UNIVERSITY OF ALBERTA

THE EFFECTS OF LOAD AND UNEXPECTED DELAY, ON BRAKING-AND-ACCELERATING VOLUNTARY FOREARM MOVEMENTS

BY

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A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science.

DEPARTMENT OF PHYSICAL EDUCATION AND SPORT STUDIES

Edmonton, Alberta

FALL 1992

ISBN 0-315-77290-5

UNIVERSITY OF ALBERTA

RELEASE FORM

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TITLE OF THESIS: The effects of load and unexpected delay, on braking-and-accelerating voluntary forearm movements.

DEGREE: Master of Science

YEAR THIS DEGREE GRANTED: 1992

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This thesis is dedicated to my family for their support and encouragement.

ABSTRACT

The purpose of this study was to examine the role of load and unexpected delays of the stretch stimulus onset during braking-and-accelerating forearm movements. The investigation focused on the effects of these factors on mechanical performance and the reflex responses of the biceps brachii and triceps brachii.

A stretch stimulus, caused by the free fall of a weight, was applied to the elbow flexors of the dominant arm of thirty male subjects. The subjects were warned of the imminent fall of the weight and were instructed to brake the movement of the load and try to move it backward as fast as possible. No visual or auditory cues were provided. To test the effect of unexpected delay, the weights in the last two trials performed by each subject were released four seconds after the warning instead of the usual two seconds expected in the previous forty trials. Each subject was tested with one of three load intensities (11, 19 or 27% of his maximum isometric strength).

The angular displacement, joint power and rectified EMG data were averaged for five 'regular' and two 'delayed' trials of each subject. Subsequently, split-plot factorial ANOVAs were conducted. The findings suggested that mechanical performance (reflected in the maximum angular displacement during the braking phase of the movement) deteriorated when the load increased and when the stretch stimulus was delayed. The reflex EMG response of the biceps was enhanced by

increased loads and was reduced by unexpected delays. The data provided support for the notion of a preparatory set of limited duration in the flexors. The reflex EMG responses of the triceps were not affected by the type (expected or unexpectedly delayed) of stimulus, and light and medium loads, but increased sharply when heavy loads were applied. The observed reflex coactivation patterns suggest the existence of two different preparatory sets, one for the flexors and a second, not affected by delays, for the extensors. Negative joint muscle power increased proportionally to the increases in the load and remained unaffected by delays but was shown to be a poor indicator of performance in this particular experimental task.

ACKNOWLEDGEMENTS

The author wishes to express his gratitude to his committee members, Dr. P. Gervais, Dr. J. Bobet and Dr. J. Yang for their advice and their critical evaluation of the thesis. The author would also like to thank Mr. A. Travlos and Mr. J. Douvis for their invaluable assistance, as well as all the volunteers who participated as subjects in the experiment.

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CHAPTER I

INTRODUCTION

Braking-and-accelerating movements, also referred to in the literature as stretch/shortening cycles, are a special category of tasks commonly found in sports and in everyday life. These movements have two distinct phases: a braking phase in which the objective is to slow down and eventually stop a moving load (external or the body weight itself) and an acceleration phase in which the objective is to accelerate the load in the opposite direction. The first phase involves primarily eccentric contraction while the second phase involves concentric contraction.

As mentioned earlier, braking-and-accelerating tasks are commonly found in sports activities. In these activities the athlete's ability to perform the task successfully may reduce the risk of injury and allow the efficient transition from the decelerating phase of one skill into the accelerating phase of another. Examples of such activities are receiving and passing a medicine ball, or landing after a front somersault and immediately starting pushing off to perform a second one. In terms of performance, braking-and-accelerating movements can be examined from many different perspectives. With respect to safety and efficiency however, a very good performance criterion is maximum angular displacement, in other words, how much the limb that receives the load will be displaced during the deceleration phase. For instance, if one fails to stop a stretching movement, the excessive limb displacement might lead to a sprained joint.

The power output of a movement is of great importance in sports since one is often interested not only in the force that is exerted, but also in the velocity of the point which is acted upon by the force. Force and velocity are two major aspects of braking-and-accelerating movements and naturally power - as their product - can provide valuable information. Factors that play an important role in the mechanical power output are the mechanics of the particular movement and the force-velocity-length relationship in the participating muscles.

Receiving a falling weight has been the task many researchers have selected in order to examine braking-and-accelerating movements (Lacquaniti et al., 1991; Lacquaniti and Maioli, 1987; Yamamoto and Ohtsuki, 1989). When examining tasks with falling weights, the inertial load and the momentum become important. The momentum of the falling weight is significant at the instant of impact whereas the inertial load maintains its significance during the concentric phase (Karst and Hasan, 1987). However, one of the most interesting effects of the impact and the subsequent eccentric contraction is the activation of the stretch reflexes. The stretch reflexes constitute the biggest portion of the muscular activity during the first 90 msec after the onset of the stretch and their contribution to the production of muscular force during that phase is very significant. Hence, stretch reflexes are an important factor for performance in fast stretching movements and certainly for braking-and-accelerating tasks. It is evident that an earlier and higher muscle activation will lead to earlier and higher force development and eventually an earlier reversal of the stretching movement. Several studies report that EMG responses to the stretch stimulus in the upper limb consist of the short latency response which occurs 15-25 msec after the stimulus, the long latency response occurring after 50-65 msec and finally the voluntary response coming after 90-115 msec (see Yamamoto and Ohtsuki, 1989).

Lacquaniti and Maioli (1989) made the distinction between early and late anticipatory responses in tasks involving catching a falling weight. They also described the role of anticipation in force development: the anticipatory activation presets the viscoelastic parameters of the muscle, which to a large extent determine the restoring forces that resist the perturbation. The mechanical and neuromuscular aspects of braking-and-accelerating movements are tied together. It has been shown that movement amplitude, duration and peak velocity play a role in phasic muscle activation (Brown and Cooke, 1990).

Undoubtedly, a very important factor in braking-and-accelerating is the sequential and temporal order of activation of the various agonist and antagonist muscles involved in the execution of the task. According to the theory of reciprocal innervation, when the agonist muscles are activated by the stimulus, the antagonists are relaxed

(Liddell and Sherrington, 1925a, 1925b). On the other hand, some recent studies indicated the existence of a coactivation pattern according to which a stretching stimulus can activate both the stretched and the shortening muscles (Lacquaniti et al., 1991; Lacquaniti and Maioli, 1987).

The magnitude of the EMG during the catching of falling weights has been shown to be adversely affected by the absence of visual information (Lacquaniti and Maioli, 1987; Yamamoto and Ohtsuki, 1989). However, providing visual information by allowing the subjects to view the falling weights results in full anticipation and makes it impossible to conduct research with unexpected perturbations.

This study focuses on the effects different loads and anticipation have on the performance - which is reflected in angular displacement and power output - in braking-and-accelerating arm movements. It is also important to know the muscle activation patterns that lead to the power generation, and this research examines them by monitoring the EMG activity in the main agonist (biceps brachii) and antagonist (triceps brachii) muscle. The emphasis is placed on the reflex EMGs, which occur during the first 90 msec after the stretch onset.

Although braking-and-accelerating limb movements occur widely in sport activities they have not been investigated adequately, especially taking into account load and anticipation. Some studies used falling loads (Lacquaniti and Maioli, 1987, 1989) but the weight of the loads was very light (up to 0.8 kg). Yamamoto and Ohtsuki (1989) used heavier weights (up to 50% of the subjects' maximum strength) but the loads did not have any momentum at impact and therefore the velocity of the moving load was slow. The present study used heavy loads (up to 27% of the subjects' maximum strength) falling with a velocity of approximately 2 m/sec. The role of unexpected delays in braking-and-accelerating arm movements has not been examined by any study to date. Does temporal anticipation in the absence of vision affect the reflex responses when the stretch stimulus is delayed? To what extent do changes in these responses correlate with the mechanical performance in braking-and-accelerating forearm movements? How does the loading interact with anticipation and does this interaction influence the reflex responses and angular displacement of the limb? Knowing how these conditions affect performance and the patterns of muscle activation, would not only help improve performance but also provide an insight into the causes of injury. Indeed, anticipation or the lack thereof may affect the rate of force development, especially in the braking phase, in a large number of tasks. These tasks range from receiving heavy baggage or resisting the pull of the arm in wrestling (Yamamoto and Ohtsuki, 1989), to several skills in gymnastics (eg. Tkatchov, Jaeger, Gaylord, front somersault) where anticipation is vital but due to excessive effort, different surface or springboard stiffness etc, contact with the bar or the ground is unexpectedly delayed. It is apparent that any significant increase in joint angular displacement during this phase would increase the risk of injury.

A. The problem and the experimental hypotheses

The purpose of this study was to investigate the effects of load and unexpected time changes on voluntary upper limb braking-and-accelerating movements. The particular movement that was examined was forearm flexion and extension.

The dependent variables were:

- Negative peak power output in the braking phase.
- Maximum angular displacement of the elbow joint.
- The integrated EMG (IEMG) (area under the curve) of the short and long latency responses (first 90 msec after the stimulus onset) for biceps brachii and triceps brachii.

The independent variables were:

- Load. The weights of the free falling masses were determined as a percentage of the maximum voluntary contraction (MVC). Three different loads were chosen.
- Expected or unexpectedly delayed arrival of moving load.

Three fundamental ideas are embedded in the design of the experiment:

i. There is agonist-antagonist coactivation.

ii. The magnitude of the load affects the reflex responses.

iii. Delaying the impact beyond the expected time affects the magnitude of reflex EMG responses.

The literature provided support for investigation of the following hypotheses related to the experimental variables:

1. Power generation will decrease as a result of delaying the onset of the stretch stimulus.
2. A moderate load (≈30% of MVC) will lead to optimal power

generation, while lower magnitudes will impair performance.

3. Elbow angular displacement will increase as a result of delaying the onset of the stretch stimulus.

4. The magnitude of the IEMG of the stretch reflex in the agonist will increase with increasing load.

5. The magnitude of the IEMG of the stretch reflex in the antagonist will increase with increasing load.

B. Limitations

1. Use of electrogoniometry. A certain degree of distortion may have been introduced especially in fast movements, because the electrogoniometer was attached to the subjects' arm and problems might have been caused by skin deformation and vibrations.

2. Use of force transducer. The force transducer that was utilized in this experiment is a displacement piezoelectric transducer, which produces "a charge rather than a voltage and this charge is normally quickly dissipated by the input impedance of the amplifier used in conjunction with this transducer" (Strong, 1973). In practice that means that in prolonged force applications in the same direction, the transducer would indicate a force magnitude lower than the real. Since, however, the duration of the movements in the experimental task was usually less than a third of a second, the extent of this problem was negligible.

3. <u>Use of surface electromyography.</u> EMG measurements in general are prone to a number of problems such as drift (low frequency time varying signal), hum (occurring at 60, 120, 180 Hz etc.) and Johnson noise.

Intersubject comparison of absolute IEMG measurements is not possible because of the many factors affecting the signal e.g. skin preparation, thickness of skin, perspiring, electrode placement etc.

C. Delimitations

Thirty healthy adult male subjects were used in this study and performed 45 trials each. The warm-up session could not be designed to provide optimal preparation for every individual. Although all subjects were given the same amount of time to warm-up and general guidelines were provided to them, the selection of the appropriate warm-up intensity was left to their personal judgement. Thus, there might be small differences in the effectiveness of the warm-up from person to person. In the experiments, the braking-and-accelerating task examined involved rotation of the forearm about the elbow joint allowing only one degree of freedom. No anticipatory action was allowed in the experiments although anticipatory action occurs widely in natural movements. This limits the ecological validity of the study.

D. Definition of terms and symbols

Joint stiffness: The first partial derivative of joint torque with respect to joint angle (Hogan, 1984).

Electromyographic (EMG) signal: The name given to the total signal detected by an electrode attached to a muscle. It is the algebraic summation of all motor unit action potential trains from all motor units within the pick-up area of the electrode(Basmajian, 1985).

Bipolar electrode: One which consists of two detection surfaces (Basmajian, 1985).

Reflex latency: The interval between a stimulus and the beginning of a reflexive (involuntary) response (Anshel et al., 1991). Usually the response to a stretch comprises two components, the myotatic reflex and the long-loop reflex.

Myotatic reflex: Occurs 15-25 msec (short latency) after a stretch stimulus and involves the spindles, gamma loop and the muscles (Schmidt, 1988).

Long-loop reflex: Occurs 50-65 msec (long latency) after a stretch stimulus and involves the spindles, cortex and the muscles (Schmidt, 1988).

In the mathematical expressions of this study, vectors will be symbolized in boldface type; for example, **A** is used to designate the vector "A". The following symbols represent:

*   Multiplication.

·   Dot product.

CHAPTER II

REVIEW OF LITERATURE

Introduction

For review purposes the literature pertaining to the subject of this study, has been divided in four groups of studies. The studies in each group deal with a particular subject:

a. Mechanical power.

b. EMG activation patterns.

c. Stiffness and viscosity.

d. Factors affecting power output and muscle activation.

A. Mechanical power

The concept of power is closely related to that of work. In mechanics (Hibbeler, 1989) if a force (F) undergoes a finite displacement along its path from point s_1 to point s_2 it produces work which is determined by the expression:

$$U_{1\text{-}2} = \int_{s_1}^{s_2} F(\cos\theta)\,ds \qquad \text{Eq. 1}$$

where $U_{1\text{-}2}$ is the work done by the force, F is the force expressed as a function of position F(s), ds is the differential segment along the path, and θ is the angle between the directions of F and ds. Work is commonly measured in Joules.

Power (P) is defined in mechanics (Hibbeler, 1989) "as the amount of work performed per unit time" or as the dot product of the force (F) and the velocity (v) of the point which is acted upon by the force. Power is commonly measured in Watts and described by the following expression:

$$P = dU/dt = \mathbf{F} \cdot \mathbf{v} \qquad \text{Eq. 2}$$

Several kinds of work - and consequently power - appear in the literature. Winter (1978, 1979) defined internal work as the mechanical work done in moving the body segments, and external as the work done against an external load or resistance. Winter (1978) also provided a definition of positive work as "the mechanical work done ... when a joint rotates in the same direction as the net muscle moment acting at the joint (concentric contraction)". In braking-and-accelerating movements positive work occurs during the second (accelerating) phase of the movement. Conversely, negative work occurs during an eccentric contraction, when the muscle torque and the joint rotation have opposite directions. Cavanagh and Kram (1985) state that external work is the work that changes the energy levels of the body segments, a definition corresponding to that given by Winter for internal work. Nevertheless, the definitions given by Winter are the most widely used in the field.

Although mechanical work and power are often used interchangeably they by no means represent the same thing. For example two people may each be able to perform the same task (same amount of work) if given enough time; however, the one who has the largest power will complete the task sooner.

In human movements there are three main sources of mechanical power. Undoubtedly the most important source is the muscular activity. Concentric muscle contraction contributes to positive work, while eccentric muscle contraction contributes to negative work (Winter, 1979; Cavagna et al., 1977; Williams and Cavanagh, 1983; Williams, 1985; Ito et al., 1983). Secondly, positive mechanical power may result from the reuse of previously stored elastic energy (Williams and Cavanagh, 1983). The storage of elastic energy takes place during muscular work, if however the muscles involved do not remain continuously active or if the interval between storage and reuse is too long, the elastic energy dissipates in heat (Williams and Cavanagh, 1983). The significance of this storage and subsequent release of energy during braking-and-accelerating movements is great but as Williams (1985) pointed out it is difficult to evaluate the amount of this energy.

The third source of mechanical power is the transfer of energy between segments, within segments, or between the moving body and equipment, footwear or running surfaces (Williams and Cavanagh, 1983; Cavanagh and Kram, 1985; Williams, 1985). This transfer of energy occurs between the arm and the falling loads in experimental tasks that involve catching.

