THE STRETCH RESPONSE OF HUMAN SKELETAL MUSCLE *IN SITU*

by

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B.Sc. University of British Columbia, 1977

THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in the Department

of

Kinesiology

SIMON FRASER UNIVERSITY

July, 1982

APPROVAL

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Degree: Master of Science (Kinesiology)

Title of thesis: The Stretch Response of Human Skeletal Muscle *In Situ*

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THE STRETCH RESPONSE OF HUMAN SKELETAL MUSCLE "IN SITU"

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ABSTRACT

When actively contracting single fibres and whole muscle *in vitro* are subjected to a ramp stretch, tension rises above isometric levels. This excess tension varies with the final muscle length, stretch amplitude and velocity of stretch. The purpose of this thesis was to examine the effect which changes in each of the above factors have on the eccentric torques produced by human skeletal muscle *in situ*.

Five subjects performed forearm supination on 5 occasions during which the muscles involved were stretched after development of maximal isometric torque. Nine different combinations of final muscle length, stretch velocity and amplitude were used. Excess torque was expressed as the ratio of torque at the end of stretch over isometric torque at the same position. The rate of decay of excess torque after stretch was also recorded. Excess torque during stretch was smaller and torque decayed much faster than has been observed in either single fibres or isolated whole muscle. It is possible that these discrepancies are the result of differences in the muscle preparation itself i.e. the amount of series elasticity, fibre composition, temperature and/or the stretch conditions used.

Excess torque increases with stretch velocity until a certain critical velocity has been reached. It decreases with an increase in stretch amplitude and is not dependent on final muscle length. The rate of decay of torque following stretch was found to be independent of final muscle length, amplitude of

stretch and stretch velocity.

The effects of stretch velocity and amplitude on excess tension indicate that cross-bridge slip may be present in muscle *in situ*. The absence of significant changes in myoelectric activity suggests that with high initial operating forces, the intrinsic mechanical properties of the supinator muscles and not reflex activity are primarily responsible for the torque response during stretch. Time constants for the rise in isometric torque were found to be considerably less than the decay time constants for all stretch trials ending at the same muscle length. The storage of mechanical energy in the series elastic component can not therefore by itself account for the changes in torque after stretch.

ACKNOWLEDGEMENTS

I would like to thank Dr. Arthur Chapman for his patient support and guidance over the past few years. I am also most grateful to Dr. Parveen Bawa for her assistance in the collection and analysis of the EMG data.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

I. Introduction

The biomechanical properties of skeletal muscle have, over the years been generally expressed as a function of sarcomere length and/or velocity of shortening, assuming a relatively high level of activation. Almost without exception the forces produced have been explained by the isometric tension-length and isotonic force-velocity relationships based on Huxley's (1957) sliding filament model of muscle contraction. Under isometric conditions the tension developed is thought to be dependent on the overlap of thick and thin filaments which in turn governs the number of effective actomyosin interactions. With concentric activity a characteristic decay in tension is observed as velocity increases due to a supposed imbalance in the rates of cross-bridge breakage and reformation. There is a net reduction in the number of active cross links present at a given instant in time and tension falls.

During an eccentric contraction the forces generated are significantly higher than those observed when isometric contractions are performed over an identical range of sarcomere lengths (Sugi, 1972; Edman et al., 1978a, 1978b; van Atteveldt and Crowe, 1980). While the force-velocity relationship predicts an increase in force with eccentric activity, it alone can not account for all of the excess tension produced when actively contracting muscle is forcibly stretched. This additional "force enhancement" is thought to be the result of temporary changes in

the above fixed muscle properties (Edman et al., 1978a; 1978b).

With prior eccentric movement muscle is able to increase it's positive work output above that obtained when it is allowed to shorten from an iscmetric contraction (Cavagna et al. 1968). Depending on the activity there may also be a considerable increase in power since the time interval over which the positive work is carried out tends to decrease as well. This enhancement of positve work and/or power is dependent not only on the amount of excess tension produced by the countermovement but also the time delay between the eccentric and concentric phases.

Regardless of whether a previous stretch is applied, muscle loses some of its ability to generate tension during "active" shortening. This process of mechanical deactivation is not caused by changes in sarcomere length alone (Edman and Kiessling, 1971). Rather it seems to be the result of changes in the myofilament system which influence the binding of activator calcium and prevents the formation of actomyosin cross-bridges (Joyce et al., 1969; Edman, 1975; 1980).

In order to distinguish between enhancement and this deactivation process, "stretch and hold" techniques have been used which prevent active shortening. These studies indicate that excess tension gradually falls away, eventually reaching isometric levels for that muscle length. Both the rise in tension during stretch and the decay of force following the termination of stretch appear to be influenced by a variety of

factors i.e. temperature, level of activation, final muscle:sarcomere length, velocity and amplitude of stretch (Sugi, 1972; Edman et al., 1978a,1978b; Curtin and Woledge, 1979; Flitney and Hirst, 1978; van Atteveldt and Crowe, 1980).

Within the literature there is little direct information on how various combinations of these factors influence eccentric tension in intact mammalian muscle. Most human movement studies deal solely with changes in the amplitude of the eccentric phase. Attempts have been made, using single fibres and isolated whole muscle , to determine the effects which changes in one or more of these conditions have on the stretch response of muscle in vitro. However due to the relatively short lifetime of each preparation and the lengthy restoration period required between successive recordings, it is difficult to examine many of the possible combinations using the same preparation (van Atteveldt & Crowe, 1980). Since the fragility of these preparations increases drastically with temperature, most studies using single fibres and isolated whole muscle are carried out at or near 0°C. This difference in temperature as well as differences in the amount of associated connective tissue and the absence of neural inputs must be taken into account when applying information obtained from isolated preparations at 0°C to intact whole muscle at 37°C.

In recent years considerable research has been conducted in the area of muscle modelling. Hatze (1981) has in fact produced a "myocybernetic" model which is capable of predicting the

changes in tension during and after stretch. Another less complicated model has been developed by Crowe *et al.* (1980). Yet much of the data used to develop these models has come from *in vivo* muscle studies. Therefore several assumptions must be made when using these models to predict the stretch response in mammalian muscle *in situ*.

Purpose

The object of this study was to examine the effects which changes in total muscle length, rate and amplitude of stretch have on the eccentric torques produced by human muscle (forearm supinators) activated voluntarily and maximally. By comparing these eccentric values with isometric torques at the same muscle lengths it was possible to obtain measures of both the excess tension generated during a ramp stretch and it's rate of decay once the stretch had been terminated. Using torque and EMG records an attempt was made to determine the various mechanisms which contribute to the enhancement of positive work and/or power in human skeletal muscle *in situ*. A comparison of the stretch response of muscle *in situ* with that of either single fibres or isolated whole muscle may lead to modifications which will facilitate the development of more accurate muscle models of human muscular contraction.

II. Review of Literature

Evidence of Enhanced Positive Work and/or Power Following Active Stretch

Using motion picture analysis and a platform sensitive to forces in the vertical direction Marey and Demeny (1885b) noted that when two jumps are performed in succession the second jump was consistently higher than the first. Although they failed to provide any qualitative results of their experiments they emphasized the benefical effect of prior eccentric movement. Similar observations were reported by Fenn (1930).

Morton (1952) compared the heights reached when standing high jumps were initiated from a squat position with those after a drop to the squat position, and he reported that performance levels were approximately 11% higher when preceeded by a downward countermovement. Anderson (1967) was able to show that the ground reaction forces were considerably greater in the second type of jump. Cavagna et al. (1971), and Komi and Bosco (1978) report that takeoff velocity for a vertical jump could be increased by a factor of 1.064 and 1.11 respectively when the countermovement was used.

Although Cavagna et al. (1971) found that the positive work done (m*g*h) was increased by approximately 10% when preceeded by eccentric activity the time interval over which this work was

done (Tw) decreased significantly. With this reduction in Tw, the power output was increased by as much as 70%.

Bosco and Komi (1979) also observed an improvement in performance with subjects who increased the amplitude of the eccentric movement by dropping from various heights (30-70 cm) before beginning their upward movement. With males the optimal drop height was 64.5 cm. while females had their best jumps when dropping from an average height of 47.6 cm.. Asmussen and Bonde-Peterson (1974) used a similar technique to demonstate that takeoff velocity could be increased by a factor of 1.11, although this enhancement tended to decrease once the drop height exceeded 40.0 cm.. Bosco and Komi (1979) included a damped drop jump where the subject was allowed to absorb some of the gravitational force over a greater change in knee angle. With this delay both the power and work done were lower than in the undamped jumps where upward movement began as soon as possible after landing from an elevated platform.

An increase in positive work through prior eccentric activity has also been observed in movement tasks involving flexion:extension of the elbow (Asmussen and Sorensen, 1971; Cnockaert, 1978; Bober et al., 1980; Cavagna et al., 1968) and forearm pronation:supination (Chapman, 1980). Cavagna et al. (1968) have shown a similar enhancement in isolated muscle fibres (toad sartorius and frog gastrocnemius).

