isometric muscle actions have been shown to be consistent across most studies (Vandenoorn et al., 1993; Vandenoorn et al., 1997; for reviews see Grange et al., 1993; Sweeney et al., 1993). Under circumstances where potentiated P_t was found to be accompanied by a prolonged twitch duration, the prolongation was attributable solely to an increased HRT time (Takamori et al., 1971), a characteristic property of muscle fatigue (Rankin et al., 1988).

Further still, Vandenboom et al. (1993) illustrated the consistency of the enhanced rate of force development property of PTP. Through employing an in vitro model, they studied contractile properties of the mouse EDL muscle at several frequencies following a 5 Hz, 20-second conditioning stimulus. Interestingly, test frequencies ranging from 1-15 Hz displayed a strong positive dF/dt_{\max} versus peak isometric force potentiation correlation; this relationship broke down, however, at frequencies above 15 Hz (20-150 Hz) due to a continued potentiation of dF/dt_{\max} and a diminution of peak force. This behavior paralleled that of prior studies that found the existence of an upper threshold of activation above which force potentiation could not be observed (Grange and Houston, 1991). The resultant force-frequency curve consequently showed an initial leftward shift at values below 20 Hz and a rightward shift at higher frequencies. Thus, a more consistent qualifier of PTP would appear to be the rate of force development and not isometric peak torque. Such a notion has recently been supported across both fatiguing and non-fatiguing conditioning stimulation protocols and will be further explained subsequently (Vandenboom et al., 1997).

1.2.2 Concentric and Eccentric Properties

When examining the effects of skeletal muscle potentiation under dynamic conditions, factors associated with movement direction have been shown to affect the degree of PTP observed. Twitch potentiation has been shown to be dependent on muscle length with a more profound augmentation of force at optimal filament overlap with little

or no effect at long sarcomere lengths (Yang et al., 1992). Human studies examining PAP have displayed similar trends, where P_t potentiation was shown to be greater at shorter muscle lengths for the knee extensors (Stuart et al., 1988) and dorsiflexors (Vandervoort et al., 1983).

One measure of concentric movement that is of particular interest when studying the effects of potentiation is that of maximum velocity of shortening or V_{max} . Past studies examining V_{max} during periods of skeletal muscle potentiation saw marked increases in mouse fast-twitch fibres with a corresponding down-regulation of actomyosin ATPase activity (Crow and Kushmerick, 1982a; 1982b), suggesting a decreased energy cost of contraction. These findings were not supported in subsequent animal studies, however (Butler et al., 1983; Persechini et al., 1985) stating that the phosphorylation process, thought to be a possible mechanism for PTP, had no effect on cross-bridge cycling rates and consequently no effect on the force-velocity relationship. In terms of human studies, maximum unloaded shortening velocities did show marginal (5%) increases 2-seconds after a 10-second MVC in comparison to resting values, but were found to be non-significant (Stuart et al., 1988).

Unfortunately, there are few studies examining PTP during shortening and lengthening muscle actions. Recently however, Sandercock and Heckman (1997) examined doublet potentiation during eccentric and concentric muscle actions of the cat soleus muscle *in situ*. Under isometric conditions, they found similar length dependencies of potentiation as previously mentioned, related to an enhanced doublet potentiation with a significant trend of increased sustained potentiation at shorter muscle

lengths. However, it was difficult to separate the effects of velocity of movement from length effects during dynamic trials. In general, it was shown that ramp decreases in muscle length decreased the absolute force produced by a doublet; ramp increases raised the absolute force of the doublet early in the ramp but later decreased the relative degree of potentiation at longer muscle lengths. The applicability of this study to PTP is limited due to the uncertainty of similar mechanisms underlying the two phenomena of doublet and posttetanic potentiation.

One study, examining contractile characteristics of phosphorylated mouse EDL muscle *in vitro*, measured evoked work (i.e., single twitch) during eccentric (lengthening) and concentric (shortening) work cycles (Grange et al., 1993). They found that concentric work was increased by 44% in comparison to the non-phosphorylated condition; corresponding twitch potentiation was 15-20%. On the other hand eccentric work, although significantly greater in absolute magnitude in comparison to concentric work, was only potentiated by 20%, and with correspondingly less twitch potentiation. On examination of the data, work potentiation was greater at shorter muscle lengths as well, displaying similar behavior seen in doublet potentiation previously described. Thus it would appear that mechanisms responsible for both doublet and posttetanic potentiation have dependencies on lengthening and shortening contractions.

1.3 Mechanisms Associated with Staircase, PTP and PAP

1.3.1 Sub-Units of the Myosin Heavy Chain

To understand current theories of skeletal muscle potentiation, it is necessary to have knowledge of the sub-units of the myosin molecule. A single myosin molecule is comprised of a heavy meromyosin (HMM) and a longer light meromyosin (LMM) component, the intersection of which form a hinge region (McComas, 1996). The HMM component is further divided into two S1 fragments, each containing one globular myosin head, and one S2 fragment which forms the attachment to the LMM chain. The myosin head is further subdivided into components classified by molecular weight. The portion of the head which binds to actin is the 50 kilodalton (kDa) subunit situated distal to an intermediate 25 kDa subunit. Finally, there is a class of 18-20 kDa light chains situated on the neck region (intersection of S1 and S2 fragments) of the myosin head that is central to current studies examining staircase, PTP or PAP.

1.3.2 Phosphorylation of the Myosin Regulatory Light Chains

Nearly three decades ago it was discovered that the 18.5 kDa light chain subunit in rabbit skeletal muscle, also known as the LC₂ subunit, could be phosphorylated (Perrie et al., 1973). Subsequently, phosphorylation of these LC₂ subunits or myosin regulatory light chains (R-LC) was found to have a close temporal correlation to PTP (Manning and Stull, 1982); staircase potentiation was also observed to be correlated with LC₂ phosphate content (Klug et al., 1982). Further still, Sweeney and Stull (1990) linked the effect of the phosphorylation process to cross-bridge kinetics by stating that R-LC phosphorylation

manifests its effect on isometric force and rate of force development by increasing f_{app} for a given level of calcium. In other words, R-LC phosphorylation resulted in more cross-bridges forming for a given level of intracellular calcium, which is analogous to an enhanced sensitivity to calcium as previously mentioned.

A phosphorylation-dependent increase in rate of tension development observed in fast rabbit (Metzger et al., 1989) and mouse (Vandenboom et al., 1995) skeletal muscle illustrated a possible physiologically relevant effect of myosin R-LC phosphorylation. The physiological relevance of PTP has more recently been proposed in terms of aiding in P_i recovery from fatigue (Fuglevand et al., 1993; Houston and Grange, 1990) and maintenance of force in light of decreased motor unit discharge frequencies (DeLuca et al., 1996).

Until recently, studies have relied on examination of the contractile properties of skeletal muscle to characterize the effects of R-LC phosphorylation. Current theories as to the effects of the phosphorylation process suggest that the added phosphate to the R-LC serves to generate a charge induced conformational change in the myosin head bringing it closer to the actin binding site (Grange et al., 1993; Sweeney et al., 1993). With advances in electron microscopy however, the effects of R-LC phosphorylation on the myosin structure have been better qualified. Levine et al. (1996) found that phosphorylation of the myosin R-LC isolated from rabbit psoas muscle resulted in an extension of the myosin heads away from the filament backbone, with a net result of “increasing disorder” of the myosin heads, independent of increased calcium levels. It was hypothesized that this increased myosin-head disorder served to increase motility,

ensuring that each head spent more time in the vicinity of the actin binding site than it would in the unphosphorylated state. Consequently, with lower levels of calcium, more actin-myosin interactions would occur and tension potentiation would result. The effect of the phosphorylation process should consequently result in a leftward shift of the pCa (i.e., $-\log[\text{Ca}^{2+}]$) vs. tension relationship; this has been shown to be the case in earlier studies where the greatest shift occurs at the midpoint of the curve (Palmer and Moore, 1989; Persechini et al., 1985), corresponding to a region associated with suboptimal levels of cytosolic Ca^{2+} for muscle contraction.

The process of phosphorylation is catalyzed by calcium dependent myosin light chain kinase (MLCK), and activated by a calcium regulatory protein calmodulin (Manning and Stull, 1982). In the presence of calcium these two components bind, resulting in the activation of MLCK which initiates the phosphorylation process and can be subsequently reversed by myosin light chain phosphatase (MLCP) (Levine et al., 1996; Manning and Stull, 1982; Moore et al., 1990; Sweeney et al., 1993). Studies linking the effects of the R-LC phosphorylation process to force potentiation have consistently suggested the phenomena of PTP or staircase to be a characteristic unique to fast-twitch fibres in animals (Close, 1972; Crow and Kushmerick, 1982a; MacIntosh and Kupsh, 1987; Manning and Stull, 1982; Moore and Stull, 1984; Tubman et al., 1996). Fast white muscles in rats have also been shown to have up to 3.5 times more MLCK activity in comparison to slow muscle (Moore and Stull, 1984). As will be subsequently discussed however, the phosphorylation process does not always result in force

potentiation, leaving the notion of R-LC phosphorylation being the sole mechanism for PTP open to debate.

1.3.3 Human Studies

As previously mentioned, animal studies have suggested that R-LC phosphorylation and the subsequent potentiation of isometric tension is a characteristic of type II muscle fibres. The most dramatic illustration of the selective phosphorylation was illustrated by Moore and Stull (1984), where they found a 2-second, 100 Hz stimulation resulted in R-LC phosphate levels jumping from approximately 0.10 mol phosphate/mol P-light chain to 0.70 mol phosphate/mol P-light chain in the mouse gastrocnemius muscle; corresponding twitch tension augmentation was on the order of 1.6 times greater than resting P_t values. The red soleus muscle, however, saw phosphate levels rise by only 77% (0.18 to 0.32 mol phosphate/mol P-light chain) after a 100 Hz, 15-second conditioning stimulus, with no corresponding P_t potentiation.

This is not to say that tension potentiation has never been observed in slow muscles in animals. Rankin and her colleagues (1988) compared the degree of staircase during a fatiguing protocol in EDL (4% SO, 54% FOG, 42% FG) to soleus (85% SO, 15% FOG) muscle of the rat. They found that with a 0.5 Hz stimulation protocol, the soleus muscle exhibited a mean maximal potentiation of 29% by the 13th to 15th twitch. Mean maximal EDL potentiation ($159 \pm 33\%$) occurred by the 23rd or 24th twitch. Contractile properties of the potentiated twitches across muscle groups differed as well. The resulting enhancement of twitch force in the soleus muscle was accompanied by

slight decreases in TPT and increases in HRT. Conversely, EDL potentiation was accompanied by reductions in both TPT and HRT. These results suggest that there may be different mechanisms for staircase potentiation across fibre types in animals.

The disparate findings of these animal studies are further confounded by human studies comparing R-LC phosphorylation of the fast (LC2F) and two slow (LC2S and LC2S¹) myosin regulatory light chains of the vastus lateralis (Houston et al., 1987; Stuart et al., 1988). Stuart et al. (1988) found no significant correlates between P_t potentiation and phosphate incorporation into individual R-LC or between percent type II fibre type distribution. In addition, although the degree of P_t potentiation had decreased one minute after the MVC, phosphate levels remained elevated.

Houston and his colleagues (1987) also found the fibre specific nature of the R-LC phosphorylation process equivocal. Following a 10-second MVC, they found that phosphate levels of the LC2F, LC2S and LC2S¹ chains had all been elevated to the same degree. As a result they suggested that MLCK and MLCP activity were similar in human fast and slow skeletal muscle, a notion which has subsequently been supported (Grange and Houston, 1991). Despite these discrepancies, PTP in human skeletal muscle has been shown to be more pronounced in muscles with shorter contraction and half relaxation times (O'Leary et al., 1997; Vandervoort et al., 1983). In addition, recent studies have also shown a strong correlation ($r = 0.81$, $p < 0.05$) between the degree of PAP and fibre type distribution in unfatigued human skeletal muscle (Hamada et al., 1998a,b). Consequently, the physiological significance of R-LC phosphorylation in human mixed muscle and the fibre type specific nature of PTP are still unclear.

1.4 Skeletal Muscle Fatigue and Potentiation

Although there is substantial evidence supporting the notion that phosphorylation of the myosin R-LC is sufficient to induce PTP, establishing a causal role has been somewhat problematic. The disputable nature of establishing an obligatory role of phosphorylation for PTP for both animal and human models is best illustrated through studies examining the interaction of PTP with fatigue.

To begin, two experimental findings seem to suggest that the process of R-LC phosphorylation may serve as a primary means of maintaining force under fatiguing conditions. There is experimental evidence suggesting that reduced availability of calcium release from the terminal cisternae of the sarcoplasmic reticulum (SR) may be the locus of excitation-contraction coupling failure during fatigue (MacIntosh and Kupsh, 1987), resulting in less Ca^{2+} being released per action potential. It has also been shown that the degree of potentiation induced as a result of R-LC phosphorylation is always greater at low intracellular calcium concentrations (Palmer and Moore, 1989; Persechini et al., 1985; Sweeney and Stull, 1990; Tubman et al., 1996). As a result, some authors have suggested that the decreases in Ca^{2+} observed during fatigue may be obligatory to permit potentiation (Grange and Houston, 1991).

Recent studies, however, have shown that potentiation brought about through a fatiguing conditioning stimulus does not have the same characteristics as that brought about by a non-fatiguing stimulus. Tubman and his colleagues (1996) observed equivalent tension potentiation values in the rat gastrocnemius muscle *in situ* under both fatiguing and non-fatiguing conditions, even though the fatigued muscles had

correspondingly lower phosphorylation levels of the myosin R-LC. Thus, phosphorylation could not be the only mechanism responsible for potentiation. Similar examples of the dissociation of P_t potentiation vs. R-LC phosphorylation linear relationship have been observed in the past (MacIntosh et al., 1993). On the other hand, other studies while not observing a decrease in R-LC phosphorylation with fatigue, still observed an uncoupling of the P_t potentiation – R-LC relationship through decreases in P_t with fatigue (Vandenboom and Houston, 1996).

Better contractile property correlates to R-LC phosphate content have been recently discovered, however. Vandenboom et al. (1997) observed a high linear correlation ($r = 0.97$, $p < 0.001$) between R-LC phosphate content and the relative rates of force development (dF/dt_{max}) potentiation across a wide range of stimulation frequencies (2.5 to 100Hz) for the mouse EDL muscle. This approach effectively demonstrated that increases in dF/dt_{max} could be uncoupled from the level of muscle fatigue, since alterations in $+ dF/dt_{max}$ consistently held its proportionality to the degree of myosin R-LC phosphorylation, whether or not the phosphorylation was elicited by a fatiguing, non-fatiguing, high or low frequency conditioning stimulus.

Alternatively, authors have suggested that PTP could be the result of increased Ca^{2+} release after a conditioning stimulus or the result of less Ca^{2+} released superimposed on an already high Ca^{2+} level (Close, 1972; Rankin et al., 1988; Takamori et al., 1971). This view has been refuted through studies involving contractile properties of skeletal muscle infused with caffeine, which increases SR Ca^{2+} release (MacIntosh and Gardiner, 1987; Vandenboom and Houston, 1996). Direct comparison of the effect of a

conditioning stimulus versus caffeine on P_t potentiation has shown caffeine to increase the duration of the twitch through an increased TPT and HRT, which is not consistent with contractile properties of a PTP twitch (MacIntosh and Gardiner, 1987; Vandenoorn and Houston, 1996). Second, a conditioning stimulus was shown to phosphorylate myosin R-LC while caffeine did not (Vandenoorn and Houston, 1996). Increased SR Ca^{2+} release or elevated Ca^{2+} levels after a conditioning stimulus, therefore, appear incompatible with the contractile properties associated with PTP. Again, these arguments are equivocal; revisiting the results of Rankin et al. (1988) at this point does illustrate that increased Ca^{2+} release as a result of prior muscle activity is not altogether unfounded during a fatiguing stimulation protocol. Although not directly measuring Ca^{2+} release, Rankin et al. (1988) found the potentiation observed with the rat soleus muscle was accompanied by slight increases in HRT, which is compatible with enhanced Ca^{2+} release.

Green and Jones (1989) examined differences in contractile characteristics of potentiated P_t between fatigued and non-fatigued quadriceps muscle and also produced similar potentiation effects as seen with caffeine. After a 10-second MVC, potentiated twitches displayed characteristic decreases in TPT and HRT. After a series of fatiguing contractions however, potentiated twitches failed to significantly decrease TPT or HRT regardless of recovery duration, with both values remaining similar to pre-fatigue values. The authors consequently questioned the viability of phosphorylation as the primary mechanism for force potentiation in fatigued muscle, and that the relationship may be circumstantial. A mechanism which better suited the contractile characteristics of the

potentiated twitches during fatigue consisted of either a fixed Ca^{2+} release by the SR superimposed on a higher cytosolic Ca^{2+} concentration or an enhanced rate of Ca^{2+} release from the SR.

Thus, it appears that twitch potentiation mechanisms need to be studied within the context of stimulus duration and frequency, or contraction duration and intensity, and in terms of fibre type as well, due to evidence suggesting similar MLCK and MLCP activity in slow and fast human skeletal muscle.

1.5 Skeletal Muscle Temperature and Potentiation

A final area of study that has not received too much attention is the effect of skeletal muscle temperature on PTP. Although the studies to date have centred on animals, this area merits attention due to the similar behavior of current data involving humans (Gossen et al., unpublished observations).

Moore et al. (1990) measured contractile properties of mouse EDL muscles at 25, 30 and 35°C and noted four primary temperature dependent changes in potentiated twitches. First, the amount of relative twitch potentiation immediately following the conditioning stimulus was directly proportional to muscle incubation temperatures, which was consistent with earlier studies (Close and Hoh, 1968b; Manning and Stull, 1982). Second, while the 35°C condition exhibited maximal potentiation immediately following the conditioning stimulus, the 25 and 30°C conditions showed maximal potentiation 10-20 seconds after stimulus cessation. Third, the rate of decline of potentiated twitch force

was directly proportional to muscle incubation temperature. Finally, the magnitude of R-LC phosphorylation was inversely proportional to muscle incubation temperature.

The cold potentiation observed in the unpotentiated resting twitches was attributed to a prolonged active state; a decline in sensitivity of the contractile element to Ca^{2+} with increasing muscle temperature on the other hand, resulted in a depressed unpotentiated twitch force. Moore et al. (1990) suggested that relative decreases in P_i potentiation would be expected with decreasing temperatures if one were to consider the notion of an enhanced effect of R-LC phosphorylation on submaximal tension generation under conditions of decreased activation of the contractile element by Ca^{2+} (Grange and Houston, 1991; Palmer and Moore, 1989).

The quantitative differences of PTP across muscle temperature can not only be attributed to mechanisms underlying E-C coupling (SR influx/efflux of Ca^{2+}), but also on the effects of temperature on the MLCK reaction (Manning and Stull, 1982). MLCK has a Q_{10} value of approximately 2.0 (Moore et al., 1990) and may be more of a factor in human studies examining temperature and PTP. Both an increase in the time course and amplitude of the Ca^{2+} transient during a twitch and a delayed inactivation of MLCK at lower muscle temperatures favored a greater fractional activation of MLCK. This increase in fractional activation was hypothesized to be sufficient to overcome any decreases in maximal catalytic rate of active MLCK at lower temperatures ($Q_{10} \sim 2.0$) resulting in a net increased rate of reaction catalysis (maximal catalytic rate x fractional activation).

On examination of the human dorsiflexor muscles across water bath temperatures of 10 and 45°C, Gossen et al. (1998) saw similar relative potentiation characteristics as those observed by Moore and his colleagues (1990), but with no changes in the twitch-tetanus ratio across bath conditions. In addition, Gossen et al. observed no cold potentiation at lower bath temperatures, unlike the inverse relation observed between P_t and muscle temperature observed *in vitro* in fast animal muscle (Close and Hoh, 1968b; Moore et al., 1990). The response of the human dorsiflexors to cooling was as expected for predominantly slow (type I) fibres, characterized by depressed P_t and prolongation of TPT and HRT (Davies et al., 1982; Ranatunga et al., 1987). In fast muscles, the increase in the ratio of potentiated (P_t^*) to unpotentiated twitch force (P_t) with increasing temperature could be attributed to a decline of P_t at higher temperatures with a comparatively small change in P_t^* (Buller et al., 1984; Close and Hoh, 1968a; Moore et al., 1990). In contrast, the human dorsiflexors exhibited an enhanced P_t at higher temperatures in addition to a greater P_t^* resulting in an increased P_t^*/P_t ratio (Gossen et al., 1998). Thus, a predominantly slow muscle has the same tendency as fast muscle to increase P_t^*/P_t with increased temperature, but the increase is a result of opposite effects of temperature on P_t and P_t^* .