B. EMG activation patterns

The electromyogram (EMG) which monitors the electrical activity of the muscles has offered valuable assistance to researchers in kinesiology. As Åstrand and Rodahl (1986) point out, the role of EMG is critical in the evaluation of the involvement of individual muscle groups. In particular, the EMG provides information pertaining to:

i. identification of the muscles or parts of a muscle that are activated;

ii. the sequential and temporal order of muscle activation in the execution of a motor task;

iii. the degree and duration of the contraction of the participating muscles (Åstrand and Rodahl, 1986).

Many studies have focused on the patterns of muscle activation. An important aspect of these patterns was the early EMG activity that often preceded the movement. In their study, Brown and Cooke (1990) asked the subjects to make visually guided flexion/extension arm movements with various temporal profiles. They found that the agonist muscle electrical activity started 30-40 ms before movement onset.

Zattara and Bouisset (1988) focused on anticipatory postural adjustments associated with the early phase of voluntary upper limb movements. The movements were performed at maximum speed and they were loaded or unloaded flexions. Zattara and Bouisset reported that under both conditions there was an average 42.2 msec delay between the EMG activity of the deltoid (prime mover) and the onset of the movement. This kind of delay is called electromechanical delay.

In the studies of Brown and Cooke (1990), and Zattara and Bouisset (1988) the subjects would begin the movement on their own, so one cannot really refer to the pre-movement EMG as anticipatory. Anticipatory contraction usually precedes an event such as landing from a height (Greenwood and Hopkins, 1976). Lacquaniti and Maioli (1989) monitored the anticipatory activation during the catching of a falling ball. The two researchers found that the anticipatory EMG had an early and a late component. The early component occurred about 130 msec after the release of the ball and its amplitude tended to decrease with increasing height of fall. The late component on the other hand, was time-locked to the moment of impact. Beginning at about 100 msec before impact, the late anticipatory EMG would lead to a small flexion up to 5° especially in higher drops. However, in a previous investigation and following similar procedures the same researchers found different results. In 1987 Lacquaniti and Maioli did not distinguish between early and late components and in addition to that, found that the onset time of the anticipatory EMG with respect to impact was independent of the height of fall.

Much of the control of muscle activity arises from receptors within the muscles and tendons, such as the muscle spindle (Kandel et al., 1991). With the muscle spindle, the muscle has an excellent instrument to measure its length, the extent of a mechanical stimulation, and the rate with which the stretch is applied. A passive stretch of the muscle, regardless of its length, can cause the muscle spindle to fire, and by reflex the same muscle will respond by contracting, thus counteracting the stretching (Åstrand and Rodahl, 1986). This is called the stretch reflex.

The stretch reflex comprises short and long latency components. Ten studies which focused on the stretch latencies reported that in the upper limb the short latency component occurs about 15-25 msec after the onset of stretch stimulus and the long latency after 50-65 msec, while the voluntary response comes after 90-115 msec (Yamamoto and Ohtsuki, 1989; Hammond, 1954; Hammond et al., 1956; Lee and Tatton, 1975; Marsden et al., 1976; Marsden et al., 1978; Marsden et al., 1981; Tarkka, 1986; Thomas et al., 1977; Wadman et al., 1980). Yamamoto and Ohtsuki (1989) investigating braking-and-accelerating movements, found that the short latency component (M1) had two peaks and an average duration of 17 msec. Whereas some other researchers had reported two separate long latency components (M2 and M3) (Lee and Tatton, 1975), Yamamoto and Ohtsuki found them to occur connected, M2 at 50-80 msec and M3 at 80-100 msec.

Lacquaniti and Maioli (1989) observed that in the agonist biceps brachii the short latency response came 18±5 msec after the onset of the stretch and a medium latency after 40-80 msec. Le Bozec et al. (1987) examined the EMG responses of elbow extensor muscles to unexpected elbow flexion and noted the absence of M1 except at high accelerations, while they found two long latency components, M2 and M3. In 1987, Lacquaniti and Maioli reported in the biceps brachii mean short latencies of 14±4 msec and medium latencies occurring over 40-80 msec after the stretch stimulus. They chose to neglect any later responses.

Among the first researchers who focused on the stretch reflex were Liddell and Sherrington. In a series of papers (1924, 1925a, 1925b) they outlined the stretch reflex and the underlying reciprocal inhibition mechanism. Their views have become the dominant theory (Åstrand and Rodahl, 1986; R.F. Schmidt, 1978; Kandel, Schwartz and Jessell, 1991) according to which, the stretch reflex response to a passive change of joint position will be twofold. Firstly, there is excitation of the stretched muscles (agonists) and secondly, inhibition (reduced activity) in the motoneurons of the passively shortening muscles (antagonists). Although reciprocal activation would normally be expected to occur in braking-and-accelerating movements, some studies reported coactivation of agonist and antagonist muscles (Lacquaniti and Maioli, 1987; Lacquaniti and Maioli, 1989) both in anticipation and as a reflex response to passive stretching.

C. Stiffness and viscosity

One explanation put forward about the role of the muscle activation patterns is the modification of the mechanical impedance in the limb. Mechanical impedance has a static component corresponding to stiffness, and a dynamic component, corresponding to viscosity (Lacquaniti, Licata and Soechting, 1982). The central nervous system (CNS) "presets" the activation patterns of the muscles in advance of the response in order to achieve the appropriate level of stiffness necessary to control the movement (Simmons and Richardson, 1988). It has also been proposed that when the subject has to move a loaded experimental apparatus then the total stiffness of the apparatus-limb system is very important. If for instance, the stiffness of the object or apparatus is small or non-existent, the stiffness required to control the response is provided only by the limb muscles. If however, the apparatus has high stiffness, there is little need for the limb muscles to provide stiffness (Simmons and Richardson, 1988). Different activation patterns seem to have their own advantages and disadvantages. A coactivation pattern is usually associated with high metabolic cost especially when used to generate low levels of limb stiffness, but is less prone to neural transmission delays. In contrast, a triphasic activation pattern (agonist-antagonist-agonist), although less energy consuming, is limited by neural transmission delays and cannot achieve high levels of stiffness (Hogan, 1984).

Lestienne (1979) looked into the effects of inertial load and velocity on the braking process of voluntary limb movements. Lestienne's research indicated that when only small velocities or inertial loads are involved, the movement can be braked by the passive viscoelastic muscle forces alone. When high forces or velocities are involved, however, there is a diphasic pattern comprising first a burst of activity in the agonist and following that, a burst in the antagonist muscles.

In contrast to Lestienne's findings, Lacquaniti and Maioli (1987, 1989) showed that coactivation is used both as anticipatory and as reflex response in order to modulate the joint stiffness and viscosity and yield motor control.

D. Factors affecting power output and muscle activation

The number of degrees of freedom is very important in terms of motor control. Brown and Cooke (1990) reported that in simple single-joint movements, muscle activation follows the triphasic pattern. Although in braking-and-accelerating movements other researchers reported a variety of activation patterns (Le Bozec et al., 1987; Yamamoto and Ohtsuki, 1989; Lestienne, 1979; Lacquaniti and Maioli, 1987, 1989) the fact remains that the study of single-joint or multi-joint movements has certain advantages and limitations. As Hogan (1985) points out, the study of single-joint movements is more practical and easy to carry out. Multi-joint movements on the other hand, are more natural and occur widely under physiological conditions. The difficulty in multi-joint movement studies arises from the highly complex neuromuscular functions, the mechanical energy transfer between segments and the variable inertial behaviour of a multiple degree of freedom system (Hogan, 1985; Winter, 1979). Examining the upper limb, Lacquaniti and Soechting (1986) reported that the electromyographic "responses in elbow muscles to applied forces depended on both elbow and shoulder motion when the whole limb was free to move" (p. 483).

Since braking-and-accelerating movements involve a moving load, the role of the momentum of this load should be examined. The linear momentum (L) is given by the equation:

$$L = mv \qquad \text{Eq. 3}$$

where m is the mass of the load and v its velocity.

Lacquaniti and Maioli (1987), studying the activation patterns when human subjects catch a ball falling from 0.4-1.2 meters, found that the onset of anticipatory activity was independent of the height of fall - and consequently of load speed or momentum. In later research using similar methods (Lacquaniti and Maioli, 1989), the mean amplitude of early anticipatory EMG in biceps decreased with increasing height of fall, whereas the late anticipatory EMG increased. Furthermore, the analysis showed that momentum - and not its individual components (mass and velocity) - is the determinant of response amplitude. With regard to short latency reflex responses, Lacquaniti and Maioli (1989) observed that the mean amplitude increased with increasing ball momentum.

When Yamamoto and Ohtsuki (1989) compared the effects a light (25% of MVC) and a heavy (50% of MVC) load had on EMG they demonstrated that under the heavy load the amplitude of M2, M3 and the voluntary contraction EMG, was significantly higher. No significant differences were found in the EMG amplitudes before the stretch stimulus and in the amplitude of M1. Yamamoto and Ohtsuki stressed the role of the gravitational load in the reflex responses. The researchers noticed the absence of two distinct bursts for M2 and M3 and attributed it to the fact that the gravitational load caused a continuous stretch instead of a brief transient step torque.

The muscular forces produced are determined to a large extent by the force-length and force-velocity (of contraction) relationships. From the numerous studies on the subject (Hill, 1938, 1950; Huxley, 1957; Winters and Stark, 1987; Winter, 1979; Chapman, 1985) it was shown that tension decreases as the shortening velocity increases. When examining the eccentric contraction it becomes clear that tension increases with increasing lengthening velocity. The force-length relationship models the maximum isometric force at different muscle lengths. The curve describing this relationship is bell-shaped as tension appears smaller at the extremes of muscle or fibre lengths and maximal in between these extremes. Naturally, the muscle length is closely linked to joint angle in vivo and hence, it is important to carefully control the starting position of the limbs in experiments involving braking-and-accelerating movements.

Movement amplitude, speed, acceleration, duration and inertial load have been proven to modulate the muscle activation patterns. Le Bozec, Evans and Maton (1987) postulated that the M2 and M3 latencies had a tendency to decrease with increasing stretch acceleration. They maintained this monotonic relationship was described by the expression:

$$y = \alpha x^{b} \qquad \text{Eq. 4}$$

where y is the latency, x the acceleration, and α and b are constants, b being a negative one. With respect to the magnitude of the M2 and M3 components, Le Bozec et al. (1987) found that the IEMG tended to increase with increasing stretch acceleration. It should be mentioned that this study dealt only with anconeus and triceps brachii muscles and that the subjects were instructed to be totally relaxed and not to resist the stretch.

Lacquaniti and Soechting (1986) demonstrated that the biceps response amplitude increased with increasing mean angle of elbow extension. Karst and Hasan (1987) suggested that the IEMG of the antagonist muscles during forearm movements was dependent upon peak velocity, movement amplitude and the total moment of inertia. Karst and Hasan came up with an expression that adequately linked all these factors and provided good predicted values for the antagonist EMG (E_{ant}). The two researchers also pointed out that the E_{ant} values were greater than those required to just brake the movement and proposed that the antagonist might also contract in order to increase joint stiffness or counteract the centrifugal forces acting on the joint. Brown and Cooke (1990) focused on the temporal profile of arm movements and in particular the ratio of the durations of acceleration to deceleration (SR). They showed that the SR affects the timing as well as the magnitudes of the components (Ag1, Ant1, Ag2) of the triphasic pattern.

The role of anticipation and anticipatory activation has already been discussed, but there are some additional points that deserve attention. One such point is the distinction between different types of warning. Lacquaniti and Maioli (1987) argued that the amplitude and time course of the anticipatory and reflex responses when catching a ball were clearly modulated by the presence of visual information. In particular they found that visual information led to significant augmentation of the EMG amplitude and that, despite the presence of audio (acoustic tone) predictive information in all experiments. In a 1989 paper, Lacquaniti and Maioli further argued that "the very existence of anticipatory responses is contingent upon the presence of vision" (p. 144). Qualitatively similar were the findings of Yamamoto and Ohtsuki (1989) regarding the reflex responses. Nevertheless, these researchers did not observe differences as large as Lacquaniti and Maioli did. Yamamoto and Ohtsuki also showed that the reflex responses - not surprisingly - were modulated by the "resist" or "assist" instructions given to the subjects.

Although the application of perturbations at random or unexpected instants has been carried out in many experiments, the insertion of unexpected perturbations earlier or later than the moment the subjects have come - through practice - to expect, has not been applied nearly as much. This method was utilized by Lacquaniti and Maioli (1987) in their study of the anticipatory and reflex coactivation of antagonist muscles in catching a falling ball. In the experiments the subjects were given two warning tones and they were trained to expect the ball to be released at the second tone. However, in a few trials randomly interspersed among control trials, the ball was not released after the second tone. Lacquaniti and Maioli delineated that in those trials the EMG remained fairly unchanged in the first 80 msec after the time at which impact normally would have occurred.

When performing any physical skill repeatedly or for a long time, fatigue starts affecting the performance and the activation patterns of the participating muscles. Moritani, Oddson and Thorstensson (1990) found that when humans performed stretch/shortening cycles of the ankle extensors - hopping - for 60 sec the "fast-twitch" medial gastrocnemius's IEMG level declined significantly in all three phases (pre-activation, eccentric, concentric) as a result of fatigue. The "slow-twitch" soleus, on the other hand, was not affected except in the concentric phase. Gollhofer, Komi, Fujitsuka and Miyashita (1987) studied the drop-jump equivalent of the arms using a special sledge apparatus. The subjects performed 100 stretch/shortening cycles and the EMG of the triceps brachii was taken. The analysis showed that there was a significant rise of the IEMG/force ratio in the eccentric phase. The concentric EMG was also negatively affected by fatigue.

Other factors that are important in terms of power output and EMG activity modulation are: adaptation through practice (Sale, 1988; Lacquaniti and Soechting, 1986), limb position e.g. forearm supination, pronation etc (Sale, 1988; Buchanan, Rovai and Rymer, 1989), whether the load release is step-like or ramp-like (influences the stretch reflexes), and the type of the load i.e. spring, inertial or gravitational (Yamamoto and Ohtsuki, 1989).

Summary

The power output of any movement is determined by movement characteristics and muscle activation. In braking-and-accelerating movements the muscular activity consists of anticipatory, stretch reflex and voluntary components.

The anticipatory component's onset is approximately 130 msec prior to the stretch stimulus and its magnitude increases with increasing magnitude of load momentum.

The electrical activity generated by the stretch reflex is reported to comprise short and long latency components, while some studies also mention a medium latency component. The mechanism of reciprocal inhibition which is associated with the stretch reflexes, is not always apparent in braking-and-accelerating tasks. Indeed, most of the studies on this type of movement report antagonist coactivation or a switching between reciprocal inhibition and antagonist coactivation, perhaps in order to more effectively control limb stiffness and viscosity.

The modulation of joint stiffness and viscosity seems to be an important function of the muscular activity and its patterns. Both power output and muscle activation are dependent upon a variety of factors, the combination of which leads to task-specific responses. The most important factors emerging from the literature are: the force-length and force-velocity relations of the muscles, the degrees of freedom in the movement, and the temporal profile of the movement.

Although the critical role of load magnitude and load momentum in braking-and-accelerating movements has been acknowledged, there is not adequate information regarding heavy loads. Anticipation of the task comes as a result of a visual or auditory warning and profoundly affects muscle activation and - consequently - force and power generation. The role of anticipation or the lack thereof in braking-and-accelerating tasks has been examined by researchers and one study (Lacquaniti and Maioli, 1987) focused on the importance of delaying an expected stretch onset indefinitely.

CHAPTER III

METHODS AND PROCEDURES

A. Subjects

Thirty normal young non-paid male subjects participated in the experiment. All subjects were screened for injuries in the arms prior to testing and gave informed consent (see appendix A).

The subjects were assigned to three groups (Appendix D, Table 1). The first group had eleven subjects who used 'light' loads equal to 10.7% (±0.49) of their isometric maximum voluntary contraction (MVC). The second group comprised nine subjects who used loads equal to 19.2% (±1.6) of their MVC ('medium') and the third group had 10 subjects who worked with 27.5% (±2.3) of their MVC ('heavy').