Mechanisms Of Force Enhancement

Over the years numerous explanations have been given for the enhancement of force following stretch. Many of the hypotheses, particularly those derived using single fibre and/or isolated muscle preparations, suggest that enhancement is due to a temporary modification of fixed muscle properties eg. transient changes in the force-length and force-velocity relationships. Studies concerned with movements produced by intact muscle systems eg. vertical jump studies, also emphasize the role of reflex activity and its affect on the recruitment and rate coding of motor units. The literature summary which follows deals with both passive and active muscle components as well as possible neural influences. Much of this work has been reviewed by Cavagna (1977).

Storage of Elastic Energy

Having calculated the work done against gravity during walking Marey and Demeny (1885a)[1] wrote:

> "A part of the negative work stored in the muscles during each falling phase is recovered during the lifting phase which follows. But it is impossible up to now to estimate the value of this recovery of work which, however certainly exists."

Cavagna and Kaneko (1977) noted that the mechanical work done in fast walking surpassed that predicted by energy expenditure data. Their results suggest a positive work to net energy

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[1] translation quoted from Cavagna, G.A. (1977). Storage and utilization of elastic energy in skeletal muscle. Exercise and Sport Science Reviews 5, 89-129.

expenditure ratio of .35 to .4 while the theoretical efficiency of the contractile process is thought to be approximately .25 (Dickinson, 1929). They attributed this difference to the release of mechanical potential energy stored in the intramuscular connective tissues and tendons as the extensor muscles of the lower leg contract eccentrically in response to gravitational and inertial forces.

This storage of energy in the muscle's passive elastic components, first discussed by Hill (1950), was found to increase linearly with speed in level running. Cavagna et al. (1964) calculated the efficiency of positive work during running to be .4 to .5 while subsequent studies (Cavagna and Kaneko, 1977; Cavagna et al., 1976) indicate this value may be as high as .8 at speeds of 32 Km/hr. They suggest that since efficiency increases with speed, the excess positive work delivered is primarily the result of stored mechanical energy rather than the transformation of chemical energy by the contractile component.

Dawson and Taylor (1973) observed a similar trend in kangeroos where oxygen consumption was shown to decrease as speed of hopping increased. The mechanical energy stored was proportional to the square of the forces acting on the elastic elements. Since these forces increase with speed of hopping, the relative importance of elastic energy in the production of positive work is greater at higher speeds. Alexander and Vernon (1975) calculated that about 40% of the positive work done by the muscles of the leg of a wallaby at each step was sustained

by the elastic recoil of the gastrocnemius and plantaris tendons.

While it is true that a muscle must be at least partially contracted before it can store elastic energy it is generally felt that the recovery of mechanical energy more than compensates for the chemical energy spent to maintain tension during stretch (Cavagna, 1977). However this recovery phase is of limited duration since, as Fenn (1930) suggested, the elastic energy is continually being dissipated as heat.

Active stretch seems to temporarily modify muscle compliance enabling a greater amount of mechanical energy to be released for a given drop in force during shortening (Cavagna and Cittero, 1974). These changes in elastic properties are short lived and tend to be more pronounced in fatigued muscle (Vigreux et al., 1980).

Margaria et al. (1963) measured oxygen consumption during successive deep knee bends with a variable interval of time between flexion and extension of the lower limbs. As the length of the interval was decreased, the efficiency increased since more of the elastic energy stored during stretching could be recovered during subsequent shortening. Since both the storage and recovery of this elastic energy are time dependant, Asmussen and Sorenson (1971) recommend that in athletic activities with a wind up, the delay between eccentric and concentric phases be minimized. As the benefical effects of the wind up will be greatest during the initial part of the movement, utilization of

stored potential energy during the concentric phase of the movement is therefore also a function of the velocity of shortening (Cavagna et al., 1968).

This ability to utilize stored elastic energy not only depends on the internal structure of the muscle but also its orientation relative to other muscles and/or limb segments. Cnockeart (1978) reports a significant difference in the work done by antagonist muscle groups, elbow flexors and extensors, during to and fro movements.

Re-synthesis of ATP During Negative Work

According to Fenn (1930) and Elftman (1966) the functional significance of stored elastic energy is negligible since the muscle " cannot retain this store of energy without continuous contraction." During the negative work phase the stored energy is continually being been lost as heat and for tension to be maintained, chemical energy is required. Instead Fenn (1930) suggests that with eccentric contraction, some of the negative work done by the muscle is temporarily stored in the form of chemical potential energy. This is the result of a shift in the creatine kinase pathway favouring the synthesis and accumulation of adenosine triphosphate (ATP).

creatine phosphate + ADP = creatine + ATP

With the onset of the concentric phase, this excess ATP is rapidly metabolized and converted back into mechanical energy allowing the muscle to increase its work production.

Having observed that the work done exceeds the difference in heat production for stretch and no stretch conditions, Abbott et al. (1951) proposed that the missing work was absorbed in chemical resynthesis. A net re-synthesis of ATP from negative work has also been suggested by Hill and Howarth (1959).

Cavagna et al. (1968) questioned the validity of Fenn's hypothesis, stating that only when these energy stores fall below a critical level, which we associate with fatigue, does potential chemical energy become a limiting factor. With short term aerobic and anerobic activities, there is an excess of chemical energy present with or without previous stretch and this therefore can not be used as an index of creatine kinase activity. Additional work by Infante et al. (1964), Gillis and Marechal (1974) and Curtin and Davies (1972) tends to support the argument put forward by Cavagna et al. (1968), and it seems unlikely that enhancement is the result of an increase in ATP synthesis.

Potentiation of Contractile Machinery

Passive elastic elements within muscle are unable to store enough mechanical energy, even when coupled with changes in compliance, to account for all of the increased positive work observed. In 1968 Cavagna et al. were unaware of the increased compliance, and suggested that a portion of the enhanced work output was due to a "potentiation of the contractile component itself." Cavagna and Citterio (1974) have since shown that with

active stretch) intact muscle is able to contract against a force greater than Po, the maximal isometric force predicted using Hill's (1938) equation. They proposed that recoil of passive elastic structures with the onset of the concentric phase allows the contractile component to shorten more slowly. This shifts the muscle upwards and to the right on the force-velocity curve. The effect is greatest near Po and decreases exponentially as shortening approaches maximum velocity (Vmax).

Using single fibres bathed in relaxing solution Guth et al. (1977) were able to show that the excess tension with stretch does indeed involve altered cross-bridge activity and is not simply due to the properties of the muscle's passive elastic elements ie. tendons, intramuscular connective tissue, etc. Cavagna and Kaneko (1977) examined the stretch over a range of temperatures and found that the increase in work done was considerably larger than would be expected from recoil of the elastic components alone. The fact that single muscle fibers, lacking much of the associated connective tissue, showed a similar shift in the force-velocity curve also suggests that some other process must be involved (Edman et al., 1978a, 1978b).

Active stretch appears to have a direct effect on the contractile machinery by modifying the molecular arrangement in some way and increasing the amount of tension which can be generated (Cavagna and Citterio, 1974; Edman et al., 1978a,1978b). It is thought that this rearrangement might open

up additional actin binding sites which were previously unable to participate in the formation of actomyosin linkages. With this increase in the number of active cross-bridges, available chemical energy could be delivered at a greater rate and the ATP stores utilized more effeciently (Cavagna et al., 1968). A given amount of work can be accomplished over a shorter period of time thereby increasing the power output and reducing the maintenance heat requirements. A reduction in ATP consumption during stretch has been shown by Curtin and Davies (1972).

If the enhancement process was the result of changes in myosin ATPase activity we would expect an increase in Vmax. While this increase is observed with increased temperature (Julian et al., 1971) and the addition of hypotonic solutions (Edman and Hwang, 1977), Vmax does not change appreciably with active stretch (Edman et al., 1978a).

Curtin and Woledge (1979) used the ratio of tension to rate of ATP splitting as an indicator of the number of actomyosin linkages present at a given instant in time, and found little difference between isometric and "prior stretch" conditions. The heat plus work (h+w) produced, an index of all chemical reactions occuring within the muscle was also not significantly different in the two types of contraction. Since the splitting of ATP is the major source of energy for the contractile process Curtin and Woledge (1979) feel that the rise in tension after active stretch is not due to a proportional increase in the number of conventional cross-bridge cycles. X-ray diffraction

studies conducted by Yagi and Matsubara (1977) failed to produce any evidence of the structural changes which would be expected if additional cross-bridges were present following active stretch. Still it may be possible to alter the force generating capability of each cross-bridge and not affect the kinetics which influence Vmax (Edman et al., 1978a).

Cooling usually causes muscle fibers (Buchtal and Kaiser, 1951) and tendons (Apter, 1972) to become stiffer. However Asmussen et al. (1976) found that the stiffness of leg extensor muscles was not measurably influenced by temperature changes in the range of 30°C to 37°C. Since stiffness was the same for a given mean tension level regardless of temperature they suggest that much of the muscle's series elasticity is located in the cross-bridges.

