Ballistic elbow extension actions in karate-trained and control subjects

BALLISTIC ELBOW EXTENSION ACTIONS IN KARATE-TRAINED AND CONTROL SUBJECTS: AGONIST PREMOVEMENT DEPRESSION (PMD) AND MOVEMENT PERFORMANCE

BY

E. PAUL ZEHR, B.P.E.

A THESIS

Submitted to the School of Graduate Studies

in partial fulfillment of the Requirements for the degree Master of Science

McMaster University

MASTER OF SCIENCE (1993) McMASTER UNIVERSITY
(Human Biodynamics) Hamilton, Ontario

TITLE: Ballistic elbow extension actions in karate-trained and control subjects: agonist premovement depression (PMD) and movement performance

AUTHOR: E. Paul Zehr, B.P.E. (McMaster University)

SUPERVISOR: Dr. D.G. Sale

NUMBER OF PAGES: vii, 78

Acknowledgments

I would like to thank my parents, Woody and Marlene, my brother Peter and my sister Pat and their families for their unconditional love and support during my years at McMaster. I would also like to thank my fiancée Lori for her love, inspiration and encouragement during the rather stressful time when both of us were busy writing up our theses.

This thesis could not have been completed without the help, support, and suggestions of my advisor Digby Sale, who always allowed me to pursue my research interests in "Eastern mysticism", and Jim Dowling, who was always there to lend a helping hand (and talk about hockey!) when I needed it most. I thank also John Moroz for his technical assistance and for helping me in my efforts to mold some of my purchases from Beaver Lumber and Canadian Tire into a useful scientific instrument.

I would like to thank all the Human Biodynamics grad students, especially Mary's Kids Gr. 1 and Gr. 2, for the good memories. In particular I want to thank my friends Marty Gibala, Jennifer O'Brien, and Kerry Dyson for always making that coffee run to the cafeteria and for listening to me down-load my frustrations!

Finally, this thesis is dedicated to all the Chito-Ryu karateka who assisted me in my undergraduate and graduate work by serving as willing subjects for my research projects. Chikara, kanarazu, tasu!

Foreword:

This thesis has been written in a format suitable for publication. The review of literature (Section 1) entitled "Ballistic movement: motor control and muscle activation" was written for publication in CANADIAN JOURNAL OF APPLIED PHYSIOLOGY. The paper (Section 2) "Ballistic elbow extension actions in karate-trained and control subjects: agonist PMD and movement performance" was written in the format for MEDICINE AND SCIENCE IN SPORTS AND EXERCISE. The differing referencing and citation styles utilized in the two papers reflect the formats required for the two publications.

TABLE OF CONTENTS

Ballistic movement: motor control and muscle activation

Running head: Ballistic movement

E. P. Zehr and D.G. Sale
Department of Kinesiology
McMaster University

Address correspondence to:
D.G. Sale
Department of Kinesiology
McMaster University
Hamilton, Ontario, CANADA
L8S 4K1

Summary

Ballistic movements have been shown to be controlled differently by the central nervous system than slow, ramp actions. It has been suggested that the cerebellum is involved primarily with ballistic actions, while the basal ganglia primarily control slower movements. These command and control differences have been shown to manifest in unique ways at the neuromuscular level. Ballistic actions evidence high firing rates, brief contraction times, and high rates of force development. A characteristic triphasic agonist-antagonist-agonist burst pattern presents itself during ballistic movement, wherein the amount and intensity of antagonist co-activation is variable. In conditions of low-grade tonic muscular activity, a premovement depression (PMD; or silent period, PMS) can occur in both agonist and antagonist muscles prior to ballistic contraction. The agonist PMD period may serve to potentiate the force and velocity of the following contraction. A selective activation of fast twitch motor units may occur in ballistic contractions under certain movement conditions. Finally, high velocity, ballistic training induces specific neuromuscular adaptations that are representative of the underlying neurophysiological mechanisms that subserve ballistic movement.

Key words: Ballistic, motor control, muscle activation, training adaptation

Introduction

Movement, or the muscular actions subservient to movement, can be classified in many different ways. A movement may be done slowly and therefore under continuous control with afferent input, or it may be executed rapidly without reference to feedback and consequent modification. These two movement classes have been termed *ramp* and *ballistic*, respectively. *Ramp* movements are executed in open-loop fashion and involve peripheral sensory feedback, while *ballistic* movements are preprogrammed and run off closed-loop (Desmedt & Godaux, 1979; Schmidt, 1988).

The present review will focus on ballistic muscular action, the initiation and control of ballistic movement, muscle activation of ballistic actions as recorded through electromyography (EMG), and will conclude with a brief treatment concerning potential neuromuscular adaptation to high velocity ballistic action training. The organization of this paper follows a logical progression from central nervous system control to peripheral neuromuscular manifestation/adaptation. As such, the first section will deal with the anatomical and neurophysiological locus of ballistic movement control. Subsequent sections will address rate gradation and firing pattern, characteristic agonist-antagonist activation, agonist (Ag) and antagonist (Ant) co-activation, agonist pre-movement depression (PMD), and potential selective motor unit activation during ballistic contractions. The final section will briefly address potential and actual peripheral neuromuscular adaptations to ballistic and high velocity training.

[A] Ballistic motor control

It has been generally accepted that the basal ganglia and the cerebellar cortex subserve different functions in the initiation and control of ramp and ballistic movements (Desmedt & Godaux, 1979). Kornhuber (1971) asserted that the basal ganglia primarily function in the generation of slow ramp movements while the cerebellum is involved largely in the pre-programming and initiation of fast ballistic movements.

Involvement of the cerebellum in voluntary movement initiation was clearly shown by Holmes (1917). This research demonstrated that lesions of the lateral cerebellar lobe induced delays in the performance of arm flexion movements in gunshot-wounded patients and acts as the partial basis for the view that the lateral cerebellum serves as a movement programmer (Brooks, 1975). The observation that patients suffering from cerebellar cortex atrophy evidence a characteristic dysfunction in the performance of ballistic eye movements (saccades) (Kornhuber, 1968; cited in Desmedt & Godaux, 1978a) has also served to support a link between cerebellar activity and ballistic movement. Recently Alfonsi et al. (1992) examined reflex activity during the performance of isotonic ballistic and isometric contractions in patients with different cerebellar ataxias and concluded that the cerebellum may play a role in modulating long-latency reflexes and thus motor control during ballistic movement. The discharge activity of monkey cerebellar Purkinje cells has been studied (Ivry et al., 1988; Mano & Yamamoto, 1980; Thach, 1968) under conditions of ballistic and rapidly alternating arm and wrist tracking movements. This research has tended to support the assertion of a specialized role of the cerebellum in the performance of rapid, ballistic movements. This specialized role, however, may not extend to movement initiation but rather to movement control during skilled, ballistic movement

(Butler et al. 1992).

Evidence for the involvement of the basal ganglia in ramp movements has primarily come from observations of Parkinson patients. Parkinson's disease itself is generally considered to result from a breakdown of the dopamine-mediated nigrostriatal interconnection within the basal ganglia of the cerebrum (Newman & Calne, 1986). The sum effect of the morphological changes in the basal ganglia is a characteristic Parkinsonian prolonged choice reaction time and slow movement initiation difficulty (Denny-Brown, 1962; Martin, 1967: Lee, 1987). As well, the anatomical connections between the basal ganglia and motor cortex suggest a special role in movement initiation (see Figure 1 in Brooks, 1975), serving to support Kornhuber's assertion (1974) that the basal ganglia are primarily involved in slow ramp movements.

DeLong and Strick (1974) examined the activity of basal ganglia, cerebellum and motor cortex neurons during slow and rapid arm movements in the monkey. These researchers found that, of observed units, 45% in the putamen, 17% in the globus pallidus, and less than 3% in the cerebellum were found to discharge preferentially during ramp movements. In contrast, less than 10% of putamen units were preferentially ballistic-related. Units in the motor cortex, as might be expected, discharged in equal proportions to ballistic and ramp movements. On the basis of their results, Delong and Strick concluded that the observed ramp-related unit activity reflected slow-movement control in the basal ganglia. It should be mentioned, though, that many basal ganglia neurons discharged during both types of movement; therefore, the basal ganglia cannot be considered to be solely involved in slow, ramp movements.

Kornhuber (1971) has further established a model which attempts to explain the activities and connections of the cerebellum and basal ganglia during voluntary movement. There exist no direct cortical projections to connect the association cortex (temporal,

occipital, and parietal lobes) with the motor cortex. Rather, the association cortex projects directly to the cerebellum and basal ganglia pre-motor areas. Kornhuber proposed that the basal ganglia and cerebellum serve as special *function generators* that interpose between decision levels (association cortex) and execution levels (motor cortex and motor neurons). Further, as has been already asserted above, the cerebellum acts as the function generator for rapid ballistic movements while the basal ganglia fulfills this role in slower, ramp movements. It has been suggested by Hamada (1981) that only the specifications for muscle and level of activation come from the motor cortex. The discharges concerned with force regulation and rate of force development would thus be the responsibility of the cerebellum and the basal ganglia as function generators. Recent research by Brotchie et al. (1991a; 1991b) on the motor function of the monkey globus pallidus suggests that the neuronal activity of the basal ganglia may depend a great deal upon the conscious and cognitive aspects of the movement being performed, with very specific discharge patterns being evidenced for different movement velocities and strategies.

Having provided background evidence for differential initiation and control of ballistic movements in the CNS, it is now necessary to turn to peripheral manifestations of this control.

[B] Muscle Activation

1) Ballistic burst duration, rate of force development, and rate gradation

The ballistic agonist burst may occur 50-100 ms before movement onset (Desmedt & Godaux, 1979) and may cause motor units to attain extremely high firing rates of 60-120 Hz (Desmedt & Godaux, 1977). These maximum firing rates are far greater than those experienced during slow ramp contractions (Bawa & Calancie, 1983; Edstrom & Grimby, 1986; Freund, 1983; Grimby, 1986; Tanji & Kato, 1973). Motor units fire in brief high-frequency bursts during ballistic contractions (Burke, 1981; Freund, 1983), which may last for 100 ms and terminate prior to the actualization of muscle force production (Desmedt & Godaux, 1979). This high frequency discharge can also be seen in the occurrence of doublet discharges in single motor unit firing (Desmedt, 1981; Tomberg et al., 1991), perhaps making use of the *catch property* (Binder-Macleod & Barker, 1991; Burke, 1981). These manifestations serve to markedly increase the rate of force development and maximal velocity of the limb being controlled during the ballistic contraction.

2) Agonist (Ag)-antagonist (Ant) burst pattern

As described in the previous section, slow and fast limb movements are controlled differently and by different anatomical structures in the CNS. These differences are readily seen at the muscle level through the recording of electromyograms (EMG). As observed by Wachholder & Altenburger (1926; cited in Halett & Marsden, 1978) slower, ramp movements evidence continuous agonist activity, while faster ballistic movements are characterized by alternating bursts of activity in agonist and antagonist muscles as observed with the surface EMG recording. This characteristic ballistic Ag-Ant-Ag EMG

manifestation is the so-called "triphasic" pattern (Angel, 1974, 1975; Basmajian, 1967; Cooke & Brown, 1990; Gottleib et al., 1970; Hopf et al., 1973; Wachholder & Altenburger, 1926), wherein there is an initial agonist burst, then a burst of activity from the antagonist, followed by a second agonist burst. As distinct from slower movements, muscular activity in ballistic movements was found by Richer (1895 a,b, cited in Desmedt & Godaux, 1978a) to evidence a brief initial agonist contraction followed by complete agonist relaxation prior to the cessation of the movement. This distinction was further emphasized by Woodworth (1899), Stetson (1905), and Stetson and McDill (1923) (all cited in Desmedt & Godaux 1978a), who also found that during certain (high speed) movements brief initial agonist contractions were followed immediately by agonist relaxation periods before the termination of the movement. Indeed, EMG recordings have provided ample evidence for the existence of a clear difference between slow and ballistic movements in agonist-antagonist activation patterns (Desmedt, 1978b). Since the initial documentation of the triphasic pattern, much research has provided further evidence of this phenomenon (Angel, 1975; Cooke & Brown, 1990; Dyhre-Poulsen et al., 1991; Gordon & Ghez, 1984; Marsden et al., 1983; Rich, 1990; Wierzbicka et al., 1986). Angel (1975) investigated the effect of unexpected load changes on the dual agonist activation in the triphasic activation of the posterior deltoid in humans. It was found that the second agonist burst (Ag2) evidenced approximately 60% of the duration of the first agonist burst (Ag1) while the minimum, or agonist silent period, was approximately 16% of Ag1 duration. When movement was artificially impeded, there was no appearance of the agonist silent period. That is, there was no distinction between Ag1 and Ag2. On the basis of the observed results, it was determined that the size of Ag2 and of the silent period depend on peripheral feedback while Ag1 is, as mentioned in the previous sections, pre-programmed centrally. However, the Ant1 burst has been shown to be potentiated by

pre-stretch and lessened by a release of tension prior to movement (Hallet & Marsden, 1979). This has allowed for the conclusion that the Ant1 burst occurs at the latency of the long-latency stretch reflex and is thus amenable to peripheral feedback and the effects of the spinal reflexes (Garland & Angel, 1971). Cooke and Brown (1990) also examined phasic muscle activation during flexion/extension movements about the elbow joint. These researchers concluded that the Ag1 burst and Ant1 burst subserved acceleration and deceleration respectively. They also suggest that the Ag2 burst serves the primary role of terminating the negative acceleration phase induced by the Ant1 burst. Similar conclusions were made by Wierzbicka et al. (1986) when they found that the agonist is primarily responsible for distance moved, with the antagonist subserving the role of reducing movement time. Furthermore, Hallett (1983) suggested that the Ag1 and Ant1 bursts range in duration from approximately 50 to 100 ms, seemingly independent of joint angle, movement amplitude, or the force against which the limb is moved.

It is important to consider that the triphasic pattern is not limited to the performance of dynamic ballistic movements only. Gordon and Ghez (1984) studied EMG patterns during isometric ballistic actions (where a rapid rate of force development is achieved) and did indeed find the presence of the characteristic triphasic burst pattern in the biceps and triceps. Of more than passing interest are the findings of Rich (1990) who investigated the agonist-antagonist activity of the forearm flexors in adults aged 30-70 y. She found that an individual subject's triphasic activity patterning are maintained well into the seventh decade of life.

According to Angel (1981) there are three possible mechanisms responsible for the agonist silence observed to occur between Ag1 and Ag2. Stretching of the antagonist during contraction of the agonist could result in homonymous reciprocal inhibition of the agonist muscle. Alternatively, build-up of tension for rapid movement could stimulate the

inhibitory Golgi tendon organ reflex causing the agonist to be shut down. The final, and most probable, explanation is that of an unloading reflex involving alpha-gamma motor neuron linkage. In the pre-movement state, during the build up of tension just prior to the movement of the limb, the agonist muscle motor neurons may be facilitated due to the stretch of the extrafusal fibres causing increased gamma activity. With contraction, there is an unloading of the extrafusal fibres causing a decrease in gamma motor neuron firing and decreased facilitation. The result would be the relative silence seen to occur between Ag1 and Ag2. Recent work by Al-Falahe et al. (1991) suggests that the influence of spindle discharge may indeed subserve important functions in the regulation of agonist and antagonist discharge during fast ballistic movements.

Recently, Brown and Gilleard (1991) observed the Ag1 burst in the transition from performance of slow movement and ballistic movement. The distinctive triphasic pattern was shown to occur most frequently when movement time was reduced to less than 400 ms. These authors found both that the triphasic burst pattern evolved out of a decrease in movement time and that the ballistic discharge pattern is not a spontaneously occurring event but rather occurs as a function of movement time constraints.

That there exists a certain overlap in the timing of Ag1, Ant1, and Ag2, thus resulting in a certain amount of co-activation or co-contraction, is also an observed feature occurring in ballistic contraction (Cooke & Brown, 1990; Marsden et al., 1983). It is to co-activation that this review now turns.

3) Co-activation of antagonist muscles

Basmajian (1978) has suggested that antagonist co-contraction is minimal in simple movements except at high velocities (i.e. ballistic movements). Antagonist muscle activity during ballistic movements has been observed to be a function of a subject's movement strategy (Waters & Strick, 1981). Waters and Strick (1981) found that antagonist co-

activation occurred when accurate termination of the movement was desired and that, correspondingly, if accuracy was not a consideration antagonist activity was markedly reduced or removed altogether. Marsden et al. (1983) studied fast flexion movements of the human thumb and fast extension movements of the elbow in order to determine antagonistic muscle function. They found that moving through a small amplitude quickly caused a large antagonist activity starting very soon after agonist activity. They concluded that the size and timing of the bursts of muscle activity were precisely adjusted to the exact nature of the task to be performed. Osternig et al. (1986) investigated the co-activation of quadriceps and hamstrings in sprinters and distance runners during low (100 degrees/s) and high (400 degrees/s) speed isokinetic exercise. These researchers found that the faster ballistic-type knee extensions induced a marked increase in hamstring co-activation in all subjects. Considerable co-activation of hip and knee flexors and extensors can also be seen in examining the reaction time sprint start EMG results of Mero and Komi (1990). These results are all in accordance with the findings of Lestienne (1979) who found that triceps activity during fast, ballistic elbow flexions was directly proportional to the velocity of movement.