B. Apparatus

The study was based on repeated measurements from human subjects. The apparatus that was used had been adapted with some modifications from Yamamoto and Ohtsuki (1989) and is illustrated in Figure 1. The system for the transmission of forces in this apparatus can be analyzed in three parts:

i. <u>Wheel-load</u>: After being released from a gate-latch, the load fell freely for 20 cm until it was hung from the wire that wrapped around the wheel. At that moment the momentum and weight of the load created torque on the wheel. Just before impact the velocity of the load was

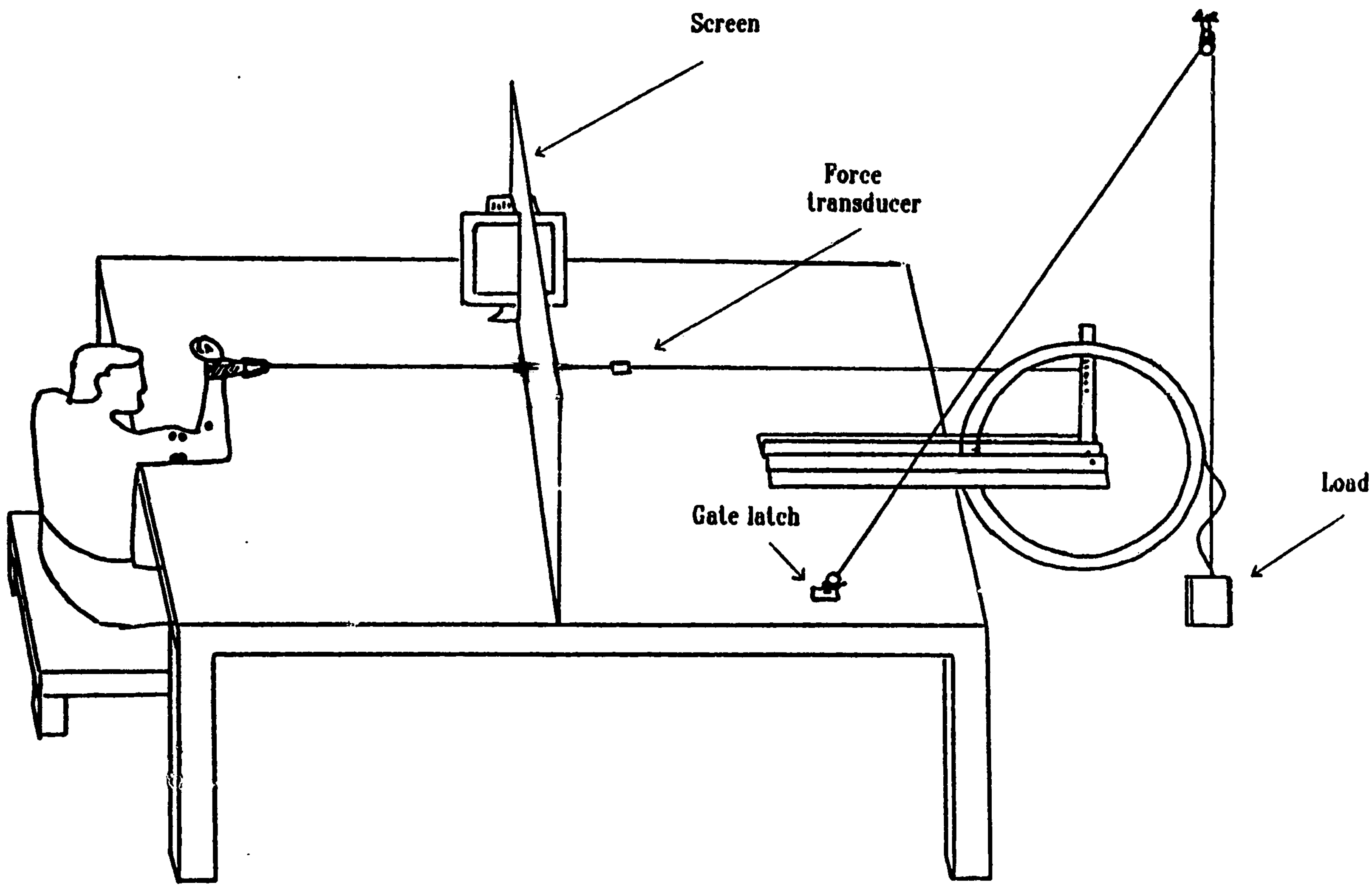

Figure 1. Experimental apparatus

approximately 2 m/sec.

ii. Wheel-lever: The lever arm was attached to the wheel and moved with it.

iii. Lever arm-subject's wrist: The lever was connected to a strap around the right wrist of the subject by a cable. The length of the wire and the point at which it was attached to the lever arm were adjustable in order for the wire to be horizontal and the subject's forearm parallel to the lever arm.

A screen was placed in front of the subject to block any visual information on the time of load release. Instead, a change in the color of a monitor screen would warn the subject that the release was imminent within the next few seconds. No audio information was provided and in order to prevent the sound of load release from warning the subjects, they were wearing headphones and listening to loud music. In addition, recorded sounds of the falling weights were played loudly on a cassette player.

Determination of maximum force and EMG: The loads were held constant for each group throughout the experiment. The maximum strength was determined in the beginning of the experiment (immediately after the warm-up) isometrically at a 90° elbow angle. A single maximum strength test was conducted with the subjects being positioned in the experimental apparatus. The subjects were instructed to flex their forearms with gradually increasing force until they reached their maximum output and maintain it for a few seconds. The tension on the cable was recorded by the force transducer and its maximum value was determined by a digital computer. The determination of the maximum EMG was done in a similar manner: the subjects performed first one maximum isometric contraction of the biceps for four seconds and afterwards one maximum contraction of the triceps. The EMG was recorded and the maximum values were determined by a digital computer.

C. Experimental procedures

The subjects were told to expect the release of the load a few seconds after the warning light was turned on by the experimenter. The subjects were also told that the exact time of release might vary randomly and were instructed not to make any premature muscle contractions before the arm was stretched. If this occurred and the forearm moved before the stimulus, the trial was repeated. The starting position was at a 90 degree elbow angle and when the weights fell, initially there was extension at the elbow joint. The subjects were instructed to reverse the movement and flex the forearm past its original position as fast as possible. Every subject performed 45 trials with one minute intervals between them. In the first 40 trials the weight was released 2 (±0.2) seconds after the warning light while in the last five trials the release occurred after 4 (±0.2) seconds.

Data acquisition: Bipolar EMG recordings were taken from the biceps brachii and triceps brachii. The 5 electrodes (four live, one ground) that were used, were surface, silver/silver chloride (Ag/AgCl), Beckman type, with adhesive collars. The bipolar electrodes were placed parallel to the muscle fibers, with 1 cm interdetection surface spacing as recommended by Basmajian (1985). Conductive paste was used on the electrodes and in order to obtain good quality EMG, skin preparation removed the skin oils and dead cells. The skin areas over which the electrodes were placed were rubbed with tissue paper and rubbing alcohol until the skin became pinkish. Special care was taken not to break or scrape the skin. The signal output of the electrodes was first fed into a battery-powered preamplifier and then to a differential amplifier with a 50 dB common mode rejection ratio (CMRR). The bandwidth of the amplifier was 20 Hz - 2000 Hz. The EMG apparatus was designed and constructed at the department of Physiology of the University of Alberta. The signal was full wave rectified and low-pass filtered at 100 Hz using an analog gain filter.

One forearm flexor and one forearm extensor had to be selected for the experiment. The two major forearm flexors that are located in the arm, are the biceps brachii and the brachialis. The biceps brachii was selected as it is the most suitable to monitor with surface EMG and can be considered representative of all elbow flexors (Le Bozec et al, 1987). Basmajian and Latif (1957) demonstrated that the biceps brachii is fairly active when loaded and supinated. The two heads of the muscle unite immediately below the middle of the arm. One live electrode was placed immediately below the point of convergence and the other 1 cm distal. The triceps brachii is the major elbow extensor (An et al., 1981). The medial head is the most active during extension, but it is hidden beneath the long and lateral heads (Woodburne, 1969) and therefore inaccessible with surface electrodes. Of the three heads, the long head was chosen for the experiments and the two electrodes were placed along the muscle, at its middle, 1 cm apart from each other.

The battery-powered electrogoniometer (Penny & Giles Biometrics Ltd.) recorded angular displacement at the elbow joint of the subjects. The two ends of the electrogoniometer were attached to the subject with adhesive tape, one on the lateral aspect of the forearm and the other on the lateral aspect of the upper arm.

A battery-powered piezoelectric force transducer (PCB Piezotronics, model 480A) was placed in the middle of the wire that connected the lever arm to the wrist of the subject and the signal was amplified.

The outputs from the electrogoniometer, the force transducer and the EMG amplifier were fed into a PCL-718 12-bit A/D board on a digital computer as well as an analog non-storage Tektronix 465 oscilloscope. The on-line sampling rate was 500 Hz and the data acquisition was performed by ASYST 3.10, a commercially available software package. The oscilloscope was used to help in the calibration and to monitor the safe operation of the equipment. The data acquisition for each trial lasted four seconds and started approximately two seconds before the release of the weight.

The subjects had a 10 minute warm-up before the experiment, to minimize the risk of muscle injuries. The warm-up consisted of biceps curls with light weights. A foamy platform was placed under the moving load to prevent it from going below a certain level. In doing so, the load was not allowed to cause full extension of the forearm.

Muscle fatigue, which may lead to muscle soreness and increase the risk of injury, was avoided by having one minute intervals between trials. According to Molbech (1963, cited in Åstrand and Rodahl, 1986) humans can sustain a rate of 30 contractions/min with 60% of their isometric MVC without any impairment due to fatigue. Therefore, the rate of one contraction/min with less than 30% of the MVC that was used in the experiment should not have fatigued the subjects. The loads used in the experiment had already been proven safe since they had been used in a similar study by Yamamoto and Ohtsuki (1989).

D. Data analysis

The raw angular displacement data obtained during the tests were smoothed using a second order critically damped Butterworth low-pass filter (Winter, 1990) with a cutoff frequency of 12 Hz. The filter was applied twice in opposite directions and the filtered data were then used to calculate the first and second time derivatives of displacement with the second finite difference method (Miller and Nelson, 1973).

The determination of stimulus onset can be done in a number of ways. Some studies relied on the detection of a statistically significant increase in angle or angular velocity (Yamamoto and Ohtsuki, 1989; Karst and Hasan, 1987) while another relied on tension rises (Yamamoto and Ohtsuki, 1989). It is important to note that the stretch reflex is activated by the muscles spindles as they react to the absolute change in length and the velocity of the length change (Kandel et al., 1991). As a result, methods that rely on angular velocity are preferable to those that rely on tension since there is always a time lag between the development of tension and movement. In this study, the stretch stimulus onset was defined as the instant at which the angular velocity exceeded 0.35 rad/s in the direction of the stretch application.

The onset and end time of the EMG activities can be determined either by visual examination of the graphs or by the detection of statistically significant increases in the EMG amplitude over the relaxed state baseline. It is also possible to rely on published reports of reflex latencies and their durations (Yamamoto and Ohtsuki, 1989), assuming they do not vary much between individuals. In this study, the reflex bursts of EMG (M1, M2, M3) could not be clearly distinguished in the averaged data and therefore the latter method appeared most suitable for the analysis of the EMG. The reflex responses were considered to occur within the first 90 msec after the stretch stimulus. Averaging was performed with the last five 'regular' and the first two 'delayed' trials of each subject. It was suspected that after the second 'delayed' trial the subjects were becoming aware of the delays which were no longer unexpected. If the instructions to avoid precontraction had been violated (indicated by forearm movement or EMG activity before the stretch onset) another trial was selected for averaging. The EMG data were integrated using a computer algorithm based on the 1/3 Simpson's rule.

Split plot factorial designs (SPF-designs) were used to analyze the data. The design that was employed to test the treatment effects on all dependent variables[1] was a SPF-3.2 design (Load x Type of stretch stimulus onset)(Kirk, 1982). The two-way ANOVA (Load x Type of stimulus onset) was performed with Load as the between subjects factor and Type of stimulus onset as the within subjects factor. In order to identify the pairs of means that are significantly different from each other, Tukey's post-hoc analyses were employed. For the ANOVAs and post-hoc analyses, the alpha (α) level of significance was set at 0.05. The analysis was performed using the SPSSx statistical software package running on an Amdahl 5870 mainframe computer of the University of Alberta.

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1 The dependent variables were negative peak joint power in the braking phase, maximum angular displacement and normalized integrated EMG in the biceps and triceps during the first 90 msec after the stretch stimulus onset.

E. Mechanical model and kinetic analysis

The mechanical system of rigid bodies consists of the forearm, hand, force transducer, wheel, lever arm, falling weight and metal wire. Terms in the equations are represented in Cartesian vector form.

The forearm, hand, wheel and lever rotate, the weight translates rectilinearly, while the force transducer translates curvilinearly.

The following assumptions were made (Hibeller, 1989; Gervais, 1986):

i. All joints in the system are considered to be frictionless pin-joints.

ii. The metal wire is inextensible and its weight is small enough to be neglected.

iii. The forearm and hand are rigid and of constant density.

iv. The center of mass remains in a fixed position within the forearm and arm during movement.

v. The mass moments of inertia for the forearm and hand remain constant during the movement.

vi. With respect to mass moment of inertia, the wheel behaves like a ring and the lever like a slender rod.

In order to calculate the mass moment of inertia and locate the center of mass of the forearm and hand, Winter's (1979) body segment data and formulae are used. The two segments - forearm and hand - are taken as one segment extending from the elbow axis to ulnar styloid. A detailed biomechanical model is presented in Appendix C. The most important calculations for the kinetic analysis of this study are the muscle torque and power. The muscle torque is calculated as follows:

$$M_m = (I_{arm@A})\alpha + (m_{arm})g(\cos\theta)(r_{AG}) + T(r_{AB})\sin\theta \qquad \text{Eq. 5}$$

where:

M_m = moment created by the muscles about the elbow axis

T = tension on the wire as measured by the force transducer; it is always horizontal

θ = angle between the forearm and the horizontal level; it is recorded by the electrogoniometer; in radians

$I_{arm@A}$ = mass moment of inertia of the forearm and hand about A (elbow axis)

g = gravitational acceleration, equal to 9.81 m/sec^2

α = angular acceleration of wheel and forearm

r_{AG} = (segment length)(0.682) m

r_{AB} = distance from the elbow axis to the wrist strap (m)

The free body as well as the impulse and momentum diagrams of the forearm/hand have been drawn in Figure 2. The joint power (P) was calculated using the following formula:

$$P = M_m\omega \qquad \text{Eq. 6}$$

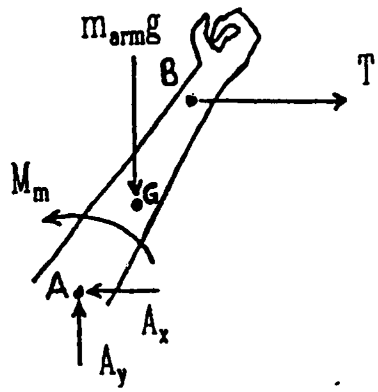

Figure 2. Free body diagram of the forearm/hand.

The movement that takes place in the system in every trial, can be divided into three distinct phases. In the first phase, the load is falling freely until the cord which connects it to the wheel is stretched. At that moment the impact produces an impulse and forces all the other segments of the system to start moving. In the second phase, the muscles of the subject produce negative work by exerting force in an effort to slow the load down. Eventually the muscle forces stop the load's downward movement - at which moment the third phase commences - and the muscles start moving the load upward producing positive work.