With recent models of muscle, cross-bridge structure has been modified to include an "instantaneous elastic element" which is responsible for short-range stiffness (Huxley and Simmons, 1971). It is felt that with active stretch cross-bridges do not simply detach spontaneously with the splitting of ATP but rather remain in a locked on position until the amplitude and/or velocity of stretch exceeds some critical level (Noble and Pollack, 1977; Sugi, 1972). At this point the actomyosin linkages "give", discharging the tension stored in the strained elastic elements. Griffths et al. (1980) feel that it is this detachment of cross bridges which accounts for the decrease in muscle stiffness observed during large amplitude

stretches. Evidence of cross-bridge slip has been reported in frog muscle fibers (Sugi, 1972), isolated whole frog muscle (van Atteveldt and Crowe, 1979) and insect fibrillar muscle (Guth et al., 1977; 1979).

Reflex Activity

With isolated muscle preparations, force enhancement can be attributed to changes in the contractile machinery and/or the storage and utilization of elastic energy. In voluntary movements such as the vertical jump, neuromuscular connections remain intact and performance may well be influenced by proprioceptive inputs as well.

It has been suggested that with prior eccentric activity it may be possible to reduce the electromechanical delay (EMD), i.e. the time lag between onset of electrical activity and the development of tension, for the concentric phase since it seemed to be a function of the rate of stretching of the series elastic component (SEC) (Norman and Komi, 1979). With vertical jump studies Bosco and Komi (1979) observed considerable EMG activity in leg extensors before the actual upward movement was initiated. This preactivation would be advantageous for two reasons. It would tend to increase muscle stiffness which in turn increases the effectiveness of force enhancement mechanisms i.e. the storage and utilization of elastic energy. It also enables the muscle to be more or less fully active at the start of the concentric phase of movement. Both of these would lead to

an increase in positive work.

EMG levels during an eccentric contraction have been shown to be greater than with a maximal isometric contraction (Schmidtbleicher et al., 1978) or a pure concentric contraction (Viitasalo and Bosco, 1982). However in an earlier study Asmussen and Sorensen (1971) found that EMG activity was maximal at or before the onset of tension development even when the muscle shortened from rest. The more gradual rise in tension with pure concentric contractions does not appear to be due to "delayed innervation" but rather differences in muscle properties.

Traditionally the neuromuscular response to external length changes has been attributed to the recruitment of additional motor units and/or an increase in the rate of firing of previously active motor units by the spinal stretch reflex. The role which each of these mechanisms plays in the overall reflex response seems to depend on a muscle's fibre composition and function (Kukulka and Clamann, 1981).

In decerebrate cats, Nichols and Houk (1973, 1976) and Hoffer and Andreasson (1981) found that with large stretches soleus muscle stiffness was greatly reduced when the dorsal roots to the muscle were cut. This was particularly evident at low operating forces and tended to become less prominent as the preload was increased. This suggests that muscle stiffness and not length may be the primary regulatory concern of the stretch reflex (Nichols and Houk, 1976; Hoffer and Andreasson, 1981). The

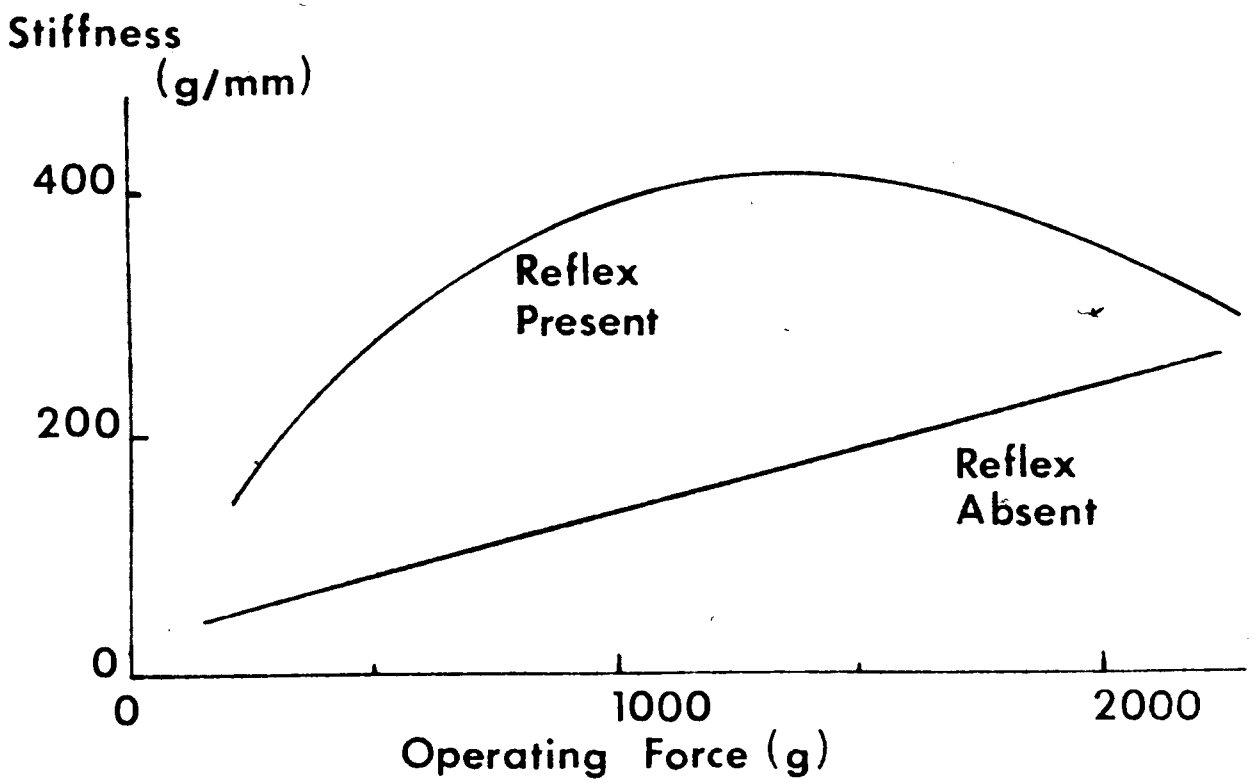
relative contribution of this recruitment of additional tension via the stretch reflex and the intrinsic mechanical stiffness of actively contracting fibres during stretch can be seen in Figure 1.

Passive stretch tends to produce considerably less spindle activity than an eccentric contraction of similar amplitude and velocity. This difference has been explained by increased fusimotor activity under eccentric conditions which may account for the rise in "net" reflex compensation at low to medium operating levels (Burke et al., 1978).

This reflex activity tends to improve the linearity of the stretch response, compensating for the non-linear mechanical properties of active soleus muscle eg. cross-bridge slip. The time interval between the onset of stretch and the appearance of reflex activity is such that it prevents mechanical yield until central long loop reflexes can take over (Nichols and Houk, 1976). In an intact limb the overall joint stiffness can also be altered through the recruitment of added force from agonists and/or antagonists (Hoffer and Andreasson, 1981).

The significance of the spinal stretch reflex as a regulatory mechanism has been questioned by several investigators. Hammond et al., (1956) feel that in man the role of the monosynaptic strech reflex is secondary to that of longer latency reflexes. Melvill Jones and Watt (1971) also stress the importance of a "functional stretch reflex" (FSR) which in man has a latency of approximately 120 msec. compared to the 40-50

Figure 1: Dependence of muscle stiffness on operating force. Data obtained in the presence of reflex and during cut nerve stimulation of cat soleus muscle. (Redrawn from Hoffer and Andreasson, 1981)


Stiffness
(g/mm)
400
200
0
0
1000
2000
Operating Force (g)
Reflex
Present
Reflex
Absent

msec. delay generally associated with the spinal reflex. While this delayed EMG activity has been observed in numerous muscle systems eg. human triceps (Dietz et al. 1981), flexor/extensor muscles of the wrist in monkey (Bawa and Tatton, 1979), the long flexors of the human thumb (Marsden et al., 1976), its origin and significance are still uncertain.

It is unlikely that 1A fibres are the only afferent pathway activated during eccentric movements. Group II spindle afferents, golgi tendon organs, joint and cutaneous mechano-receptors will surely be involved in the overall response. In fact it maybe the reflex excitation of separately responding motor neuron subpopulations which account for the series of peaks in the averaged EMG (M1,M2,M3) following joint displacement (Bawa and Tatton, 1979). Hagbarth et al. (1981) tend to disagree, stating that the 'burst intervals' observed can not be explained by stepwise recruitment of different mechano-receptor populations or the synchronous firing of receptors within a single population.