The learning of a new motor task requiring high speed movement has also been shown to cause an increase in antagonist co-activation (Engelhorn, 1983; McGrain, 1980). In addition, this research has shown that the necessity of increased joint angular velocity (as in ballistic movement) will cause a marked increase in EMG activity of antagonist muscles acting at that joint (McGrain, 1980). The functional significance of this observed antagonist co-activation during ballistic movements may be to prevent injury and maintain joint integrity (Osternig et al., 1986; Tyler & Hutton, 1986). However, the exact amount of functional interference caused by co-contraction during a ballistic movement cannot be solely based upon examinations of the surface EMG. As reported by

Norman and Komi (1979), the electromechanical delay (EMD) interacts in a complex fashion with muscle fibre type composition, contraction type (eccentric or concentric), and the movement velocity. These researchers concluded that the EMD must be considered when the phasic relations between muscle force or joint torque generation from different muscles are inferred from EMG recordings.

4) Agonist premovement depression (PMD)

In a prior section, it was suggested that a large agonist EMG burst (Ag1) serves to begin a ballistic limb movement. However, it has been shown that a decrease, rather than an increase in agonist activity may occur 40-50 ms in advance of the Ag1 burst (Ikai, 1955; cited in Conrad et al., 1983; Yabe , 1976). In a situation of low-level tonic co-contraction preceding voluntary ballistic movement, a premovement inhibition of the antagonist has also been observed (Hufschmidt & Hufschmidt, 1954). Agonist premovement depression (PMD) can only be seen when there exists some low-grade tonic activity of the muscles prior to ballistic movement execution. The duration of the agonist EMG depression has been shown to span a wide and variable range from a low of 40 ms (Gatev 1972) to a high of 100 ms (Yabe 1976a). Other studies place the PMD duration at more intermediate values of 50-80 ms (Conrad et al., 1983; Yabe, 1976b; Zehr & Sale, 1993a,b).

The frequency of occurrence of the PMD phenomenon has also been shown to be quite variable, both within subject and between subjects (Shibata and Moritani, 1991, cited in Moritani, 1993). Gatev (1972) reported that the PMD occurred in ~25% of elbow extension movements, while Tanii (1983) reported an inter-subject range of ~11% to ~95% frequency of occurrence for subjects performing back extension movements. Palmer et al. (1991) found a 30% occurrence during elbow extension movements of Parkinson patients. The occurrence of the PMD phenomenon has also been shown to

depend on the movement intent of the subject. Mortimer et al. (1987) found that the PMD occurred in 58% of subject-paced trials and only 29% in reaction time trials. As well, Kawahats and Miyashita (1983) found that the PMD occurred most frequently (53%) prior to a swift, well co-ordinated movement.

The PMD phenomenon (also PMS; premovement silence, see Conrad et al., 1983) has been examined in the human knee flexors and extensors during explosive vertical jumping (Kawahats & Miyashita, 1983; Yabe, 1976). The experimental set-up used by Yabe (1976) involved subjects vertical jumping as quickly as possible from a position allowing slight knee flexion (pre-contraction of the agonist) following a given reaction time (RT) stimulus. It was found that the knee extensors evidenced the most consistent PMS appearance. The PMS period occurred, as a percentage of total trials, 53% in rectus femoris, 61% in vastus medialis, and 72% in the vastus lateralis. Kawahats and Miyashita (1983) utilized a similar set-up and observed the occurrence of the PMD in both knee extensors and flexors but that it was restricted to a limited knee joint angle where tension requirement was low. The occurrence of PMD has also been studied during ballistic elbow movements (Conrad et al., 1983; Mortimer et al., 1987; Walter, 1988). Walter (1988) had subjects perform rapid dynamic elbow extensions and flexions, and static flexions while EMG activity of the biceps and triceps was recorded. His results give support to the idea that the PMD occurs as a general control mechanism both in flexors and extensors. Also of interest is the observation that the PMD is observed in static contractions wherein there is a rapid rate of force of development and may subserve the same functions as in dynamic contraction.

Nishizono and Kato (1987) examined the PMD occurrence during the highly skilled act of the release in archery. Three different skill levels were analyzed and it was found that the occurrence of PMD was highest in the most highly skilled group with a

continuous decrease in appearance rate towards the lesser skilled groups. This would tend to point to a potential learning effect on the PMD, an observation also alluded to by Mortimer et al. (1987).

Shibata and Moritani (1991; cited in Moritani, 1993) found that the occurrence of agonist PMD resulted in a significantly greater maximal rate of force development during ballistic plantar flexion movements. These researchers also observed a significant reduction in H-wave amplitude occurring approximately 40 ms immediately prior to the occurrence of PMD and preceding force development by ~50 to 60 ms.

Mortimer et al. (1987) have suggested several physiological mechanisms to account for the PMD period. They suggest disfacilitation of tonically active motor neurons caused by inhibition of supra-spinal centres, post synaptic inhibition produced by spinal interneurons, or primary afferent depolarization causing presynaptic inhibition. These researchers conclude that pre-synaptic inhibition and disfacilitation are most probable. Many other observations support the proposition that disfacilitation, through alpha-gamma linkage and a neural switching mechanism, may be quite likely (Conrad et al., 1983; Kawahats & Miyashita, 1983; Ward, 1978; Yabe, 1976). Shibata and Moritani (1991; cited in Moritani 1993) suggest that PMD latencies are too short to allow for post synaptic inhibition via spinal interneuronal networks operating in parallel with alpha motoneuron activation. They suggest that PMD could be achieved by alpha motoneuron inhibition through the action of spinal interneurons activated by the cortico-spinal tract, a hypothesis supported by their findings of decreased H-reflex amplitude during the silent period (see above).

The functional significance for the PMD period is most likely the increase in muscular peak force and peak rate of force development caused by allowing motor neurons to be brought into a nonrefactory state (Conrad et al., 1983; Mortimer &

Eisenberg, 1982; Tanii, 1984) thereby allowing for a very short first interspike interval. Thus when the Ag1 burst occurs, the activated muscle can rise quickly in force development in static contraction (Walter, 1988) or produce high accelerations and power (Tanii, 1984) in dynamic movement (Shibata & Moritani, 1991; cited in Moritani, 1993).

It has been postulated that the PMD may be a learned response, and that it may be most likely to occur in self-initiated movements demanding high instantaneous force. Walter (1989) did show that subjects could gain some measure of voluntary control over the agonist PMD with biofeedback training and that control over PMD could thus be acquired. Also, Moritani (1993) suggests that the variability of PMD in terms of inter- and intra-subject occurrence and duration, points to a potential learning effect or learned motor response, rather that a natural and automatically occurring movement component. However, Zehr and Sale (1993a,b) examined a group of moderately-trained karate practitioners and found only an approximately 27% frequency of occurrence and a large inter-subject variability in PMD occurrence. As the values of these ballistically habituated subjects are not different from values of untrained subjects already shown in the literature, this would tend to argue against a naturally occurring training effect. What might be found in very highly skilled subjects, such as black belt level karate practitioners, though, is as yet unknown.

As was mentioned at the beginning of this section, the PMD period is only seen prior to rapid movement, and only when there is some tonic background activity of the involved muscles. Recently Mellah et al. (1990) examined the changes occurring in motor unit excitability during movement preparation. They found low-grade activity (as occurs prior to many skilled activities such as in the co-contraction prior to or during ballistic movements outlined above) occurring prior to forearm flexion movements in monkeys to be due to slow motor unit activity. Their results also show that the motor units active

during movement execution were of the fast motor unit type. Mellah et al. (1990) concluded that the slow motor unit activity in the preparatory phase may enhance the efficiency of the fast motor units during the following ballistic contraction by increasing muscle stiffness. As well, the slow motor units may have contributed to the build-up of nervous activity for the following movement and thus affect central neuronal excitability. As such, these results point to further potential effects of the PMD on ballistic movement execution. Of pertinence to the following section, is the extent to which the results of Mellah et al. (1990) and the PMD literature may reflect selective recruitment.

5) Selective recruitment of fast motor units

When continuous contractions of different forces are performed, agonist motor units are recruited in a consistent order from smaller soma, slow contracting to larger soma, faster contracting motor units, according to the so-called *size principle of motor unit recruitment* proposed by Henneman et al. (1965). The extent to which this *size principle* pertains to ballistic movements is unclear. Desmedt (1981; Desmedt & Godaux, 1976) has indicated that with fast ballistic contractions the recruitment order is maintained while the force recruitment threshold for a given motor unit decreases markedly. This quantity, the ballistic force threshold, is calculated on the basis of the mean between maximum peak force for which the motor unit never discharges and the mean peak force for which it always discharges (Desmedt, 1981). Tanji and Kato (1973a,b) found that individual motor units were recruited at lower thresholds in faster contractions in the human abductor digiti minimi. As well, Burke (1981) has raised the possibility that major restructuring of motor unit recruitment thresholds may take place under certain conditions (i.e. as in ballistic movement). While the recruitment thresholds may be changed, Stein and Bertoldi (1981) suggest that the orderly recruitment of motor neurons according to size definitely applies during ballistic voluntary contractions.

Grimby and Hannerz (1977), using bipolar needle electrodes, recorded motor units from human toe extensors during voluntary contraction. These authors identified two main motor unit types; continuously firing long interval and intermittently firing short interval motor units. In prolonged constant-strength contractions and rapid alternating movements, both unit types were active and played similar roles. However, in twitch contractions, the intermittently firing units could be selectively activated if the muscle was relaxed, and if a great effort with short contraction duration was intended (i.e. rapid, ballistic contraction). They suggested that the intermittently firing units be ascribed to type II, fast twitch motor units and that the recruitment order of type I and type II motor units is adapted to the contraction mode. Also, if the muscle was slightly contracted or stretched prior to the ballistic contraction, or if lesser effort and longer contraction duration was intended, selective activation of type I (continuously firing long interval motor units) was evidenced (Grimby & Hannerz, 1981). Grimby and Hannerz (1981) further suggest that this observed selective activation may not be seen in studies of isometric twitch tension because of the necessity of a pre-existing contraction.

Nardone and Schieppati (1988) observed a shift in activity from human soleus to gastrocnemius lateralis during voluntary lengthening contractions of increasing velocity. These authors concluded that voluntary lengthening of the triceps surae is accomplished through derecruitment of slow motor units concomitant with selective activation of fast motor units, a manifestation influenced by movement velocity. Nardone et al. (1989) further examined this phenomenon during active lengthening of the soleus, gastrocnemius lateralis, and gastrocnemius medialis. These authors identified 3 populations of units; those active during shortening (S), those active during shortening and lengthening (S+L), and those active only during lengthening (L). Activity of L units, which were concluded to be associated with high-threshold motor neurons, showed a great dependence upon

velocity of lengthening. Most L units could be recruited voluntarily only when performing ballistic isotonic or isometric contractions. At the same time, most S and S+L units were derecruited during the contractions involving L unit selective activation. The authors concluded that the morphological and functional features of the L units (i.e. short 1/2 relaxation time) determined the observed selective activation.

It can be seen from the above that there exists a certain controversy over the extent to which selective recruitment of large soma, fast twitch motor units may occur (if at all) in ballistic contractions. At this juncture it is important to keep in mind certain methodological and measurement considerations that must be noted when discussing potential violations of the size principle in ballistic movement. Desmedt (1981) has suggested that the differing motor unit axonal conduction velocities must be considered when discussing muscle recordings and the size principle. As the force recruitment threshold is markedly reduced (see above) both type I and type II motor units may fire almost simultaneously. Due to the larger axon and correspondingly larger axonal conduction velocity of the type II unit, it will appear to discharge ahead of the type I unit. It is important also to consider the difference between selective recruitment of motor units in pools from synergistic motor unit pools being selectively activated during certain movements (Desmedt ,1981). This involves consideration of research similar to that of Nardone and Schieppati (1988) briefly mentioned above, but is not the focus of the present paper.

This review now turns to a brief description of potential neuromuscular adaptations that may be induced by ballistic contraction training.

[C] Neuromuscular adaptation to ballistic training

Given the specific burst patterns, co-ordination strategies, and control schemes experienced during the performance of ballistic movements, it might be expected that this type of training or activity simulating these contractions would induce very specific neuromuscular adaptations that may extend even to fibre-type transformation. By means of direct, intermittent high frequency (100 Hz) muscle stimulation, Lomo et al. (1974, 1980) induced a transformation from a slow to a fast muscle in denervated rat soleus. This transformation was considered on the basis of isometric twitch time to peak tension (TPT), half-relaxation time (1/2 RT), and twitch to tetanus ratio (TTR), as well as post-tetanic potentiation characteristics, position of tension frequency curve, and sag appearance during continuous tetanus. These researchers concluded that it is the stimulation pattern that is of central importance for inducing changes in the contractile characteristics of the activated muscle. Although there has been little evidence to support such extreme fibre-type conversion in mammalian muscle (Edstrom & Grimby, 1986), recent research by Jansson et al.(1990) has indicated that it may be possible to achieve fibre-type transformation with high intensity, high velocity training. These authors had their subjects perform repeated Wingate 30 s sprint tests on the cycle ergometer for 4-6 weeks and noted a significant decrease in type I and a significant increase in type II muscle fibres. They conclude that the effect of sprint training may be related to an increased stimulation frequency seen through the change in fibre activation pattern with training.

Casabona et al. (1990) examined differences in H-reflex between athletes trained for explosive contractions and untrained control subjects. These researchers found a significantly smaller ratio of the maximal reflex response to the maximal direct response in

athletes trained for explosive-type movements. This difference was due to a smaller amplitude of the maximal reflex response (H-reflex). They tentatively conclude that this difference could be due to training-induced transformation of small and slow motor neurons into large and fast ones. Hainaut et al. (1981) had subjects train isometrically or fast isotonically for 3 months. Following cessation of training, it was found that the fast isotonically trained subjects had an accelerated twitch time course and a shorter contraction time than the isometrically trained subjects. In a similar study, Duchateau and Hainaut (1984) found that dynamically (fast isotonic) trained subjects evidenced a rate of tension development that was 13% greater than those who trained isometrically. Maximal velocity of shortening was also increased by 21% following dynamic training whereas there was no change after isometric training. Interestingly, only dynamic training reduced the twitch time to peak (by 11%) thus reducing the peak twitch force seen in the dynamic trained subjects. These researchers concluded that human muscle has the capacity to adapt differently to isometric or dynamic training. Cracraft and Petajan (1977) found that dynamic and isometric training regimens could induce changes in individual motor unit firing patterns that could effect muscular adaptation. This conclusion was based on their finding that differential training regimens could produce changes in motor unit firing patterns and thus effect change in the muscle fibres of the alpha motor neurons involved in training.

That there exists a specific effect of ballistic or high velocity training upon strength or muscular adaptation has been repeatedly shown (Coyle et al., 1981; Dudley et al., 1990; Moritani, 1993; Sale, 1987, 1988; Sale & MacDougall, 1981). Hakkinen et al. (1985) investigated the influence of explosive-type strength training on isometric force- and relaxation-time and on the EMG of leg extensor muscles. They had subjects train in explosive jumping exercise with and without load for a 24 week period. Following

training subjects showed greater improvement in fast force production than in maximal strength. These fast force production gains were concomitant with increased neural activation and fast twitch: slow twitch fibre area ratio. It was concluded that explosive type ballistic jumping training can cause significant neural and selective muscular adaptations resulting in improved performance. Similar results were observed by Hakkinen and Komi (1986) when they compared explosive type strength training to heavy resistance training. Explosive training again evidenced a considerable shortening in the time of force production following training, indicative of a specific high-velocity training adaptation.

It is intuitively obvious that neural factors and muscular adaptations interact in variable ways to produce a training effect following any kind of training, especially strength or power training. It has been repeatedly shown that the gains in strength or power seen to follow training have an initial neural basis (Cannon & Cafarelli, 1987; Hakkinen & Komi, 1983; Moritani & Devries, 1979; Rutherford & Jones, 1986; Sale, 1988; Thorstensson et al., 1976). Training has been shown to affect motor neuron excitability in man, causing an increased ability to raise excitability during effort (Sale et al., 1983). As well, Milner-Brown et al. (1975) showed that training could potentially affect supra-spinal connections from the motor cortex to spinal motor neurons to produce synchronization of motor units during contractions. Further, ballistic training may induce further neural adaptations involving reflex responses. Mortimer and Webster (1983) found karate-trained (ballistic athletes) to manifest larger increases in the gain of long-latency myotatic pathways preceding movement, greater limb acceleration, and briefer rise times in initial agonist (Ag1) burst than untrained subjects. It can be seen from these observations that the potential for ballistic movement, so specialized in neuromuscular manifestation, to induce specific training adaptations is enormous.