The thermal dependence of peak force during tetanus (P_o) has been shown to be less dependent on fibre type than P_t , particularly at higher temperatures (Bennett, 1984). At lower temperatures a consistent finding is a decreased P_o (Bennett, 1984; Close and Hoh, 1968b). There have been some discrepancies in the literature, however, due to both linear decreases (Close and Hoh, 1968b) and no changes (Moore et al., 1990) in force

output exhibited for mouse EDL muscle between 25-35°C. With similar P_i characteristics observed across temperatures, this would result in different twitch tetanus ratios and different conclusions regarding Ca^{2+} sensitivity across temperature. There is also the issue of different temperature dependencies of skeletal muscle between species. Different plateaus of P_o across a range of temperatures have been associated with experimental temperatures proximal to a physiologically relevant functional ranges that are species specific (Rall and Woledge, 1990). Gossen et al. (1998) observed a 30% decrease in P_o between 10 and 45°C water bath temperatures, despite M-wave data ruling out nerve block. These observations are similar to decreases found in P_o in the first dorsal interosseus subjected to reductions in muscle temperature from 30 to 15°C, which were associated with decreases in the force output per crossbridge with at lower temperatures (Rall and Woledge, 1990). Thus, the changes in twitch-tetanus ratios observed in animal, and not human studies, may due to the different force-temperature dependencies across species, and the corresponding location of the physiologically relevant range over which P_o plateaus; this possibility illustrates the need to study the temperature dependency of PTP at skeletal muscle temperatures relative to physiological relevant or optimal conditions, and not simply across absolute temperature ranges. This approach may better serve to isolate the relative temperature dependencies of MLCK, MLCP and calcium sensitivity, yielding a better model illustrating the thermal dependence of components responsible for PTP.

1.6 Conclusion

Although the phosphorylation process has been repeatedly shown to occur in both animal (Klug et al., 1982; Perrie et al., 1973; Manning et al., 1982; Moore and Stull, 1984) and human (Green and Jones, 1989; Stuart et al., 1988) skeletal muscle, the physiological significance of the process and its relation to the phenomenon of PTP has yet to be established. With recent studies illustrating R-LC is associated with conformational changes of the myosin heads (Levine et al., 1996), it does become increasingly plausible that LC₂ phosphorylation in animals does result in an enhanced number of cross-bridges forming at suboptimal Ca²⁺ levels. Human studies examining PTP or PAP do exhibit similar dependencies as animals in terms of muscle length, movement type, potentiation duration and, in some respects, temperature. The similarities diverge, however, on examination of fibre-type specificity of R-LC phosphorylation; in addition, both human and animal models do not show a consistent P_t potentiation-phosphorylation relationship, while dF/dt_{max} has been shown to be a more reliable correlate.

As a result, future studies need to centre on more time-dependent measures of performance augmentation in potentiated skeletal muscle, and to establish the degree to which the phosphorylation process is responsible for these changes. These changes also need to be qualified in terms of fibre type, muscle temperature, movement condition and degree and type of fatigue. Although animal studies have illustrated possible functional

manifestations of R-LC phosphorylation, the unique characteristics of phosphorylation in human skeletal muscle and the corresponding physiological significance of these processes need to receive further scrutiny.

Chapter 2: Effect of Postactivation Potentiation on Isotonic Knee Extension Performance

2.0 Abstract

Maximal isotonic knee extension performance was studied during periods of twitch postactivation potentiation immediately following a 10-s maximal voluntary isometric contraction (MVC), and during control conditions. Six men and 4 women attended 4 sessions consisting of a habituation, 2 treatment and twitch control trials. Using a Biodex dynamometer, isotonic loads were randomized and set at 15%, 30%, 45% and 60% of MVC peak torque attained during habituation. Each treatment trial consisted of a unilateral knee extension protocol. Two of the four loads were tested per treatment session. After an evoked baseline twitch had been established on a leg (via the femoral nerve), one of either a control (CON) or potentiated (PAP) protocol was performed. CON consisted of an initial twitch, a maximal isotonic knee extension (kick #1), a second twitch, a second knee extension at the same load (kick #2), and a final twitch. After a 15 min. rest, the PAP trial was performed. PAP was identical to CON except following the resting twitch, a 10-s MVC was used to elicit potentiation. Each protocol lasted 60-s. The twitch control trial was identical to the PAP trial except that twitches were substituted for isotonic knee extensions. After kick #2 in CON, twitches showed significant potentiation above baseline ($29.4 \pm 2.1\%$, $p < 0.0001$). During PAP, all twitches after the MVC were potentiated to a greater extent than in CON trial ($p < 0.001$). Despite significant twitch potentiation during PAP, there was no change in the velocity attained with isotonic loads. Peak power exhibited a condition main effect due to reductions in peak power from CON to PAP (CON, 462.2 ± 16.2 W; PAP, 435.1 ± 14.1 W, $p < 0.05$) and a kick order effect due to an increase from kick #1 to kick #2 (kick #1, 441.3 ± 15.2 W; kick #2, 456.0 ± 15.3 W, $p < 0.03$). Similar reductions were found for muscle activation at peak power (CON, $8.0 \pm 0.3\%$; PAP, $7.5 \pm 0.3\%$, $p < 0.02$). No corresponding changes in movement times or work attained up to the point of peak power were evident, however. Although increases in knee extension performance were not observed during periods of maximal twitch potentiation, further analysis revealed increases in work to peak power during CON and maintenance of work during PAP, despite reductions in peak power, may reflect a compensatory effect of potentiation during movement. Thus potentiation of work may better reflect effects of PAP during volitional movements than does the load-velocity relationship.

2.1 Introduction

The force of a muscle twitch contraction is increased after a conditioning tetanus (posttetanic potentiation or PTP), maximal voluntary contraction (postactivation potentiation or PAP) or repeated sub-tetanic stimuli (staircase or treppe). For simplicity all the aforementioned forms of potentiation will be referred to as PAP. PAP is likely the result of phosphorylation of the myosin regulatory light chains via myosin light chain kinase (Sweeney et al., 1993) which theoretically increases the interaction between the myosin cross-bridge and the thin filament (Persechini et al., 1985). Under isometric conditions, PAP transiently increases twitch peak torque, rate of torque development and decreases time to peak torque of the twitch (Grange et al., 1993, O'Leary et al., 1997, Sweeney et al., 1993). Other studies examining the effect of PAP across various stimulation frequencies in unfatigued muscle found both peak force and rate of force development to be increased at low frequencies (≤ 15 Hz) while only rate of force development to be enhanced at higher stimulation frequencies (Vandenboom et al., 1993).

The majority of the current literature characterizing the effects of PAP has used either isometric twitch models or a range of stimulation frequencies; few have examined the interaction between PAP and maximal voluntary dynamic actions in intact human muscle. Studies that have examined the effects of PAP on maximal unloaded velocity of shortening (V_{\max}) during knee extension found no significant effects (Stuart et al., 1988), in agreement with observations in small mammals (Butler et al., 1983; Persechini et al.,

1985). Combining these findings with past isometric data suggests a negligible effect of PAP at the end-points of the load-velocity curve (i.e., isometric maximum force (P_0) and V_{max} , Fig. 1). Literature examining the effects of PAP at intermediate points on the load-velocity is scarce. Utilizing low frequency isotonic twitch contractions, Grange et al. (1995) found potentiation of both net displacement and shortening velocity (coincident with increases in R-LC phosphate content). Potentiation of displacement was more pronounced at higher loads (20-82% potentiation from 0- 75% of peak isometric twitch force, respectively) and was accompanied by increases in shortening velocities independent of muscle elastic elements. Using the work cycle technique with mouse fast muscle, Grange et al. (1998) found both concentric work and mean power could be potentiated as much as 50% across muscle displacement lengths ranging from 5-13% L_0 . Further still, a prior study using similar muscle preparations found concentric work was potentiated to a greater extent than eccentric work during periods of myosin light chain phosphorylation (44% CON; 20% ECC), reflecting a direction of contraction dependency of PAP (Grange et al., 1993).

These *in vitro* animal paradigms, however, quantified the effects of PAP (and myosin regulatory light-chain phosphorylation) using modified evoked twitch methods that are difficult to generalize to voluntary human movement, particularly to maximal contractions in which motor unit firing rates would be stimulating muscle fibres at frequencies above those at which potentiation of isometric force occurs. Nevertheless, Güllich and Schmidtbleicher (1995) have shown improvements in jumping and loaded arm pushing performance following a brief maximal voluntary isometric contraction

(MVC). The "loads" in these tasks are between the V_{\max} and P_o extremes in Figure 1. It was suggested that improvements were the result of enzymatic and/or neuromuscular activation alterations. PAP was briefly mentioned as a possible performance enhancement mechanism through augmentation of cross-bridge cycling rates, which has been shown not to be a cause of potentiation (Butler et al., 1983; Persechini et al., 1985). More recently, Young et al. (1998) supported Güllich and Schmidtbleicher's findings by showing increases in jump performance after heavy squats. Performance increases in both cases were small, however (2-3%). Beyond this, little is known of the effects of PAP on dynamic torque production in humans; more specifically, the way in which PAP affects maximal voluntary efforts across a series of submaximal loads.

The purpose of this study will be to examine the effects of postactivation potentiation on various performance measures of the knee extensor muscles. More specifically, changes in the load-velocity relationship, power, work and activation following PAP will be investigated. It is hypothesized that if PAP can increase rate of force development of high frequency tetanic contractions (Vandenboom et al., 1995; 1993), it could increase the acceleration and thus the velocity attained with loads between the extremes of the load-velocity relation, thereby reducing the curvature but not changing the end-points of the load-velocity relationship (Fig. 1). Any enhancements in velocity would also translate into increased peak power and work.

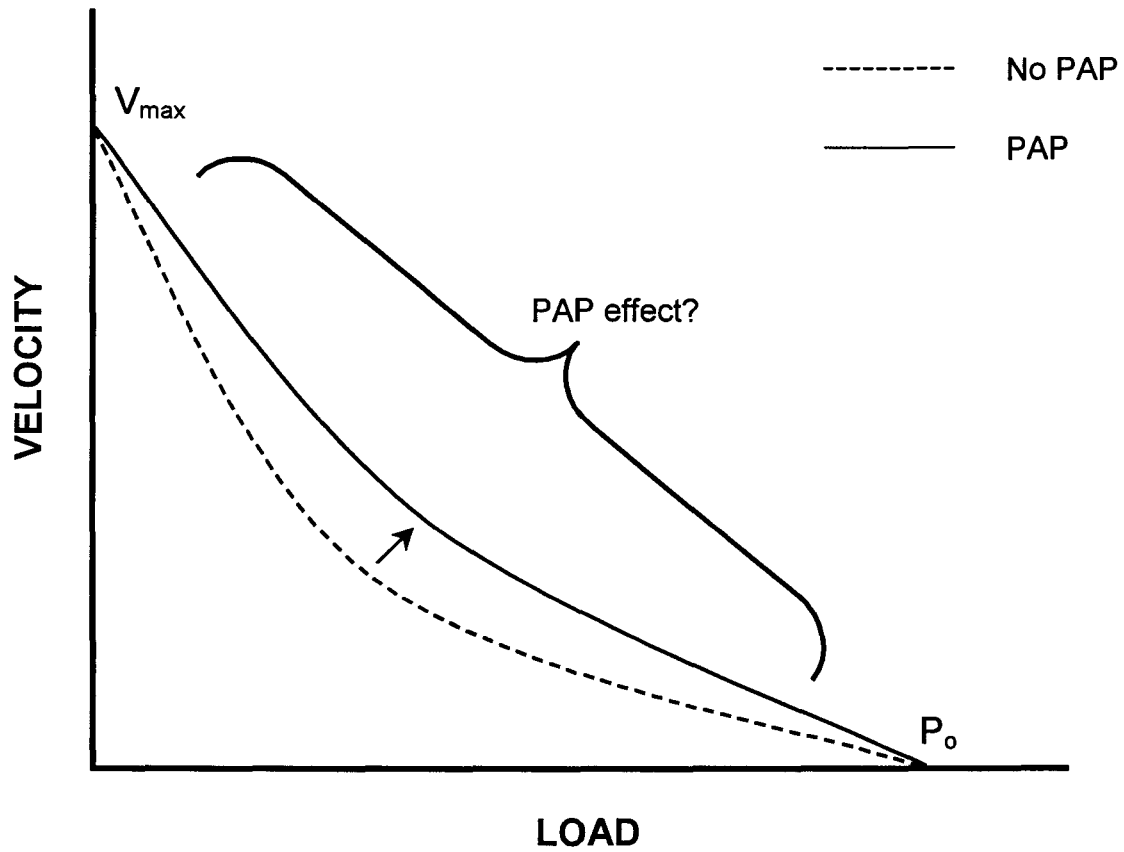


Figure 1: Hypothesized effect of PAP on the load-velocity relation. The load-velocity relation before (solid line) and after (dashed line) is shown. PAP reduces the curvature of, but does not alter the end points (maximum unloaded shortening velocity, V_{max} ; isometric maximum torque, P_o) of the load-velocity relation.

2.2 Methods

2.2.1 Subjects

Ten moderately active subjects (6 male, 4 female) between 22 and 35 years of age volunteered for the study after giving informed, written consent. To be eligible for participation, all subjects had to be free of lower leg injury. The study carried the approval of McMaster University's Human Ethics Committee.

2.2.2 Design

Postactivation potentiation was studied in the human knee extensors, comprised of the vastus lateralis, vastus medialis, vastus intermedius and rectus femoris muscles (Snell, 1995). The experimental protocol is illustrated in Figure 2. In general, a protocol was designed such that PAP could be quantified through isometric twitches. As a result, an alternating series of 4 evoked twitches (baseline plus 3 trial twitches) and 2 isotonic knee extensions (i.e., kicks) were performed to monitor the degree of PAP during each trial. The trials were repeated across two conditions: a control trial (CON) and potentiation trial (PAP); the protocol for each was identical except that in the PAP trial the initial twitch was followed by a 10-s maximal isometric knee extension (MVC). Each subject was required to repeat the experimental protocol across four isotonic loads (15%, 30%, 45% and 60% of maximal isometric torque). Consequently, this study was a 3 factor repeated measures design (4 load, 2 conditions [CON/PAP], 2 kicks).

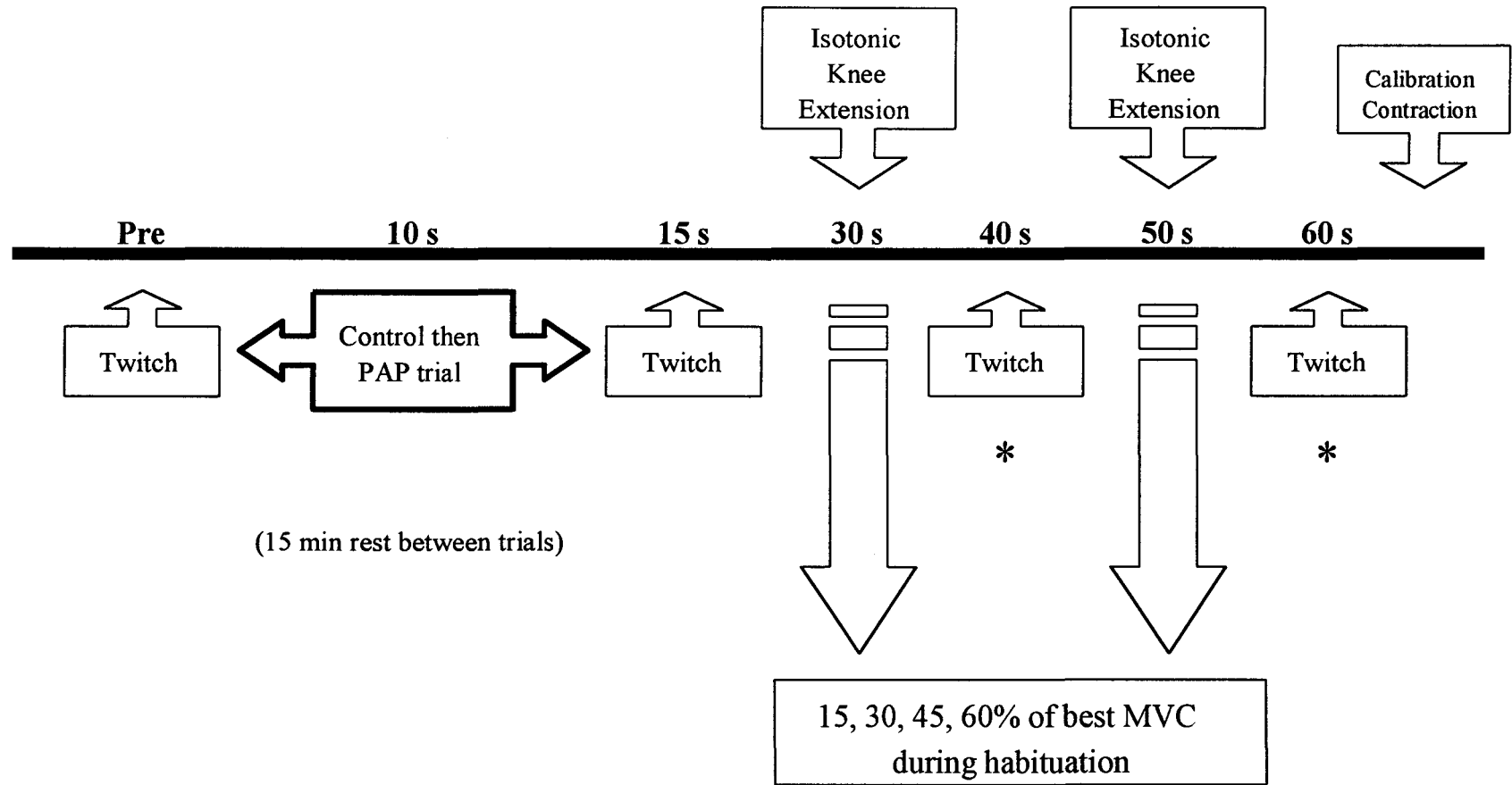


Figure 2: A schematic of the control and treatment protocol. Each test session will consist of an initial control trial followed by a PAP trial. Twitches marked by ‘*’ will determine the degree to which PAP is affected by the isotonic extensions.

2.2.3 Experimental Apparatus

Isotonic loads were preset using a Biodex System 3 dynamometer (Biodex Medical Systems, Shirley, New York). Prior to being seated, EMG electrodes (Meditrace Ag/AgCl Pellet ECG) were affixed to the knee flexors of both legs proximal to the belly of the hamstrings with a 2 cm centre-to-centre spacing to measure the degree of co-activation. Prior to electrode attachment, skin surfaces were shaven, abraded and swabbed with alcohol pads. Once leads were attached, the electrodes were then bordered by dense foam (~ 1.5 cm thick) and secured with a tensor to minimize movement artifact during knee extension. In addition, the electrode positioning on the knee flexors was carried out to minimize movement artifact during knee extension; this was accomplished by positioning the electrodes proximal to the belly of the hamstring muscles. Following placement of knee flexor EMG electrodes, the remaining preparation was done while the subjects were seated in the dynamometer. Once the subject was seated properly, the electrodes for the knee extensors were positioned. To monitor agonist EMG activity, electrodes (Meditrace Ag/AgCl ECG) were used in a monopolar configuration. The active electrode was placed on the vastus medialis approximately 8 cm proximal from the knee. The reference electrode was positioned on the patellar ligament immediately inferior to the apex of the patella while the ground was placed over the tibia midway between the knee and ankle joints. The thigh was then secured using leg restraints and the ankle was secured to the leg armature by a velcro strap. The seat position was

adjusted so that the knee's axis of rotation (tibio-femoral joint) was aligned with the dynamometer's armature axis of rotation with the knee at 90° (visually estimated).

Electrical stimulation electrodes were then positioned on the first leg to be tested. The anode was constructed out of a 6 cm x 4 cm lead plate that was coated with electrode gel (Spectra 300) and placed on the proximal quarter of the lateral thigh using surgical tape (Blenderm). The cathode (1.5 x 1.5 cm lead plate with 1.5 cm thick dense foam fastened to one side) was then initially positioned immediately medial to the tendon of the sartorius in the inguinal fold by the subject; subjects were instructed to apply minimal pressure so as to mimic the pressure that tape would exert on the electrode. A 0.25 ms square wave pulse from a Grass S11 stimulator (Grass Instruments, Quincy, Mass.) was used to evoke a minimal twitch response. All twitches were evoked with the dynamometer in the off mode to minimize signal noise. The subject was then instructed to move the cathode medially down the inguinal fold by approximately 1 cm. Using the same minimal stimulation intensity, a subsequent twitch was evoked. This procedure was repeated until the electrode was positioned directly over the femoral nerve, which was reflected by the largest twitch response. The cathode was then taped into place and a velcro belt secured over the subject's waist to minimize movement in the dynamometer seat. The stimulus intensity was then increased until a maximal twitch response was evoked.