CHAPTER IV

RESULTS AND DISCUSSION

The effects of varying load intensities and levels of anticipation on mechanical performance and reflex responses were investigated using measurements from thirty subjects. An example of the raw, kinematic and kinetic data from a single trial is given in Figure 4. In this example, the subject had to stop and reverse the movement of a heavy (27% of MVC) load. The vertical line denotes the onset of the stretch which arrived at the expected time frame. There was no anticipatory EMG activity in either biceps or triceps. Immediately after the impact, the tension on the wire started rising causing at the same time the forearm to move, as is indicated by the increase in elbow angle. This fast displacement invoked the stretch reflexes on biceps: the first small peak occurred in the interval 12-26 msec after the stimulus and corresponds to M1, while the second response, clearly larger, occurred over the interval 42-86 msec and corresponds to M2. The activity of the triceps increased in the period 34-78 msec after the stimulus, showing a pattern of long latency reflex coactivation with the flexor. The magnitudes of the EMGs in the graph are not normalized. It should be mentioned that in large proportion of the trials recorded, the M1 and M2 components were not as clearly defined as in this example. Generally, the data were characterized by a wide variety of responses. Figure 6 is an illustration representative of those trials in which the kinematic data were 'noisy' and caused problems in the calculation of muscle torque and power while Figure 5 has the graphs of a trial in which the weights hit the ground. The trials in which the weights touched the ground could not be discarded, because they were very good examples of poor performance. Although an analysis specific to muscle torques was not conducted, a general observation was that the muscle torque rose sharply a few msec after the stretch and reach a plateau for the rest of the movement. As is depicted in Figures 7, 8 and 9, averaging the EMGs usually would result in a curve in which it was impossible to distinguish the separate reflex components. To assure that the reflex responses were included, the analysis relied on the commonly accepted assumption that reflex responses in the arm occur within the first 90 msec after the stimulus onset (Yamamoto and Ohtsuki, 1989). Consequently, the reflex responses were examined as one unit occurring during the first 90 msec after the stretch stimulus. Figures 7 and 8 show the 'regular' and 'delayed' EMG averages of one subject under heavy load, while Fig. 9 has the graph of the 'regular' EMG average of another subject performing under medium load intensity. Cutaneous stimuli from early tension in the wrist strap might have affected the reflex responses in several trials and caused the problems in the temporal 'alignment' of the EMGs.

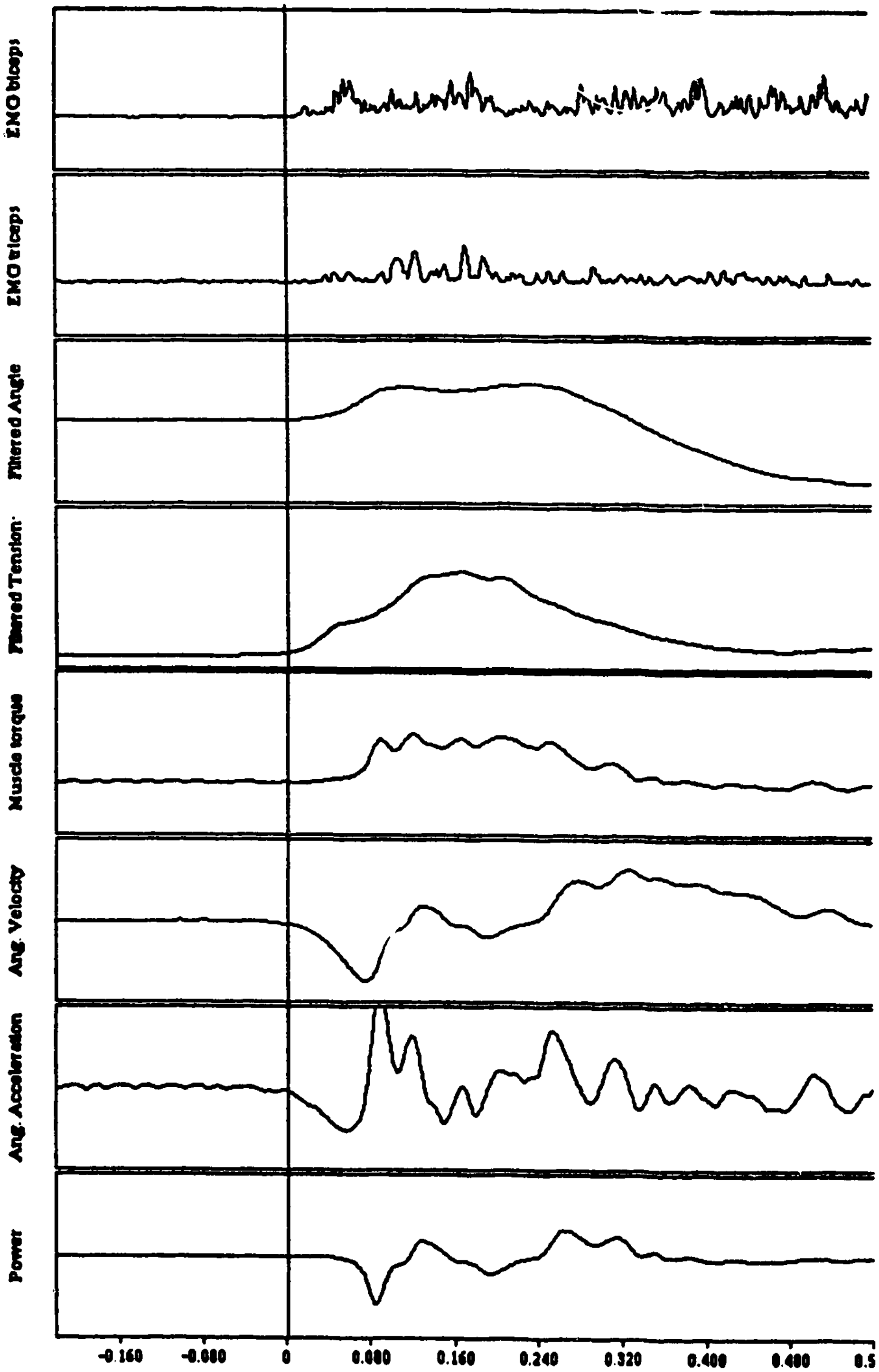

Fig. 4: Raw, kinematic and kinetic data from a single regular trial using heavy loads. The values in the graph range: EMG biceps and triceps 0 to 4097 A/D units, angle 95 to 200°, tension -70 to 721 N, muscle torque -100 to 200 Nm, angular velocity -5 to 5 rad/s, angular acceleration -300 to 300 rad/s^2 and joint muscle power -700 to 700 Watt. The vertical line denotes the stimulus onset.

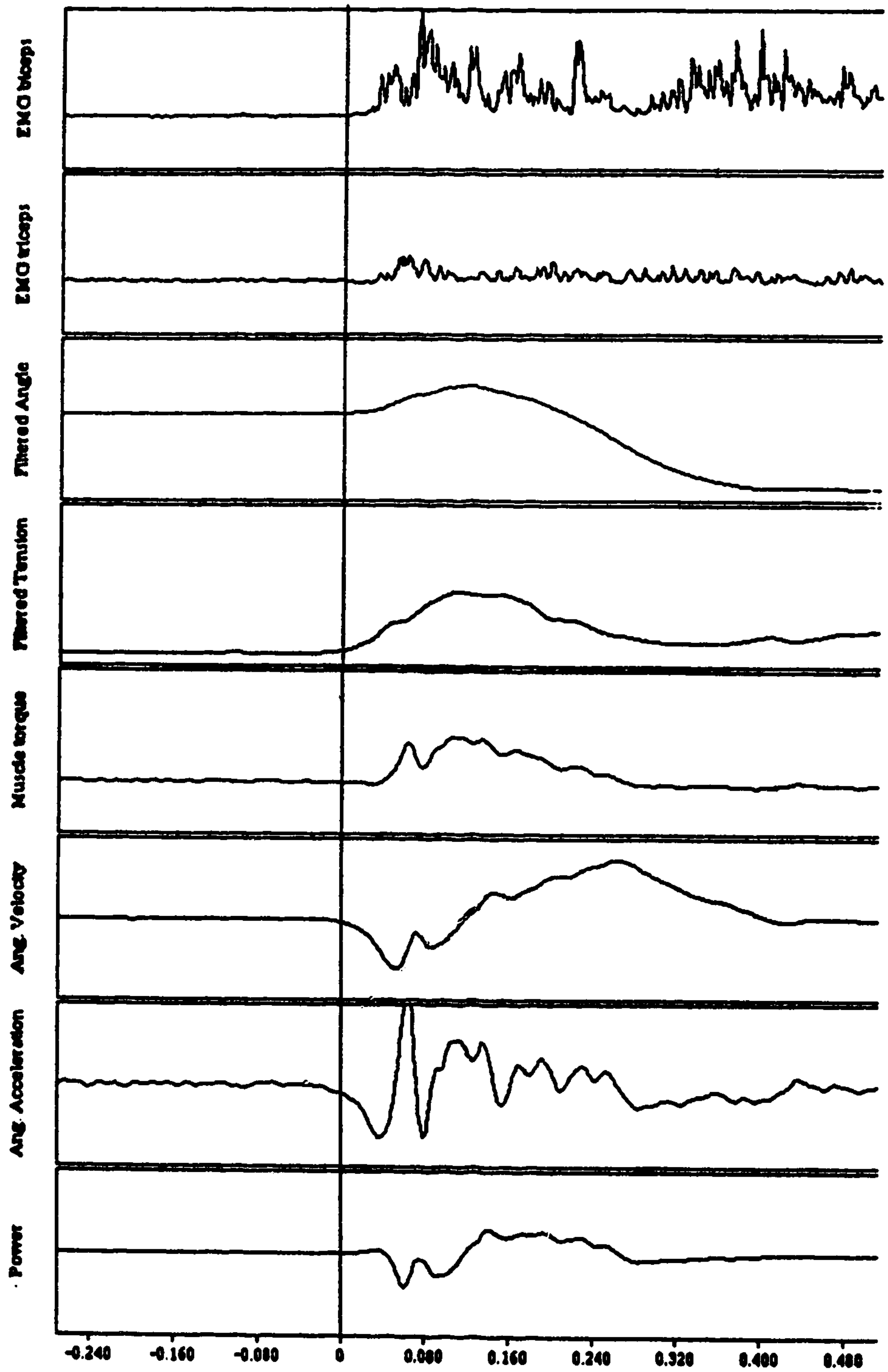

Fig. 5: Raw, kinematic and kinetic data from a single regular trial using medium loads. The values in the graph range: EMG biceps and triceps 0 to 4097 A/D units, angle 95 to 200°, tension -70 to 721 N, muscle torque -100 to 200 Nm, angular velocity -5 to 5 rad/s, angular acceleration -300 to 300 rad/s^2 and joint muscle power -700 to 700 Watt. The vertical line denotes the stimulus onset. In this trial the weights hit the ground.

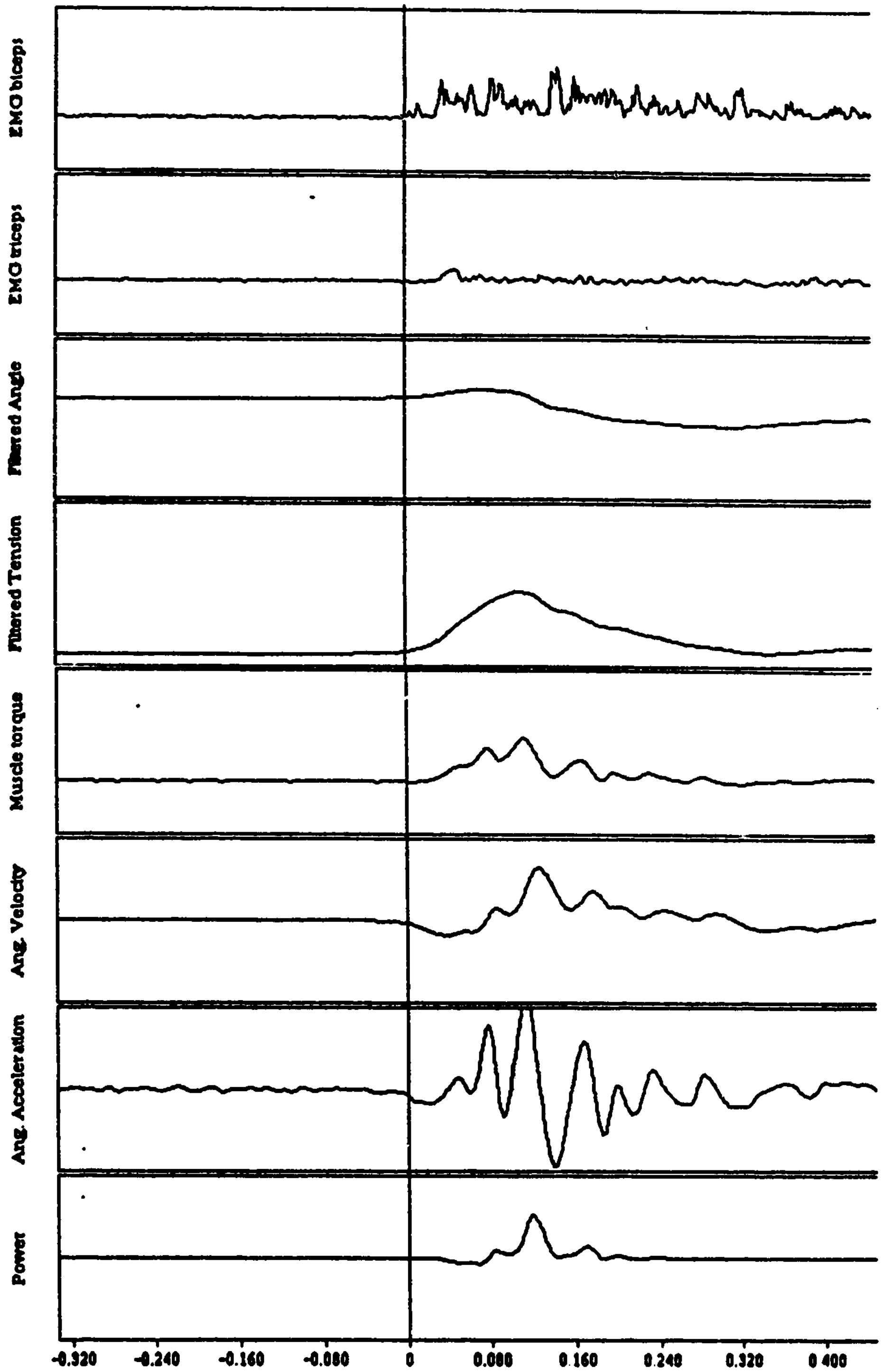

Fig. 6: Raw, kinematic and kinetic data from a single regular trial using light loads. The values in the graph range: EMG biceps and triceps 0 to 4097 A/D units, angle 95 to 200°, tension -70 to 721 N, muscle torque -100 to 200 Nm, angular velocity -5 to 5 rad/s, angular acceleration -300 to 300 rad/s^2 and joint muscle power -700 to 700 Watt. The vertical line denotes the stimulus onset. The kinematic data in this trial are 'noisy'.