Recently, Behm and Sale (1993) examined the responses of men and women to ballistic isometric and ballistic high velocity isokinetic training. During all training contractions, subjects were instructed to contract as rapidly and forcefully as possible and to then relax as quickly as possible. Following 16 wks of training, isokinetic peak torque results exhibited a velocity-specific training effect with the greatest peak torque increase seen at the highest testing velocity. Voluntary isometric peak torque and rates of torque development and relaxation all increased after training. Their results show that the velocity specific response to the isometric and concentric isokinetic training was the same. Behm and Sale (1993) suggest that their results indicate that it was the intent to contract ballistically rather than the actualized contraction velocity that determined the velocity specific response. The observation that the changes in contractile properties that are considered to increase high velocity ballistic strength performance did not depend on the actual training velocity serves to show the potential for ballistic training to effect specific neuromuscular adaptation.

References

Al-Falahe, N. A., Nagaoka, M., and Vallbo, A.B.(1991) . Dual response from human muscle spindles in fast voluntary movements. **Acta Physiol Scand** 141:363-371.

Alfonsi, E., Bono, G., Mertel, J., Sandrini, G., Bollani, E., and Moglia, A. (1992). Electrically elicited short and long-latency responses of intrinsic hand muscles in hereditary ataxias. Effects of isometric and ballistic isotonic voluntary contractions. **EMG Clin. Neurophysiol.** 32: 435-442.

Angel, R.W.(1974). Electromyography during voluntary movement: the two-burst pattern. **EEG Clin. Neurophysiol.** 36:493-498.

Angel, R.W. (1975). Myoelectric patterns associated with ballistic movement: effect of unexpected changes in load. **J. Hum. Mov. Stud.** 1:96-103.

Angel, R.W. (1981). Electromyographic patterns during ballistic movements in normals and hemiplegic patients. In: J.E. Desmedt (Ed.), **Progress in clinical neurophysiology volume 9: Motor unit types, recruitment and plasticity in health and disease,** pp.347-357. Basel: Karger.

Basmajian, J.V.(1967). Control of individual motor units. **Am. J. Phys. Med.** 46:480-486.

Basmajian, J.V. (1978). **Muscles alive: Their functions revealed by electromyography,** (4th ed.). Baltimore: Williams & Wilkins.

Bawa, P., and Calancie, B. (1983). Repetitive doublets in human flexor carpi radialis muscle. **J. Physiol.** 339:123-132.

Behm, D.G., and Sale, D.G. (1993). Intended rather than actual movement velocity determines velocity-specific training response. **J. Appl. Physiol.** 74(1): 359-368.

Binder-Macleod, S.A., and Barker, C.B. (1991). Use of a catchlike property of human skeletal muscle to reduce fatigue. **Muscle Nerve** 14:850-857.

Brooks, V.B. (1975). Roles of cerebellum and basal ganglia in initiation and control of movements. **Can. J. Neurolog. Sci.** 265-277, August.

Brotchie, P., Iansek, R., and Horne, M.K. (1991a). Motor function of the monkey globus

pallidus: 1. Neuronal discharge and parameters of movement. **Brain** 114:1667-1683.

Brotchie, P., Iansek, R., and Horne, M.K. (1991b). Motor function of the monkey globus pallidus. 2. Cognitive aspects of movement and phasic neuronal activity. **Brain** 114: 1685-1702.

Brown, J.M.M., and Gilleard, W. (1991). Transition from slow to ballistic movement: development of triphasic electromyogram patterns. **Eur. J. Appl. Physiol.** 63: 381-386.

Burke, R.E. (1981). Motor unit recruitment: what are the critical factors? In: J.E. Desmedt (Ed.), **Progress in clinical neurophysiology Vol. 9: Motor Unit types, recruitment and plasticity in health and disease**, pp. 61-84. Karger: Basel.

Butler, E.G., Horne, M.K., and Hawkins, N.J. (1992). The activity of monkey thalamic and motor cortical neurones in a skilled, ballistic movement. **J. Physiol.** 445: 25-48.

Cannon, R.J., and Cafarelli, E. (1987). Neuromuscular adaptations to training. **J. Appl. Physiol.** 63(6): 2396-2402.

Casabona, A., Polizzi, M.C., and Perciavalle, V. (1990). Differences in H-reflex between athletes trained for explosive contractions and non-trained subjects. **Eur. J. Appl. Physiol.** 61:26-32.

Conrad, B., Benecke, R., and Goehmann, M. (1983). Premovement Silent period in fast movement initiation. **Exp. Brain Res.** 51: 310-313.

Cooke, J.D., and Brown, S.H. (1990). Movement-related phasic muscle activation. II. Generation and functional role of the triphasic pattern. **J. Neurophysiol.** 63(3): 465-472.

Coyle, E.F., Feiring, D.C., Rotkis, T.C., Cote III, R.W., Roby, F.B., Lee, W., and Wilmore, J.H. (1981). Specificity of power improvements through slow and fast isokinetic training. **J. Appl. Physiol.** 51(6): 1437-1442.

Cracraft, J.D., and Petajan, J.H. (1977). Effect of muscle training on the pattern of firing of single motor units. **Am. J. Phys. Med.** 56(4): 183-193.

DeLong, M.R., and Strick, P.L. (1974). Relation of basal ganglia, cerebellum, and motor cortex units to ramp and ballistic limb movements. **Brain Res.** 71:327-335.

Denny-Brown, D. (1962). **The Basal ganglia.** London: Oxford University Press.

Desmedt, J.E. (1981). The size principle of motoneuron recruitment in ballistic or ramp voluntary contractions in man. In: J.E. Desmedt (Ed.), **Progress in clinical neurophysiology Vol. 9: Motor unit types, recruitment and plasticity in health and disease**, pp. 97-136. Karger: Basel.

Desmedt, J.E., and Godaux, E. (1977). Ballistic contractions in man: characteristic recruitment pattern of single motor units of the tibialis anterior muscle. **J. Physiol.** 264: 673-693.

Desmedt, J.E., and Godaux, E. (1978a). Ballistic skilled movements: load compensation and patterning of the motor commands. In: J.E. Desmedt (Ed.), **Progress in clinical neurophysiology Vol. 4.: Cerebral motor control in main: long loop mechanisms**, pp. 21-55. Karger: Basel.

Desmedt, J.E., and Godaux. E. (1978b). Ballistic contractions in fast or slow human muscle: discharge patterns of single motor units. **J. Physiol.** 285:185-196.

Desmedt, J.E., and Godaux, E. (1979). Voluntary motor commands in human ballistic movements. **Ann. Neurol.** 5: 415-421.

Duchateau, J., and Hainaut, K. (1984). Isometric or dynamic training: differential effects on mechanical properties of a human muscle. **J. Appl. Physiol.** 56(2): 296-301.

Dudley, G.A., Harris, R.T., Duvoisin, M.R., Hather, B.M., and Buchanan, P. (1990). Effect of voluntary vs. artificial activation on the relationship of muscle torque to speed. **J. Appl. Physiol.** 69(6): 2215-2221.

Dyhre-Poulsen, P., Simonsen, E.B., and Voigt, M. (1991). Dynamic control of muscle stiffness and H-reflex modulation during hopping and jumping in man. **J. Physiol.** 437: 287-304.

Edstrom, L., and Grimby, L. (1986). Effect of exercise on the motor unit. **Muscle Nerve** 9: 104-126.

Engelhorn, R. (1983). Agonist and antagonist muscle EMG activity pattern changes with skill acquisition. **Res. Q.** 54(4): 315-323.

Freund, H.J. (1983). Motor unit and muscle activity in voluntary motor control. **Physiol. Rev.** 63: 387-436.

Garland, H., and Angel, RW. (1971). Spinal and supraspinal factors in voluntary movement. **Exp. Neurol.** 33: 343-350.

Gordon, J., and Ghez, C. (1984). EMG patterns in antagonist muscles during isometric contraction in man: relations to response dynamics. **Exp. Brain Res.** 55: 167-171.

Gottlieb, G.L., Agarwal, G.C., and Stark, L. (1970). Interactions between voluntary and postural mechanisms of the human motor system. **J. Neurophysiol.** 33: 365-381.

Grimby, L. (1986). Single motor unit discharge during voluntary contraction and locomotion. In: N.L. Jones, N. McCartney, and A.J. McComas (Eds.), **Human Muscle Power**, pp. 111-130. Champaign, Illinois: Human Kinetics Publishers.

Grimby, L., and Hannerz, J. (1977). Firing rate and recruitment order of toe extensor motor units in different modes of voluntary contraction. **J. Physiol.** 264: 865-879.

Grimby, L., and Hannerz, J. (1981). Flexibility of recruitment order of continuously and intermittently discharging motor units in voluntary contraction. In: J.E. Desmedt (Ed.), **Progress in clinical neurophysiology Vol. 9: Motor unit types, recruitment and plasticity in health and disease**, Karger: Basel.

Hainaut, K., Duchateau, J., and Desmedt, J.E. (1981). Differential effects on slow and fast motor units of different programs of brief daily muscle training in man. In: J.E. Desmedt (Ed.), **Progress in clinical neurophysiology Vol.9: Motor unit types, recruitment and plasticity in health and disease**, pp. 241-249. Karger: Basel.

Hakkinen, K., and Komi, P.V. (1983). Electromyographic changes during strength training and detraining. **Med. Sci. Sports Exerc.** 15(6): 455-460.

Hakkinen, K., and Komi, P.V. (1986). Training-induced changes in neuromuscular performance under voluntary and reflex conditions. **Eur. J. Appl. Physiol.** 55: 147-155.

Hakkinen, K., Komi, P.V., and Alen, M. (1985). Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. **Acta Physiol. Scand.** 125: 587-600.

Hallett, M. (1983). Analysis of abnormal voluntary and involuntary movements with surface electromyography. In: J.E. Desmedt (Ed.), **Advances in Neurology Vol. 39: Motor control mechanisms in health and disease**, pp. 907-914. New York: Raven.

Hallett, M., and Marsden, C.D. (1979). Ballistic flexion movements of the human thumb. **J. Physiol.** 294: 33-50.

Hamada, I. (1981). Correlation of monkey pyramidal tract neuron activity to movement velocity in rapid wrist flexion movement. **Brain Res.** 230: 384-389.

Henneman, E., Somjen, G., and Carpenter, D.O. (1965). Functional significance of cell size in spinal motoneurons. **J. Neurophysiol.** 28: 560-580.

Holmes, G. (1917). The symptoms of acute cerebellar injuries due to gunshot injuries. **Brain** 40: 461-535.

Hopf, H.C., Lowitzsch, K., and Schlegel, H.J. (1973). Central versus proprioceptive influences in brisk voluntary movements. In: J.E. Desmedt (Ed.), **New Developments in electromyography and clinical neurophysiology Vol. 3,** pp. 273-276. Karger: Basel.

Hufschmidt, H.J., and Hufschmidt, T. (1954). Antagonist inhibition as the earliest sign of a sensory-motor reaction. **Nature** Sept. 25, p. 607.

Ikai, M. (1955). Inhibition as an accompaniment of rapid voluntary action. **Nippon Seirigaku Zasshi [Physiol. Soc. Jpn]** 17: 292-298.

Ivry, R.B., Keele, S.W., and Denier, C. (1988). Dissociation of the lateral and medial cerebellum in movement timing and movement execution. **Exp. Brain Res.** 73: 167-180.

Jansson, E., Esbjornsson, M., Holm, I., and Jacobs, I. (1990). Increase in the proportion of fast-twitch muscle fibres by sprint training in males. **Acta Physiol. Scand.** 140: 359-363.

Kawahats, K., and Miyashita, M. (1983). Electromyogram premotion silent period and tension development in human muscle. **Exp. Neurol.** 82: 287-302.

Kornhuber, H.H. (1968). Neurologie des kleinhirns. **Zentbl. ges. Neurol. Psychiat.** 191: 13.

Kornhuber, H.H. (1971). Motor functions of cerebellum and basal ganglia: the cerebellocortical saccadic ramp (voluntary smooth speed movement) generator. **Kybernetik** 8: 157-162.

Kornhuber, H.H. (1974). The vestibular system and the general motor system. In: H.H. Kornhuber (Ed.), **Handbook of sensory physiology, Vol. 6, Part 2,** pp. 581-620. Berlin: Springer.

Lee, R.G. (1987). Physiology of the basal ganglia and pathophysiology of Parkinson's disease. **Can. J. Neurol. Sci.** 14: 373-380.

Lestienne, F. (1979). Effects of inertial load and velocity on the braking process of voluntary limb movements. **Exp. Brain Res.** 35: 407-418.

Lomo, T., Westergaard, R.H., and Dahl, H.A. (1974). Contractile properties of muscle: control by pattern of muscle activity in the rat. **Proc. R. Soc. Lond.** 187: 99-103.

Lomo, T., Westergaard, R.H., and Engebretsen, L. (1980). Different stimulation patterns affect contractile properties of denervated rat soleus muscle. In: D. Pette (Ed.), **Plasticity of muscle,** pp. 297-309. Berlin: Gruyter.

Marsden, C.D., Obeso, J.A., and Rothwell, J.C. (1983). The function of the antagonist muscle during fast limb movements in man. **J. Physiol.** 335: 1-13.

Martin, J.P. (1967). **The Basal ganglia and posture.** London: Pitman Medical.

McGrain, P. (1980). Trends in selected kinematic and myoelectric variables associated with learning a novel motor task. **Res. Q.** 51(3): 509-520.

Mellah, S., Rispal-Padel, L., and Riviere, G. (1990). Changes in excitability of motor units during preparation for movement. **Exp. Brain Res.** 82: 178-186.

Mero, A., and Komi, P.V. (1990). Reaction time and electromyographic activity during a sprint start. **Eur. J. Appl. Physiol.** 61: 73-80.

Milner-Brown, H.S., Stein, R.B., and Lee, R.G. (1975). Synchronization of human motor units: possible roles of exercise and supraspinal reflexes. **EEG and Clin. Neurophysiol.** 38: 145-254.

Moritani, T. (1993). Neuromuscular adaptations during the acquisition of muscle strength, power and motor tasks. **J. Biomechanics** 26(suppl.1): 95-107.

Moritani, T., and Devries, H.A. (1979). Neural factors versus hypertrophy in the time course of muscle strength gain. **Am. J. Phys. Med.** 58(3): 115-129.

Mortimer, J.A., and Eisenberg, P. (1982). Pre-movement silence in agonist muscles

preceding maximum power arm movements. **Soc. Neurosci. Abstr.** 8: 730.

Mortimer, J.A., Eisenberg, P., and Palmer, S.S. (1987). Premovement silence in agonist muscles preceding maximum efforts. **Exp. Neurol.** 98: 542-554.

Mortimer, J.A., and Webster, D.D. (1983). Dissociated changes of short- and long-latency myotatic responses prior to a brisk voluntary movement in normals, in karate experts, and in Parkinsonian patients. In: J.E. Desmedt (Ed.), **Advances in neurology Vol. 39: Motor Control Mechanisms in health and disease**, pp. 541-554. New York: Raven.

Nardone, A., and Schieppati, M. (1988). Shift of activity from slow to fast muscle during voluntary lengthening contractions of the triceps surae muscles in humans. **J. Physiol.** 395: 363-381.

Nardone, A., Romano, C., and Schieppati, M. (1989). Selective recruitment of high-threshold human motor units during voluntary isotonic lengthening of active muscles. **J. Physiol.** 409: 451-471.

Newman, R. P., and Calne, D.B. (1986). Parkinsonism: Physiology and pharmacology. In: N.S. Shah and A.G. Donald (Eds.), **Movement disorders**, pp. 39-52. New York: Plenum Medical.

Nishizono, H., and Kato, M. (1987). Inhibition of muscle activity prior to skilled voluntary movement. In: B. Jonsson (Ed.), **Biomechanics X-A**, pp. 455-458. Champaign, Illinois: Human Kinetics.

Norman, R.W., and Komi, P.V. (1979). Electromechanical delay in skeletal muscle under normal movement conditions. **Acta Physiol. Scand.** 106: 241-248.

Osternig, L.R., Hamill, J., Lander, J.E., and Robertson, R. (1986). Co-activation of sprinter and distance runner muscles in isokinetic exercise. **Med. Sci. Sports Exerc.** 18(4): 431-435.