All isometric and isotonic analogue signals were fed directly from the dynamometer head into a 12 bit A/D converter, then into a computer sampling at 2000 Hz per channel using CODAS software (DATAQ electronics). Measures included torque

and armature angle as well as agonist and antagonist EMG. EMG was collected using a bandwidth ranging from 4 to 2000 Hz (Grass P5 series amplifier, Grass Instruments, Quincy, Mass.) and subsequently digitally filtered during processing. To monitor acceleration in the sagittal plane, a 50-G accelerometer (ADXL150EM3, Analog Devices, Norwood, Mass.) was attached to the end of the dynamometer armature.

2.2.4 Protocol

The experimental protocol utilized an isotonic knee extension model in the assessment of any effects of PAP. The isotonic mode on the dynamometer consisted of a minimal load at which the armature began to move; the torque levels produced by the subjects could exceed this preset load, however. This is analogous to a leg extension with a free weight with reduced inertia at higher loads, in contrast to a true isotonic load where the unit would continue to accelerate to keep the load constant during the entire range of motion. It was felt that an isotonic model better represented the types of actions performed in daily living and would create a more relevant PAP characterization scheme in comparison to an isokinetic approach. Furthermore, since isometric rates of force development have been shown to be more consistently correlated with PAP (Vandenboom et al., 1993), an isokinetic protocol may fail to reveal whether or not greater velocities are present during dynamic actions. In addition, the time at which peak torque is achieved during high velocity isokinetic actions may occur at a point well past L_0 , resulting in a peak torque close to the end of the joint's range of motion. This artifact would be reduced using isotonic contractions. Isometric twitches were evoked as an

index of the degree of PAP present at key points during data collection. To minimize the number of visits required, testing was performed on both legs each session (i.e., one condition per leg explained below). Consequently, subjects attended 4 sessions consisting of 1 habituation, 2 test and 1 twitch potentiation control run. Subjects were instructed to refrain from any heavy lower leg exercises or caffeine consumption 24 hours prior to testing.

2.2.5 Experimental Trials

Session #1: Habituation session. The primary purpose of this session was to habituate the subject to electrical stimulation of the knee extensors and to maximal isotonic and isometric contractions on the dynamometer. Subjects were required to perform 3 10-s maximal isometric knee extensions (MVCs) per leg. Isotonic loads for subsequent test sessions were derived as a percentage of the best MVC peak torque achieved with each leg. Following the MVCs, each subject was required to do a minimum of 3 practice maximal isotonic knee extensions at each of the 4 loads. In addition, if the subject had not previously experienced knee extensor twitches, the protocol used to elicit a maximal twitch response was also done during this session.

Sessions #2 and #3: Treatment and Control Sessions. The treatment and control protocols are illustrated in Figure 2. Each test session consisted of a control trial (CON) followed by a PAP trial separated by a 15 min. rest. Isometric twitches were evoked on the knee extensors using the Biodex dynamometer (90° tibio-femoral joint angle which was sufficient to remove the excess play of the armature that would "contaminate"

twitches) and were used as an index of PAP. The twitches evoked after each isotonic contraction determined whether PAP was increasing or decreasing (fatigue effects) as a result of these actions. The time line indicated in Figure 2 indicates the delay between measurement intervals within each trial. Since isometric twitches were used to quantify the amount of PAP during the dynamic actions, they were evoked as close as possible to each isotonic knee extension. Due to constraints of the apparatus, the time interval between the evoked twitch and isotonic contraction could be no less than 10 seconds. Each lab visit entailed one randomized control plus isotonic load set per leg (i.e. two loads per session). Isotonic loads were chosen from a randomized set of 4 values comprised of 15%, 30%, 45%, and 60% of the highest MVC torque obtained during the habituation session for the leg to be tested.

The last measure for each leg for each load was a ramp isometric knee extension (4-s total), which was used to establish a torque to EMG relationship during knee extension (see below). In addition, a normalizing isokinetic eccentric contraction of the knee flexors at 180°/s was done at the end of session 3. This normalizing maximal eccentric action was done to provide a relevant reference when quantifying the amount of co-activation present during knee extension, in comparison to an isometric action. Subjects were allowed 3 submaximal eccentric actions (.50%) at 60, 90 and 120°/s prior to the maximal effort. A ratio of isotonic knee flexor AEMG during knee extension to maximal eccentric knee flexor AEMG would be used as an index of co-activation during each isotonic contraction.

Session #4: Isometric Control Session. Due to the possibility of significant degradation of potentiation during the period after the MVC to before the isotonic action, and the added confounder of additional time delays between switching from an isometric to isotonic mode during each measurement interval, an isometric control trial was included. Each of these trials was comprised of a protocol identical to the PAP trials except that isometric twitches were evoked in place of isotonic contractions. This provided an estimate of the amount of potentiation during each isotonic contraction. In addition, comparisons between twitches evoked prior to and after each contraction during sessions 2 and 3 and the potentiation control trial served to verify the consistency of potentiation within subjects across test days (Fig. 3).

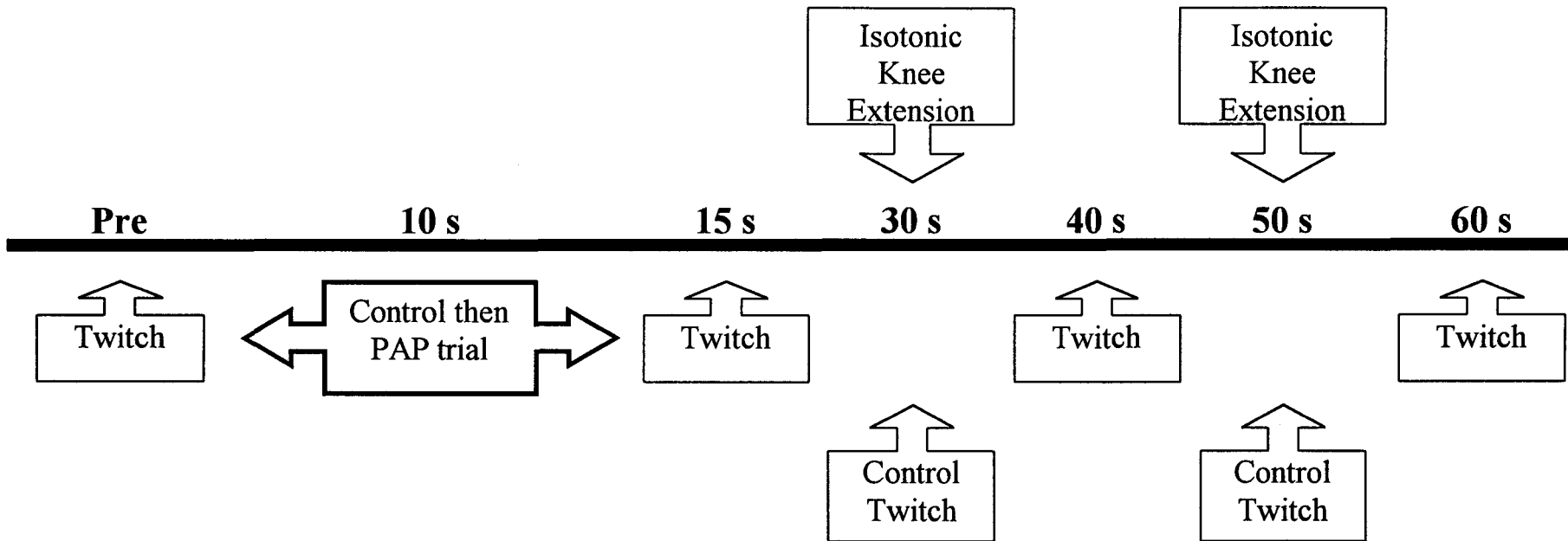


Figure 3: Schematic of twitch potentiation control protocol. Control twitches were evoked in place of isotonic knee extensions for both legs.

2.2.6 Electromyography (EMG) Processing

Both voluntary and evoked (M-wave) EMG were monitored. Voluntary EMG determined whether any performance enhancements were in part due to increased motor unit activation, whereas the M-wave amplitude controlled for any changes in the muscle action potential. Prior to analysis, all voluntary EMG was highpass filtered (cutoff frequency = 10 Hz) with custom software to remove movement artifact. EMG was subsequently processed using two different methods.

To obtain a measure of activation across an entire movement, average EMG (AEMG) was calculated by rectifying the signal during each knee extension and averaging all data points; values were then normalized to M-wave amplitude and expressed as a percent of the latter. A second method of EMG processing was used to obtain instantaneous activation during a trial, and could be considered an indirect measure of the intensity of the active state (Dowling, 1997). The ramp contraction served as a calibration trial to establish a relation between torque and EMG. Rectified EMG was smoothed using a critically damped lowpass filter (SEMG) with a cutoff frequency (f_c) and nonlinearity (n) adjustment chosen such that when SEMG was plotted as a function of the torque the relationship was linear. All subsequent control and PAP trials specific to the appropriate calibration ramp contraction were processed using the determined f_c and n (for a review of the process, see Dowling, 1997). Since the M-wave amplitude is representative of 100% activation of motor units, instantaneous voluntary activation

values (e.g. activation at peak torque) were normalized to baseline M-wave amplitudes and expressed as a percent peak activation.

When M-wave amplitude was analyzed, however, an average upward shift of 9.8% across loads was present in comparison to PRE values (Figure 5). The shift, however, was present in both the CON and PAP conditions, indicative of a methodological error. The reason for the increase can be attributed to two sources of error including software calibration and hardware gain. Due to the need for better resolution in establishing a baseline twitch, two separate calibration files were required for data acquisition (baseline twitch and CON/PAP files) with different reference hardware gain settings for each. Since both voluntary and evoked EMG were recorded throughout CON and PAP trials, hardware gains were repeatedly toggled for optimal resolution; gain adjustments were then carried out through multiplication of the raw data signal within a spreadsheet. The combination of innate errors in software calibration and errors across hardware gains resulted in the systematic upward shift of M-wave amplitudes. This is reflected by the consistency of normalized M-wave data. Thus it can be said that during both CON and PAP, no effect of M-wave amplitude was present.

2.2.7 Measurements

Twitch response measurements included peak twitch torque (P_t) and muscle compound action potential (M-wave) amplitude. Dynamic performance measures included peak torque, velocity and joint angle at peak torque, peak power, work attained

up to the point of peak power (hereafter referred to as work to peak power) and peak velocity. Velocity was calculated by differentiating angular displacement using custom residual analysis software ($f_c = 20$ Hz). Velocity was then multiplied by torque to derive power. The power-time curve was then integrated to obtain work. Activation was derived from computer analysis of agonist EMG and consisted of activation at peak torque, activation at peak power and AEMG.

2.2.8 Statistics

A 3 factor mixed analysis of variance (ANOVA) on all isotonic measures (4 load x 2 condition [control/PAP] x 2 kicks per condition) was performed. To assess whether there was any effect on the peak torque-velocity relationship across conditions a 2 variable (peak torque, velocity at peak torque) multivariate analysis of variance (MANOVA) was performed. Significance for multivariate and all subsequent univariate analyses was set at $p \leq 0.05$.

Twitch response measurements were analyzed through a three factor ANOVA (4 load x 2 condition x 4 time) to assess the degree of isometric twitch potentiation across trials. Descriptive statistics include means \pm standard error (SE). All statistical analysis was done using the Statistica[®] software package (StatSoft Inc., Tulsa, OK).

2.3 Results

2.3.1 Twitch Contractile Properties

Resting twitch peak torque (P_t) values were consistent (see Appendix 1) with no differences across loads or conditions. Figure 4 compares twitches from CON, PAP and twitch control trials. As seen in Figure 4A, significant potentiation was induced and maintained throughout the PAP trial. CON also showed significant twitch potentiation after the first kick for all loads ($p < 0.01$). The twitch control trial time points that correspond to isotonic knee extension during PAP (Fig. 4B) showed that the 10-s MVC induced a significant potentiation effect throughout the measurement period, even in the absence of isotonic knee extensions (time effect, twitch times corresponding to kick #1, $52.6 \pm 3.7\%$; twitch times corresponding to kick #2, $43.2 \pm 2.9\%$, $p < 0.01$).

P_t normalized to resting values (PRE) exhibited a significant load x condition x time interaction ($p < 0.05$) with 60% exhibiting a greater relative potentiation than 15% or 30% after the second knee extension ($p < 0.01$) (15%, $24.8 \pm 3.4\%$; 30%, $22.7 \pm 3.3\%$; 45%, $30.6 \pm 2.4\%$; 60%, $39.5 \pm 5.1\%$) (Fig. 4A, left). Immediately following MVC, 60% showed a greater relative potentiation than 30% or 45% ($p < 0.01$) (15%, $67.9 \pm 14.5\%$; 30%, $66.2 \pm 6.9\%$; 45%, $63.4 \pm 4.1\%$; 60%, $77.3 \pm 10.1\%$) (Fig. 4A, right).

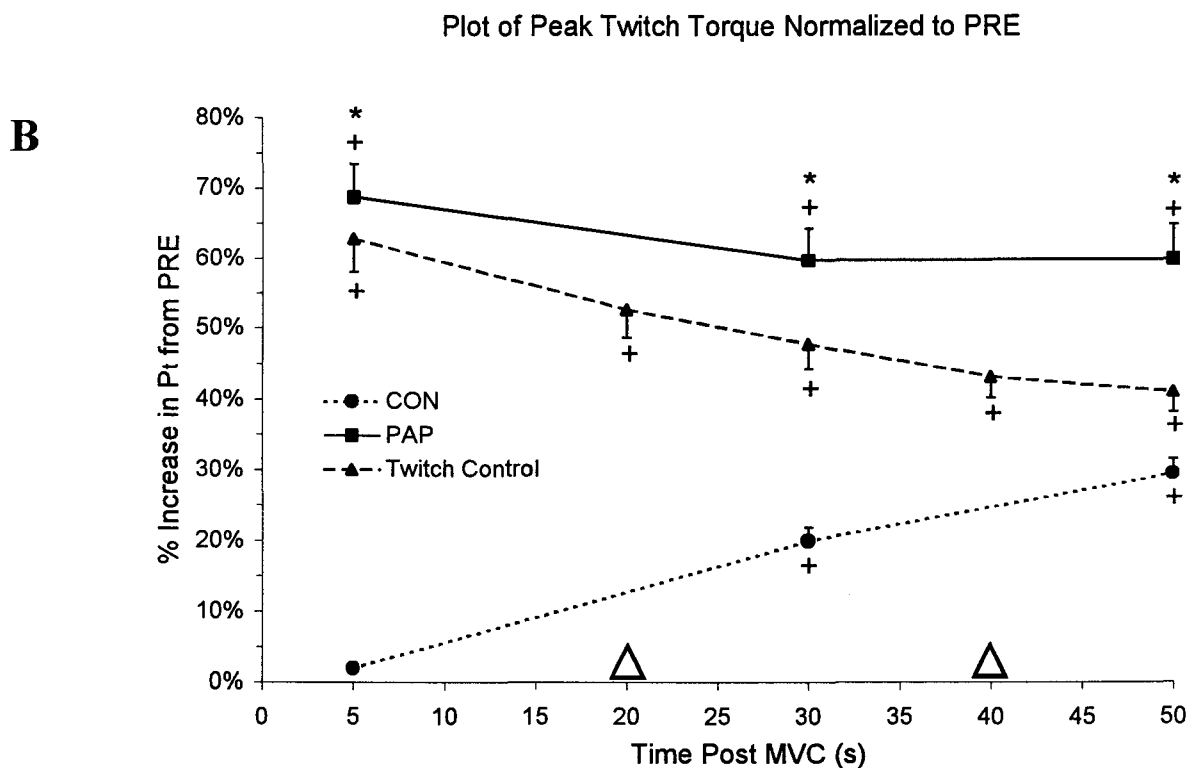
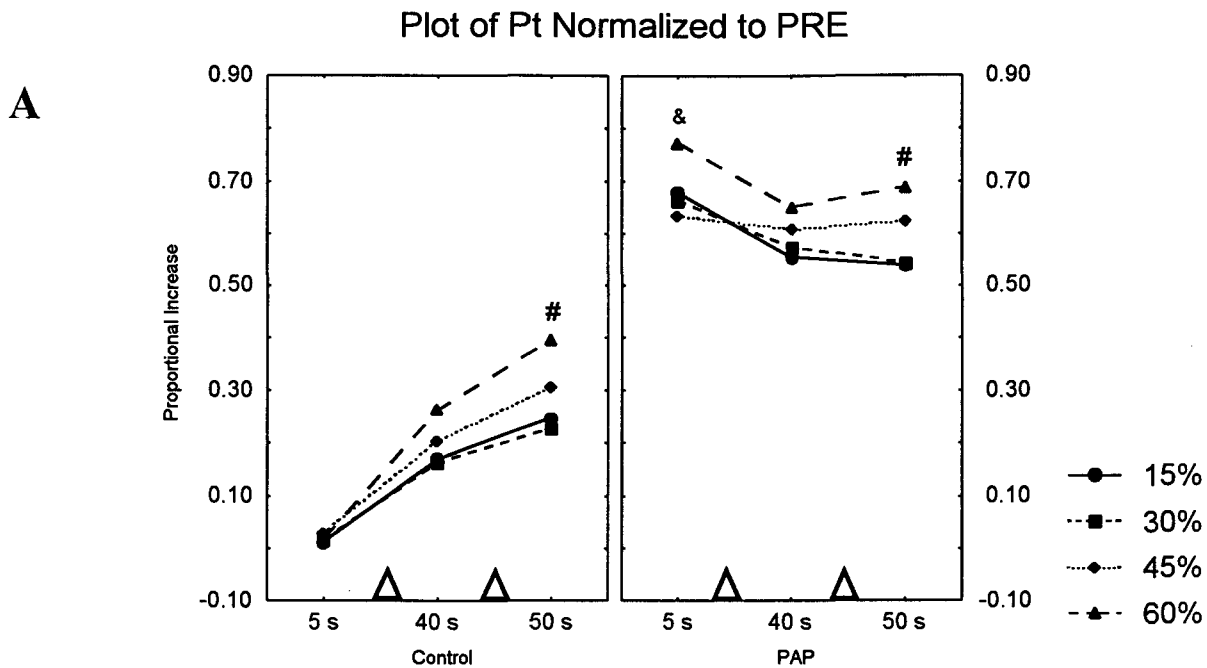


Figure 4: **A:** Plot of peak twitch torque (P_t) normalized to resting twitch (PRE) during control (CON) and potentiation (PAP). Ordinate values are proportional increases from PRE (i.e., 0.50 = 50%). # different from 15% and 30% load, $p < 0.05$. & different from 30% and 45% load, $p < 0.05$. **B:** P_t normalized to PRE during CON, PAP collapsed across load and twitch control trials collapsed across leg (observations per mean for CON and PAP = 40; twitch control = 20). Δ represent time points where knee extension occurred during CON and PAP. Means are collapsed across load (condition \times time interaction, $p < 0.001$). * different from CON, $p < 0.001$. + different from PRE, $p < 0.01$.

Resting M-wave amplitudes were also found to be consistent across trials (Appendix 1). A time effect for absolute values was found (Fig. 5) ($p < 0.0001$) as a result of the aforementioned methodological error. When amplitudes following the resting M-wave were normalized to PRE, however, no significance condition or time effects were present.