Pompeiano (1960) found the relationship between soleus muscle stiffness and the amplitude of stretch to be the same before and after the dorsal roots had been cut. Grillner (1972) also suggests that the visco-elastic properties of pre-activated muscle alone must be able to compensate for sudden changes in load and/or length since at high velocities the monosynaptic time delays are too long for reflex compensation. Dietz et al., (1980) point out that the stretch velocities and amplitudes used

in many of these studies represent a small fraction of those which occur naturally in man. Even so several studies (Marsden et al., 1976 ; Goodwin et al., 1978) have shown short latency EMG responses and a clear increase in the size of the spinal reflex with speed of stretch, even at the slower velocities.

Factors Influencing the Enhancement of Force

The following section deals briefly with those factors which, by altering the mechanical properties of the muscle or the response of proprioceptors, may influence the forces produced during an eccentric contraction. The conditions present during stretch will also ultimately determine the amount of positive work done and/or power generated if the muscle is then allowed to shorten.

Muscle Properties

Temperature

There is considerable disagreement within the literature concerning the effect of temperature on muscular performance. Asmussen et al. (1976) report a significant positive correlation between muscle temperature (Tm) and maximal isometric strength while Binkhorst et al. (1977) suggest the two are independent of one another. Bergh (1980) found that isometric strength was not affected by Tm while maximal dynamic strength showed an increase of 4-5% per degree Tm. This effect was observed over a range of concentric velocities, producing a shift to the left on the force-velocity curve. Since force enhancement is a measure of the tension generated above the isometric maximum for a given muscle length, it will no doubt be influenced by temperature as

well.

Bergh and Ekblom (1979) found in both the vertical jump and sprinting, performance levels were lower at sub-normal body temperatures and increased by 4.2% and 5.1% per degree respectively at elevated temperatures. Since there was no apparent change in EMG activity it is suggested that the variations in temperature had little if any effect on temporal coordination in these activities.

Evidence presented by Asmussen et al. (1976) on the other hand indicates that the ability to store mechanical energy during an eccentric contraction ie. a downward countermovement, is enhanced by a reduction in temperature. While the positive work done at cooler temperatures was reduced due to a slower development of tension, the effeciency of movement was improved. Under warm conditions (37°C) approximately 4.45% of the absorbed energy was re-used while the figure was 11.2% in the cold condition (32°C). Studies using single fibres have produced more dramatic results. Sugi (1972) found force enhancement increased by as much as 50% when the temperature was lowered from 20° to 0°C.

The decay in tension is much more rapid in mammalian muscle at 37°C (Stevens et al., 1980) than with frog muscle at 0°C. Single fibre studies by Edman et al. (1978) indicate that 0°C excess tension may persist for 4.3 seconds after stretch. Subsequent work by the same authors (Edman et al., 1978b) has shown evidence of enhancement 7.0 seconds after the stretch has

been completed. Again this may not be due to temperature differences alone but also differences in series elasticity.

Since both the development and decay of tension during isometric contractions is slower at reduced temperatures, it has been suggested that the biochemical processes ie. ATP splitting associated with each cross-bridge cycle must be altered in some way. The result is an increased stiffness and resistance to any change in length, be it shortening or stretch. Muscle relaxation is also slowed, and stiffness persists for a longer period allowing more of the elastic energy to be carried over into the positive work phase (Asmussen et al., 1976).

Level of Activation

When examining force enhancement, muscle activation is generally assumed to be maximal prior to stretch. Joyce et al. (1969) state that the rates of stimulation used in most muscle studies are considerably higher than those present during normal movements. Using intact cat soleus they discovered that for a given amplitude and velocity of stretch, the enhancement profile varied with the rate of stimulation. At higher rates (35 pps) the tension consistently rose above isometric levels, while lower stimulus rates (5-15 pps) produced forces less than isometric for that muscle length.

With high stimulus rates the rapid turnover of cross-bridges more than makes up for any mechanical slippage. As the level of activation decreases there is a reduction in the

number of cross-bridge cycles per unit time. The slippage can no longer be adequately compensated for and tension falls below isometric.

Since much of the short range stiffness is contained within the active cross-bridges, submaximally contracting muscle tends to be more compliant. While this allows more of the absorbed elastic energy to be recovered, the forces involved in stretching the muscle are smaller. With fewer motor units activated the end result is an overall reduction in recoverable elastic energy (Cavagna, 1977). Still Cnockeart and Goubel (1975) report that a submaximally active human biceps which has been stretched is able to accomplish the same amount of work as a maximally active muscle which has not been previously stretched.

Both Fenn (1930) and Grillner (1972) found the forces present following active stretch to be lower or the same as isometric for a given muscle length. Cavagna (1977) suggests that the above observations can be explained by differences in the number of active motor units present under each condition. Bawa (1981a) agrees that differences in the level of activation may account for this discrepancy. However at or near maximal activity it would be the result of differences in firing frequency not the number of motor units recruited.

Stretch Parameters

Belagyi et al. (1979) found that the profile of force enhancement (P/Po) varied depending on the time characteristics of the applied stretch (ie. linear; parabolic; exponential). In most instances however active stretch is assumed to represent a linear or ramp change in muscle length. Under these conditions force enhancement (P/Po) has been shown to be a function of both the ramp parameters (ie. magnitude and velocity of stretch) and absolute muscle:sarcomere length.

Using bundles of frog semitendinosus fibres Sugi (1972) found that short stretches (less than 10% length) carried out at a velocity of 15cm./sec (10L/sec) produced a smooth rise in tension through out the eccentric phase and a gradual decay back towards isometric during the hold phase. The magnitude of peak tension increased with both velocity and amplitude of stretch while the decay in tension was more rapid as stretch velocity increased.

Due to the time required for reformation of actomyosin linkages there is a proportional decrease in their number as velocity increases (Sugi, 1972; Joyce et al., 1969). With slow to moderate stretch velocity, it is felt that this reduction in number is overshadowed by the enhanced force generated by each strained cross-bridge. However as velocity increases (30-80 cm./sec.; 20-50 L/sec.) more and more cross-bridges are forcibly detached allowing the extended elastic components to recoil before the stretch can be completed. As the cross links "give"

there is a sharp initial drop in tension , and then a gradual decay phase similar to that observed at slower stretch velocities. With still faster velocities (80-150 cm./sec.; 70-100 L/sec.) this initial drop is more pronounced, and tension falls below isometric for that muscle length. Tension then climbs slowly back towards isometric. Sugi (1972) reports that both the magnitude of the initial drop and the time required for this secondary rise in tension increases with amplitude of stretch.

Van Atteveldt and Crowe (1980) re-examined the work of Sugi (1972) and Edman et al. (1976;1978a;1978b) using isolated whole muscle (frog sartorious). While the same general patterns emerged there were several differences. Muscle slip was observed at much lower velocities of stretch and the decay in enhancement was considerably slower than with fibre preparations. Since the muscle used was dissected out complete with its tendinous insertions, the authors conclude that the differences are the result of additional series elastic connections.

Flitney and Hirst (1978) used intact frog sartorious muscle and found that enhancement increased with speed of stretch until a certain temperature dependent critical velocity was reached. Beyond this point (3.8 mm/sec.; .14 L/sec at 0 C) tension was more or less independent of any further increase up to 64 mm/sec. ; 2.35 L/sec. Filament displacement greater than 11-12 nm. (1.2% of muscle length) produced an abrupt increase of approximately 33 mm. in muscle length. They state that this is

considerably larger than most estimates for the working range of a single cross-bridge. However in reviewing some of the work in this area, Noble and Pollack (1977) noticed that the magitude of these sudden length changes was generally in the range of 40-45 nm. per sarcomere. They suggest this value is similar to the helical repeat spacing of cross-bridges along the thick filament.

In discussing their results both Flitney and Hirst (1978) and Griffths et al. (1980) emphasize the fact that the amount of elasticity in single fibres is considerably less, approximately 20%, of that in whole muscle. Griffths et al. (1980) used single fibres in which the sarcolemma had been mechanically removed and observed slippage at stretch amplitudes of 1.2%. Similar results were reported by Flitney and Hirst (1978) using isolated whole muscle. On the other hand Edman et al. (1978a;1978b) subjected single fibres to stretches much greater than 12 nm without any indication of the cross-bridges giving out. While Sugi (1972) observed slip, it was not evident until the amount of stretch rose above 6.0% L.

Total Muscle Length

With single fibres from frog semitendinosus muscle Edman et al. (1978a;1978b) found the rise to peak tension during stretch to be dependent on velocity but not magnitude of stretch or degree of filament overlap. After stretch, excess tension was independent of stretch velocity and at sarcomere lengths between

1.9 and 2.3 um. it was also independent of stretch amplitudes greater than 25 nm. per sarcomere. At lengths greater than 2.3 um, percentage excess tension increased with muscle length.

Having accounted for changes in resting tension Hill (1977) demonstated that the excess tension maintained after stretch increases with muscle length. Peak tension on the other hand tends to decrease at longer muscle lengths. van Atteveldt and Crowe (1980) report that excess tension remains more or less constant at shorter muscle lengths up to Lo, the length corresponding to maximum isometric force. Once this value has been surpassed enhancement tends to increase with length over the downward portion of the isometric force-length curve.