Rich, N.C. (1990). Electromyography of rapid forearm flexion and extension and aging. **Int. J. Aging Hum. Dev.** 31(1): 11-29.

Richer, P. (1895a). Du mode d'action des muscles antagonistes dans les mouvements tres rapides, alternativement de sens inverse. **C. Seanc. Soc. Biol.** 47: 171-173.

Richer, P. (1895b). Note sur la contraction du muscle quadriceps dans l'acte de donner un coup de pied. **C. Seanc. Soc. Biol.** 47: 204-205.

Rutherford, O.M., and Jones, D.A. (1986). The role of learning and co-ordination in strength training. **Eur. J. Appl. Physiol.** 55: 100-105.

Sale, D.G. (1987). Influence of exercise and training on motor unit activation. In: K.B. Pandolf (Ed.), **Exerc. Sport Sci. Rev.**, Vol. 15, pp. 95-151. New York: Macmillan.

Sale, D.G. (1988). Neural adaptation to resistance training. **Med. Sci. Sports Exerc.** 20(5): S135-S145.

Sale, D.G., and MacDougall, J.D. (1981). Specificity in strength training: a review for the coach and athlete. **Can. J. Appl. Sports Sci.** 6(2): 87-92.

Sale, D.G., MacDougall, J.D., Upton, A.R.M., and McComas, A.J. (1983). Effect of strength training upon motoneuron excitability in man. **Med. Sci. Sports Exerc.** 15(1): 57-62.

Schmidt, R.A. (1988). **Motor control and learning** (2nd ed.). Champaign, Illinois: Human Kinetics.

Stein, R.B., and Bertoldi, R. (1981). The size principle: a synthesis of neurophysiological data. In: J.E. Desmedt (Ed.), **Progress in clinical neurophysiology Vol. 9: Motor unit types, recruitment and plasticity in health and disease**, pp. 85-96. Karger: Basel.

Stetson, R.H. (1905). A motor theory of rhythm and discrete succession. **Psychol. Rev.** 12: 250-270.

Stetson, R.H., and McDill, J.A. (1923). Mechanism of the different types of movement. **Psychol. Monogr.** 32: 18-40.

Tanii, K. (1984). Occurrence of a rhythmic slower wave in EMG prior to a rapid voluntary movement. **EEG Clin. Neurophysiol.** 57: 435-440.

Tanji, J., and Kato, M. (1973a). Recruitment of motor units in voluntary contractions of a finger muscle in man. **Exp. Neurol.** 40: 759-770.

Tanji, J. and Kato, M. (1973b). Firing rate of individual motor units in voluntary contraction of abductor digiti minimi muscle in man. **Exp. Neurol.** 40: 771-783.

Thach, W.T. (1968). Discharge of Purkinje and cerebellar nuclear neurons during rapidly

alternating arm movements in the monkey. **J. Neurophysiol.** 31: 785-797.

Thorstensson, A., Karlsson, J., Viitasalo, J.H. T., Luhtanen, P., and Komi, P.V. (1976). Effect of strength training on EMG of Human skeletal muscle. **Acta Physiol. Scand.** 98: 232-236.

Tomberg, C., Levarlet-Joyce, H., and Desmedt, J.E. (1991). Reaction time recording methods: reliability and EMG analysis of patterns of motor commands. **EEG and Clin. Neurophysiol.** 81: 269-278.

Tyler, A.E., and Hutton, R.S. (1986). Was Sherrington right about co-contractions? **Brain Res.** 370: 171-175.

Wachholder, K., and Altenberger, H. (1926). Beitrage zur physiologie der Wilkurlicken Bewegung IX Mitteilung. Fortlaufende Hin- und Herbewegunen. **Pflug. Arch.** 214: 625-641.

Walter, C.B. (1988). The influence of agonist premotor silence and the stretch-shortening cycle on contractile rate in active skeletal muscle. **Eur. J. Appl. Physiol.** 57: 577-582.

Walter, C.B. (1989). Voluntary control of agonist premotor silence preceding limb movements of maximal effort. **Percept. Motor Skills** 69: 819-826.

Ward, T. (1978). Muscle state: reaction and movement time in elbow extension. **Arch. Phys. Med. Rehabil.** 59: 377-383.

Waters, P., and Strick, P.L. (1981). Influence of 'strategy' on muscle activity during ballistic movements. **Brain Res.** 207: 189-194.

Wierzbicka, M.M., Wiegner, A.W., and Shahani, B.T. (1986). Role of agonist and antagonist muscles in fast arm movements in man. **Exp. Brain Res.** 63: 331-340.

Woodworth, R.S. (1899). The accuracy of voluntary movements. **Psychol. Monog.** 3:1-114.

Yabe, K. (1976). Premotion silent period in rapid voluntary movement. **J. Appl. Physiol.** 41(4): 470-473.

Yamamoto, K., and Mano, N. (1980). Simple-spike activity of cerebellar Purkinje cells related to visually guided wrist tracking movement in the monkey. **J. Neurophysiol.** 43(3): 713-728.

Zehr, E.P., and Sale, D.G. (1993a). Ballistic elbow extension movements in moderately trained karate practitioners: peak torque, velocity, acceleration, and the agonist pre-movement silence. **Med. Sci. Sports Exerc.** 25(5): S131.

Zehr, E.P., and Sale, D.G. (1993b). Loaded ballistic elbow extension actions in moderately trained karate practitioners: agonist PMD and movement performance between the hands. Submitted to **Eur. J. Appl. Physiol.**

Ballistic elbow extension actions in karate-trained and control subjects: agonist premovement depression (PMD) and movement performance

Running Head: Ballistic elbow extension in karate-trained and control subjects

E. P. Zehr, D.G. Sale, and J.J. Dowling
Department of Kinesiology
McMaster University

Address correspondence to:
D.G. Sale
Department of Kinesiology
McMaster University
Hamilton, Ontario, CANADA
L8S 4K1

Abstract

This study was conducted to determine differences in movement performance and the occurrence of agonist EMG premovement depression (agonist PMD) in highly trained karate practitioners and untrained control subjects. Isometric and ballistic elbow extension actions under two loading levels (0 and 10% of isometric MVC) were performed. Measures of peak torque, rate of torque development, peak and integrated biceps and triceps EMG, peak velocity, acceleration, and movement time were calculated. Karate-trained subjects produced significantly higher isometric (65.2 vs. 49.3 N·m; $p<0.005$) and ballistic (10% MVC load, 26.5 vs. 18.9 N·m; $p<0.01$) peak torques. Karate-trained subjects also had significantly higher ($p<0.001$) peak rate of torque development under both loading levels (0% and 10%; 209.9 and 470.0 $N \cdot m \cdot s^{-1}$) than control (138.8 and 312.1 $N \cdot m \cdot s^{-1}$). Peak acceleration was also significantly higher in the karate-trained as compared to the control subjects at both the 0% load (187.9 vs. 110.3 $rad \cdot s^{-2}$; $p<0.01$) and 10% MVC load (162.9 vs. 101.2 $rad \cdot s^{-2}$; $p<0.05$). Agonist PMD occurred sporadically in both groups occurring in 10% of trials for karate-trained and 5.4% of trials for control subjects. The average PMD duration was 57.8 ms for the karate-trained and 45.4 ms for the control (NS) group. The superior movement performance of the karate-trained subjects could not be explained in terms of agonist PMD. It is concluded that agonist PMD should not be considered to be a naturally occurring training effect or learned motor response.

TORQUE, VELOCITY, ACCELERATION, EMG

Introduction

Ballistic movements can be considered to be those movements that are performed as rapidly as possible, without reference to peripheral feedback. When a ballistic movement is performed upon a background of tonic muscular activity, a brief reduction may occur in the agonist EMG activity subsequent to phasic activation. This is known as the agonist premovement depression (PMD; 27) or premovement silence (PMS; 17,22). Investigations of the PMD phenomenon have included subjects ranging from children of 2.5 y (8) to 71 y old Parkinson patients (17), and have encompassed elbow flexion/extension (3,8,14,17,22,26), knee extension/vertical jump (10,25), rapid back extension (21), and plantar flexion (12) movements.

Results from investigations of the agonist EMG depression have revealed that the PMD phenomenon evidences several distinct characteristics. The duration of the agonist EMG silence has been shown to span a wide range from a low of 40 ms (8) to a high of 100 ms (25). Other studies place the PMD duration at more intermediate values of 50-85 ms (3,26,27). The frequency of occurrence of the PMD phenomenon has also been shown to be quite variable. Gatev (8) reported that the PMD occurred in ~25% of elbow extension movements, while Tanii (21) reported a range of ~11% to 95% inter-subject frequency of occurrence for subjects performing back extension movements. Palmer et al. (17) found a 30% frequency of occurrence during elbow extension movements of Parkinson patients. Zehr and Sale (27) recently found agonist PMD to occur on 27% of trials, also during ballistic elbow extension. The occurrence of the PMD phenomenon has also been shown to depend on the intent of the movement. Mortimer et al. (14) found that the PMD occurred in 58% of subject-paced trials, and only 29% in reaction time trials. As

well, Kawahats and Miyashita (11) found that the PMD occurred most frequently (53%) prior to a swift, well co-ordinated movement.

The functional significance of the PMD phenomenon has been shown to be an increase in the velocity and acceleration of the following limb movement. High correlations have been shown between movements evidencing the PMD and high peak accelerations and velocities (3,14,17,22), as well as with increased rate of force development (22). The only exception to this was shown by Palmer et al. (17) when their Parkinson patients evidenced the PMD but failed to demonstrate any significant kinematic potentiation.

The PMD phenomenon itself, while marginally described, is poorly understood. It is purported to be under the control of the CNS (22) and may involve some type of neural switching mechanism (25,26). It has been hypothesized that the silencing of the tonic background EMG activity may serve to increase subsequent force production by bringing all motor neurons into a non-refractory state immediately prior to phasic activation (3,14,21). As such, it has been postulated that the PMD may be a learned response, and that it may be most likely to occur in self-initiated movements demanding high instantaneous force (14). Walter (23) showed that subjects could gain some measure of voluntary control over the agonist PMD with biofeedback training and that control over PMD could thus be acquired. Zehr and Sale (27) examined agonist PMD during ballistic elbow performance in moderately trained karate practitioners. They found PMD occurrences and durations that were very similar to that of untrained subjects reported in the literature, observations that may argue against a naturally occurring PMD training effect. However, there have been no studies published that have addressed the issues of PMD occurrence and functional significance in relation to chronic ballistic training adaptations in highly skilled subjects. The purposes of the present study were first, to

evaluate potential differences between an untrained control group and a highly trained group of karate practitioners habituated to ballistic movements in the occurrence and kinematic and dynamic significance of agonist premovement depression during ballistic elbow extension movements. The second purpose was to examine any differences in terms of movement kinematics and dynamics that might exist between the control and the karate-trained subjects.

Methods

Study design

This study was a cross-sectional study consisting of 22 subjects and carried the approval of the McMaster University Ethics Committee.

Subjects

All subjects participated with informed written consent. The control group comprised 13 recreationally active males who had not undertaken any specific upper-body training or whole-body strength training. Nine males who had trained in Chito-Ryu karate for a minimum of 10 y and who had achieved a minimum karate rank of shodan (first degree black belt) formed the ballistic-trained group. This group had a mean training experience of (16.3 ±4.5 y) and comprised 2 shodan, 1 nidan (second degree), 5 sandan (third degree), and 1 godan (fifth degree). Subject anthropometric characteristics for trained and control groups can be found in Table 1.

Experimental methodology

Subjects came to the laboratory to perform isometric and ballistic elbow extension actions on a specially designed arm manipulandum.

Apparatus

An apparatus (Fig.1) was constructed and instrumented to record displacement, torque production, and electromyographic (EMG) activity during static isometric and ballistic elbow extension actions. Subjects sat on a seat with the upper arm to be tested resting vertically just proximal to the olecrannon process on a horizontal elbow support. The forearm was strapped into a separate forearm support (aircraft aluminum) fixed directly to the steel rotatory axis of a light alloy loading wheel so the elbow joint

approximated this rotatory axis. Subjects placed the other arm in a relaxed fashion on their laps during all testing. The upper arm was thus in ~76° of shoulder flexion, while the forearm was maintained at 90° from the horizontal (manipulandum locked for isometric and initial position for ballistic movements) and fixed to the rotatatory arm manipulandum. For movements against preload, weights (equivalent to 10% MVC; see below) were attached to the loading wheel with cording and damped against oscillation with surgical tubing. Amplified torque sensor and displacement potentiometer signals, along with amplified (bandpass 3 Hz to 10 kHz) EMG signals were fed into a 12 bit A/D converter (Dataq Electronics) and then into a microcomputer sampling at 1250 Hz and running CODAS data acquisition software (Dataq Electronics).

Maximal Voluntary Contraction

Isometric maximal voluntary contractions (MVC) were performed initially to determine isometric peak torque such that preloads equal to 0 and 10% of this value could be set for each subject during the ballistic actions. For the isometric actions, the arm support was brought to a position of 90° relative to the horizontal, locked in place, and the torque amplifier was set to zero. The subject then sat in the modified elbow jig apparatus, wherein the upper arm just proximal to the elbow was supported by the main frame and the lower arm was strapped into the aluminum manipulandum. Three maximal isometric elbow extension actions were then performed. Subjects were allowed 1 min recovery between MVCs. Following the determination of MVC peak torque (average of 3 trials), the arm manipulandum was released to allow free movement and the subjects were allowed ~3-5 min rest before the next phase of the experiment.

Ballistic Actions

On ballistic extension trials, subjects extended from the initial 90° position (see Fig. 1) to ~190° where the hands of the subjects contacted a soft foam striking pad

(Century Martial Arts) and thus the amplitude of each extension was approximately 100°. The subjects performed 10 maximal effort ballistic elbow extension movements with no preload on the loading wheel (0% MVC preload) and 10 with a preload equal to ~10% of isometric MVC extension peak torque. The order of performance was randomized for each subject. Previous research (27) has indicated that a 10% MVC preload induces an acceptable background agonist premovement activation level. The subject was asked to maintain joint position (and thus muscle activation in the 10% MVC preload trials) at 90° until ballistic extension. Subjects were instructed to move on their own cue, but as "rapidly and forcefully as possible", to move "explosively and with as high a velocity as possible" (i.e. ballistically) and to strike the foam pad. The subjects were asked to leave the forearm in extension until requested to return the forearm to a resting posture of complete flexion. Subjects were allowed ~30 to 45 s rest between each trial, and ~3-5 min between each ballistic phase of the experiment.

Electromyography

The skin surface over the biceps and triceps brachii of the appropriate arm was shaved, abraded with steel wool, cleaned with alcohol and prepared with 5 EMG (bipolar configuration; 2 biceps, 2 triceps, 1 ground) electrodes (pediatric ECG electrodes; Red Dot, 3M). Maximal agonist EMG activation, and antagonist co-activation were recorded during MVC and ballistic actions.

Data analysis and statistical methods

MVC

All MVC trials were analyzed for peak torque, and maximum biceps and triceps integrated EMG (IEMG) and peak EMG (EMG_{peak}) (from full wave rectified and smoothed signal, see below) activity. Trial averages were used for analysis.

Ballistic Actions

Ballistic movement trials were averaged for each subject and experimental condition (10% preload or no preload) and analyzed for peak torque, rate of torque development, velocity, acceleration, and biceps and triceps EMG activity (IEMG and EMG_{peak}), and movement time (MT) (Fig. 2a shows schematic, and Fig. 2b an example of one 10% MVC preload trial in a karate-trained subject). As well, electromechanical delay (EMD; the time elapsed between the onset of the phasic agonist EMG activation and the initial rise in torque production) and electrodisplacement delay (EDD; time elapsed from the onset of the phasic agonist EMG activation and initial displacement) were calculated on ballistic trials (Fig. 2a). EMG recordings were integrated (IEMG) and full-wave rectified (FWR). The FWR EMG was also smoothed using a 25 point moving average function. EMG_{peak} was obtained directly from the smoothed FWR EMG signal, while the IEMG was subjected to further analysis. Ballistic and isometric IEMG values were divided by the appropriate durations to determine an average EMG (AEMG). The ballistic values were then normalized to the isometric MVC maximum values for each subject. This normalization procedure was applied to agonist AEMG and EMG_{peak}. The torque and displacement recordings were filtered using a moving average function (10 points for torque, 30 for velocity; CODAS software, Dataq Electronics) and differentiated to provide values of instantaneous rate of torque development (RTD) and peak velocity, respectively. The velocity recordings were again smoothed with a 30 point moving average function and differentiated to provide a signal for determination of instantaneous peak acceleration. Occasionally, analysis of ballistic trials would reveal a flexion counter-movement immediately prior to the ballistic elbow extension. Also, under 10% MVC preload conditions, some subjects would sporadically perform a slow ramp rather than a ballistic movement. Thus, twenty-three 0% MVC (10.4% of all trials) and thirty-four 10%

MVC trials (15.5% of all trials) evidencing these characteristics were discarded from the analysis.