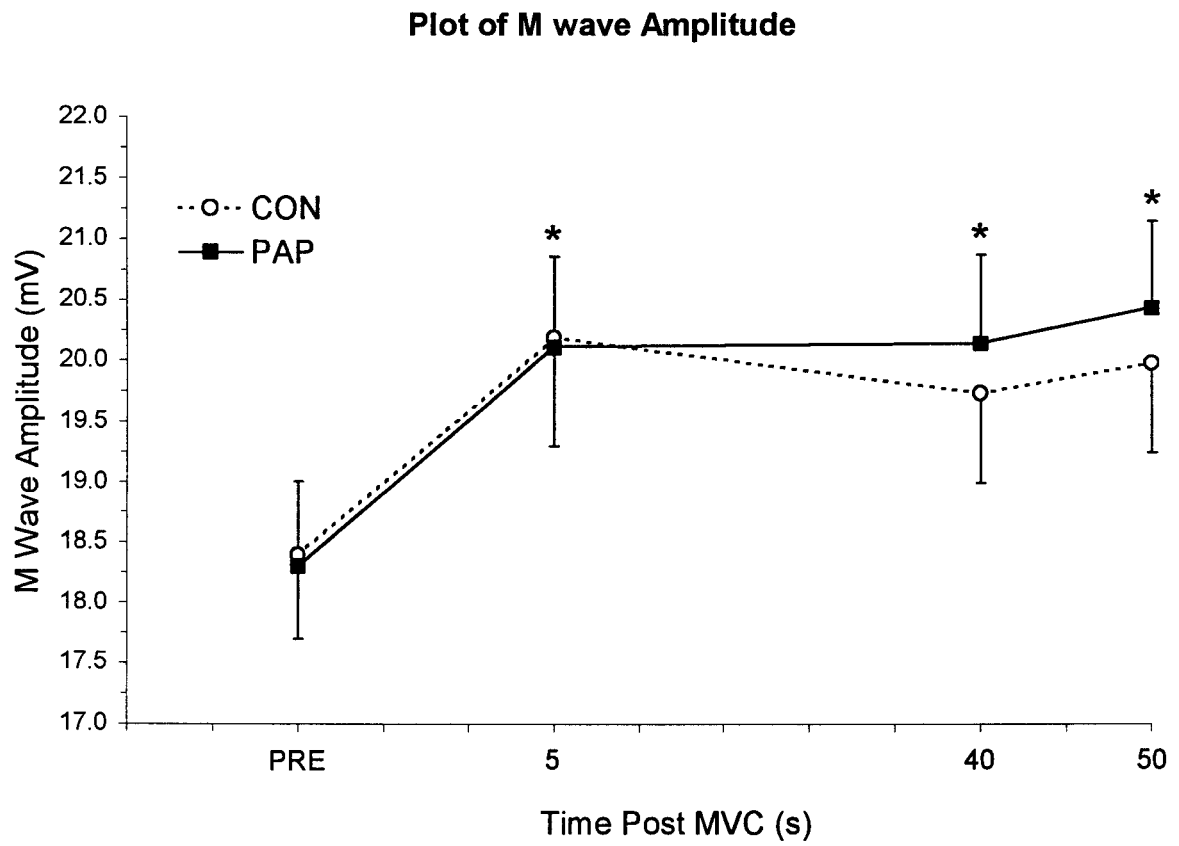


Figure 5: Plot of condition x time for absolute M-wave amplitude. Means are \pm SE with number of observations per mean = 40.

2.3.2 Dynamic Measures

Example baseline torque-time plots for all loads and corresponding EMG are illustrated in Figure 6. Despite significant twitch potentiation during both CON and PAP, dynamic measures showed no increases across conditions. Table 1 displays the means for peak torque achieved during each trial.

Table 1: Means of peak torque (N•m) achieved. Means are \pm SE (N = 10 per mean).

Load (% MVC)	Peak Torque CON (N•m)			
	15	30	45	60
Kick #1	72.9 \pm 6.7	88.2 \pm 6.0	108.1 \pm 8.2	140.7 \pm 13.5
Kick #2	69.6 \pm 6.6	89.3 \pm 6.1	108.7 \pm 8.0	141.4 \pm 13.4
	Peak Torque PAP (N•m)			
Kick #1	67.6 \pm 6.1	87.6 \pm 6.8	107.4 \pm 8.2	138.9 \pm 13.0
Kick #2	68.3 \pm 5.7	88.8 \pm 6.2	109.3 \pm 7.9	138.9 \pm 13.8

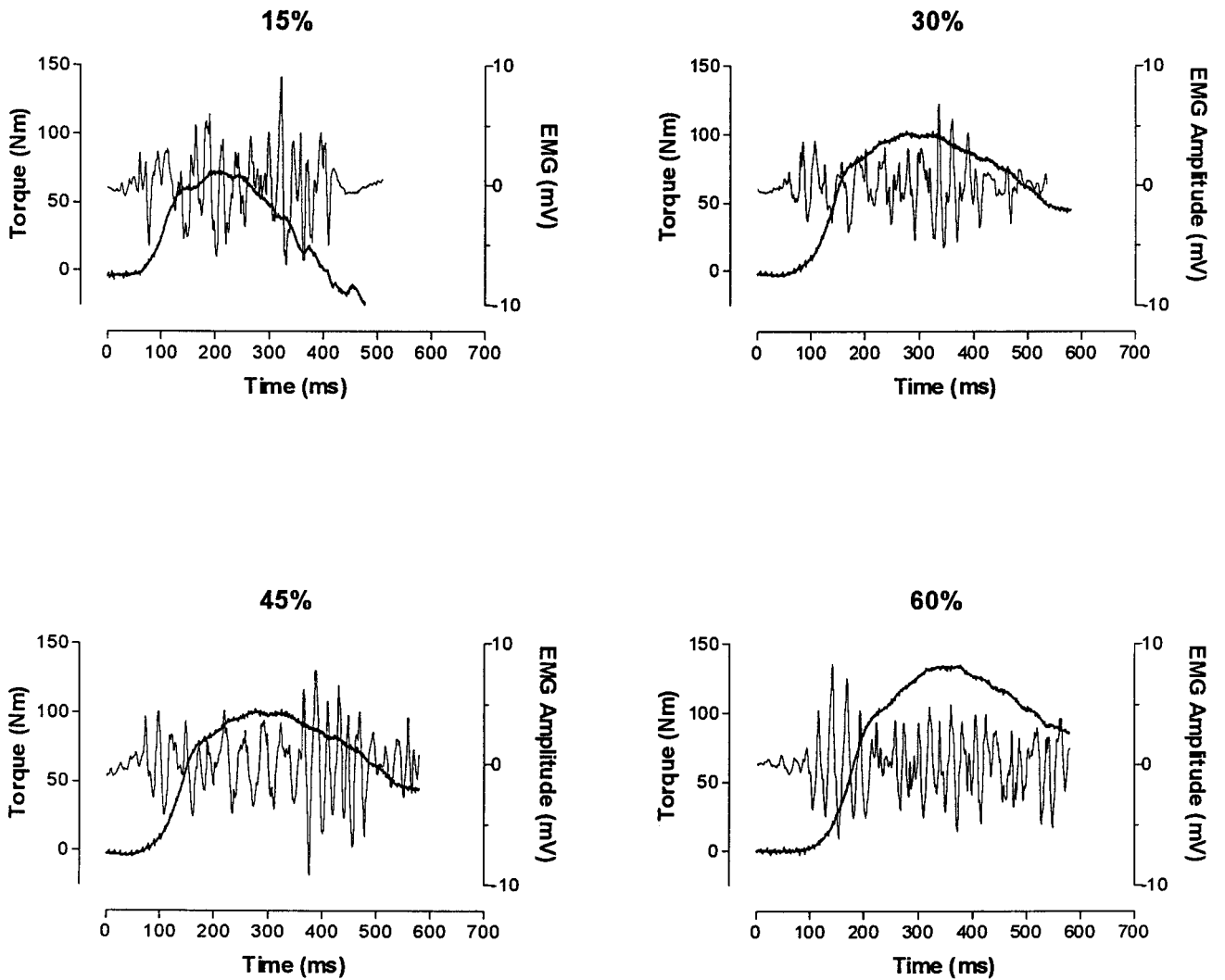


Figure 6: Example torque-time curves of knee extensions across 4 test loads (CON trials).

Similarly, other than a load main effect (MANOVA, $p < 0.01$; peak torque, $p < 0.0001$; velocity at peak torque, $p < 0.001$), there were no effects or interactions on either the peak torque-velocity (Fig. 7) or load-peak velocity (Fig. 8) relationships. Both plots, however, do show a tendency to shift leftward during PAP. In addition, peak velocity exhibited a main effect for condition as a result of decreases from CON to PAP (CON, 341.6 ± 10.6 °/s; PAP, 326.7 ± 10.6 °/s, $p < 0.03$), but showed a kick order main effect due to increases from kick #1 to kick #2 (kick #1, 328.1 ± 10.7 °/s; kick #2, 340.2 ± 10.6 °/s, $p < 0.02$). It should be noted that coincident with peak torque and velocity at peak torque, there was a significant effect of load on joint angle as well ($p < 0.001$). The range over which subjects achieved peak torque across loads, however, was small and subjects were generally able to achieve peak torque within 14° of angular displacement from onset of movement.

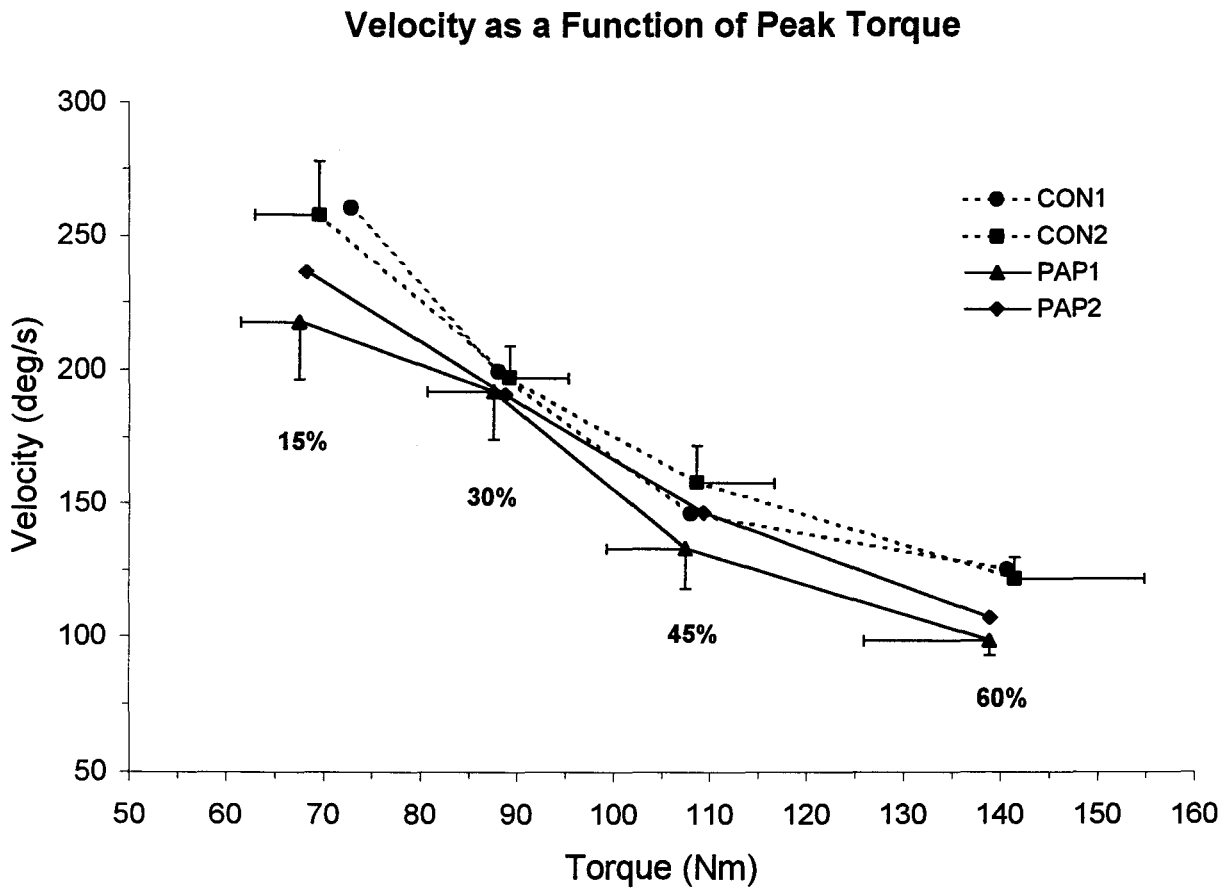


Figure 7: Torque-Velocity at Peak Torque relationship. CON1 and CON2 represent knee extensions 1 and 2 in CON respectively; PAP1 and PAP2 similarly represent kicks 1 and 2 in PAP. Values are means ($N = 10$) \pm SE. Some error bars (CON1 and PAP2) are omitted for clarity.

Peak-Velocity as a Function of Applied Load

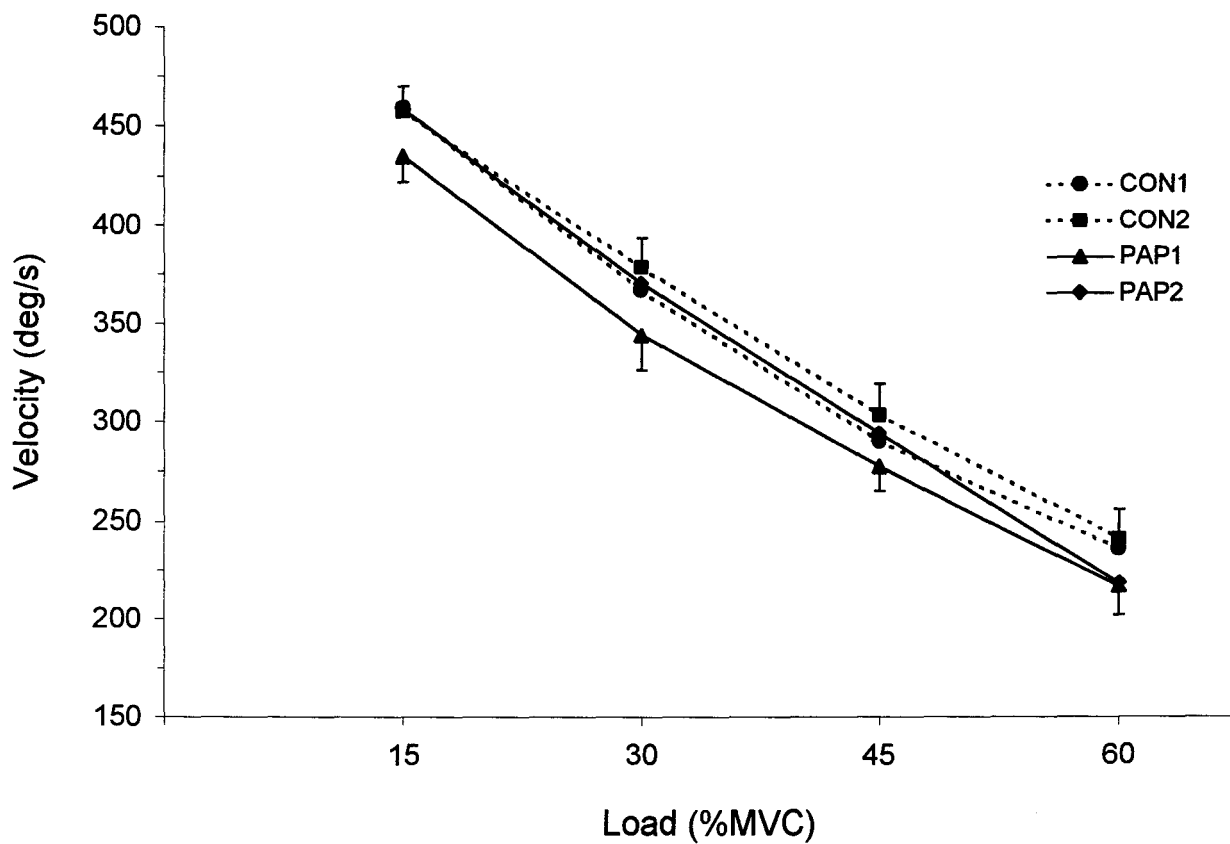


Figure 8: Plot of peak velocity as a function of applied load. Means are \pm SE with 10 observations for each mean. Some error bars are omitted for clarity

Peak power and work to peak power both showed no significant interactions (peak power, $p = .58$; work to peak power, $p = 0.13$). Any differences in peak power were due to main effects across load, condition (i.e., CON/PAP) or kick number. As with peak velocity, the condition main effect for peak power was a result of a decrease from CON to PAP (CON, 462.2 ± 16.2 W; PAP, 435.1 ± 14.1 W, $p < 0.05$). In contrast, the kick order main effect for peak power was a result of an increase from kick #1 to kick #2 (CON, 441.3 ± 15.2 W; PAP, 456.0 ± 15.3 W, $p < 0.05$). No differences in movement times to peak power were displayed across conditions, however (CON, 197.8 ± 4.3 ms; PAP, 199.2 ± 5.6 ms, $p = 0.78$). In addition, despite the lessening of peak power across conditions, no corresponding changes for work to peak power across conditions (CON, 44.5 ± 2.0 J; PAP, 43.1 ± 1.6 J, $p = 0.34$) or kick order (kick #1, 42.7 ± 1.8 J; kick #2, 44.9 ± 1.8 J, $p = 0.09$) were apparent.

To see the degree to which recovery from the MVC may have contributed to the peak power kick order effect, or if there was a potentiating effect on peak power during CON, two retrospective ANOVAs were performed separately across CON and PAP. Similarly, separate ANOVAs on work to peak power and movement time to peak power were performed. It was found that peak power increased significantly from the first to second knee extension in PAP only (Table 2). Interestingly, during CON, while peak power showed no kick order main effect, work to peak power exhibited a significant increase ($p < 0.03$). Conversely, during PAP, while peak power from the first to second kick showed a significant increase, work to peak power was unchanged ($p = 0.66$).

Table 2: Means for peak power, work to peak power and time to peak power for kick order main effects.

	CON		PAP	
	Kick #1	Kick #2	Kick #1	Kick #2
Peak Power (W)	456.9 ± 23.5	467.6 ± 22.9	425.7 ± 19.5	444.5 ± 20.8*
Work to Peak Power (J)	42.7 ± 2.7	46.4 ± 2.8*	42.7 ± 2.4	43.5 ± 2.3
Time to Peak Power (ms)	195 ± 7	201 ± 6	198 ± 7	200 ± 9

For all the above values, ANOVAs were performed separately across CON and PAP, thus no direct comparisons across conditions were made in the above values. Only peak power in PAP showed a significant increase from the first to second kick (CON, $p = 0.14$; PAP, $p < 0.05$). Work to peak power, however, increased from kick #1 to kick #2 in CON ($p < 0.03$) and did not change during PAP ($p = 0.66$), despite a lower peak power during kick #1. Movement times remained unchanged across all trials. * different from kick #1 within condition, $p < 0.05$.

2.3.3 Activation and AEMG

An example of the EMG calibration process and the corresponding torque/activation relationship are illustrated in Figure 9. Activation at peak torque changed only across conditions, showing an overall decrease from CON to PAP (CON, $7.2 \pm 0.3\%$; PAP, $6.6 \pm 0.3\%$, $p < 0.01$). Activation at peak power showed a similar condition main effect, exhibiting a significant depression during PAP (CON, $8.0 \pm 0.3\%$; PAP, $7.5 \pm 0.3\%$, $p < 0.02$). In contrast, both peak activation (CON, $8.9 \pm 0.3\%$; PAP, $8.5 \pm 0.3\%$, $p = 0.09$) (Figure 10) and AEMG (CON, $4.9 \pm 0.2\%$; PAP, $4.5 \pm 0.3\%$, $p = 0.26$) showed no condition main effects, although both did show a slight tendency toward depression across conditions. No other interactions or main effects for activation at peak torque, activation at peak power, peak activation or AEMG were present.

Despite the precautions taken, antagonist EMG had excessive movement artifact due to the variable pressure on the electrodes during each knee extension. As a result, no subsequent analysis was carried out.