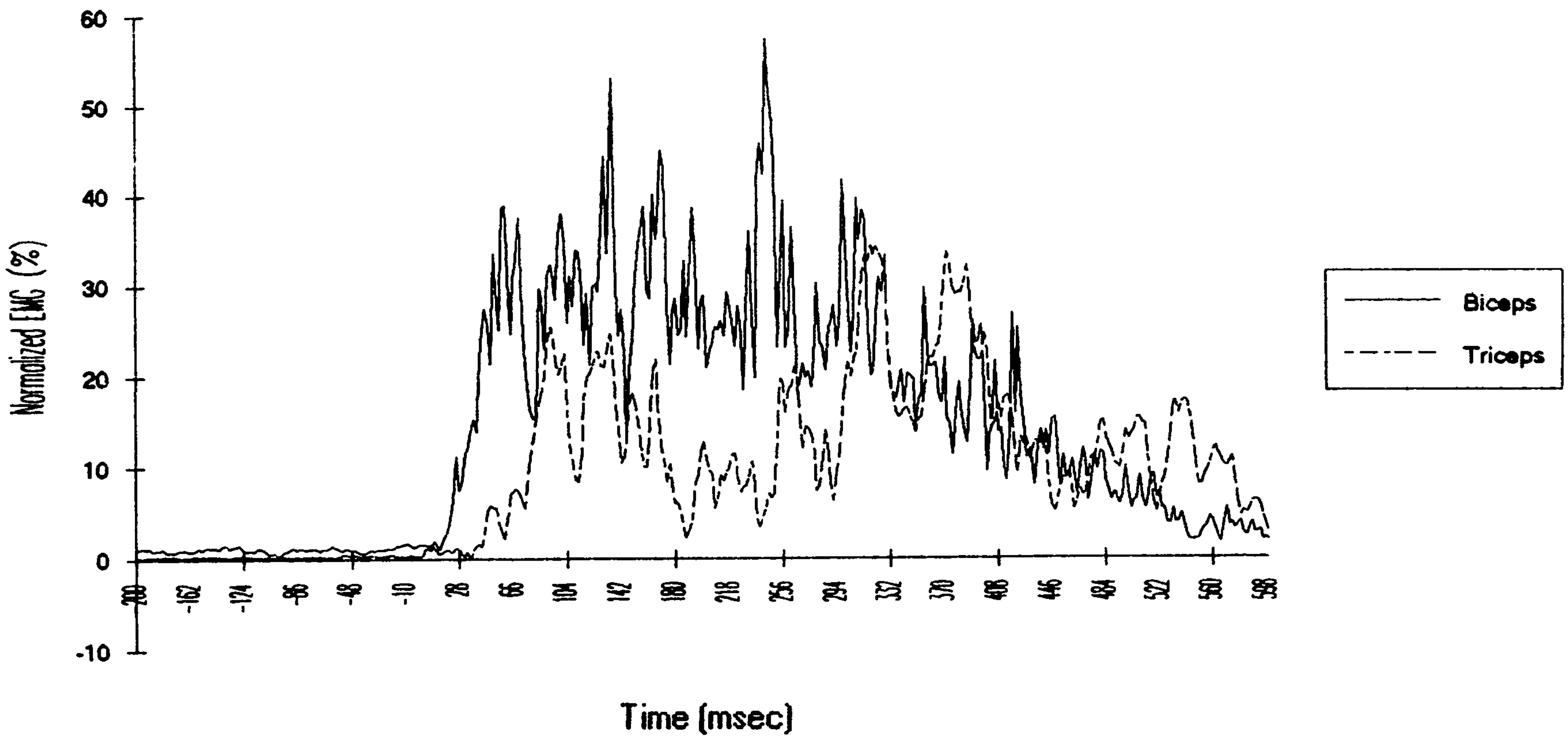

Fig. 7: Normalized EMG averaged over 5 regular trials in the 'heavy' load condition.

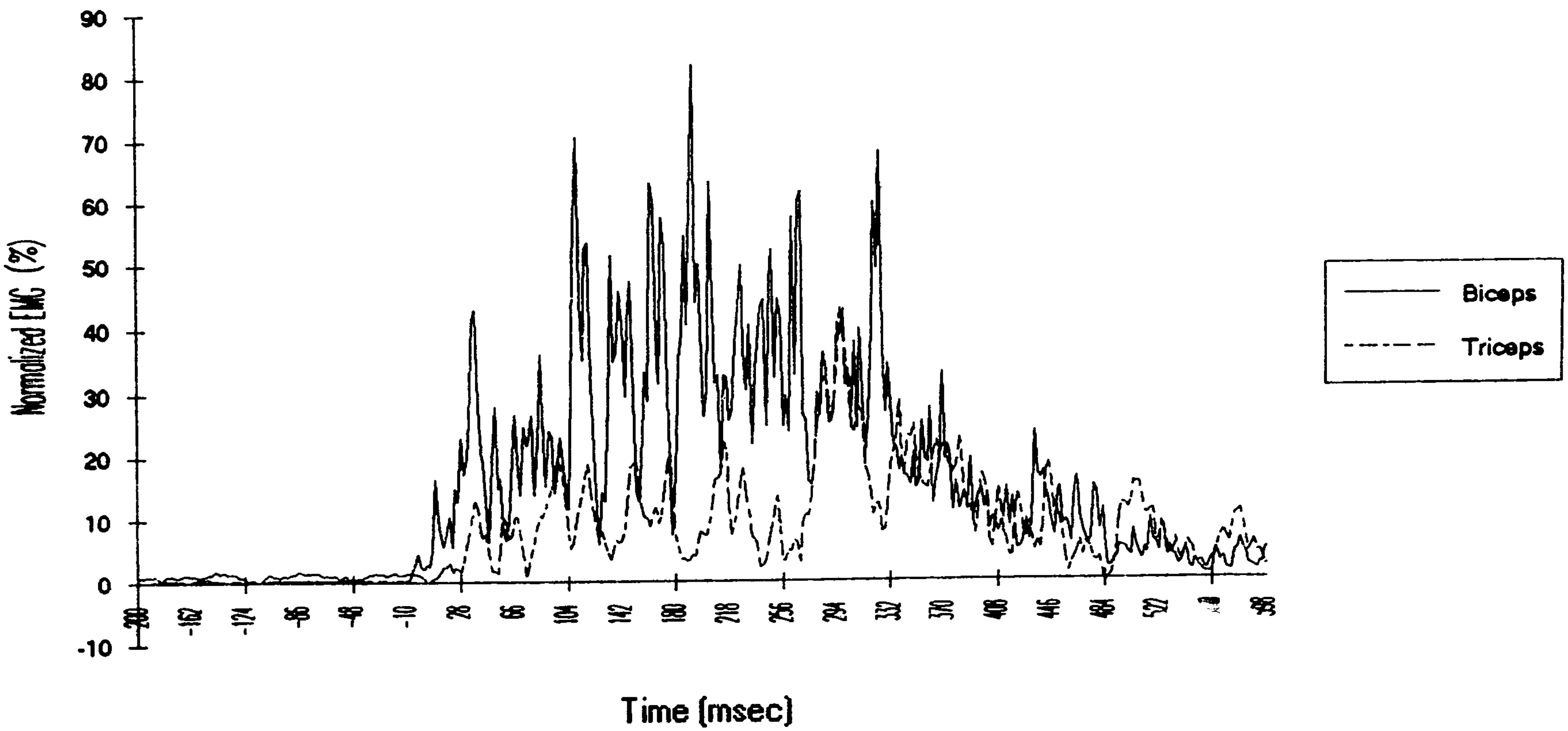

Fig. 8: Normalized EMG averaged over 2 delayed trials in the 'heavy' load condition.

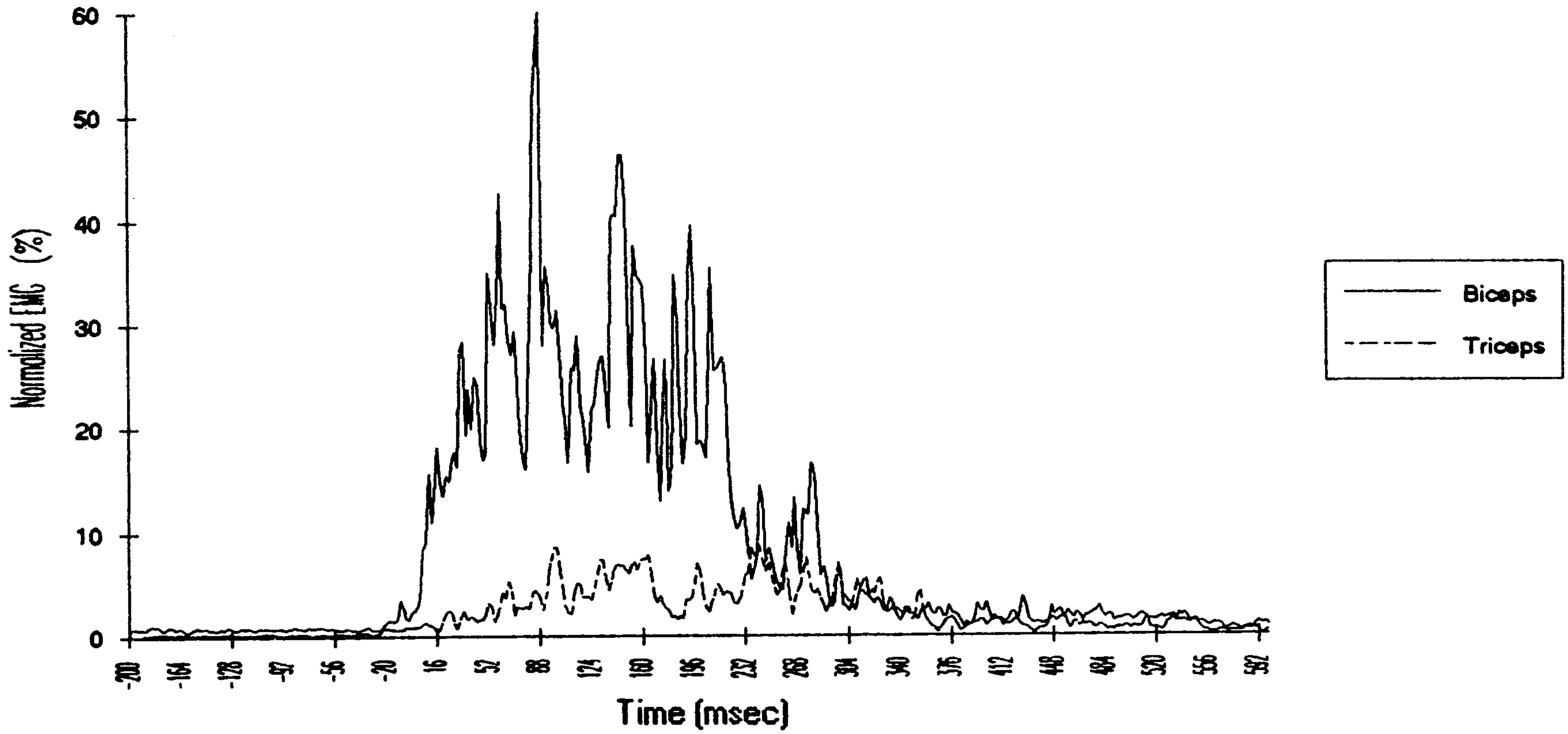

Fig. 9: Normalized EMG averaged over 5 regular trials in the 'medium' load condition.

A. Angular displacement at the elbow

The ANOVA showed a significant Type main effect ($\underline{F}(2,27)=6.03$, $\underline{p}<0.02$, $\underline{M.S.Error}=0.02$) and a significant Load by Type of stimulus onset interaction ($\underline{F}(2,27)=7.49$, $\underline{p}<0.003$, $\underline{M.S.Error}=0.0039$). The main effect of Load did not reach significant levels. The means and the standard deviations of the analyzed data are reported in Appendix D (Table 3) and the summary of the ANOVA in Table 4 of Appendix D. The means are plotted in Figure 10.

Tukey's post-hoc analysis revealed that the angular displacement in the regular trials under light and medium load were significantly different from the displacement in the 'delayed' trials under medium load.

The delay of the falling of the weights by approximately two seconds was expected to lead to a drop in performance. Since - for the purposes of this study - performance is reflected in angular displacement, it was hypothesized that the delay would result in larger angular displacement. The results of the experiment suggest that the hypothesis is tenable.

The analysis revealed a significant interaction of Load by Type of stimulus onset. It appears that the increase in angular displacement in the 'delayed' trials, was more pronounced in the medium load (angle in 'delayed' trials 40% larger than in regular trials) and it existed in the light load (23%) but not in significant levels. On the heavy load

Load	Regular	Delayed
Light	0.246644	0.304286
Medium	0.270091	0.377483
Heavy	0.355646	0.31009

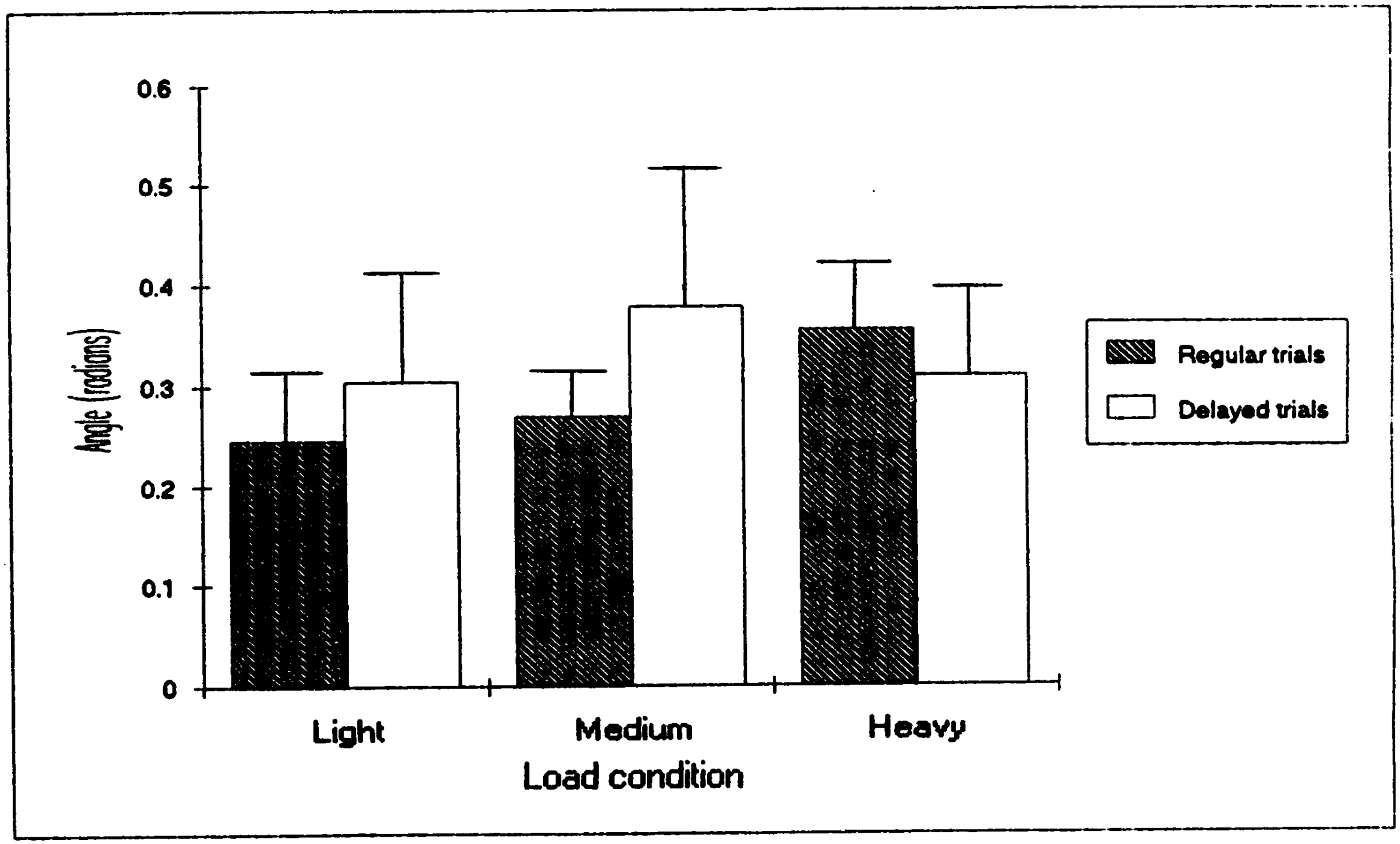

Fig. 10: Means and SDs of maximum angular displacement vs load.

condition there was actually a 19% reduction in the angular displacement in the 'delayed' trials but once again it was not significant.

The results suggest that delays in the perturbation compromise the ability to stop the stretching movement in light and medium load conditions. In these conditions the delays lead to larger displacement and increase the risk of injury. However, athletes or workers performing braking-and-accelerating movements with heavy loads are not affected negatively and they may even improve their performance and reduce the risk of sprain injury in the 'delayed' trials.