Agonist EMG premovement depression (PMD)

Ballistic 10% MVC preload trials were analyzed (raw and rectified EMG) for PMD occurrence, duration, and movement potentiation. The term agonist premovement depression was applied instead of PMS (14) because PMD more closely reflects the documented premovement changes in agonist activation, encompassing both complete quiescence and/or marked depression in the tonic agonist EMG immediately prior to the phasic ballistic discharge (see Fig. 2a,b).

Statistical Analysis

One-factor (group) and 2-factor (group x preload) between, within split-plot analysis of variance (ANOVA), were used to analyze the data, with statistical significance set at $p \leq 0.05$. Descriptive statistics include means ± standard deviation (SD).

Results

Physical Characteristics

In order to determine whether the karate and control groups differed significantly in age and anthropometry, statistical analysis was performed on the anthropometric data. There were no significant differences between groups in age, height, and biceps and triceps skinfolds. The karate subjects had a significantly ($p<0.04$) greater mass and a significantly ($p<0.001$) larger arm girth than control subjects (refer to Table 1).

Isometric Strength (MVC)

Isometric peak torque. Control subjects produced a peak torque of 49.3 ±7.9 N.m (mean, ± SD) and karate practitioners 65.2 ±16.0 N.m. The value for the karate subjects was significantly higher than control ($p<0.005$).

Ballistic Actions

Mechanical Properties

Peak torque. There were main effects for group and preload ($p<0.001$), as well as a group x preload interaction ($p<0.001$) for peak torque production. Karate and control subjects produced peak torque values of 4.3 ±0.8 and 3.3 ±0.7 N·m (NS) and 26.5 ±4.6 and 18.9 ±2.5 N.m ($p<0.01$) at the 0 and 10% preload levels, respectively (Fig. 3, top). There were no main effects for preload or group in the time to peak torque. However, control subjects evidenced a 9% longer time to peak torque in the 10% preload condition.

Rate of torque development (RTD). There were main effects for preload and group ($p<0.001$) in peak RTD. A group x preload interaction just failed to reach significance ($p=0.065$), as karate practitioners increased from 209.9 ±87.0 to 470.0 ±

100.4 $N \cdot m \cdot s^{-1}$ and control subjects from 138.8 ±37.1 to 312.1 ±80.8 $N \cdot m \cdot s^{-1}$ under preload conditions 0 and 10% MVC, respectively (Fig. 3, bottom). There was a main effect for preload ($p<0.02$) but no main effect for group in time to peak RTD, as this value decreased from 116.0 to 93.8 ms from 0 to 10% MVC preload.

Velocity. There was a main effect for preload ($p<0.001$), but no group main effect, as mean peak velocity decreased from 16.4 to 12.3 $rad \cdot s^{-1}$ across preload levels 0 to 10% MVC (Fig.4 top). There was also a main effect for preload ($p<0.001$) but not for group in time to peak velocity. Karate subjects produced peak velocities that were 6 and 4% greater than control at 0 and 10% preloads respectively. Time to peak velocity increased from 190.6 to 231.3 ms from 0% preload to 10% MVC preload, with karate-trained subjects evidencing 7 and 8% shorter intervals than control.

Acceleration. There were main effects for preload ($p<0.001$) and group ($p<0.03$) and a group x preload interaction ($p<0.04$) in peak acceleration values. Karate and control subjects produced peak accelerations of 187.9 ±25.3 and 162.9 ±18.7 $rad \cdot s^{-2}$ ($p<0.01$) and 110.3 ±15.2 and 101.2 ±15.1 $rad \cdot s^{-2}$ ($p<0.05$) in the 0% and 10% preload conditions, respectively (Fig. 4, bottom). There was no main effect for group, but a significant main effect ($p<0.001$) for preload in time to peak acceleration which increased from 109.6 to 148.3 ms from 0 to 10% MVC preload. Karate subjects were 9 and 11% faster in achieving peak torque than control across preload conditions.

Movement time. There was a main effect for preload ($p<0.001$) in movement time. Karate subjects increased from 147.4 ±8.9 to 199.9 ±10.7 ms and control from 161.8 ±23.1 to 210.6 ±30.8 ms across preload conditions 0 and 10% respectively (Fig. 5, top). Karate-trained subjects achieved 9 and 5% shorter movement times than control across the preload conditions.

Electromechanical Delay (EMD). There was a main effect for preload ($p<0.01$)

in EMD with karate and control subjects demonstrating values of 16 ±8.3 and 17.6 ±6 ms and 11.4 ±7.2 and 10.8 ±8.8 ms in the 0% and 10% MVC preload conditions (Fig. 5, middle).

Electrodisplacement Delay (EDD). There were no main effects or interactions in EDD (Fig. 5, bottom) with an average value (collapsed across group and preload) of 54.1 ms being evidenced.

Electromyography

Agonist PMD

PMD occurrence. PMD occurred sporadically in the present experiment. Agonist PMD occurred in 10% of all possible trials in the karate and in 5.4% of all possible trials in the control group. Three of 9 karate subjects evidenced PMD, giving an inter-subject range of 20% to 50%, while 4 of 13 control subjects showed agonist PMD with an inter-subject occurrence range of 10% to 20%. Individual subject PMD percent-of-trial occurrences can be found in Table 2.

PMD duration. The duration of PMD in the karate-trained group was 57.8 ±16.3 ms and 45.4 ±12.9 ms in the control group (NS).

PMD movement potentiation. As detailed above, agonist PMD occurred only sporadically in the present experiment, thus providing a very low number of trials upon which to attempt an analysis of any potentiating effects. Therefore no analysis of movement potentiation could be conducted in this experiment.

Muscle activation

Agonist AEMG. There were no effects for group or preload in AEMG, with karate and control subjects producing 1.57 and 1.74, and 1.65 and 1.62 mV across preload levels. There was no effect for preload in AEMG (ballistic/isometric) ratio (Fig. 6, top). A main effect for group just failed to reach significance ($p=0.079$), with karate-

trained subjects evidencing ratios that were 46 and 29% higher than control across preload levels 0 and 10% MVC.

Agonist EMG_{peak}. There were no effects for group or preload in EMG_{peak} with karate and control subjects evidencing values of 1.37 and 1.41, and 1.30 and 1.62 across preload levels. There were no effects for preload or group ($p=0.098$) in EMG_{peak} values (Fig. 6, bottom). Karate-trained subjects had ratios that were 39 and 20% greater than control at the 0 and 10% MVC preloads, respectively.

Antagonist Activation. There was no effect for group in antagonist AEMG or EMG_{peak}. Karate subjects had mean AEMG values of 0.17 and 0.21 mV and control subjects 0.19 and 0.18 mV across preload levels (NS). EMG_{peak} values showed a main effect for preload ($p<0.05$). Values for karate-trained subjects were 0.29 and 0.25 mV and for control 0.31 and 0.24 mV across preloads 0 and 10% MVC, respectively.

Discussion

In the present study highly skilled karate practitioners evidenced significantly higher peak torques (isometric and ballistic), higher peak rate of torque development, and higher peak accelerations than control subjects. Although karate training involves the repetitive practice of movements requiring high accelerations and necessitates both strength and power (7,15), superior strength and movement performance in karate practitioners during ballistic actions was a heretofore undocumented finding.

The karate practitioners in the present research demonstrated statistically superior performances only on the above-mentioned measures, and, while there were trends towards superior performance, there were no significant differences on other measures such as peak velocity and movement time. As well, there were no differences in the time to reach peak values of torque, RTD, velocity, and acceleration. It must be recalled that the preload set on the loading wheel was the same for all subjects at 0% MVC, but was scaled relative to each subject's isometric strength at the 10% MVC preload condition. The karate-trained subjects had significantly higher (~32%; $p<0.005$) isometric MVC peak torques and thus had correspondingly higher preloads to ballistically extend against during the 10% MVC trials. Therefore, while the relative performance of the karate-trained subjects was statistically equivalent to that of the control subjects on some measures, the absolute performance of the karate practitioners can be considered to be superior. In kinetic terms, karate subjects moved heavier loads at velocities similar to those of control and thus generated greater momentum (mass x velocity) and greater kinetic energy (1/2 mass x velocity^2). As one of the major goals of karate training is to develop the capacity to generate momentum and kinetic energy in the striking hand or foot (9) it should not

then be surprising that the very highly trained karate practitioners in the present study demonstrated superior absolute and relative ballistic movement performance.

This study involved the performance of open-ended ballistic elbow extension movements requiring no volitional termination. That is, the subjects were told to extend their arms ballistically and to strike the soft foam pad with their hands. Thus the movement can be considered to be a single degree of freedom gross motor action. Proper execution of karate technique requires gross motor skill and overall strength and power in conjunction with fine motor skill and accuracy (7,9,15). Also, the expression of superior performance for traditionally strength and power trained athletes has been shown to depend on the similarity of the testing procedures to the training actions (specificity of training; 19). The movement strategies utilized by different subjects may depend on their skill on a given task (4). It is thus possible that the lack of a performance difference between control and karate-trained subjects in measures such as movement time and peak velocity might also be due to the specific skill-oriented nature of karate practice. The task itself may have thus reduced the skill component required in the present research and acted to equalize the relative performance between karate-trained and control subjects on some measures. Had we chosen a more complicated movement more closely replicating karate technique and requiring greater skill (i.e. "strike to the target and just touch it lightly") there may indeed have been even greater differences demonstrated between control and karate-trained subjects.

In the present research, the very low level antagonist co-activation levels and lack of a consistent observation of a triphasic EMG pattern, supposed characteristics of ballistic actions, are likely also due to the task employed. It has been indicated elsewhere that antagonist activity may be markedly decreased when accurate movement termination is not a consideration (24). Further, in experiments involving alteration of limb loading

and amplitude of movement, the second agonist burst has been shown to be subsumed by the prolongation of the initial agonist discharge such that only one agonist discharge may be discerned (1,2). The results of this study fit in well with these previous observations.

It should be recalled at this point that one of the major aims of this experiment was to evaluate the effect that agonist PMD might have on the movement performances of the karate-trained and control subjects. As previous research has indicated that the occurrence of agonist PMD is highly correlated with high peak accelerations and rate of torque development (3,14), the experimental protocol in this study was designed such that the conditions in which subjects could perform a movement involving high peak torques, RTD, accelerations, and velocities could be maximized. Thus, it was hypothesized, the conditions under which PMD would occur should also be maximized. The most agreed upon mechanism through which PMD has been suggested to operate is a supra-spinal neural switching mechanism potentially allowing for a near-synchronous motor unit discharge (12,14). Yet, even though the karate practitioners evidenced superior performance on those movement parameters that should be most affected by agonist PMD (peak torque, RTD, and acceleration), there was no difference between groups in agonist PMD occurrence or duration. In fact, as mentioned in the Results, agonist PMD occurred so infrequently in both groups that no attempt at determining any movement potentiation could be conducted. This raises two questions: 1) why did agonist PMD not occur more frequently in both groups?; and 2) if PMD is a training effect or learned motor response (12,14,23) why did the very highly skilled karate practitioners not evidence a striking difference in PMD occurrence and duration from that of the control group?

With regard to the overall occurrences of agonist PMD in both groups, the values of 10% and 5.4% (karate and control) fall on the low end of the extremely wide range reported in the literature (12,21,23). Also, recent results from a study in our laboratory

making use of a similar methodology and moderately trained karate practitioners evidenced a PMD occurrence rate of ~27% of trials (27). However this investigation revealed an extremely wide range in inter-subject frequency of occurrence, further supporting the extreme variability of this phenomenon (27). As agonist PMD has continually been characterized by its variability of occurrence (12), perhaps it should not be considered surprising that we observed such a low frequency of occurrence in this research.

With regard to the lack of a group effect in agonist PMD, it has been suggested that PMD occurrence may be a training effect or learned motor response (12). Also, as it has been shown that with specific biofeedback training subjects can acquire volitional control over PMD (23), it is surprising that there are no differences between a very highly trained group habituated to ballistic movement performance and an untrained, recreationally active control group. As previously mentioned, the most accepted mechanism through which agonist PMD is thought to act to potentiate the subsequent ballistic actions is to allow for a near-synchronous discharge of motor units (12). An untrained subject, then, attempting to perform a ballistic action necessitating high force and rate of force production would need to make use of a mechanism that would allow for this; namely, synchronous motor unit activation concomitant with short first inter-spike interval (the "ballistic discharge"; 6). However, previous research involving strength-trained subjects has shown that such training can induce specific neural adaptation allowing for a more synchronous motor unit activation during voluntary effort (11) and increased motor unit excitability (18,20). Also, karate training has been shown to induce neural adaptations representative of increased motor unit excitability (13) and thus potentially increased motor unit activation. It is thus possible that the highly trained karate practitioners, already habituated to ballistic actions, had experienced training effects

that encompassed increased motor unit excitability and activation. The normalized (ballistic/isometric) agonist AEMG and EMG_{peak} (Fig. 6) ratios show higher values for the karate-trained group, thus supporting the assertion that karate subjects evidenced a superior capacity to activate the triceps brachii during ballistic actions. When these subjects performed the loaded ballistic actions, then, they did not need to make use of a mechanism (agonist PMD) that would allow for a compressed or synchronous motor unit discharge because they already could fully activate all their motor units. This would explain the lack of a striking PMD occurrence in the trained subjects. Our results, then, suggest that agonist PMD is not a naturally occurring training adaptation or learned motor response. Previous work in our laboratory (27) involving moderately trained karate practitioners evidenced a similar PMD occurrence to that of untrained subjects shown already in the literature, thus also serving to dispel the notion that agonist PMD may occur as a natural training adaptation. We have demonstrated that even in highly skilled subjects agonist PMD occurs extremely variably. Further, while we found superior movement performances in the trained group on those measures that should have been most sensitive to the occurrence of agonist PMD, agonist PMD can not be considered to be the locus of this superior performance. As such, based on the results of the present research involving highly trained karate subjects habituated to ballistic movement, agonist PMD should not be considered to be a naturally occurring training effect or learned motor response.

Another aspect of movement performance may be differences in the consistency and variability of ballistic actions performed by trained and untrained subjects. Subjects possessing greater skill would then evidence decreased variability (5,16). When the coefficients of variation (S.D./mean x 100) for ballistic peak torque were evaluated for karate-trained and control subjects in the present research, there was no significant difference found between groups. This indicates no difference in relative performance

between groups, but because the mean values for the karate-trained subjects were higher than control, there was a difference in absolute force variability, thus suggesting a difference in force variability between the two groups. While the variability of ballistic movement was not the focus of the present study, a future paper will address the issue of ballistic movement variability in karate-trained and control subjects.

Table 1: Physical characteristics of subjects

	Control	Karate
Height (cm)	180.2 ±7.5	179.1 ±5.2
Weight (kg)	83.0 ±13.0	94.3 ±9.3 *
Age (y)	31.9 ±6.6	37.6 ±8.5
Triceps skinfold (mm)	12.1 ±6.6	13.4 ±3.1
Biceps skinfold (mm)	5.4 ±2.0	7.2 ±3.0
Arm girth (cm)	30.7 ±2.7	34.7 ±1.1 **

Values are means ±S.D. Statistical differences between groups are indicated by * ($p<0.04$) and ** ($p<0.01$).

Table 2: Agonist PMD occurrence

Control	Karate
0	0
0	50
0	20
10	0
10	20
40	0
0	0
0	0
0	0
10	
0	
0	
0	

Values are PMD occurrences as a percentage of total trials for individual subjects.

Figure Legends

Figure 1: Diagram of the experimental apparatus showing subject forearm, loading wheel and affixed weights, and striking pad.