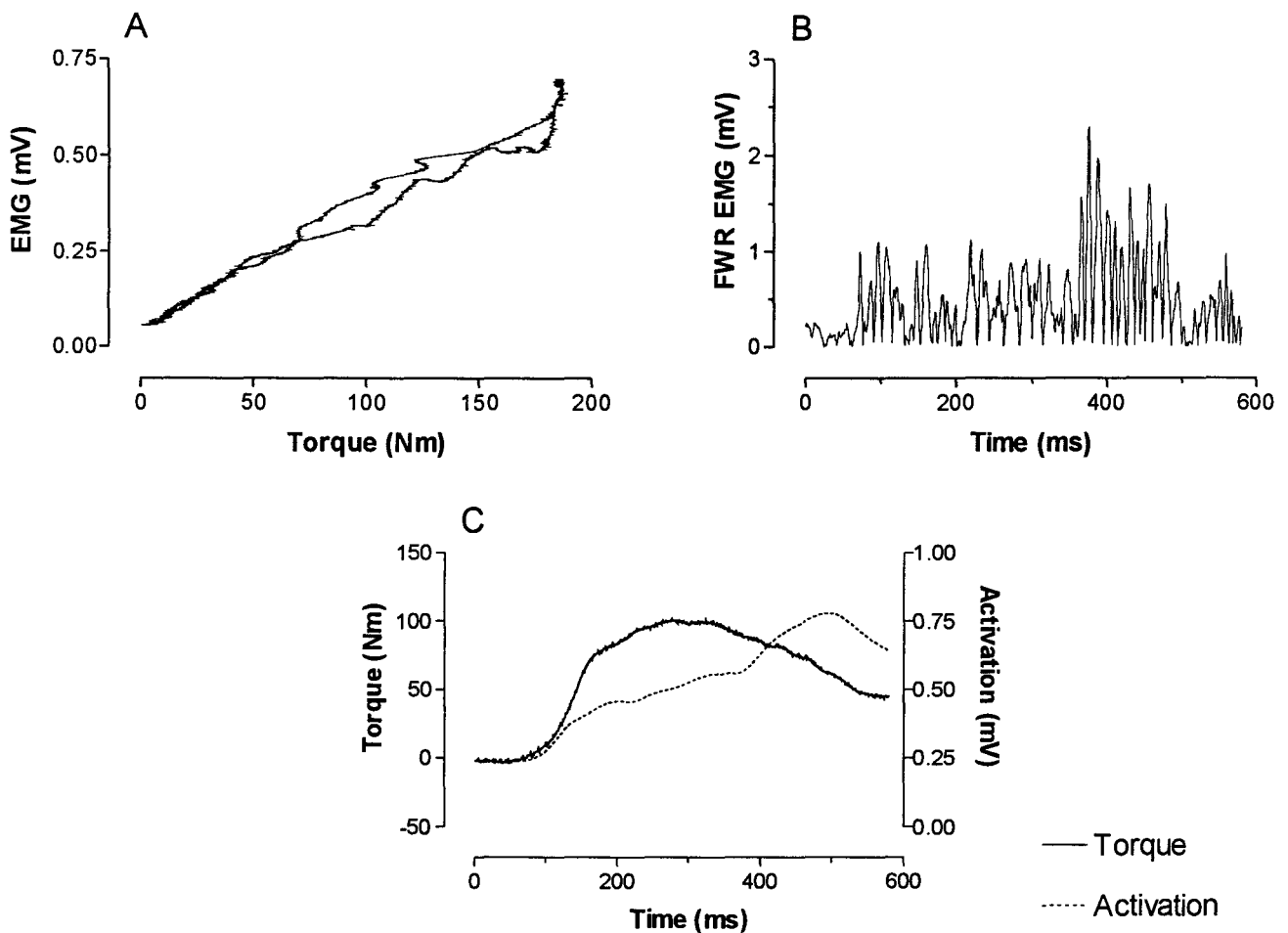


Figure 9: Example plots illustrating various steps in deriving the prediction of instantaneous activation at a load of 30% MVC. **A.** The Torque/EMG relationship after an appropriate f_c (2.2 Hz) and non-linearity ($n = 0.70$) has been determined (i.e. calibration). The calibration process was repeated for every load condition. **B.** Full wave rectified (FWR) EMG of a subject's isotonic extension at 30% MVC after highpass filtering ($f_c = 10\text{Hz}$). Average EMG (AEMG) was derived from calculating the mean of all points. **C.** Using a critically damped 2nd order lowpass filter, the f_c and n determined from (A) was applied to (B) to derive a torque/activation relationship. From the derived activation curve, various instantaneous estimates of the 'active state' of the muscle could be determined. The difference between activation and torque in 'C' can be attributed to changes in moment arm, muscle length and shortening velocity. It is interesting to note that maximum activation in this example was not achieved until late in the movement.

Plot of Activation Condition Main Effects

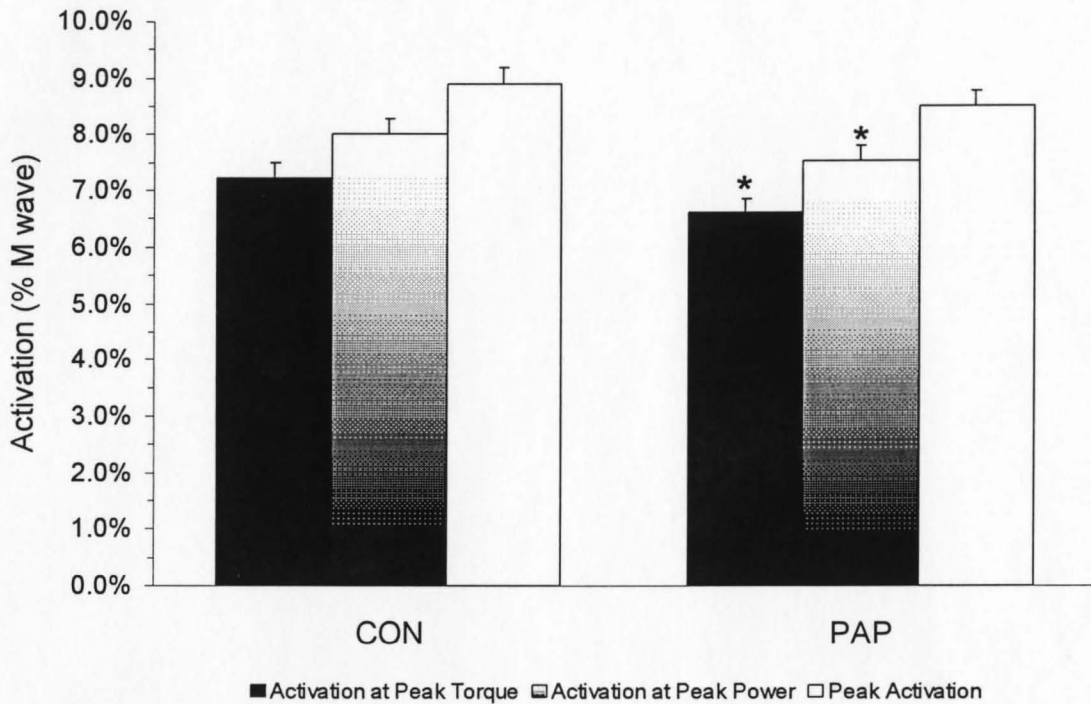


Figure 10: Comparison of activation condition effects. Although activation at peak torque and activation at peak power did exhibit significant reductions from CON to PAP, peak activation (as with AEMG) did not show significant reductions across conditions. Values are means \pm SE with N = 80 observations per mean. * different from CON, $p < 0.05$.

2.4 Discussion

Investigation of the significance of postactivation potentiation (PAP) during dynamic movements has been limited. Isometric animal models investigating mechanisms associated with PAP have found rates of force development consistently enhanced with phosphorylation of myosin light chains. These increases in rates of force development have been shown to hold across a large range of stimulation frequencies (Vandenboom et al., 1993; 1995; 1997) and during fatigue (Vandenboom et al., 1997). Load clamping (Grange et al., 1995) and work cycle methods (Grange et al., 1993; 1998) have shown that work and power better reveal the effects of potentiation than does isometric peak force during low frequency stimulation. There appears to be no effect of PAP on peak isometric force (Vandenboom et al., 1993) or peak unloaded velocity of shortening (V_{max}) (Butler et al., 1983; Grange et al., 1993), however. Likewise, PAP in humans has been found to have no effect on V_{max} (Grange et al., 1993; Stuart et al., 1988). In the current study, it was hypothesized that, as a result of increased rates of torque development, PAP would increase the velocity attained with loads between the extremes of the load-velocity relation occurred. When a series of four loads were tested, however, the results indicated that no enhancements occurred in maximal isotonic knee extension performance during periods of maximal twitch potentiation, nor were there significant changes in the load-velocity relation. The load-velocity relation actually

exhibited a slight leftward shift during PAP rather than the hypothesized rightward shift (compare Fig. 7 with Fig. 1).

A recurring finding was that any changes in performance from CON to PAP were a result of decreases in dynamic measures rather than increases. Unlike studies examining the effects of potentiation on evoked force-velocity characteristics of small mammals at low frequencies (Grange et al., 1995; 1998), maximal volitional efforts during PAP did not result in a rightward shift in the force-velocity relation as hypothesized. In contrast, although effects of PAP on the peak load-velocity (Fig. 7) and load-peak velocity (Fig. 8) relations showed no significant change from CON to PAP, there was a tendency for the curves to shift leftward. When main effects were examined, the consequences of the shift were manifest through significant decreases in peak power and peak velocity from CON to PAP. Thus the 10-s MVC that has been shown to be optimal for inducing immediate maximal twitch potentiation (Green and Jones, 1989; Vandervoort et al., 1983) actually lead to reductions in maximal voluntary performance in the time frame studied.

A 10-s MVC of the knee extensors is typically associated with an immediate 2-fold increase in phosphate content of both slow and fast myosin light chains of the vastus lateralis (Houston and Grange, 1990; Houston et al., 1987; Stuart et al., 1988). The most consistent contractile correlate to myosin light chain phosphorylation has been shown to be rate of force development (Vandenboom et al., 1993; 1997). In this study, however, any potentiation of rate of torque development was not sufficient to overcome the fatigue induced by the MVC and consequently did not translate into enhanced velocity or power.

The observation of substantial twitch potentiation concurrent with depressed maximal performance parallels findings of Green and Jones (1989) who found the existence of low frequency force potentiation concurrent with depression of peak tetanic force. In light of no significant change in peak activation and AEMG, and because depression of activation at peak torque and at peak power across conditions was small, the depression in peak power and peak velocity was primarily due to peripheral fatigue. Central fatigue cannot be ruled out, however; recovery of the central nervous system has been considered in previous studies examining enhancement of power performance following 5 RM squats (Young et al., 1998).

Another significant finding was that twitch potentiation was induced as a result of the kicks during CON (Fig. 4A). Past studies that have shown progressively enhanced torque early in isokinetic fatigue tests (Thorstensson and Karlsson, 1976; Tesch and Wright, 1983) and performance increases during repeated jumps (McArdle et al., 1996) suggest that enhancements in performance can be induced without the heavy loads utilized by Güllich and Schmidbleicher (1995) and Young et al. (1998). It is possible that there would have been a right shift in the load-velocity relationship had the potentiation been induced through repeated knee extensions and not by a maximal isometric effort.

Despite the lack of findings showing performance enhancement during twitch potentiation, there are factors that suggest there was a compensatory mechanism at work during CON and PAP as revealed in the retrospective analyses of peak power and work (Table 2). First, it was shown that during PAP, there was a significant decrease in peak

power in kick #1 in comparison to kick #2; conversely, there were no changes in movement time to peak power. Second, during CON, there was no difference in peak power from the first to second kick, yet work showed a significant increase from kick #1 to kick #2. As with PAP, movement times to peak power remained unchanged across kicks. Thus, it is feasible that these different interactions between peak power and work to peak power during CON and PAP may be an unexpected reflection of the effects of potentiation on maximal efforts. Potentiation has been shown to be an effective compensatory mechanism for fatigue (Alway et al., 1987; Garner et al., 1989; Gibson et al., 1988; Grange and Houston, 1991; Green and Jones, 1989, MacIntosh et al., 1993; Rankin et al., 1988; Tubman et al., 1996).

Further still, *in vitro* studies have shown consistent enhancements of rates of force development across a wide range of stimulation frequencies regardless of decreases in peak tetanic force (Vandenboom et al., 1993, 1995, 1997). Yet these rates of force development across stimulation frequencies (20 – 150 Hz) were limited to the initial rising phase of force development (i.e., first 50 ms), due to the limited force-potentiating influences of myosin phosphorylation at higher myoplasmic Ca^{2+} levels (Vandenboom et al., 1993). It is possible, during the PAP trial, that the rates of torque development were enhanced early during kick #1 and translated into a proportionately larger work to peak power. Similarly, during CON, the enhancement of work to peak power during kick #2, despite no increases in the peak power attained, would reflect the work potentiating effect of kick #1 on kick #2. Thus it seems that the protocol utilized served to illustrate the effect of twitch potentiation on isotonic work, not on the load-velocity relationship or

peak power as initially hypothesized. On examination of current literature, this effect of potentiation on isotonic work should be expected. Using load clamping of mouse fast muscle, Grange et al. (1995) showed that during a period of 5-fold increase of R-LC phosphorylation content above baseline values, net muscle displacement was increased by 20% while shortening velocity was increased by 11%. A subsequent study using the work cycling technique (Grange et al., 1998) showed that dynamic conditions better revealed potentiation than did isometric twitch measures as was illustrated through a maximal potentiation of work and power of 50% when isometric P_t showed maximal potentiation levels of only 14%.

In conclusion, it was found that dynamic performance coincident with PAP was not increased in a load dependent manner as hypothesized. Enhancement in work during CON, however, was present in an unfatigued state, concurrent with twitch potentiation. In contrast, during PAP, work was maintained despite decreases in peak power. Although a clear increase in dynamic performance as a result of potentiation was not established, an alternate approach to the induction of PAP may have revealed a progressively greater potentiation of work and power prior to the onset of fatigue.

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Appendix 1: Supplementary Tables

Load (% MVC)	CON (N•m)	PAP (N•m)	CON Right Leg (N•m)	CON Left Leg (N•m)
15	31.9 ± 3.1	31.6 ± 2.8		
30	30.5 ± 2.7	28.4 ± 2.6		
45	29.6 ± 2.0	29.1 ± 2.4	32.9 ± 3.2	31.9 ± 3.0
60	30.3 ± 2.7	29.3 ± 2.7		

Table A - 1: Baseline P_t values. No significant overall load x condition x time ($F(9,81) = 0.92$, $p = 0.51$) or PRE condition x time ($p = 0.99$) differences were present. Values are means \pm SE with $n=10$ for each mean. Resting P_t values for the twitch control trials also showed no difference ($p = 0.91$).

Load (% MVC)	PRE CON (mV)	PRE PAP (mV)
15	17.4 \pm 1.3	17.6 \pm 1.4
30	18.3 \pm 1.2	17.8 \pm 1.0
45	18.6 \pm 1.3	18.4 \pm 1.4
60	19.4 \pm 1.4	19.4 \pm 1.3

Table A - 2: Baseline M-wave amplitude. No differences interactions were present across factors (load x condition, $F(3, 27) = 1.36$, $p = 0.28$; load x time, $F(9,81) = 1.10$, $p = 0.37$; load x condition x time; $F(9, 81) = 0.99$, $p = 0.46$).

	Time POST MVC (s)				
	5	20	30	40	50
Twitch Control	62.6 ± 4.7%	52.6 ± 3.8%	47.7 ± 3.5%	43.2 ± 3.0%	41.0 ± 2.9 %
PAP Twitch	68.7 ± 4.8%	Extension #1	59.6 ± 3.9%	Extension #2	59.8 ± 4.0%

Table A – 3: Comparison of twitch control P_t collapsed across legs to P_t during PAP trial collapsed across load. Number of observations per mean for twitch control = 20; number of observations per mean for PAP trial = 40.

Time(s)	Amplitude (mV)	% from PRE
PRE	18.3 ± 0.4	N/A
5	20.1 ± 0.1*	10.0 ± 1.4
30	19.9 ± 0.0*	8.9 ± 1.5
50	20.2 ± 0.0*	10.4 ± 1.5
Grand Mean		9.8 ± 0.9

Table A - 4: Means ± SE with number of observations per mean = 80 (grand mean, N = 240). * different from PRE for time main effect, $p < 0.001$.

Load (% MVC)	Peak Velocity (°/s)
15	452.4 ± 6.4*
30	364.9 ± 7.8*
45	291.1 ± 6.7*
60	228.2 ± 7.2*

Table A - 5: Means for peak velocities achieved during trials (i.e. load main effect, $F(3,27) = 105.2$, $p < 0.00001$). Values are \pm SE with $N = 40$ for each mean. * different from all other conditions, $p < 0.001$.

	Load (% MVC)				Row Means (ms)
	15	30	45	60	
Time to TOR (ms)	166 ± 10	215 ± 12	259 ± 15	270 ± 28	227 ± 11
Time to PWR (ms)	162 ± 7	190 ± 9	223 ± 10	204 ± 19	195 ± 7
Time to ACT_PEAK (ms)	277 ± 22	340 ± 26	396 ± 25	440 ± 58	363 ± 19*

Table A - 6: Baseline (i.e. CON, extension #1) times to peak torque (TOR), peak power (PWR) and peak activation (ACT_PEAK) are summarized below. Means are \pm SE. * different from TOR and PWR ($p < 0.001$).

Appendix 2: ANOVA Summary Tables

Peak Twitch Torque ANOVAs

Summary of all Effects; (Peak Twitch Torque - Absolute Values)
1-LOAD, 2-CON/PAP, 3-TIME

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	137.3	27	41.883	3.277	0.036 *
2	1	6367.7	9	126.688	50.263	0.000057 *
3	3	2830.1	27	37.851	74.768	0.000000 *
12	3	20.0	27	20.660	0.968	0.42
13	9	19.0	81	8.253	2.297	0.024 *
23	3	1283.3	27	24.778	51.793	0.000000 *
123	9	5.2	81	5.613	0.920	0.51

**Tukey HSD test; Twitch Pt Absolute
Probabilities for Post Hoc Tests
INTERACTION: Condition x Time**

CON/PAP	Time		{1}	{2}	{3}	{4}	{5}	{6}	{7}	{8}
			30.6	31.1	36.5	39.4	29.6	49.5	47.0	47.1
1	1 (PRE CON)	{1}		1.0	0.0005	0.000	0.9857	0.0001	0.0001	0.0001
1	2	{2}	0.9998		0.0012	0.000	0.8846	0.0001	0.0001	0.0001
1	3	{3}	0.0005	0.001		0.183	0.0002	0.0001	0.0001	0.0001
1	4	{4}	0.0001	0.000	0.1827		0.0001	0.0001	0.0001	0.0001
2	1 (PRE PAP)	{5}	0.9857	0.885	0.0002	0.000		0.0001	0.0001	0.0001
2	2	{6}	0.0001	0.000	0.0001	0.000	0.0001		0.3800	0.3889
2	3	{7}	0.0001	0.000	0.0001	0.000	0.0001	0.3800		1.0000
2	4	{8}	0.0001	0.000	0.0001	0.000	0.0001	0.3889	1.0000	

Summary of all Effects; (Peak Twitch Torque - Normalized to PRE)
1-LOAD, 2-CON/PAP, 3-TIME

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	0.131	27	0.119	1.096	0.37
2	1	12.500	9	0.101	123.173	0.00000 *
3	2	0.174	18	0.012	14.090	0.00021 *
12	3	0.004	27	0.084	0.045	0.99
13	6	0.015	54	0.005	2.838	0.018 *
23	2	0.717	18	0.010	72.010	0.00000 *
123	6	0.009	54	0.004	2.394	0.040 *

Tukey HSD test; Pt Normalized to PRE
Probabilities for Post Hoc Tests
INTERACTION: Condition x Time

Load	Condition	Time	{1} 1.9%	{2} 19.9%	{3} 29.4%	{4} 68.7%	{5} 59.6%	{6} 59.8%
....	1	1		0.0002	0.0002	0.0002	0.0002	0.0002
....	1	2	0.0002		0.0055	0.0002	0.0002	0.0002
....	1	3	0.0002	0.0055		0.0002	0.0002	0.0002
....	2	1	0.0002	0.0002	0.0002		0.0079	0.0097
....	2	2	0.0002	0.0002	0.0002	0.0079		1.0000
....	2	3	0.0002	0.0002	0.0002	0.0097	1.0000	

Summary of all Effects; (Twitch Control Trial - Absolute Values)
1-LEG, 2-TIME

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	383.9	9	51.567	7.445	0.023 *
2	6	822.8	54	12.197	67.456	0.00000 *
12	6	5.7	54	1.804	3.185	0.0095 *

Isotonic Peak Torque, Peak Power, Work to Peak Power, Joint Angle and Velocity ANOVAs

Summary of all Effects; (Torque x Velocity MANOVA) 1-LOAD, 2-CON/PAP, 3-KICK#

	Wilks' Lambda	Rao's R	df 1	df 2	p-level
1	0.014	47.03	6	4	0.0012 *
2	0.571	3.00	2	8	0.11
3	0.928	0.31	2	8	0.74
12	0.296	1.59	6	4	0.34
13	0.591	0.46	6	4	0.81
23	0.784	1.10	2	8	0.38
123	0.346	1.26	6	4	0.43