Stretch Conditions and Reflex Activity

When muscle is exposed to a ramp and hold displacement series the response of the spindles has traditionally been given as a function of acceleration, velocity and position or muscle length. Recent work by Houk et al. (1981) suggests that the firing pattern is not simply a sum of these three components. They feel it can be more accurately descibed as "a product relation between muscle length and a low fractional power of velocity".

Spindle activity tends to vary with the level of activation or preload. Lengthening contractions produce a higher spindle discharge rate than a passive stretch of similar amplitude and velocity (Burke et al., 1978). The sensitivity of the stretch

reflex is greatest for small changes in muscle length and/or load (Matthews, 1972). This may be due to the fact that following the initial reflex burst the excitability of the segmental pathway is temporarily reduced (100-150 msec) due to a "rebound" membrane hyperpolarization (Gottlieb et al., 1970).

Few studies have examined how reflex activity changes with temperature. A study by P. Bawa (currently in progress) indicates that a reduction in temperature produces little change in the amplitude of the stretch response but causes it to be shifted in time.

Summary

An enhancement in positive work is observed when muscle is forcibly stretched from the isometric condition. This enhancement has been attributed to a variety of mechanisms. Some earlier hypotheses ie. the re-synthesis of ATP during the negative work phase have been more or less abandoned and at present it appears that the enhancement of positive work is the result of (a) the storage of mechanical energy in passive elastic structures (b) changes in the contractile properties of the actomyosin cross-bridges and, if the neural connections are intact, (c) reflex activity. These mechanisms appear to be sensitive to changes in the characteristics of the applied stretch, filament overlap, muscle temperature and level of activation. Changes in one or more of these factors does not necessarily have the same effect on the enhancement profile in

all types of muscle prepartions (*in vivo* or *in vitro*). Differences in series elasticity, chemical preparation, fatique, fibre composition, etc. will alter the development and decay of excess tension. Yet certain generalities have been made about the response of isolated muscle to active stretch.

Eccentric force tends to increase with velocity of stretch. This relationship eventually breaks down at high velocities as cross-bridges begin to slip. Increases in amplitude of stretch and total muscle length particularly at lengths greater than Lo, seem to produce a larger enhancement effect. With muscle *in situ* an increase in velocity of stretch or muscle length also produces an increase in reflex activation. The rate of decay in tension during the hold phase is thought to be a function of prior stretch velocity and total muscle length. A reduction in temperature and/or an increase in level of activation tends to magnify the enhancement process by increasing the tension generated during stretch while prolonging the decay of excess tension after stretch.

III. Methods

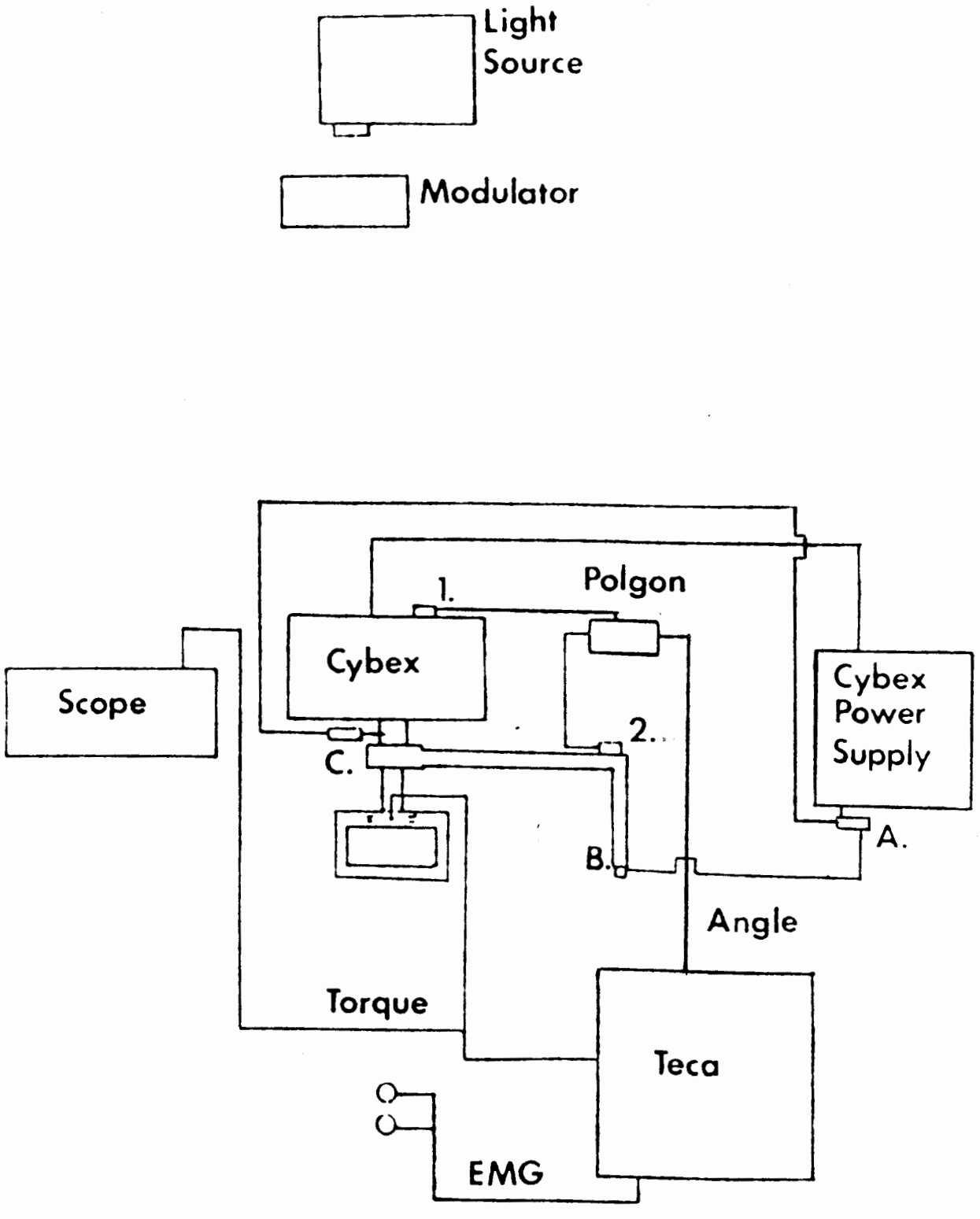
Subjects

The subject population consisted of five healthy males, each right handed, ranging in age from 22 to 40 years. The subjects had no known musculo-skeletal abnormalities which might restrict their range of movement and/or torques during supination. One of the subjects (AC) is a proficient squash player and was therefore somewhat more familiar with the action of supination/ pronation under a variety of conditions of activation and velocity. In addition this subject (AC) had performed the actions many times during experiments which utilized the present apparatus.

Apparatus

The experimental set up is summarized schematically in Figure 2. A polarized light goniometer (POLGON) (c.f. Grieve, 1969) which has a range of 180° was used to monitor changes in angular displacement. The device consists of a modulator unit which plane polarizes the light emitted from an incandescent source and a pair of photo sensors. One of the sensors acted as a stationary reference while the other was attached to the arm of a Cybex II Isokinetic Dynamometer (Lumex Inc., New York). Movement of one sensor relative to the other produces a phase

Figure 2: Schematic diagram of experimental apparatus.


Light
Source
Modulator
Polgon
1.
Cybex
Scope
2.
C.
Cybex
Power
Supply
A.
B.
Angle
Torque
Teca
EMG

shift which is converted by the POLGON module (PG6) into a voltage signal. The output was presented on the oscilloscope of a Teca (model TE4, White Plains, New York.) recorder which also produces a hard copy of the data on UV sensitive paper. Calibration of the POLGON was carried out using a carpenter's level to position the handle at 0° and 90°. Zero degrees represented the position in which the handle was horizontal with the forearm in pronation and the palm facing downwards.

Torques were measured using strain gauges (Micro-Measurements Ltd. Romulus, Mich.) attached to the handle in a Wheatstone bridge configuration. The linearity of these gauges has been previously verified. This signal was passed through an AD6 amplifier contained within the Teca unit. Due to the electronic configuration of the POLGON there is a 40 msec. delay between angular displacement and both the torque and EMG traces on the UV paper. This delay has been corrected for in all Figures.

Raw EMG from the biceps was obtained using surface cup-type electrodes (IMI Ltd., Newport, Calif.) and a Teca AA6 MK III amplifier. The signal was filtered using a low frequency cut-off of 32 Hz and a high frequency cut-off of 1.6 KHz. The output from this unit was then rectified and passed through a simple R.C. type filter with a time constant of 200 msec (Teca I6 integrator module).