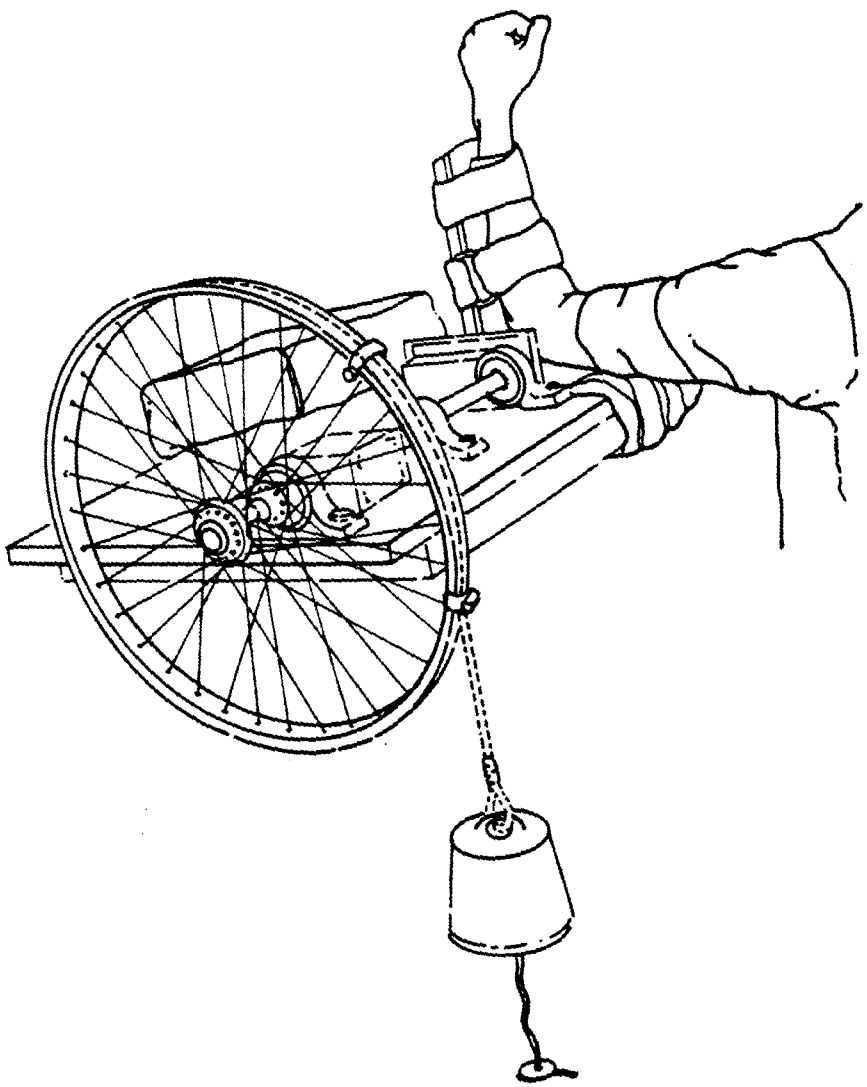
Figure 2: a) Illustrated is a schematic diagram showing simulated data and experimental parameters for a ballistic extension action. b) Shown is an example of one trial of a karate-trained subject performing a 10% MVC preload extension action and evidencing agonist PMD (triceps). Channel 1 is triceps EMG (± 1.25 mV full scale), channel 2 is biceps EMG (± 1.25 mV full scale), channel 3 is torque (40 N·m full scale), and channel 4 is displacement (80 to 200° full scale). Time interval between vertical dotted lines is equal to 25.6 ms and total elapsed time (as indicated by TBR) is 453.2 ms.

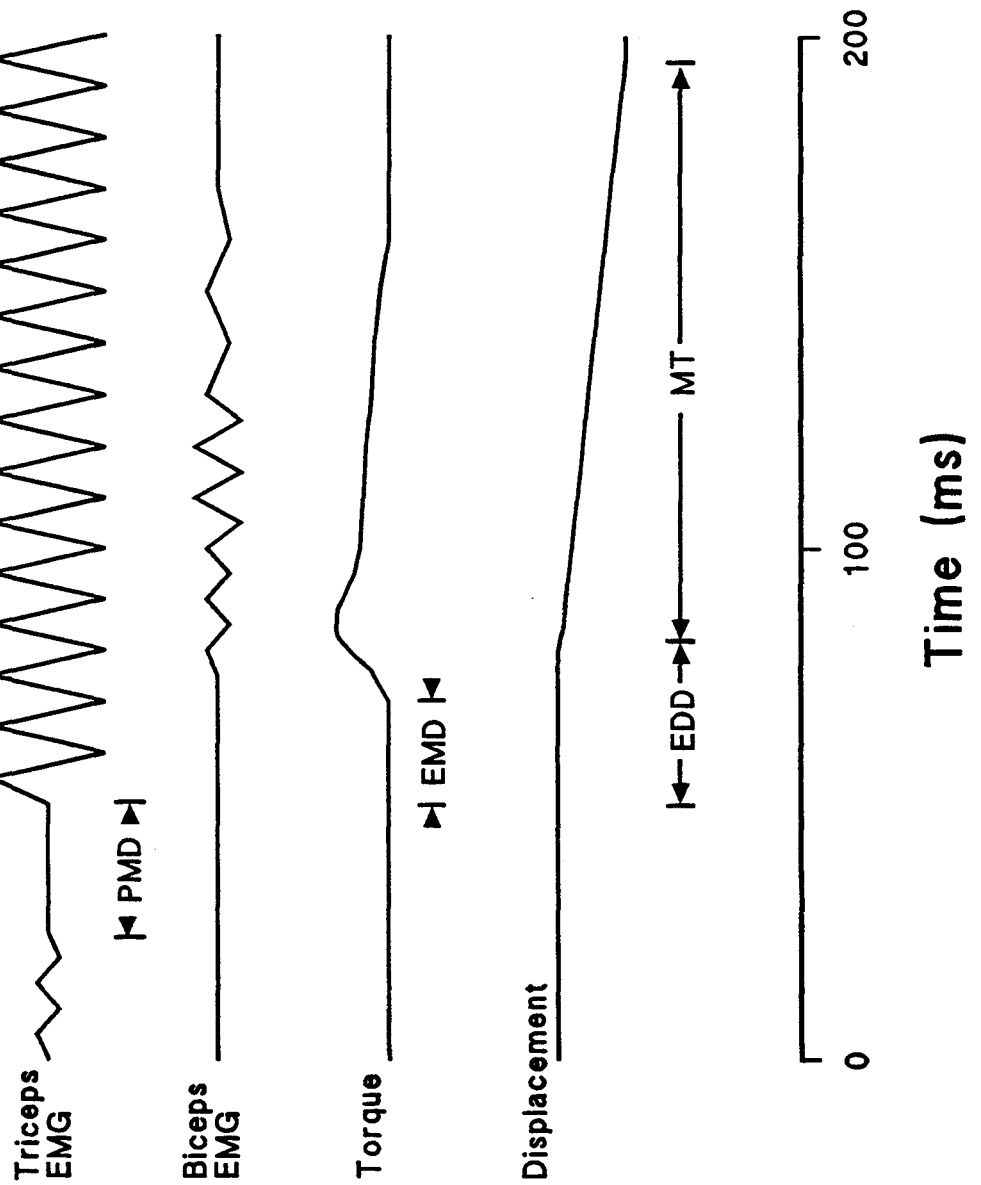
Figure 3: The top panel shows ballistic peak torque values for karate and control subjects. Values are means ± SD. There were main effects for group and preload ($p<0.001$) and a group x preload interaction ($p<0.001$). The bottom panel shows peak RTD. There were main effects for group and preload ($p<0.001$). ** indicates post hoc significant differences at $p<0.01$.

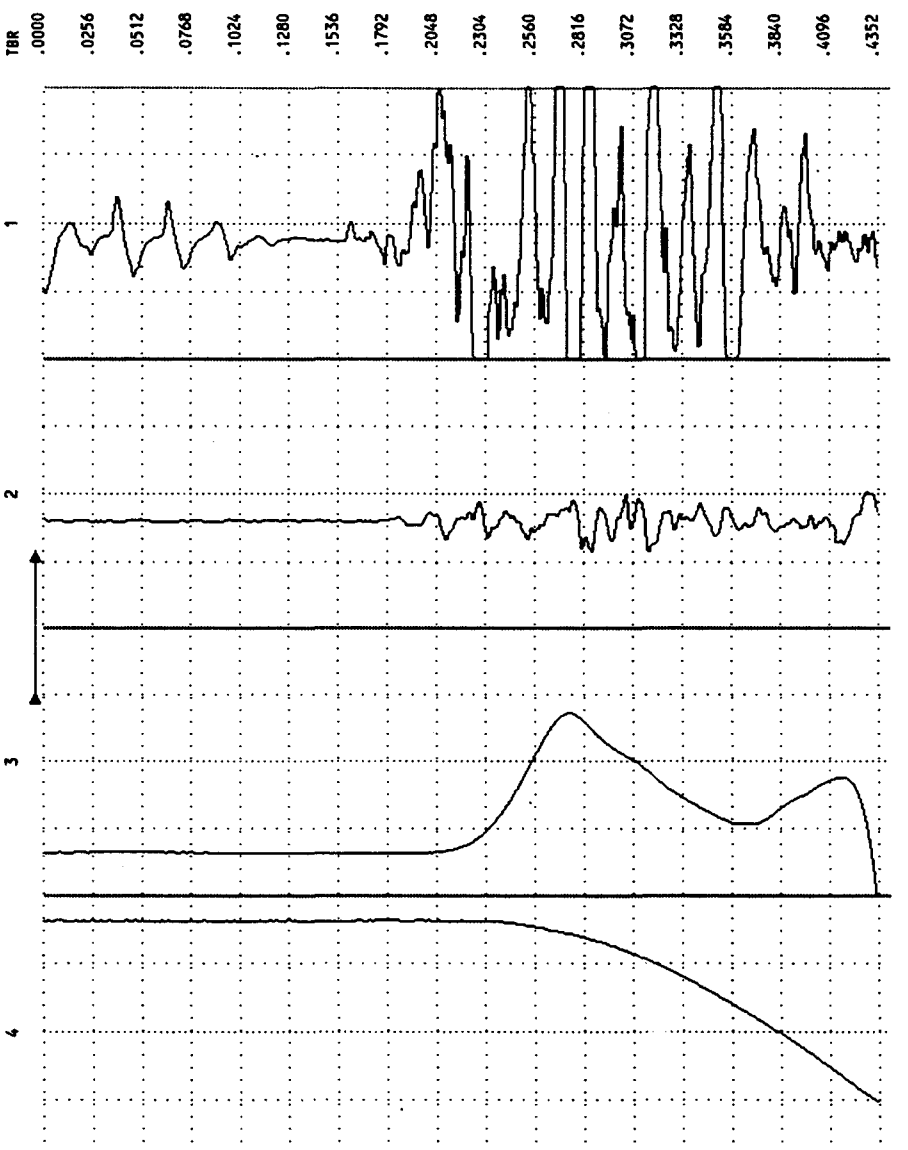
Figure 4: Peak velocities for karate and control subjects are shown at top. Values are means ± SD. There was a main effect for preload in peak velocity ($p<0.001$). The bottom panel is peak acceleration. There were main effects for group ($p<0.03$) and preload ($p<0.001$), and a group x preload interaction ($p<0.04$). Significant differences at $p<0.01$ are shown by **.

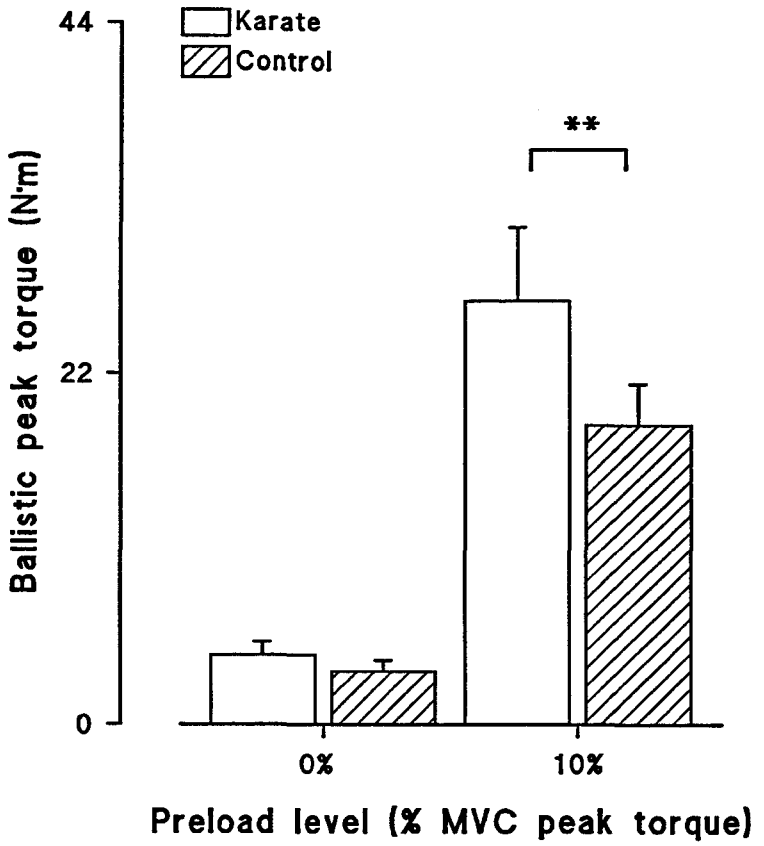
Figure 5: A preload main effect ($p<0.001$) in movement time for karate and control subjects is shown at top. A main effect for preload ($p<0.01$) in EMD is shown in the middle panel. The bottom panel illustrates EDD values for both groups. Values are means ± SD.

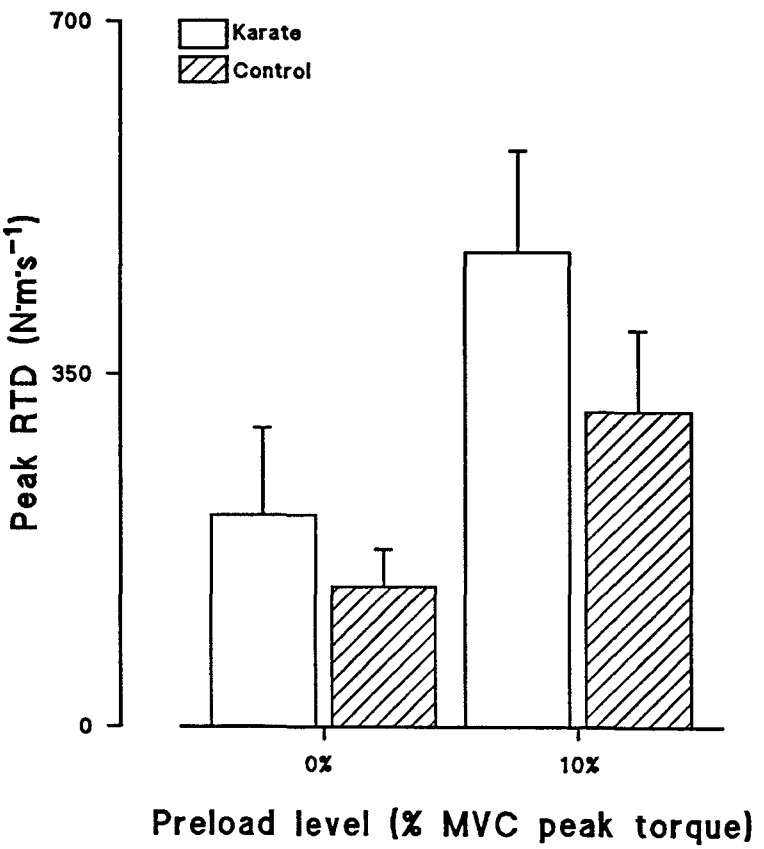
Figure 6: Agonist AEMG (top) and EMG_{peak} (bottom) ballistic/isometric activation ratios for karate and control subjects are illustrated in the figure. There were no main effects for group or preload. Values are means ± SD.