The angular displacement depends on the forces developed by the muscles involved in the task. Therefore, it is important to examine angular displacement in conjunction with muscular activity. The averaged EMG graphs show that the reflex responses are a major contributor to the total electromyographic activity on the biceps, and although they last for approximately half the duration of the total movement (braking-and-accelerating until the forearm returned to its starting position), it is during that first half that the stretching movement takes place.

B. Reflex electromyographic activity on the biceps

It was hypothesized that the magnitude of the reflex IEMG on the biceps would rise when heavier loads were used. The ANOVA yielded a significant main effect of Load ($F(2,27)=4.22$,

$p<0.025$, M.S.Error=0.61) and revealed a main effect of the Type of stimulus onset ($F(1,27)=5.28$, $p<0.03$, M.S.Error=0.16) but showed no interaction between Load and Type of stimulus onset. The means and standard deviations of the analyzed data are reported in Table 5 (Appendix D) and a summary of the ANOVA is provided in Table 6 (Appendix D). Figure 11 has the graph of the means.

Post-hoc analysis suggested that the significance of the main effect of Load was due to the fact that the reflex responses of the biceps under light load were significantly lower than the responses under medium load.

The ANOVA indicated that the load affects the magnitude of the reflex responses in the biceps and the difference in the IEMG responses is more evident between the light and medium load intensities where the higher loads are accompanied by higher IEMGs. These findings confirm the experimental hypothesis and are in agreement with the findings of Lacquaniti and Maioli (1989).

The magnitude of the IEMG of the stretch reflex is also influenced by the type of stretch stimulus. In general, the IEMG during the first 90 msec after the stimulus onset is attenuated by an average of 15% when the stimulus is delayed. This drop in reflex EMG activity is very important because it is linked to the drop in performance (increase in angular displacement) when the stretch stimulus is delayed.

Load	Regular	Delayed
Light	1.333364	0.948727
Medium	1.998111	1.629556
Heavy	1.6477	1.681

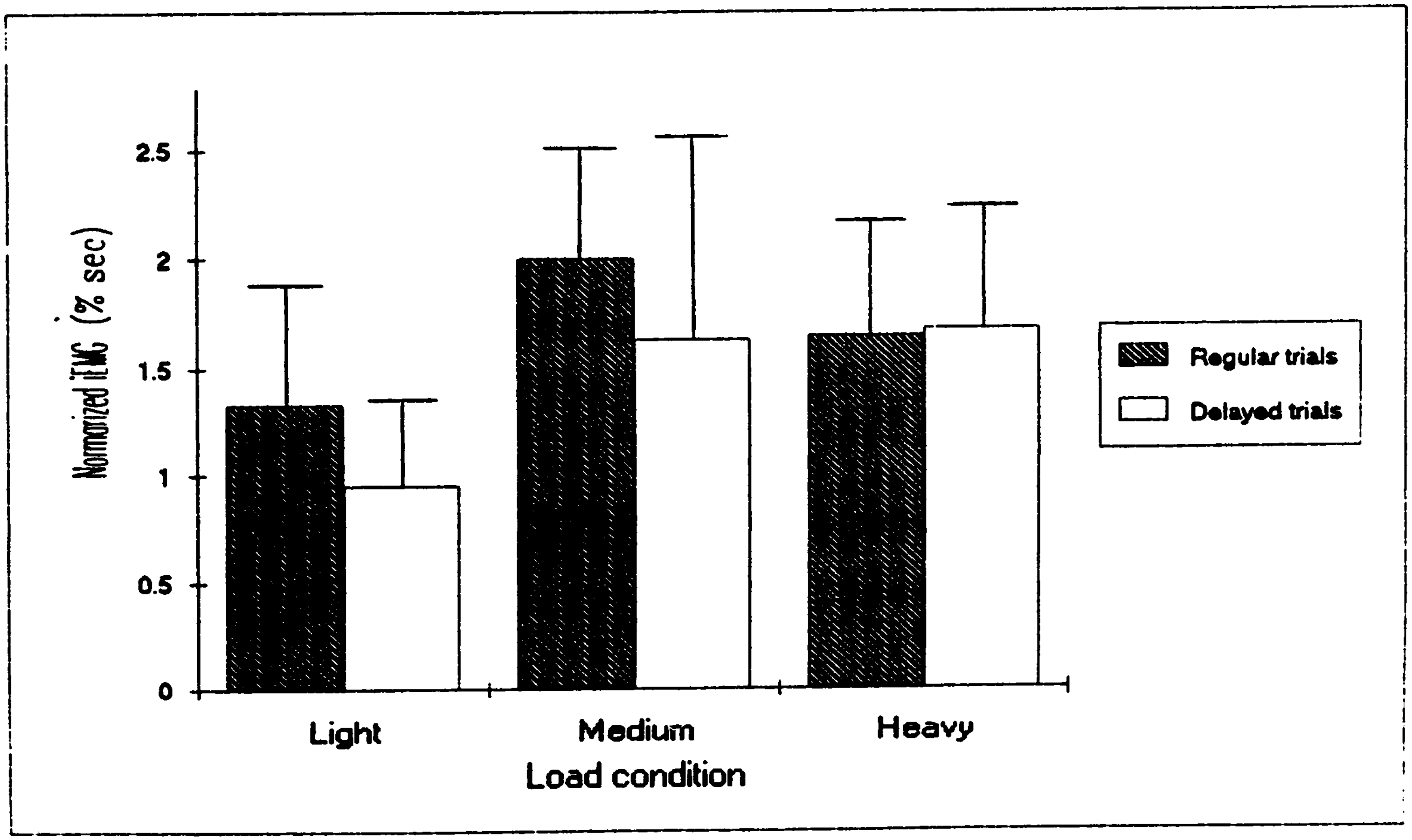

Fig. 11: Means and SDs of normalized EMG reflex responses on the biceps (integrated over 90ms) vs load.

In order to understand how the reflex EMG is affected by different load intensities and levels of anticipation, one should go back to the concept of preparatory set. Evarts et al. (1984) defined it as a state of readiness to receive a stimulus that has not yet arrived or a state of readiness to make a movement. In the present study, the preparatory set is considered to apply to the action of the two muscle groups investigated. Prochazka (1989) suggested the existence of a "fusimotor set" saying that the central nervous system presets the sensitivity of the muscle spindles through the control of the gamma motor neurons. According to this approach, as the speed, novelty and difficulty of a movement increases, so do the preset sensitivity of the muscle spindles and the gain of the stretch reflex responses. The fusimotor set hypothesis provides a possible explanation for the effects of load on biceps EMG reflex responses: the increased loading presents additional difficulty to the task and the gain is set higher. The notion of fusimotor set can be adopted in the explanation of reflex EMG and performance changes due to anticipation. Yamamoto and Ohtsuki (1989) postulated that the preparatory set which was based on temporal anticipation due to the visual information could not be maintained for even a few seconds. Yamamoto and Ohtsuki (1989) did not specifically test the effects of delaying the stimulus onset. This study however, focused on these effects and it seems that indeed, the preparatory set - in the particular experimental task - begins to lose its effectiveness after just a two second delay. To better comprehend that, one should bear in mind two important features of the experimental procedures:

- No anticipatory action or muscle activation was allowed prior to the stimulus onset. Thus, the gain of the reflex responses was not influenced by any anticipatory activity.
- In the 'delayed' trials, the subjects were still under the instructions to wait for the falling of the weights. If the state of readiness could be maintained for as long as the subjects wanted, no differences would have been observed when the stretch stimulus was delayed.

From the graph in Figure 11 it can be seen that EMG behaviour in the heavy load condition does not conform with the general trend. The first deviation from the general trend comes from the magnitude of the IEMG in the delayed trials, which is almost at the same level as in the regular trials instead of being lower. One possible explanation is that the higher difficulty and psychological stress associated with the heavy loads, enhance the preparatory set's efficiency by extending its duration. In fact Figure 10 shows that the angular displacement was slightly reduced in delayed trials under heavy load. The second deviation from the general trend is that the magnitude of the IEMG in the heavy load condition is lower than the magnitude in the medium load condition (Fig. 11). Although this difference is not statistically significant, the mere fact that the IEMG in the heavy load failed to exceed the IEMGs of the medium load is puzzling since it is in contrast with the general trends observed in this and other studies (Lacquaniti and Maioli, 1989; Yamamoto and Ohtsuki, 1989). These observations should be examined in the light of the angular displacement data (Table 3, Appendix D) which also show similar mean values under heavy and medium conditions. Practical logic suggests that when an individual receives increased load, the angular displacement should also be increased. Since however, different subjects were tested for each load condition, the behaviour observed in this experiment could be attributed to intersubject differences. Another possible explanation is based on the force-velocity relationship of the muscle and on the fact that after the impact the heavy loads continued their downward movement with higher velocity than the lighter loads. Since higher stretching velocities allow for the development of higher forces it is possible that under heavy loads the flexors could produce high forces with lower levels of activation. The non-linear nature of the force-velocity relationship could account for the absence of this behaviour in the light and medium load intensity conditions.

C. Reflex electromyographic activity on the triceps

The experimental hypothesis predicted an increase in the reflex EMG activity of the triceps when heavier loads were used. This implied an agonist-antagonist coactivation pattern during the braking phase of the movement. The ANOVA indicated a significant Load main effect ($\underline{F}(2,27)=6.33$, $\underline{p}<0.0056$, M.S.Error=0.006). The main effect of the Type of stimulus onset and the Load x Type interaction were not statistically significant. Table 7 (Appendix D) lists the means and standard deviations of the analyzed data, while Table 8 (Appendix D) gives a summary of the ANOVA. The means are plotted in Fig. 12.

Tukey's post-hoc analysis showed that the EMG of the triceps under heavy load was significantly different from the EMG responses under light and medium load conditions.

Increases in the load resulted in enhanced reflex activity on the triceps. This effect was not present between light and medium loads which seem to have similar IEMG magnitudes in the first 90 msec after the onset of the stretch stimulus. However, when the load is increased from light or medium to heavy, the reflex response on the triceps (antagonist) almost doubles. At that load, the muscular activity is high enough to be considered a form of coactivation although the level of antagonistic activation is not as high as the agonistic. This type of nonlinear relationship between load and antagonist reflex EMG has been observed also by Lacquaniti and Maioli (1989). Although the loads they used were much lighter than in this study, they had three different load intensities and the short latency EMGs

Load	Regular	Delayed
Light	0.218909	0.210727
Medium	0.256444	0.203556
Heavy	0.4755	0.4629

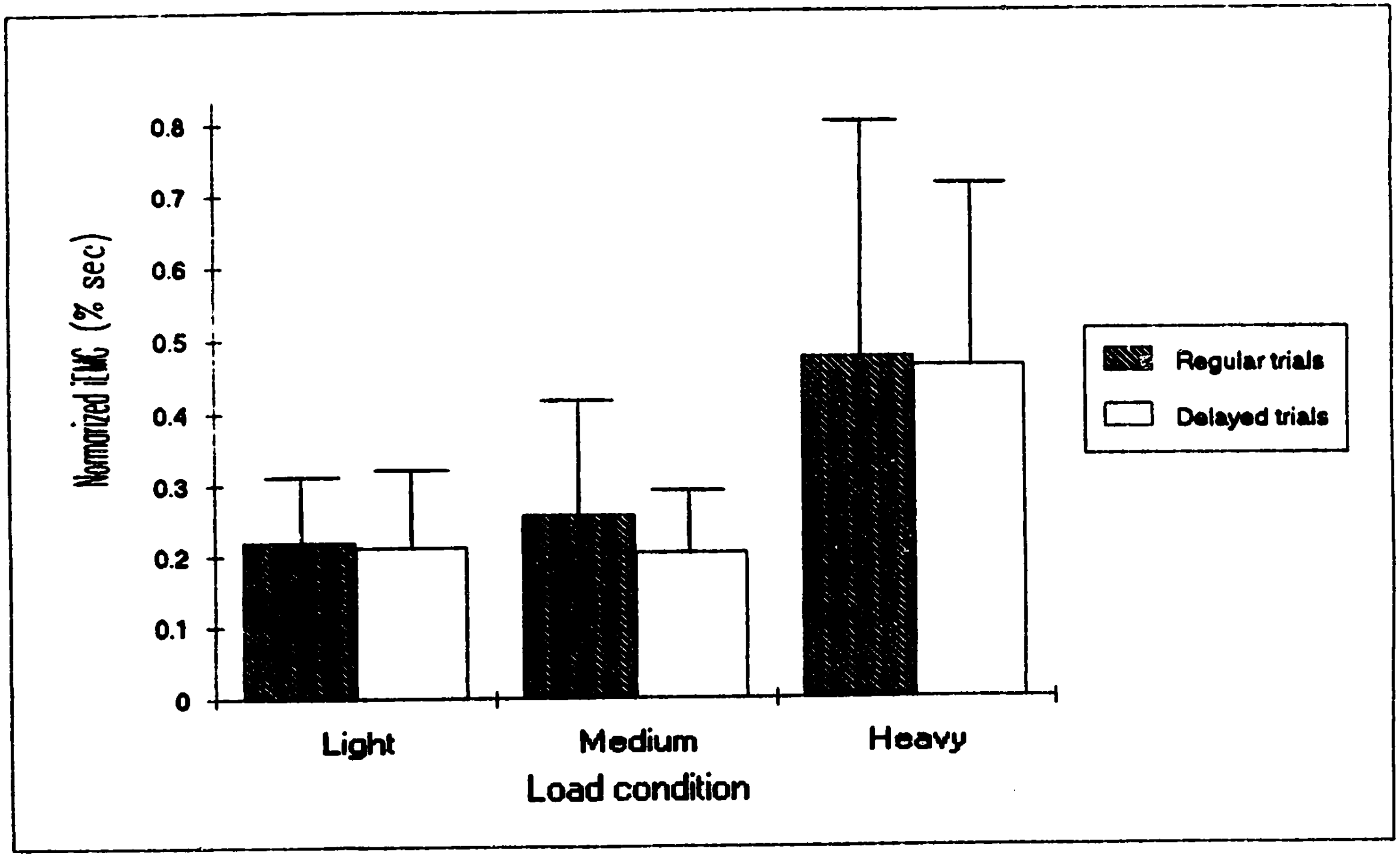

Fig. 12: Means and SDs of normalized EMG reflex responses on the triceps (integrated over 90ms) vs load.

for them were 11, 13 and 55 μV. In general the findings of the present study and the one conducted by Lacquaniti and Maioli (1989) are in agreement since in both studies the reflex activity increased with the intensity of the perturbation both in the agonist and antagonist muscles. The notion of reflex coactivation has been supported in the past (Lacquaniti and Maioli, 1987, 1989; Lacquaniti, Borghese and Carrozzo, 1991) but is in conflict with the traditional notion of reciprocal activation. Coactivation is considered to lead to increased joint angular stiffness and viscosity, while reciprocal activation is geared to net joint torque production (Hasan, 1986; Lacquaniti and Maioli, 1987, 1989; Lacquaniti, Borghese and Carrozzo, 1991). Each pattern of activation is reported to have certain advantages and disadvantages: coactivation has a higher metabolic cost but responds faster to kinematic changes than reciprocal innervation which has conduction delays in neural feedbacks (Hogan, 1984; Lacquaniti and Maioli, 1987).

It has been shown by other researchers (Lacquaniti and Maioli, 1987) that the reflex coactivation is centrally preset and it has been postulated that the stimulation of Ia afferents from the arm muscles can evoke excitation of the α-motoneurons not only from homonymous and synergistic muscles but also of antagonist muscles. Acceptance of the aforementioned notion of centrally preset reflex coactivation, raises the question whether a unique preparatory set controls the coactivation or each muscle group uses its own set. The present study provides evidence for the latter for two main reasons:

- Despite the trend of the reflex IEMGs to increase with increasing load in both biceps and triceps, the two EMGs do not correlate. For example, whereas the responses on the biceps increased significantly from the light to the medium load condition the responses on the triceps remained unaffected. It could be argued however, that this indicates the absence of a linear relationship but not the absence of a nonlinear one.
- The Type of stimulus onset in this study does not have any effect on the responses of the triceps. Hence, there must be two different preparatory sets: the one for the antagonist (triceps brachii) can be maintained for a long period of time, while the one for the agonist (biceps brachii) is significantly affected by delays.