With several minor modifications the constant velocity qualities of the Cybex II unit can be used to produce ramp and

hold stretches of various amplitudes. Three switches were installed which made it possible to override the standard speed control and limit the movement of the Cybex arm. With switch A in the on position, movement of the Cybex arm could be stopped manually by depressing button B or automatically, when switch C was tripped by a metal paddle attached to the Cybex potentiometer. With switch A off, switches B and C no longer influenced the Cybex circuitry and the arm could be re-positioned.

Procedure

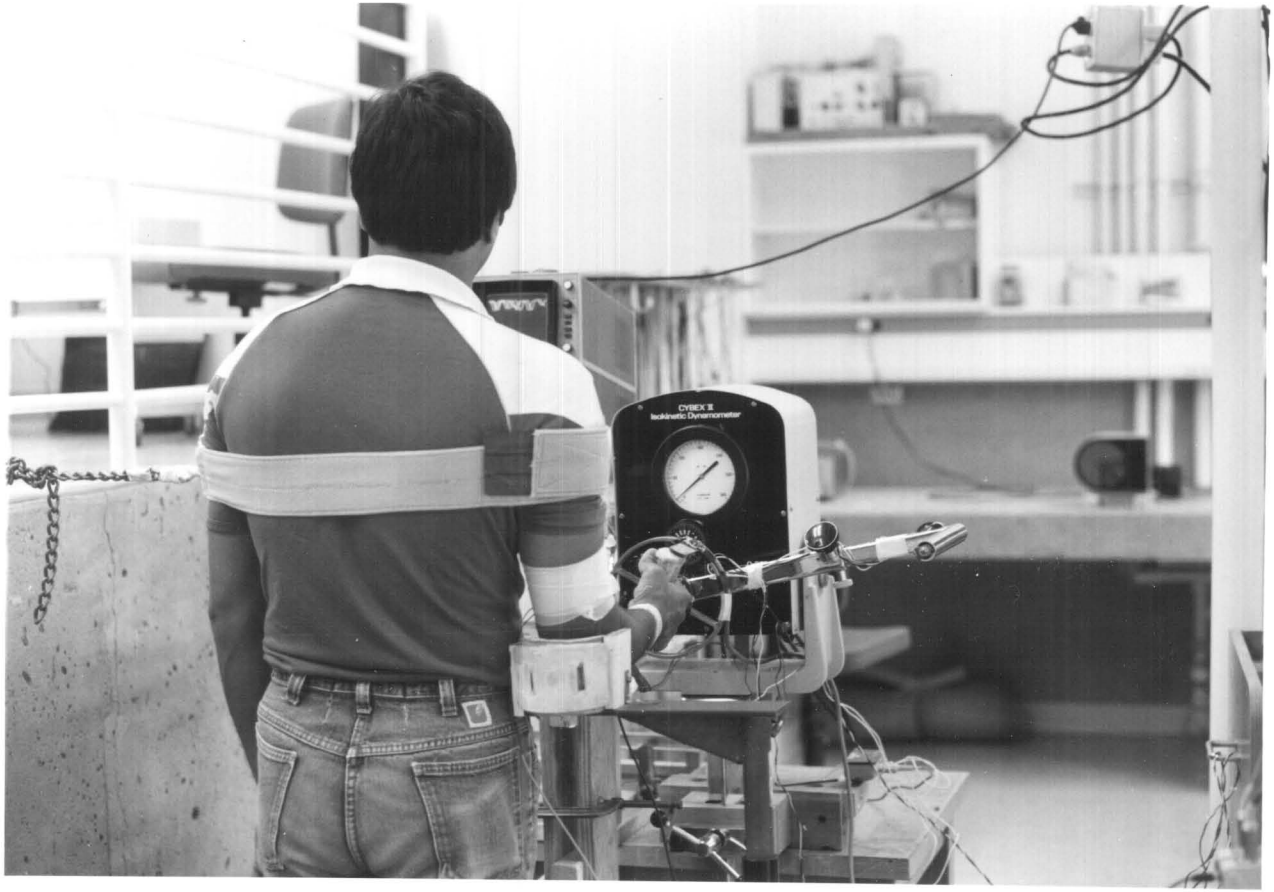
EMG electrodes were attached to the biceps and a ground strap was fixed across the wrist. An attempt was made to place the electrodes in the same position each test day. The handle and elbow support were adjusted to produce 90 degrees of elbow flexion and the subject was positioned as shown in Figures 3 and 4.

During forceful supination there is a tendency to dip the right shoulder. A harness was used to keep the subject as upright as possible. This also held the upper arm tight against the side of the chest, preventing unwanted rotation of the shoulder.

Prior to each trial hand position was checked to ensure that the subject's middle finger lined up with the axis of handle rotation. The handle was placed at 10° (θF1) and a record of isometric torque at that angle was taken. The handle was then

Figure 3: Positioning of subject. Side view.

Figure 4: Positioning of subject. Rear view.


CYBEX II
Isokinetic Dynamometer

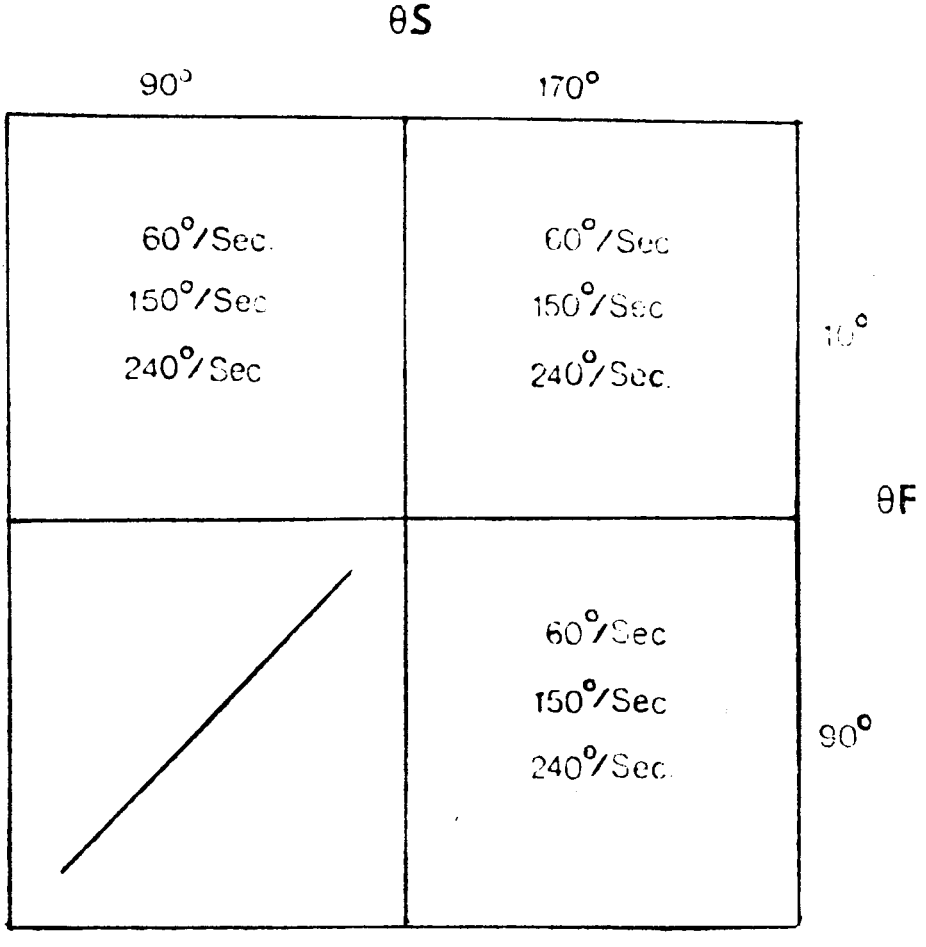
passively rotated to position θS1 (90°) and the desired stretch velocity (60, 150, 240 °/sec.) was dialed in on the Cybex. With button B depressed the Cybex arm was fixed and the subject supinated maximally. Pre-stretch isometric levels for each position were kept as constant as possible by providing the subject with a visual display of torque on an oscilloscope.

When button B was released, the Cybex arm was rotated counter clockwise until the handle reached θF1 (10°). At this point switch C was tripped, reducing the cicuit resistance to zero. This prevented any further movement of the handle. During this hold phase the subject maintained a maximal contraction of forearm supination for a period of 4 to 5 seconds. This was long enough for the torque to drop back to the isometric level at the muscle length corresponding to θF1. Stretches from θS1 to θF1 were then carried out at the other velocities.

An isometric reading was taken prior to each stretch trial, leaving approximately 1 minute between contractions. In an attempt to reduce the effects of fatigue a longer rest period of 5 minutes was used between stretch trials. The above procedure was repeated starting from position θS2 (170°) and finishing at θF1 (10°) and again from θS2 (170°) to θF2 (90°) (Table 1.). With each degree of stretch there was approximately an 0.016 cm. change in biceps muscle length (Appendix A).