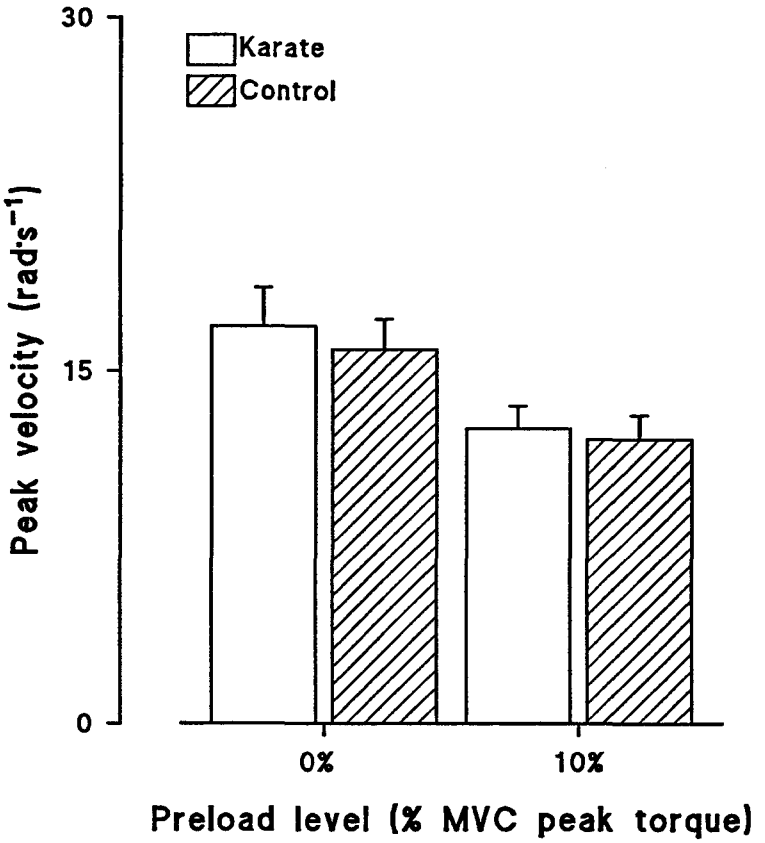


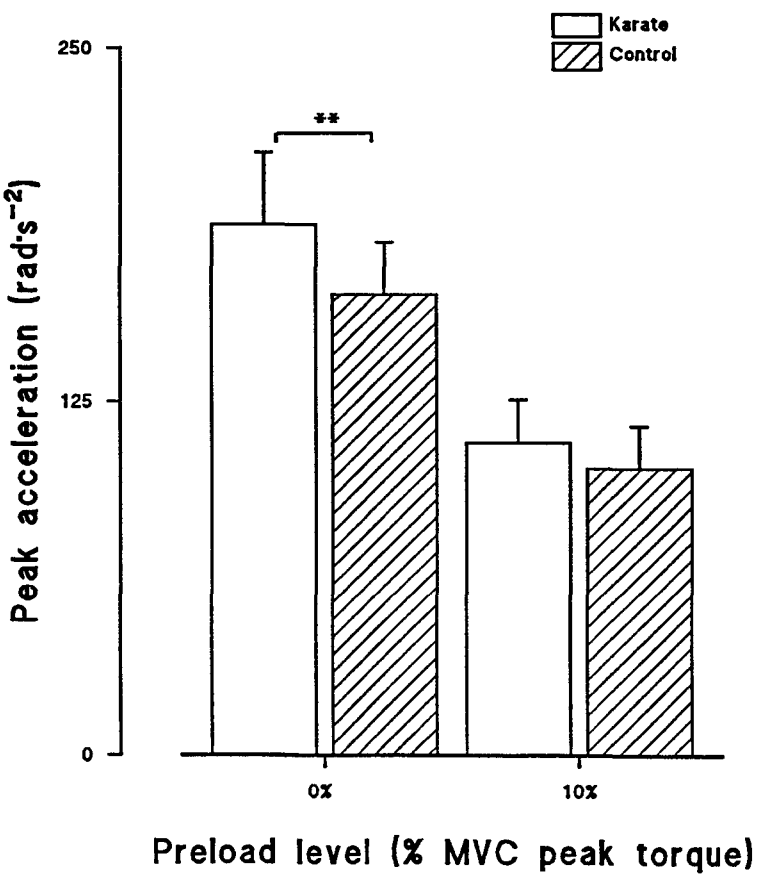

Triceps EMG
PMD
Biceps EMG
Torque
EMD
Displacement
EDD
MT
0
100
200
Time (ms)

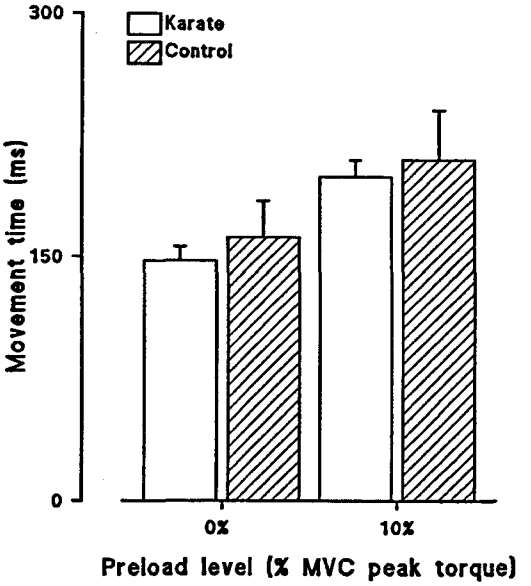

TBR
.0000
.0256
.0512
.0768
.1024
.1280
.1536
.1792
.2048
.2304
.2560
.2816
.3072
.3328
.3584
.3840
.4096
.4352
1
2
3
4

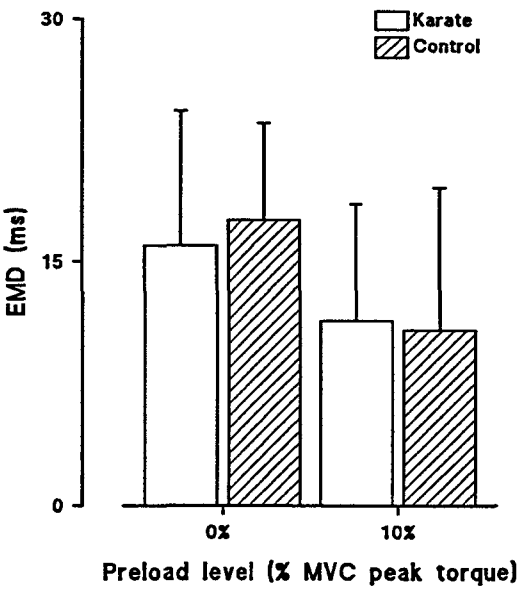

44
22
0
Karate
Control
**
Ballistic peak torque (N'm)
0%
10%
Preload level (% MVC peak torque)

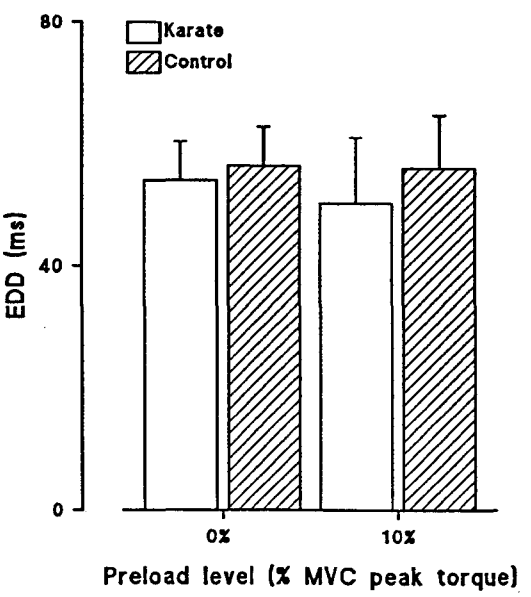

700
350
0
Karate
Control
Peak RTD (N'm's^{-1})
0%
10%
Preload level (% MVC peak torque)

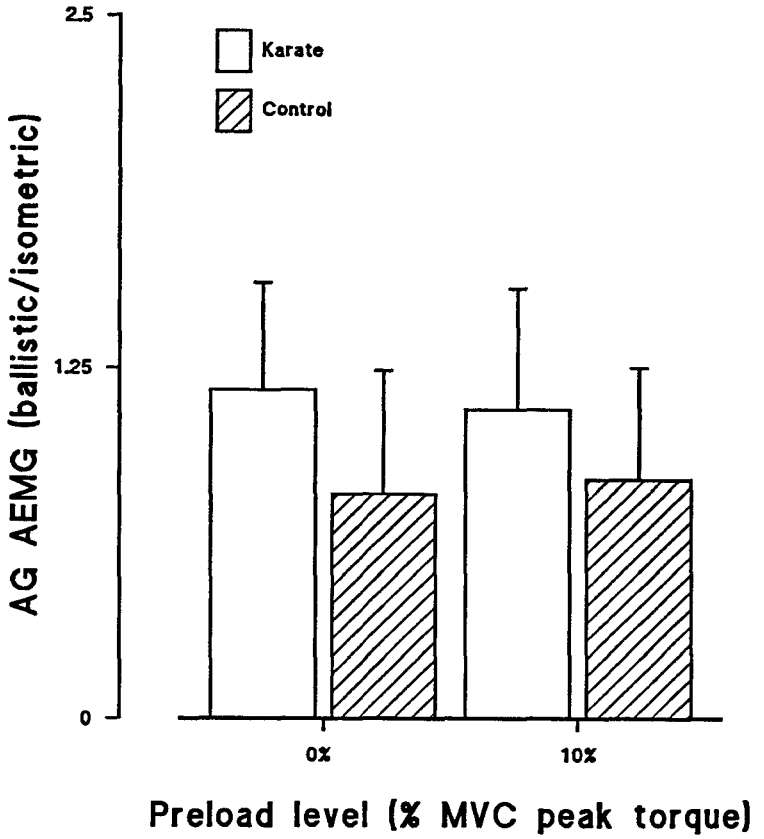

30
15
0
Karate
Control
Peak velocity (rad·s⁻¹)
0%
10%
Preload level (% MVC peak torque)

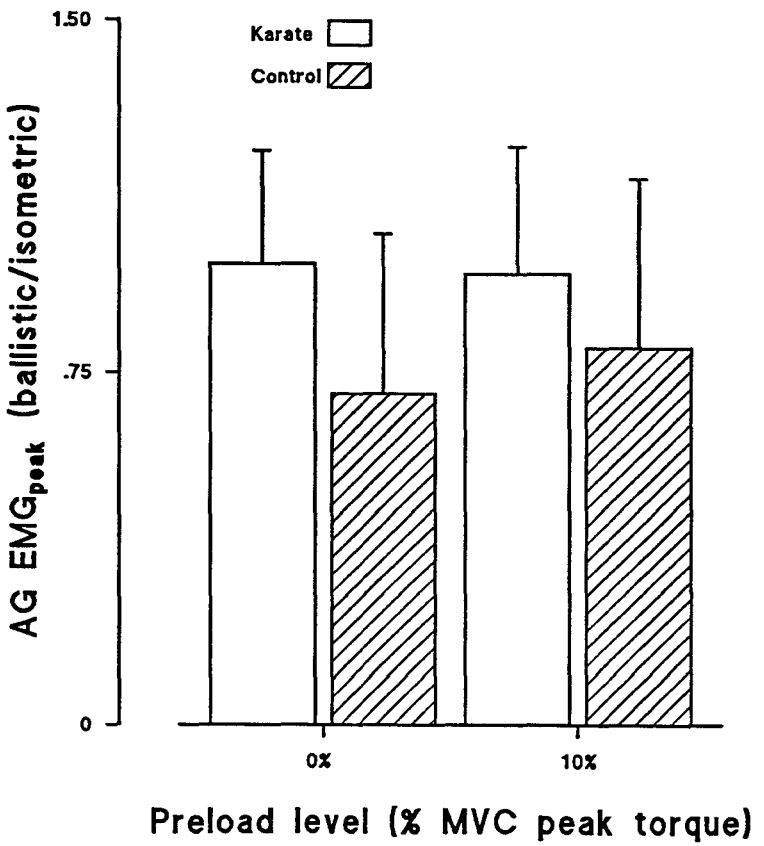

250
125
0
Karate
Control
**
Peak acceleration (rad·s⁻²)
0%
10%
Preload level (% MVC peak torque)


300
150
0
Movement time (ms)
Karate
Control
0%
10%
Preload level (% MVC peak torque)


30
15
0
EMD (ms)
Karate
Control
0%
10%
Preload level (% MVC peak torque)


80
40
0
EDD (ms)
Karate
Control
0%
10%
Preload level (% MVC peak torque)


2.5
1.25
0
Karate
Control
AG AEMG (ballistic/isometric)
0%
10%
Preload level (% MVC peak torque)


1.50
.75
0
Karate
Control
AG EMGpeak (ballistic/isometric)
0%
10%
Preload level (% MVC peak torque)

Acknowledgments
John Moroz provided technical assistance. This study was supported by the Natural Sciences and Engineering Research Council of Canada.

References

1. Benecke R., H.M. Meinck, and B. Conrad. Rapid goal-directed elbow flexion movements: limitations of the speed control system due to neural constraints. *Exp. Brain Res.* 59: 470-477, 1985.

2. Brown, S.H., and J.D. Cooke. Amplitude- and instruction-dependent modulation of movement-related electromyogram activity in humans. *J. Physiol.* 55: 523-527, 1984.

3. Conrad, B., R. Benecke, and M. Goehmann. Premovement silent period in fast movement initiation. *Exp. Brain Res.* 51:310-313, 1983.

4. Corcos, D.M., G.L. Gottlieb, and G.C. Agarwal. Accuracy constraints upon rapid elbow movements. *J. Motor Behav.* 20(3): 255-272, 1988.

5. Darling, W.G., and J.D. Cooke. Changes in the variability of movement trajectories with practice. *J. Motor Behav.* 19(3): 291-309, 1987.

6. Freund, H.J. Motor unit and muscle activity in voluntary motor control. *Physiol. Rev.* 63: 387-436, 1983.

7. Funakoshi, G. *Karate-do Kyohan* (2nd Edition). Kodansha Int.: Tokyo, 1990.

8. Gatev, V. Role of inhibition in the development of motor co-ordination in early childhood. *Develop. Med. Child Neurol.* 14:336-341, 1972.

9. Ingber, L. *Karate kinematics and dynamics.* Unique Publications Inc.: California, 1981.

10. Kawahats, K. and M. Miyashita. Electromyogram premotion silent period and tension development in human muscle. *Experimental Neurology* 82:287-302, 1983.

11. Milner-Brown, H.S., R.B. Stein, and R.G. Lee. Synchronization of human motor units: possible roles of exercise and supraspinal reflexes. *EEG Clin. Neurophysiol.* 38: 145-254, 1975.

12. Moritani, T. Neuromuscular adaptations during the acquisition of muscle strength, power and motor tasks. *J. Biomechanics* 26(suppl.1):95-107, 1993.

13. Mortimer, J.A., and D.D. Webster. Dissociated changes of short- and long-latency myotatic responses prior to a brisk voluntary movement in normals, in karate experts, and in Parkinsonian patients. In: *Advances in neurology vol. 39: motor control mechanisms in health and disease*, J.E. Desmedt (Ed.). New York: Raven, 541-554, 1983.

14. Mortimer, J.A., P. Eisenberg, and S.S. Palmer. Premovement silence in agonist muscles preceding maximum efforts. *Experimental Neurology* 98:542-554, 1987.

15. Nakayama, M. *Dynamic Karate.* Kodansha International: Tokyo, 1983.

16. Newell, K.M., and D.M. Corcos. Issues in variability and motor control. In: *Variability and motor control,* K.M. Newell and D.M. Corcos (Eds.). Champaign, Illinois: Human Kinetics, 1-12, 1993.

17. Palmer, S.S., P. Eisenberg, and J.A. Mortimer. Premovement silence of EMG activity prior to movement in Parkinson patients. *EMG Clin. Neurophysiol.* 31:425-431, 1991.

18. Sale, D.G. Neural adaptation to strength training. In: *Strength and power in sport,* P.V. Komi (Ed.). London: Blackwell Scientific, 249-265, 1992.

19. Sale, D.G., and J.D. MacDougall. Specificity in strength training: a review for the coach and athlete. *Can. J. Appl. Sports Sci.* 6(2): 87-92, 1981.

20. Sale, D.G., J.D. MacDougall, A.R.M. Upton, and A.J. McComas. Effect of strength training upon motoneuron excitability in man. *Med. Sci. Sports Exerc.* 15(1): 57-62, 1983.

21. Tanii, K. Occurrence of a rhythmic slower wave in EMG prior to a rapid voluntary movement. *EEG Clin. Neurophysiol.* 57:435-440, 1984.

22. Walter, C.B. The influence of agonist premotor silence and the stretch-shortening cycle on contractile rate in active skeletal muscle. *Eur. J. Appl. Physiol.* 57:577-582, 1988.

23. Walter, C.B. Voluntary control of agonist premotor silence preceding limb movements of maximal effort. *Perceptual and Motor Skills* 69:819-826, 1989.

24. Waters, P., and P.L. Strick. Influence of 'strategy' on muscle activity during ballistic movements. *Brain Res.* 207: 189-194, 1981.

25. Yabe, K. Premotion silent period in rapid voluntary movement. *J. Appl. Physiol.*

41(4):470-473, 1976a.

26. Yabe, K. Electromyographic silent period preceding a rapid voluntary movement. In: *Proc. 5th Intern. Congr. Biomechanics,* P.V. Komi (Ed.). Baltimore, Md: University Park Press, pp. 75-81, 1976b.

27. Zehr, E.P., and D.G. Sale. Ballistic elbow extension movements in moderately trained karate practitioners: peak torque, velocity, acceleration, and the agonist pre-movement silence. *Med. Sci. Sports Exerc.* 25(5):S131, 1993.

Appendix

Appendices 1-3. Tabled method error, Pearson r correlation coefficients, and significant p values for day 1-day 2 reliability calculations (6 control subjects).

Appendices 4-9. Tabled raw data (subject means) for isometric and ballistic (0% MVC and 10% MVC preload) conditions.