The fact that agonist-antagonist coactivation takes place in braking-and-accelerating movements could be important to athletes who have suffered injuries in the antagonists muscles. If the athletes or their trainers are under the false impression that the shortening muscles are not actively involved in a braking-and-accelerating exercise they could possibly aggrevate existing injuries in these muscles. For instance, under heavy loads the level of activation of the antagonists goes up to about one third of the level of the agonists. This is illustrated in Figures 7 and 9 which show the EMG averages in 'regular' trials for two subjects. The amplitude of the antagonist EMG in the subject that used the heavy load (Fig. 7) is considerably higher than that of the other subject who used medium load (Fig. 9).

D. Peak negative muscle power

The ANOVA revealed a significant Load main effect ($\underline{F}(2,27)=8.11$, $\underline{p}<0.00174$, $\underline{M.S.Error}=7953.2$). The main effect of the Type of stimulus onset and the Load x Type interaction were not statistically significant. The means and standard deviations of the analyzed data are listed in Table 9 (Appendix D), while Table 10 (Appendix D) gives a summary of the ANOVA. The means are plotted in Fig. 13.

The load was found to have a significant effect on negative power amplitude. Table 9 (Appendix D) shows that increases in negative power amplitude were proportional to the increases in the load. The highest power amplitude was found in the heavy load condition which used a 27% of MVC load intensity. This confirms the experimental hypothesis which predicted that the highest power amplitude would occur at 30% of MVC.

Since all the weights were falling from the same height and their velocity at the time of impact was the same, the original determinant of muscle power was the inertial load. The rise in power amplitude under heavy load can be attributed to two factors: First, the higher levels of activation that

Load	Regular	Delayed
Light	208.56	210.91
Medium	254.3	272.04
Heavy	334.99	306.47

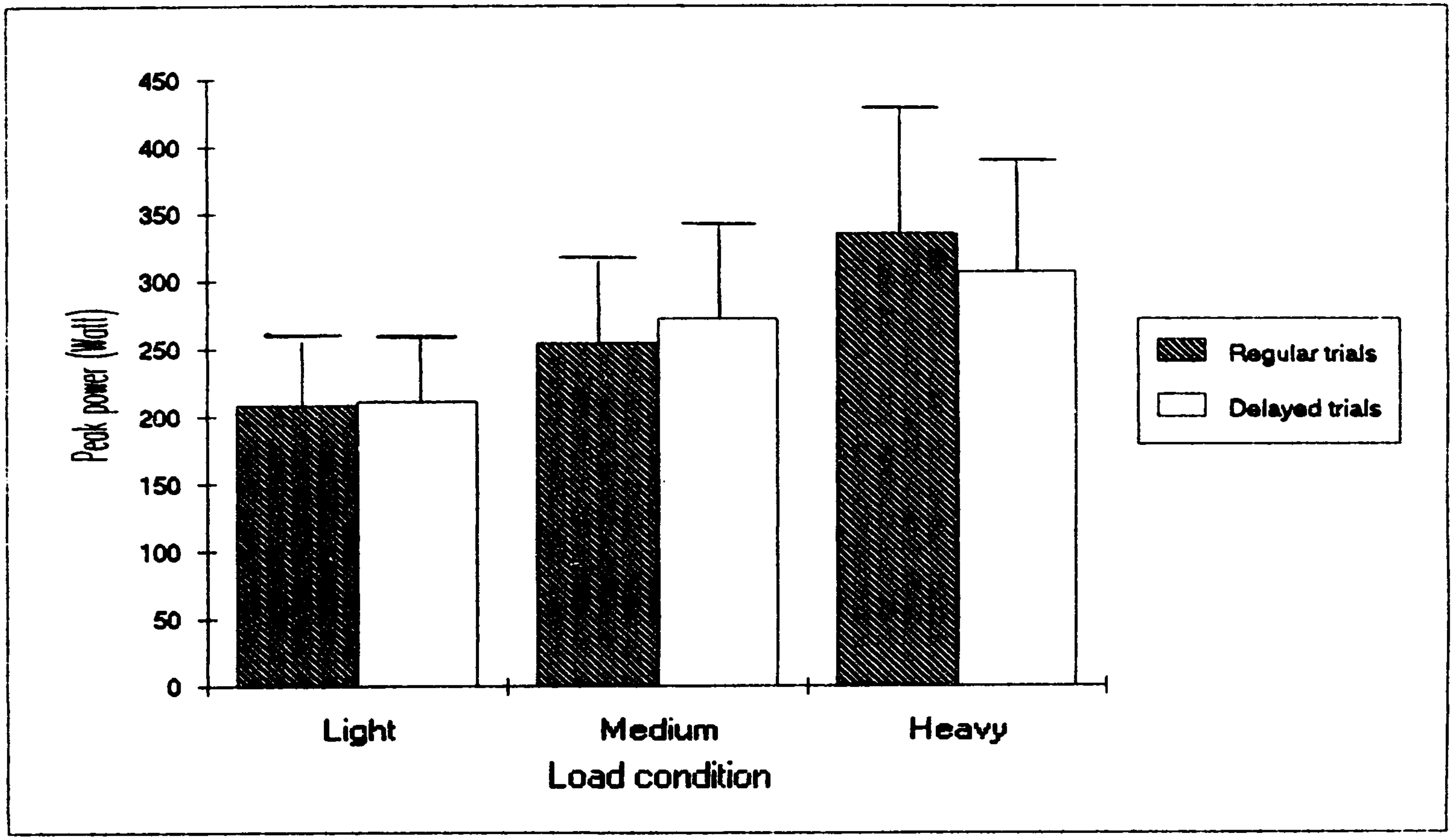

Fig. 13: Means and SDs of peak joint muscle power vs load.

were observed in the biceps probably led to higher torques at the elbow joint. Second, as the displacement data indicate, it was more difficult for the subjects to stop the heavier weights. As a result, higher angular velocities were present during the braking phase in the heavy load condition. Since joint power is the product of torque and angular velocity, both variables contributed to higher power amplitude.

The results show that peak power in the first phase of the movement remained unaffected by the delays in the fall of the weights. This happened despite the fact that delays had an effect on angular displacement and velocity, as well as on muscle activity and therefore, torques. Nevertheless, lower torques that might have come as the result of delays, probably led to higher angular velocities and it is possible that it was this trade-off that kept peak power at approximately the same levels.

In the design of the study and the formation of the experimental hypotheses, the mechanical power output of the muscles acting on the elbow joint in the braking phase was selected as an indicator of performance. Performance from a joint power perspective can be quantified and examined in terms of amplitude, work (integrated power) or temporal profile. Unfortunately, during the process of data collection and analysis it became apparent that mechanical power could not be utilized as was originally intended. The following paragraphs will describe briefly each of the three different ways the power analysis could be carried out and will explain why they all failed to meet the objectives of the study.

Amplitude: Amplitude analysis was expected to provide two peaks for the movement: one negative during the first phase and one positive during the second phase. Although that pattern was observed, the usefulness of this method was questionable since the peak value would provide information for only a very short portion of the movement. There was no guarantee that even if the amplitude was high at some point, the power production would not be low for the rest of the movement. In addition, the knowledge of the magnitude of the power peak would be inadequate without any information about its temporal locus.

Work (integrated power): It would appear that given the same load, the less work a subject produced the better he performed. In order to understand that, one would have to realize that the work produced by the muscles during the first phase (braking) was used to absorb/counteract the kinetic energy of the moving load. As the load was moving down, more of its potential energy was transformed into kinetic energy which eventually would have to be absorbed by the muscles. So if two subjects were trying to catch the same load, the subject who managed to stop the weights with a shorter displacement would have performed better because: a) he achieved the first objective of the task (brake the movement of the falling weights) with a smaller displacement, and

b) his movement was mechanically less energy consuming.

The problem with this approach occurred in the trials in which the subject produced a very small force or no force at all. In those trials the weight would hit the ground and although the performance was poor, the small amount of muscle work would falsely indicate a good performance.

Temporal analysis could examine when the power peaks occurred. However, knowing when the peak occurred would not reveal anything about its amplitude or the magnitude of power before and after it.

The inability of power to serve as an indicator of performance in this experiment, can be illustrated by the following example: in two regular trials of the same subject, the angular displacement in the first trial was 0.114 rad and in the second was 0.279 rad. Despite these huge differences in displacement, the power output in both trials showed great similarities in terms of peak values (-209 compared to -188 Watts) and temporal profile (both peaks occurred approximately 77 msec after the stimulus). It should be stressed that although the three aforementioned indeces of power could not be fruitfully utilized in this study, this does not deny the value of power and its many indeces in general biomechanical studies. In fact, many of the problems associated with the use of negative peak power in this research, were due to the nature of the particular experimental task, the existence of noisy kinematic data and the great variance observed in the data.

CHAPTER V

SUMMARY AND CONCLUSIONS

A. Summary

The purpose of the study presented herein was to investigate the effects of load and unexpected time changes of the stimulus onset, on braking-and-accelerating voluntary arm movements, and in particular, on the mechanical performance and on the reflex EMG responses of the agonist and antagonist muscles involved in the task.

A stretch was applied to the dominant arm elbow flexor (biceps brachii) by the free fall of a weight. The thirty male subjects were instructed to resist the movement of the load which would tend to extend their elbow, and try to reverse it by flexing the arm as fast as possible. The subjects were also told to expect the release a few seconds after a warning light was turned on. In the absence of any other visual or auditory information, the subjects practiced catching the falling weights two seconds after the warning, with the exception of the last two trials where the weights fell with a delay of four seconds. Each subject was using a load - constant across trials - which could be 11% (light), 19% (medium) or 27% (heavy) of his isometric MVC score. The angular displacement and rectified EMG data were averaged for the last five regular and the first two delayed trials of each subject.

B. Conclusions

Based on the results of this study, within the limitations of this research, the following conclusions are warranted:

- Maximum angular displacement tends to increase when the load increases and when the stretch stimulus is unexpectedly delayed. Nevertheless, when the load is heavy, performance remains unaffected by unexpected delays in the stretch.
- Increased loads - especially at the light and medium levels - are accompanied by increased EMG activity on the biceps, while delays in the stimulus result in reduced activity. The data provide support for the notion of a preparatory set of limited duration (in the particular experimental task).
- Reflex EMG activity on the antagonists (triceps) seems to be unaffected by light and medium loads, and the type of stretch stimulus (expected/unexpected), but increases sharply when heavy loads are applied. The observed pattern of agonist-antagonist coactivation points to the possibility that the reflex responses of both flexors and extensors are preset by two independent preparatory sets.
- Joint power output as an indicator of performance is not suitable for the experimental task employed in this study.

C. Recommendations for future research

1. The present study focused on the magnitudes of the kinematic and electromyographic data but it left unexplored their temporal characteristics. A study must be designed to concentrate on the temporal profiles.
2. The kinetic analysis should be the subject of further research. Although an analysis of power seems elusive, a torque analysis is more promising and will likely provide a link between the kinematic and electromyographic data.
3. The relative contributions of reflex and voluntary muscular activity should be determined in order to be able to draw clear and substantiated conclusions about their role on the outcome of the movement.
4. The issue of agonist-antagonist coactivation patterns or reciprocal inhibition remains unresolved and controversial.
5. Certain results of this study regarding angular displacement and reflex responses on biceps, were either not statistically significant or represent interestingly surprising trends. It is recommended that future research be conducted to investigate these results in depth.

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APPENDIX A

Informed consent form

EXPLANATION OF THE STUDY

Title: The effects of load, anticipation and unexpected time changes, on braking-and-accelerating voluntary arm movements.

The purpose of this study is to examine the power output and the muscle activation in braking-and-accelerating movements of the arm. These movements involve braking the movement of a moving load and accelerating it in the opposite direction. The experiment will focus on the effects of different loads and anticipation.

The experimental session will last about an hour. In the beginning you will have a 10 minute warm-up of the upper body and the right arm. Then you will sit in front of a table and have your arm strapped to its surface. An electrogoniometer will be attached to your elbow to measure the angle in this joint. Three areas in your upper arm (each about 3-4 cm long) will be rubbed vigorously with tissue paper and alcohol to clean the skin. The skin will turn pink from the rubbing and may be slightly irritated. In some cases an allergy may occur. In the cleaned skin, five electrodes will be placed in order to record the electrical activity of your muscles. These are adhesive surface electrodes used only to record. A strap will be placed in your right wrist and through a cable will be connected to a falling load. A light will warn you that the load is about to be released in a few seconds. The exact time of release will vary. After a short free fall the load will be suspended from the cable that goes to your strap. You are supposed to resist the movement of the load (which will tend to extend your arm) and try to reverse it (flex your arm) as fast as you can. You will be asked to perform 35 trials with one minute intervals between them. Apart from these trials, your maximum strength will be measured in a static contraction after the warm-up.

It is possible that your arm muscles will be sore for a couple of days due to the intensity of the exercise. There is a very remote possibility of equipment malfunction that could result in electric shock. The equipment has been inspected and a number of other measures taken to minimize that risk. You are advised not to take part in this study if you have ever had serious injuries or pain in your right arm.

Date: ______ ______________________________

Subject's signature   Investigator's signature
Nikitas Tsaousidis

Note: If you have any questions or concerns I would be pleased to answer or discuss them with you. I may be reached at 492-2018 (work) or 431-0633 (home).

INFORMED CONSENT FORM
Biomechanics Laboratory
Department of Physical Education & Sport Studies
University of Alberta

Title: The effects of load, anticipation and unexpected time changes, on braking-and-accelerating voluntary arm movements.

Researcher: Nikitas Tsaousidis

I, ______________________________
Participant's name

authorize the University of Alberta, Department of Physical Education and Sport Studies to administer, for the purposes of research only, the test outlined below. I understand that I am free to withdraw consent and to discontinue participation in the project at any time and for any reason. The investigation and the participant's part in the study have been defined to me in a written description of the procedures and I understand the explanation. I have also been given an opportunity to ask questions concerning the tests. I understand that the recorded and derived data will remain the property of the researcher, will remain confidential and will be used for research purposes only.

Tests to be performed:
1. Subject's maximum isometric strength.
2. 35 trials of fast braking-and-accelerating arm movements during which force, angle and EMG data will be recorded.

I acknowledge that I have read this form and understand the test procedures to be performed and the inherent risks, and I give my consent for participation.

Date:______________________ Signature:______________________

APPENDIX B

Table of means of the dependent variables for each subject

sub	Load	angle (5)	angle (2)	iEMG5b	iEMG5t	iEMG2b	iEMG2t
10	1	0.21287	0.29826	1.873	0.117	0.728	0.143
12	1	0.31868	0.36767	0.796	0.374	0.57	0.285
16	1	0.27913	0.25231	0.945	0.321	1.314	0.417
20	1	0.19475	0.24778	0.878	0.303	0.756	0.279
21	1	0.20325	0.23155	1.225	0.232	1.144	0.195
22	1	0.31499	0.44485	0.675	0.215	0.504	0.18
23	1	0.31389	0.52152	1.77	0.149	0.699	0.323
26	1	0.17822	0.2281	1.421	0.225	0.864	0.132
28	1	0.37475	0.37173	0.96	0.087	0.714	0.046
33	1	0.17872	0.2438	2.269	0.273	1.525	0.227
42	1	0.14383	0.13958	1.855	0.112	1.618	0.091
8	2	0.3079	0.66458	1.548	0.128	0.907	0.08
9	2	0.19997	0.19537	1.986	0.14	2.645	0.18
19	2	0.22126	0.42856	2.01	0.244	1.072	0.138
30	2	0.25203	0.31049	1.57	0.187	0.712	0.191
35	2	0.30297	0.34052	1.583	0.186	1.346	0.257
37	2	0.28383	0.31439	2.407	0.425	3.313	0.394
38	2	0.28095	0.55444	2.376	0.647	0.563	0.24
39	2	0.30888	0.27246	1.482	0.169	1.157	0.183
40	2	0.27303	0.31654	3.021	0.182	2.951	0.169
14	3	0.39	0.29249	1.249	0.516	1.776	0.535
15	3	0.32785	0.237	1.064	0.67	1.651	0.918
18	3	0.25073	0.16953	2.891	0.21	2.716	0.255
24	3	0.31399	0.25613	1.541	0.418	1.514	0.468
27	3	0.38332	0.37619	1.13	0.345	0.989	0.424
29	3	0.44578	0.37981	1.546	0.731	1.134	0.706
31	3	0.435	0.4086	1.898	0.315	2.087	0.324
32	3	0.29977	0.34266	1.777	1.22	1.306	0.68
34	3	0.2773	0.22061	2.033	0.115	2.379	0.153
41	3	0.43272	0.41788	1.348	0.215	1.258	0.166
	1	0.246644	0.304286	1.333364	0.218909	0.948727	0.210727
	2	0.270091	0.377483	1.998111	0.256444	1.629556	0.203556
	3	0.355646	0.31009	1.6477	0.4755	1.681	0.4629

Displacement and electromyographic averages over 5 (regular) and 2 (delayed) trials for each subject and across subjects.