At the start of each test session, a set of submaximal trials were carried out from θS=170° to θF =10° at the highest stretch velocity (240°/sec.). The purpose of these additional

Table 1: Summary of stretch conditions.


θS
90°
170°
60°/Sec.
150°/Sec
240°/Sec
60°/Sec
150°/Sec
240°/Sec.
10°
θF
60°/Sec
150°/Sec
240°/Sec.
90°

stretches was to establish if reflex activity changed with the level of prior isometric contraction or preload. Each subject repeated this complete series of stretches 5 times over a period of two to three weeks.

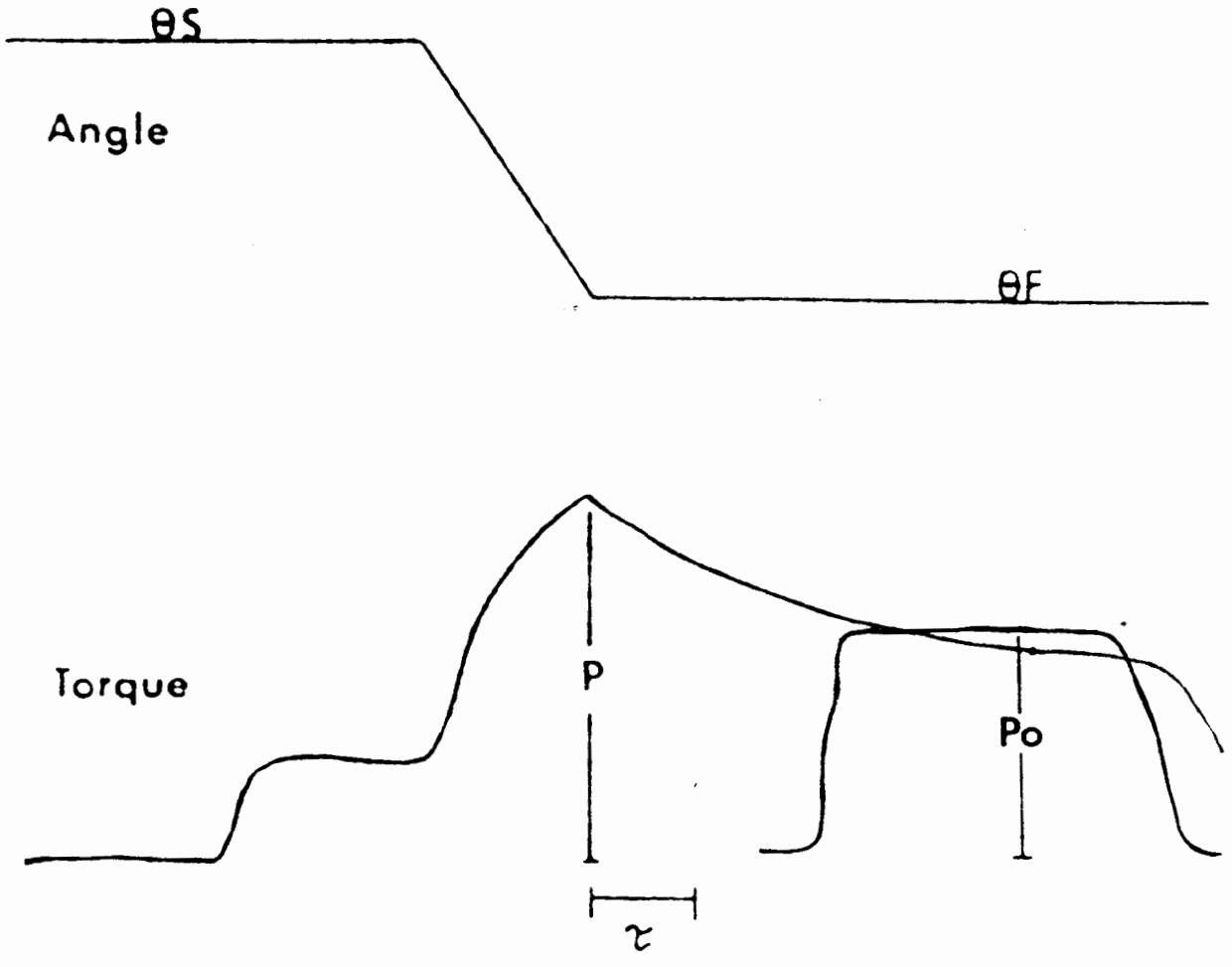
From the pilot study it was evident that initially the subjects might have difficulty producing a maximal voluntary contraction for each trial. This was particularly true of supination at θS2 (170°). For this reason the subjects were allowed to become familiar with the task prior to the actual test trials. In order to minimize the learning effect and prevent the subjects from anticipating the expected response, stretch amplitudes and velocities used during each series were randomized.

Since P/Po is a ratio of eccentric tension over isometric tension at the same muscle length, it is important that the conditions be the same for each contraction. While an attempt was made to standardize the conditions for each trial it was assumed that those factors over which we had little control (eg. muscle temperature) remained more or less constant thoughout the experiment.

Analysis

Figure 5. shows a composite of the torque data collected during a prior isometric contraction at θF1 and a stretch trial with a final position of θF1.

Figure 5: Sample record to show how P/Po and decay constants (γ) were measured. The top trace represents the change in angle from θ S to θF. Stretch velocities were calculated by differentiating the angular displacement trace.


θS
Angle
θF
Torque
P
Po
τ

Angular Velocity

Although the Cybex II isokinetic dynamometer was originally developed for rehabilitation purposes, over the past decade it has frequently been used as a research tool. Recently however the validity of some of these Cybex studies has been questioned. Failure to take gravitional effects into account may produce considerable errors when conducting various muscle performance tests (Winter et al., 1981). There also appear to be calibration problems which make it "necessary to calibrate the Cybex II isokinetic dynamometer every testing day and at every test speed" (Olds et al. 1981).

Having compared the actual stretch velocities from the UV traces with those dialed in on the Cybex it was apparent that the Cybex speed control was not properly calibrated. Rather than take the time to calibrate the Cybex each test day the pre-determined velocities were selected on the Cybex control but the actual strech velocities were calculated through numerical differentiation of the angular displacement traces. Since none of the Cybex transducers were used, calibration errors in torque and/or angle were not a problem. The differences between the pre-selected values and the true stretch velocities are shown below.

	Cybex	Corrected Value ±SD
Velocity 1.	60°/sec.	63.1±.88°/sec.
Velocity 2.	150°/sec.	144.5±.81°/sec.
Velocity 3.	240°/sec.	227.0±1.6°/sec.

P/Po

Peak torque (P) from the stretch trials and isometric torques (Po) were measured as displacements from the zero baseline. Peak torque following stretch was then divided by the average isometric torque at the same finishing position. The ratio P/Po represents peak eccentric/isometric torque. While this ratio is not a true measure of force enhancement "per se" it can be used to show how the enhancement mechanisms as a whole behave under the various stretch conditions.

Decay Constants

The decay in torque after stretch is expressed as a decay time constant ( ). One time constant represents the time required for torque to drop exponentially by 63% of the difference between P and Po. Due to the fixed visco-elastic properties of muscle the decay in torque after stretch due to recoil of the SEC should have a similar profile to the rise in torque during an isometric contraction at the same muscle length. By determining the time constant for isometric torque and comparing it with the decay time constant for the hold phase it should be possible to make some general qualitative statements about the separate contributions of storage of elastic energy and enhancement of the contractile machinery. If storage of elastic energy was the only factor responsible for

the enhancement of torque after stretch then one would expect the isometric rise time constant to be more or less the same as the decay constant.

EMG

Changes in the rectified smoothed EMG were used to indicate any gross reflex increase in motor unit activity. With a filter time constant of 200 msec. it is difficult to make any conclusive statements about the reflex response to stretch since short latency bursts will most likely be masked by the EMG processing. Still if reflex recruitment and/or intensity coding of motor units are contributing to the rise in tension during stretch eccentric EMG should be greater than isometric EMG at the same muscle length. The EMG response at the end of stretch were therefore measured and compared with the isometric levels at the same finishing position.

Statistics

The means for both P/Po and the decay constants for each of the subject's 5 trials were calculated. The variances of the group response, for each experimental condition, were analysed using a two way ANOVA (BMDP2V: Analysis of variance and covariance with repeated measures, Health Sciences Computing Facility, UCLA). When the ANOVA showed significance the differences between the means were examined using Tukey's test

for pairwise comparisons among means.

IV. Results

Torque and EMG data was collected for the 9 different stretch conditions outlined in Table 1. These stretch trials were repeated 5 times by each subject and the mean values for P/Po and decay constants were calculated.