Appendix 1

DAY 1-DAY 2 ISOMETRIC RELIABILITY CALCULATIONS

VARIABLE	METHOD ERROR	PEARSON R	P VALUE
PEAK TORQUE	3.1	0.978	NS
AG IEMG	38.1	0.596	NS
AG EMG_{PEAK}	47.6	0.522	NS
ANT IEMG	29.5	0.390	NS
ANT EMG_{PEAK}	23.7	0.881	0.017

Appendix 2:

DAY 1-DAY 2 RELIABILITY CALCULATIONS FOR 0% MVC PRELOAD

VARIABLE	METHOD ERROR	PEARSON R	P VALUE
EMD	24.3	-0.311	.048
EDD	12.7	-0.246	NS
MT	5.5	0.544	NS
PEAK VELOCITY	3.4	0.864	NS
PEAK ACCELER.	4.0	0.932	NS
PEAK TORQUE	9.2	0.829	NS
PEAK RTD	14.1	0.175	NS
AG IEMG	25.8	0.666	NS
AG EMG_{peak}	29	0.537	NS
ANT IEMG	30.9	0.495	NS
ANT EMG_{peak}	21.8	0.785	0.013

Appendix 3:

DAY 1-DAY 2 RELIABILITY CALCULATIONS FOR 10% MVC PRELOAD

VARIABLE	METHOD ERROR	PEARSON R	P VALUE
EMD	47.8	-0.482	NS
EDD	9.5	0.026	NS
MT	3.9	0.425	NS
PEAK VELOCITY	2.8	0.9	NS
PEAK ACCELER.	6.5	0.829	NS
PEAK TORQUE	7.2	0.877	NS
PEAK RTD	11.1	0.764	NS
AG IEMG	23.9	0.719	NS
AG EMG_{peak}	29.0	0.6	NS
ANT IEMG	48.7	0.110	NS
ANT EMG_{peak}	36.8	0.201	NS

Appendix 4: Isometric data for karate-trained subjects

Isometric: Karate	PGZ	EPZ	MS	JH	JP	SL	TS	PG	JW
Peak Torque (N.m)	79.23	63.71	50.21	69.04	92.13	72.27	65.02	57.24	37.67
AG IEMG (mV.s)	1.023	0.769	0.482	0.515	0.591	0.809	0.763	0.491	0.315
AG PEAK EMG (mV)	2.054	1.931	1.045	1.139	1.378	1.703	1.660	0.998	0.937
ANT IEMG (mV.s)	0.358	0.340	0.240	0.402	0.307	0.175	0.187	0.128	0.339
ANT PEAK EMG (mV)	0.821	0.461	0.322	0.716	0.601	0.324	0.364	0.238	0.754

NB: Time period over which AG and ANT IEMG were calculated is equal to 1 s.

Appendix 4: Isometric data for karate-trained subjects

Isometric: Karate	PGZ	EPZ	MS	JH	JP	SL	TS	PG	JW
Peak Torque (N.m)	79.23	63.71	50.21	69.04	92.13	72.27	65.02	57.24	37.67
AG IEMG (mV.s)	1.023	0.769	0.482	0.515	0.591	0.809	0.763	0.491	0.315
AG PEAK EMG (mV)	2.054	1.931	1.045	1.139	1.378	1.703	1.660	0.998	0.937
ANT IEMG (mV.s)	0.358	0.340	0.240	0.402	0.307	0.175	0.187	0.128	0.339
ANT PEAK EMG (mV)	0.821	0.461	0.322	0.716	0.601	0.324	0.364	0.238	0.754

NB: Time period over which AG and ANT IEMG were calculated is equal to 1 s.

Appendix 5: Isometric data for control subjects

Isometric: Control	JM	SR	BH	TO	JD	JLA	RD	KD	BJ
Peak Torque (N.m)	54.82	37.59	44.19	46.00	60.64	49.35	54.32	36.01	62.61
AG IEMG (mV.s)	0.711	0.566	0.604	0.677	1.213	0.580	2.310	1.586	0.902
AG PEAK EMG (mV)	1.589	1.137	2.446	1.394	2.585	1.314	5.728	3.182	2.242
ANT IEMG (mV.s)	0.213	0.345	0.182	0.294	0.355	0.123	0.297	0.181	0.161
ANT PEAK EMG (mV)	0.301	0.638	0.434	0.382	0.553	0.236	0.883	0.672	0.349

Isometric: Control (cont'd.)	JL	AE	TL	DE
Peak Torque (N.m)	48.35	44.91	52.77	49.56
AG IEMG (mV.s)	0.546	2.140	0.465	1.240
AG PEAK EMG (mV)	1.064	4.203	1.000	2.486
ANT IEMG (mV.s)	0.152	0.320	0.187	0.142
ANT PEAK EMG (mV)	0.314	0.616	0.222	0.267

NB: Time period over which AG and ANT IEMG were calculated is equal to 1 s.

Appendix 6: Ballistic 0% MVC preload data for karate-trained subjects

Ballistic 0%MVC: Karate-trained subjects	PGZ	EPZ	MS	JH	JP	SL	TS	PG	JW
EMD (ms)	0.00	16.48	15.38	23.80	14.08	16.30	13.36	30.72	13.80
EDD (ms)	44.91	49.20	60.53	56.80	48.96	53.10	57.44	64.72	50.40
AG DURATION (ms)	201.71	194.80	212.89	189.00	195.76	193.40	208.16	203.52	198.80
AG IEMG (mV.s)	0.142	0.151	0.096	0.139	0.136	0.154	0.256	0.084	0.115
AG PEAK EMG (mV)	1.169	1.775	0.903	1.349	1.365	1.486	2.265	0.867	1.118
ANT DURATION (ms)	182.17	205.20	194.13	175.60	189.68	169.20	202.96	203.52	193.10
ANT IEMG (mV.s)	0.013	0.046	0.042	0.048	0.026	0.024	0.027	0.018	0.049
ANT PEAK EMG (mV)	0.259	0.308	0.271	0.371	0.240	0.178	0.455	0.176	0.310
MT (ms)	156.80	159.76	152.36	132.20	146.80	140.30	150.72	138.80	148.40
PEAK VELOCITY (rad.s-1)	16.03	14.22	16.48	19.83	18.26	17.88	15.61	17.12	16.97
TIME TO PK. VELOCITY (ms)	182.51	193.36	195.11	170.60	178.64	174.10	192.16	186.96	181.70
PK. ACCELERATION (rad.s-2)	168.00	164.60	171.56	242.25	170.80	208.38	178.30	199.80	187.25
TIME TO PK. ACC. (ms)	84.91	79.68	121.51	121.00	111.52	117.50	102.48	102.32	96.80
PEAK TORQUE (N.m)	3.71	3.48	3.07	4.75	1.192na	5.36	5.15	4.82	4.25
TIME TO PT (ms)	89.37	113.92	120.62	124.00	98.80	111.70	175.68	131.36	113.30
PEAK RTD (N.m.s-1)	178.43	206.80	112.78	152.00	145.90	178.00	402.30	270.10	242.38
TIME TO PK. RTD (ms)	89.14	177.68	85.78	101.20	60.24	69.00	185.12	154.00	155.20

Appendix 7: Ballistic 10% MVC preload data for karate-trained subjects

Ballistic 10% MVC: Karate-trained subjects	PGZ	EPZ	MS	JH	JP	SL	TS	PG	JW
PMD DURATION (ms)	na	54.08	52.40	na	72.40	na	na	na	na
EMD (ms)	0.00	17.20	12.00	2.67	15.28	8.98	13.28	9.51	23.43
EDD (ms)	30.29	55.44	59.29	49.73	50.08	39.56	63.92	43.87	59.43
AG DURATION (ms)	234.29	255.44	259.64	252.93	249.68	237.87	272.88	236.44	235.54
AG IEMG (mV.s)	0.181	0.163	0.115	0.191	0.171	0.190	0.138	0.119	0.152
AG PEAK EMG (mV)	1.567	1.481	0.922	1.412	1.207	1.578	1.070	1.005	1.431
ANT DURATION (ms)	240.80	255.36	252.09	252.93	250.00	233.42	272.88	236.44	235.54
ANT IEMG (mV.s)	0.021	0.066	0.050	0.154	0.029	0.030	0.035	0.021	0.057
ANT PEAK EMG (mV)	0.187	0.347	0.245	0.391	0.207	0.173	0.221	0.138	0.300
MT (ms)	210.51	208.88	200.36	203.33	199.60	198.31	208.96	192.58	176.11
PEAK VELOCITY (rad.s-1)	11.78	11.18	12.97	13.51	12.56	12.98	11.15	12.80	13.85
TIME TO PK. VELOCITY (ms)	200.57	238.72	236.80	226.80	224.48	212.71	240.32	212.27	212.57
PK. ACCELERATION (rad.s-2)	111.14	89.80	100.89	117.17	102.80	116.33	92.60	128.22	133.86
TIME TO PK. ACC. (ms)	136.57	96.96	150.58	161.20	142.48	133.60	155.68	139.47	141.03
PEAK TORQUE (N.m)	29.38	26.73	20.28	32.77	34.11	31.58	24.21	25.72	21.16
TIME TO PT (ms)	104.10	96.08	128.98	141.60	103.68	116.00	123.84	133.33	112.00
PEAK RTD (N.m.s-1)	500.00	558.40	318.11	424.33	667.90	490.11	402.60	440.44	428.43
TIME TO PK. RTD (ms)	77.90	77.44	91.64	93.07	77.60	85.42	94.80	80.18	92.23

Appendix 8: Ballistic 0% MVC preload data for control subjects

Ballistic 0%MVC: Control subjects	JM	SR	BH	TO	JD	JLA	RD	KD	BJ
EMD (ms)	15.55	7.12	15.73	11.92	16.98	30.20	14.24	20.89	21.42
EDD (ms)	47.53	50.72	55.20	57.76	58.40	60.50	52.24	55.73	72.89
AG DURATION (ms)	199.22	203.36	179.47	209.76	205.96	200.10	198.24	225.87	304.09
AG IEMG (mV.s)	0.092	0.096	0.201	0.115	0.157	0.094	0.242	0.159	0.094
AG PEAK EMG (mV)	0.933	0.914	2.502	1.135	1.647	0.992	2.143	1.264	0.943
ANT DURATION (ms)	217.33	188.32	186.40	188.72	168.00	185.70	194.00	223.64	233.87
ANT IEMG (mV.s)	0.045	0.058	0.023	0.042	0.051	0.012	0.034	0.059	0.039
ANT PEAK EMG (mV)	0.429	0.361	0.190	0.310	0.481	0.102	0.466	0.511	0.264
MT (ms)	170.22	153.36	146.58	159.19	152.00	145.90	146.00	174.58	231.20
PEAK VELOCITY (rad.s-1)	13.51	16.79	16.52	15.75	17.57	15.82	16.71	14.89	15.22
TIME TO PK. VELOCITY (ms)	185.87	187.28	183.20	177.14	193.33	188.50	179.92	209.96	286.40
PK. ACCELERATION (rad.s-2)	147.11	159.50	176.67	158.80	169.89	158.50	190.70	138.22	135.67
TIME TO PK. ACC. (ms)	79.64	91.36	107.82	116.16	105.24	100.60	119.84	101.42	217.87
PEAK TORQUE (N.m)	2.89	3.50	4.12	3.17	2.83	3.40	3.88	1.97	1.83
TIME TO PT (ms)	101.11	130.00	104.89	106.24	104.71	99.30	102.16	103.91	231.56
PEAK RTD (N.m.s-1)	147.33	168.10	126.33	115.80	113.33	167.13	122.30	122.44	54.56
TIME TO PK. RTD (ms)	132.44	134.16	69.33	83.52	71.91	89.20	87.44	106.49	178.13

Appendix 8 cont'd:

Ballistic 0% MVC: Control subjects (cont'd)	JL	AE	TL	DE
EMD (ms)	12.36	25.16	18.88	17.78
EDD (ms)	53.51	53.87	61.28	53.07
AG DURATION (ms)	205.33	195.38	225.84	212.98
AG IEMG (mV.s)	0.070	0.218	0.168	0.176
AG PEAK EMG (mV)	0.594	2.084	1.637	1.632
ANT DURATION (ms)	206.40	179.29	203.12	206.67
ANT IEMG (mV.s)	0.014	0.042	0.029	0.046
ANT PEAK EMG (mV)	0.104	0.298	0.183	0.284
MT (ms)	157.33	141.49	165.68	159.91
PEAK VELOCITY (rad.s-1)	15.56	18.26	16.16	14.22
TIME TO PK. VELOCITY (ms)	191.02	176.89	207.60	196.62
PK. ACCELERATION (rad.s-2)	166.11	200.22	151.90	163.89
TIME TO PK. ACC. (ms)	91.20	122.93	133.60	107.64
PEAK TORQUE (N.m)	3.76	3.72	3.74	3.73
TIME TO PT (ms)	90.49	124.44	156.80	111.82
PEAK RTD (N.m.s-1)	122.67	180.56	188.90	174.67
TIME TO PK. RTD (ms)	74.31	61.96	190.16	181.24

Appendix 9: Ballistic 10% MVC preload data for control subjects

Ballistic 10% MVC: Control subjects	JM	SR	BH	TO	JD	JLA	RD	KD	BJ
PMD DURATION (ms)	na	na	na	68.80	46.40	38.60	na	na	na
EMD (ms)	13.33	5.14	6.20	3.60	6.29	31.20	9.69	8.64	0.00
EDD (ms)	57.07	52.80	45.10	52.20	50.40	63.73	51.64	48.72	72.40
AG DURATION (ms)	253.87	243.20	236.50	265.70	256.46	262.80	247.20	272.00	379.00
AG IEMG (mV.s)	0.150	0.144	0.203	0.200	0.197	0.105	0.341	0.228	0.114
AG PEAK EMG (mV)	1.262	1.250	2.061	1.526	1.834	0.839	2.850	1.646	0.864
ANT DURATION (ms)	261.84	236.34	234.60	241.30	235.31	252.67	231.64	265.36	366.70
ANT IEMG (mV.s)	0.054	0.062	0.029	0.062	0.062	0.015	0.047	0.021	0.053
ANT PEAK EMG (mV)	0.272	0.335	0.172	0.320	0.325	0.236	0.251	0.131	0.219
MT (ms)	210.00	190.40	195.50	213.50	209.71	199.07	195.56	223.28	306.60
PEAK VELOCITY (rad.s-1)	10.49	13.45	12.68	11.41	11.92	11.37	12.52	11.17	10.74
TIME TO PK. VELOCITY (ms)	226.84	222.29	215.80	237.40	234.74	225.60	221.51	246.56	355.70
PK. ACCELERATION (rad.s-2)	85.67	113.57	115.75	94.38	95.00	96.33	118.78	77.70	78.00
TIME TO PK. ACC. (ms)	122.67	145.37	141.20	158.10	146.63	146.80	144.00	144.48	287.20
PEAK TORQUE (N.m)	21.09	16.55	18.75	17.13	20.11	17.87	23.38	13.63	18.39
TIME TO PT (ms)	109.60	116.34	126.40	126.10	118.74	116.00	117.60	103.20	257.40
PEAK RTD (N.m.s-1)	384.44	288.86	285.00	263.75	269.57	339.67	416.11	226.30	126.25
TIME TO PK. RTD (ms)	84.62	116.23	83.20	92.30	84.69	85.73	87.91	80.00	203.40

Appendix 9 cont'd:

Ballistic 10% MVC: Control subjects (cont'd)	JL	AE	TL	DE
PMD DURATION (ms)	48.00	na	na	na
EMD (ms)	16.30	7.44	7.64	25.10
EDD (ms)	61.00	45.12	59.20	68.40
AG DURATION (ms)	256.10	230.64	258.22	281.40
AG IEMG (mV.s)	0.090	0.269	0.211	0.295
AG PEAK EMG (mV)	0.710	2.372	1.738	2.139
ANT DURATION (ms)	257.60	228.24	236.53	281.40
ANT IEMG (mV.s)	0.018	0.052	0.040	0.063
ANT PEAK EMG (mV)	0.112	0.273	0.206	0.281
MT (ms)	196.60	185.52	199.02	213.00
PEAK VELOCITY (rad.s-1)	12.26	13.29	13.70	11.74
TIME TO PK. VELOCITY (ms)	230.60	207.36	237.33	255.20
PK. ACCELERATION (rad.s-2)	103.63	117.50	119.84	99.00
TIME TO PK. ACC. (ms)	144.80	132.40	159.56	166.20
PEAK TORQUE (N.m)	18.72	20.09	21.66	17.89
TIME TO PT (ms)	110.60	98.32	133.16	130.50
PEAK RTD (N.m.s-1)	391.25	384.60	364.44	317.38
TIME TO PK. RTD (ms)	89.40	75.52	103.91	140.10