MAIN EFFECT: LOAD 1-LOAD, 2-CON/PAP, 3-KICK#

	Mean sq Effect	Mean sq Error	F(df1,2) 3,27	p-level
Peak Torque	36220.1	757.5	47.8	0.0000 *
Velocity at Peak Torque	129471.0	1792.2	72.2	0.0000 *

Summary of all Effects; (Peak Isotonic Torque - Absolute Values) 1-LOAD, 2-CON/PAP, 3-KICK

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	36207.5	27	758.20	47.75	0.00000 *
2	1	89.5	9	30.73	2.91	0.12
3	1	6.1	9	24.52	0.25	0.63
12	3	23.2	27	14.41	1.61	0.21
13	3	14.0	27	16.02	0.87	0.47
23	1	13.4	9	10.56	1.27	0.29
123	3	10.9	27	11.42	0.96	0.43

Summary of all Effects; (Peak Power) 1-LOAD, 2-CON/PAP, 3-Kick

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	33422.4	27	8815.8	3.79	0.022 *
2	1	30024.5	9	5270.7	5.70	0.041 *
3	1	8963.6	9	1316.4	6.81	0.028 *
12	3	3527.9	27	1928.1	1.83	0.17
13	3	347.2	27	1177.8	0.29	0.83
23	1	579.7	9	881.0	0.66	0.44
123	3	272.7	27	405.6	0.67	0.58

Summary of all Effects; (Time to PWR - CON Trial Only)
1-LOAD, 2-KICK#

	df	MS	df	MS	F	p-level
	Effect	Effect	Error	Error		
1	3	9249.9	27	1304.9	7.09	0.0012 *
2	1	729.0	9	1085.3	0.67	0.43
12	3	528.4	27	310.6	1.70	0.19

Summary of all Effects; (Time to PWR - PAP Trial Only)
1-LOAD, 2-KICK#

	df	MS	df	MS	F	p-level
	Effect	Effect	Error	Error		
1	3	9995.0	27	2804.9	3.56	0.027 *
2	1	63.9	9	436.5	0.15	0.71
12	3	1859.9	27	992.2	1.87	0.16

Summary of all Effects; (Peak Power - CON Trial Only)
1-LOAD, 2-KICK #

	df	MS	df	MS	F	p-level
	Effect	Effect	Error	Error		
1	3	22957.0	27	5400.3	4.25	0.014 *
2	1	2492.2	9	924.4	2.70	0.14
12	3	505.4	27	548.2	0.92	0.44

Summary of all Effects; (Peak Power - PAP Trial Only)
1-LOAD, 2-KICK #

	df	MS	df	MS	F	p-level
	Effect	Effect	Error	Error		
1	3	13993.2	27	5343.5	2.62	0.071
2	1	7051.1	9	1273.0	5.54	0.043 *
12	3	114.5	27	1035.2	0.11	0.95

**Summary of all Effects; (Work to Point of Peak Power)
1-LOAD, 2-CON/PAP, 3-KICK #**

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	2586.1	27	184.9	13.98	0.00001 *
2	1	91.5	9	91.9	1.00	0.34
3	1	210.5	9	59.1	3.56	0.092
12	3	46.2	27	50.1	0.92	0.44
13	3	18.8	27	37.6	0.50	0.69
23	1	93.1	9	38.7	2.40	0.16
123	3	63.7	27	30.6	2.08	0.13

**Summary of all Effects; (Work to Peak Power - CON Trial Only)
1-LOAD, 2-KICK #**

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	1230.7	27	154.1	7.99	0.00057 *
2	1	291.7	9	41.1	7.10	0.026 *
12	3	38.4	27	36.9	1.04	0.39

**Summary of all Effects; (Work to Peak Power - PAP Trial Only)
1-LOAD, 2-KICK #**

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	1401.7	27	80.9	17.33	0.00000 *
2	1	11.8	9	56.7	0.21	0.66
12	3	44.0	27	31.3	1.41	0.26

Summary of all Effects; (Velocity at Peak Torque)
1-LOAD, 2-CON/PAP, 3-KICK #

	df	MS	df	MS	F	p-level
	Effect	Effect	Error	Error		
1	3	129471.0	27	1792.2	72.2	0.00000 *
2	1	12880.9	9	2681.8	4.8	0.056
3	1	1159.9	9	2582.2	0.45	0.52
12	3	1236.3	27	1491.8	0.83	0.49
13	3	391.0	27	1112.7	0.35	0.79
23	1	737.0	9	1109.1	0.66	0.44
123	3	236.8	27	1307.2	0.18	0.91

Summary of all Effects; (Peak Velocity)
1-LOAD, 2-CON/PAP, 3-KICK #

	df	MS	df	MS	F	p-level
	Effect	Effect	Error	Error		
1	3	373315.3	27	3548.7	105.2	0.00000 *
2	1	8875.9	9	1278.3	6.94	0.027 *
3	1	5816.5	9	657.6	8.84	0.016 *
12	3	221.3	27	688.1	0.32	0.81
13	3	437.0	27	278.1	1.57	0.22
23	1	901.1	9	429.0	2.10	0.18
123	3	395.1	27	279.3	1.41	0.26

Summary of all Effects; (Joint Angle at Peak Torque)
1-LOAD, 2-CON/PAP, 3-KICK#

	df	MS	df	MS	F	p-level
	Effect	Effect	Error	Error		
1	3	340.9	27	19.9	17.13	0.00000 *
2	1	64.3	9	11.5	5.61	0.042 *
3	1	16.8	9	18.3	0.92	0.36
12	3	6.6	27	12.9	0.51	0.68
13	3	0.5	27	9.0	0.05	0.98
23	1	11.0	9	11.6	0.95	0.36
123	3	1.4	27	9.8	0.15	0.93

Movement Time ANOVAs

Summary of all Effects; (Time to peak torque, peak power and peak activation for first extension during CON)
1-LOAD, 2-Dependent Measure (TOR, PWR, Act_Peak)

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	73437.7	27	7546.9	9.73	0.00016 *
2	2	300542.6	18	3359.9	89.45	0.00000 *
12	6	4530.3	54	3371.3	1.34	0.25

Tukey HSD Test
Probabilities for Post Hoc Tests
MAIN EFFECT: TIME

Time to...		{1}	{2}	{3}
		227.4 ms	201.4 ms	362.9 ms
Peak Torque	{1}		0.140	0.0001
Peak Power	{2}	0.140		0.0001
Peak Activation	{3}	0.000	0.000	

Tukey HSD Test
Probabilities for Post Hoc Tests
MAIN EFFECT: LOAD

Load		{1}	{2}	{3}	{4}
		201.6 ms	247.9 ms	295.1 ms	311.1 ms
15% 1 {1}		0.189	0.002	0.000
30% 2 {2}	0.189		0.179	0.042
45% 3 {3}	0.002	0.179		0.890
60% 4 {4}	0.000	0.042	0.890	

Summary of all Effects; (Time to Peak Power)
1-LOAD, 2-CON/PAP, 3-KICK #

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	18575.0	27	3694.2	5.03	0.0067 *
2	1	78.4	9	991.3	0.08	0.78
3	1	612.3	9	746.0	0.82	0.39
12	3	669.9	27	415.5	1.61	0.21
13	3	1100.6	27	606.8	1.81	0.17
23	1	180.6	9	775.8	0.23	0.64
123	3	1287.8	27	695.9	1.85	0.16

Activation, AEMG and M-Wave ANOVAs

Summary of all Effects; (Activation at Peak Torque)
1-LOAD, 2-CON/PAP, 3-KICK#

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	9.71E-05	27	6.49E-05	1.50	0.24
2	1	9.71E-05	9	8.89E-06	10.93	0.0091 *
3	1	1.07E-05	9	2.47E-06	4.35	0.067
12	3	1.28E-06	27	9.40E-06	0.14	0.94
13	3	1.02E-06	27	6.20E-06	0.16	0.92
23	1	6.32E-06	9	4.80E-06	1.32	0.28
123	3	2.18E-06	27	7.56E-06	0.29	0.83

Summary of all Effects; (Activation at Peak Power)
1-LOAD, 2-CON/PAP, 3-KICK#

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	1.26E-03	27	1.10E-03	1.14	0.35
2	1	9.77E-04	9	9.29E-05	10.51	0.010 *
3	1	5.97E-05	9	1.21E-04	0.49	0.50
12	3	7.21E-05	27	1.34E-04	0.54	0.66
13	3	2.49E-05	27	8.06E-05	0.31	0.82
23	1	1.85E-05	9	8.66E-05	0.21	0.65
123	3	1.71E-04	27	7.74E-05	2.21	0.11

Summary of all Effects; (Peak Activation)
1-LOAD, 2-CON/PAP, 3-KICK #

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	3	8.49E-05	27	6.56E-05	1.29	0.30
2	1	4.20E-05	9	1.13E-05	3.73	0.085
3	1	7.99E-06	9	9.16E-06	0.87	0.37
12	3	1.12E-05	27	6.00E-06	1.87	0.16
13	3	4.04E-06	27	4.21E-06	0.96	0.43
23	1	8.35E-07	9	3.05E-06	0.27	0.61
123	3	9.68E-06	27	3.34E-06	2.90	0.053

Summary of all Effects; (M-wave Amplitude)
1-LOAD, 2-CON/PAP, 3-TIME

	df	MS	df	MS		
	Effect	Effect	Error	Error	F	p-level
1	3	47.66	27	32.45	1.47	0.245
2	1	2.37	9	13.55	0.17	0.686
3	3	61.77	27	4.49	13.74	0.000012 *
12	3	12.49	27	9.19	1.36	0.276
13	9	2.73	81	2.48	1.10	0.372
23	3	1.78	27	3.08	0.58	0.634
123	9	2.14	81	2.17	0.99	0.459

Summary of all Effects; (M-wave Normalized to PRE)
1-LOAD, 2-CON/PAP, 3-TIME

	df	MS	df	MS		
	Effect	Effect	Error	Error	F	p-level
1	3	3.55%	27	3.68%	0.96	0.42
2	1	4.06%	9	4.48%	0.91	0.37
3	2	0.48%	18	0.91%	0.53	0.60
12	3	3.41%	27	2.52%	1.35	0.28
13	6	0.73%	54	0.53%	1.39	0.24
23	2	0.68%	18	1.01%	0.67	0.52
123	6	0.51%	54	0.60%	0.84	0.54

Summary of all Effects; (AEMG)
1-LOAD, 2-CON/PAP, 3-KICK#

	df	MS	df	MS		
	Effect	Effect	Error	Error	F	p-level
1	3	0.3309%	27	0.0392%	8.45	0.00040 *
2	1	0.0497%	9	0.0344%	1.44	0.26
3	1	0.0916%	9	0.0316%	2.90	0.12
12	3	0.0214%	27	0.0186%	1.15	0.35
13	3	0.0028%	27	0.0230%	0.12	0.95
23	1	0.0019%	9	0.0153%	0.13	0.73
123	3	0.0200%	27	0.0138%	1.45	0.25

Appendix 3: Raw Data

MVC PT - Absolute

Subject	Habituation						PAP			
	MVC1L	MVC2L	MVC3L	MVC1R	MVC2R	MVC3R	MVC 15%	MVC 30%	MVC 45%	MVC 60%
1	217	250	231	315	315	310	257	235	232	306
2	199	204	212	205	228	242	182	161	252	186
3	233	256	244	259	281	283	202	284	243	296
4	254	256	252	235	257	257	219	233	270	199
5	111	114	120	127	118	121	104	90	106	118
6	164	168	172	142	146	145	180	163	158	167
7	148	149	151	155	155	160	164	164	164	162
8	192	186	185	181	195	179	208	147	197	203
9	202	205	199	302	249	254	295	198	256	359
10	145	141	130	129	119	118	121	120	114	131
Mean	187	193	190	205	206	207	193	179	199	213
SD	44	51	46	70	70	70	57	58	61	81
SE	15	17	15	23	23	23	19	19	20	27

Twitch PT - Absolute

Subject	T01 15%	T02 15%	T03 15%	T04 15%	T05 15%	T06 15%	T07 15%	T08 15%	T01 30%	T02 30%	T03 30%	T04 30%	T05 30%	T06 30%	T07 30%	T08 30%
1	46.06	45.20	52.82	57.32	41.60	66.50	56.97	55.59	36.60	37.73	44.32	48.66	30.94	58.72	51.78	52.82
2	49.12	48.32	44.85	50.05	41.60	77.58	73.77	74.64	47.37	48.49	53.68	56.63	46.12	70.66	68.06	67.54
3	30.53	31.31	33.22	37.90	44.50	52.13	50.22	46.75	35.24	35.50	41.04	43.12	33.17	62.52	58.53	54.72
4	33.85	34.29	40.35	43.64	30.90	60.09	57.49	56.11	35.63	36.34	38.60	38.94	32.85	56.29	49.35	49.87
5	28.69	28.88	33.57	34.95	33.05	39.98	43.80	41.20	29.81	29.96	36.02	38.10	26.55	35.50	39.83	36.89
6	20.41	21.13	26.32	27.36	28.34	35.85	32.38	31.17	18.02	18.47	21.42	21.77	16.90	32.18	26.45	26.28
7	20.50	21.47	22.51	23.55	19.33	33.60	28.57	28.57	21.24	21.13	26.67	27.19	20.50	34.81	34.81	32.90
8	33.54	33.39	38.60	41.20	20.20	54.40	43.97	46.23	29.73	28.57	33.08	36.54	27.65	41.39	39.83	39.83
9	32.57	33.25	45.37	44.33	30.43	57.15	56.97	54.38	26.55	27.49	34.61	37.04	24.93	44.84	42.93	43.28
10	24.16	24.59	30.13	33.60	26.53	37.75	37.23	39.48	24.70	26.11	23.85	25.59	24.81	33.74	34.26	33.91
Mean	31.9	32.2	36.8	39.4	31.6	51.5	48.1	47.4	30.5	31.0	35.3	37.4	28.4	47.1	44.6	43.8
SD	9.6	9.1	9.4	10.2	8.7	14.6	13.7	13.6	8.6	8.8	9.8	10.7	8.1	13.9	12.5	12.4
SE	3.2	3.0	3.1	3.4	2.9	4.9	4.6	4.5	2.9	2.9	3.3	3.6	2.7	4.6	4.2	4.1

Twitch PT - Rel to PRE

Subject	T02 15%	T03 15%	T04 15%	T06 15%	T07 15%	T08 15%	T02 30%	T03 30%	T04 30%	T06 30%	T07 30%	T08 30%	T02 45%	T03 45%	T04 45%	T06 45%
1	-1.9%	14.7%	24.4%	59.9%	36.9%	33.6%	3.1%	21.1%	33.0%	89.8%	67.4%	70.7%	4.9%	29.3%	42.1%	58.8%
2	-1.6%	-8.7%	1.9%	86.5%	77.3%	79.4%	2.4%	13.3%	19.5%	53.2%	47.6%	46.4%	-1.1%	17.9%	27.4%	71.5%
3	2.6%	8.8%	24.1%	17.1%	12.9%	5.1%	0.7%	16.5%	22.4%	88.5%	76.5%	65.0%	2.1%	13.2%	26.0%	83.5%
4	1.3%	19.2%	28.9%	94.5%	86.1%	81.6%	2.0%	8.3%	9.3%	71.4%	50.2%	51.8%	0.2%	29.4%	39.3%	69.3%
5	0.7%	17.0%	21.8%	21.0%	32.5%	24.7%	0.5%	20.8%	27.8%	33.7%	50.0%	38.9%	6.9%	15.2%	25.4%	65.6%
6	3.5%	29.0%	34.1%	26.5%	14.3%	10.0%	2.5%	18.9%	20.8%	90.4%	56.5%	55.5%	8.5%	15.7%	25.3%	49.2%
7	4.7%	9.8%	14.9%	73.8%	47.8%	47.8%	-0.5%	25.6%	28.0%	69.8%	69.8%	60.5%	2.3%	19.1%	18.2%	71.0%
8	-0.4%	15.1%	22.8%	169.3%	117.7%	128.9%	-3.9%	11.3%	22.9%	49.7%	44.1%	44.1%	1.2%	16.2%	29.9%	68.8%
9	2.1%	39.3%	36.1%	87.8%	87.2%	78.7%	3.5%	30.4%	39.5%	79.9%	72.2%	73.6%	1.1%	25.1%	35.8%	59.9%
10	1.8%	24.7%	39.1%	42.3%	40.3%	48.8%	5.7%	-3.4%	3.6%	36.0%	38.1%	36.7%	2.3%	21.8%	36.2%	36.8%
Mean	1.3%	16.9%	24.8%	68.8%	55.3%	53.9%	1.6%	16.3%	22.7%	66.2%	57.2%	54.3%	2.8%	20.3%	30.6%	63.4%
SD	2.1%	12.9%	10.9%	48.6%	34.9%	38.4%	2.6%	9.6%	10.5%	21.8%	13.3%	13.0%	3.0%	5.9%	7.5%	13.1%
SE	0.7%	4.3%	3.6%	16.2%	11.6%	12.8%	0.9%	3.2%	3.5%	7.3%	4.4%	4.3%	1.0%	2.0%	2.5%	4.4%

Subject	T01 45%	T02 45%	T03 45%	T04 45%	T05 45%	T06 45%	T07 45%	T08 45%	T01 60%	T02 60%	T03 60%	T04 60%	T05 60%	T06 60%	T07 60%	T08 60%
1	31.18	32.70	40.33	44.32	36.66	58.20	59.76	62.36	34.56	36.02	50.22	54.20	33.84	70.31	61.82	62.86
2	42.01	41.55	49.53	53.52	43.54	74.68	74.51	71.38	50.18	50.57	53.86	58.03	48.82	74.85	75.20	72.77
3	31.18	31.83	35.30	39.29	27.27	50.05	47.79	47.96	29.15	30.13	43.99	45.37	28.30	63.21	59.05	58.19
4	35.07	35.15	45.37	48.84	34.38	58.19	57.49	59.57	35.59	35.47	40.16	46.58	34.65	57.33	54.38	52.82
5	27.18	29.06	31.31	34.09	25.72	42.59	40.68	40.33	30.20	29.79	37.23	42.08	28.73	38.44	36.02	46.76
6	21.66	23.51	25.07	27.15	19.59	29.23	28.36	28.36	20.64	21.65	27.36	29.61	20.24	37.93	35.15	33.77
7	19.59	20.04	23.33	23.16	18.61	31.83	29.58	28.71	20.92	20.56	22.47	24.37	19.13	29.75	28.19	25.59
8	29.04	29.40	33.74	37.73	29.04	49.01	43.80	45.02	29.81	29.44	35.67	42.25	27.85	48.14	36.89	45.37
9	32.52	32.87	40.68	44.15	29.99	47.96	52.30	53.69	26.95	28.75	39.48	43.99	27.16	60.78	60.26	59.92
10	26.58	27.19	32.38	36.19	26.21	35.85	38.10	39.14	25.40	25.93	29.58	33.57	23.98	35.82	35.13	35.65
	29.6	30.3	35.7	38.8	29.1	47.8	47.2	47.7	30.3	30.8	38.0	42.0	29.3	51.7	48.2	49.4
	6.5	6.0	8.4	9.4	7.6	13.8	14.3	14.2	8.6	8.6	9.9	10.4	8.5	15.8	15.8	14.7
	2.2	2.0	2.8	3.1	2.5	4.6	4.8	4.7	2.9	2.9	3.3	3.5	2.8	5.3	5.3	4.9

Subject	T07 45%	T08 45%	T02 60%	T03 60%	T04 60%	T06 60%	T07 60%	T08 60%
1	63.0%	70.1%	4.2%	45.3%	56.8%	107.8%	82.7%	85.8%
2	71.1%	63.9%	0.8%	7.3%	15.6%	53.3%	54.0%	49.1%
3	75.2%	75.9%	3.4%	50.9%	55.6%	123.4%	108.7%	105.6%
4	67.2%	73.3%	-0.3%	12.8%	30.9%	65.5%	56.9%	52.4%
5	58.2%	56.8%	-1.4%	23.3%	39.3%	33.8%	25.4%	62.8%
6	44.8%	44.8%	4.9%	32.6%	43.5%	87.4%	73.7%	66.8%
7	58.9%	54.3%	-1.7%	7.4%	16.5%	55.5%	47.4%	33.8%
8	50.8%	55.0%	-1.2%	19.7%	41.7%	72.9%	32.5%	62.9%
9	74.4%	79.0%	6.7%	46.5%	63.2%	123.8%	121.9%	120.6%
10	45.4%	49.3%	2.1%	16.5%	32.2%	49.4%	46.5%	48.7%
	60.9%	62.2%	1.7%	26.2%	39.5%	77.3%	65.0%	68.8%
	11.3%	11.9%	3.0%	16.5%	16.2%	32.0%	31.6%	27.3%
	3.8%	4.0%	1.0%	5.5%	5.4%	10.7%	10.5%	9.1%