APPENDIX C

Biomechanical model

In order to calculate the mass moment of inertia and locate the center of mass of the forearm and hand, Winter's (1979) body segment data and formulae are used. The two segments - forearm and hand - are taken as one segment extending from the elbow axis to ulnar styloid:

$$I_{arm@A} = (0.022)(\text{body mass})[(0.827)(\text{segment length})]^2 \ \text{kg}\cdot\text{m}^2 \qquad \text{Eq. 7}$$

where:

$I_{arm@A}$ = mass moment of inertia of the forearm and hand about A (elbow axis)

0.022 = (segment weight)/(total body weight)

0.827 = (radius of gyration)/(segment length) for elbow

The distance (r_{AG}) between the elbow axis and the segment's center of gravity is:

$$r_{AG} = (\text{segment length})(0.682) \ \ \text{m} \qquad \text{Eq. 8}$$

Also:

$$I_{wheel@D} = (m_{wheel})(r_{wheel})^2 \ \ \text{kg}\cdot\text{m}^2 \qquad \text{Eq. 9}$$

where:

$I_{wheel@D}$ = wheel's mass moment of inertia about its axis of rotation at D

m_{wheel} = mass of the wheel in kg

r_{wheel} = wheel's radius in m

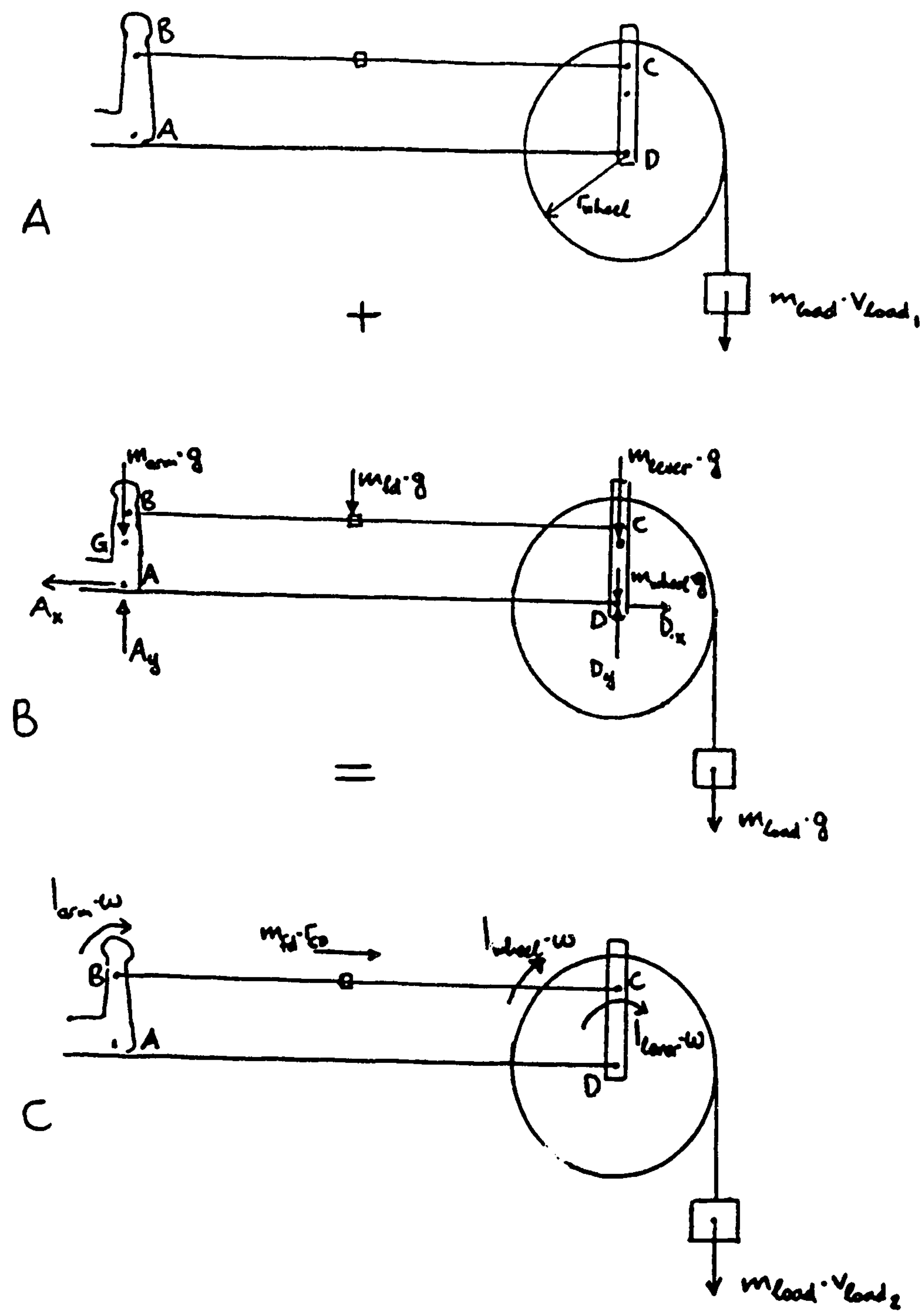

Fig. 3: A is the impulse and momentum diagram of the system before the impact. B is the free body diagram before impact. C is the final momentum diagram after impact.

The mass moment of inertia of the lever about D is:

$$I_{leverD} = (1/3)(m_{lever})(\text{lever's length})^2 \quad kg \cdot m^2 \qquad \text{Eq. 10}$$

where:

m_{lever} = lever's mass in kg

First phase: Just before impact, the load is moving downward with a speed v_{load} which is determined as follows:

$$\frac{1}{2}(m_{load})(v_{load})^2 = (m_{load})gh \Leftrightarrow (v_{load})^2 = 2gh \Rightarrow v_{load} = \sqrt{2gh} \quad m/sec$$

Eq. 11

where: m_{load} = mass of the load in kg

h = height of load's free fall in m

g = gravitational acceleration, equal to 9.81 m/sec^2

Since the wire is inextensible and the time of impact very short ($\Delta t \approx 0$), the weights of the load, forearm and lever can be considered nonimpulsive. Applying the principle of angular impulse and momentum (Fig. 3), we have:

$$(\circlearrowleft +) \quad \Sigma H_1 + \Sigma \int M dt = \Sigma H_2 \qquad \text{Eq. 12}$$

where:

ΣH_1 = sum of the system's angular momentum prior to impact

ΣH_2 = sum of the system's angular momentum after impact

Expanding Eq. 12:

$$-(m_{load})(r_{wheel})(v_{load}) + \int M_A dt =$$

$$-(\omega I_{arm@A}) - (\omega I_{wheel@D}) - (\omega I_{lever@D}) - \omega m_{fd}(r_{CD})^2$$

$$\Leftrightarrow -(m_{load})(r_{wheel})(v_{load}) + \int M_A dt = -\omega[I_{arm@A} + I_{wheel} + I_{lever@D} + m_{fd}(r_{CD})^2] \qquad \text{Eq. 13}$$

where:

ω = angular velocity of wheel and forearm

m_{fd} = mass of the force transducer in kg (negligible)

r_{CD} = distance between C (point where the wire coming from the forearm attaches to the lever arm) and D.

It should be noted that not all of the angular momenta in the equation are calculated about the same fixed reference point (D). Nevertheless, this approach is valid because the angular momenta ($I\omega$) are free vectors, and therefore, they may be applied at any point on the body. The angular impulse can be eliminated from the equation because $\Delta t \approx 0$ and the couple moment M_A about the elbow axis is also very small since the muscles have not had the time to contract yet. Therefore solving the equation with respect to ω we have:

$$\omega = \frac{(m_{load})(r_{wheel})(v_{wheel})}{I_{arm@A} + I_{wheel} + I_{lever@D} + m_{fd}(r_{CD})^2} \qquad \text{Eq. 14}$$

<u>Second phase</u>: The free body as well as the impulse and momentum diagrams of the forearm/hand have been drawn in Figure 2 (chapter III) to clarify the application of the principle of angular impulse and momentum about the elbow axis.

$$(\;+) \quad H_3 + \int M_A dt = H_4 \qquad \text{Eq. 15}$$

$$-(I_{arm@A})\omega_1 - \int(m_{arm})g(\cos\theta_1)(r_{AG})dt - \int T(r_{AB})(\sin\theta_1)dt + \int M_m dt =$$
$$-(I_{arm@A})\omega_2 \qquad \text{Eq. 16}$$

where:

H_3 = angular momentum of the forearm/hand about A, before the application of the couple moments

H_4 = angular momentum of the forearm/hand about A, after the application of the couple moments

M_m = moment created by the muscles about the elbow axis

T = tension on the wire as measured by the force transducer; it is always horizontal

θ = angle between the forearm and the horizontal level; it is recorded by the electrogoniometer; in radians

The reaction forces at A (A_x and A_y) are not producing any moment about A, and therefore, they were not included in Eq. 16.

Third phase: It is similar to the second phase except for the direction of the movement:

$$(\;+) \quad H_3 + \int M_A dt = H_4 \qquad \text{Eq. 17}$$

$$(I_{arm@A})\omega_1 - \int(m_{arm})g(\cos\theta_1)(r_{AG})dt$$
$$- \int T(r_{AB})(\sin\theta_1)dt + \int M_m dt = (I_{arm@A})\omega_2 \qquad \text{Eq. 18}$$

APPENDIX D

Tables of means and SDs, ANOVAs and correlation coefficients.

Table 1. Means, standard deviations and ranges of the normalized loads (% of MVC score) in each Load class.

	Light load n=11	Medium load n=9	Heavy load n=10
Mean	10.741	19.222	27.46
Stand.Dev.	0.4913	1.5738	2.3167
Range	10 - 11.7	18.1 - 22.2	24.7 - 32

Table 2: Pearson product correlations (r) among biceps reflex EMG, triceps reflex EMG and angular diasplacement across Type of stretch stimulus onset for each Load.

<u>**Light**</u>

	Regular		Delayed	
	Biceps reflex iEMG	**Triceps reflex iEMG**	**Biceps reflex iEMG**	**Triceps reflex iEMG**
Angular displacement	-0.5614 (11) P=0.036	-0.0275 (11) P=0.468	-0.7344 (11) P=0.005	0.2094 (11)P=0.268
Biceps reflex iEMG		-0.3983 (11) P=0.113		0.0145 (11)P=0.483

<u>**Medium**</u>

	Regular		Delayed	
	Biceps reflex iEMG	**Triceps reflex iEMG**	**Biceps reflex iEMG**	**Triceps reflex iEMG**
Angular displacement	-0.1855 (9) P=0.316	0.1096 (9) P=0.389	-0.5474 (9) P=0.064	-0.3548 (9) P=0.174
Biceps reflex iEMG		0.4248 (9) P=0.127		0.4664 (9) P=0.103

<u>**Heavy**</u>

	Regular		Delayed	
	Biceps reflex iEMG	**Triceps reflex iEMG**	**Biceps reflex iEMG**	**Triceps reflex iEMG**
Angular displacement	-0.5425 (10) P=0.053	-0.0025 (10) P=0.497	-0.6809 (10) P=0.015	-0.0107 (10)P=0.488
Biceps reflex iEMG		-0.2594 (10) P=0.235		-0.4379 (10)P=0.103

Table 3. Means and standard deviations for angular displacement at the elbow (rad) for different loads across type of stretch stimulus onset

		Regular	Delayed	
Light n=11	Mean St. Dev.	0.247 0.0758	0.304 0.1111	0.275
Medium n=9	Mean St. Dev.	0.270 0.0387	0.377 0.1475	0.324
Heavy n=10	Mean St. Dev.	0.356 0.0708	0.310 0.0869	0.333
		0.290	0.328	0.309

Table 4. Summary ANOVA of angular displacement at the elbow for Load and Type of stretch stimulus onset

Sources	SS	df	MS	F	P	
Load	0.04	2	0.02	1.45	0.25306	
Error term	0.37	27	0.01	-	-	
Type	0.02	1	0.02	6.03	0.02081	*
Load x Type	0.06	2	0.03	7.49	0.00259	*
Error term	0.37	27	0.00392	-	-	

Table 5. Means and standard deviations for reflex iEMG on biceps brachii for different loads across type of stretch stimulus onset

		Regular	Delayed	
Light n=11	Mean St. Dev.	1.333 0.5356	0.949 0.3877	1.141
Medium n=9	Mean St. Dev.	1.998 0.5222	1.630 1.0445	1.814
Heavy n=10	Mean St. Dev.	1.648 0.5429	1.681 0.5636	1.664
		1.638	1.397	1.517

Table 6. Summary ANOVA of reflex iEMG on biceps brachii for Load and Type of stretch stimulus onset

Sources	SS	df	MS	F	P	
Load	5.13	2	2.56	4.22	0.02538	*
Error term	16.40	27	0.61	-	-	
Type	0.86	1	0.86	5.28	0.02959	*
Load x Type	0.56	2	0.28	1.73	0.19618	
Error term	4.39	27	0.16	-	-	

Table 7. Means and standard deviations for reflex iEMG on triceps brachii for different loads across type of stretch stimulus onset

		Regular	Delayed	
Light n=11	Mean St. Dev.	0.219 0.0942	0.211 0.1091	0.215
Medium n=9	Mean St. Dev.	0.256 0.1711	0.204 0.0883	0.230
Heavy n=10	Mean St. Dev.	0.475 0.3289	0.463 0.2510	0.469
		0.316	0.293	0.304

Table 8. Summary ANOVA of reflex iEMG on triceps brachii for Load and Type of stretch stimulus onset

Sources	SS	df	MS	F	P	
Load	0.82	2	0.41	6.33	0.00558	*
Error term	1.75	27	0.06	-	-	
Type	0.008985	1	0.008985	0.82	0.37355	
Load x Type	0.005768	2	0.002884	0.26	0.77082	
Error term	0.30	27	0.01	-	-	

Table 9. Means and standard deviations for peak joint power in the braking phase for different loads across type of stretch stimulus onset

		Regular	Delayed	
Light n=11	Mean St. Dev.	208.56 48.97	210.91 55.76	209.735
Medium n=9	Mean St. Dev.	254.30 63.26	272.04 69.76	263.17
Heavy n=10	Mean St. Dev.	334.99 92.56	306.47 81.25	320.73
		264.43	261.1	0.309

Table 10. Summary ANOVA of peak joint power in the braking phase for Load and Type of stretch stimulus onset

Sources	SS	df	MS	F	P	
Load	129027	2	64533.7	8.11	0.00174	*
Error term	214736	27	7953.2	-	-	
Type	117.87	1	117.87	0.07	0.79834	
Load x Type	5344.9	2	2672.48	1.51	0.23908	
Error term	47801	27	1770.4	-	-	