Twitch Control PT Left Leg - Absolute

Twitch Control PT Right Leg - Absolute

Subject	PRE	5 s	20 s	30 s	40 s	50 s	2 min	3 min	4 min	5 min	PRE	5 s	20 s	30 s	40 s	50 s	2 min	3 min	4 min	5 min
	PT.T01	PT.T02	PT.T03	PT.T04	PT.T05	PT.T06	PT.T07	PT.T08	PT.T09	PT.T10	PT.T01	PT.T02	PT.T03	PT.T04	PT.T05	PT.T06	PT.T07	PT.T08	PT.T09	PT.T10
1	41.30	62.19	58.20	56.64	55.60	55.08	50.39	47.10	45.88	46.58	46.40	71.35	69.44	68.23	65.81	66.67	60.96	59.40	58.36	56.97
2	50.18	74.44	70.64	71.96	71.24	66.95	65.62				50.70	73.31	72.24	71.49	72.28	71.13	69.18			
3	28.28	53.69	47.10	45.71	44.49	44.67	41.72	39.46	37.04	39.14	30.87	62.69	58.01	55.07	50.39	49.87	46.24	42.60	39.83	39.14
4	36.60	57.16	52.65	50.91	50.74	50.39	48.14	44.67	44.32	42.41	35.96	60.78	57.15	54.55	52.65	51.61	50.05	46.93	45.03	43.29
5	27.18	49.01	43.45	42.59	41.37	40.16	38.08	35.30	34.09	32.01	32.45	47.97	45.20	43.99	43.47	42.77	39.83	38.96	37.41	36.54
6	20.33	34.09	29.75	28.71	26.80	26.28	24.20	23.51	23.85	24.20	20.55	36.02	32.90	31.34	30.13	28.57	25.98	25.63	25.46	25.63
7	21.01	28.88	29.06	28.19	27.49	27.15	25.07	24.20	23.51	22.98	20.66	32.04	29.44	28.05	27.19	27.19	24.94	25.11	23.55	23.21
8	37.83	55.25	51.26	49.53	49.18	47.79	46.40	45.36	44.32	44.84	35.17	59.57	52.30	50.39	49.35	48.32	47.97	46.06	46.06	45.03
9	30.76	54.90	53.86	49.87	48.31	48.14	41.89	41.20	38.60	38.42	32.85	66.85	63.38	61.65	58.53	56.97	49.70	46.58	43.81	43.29
10	25.62	35.99	35.13	34.78	33.39	33.39	30.44	29.92	29.60	28.36	23.66	32.73	34.29	33.08	31.69	32.38	30.48	30.13	29.27	28.05
Mean	31.9	50.6	47.1	45.9	44.9	44.0	41.2	36.7	35.7	35.4	32.9	54.3	51.4	49.8	48.1	47.5	44.5	40.2	38.8	37.9
SD	9.5	14.0	13.1	13.3	13.5	12.7	12.6	9.0	8.6	8.9	10.0	15.9	15.4	15.4	15.2	15.1	14.5	11.4	11.2	10.8
SE	3.2	4.7	4.4	4.4	4.5	4.2	4.2	3.2	3.0	3.1	3.3	5.3	5.1	5.1	5.1	5.0	4.8	4.0	4.0	3.8

Twitch Control PT Left - Relative

Twitch Control PT Right - Relative

Subject	5 s	20 s	30 s	40 s	50 s	2 min	3 min	4 min	5 min	5 s	20 s	30 s	40 s	50 s	2 min	3 min	4 min	5 min
	PT.T02	PT.T03	PT.T04	PT.T05	PT.T06	PT.T07	PT.T08	PT.T09	PT.T10	PT.T02	PT.T03	PT.T04	PT.T05	PT.T06	PT.T07	PT.T08	PT.T09	PT.T10
1	50.6%	40.9%	37.1%	34.6%	33.4%	22.0%	14.0%	11.1%	12.8%	53.8%	49.7%	47.0%	41.8%	43.7%	31.4%	28.0%	25.8%	22.8%
2	48.3%	40.8%	43.4%	42.0%	33.4%	30.8%				44.6%	42.5%	41.0%	42.6%	40.3%	36.4%			
3	89.9%	66.5%	61.6%	57.3%	58.0%	47.5%	39.5%	31.0%	38.4%	103.1%	87.9%	78.4%	63.2%	61.5%	49.8%	38.0%	29.0%	26.8%
4	56.2%	43.9%	39.1%	38.6%	37.7%	31.5%	22.0%	21.1%	15.9%	69.0%	58.9%	51.7%	46.4%	43.5%	39.2%	30.5%	25.2%	20.4%
5	80.3%	59.9%	56.7%	52.2%	47.8%	40.1%	29.9%	25.4%	17.8%	47.8%	39.3%	35.6%	34.0%	31.8%	22.7%	20.1%	15.3%	12.6%
6	67.7%	46.3%	41.2%	31.8%	29.3%	19.0%	15.6%	17.3%	19.0%	75.3%	60.1%	52.5%	46.6%	39.0%	26.4%	24.7%	23.9%	24.7%
7	37.5%	38.3%	34.2%	30.8%	29.2%	19.3%	15.2%	11.9%	9.4%	55.1%	42.5%	35.8%	31.6%	31.6%	20.7%	21.5%	14.0%	12.3%
8	46.0%	35.5%	30.9%	30.0%	26.3%	22.7%	19.9%	17.2%	18.5%	69.4%	48.7%	43.3%	40.3%	37.4%	36.4%	31.0%	31.0%	28.0%
9	78.5%	75.1%	62.1%	57.1%	56.5%	36.2%	33.9%	25.5%	24.9%	103.5%	92.9%	87.7%	78.2%	73.4%	51.3%	41.8%	33.4%	31.8%
10	40.5%	37.1%	35.8%	30.3%	30.3%	18.8%	16.8%	15.5%	10.7%	38.3%	44.9%	39.8%	33.9%	36.9%	28.8%	27.3%	23.7%	18.6%
Mean	59.5%	48.4%	44.2%	40.5%	38.2%	28.8%	23.0%	19.6%	18.6%	66.0%	56.7%	51.3%	45.9%	43.9%	34.3%	29.2%	24.6%	22.0%
SD	18.3%	13.8%	11.6%	11.1%	11.7%	10.1%	9.3%	6.7%	8.8%	22.9%	19.0%	17.9%	14.5%	13.4%	10.4%	7.1%	6.5%	6.7%
SE	6.1%	4.6%	3.9%	3.7%	3.9%	3.4%	3.3%	2.4%	3.1%	7.6%	6.3%	6.0%	4.8%	4.5%	3.5%	2.5%	2.3%	2.4%

Isotonic Peak Torque - Absolute

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
1	104.8	99.2	90.9	92.8	109.6	109.6	106.8	108.5	142.0	141.1	136.8	138.3	186.9	184.6	183.6	185.1
2	78.8	87.6	81.9	81.9	94.7	100.8	103.4	100.4	128.5	130.0	127.6	133.5	144.6	143.0	139.0	138.2
3	84.6	89.3	76.9	78.7	119.0	119.5	117.4	117.6	130.4	129.2	136.1	130.5	186.5	188.8	180.8	182.3
4	63.9	68.4	64.1	71.2	99.7	100.4	102.4	100.2	130.2	131.1	126.6	129.2	155.2	154.8	154.6	154.1
5	49.7	52.3	44.7	51.4	65.3	64.6	53.7	63.9	70.7	77.3	73.5	76.4	94.4	96.8	95.2	92.3
6	58.7	58.4	59.7	58.7	79.2	80.1	78.1	76.4	94.3	90.5	90.3	92.5	101.6	102.7	100.1	99.1
7	88.8	51.8	56.6	52.1	73.1	73.8	74.6	73.1	85.8	85.3	85.3	85.6	103.0	100.5	103.1	101.0
8	72.4	67.9	76.4	73.8	85.0	88.5	90.7	90.9	100.7	105.4	108.0	110.2	134.0	136.6	140.4	141.7
9	88.7	83.5	87.6	83.1	90.6	88.0	86.6	91.7	115.6	117.4	113.7	116.8	200.0	202.6	195.0	201.7
10	38.6	37.6	36.9	39.3	65.5	67.7	62.2	65.3	82.3	79.5	76.4	80.4	100.9	104.0	97.1	93.6
Mean	72.9	69.6	67.6	68.3	88.2	89.3	87.6	88.8	108.1	108.7	107.4	109.3	140.7	141.4	138.9	138.9
SD	20.2	19.9	18.2	17.1	18.1	18.2	20.5	18.5	24.5	24.1	24.5	23.8	40.4	40.3	38.9	41.4
SE	6.7	6.6	6.1	5.7	6.0	-6.1	6.8	6.2	8.2	-8.0	8.2	7.9	13.5	-13.4	13.0	13.8

Joint Angle at Peak Torque (deg)

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
1	108	96	107	102	101	104	102	99	102	100	96	97	99	96	95	97
2	101	99	94	104	101	100	93	101	97	99	94	94	95	102	94	94
3	103	104	105	101	101	99	98	99	96	95	98	96	96	96	96	95
4	104	105	103	106	101	98	105	101	94	98	96	95	100	103	97	99
5	113	117	110	107	107	106	95	111	105	101	103	110	104	98	97	104
6	102	102	105	98	101	104	102	100	97	96	96	95	94	95	94	96
7	94	104	102	101	99	101	101	101	96	97	97	98	95	96	96	96
8	103	106	103	104	107	98	105	101	94	97	96	97	98	94	95	94
9	109	113	95	110	103	106	102	104	103	110	97	100	96	97	96	96
10	105	102	95	95	100	103	99	98	102	99	109	110	100	97	95	95
Mean	104	105	102	103	102	102	100	102	99	99	98	99	98	97	95	97
SD	5	6	6	4	3	3	4	4	4	4	5	6	3	3	1	3
SE	2	2	2	1	1	1	1	1	1	1	2	2	1	1	0	1

Velocity at Peak Torque (deg/s)

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
1	340.7	155.4	289.4	244.6	211.9	236.0	212.6	173.7	192.8	171.6	122.1	124.6	169.1	127.2	114.6	132.6
2	232.8	210.6	137.1	262.2	185.7	167.6	92.5	173.3	157.0	171.9	68.4	96.1	105.5	173.4	85.5	90.0
3	270.2	288.6	261.5	252.6	205.0	190.5	171.1	186.1	124.9	114.4	146.1	120.6	118.2	104.4	98.8	94.4
4	205.6	235.6	205.2	259.6	176.9	134.5	220.3	189.1	61.5	138.6	92.5	98.0	87.2	115.9	78.3	67.0
5	326.6	354.3	249.2	256.4	225.7	225.7	279.3	257.1	176.5	176.3	173.5	235.9	203.2	134.0	122.0	172.7
6	230.2	238.1	275.7	132.0	195.6	211.4	184.8	178.7	119.6	118.7	90.4	100.3	99.7	117.5	89.5	121.2
7	109.5	224.9	234.0	201.4	159.2	193.4	170.2	194.8	106.6	128.5	117.4	133.8	85.3	91.0	95.2	101.5
8	260.2	285.3	265.3	269.7	244.5	162.2	253.8	193.4	128.5	145.3	152.2	155.8	124.0	92.0	119.5	94.1
9	324.8	340.3	122.6	329.5	223.3	245.1	193.1	223.8	208.4	258.8	147.2	160.0	109.4	130.0	78.4	101.7
10	301.2	246.3	136.3	154.6	161.4	203.2	143.6	136.6	183.4	155.0	221.1	236.3	149.3	131.3	101.3	92.9
Mean	260.2	257.9	217.6	236.3	198.9	197.0	192.1	190.7	145.9	157.9	133.1	146.1	125.1	121.7	98.3	106.8
SD	70.1	60.2	63.5	58.2	28.4	34.9	53.6	32.0	45.6	41.8	44.9	52.3	38.0	24.0	16.1	29.1
SE	23.4	20.1	21.2	19.4	9.5	11.6	17.9	10.7	15.2	13.9	15.0	17.4	12.7	8.0	5.4	9.7

Peak Velocity - Absolute (deg/s)

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
1	503.7	495.0	430.3	461.8	470.4	466.1	425.7	455.5	336.6	350.9	312.8	318.3	295.3	279.2	269.8	281.7
2	407.7	423.3	414.7	456.2	304.7	318.8	335.7	315.7	335.9	340.5	274.8	272.4	243.4	242.6	211.9	154.3
3	513.4	504.7	502.4	500.2	366.6	394.1	354.9	388.2	296.0	291.0	286.7	261.4	193.8	201.9	184.7	195.8
4	367.1	405.0	397.7	433.3	309.8	349.5	364.6	357.2	214.8	227.7	220.2	252.8	143.4	172.0	142.9	157.7
5	493.4	497.5	375.8	466.5	376.5	377.9	223.6	371.9	276.5	328.4	296.1	329.7	309.6	322.3	303.3	293.6
6	449.2	438.6	427.3	442.8	358.7	360.8	340.8	350.1	256.4	237.4	221.5	241.8	245.3	244.3	202.5	211.2
7	400.9	408.3	417.4	396.1	317.7	337.7	337.0	337.0	266.3	269.7	257.0	265.6	210.6	200.9	209.7	211.8
8	501.8	483.4	485.1	482.0	371.0	382.7	337.7	377.4	244.9	283.9	305.7	311.0	230.8	231.6	229.2	234.8
9	468.1	437.6	459.7	470.2	443.8	438.5	394.9	414.5	333.6	352.9	277.2	342.6	223.3	248.2	191.9	224.6
10	484.9	481.8	438.9	472.8	347.1	359.2	326.6	333.6	338.4	351.7	321.5	342.6	266.6	272.9	222.1	217.8
Mean	459.0	457.5	434.9	458.2	366.6	378.5	344.2	370.1	289.9	303.4	277.4	293.8	236.2	241.6	216.8	218.3
SD	50.9	38.9	38.6	28.8	54.5	45.1	52.5	41.6	44.9	48.1	35.4	38.9	48.5	43.6	44.6	45.2
SE	17.0	13.0	12.9	9.6	18.2	15.0	17.5	13.9	15.0	16.0	11.8	13.0	16.2	14.5	14.9	15.1

Peak Power (Watts) - Absolute

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
1	722.2	684.1	528.9	558.8	736.9	724.6	637.7	708.1	683.3	709.4	626.2	606.1	854.1	809.3	760.9	795.4
2	412.2	445.8	429.6	493.7	410.4	435.7	462.3	439.0	607.4	621.1	463.6	478.4	556.0	528.1	456.1	333.3
3	550.1	589.0	502.3	478.7	605.9	657.3	571.7	626.2	583.0	563.6	594.4	509.2	540.2	541.8	516.5	543.2
4	332.7	381.8	356.6	416.0	432.1	471.8	502.3	510.8	411.6	456.5	426.9	491.3	376.5	442.6	365.2	399.8
5	304.6	338.3	216.5	298.0	320.9	323.5	348.7	332.6	260.9	352.6	307.3	334.4	427.0	456.1	404.6	401.4
6	322.4	323.0	308.1	320.9	386.6	389.2	356.8	373.2	362.0	322.5	311.6	316.0	358.8	370.2	286.8	302.3
7	315.0	268.8	304.8	274.9	316.2	328.1	333.2	319.1	309.7	310.6	300.4	322.3	332.6	314.8	314.9	326.8
8	471.5	424.2	469.7	441.2	447.1	463.9	463.2	469.3	363.8	428.9	469.6	506.2	470.4	464.3	449.4	497.2
9	561.8	529.9	521.4	539.8	542.7	515.2	444.0	490.5	549.6	568.8	457.7	568.8	692.4	775.1	581.1	685.7
10	251.2	239.3	214.0	242.2	305.6	332.8	287.0	288.0	390.3	410.5	356.4	424.7	399.0	389.0	319.3	315.3
Mean	424.4	422.4	385.2	406.4	450.4	464.2	440.7	455.7	452.2	474.4	431.4	455.7	500.7	509.1	445.5	460.0
SD	149.4	143.0	121.6	114.7	140.1	137.3	111.3	135.9	142.4	135.3	115.6	103.1	165.6	164.3	145.0	169.5
SE	49.8	47.7	40.5	38.2	46.7	45.8	37.1	45.3	47.5	45.1	38.5	34.4	55.2	54.8	48.3	56.5

Work to Peak Power (Joules) - Absolute

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
1	57.2	61.7	41.6	44.1	77.1	79.2	75.1	75.3	72.5	75.0	73.2	53.0	85.4	80.4	80.0	66.6
2	31.0	33.0	41.8	38.0	36.8	39.8	49.3	48.1	46.9	62.5	62.7	46.0	49.3	47.5	51.3	43.4
3	73.5	73.8	48.7	49.7	47.1	60.4	40.9	49.3	55.3	77.8	56.0	55.9	34.7	46.5	51.4	52.5
4	26.7	28.0	36.0	31.3	38.4	52.8	48.9	50.0	58.8	50.5	58.0	50.0	66.8	73.0	50.7	72.1
5	23.2	24.7	17.5	21.7	31.5	33.0	31.3	26.9	23.5	37.4	36.1	42.7	50.6	46.1	39.6	45.0
6	22.0	22.9	22.4	22.4	32.5	29.6	37.2	32.2	38.7	30.9	34.3	41.0	38.9	33.4	31.1	36.1
7	18.7	23.8	25.2	23.0	31.4	34.9	38.3	25.1	25.2	27.0	33.2	33.4	39.7	34.8	41.8	41.3
8	31.0	34.7	29.2	29.4	41.1	44.3	36.8	47.5	41.8	40.8	39.9	43.3	50.3	50.4	28.6	43.7
9	34.0	32.5	33.8	33.6	37.8	66.6	58.6	56.6	51.2	57.3	39.3	56.9	66.6	59.6	65.1	75.4
10	13.4	18.8	16.1	12.0	24.2	34.5	32.8	32.4	44.1	36.1	44.2	46.1	38.1	58.9	29.4	45.1
Mean	33.1	35.4	31.2	30.5	39.8	47.5	44.9	44.3	45.8	49.5	47.7	46.8	52.0	53.1	46.9	52.1
SD	18.5	18.0	11.0	11.4	14.5	16.6	13.5	15.5	14.9	18.1	13.8	7.3	16.2	15.2	16.5	14.0
SE	6.2	6.0	3.7	3.8	4.8	5.5	4.5	5.2	5.0	6.0	4.6	2.4	5.4	5.1	5.5	4.7

Time to Peak Power (ms)

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
1	173	191	157	179	228	217	228	203	217	217	233	190	173	188	191	172
2	174	141	168	147	226	191	156	214	212	198	217	159	190	183	212	208
3	154	205	195	163	157	194	172	170	215	266	184	205	129	150	120	158
4	201	212	221	169	198	220	188	208	275	252	255	207	344	336	318	495
5	166	166	202	175	211	205	173	221	268	211	259	261	214	218	222	224
6	160	161	145	160	168	177	219	188	208	221	211	210	196	179	222	196
7	154	177	177	188	173	206	247	153	176	194	197	200	208	217	228	239
8	157	190	145	155	196	186	173	197	227	216	171	198	204	175	124	159
9	168	139	151	149	179	222	234	215	219	193	197	197	216	172	170	196
10	120	169	156	140	163	237	254	235	218	197	245	240	165	252	204	269
Mean	162	175	171	162	190	205	204	200	223	216	217	207	204	207	201	231
SD	20	25	26	15	26	19	36	25	29	25	30	28	56	54	57	99
SE	7	8	9	5	9	6	12	8	10	8	10	9	19	18	19	33

Time to Peak Activation (ms)

Subject	CON1 15%			CON1 30%			CON1 45%			CON1 60%		
	Initial TOR	Final ACT	Time ms	Initial TOR	Final Pt	Time ms	Initial TOR	Final Pt	Time ms	Initial TOR	Final Pt	Time ms
TJ	196	654	229	106	809	352	162	983	411	190	350	80
AB	125	713	294	114	615	251	47	758	356	137	792	328
PL	113	782	335	131	938	404	72	775	352	58	1167	555
TW	163	429	133	93	1044	476	89	1048	480	23	1563	770
BO	118	798	340	107	724	309	81	876	398	95	1104	505
JD	129	778	325	144	917	387	19	967	474	135	985	425
LG	172	671	250	147	799	326	140	571	216	33	1004	486
JC	136	837	351	196	811	308	168	1001	417	169	906	369
KM	147	665	259	103	522	210	81	929	424	133	1058	463
KH	106	613	254	38	790	376	68	933	433	141	976	418
Mean			277			340			396			440
SD			67			77			76			175
SE			22			26			25			58

Twitch M-Wave Amplitude - Absolute

Subject	T01 15%	T02 16%	T03 16%	T04 16%	T05 16%	T06 16%	T07 16%	T08 16%	T09 16%	T10 16%	T01 30%	T02 30%	T03 30%	T04 30%	T05 30%	T06 30%	T07 30%	T08 30%	T09 30%	T10 30%	T01 45%	T02 45%	T03 45%	T04 45%	T05 45%	T06 45%	T07 45%	T08 45%	T09 45%	T10 45%	T01 60%	T02 60%	T03 60%	T04 60%	T05 60%	T06 60%	T07 60%	T08 60%	T09 60%	T10 60%
1	19.4	22.5	23.4	24.3	19.8	25.2	25.5	25.4	18.2	20.3	21.4	21.5	18.4	22.6	23.1	23.9	18.7	20.7	20.8	21.3	16.8	20.2	21.4	20.9	20.9	23.1	24.0	25.2	21.1	24.8	25.5	25.6								
2	21.7	25.0	23.3	23.3	22.2	26.8	24.8	24.9	23.3	25.3	22.0	23.1	22.7	21.8	14.9	15.8	24.4	26.2	23.3	15.9	24.7	27.8	28.9	27.7	24.9	27.6	27.7	27.8	24.5	27.5	28.6	28.4								
3	17.5	17.4	15.0	19.7	19.3	23.9	23.2	23.2	19.5	21.5	22.2	22.6	17.5	22.5	24.0	23.9	17.8	17.8	18.5	20.1	17.9	23.1	22.1	22.3	19.5	21.3	22.6	22.5	19.9	23.6	23.5	21.9								
4	15.5	17.2	17.7	18.0	15.5	19.3	19.3	19.2	21.8	23.8	22.3	21.1	20.6	22.3	22.2	23.5	17.6	16.6	19.7	20.0	17.7	20.2	20.2	20.7	20.8	21.8	23.7	24.0	20.3	23.8	23.8	23.5								
5	14.0	15.6	14.3	13.5	14.3	14.6	16.5	13.0	14.4	16.7	10.6	12.6	14.5	9.0	13.4	11.2	16.7	18.6	18.8	18.9	15.9	18.7	19.4	19.3	17.9	20.0	19.0	20.9	18.1	12.8	11.8	20.2								
6	15.1	16.6	16.9	17.2	15.1	18.2	17.5	17.5	14.6	16.2	15.7	16.0	14.0	17.3	16.3	16.4	18.1	19.9	20.0	20.4	18.8	11.6	14.9	17.1	17.6	19.0	19.8	20.1	17.9	21.2	20.5	20.2								
7	15.3	16.9	17.4	17.8	15.6	18.6	18.1	18.4	14.9	16.4	17.0	17.4	15.1	18.2	17.8	18.3	13.4	14.5	14.9	14.9	13.5	15.9	15.2	15.3	16.6	17.5	17.6	18.6	17.3	19.3	17.9	18.8								
8	25.8	28.8	27.5	29.3	26.6	28.9	25.5	28.1	22.6	25.2	24.5	24.7	21.9	20.7	21.5	18.7	26.8	29.4	30.1	30.4	26.7	33.1	32.2	32.2	26.8	29.4	29.7	28.6	26.0	17.2	19.9	22.7								
9	15.2	16.9	16.7	11.0	13.4	13.0	17.1	16.9	18.2	19.5	19.7	20.0	17.8	19.6	20.6	20.1	16.1	17.7	17.4	17.8	16.0	13.8	17.5	18.4	13.7	15.3	15.0	14.8	14.6	14.1	14.8	16.9								
10	14.3	16.2	16.5	16.6	14.3	17.2	15.4	15.4	15.0	16.2	10.8	14.1	15.4	19.0	18.3	16.4	16.1	17.5	15.1	16.5	15.9	16.9	16.8	17.5	15.1	16.4	16.4	16.3	14.5	17.9	15.3	16.2								
Mean	17.4	19.3	18.9	18.1	17.8	20.6	20.3	20.3	18.3	20.1	18.6	19.3	17.8	19.3	19.2	18.8	18.6	20.2	19.9	19.8	16.4	20.3	20.9	21.2	19.4	21.1	21.5	21.9	19.4	20.2	20.2	21.4								
SD	3.8	4.8	4.3	6.4	4.3	6.3	4.0	4.8	3.6	3.7	4.9	4.1	3.1	4.1	3.6	4.2	4.0	4.4	4.4	4.3	4.1	6.4	6.7	6.2	4.1	4.6	4.8	4.6	3.8	4.8	6.3	3.8								
SE	1.3	1.6	1.4	1.8	1.4	1.8	1.3	1.6	1.2	1.2	1.8	1.4	1.0	1.4	1.2	1.4	1.3	1.6	1.6	1.4	2.1	1.9	1.7	1.4	1.6	1.6	1.6	1.3	1.6	1.8	1.3									

Twitch M-Wave Amplitude - Relative

Subject	T02 16%	T03 16%	T04 16%	T05 16%	T06 16%	T07 16%	T08 16%	T09 16%	T10 16%	T02 30%	T03 30%	T04 30%	T05 30%	T06 30%	T07 30%	T08 30%	T02 45%	T03 45%	T04 45%	T05 45%	T06 45%	T07 45%	T08 45%	T02 60%	T03 60%	T04 60%	T05 60%	T06 60%	T07 60%	T08 60%
1	15.92%	20.31%	25.03%	27.13%	28.74%	28.20%	11.52%	17.50%	18.37%	22.82%	25.04%	29.89%	10.80%	11.15%	13.65%	19.85%	27.41%	24.18%	10.25%	14.65%	20.38%	17.46%	21.00%	21.19%						
2	15.27%	7.83%	7.83%	20.83%	11.64%	12.24%	9.60%	-5.57%	-0.83%	-3.93%	-34.37%	-30.19%	7.52%	-4.56%	-34.74%	12.55%	17.23%	12.07%	10.94%	11.48%	11.96%	12.02%	16.51%	15.65%						
3	-0.62%	-14.48%	12.61%	23.45%	20.49%	20.49%	10.71%	14.13%	16.05%	28.26%	37.00%	36.63%	0.00%	3.64%	12.88%	28.75%	23.06%	24.47%	9.13%	16.04%	15.70%	18.48%	17.87%	9.72%						
4	10.61%	13.95%	15.66%	24.25%	24.60%	23.74%	9.12%	2.49%	-3.05%	8.24%	7.98%	14.04%	11.08%	11.91%	13.58%	14.48%	14.48%	17.42%	4.59%	13.62%	14.89%	17.14%	17.72%	15.70%						
5	11.13%	2.23%	-3.83%	2.33%	15.96%	-8.96%	15.92%	-26.01%	-12.00%	-38.18%	-7.77%	-23.34%	11.26%	12.54%	13.25%	18.14%	22.07%	21.90%	11.46%	6.18%	16.89%	-29.06%	-34.88%	11.68%						
6	9.95%	11.98%	13.66%	20.08%	15.51%	15.68%	10.76%	7.42%	9.31%	22.94%	16.13%	16.80%	9.84%	10.50%	12.84%	-38.22%	-20.65%	-8.81%	7.98%	12.44%	14.10%	18.60%	14.43%	12.58%						
7	10.15%	13.82%	16.33%	19.59%	16.42%	18.30%	10.03%	13.79%	16.47%	20.36%	18.07%	20.89%	8.46%	11.64%	11.34%	17.81%	12.71%	13.40%	5.43%	5.75%	11.82%	11.94%	3.87%	9.02%						
8	11.51%	6.50%	13.35%	8.55%	-4.31%	5.46%	11.26%	8.51%	9.22%	-5.55%	-1.99%	-14.66%	9.95%	12.38%	13.69%	23.97%	20.69%	20.64%	9.72%	10.77%	6.78%	-33.67%	-23.28%	-12.72%						
9	11.55%	10.14%	-27.68%	-2.90%	27.61%	26.11%	7.01%	8.10%	9.58%	9.97%	15.33%	12.95%	9.70%	8.22%	13.50%	9.03%	14.74%	11.46%	9.42%	8.25%	-3.46%	1.94%	16.30%							
10	13.33%	15.19%	16.03%	21.00%	7.91%	15.01%	8.28%	-28.11%	-5.88%	23.05%	18.42%	6.13%	9.14%	-5.95%	2.59%	19.08%	6.00%	10.45%	8.65%	9.00%	8.21%	23.86%	5.81%	12.13%						
Mean	10.8%	8.7%	8.9%	16.3%	16.6%	16.6%	10.3%	1.2%	6.7%	8.8%	9.4%	6.9%	8.0%	7.1%	7.0%	10.3%	13.2%	16.0%	9.0%	10.9%	12.9%	6.3%	4.1%	11.1%						
SD	4.6%	9.6%	14.8%	10.0%	9.8%	11.0%	2.4%	16.3%	10.6%	20.2%	20.0%	22.6%	3.3%	7.1%	16.0%	20.4%	13.6%	8.9%	2.4%	3.4%	4.3%	20.7%	16.6%	9.1%						
SE	1.6%	3.2%	4.9%	3.3%	3.3%	3.7%	0.8%	6.4%	3.6%	6.7%	6.7%	7.6%	1.1%	2.4%	6.0%	6.8%	4.6%	3.3%	0.8%	1.1%	1.4%	6.9%	6.3%	3.0%						

Activation at Peak Torque - Normalized to M-Wave

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
TJ	8.85%	6.79%	5.23%	3.94%	9.17%	10.75%	9.63%	8.83%	6.64%	6.32%	6.54%	6.30%	8.80%	8.57%	8.55%	9.47%
AB	4.54%	5.13%	3.97%	5.18%	3.53%	3.16%	3.65%	4.18%	6.23%	5.77%	4.46%	4.30%	5.79%	5.05%	4.44%	3.60%
PL	7.49%	7.24%	9.49%	6.57%	2.14%	2.01%	2.21%	3.45%	11.56%	10.66%	10.66%	8.65%	10.27%	8.99%	7.13%	
TW	8.65%	7.16%	8.51%	8.53%	9.61%	7.28%	9.45%	8.61%	7.13%	8.60%	6.81%	5.41%	8.71%	10.48%	9.46%	9.48%
BO	5.31%	6.37%	4.43%	4.38%	6.68%	5.87%	3.74%	4.81%	6.43%	6.43%	7.67%	7.34%	5.79%	5.00%	5.00%	5.37%
JD	5.98%	6.96%	7.02%	4.57%	12.67%	12.73%	10.86%	11.43%	8.90%	6.67%	6.91%	7.08%	11.86%	8.67%	8.58%	8.49%
LG	5.12%	5.07%	4.03%	4.85%	7.03%	8.08%	7.57%	5.56%	8.55%	7.27%	6.54%	7.75%	7.34%	5.32%	7.28%	6.96%
JC	8.56%	4.85%	5.58%	6.27%	8.09%	6.55%	9.67%	9.19%	7.52%	9.39%	7.96%	8.73%	8.72%	8.62%	8.89%	10.53%
KM	7.88%	7.64%	7.42%	8.32%	10.65%	7.35%	7.36%	7.36%	7.01%	9.42%	6.90%	6.87%	10.40%	10.55%	6.10%	8.08%
KH	5.80%	4.48%	1.96%	4.13%	2.88%	4.35%	2.12%	2.31%	4.73%	4.48%	4.88%	4.93%	5.69%	4.39%	4.56%	4.23%
Mean	6.8%	6.2%	5.8%	5.7%	7.2%	6.8%	6.6%	6.6%	7.5%	7.5%	6.9%	6.7%	8.1%	7.7%	7.2%	7.3%
SD	1.6%	1.2%	2.3%	1.7%	3.5%	3.3%	3.4%	2.9%	1.9%	1.9%	1.7%	1.5%	2.1%	2.5%	2.0%	2.3%
SE	0.5%	0.4%	0.8%	0.6%	1.2%	1.1%	1.1%	1.0%	0.6%	0.6%	0.6%	0.5%	0.7%	0.8%	0.7%	0.8%

Activation at Peak Power - Normalized to M-Wave

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
TJ	9.51%	7.26%	6.01%	4.74%	11.21%	10.73%	11.41%	9.63%	6.19%	6.74%	8.58%	6.39%	8.04%	9.24%	9.67%	6.77%
AB	5.18%	5.63%	4.31%	5.90%	4.60%	3.52%	4.90%	4.87%	7.25%	6.69%	6.10%	6.11%	6.13%	5.20%	5.25%	4.27%
PL	9.54%	11.05%	10.32%	10.16%	2.69%	2.96%	2.67%	4.25%	12.12%	12.61%	10.01%	10.68%	7.70%	10.27%	8.87%	7.47%
TW	7.91%	7.68%	8.27%	8.97%	9.96%	10.25%	10.60%	9.76%	10.17%	7.99%	8.90%	6.63%	8.90%	11.55%	8.81%	10.13%
BO	5.65%	6.45%	4.57%	5.08%	6.85%	5.60%	4.98%	5.09%	6.45%	7.29%	9.44%	7.52%	5.94%	6.86%	7.19%	6.79%
JD	7.41%	7.94%	7.90%	6.07%	12.95%	12.95%	11.03%	12.40%	9.54%	8.19%	10.27%	9.53%	11.23%	10.41%	10.54%	9.60%
LG	6.50%	5.59%	4.88%	5.60%	8.64%	8.48%	8.05%	6.01%	8.01%	7.63%	8.05%	8.25%	7.32%	6.76%	7.21%	6.45%
JC	9.83%	7.06%	6.85%	8.13%	8.76%	9.98%	10.91%	10.91%	9.42%	11.74%	10.48%	10.47%	9.22%	10.96%	9.29%	11.48%
KM	8.62%	7.59%	8.14%	8.41%	10.47%	7.40%	5.79%	7.27%	7.95%	9.27%	7.52%	7.75%	11.92%	11.51%	7.97%	8.93%
KH	6.16%	5.63%	3.90%	5.64%	3.71%	4.41%	3.89%	3.42%	6.15%	5.38%	5.61%	5.51%	5.93%	5.13%	5.53%	4.03%
Mean	7.6%	7.2%	6.5%	6.9%	8.0%	7.6%	7.4%	7.4%	8.3%	8.4%	8.5%	7.9%	8.2%	8.8%	8.0%	7.6%
SD	1.7%	1.6%	2.1%	1.9%	3.4%	3.4%	3.4%	3.1%	2.0%	2.3%	1.7%	1.8%	2.1%	2.6%	1.7%	2.4%
SE	0.6%	0.5%	0.7%	0.6%	1.1%	1.1%	1.1%	1.0%	0.7%	0.8%	0.6%	0.6%	0.7%	0.9%	0.6%	0.8%

Peak Activation - Normalized to M-Wave

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
TJ	10.08%	7.31%	6.20%	5.13%	13.39%	11.30%	11.61%	9.94%	7.21%	8.01%	9.15%	7.53%	9.49%	9.62%	9.96%	10.26%
AB	5.46%	5.64%	4.71%	5.99%	4.60%	5.20%	5.12%	5.01%	7.33%	6.81%	6.66%	6.66%	6.13%	5.66%	5.56%	5.20%
PL	10.00%	11.12%	10.43%	10.22%	3.06%	4.15%	3.72%	4.84%	12.68%	12.95%	10.92%	12.64%	8.79%	11.85%	10.24%	9.21%
TW	9.94%	10.10%	9.69%	9.43%	10.02%	11.78%	12.66%	11.00%	10.40%	10.37%	9.58%	7.90%	10.74%	11.63%	10.90%	10.66%
BO	7.34%	7.28%	5.33%	6.59%	8.02%	6.29%	7.53%	6.87%	7.03%	8.28%	9.46%	8.02%	8.09%	8.35%	8.82%	8.71%
JD	11.06%	8.52%	9.32%	6.73%	14.15%	13.08%	13.00%	12.94%	9.65%	9.70%	10.85%	10.02%	12.79%	11.91%	11.12%	11.06%
LG	7.39%	5.67%	5.08%	5.70%	9.36%	8.69%	8.34%	6.25%	8.58%	8.97%	8.70%	9.35%	7.46%	7.10%	7.30%	6.96%
JC	10.73%	9.12%	9.67%	10.36%	10.99%	11.75%	12.50%	12.81%	11.66%	11.74%	12.43%	12.45%	9.25%	12.74%	12.07%	13.13%
KM	9.91%	9.46%	8.65%	8.92%	10.69%	8.26%	7.70%	7.50%	8.85%	9.42%	9.05%	8.17%	12.45%	11.81%	8.54%	8.96%
KH	6.86%	5.71%	4.77%	5.95%	4.11%	4.49%	4.35%	4.64%	6.47%	6.65%	6.39%	7.02%	6.33%	5.24%	6.38%	4.26%
Mean	8.9%	8.0%	7.4%	7.5%	8.8%	8.5%	8.7%	8.2%	9.0%	9.3%	9.3%	9.0%	9.2%	9.6%	9.1%	8.8%
SD	1.9%	2.0%	2.4%	2.0%	3.8%	3.4%	3.6%	3.2%	2.1%	2.0%	1.8%	2.1%	2.3%	2.8%	2.2%	2.7%
SE	0.6%	0.7%	0.8%	0.7%	1.3%	1.1%	1.2%	1.1%	0.7%	0.7%	0.6%	0.7%	0.8%	0.9%	0.7%	0.9%

AEMG Normalized to M-wave (%)

Subject	CON1 15%	CON2 15%	PAP1 15%	PAP2 15%	CON1 30%	CON2 30%	PAP1 30%	PAP2 30%	CON1 45%	CON2 45%	PAP1 45%	PAP2 45%	CON1 60%	CON2 60%	PAP1 60%	PAP2 60%
1	6.0%	3.6%	3.2%	2.3%	4.8%	3.3%	3.4%	2.9%	4.0%	4.1%	3.6%	3.1%	7.3%	6.3%	5.2%	4.6%
2	2.4%	2.1%	2.1%	2.8%	2.7%	2.4%	2.6%	2.2%	3.4%	3.1%	3.2%	3.3%	3.1%	3.5%	4.5%	3.5%
3	6.1%	6.4%	7.0%	5.0%	4.4%	4.0%	3.7%	4.4%	5.6%	5.2%	4.3%	4.2%	6.2%	7.2%	5.4%	4.4%
4	4.8%	3.8%	4.2%	4.1%	6.3%	5.1%	4.5%	4.7%	3.9%	3.6%	3.9%	2.8%	4.9%	5.0%	4.6%	4.1%
5	4.0%	3.6%	2.4%	3.3%	4.6%	3.6%	3.4%	3.8%	3.8%	3.7%	5.9%	6.8%	5.5%	9.5%	6.1%	7.7%
6	6.7%	4.9%	5.5%	3.5%	5.9%	4.8%	4.3%	4.1%	5.8%	4.3%	6.0%	5.3%	9.4%	5.8%	5.8%	5.7%
7	3.8%	3.0%	2.5%	2.9%	4.7%	4.1%	3.8%	2.9%	4.8%	4.3%	3.8%	4.2%	6.5%	4.1%	4.8%	4.6%
8	6.8%	4.1%	4.5%	4.7%	4.9%	4.7%	6.0%	4.9%	8.3%	8.3%	8.3%	8.6%	7.9%	10.5%	9.0%	12.6%
9	4.1%	3.9%	3.3%	5.0%	6.7%	6.1%	2.7%	3.5%	2.7%	2.9%	2.6%	2.2%	5.6%	4.8%	2.9%	3.4%
10	4.4%	3.1%	2.5%	3.0%	3.8%	3.6%	3.5%	3.4%	4.3%	4.2%	6.6%	5.1%	4.8%	3.2%	17.1%	3.1%
Mean	4.9%	3.9%	3.7%	3.7%	4.9%	4.2%	3.8%	3.7%	4.7%	4.4%	4.8%	4.6%	6.1%	6.0%	6.6%	5.4%
SD	1.4%	1.2%	1.6%	1.0%	1.2%	1.0%	1.0%	0.9%	1.6%	1.5%	1.8%	2.0%	1.8%	2.5%	4.0%	2.9%
SE	0.5%	0.4%	0.5%	0.3%	0.4%	0.3%	0.3%	0.3%	0.5%	0.5%	0.6%	0.7%	0.6%	0.8%	1.3%	1.0%