Torque Changes During Stretch

While the shape of the torque records differed slightly with each subject the same basic patterns were observed overall (Figures 6 and 7). With the onset of stretch torque rose sharply. Both the magnitude and duration of this initial rise varied depending on the stretch conditions. With short quick stretches from 90°-10° or 170°-90° this steep phase persisted throughout the stretch and torque peaked as the movement stopped. With slower velocities there was a much more gradual increase following the initial rise in torque. For any given velocity an increase in the amplitude of stretch (170°-10° vs. 90°-10°) had a similar effect. Torque rose sharply at first and then more slowly during the latter part of the movement. In both cases peak eccentric torques were recorded as movement ceased. When a reduction in velocity was coupled with an increase in the amplitude of stretch ie. 170°-10° torque response was much more rounded reaching a peak before the stretch could be completed.

In many of the trials subjects RB and BC in particular, consistantly showed a slight drop in torque midway through the

Figure 6: Sample record of angular displacement (top trace), torque response (middle trace) and rectified:averaged EMG (bottom trace) during and after medium velocity stretch from $\theta S2$ (170°) to $\theta F2$ (10°). This sort of response was typical of those produced by subjects AC, AP and CT.

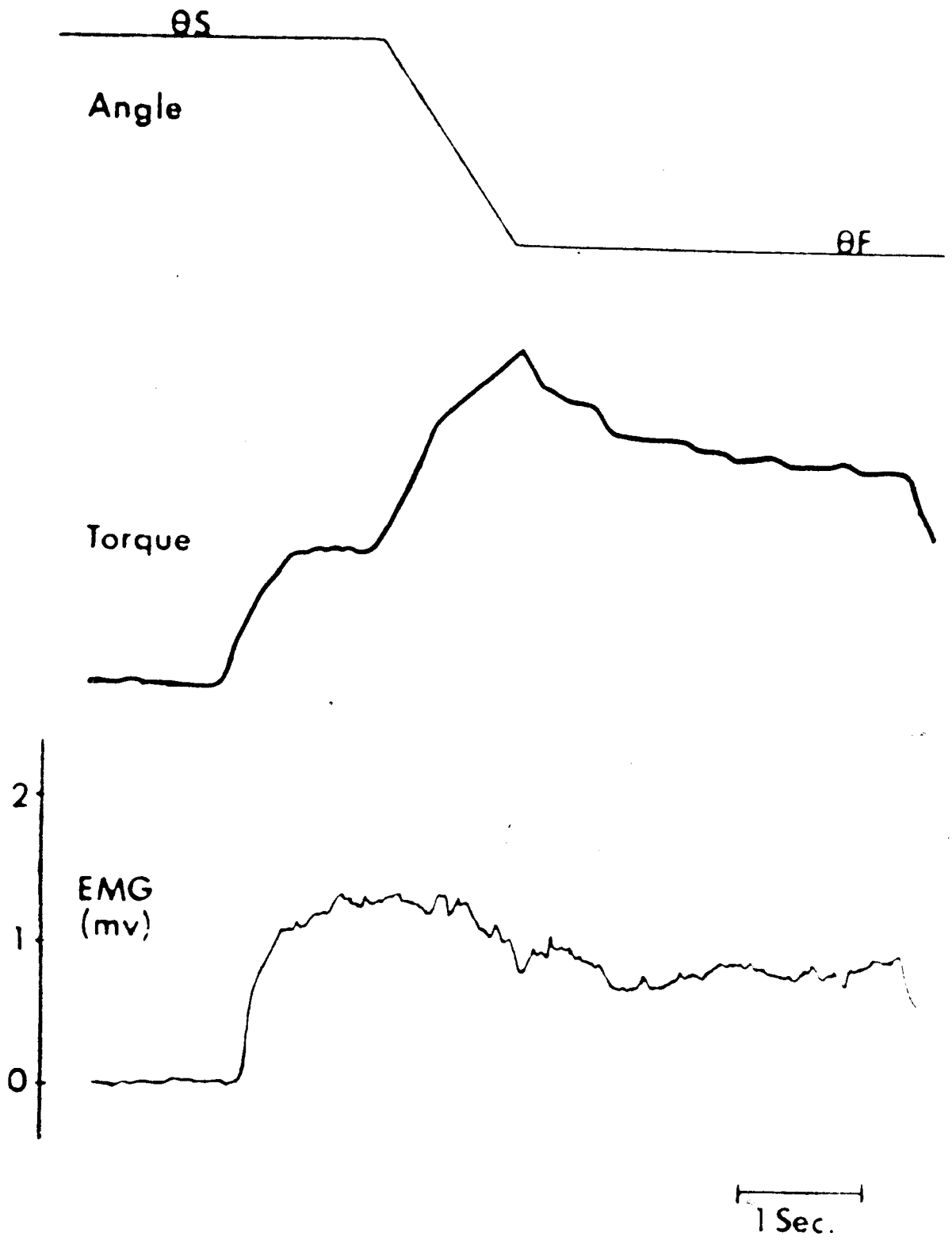
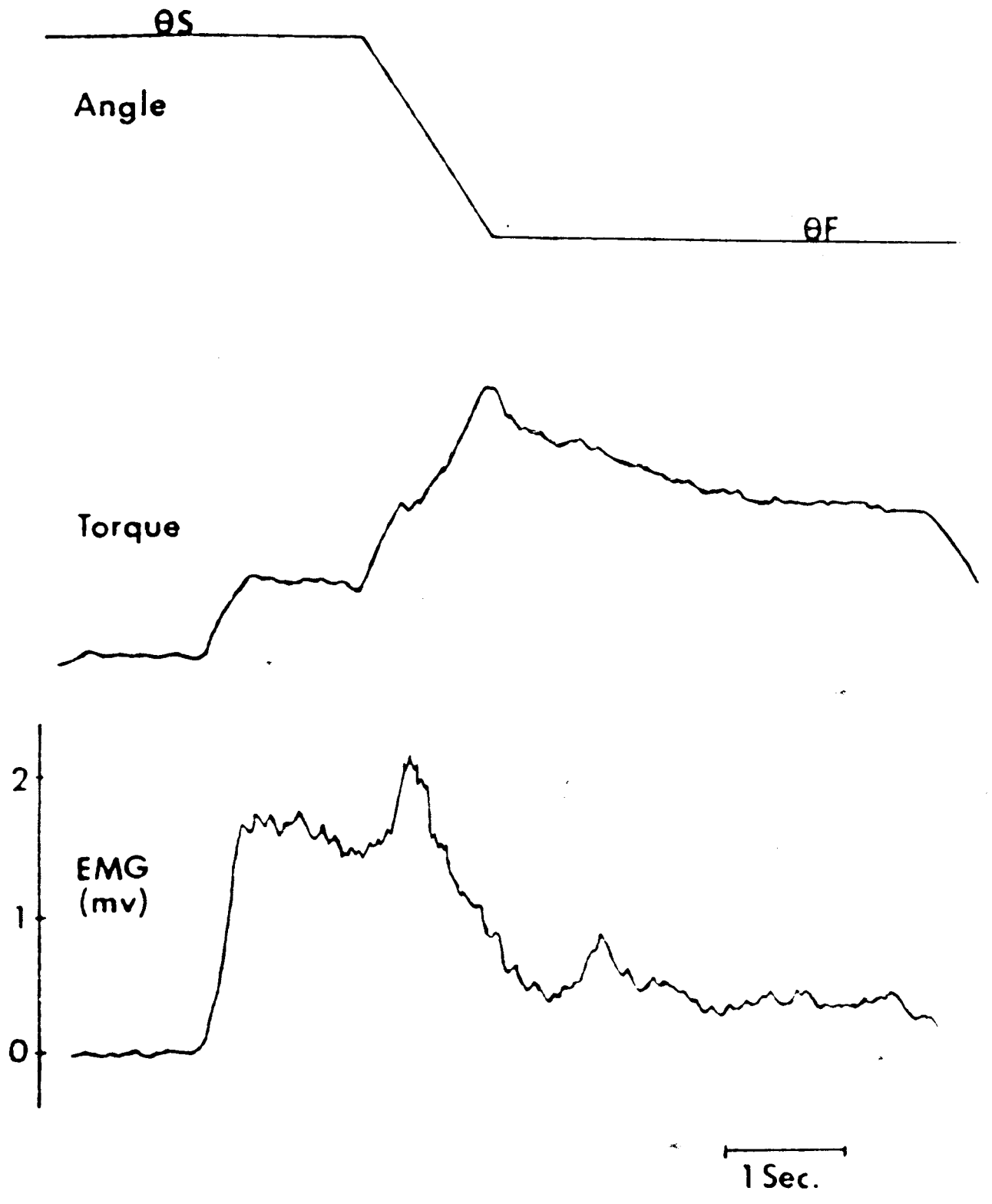

θS
Angle
θF
Torque
2
EMG
(mv)
1
0
1 Sec.

Figure 7: Sample record of angular displacement (top trace), torque response (middle trace) and rectified:averaged EMG (bottom trace) during and after medium velocity stretch from θS2 (170°) to θF2 (10°). This sort of response was typical of those produced by subjects BC and RB.


θS
Angle
θF
Torque
2
1
0
EMG
(mv)
1 Sec.

movement. Torque then recovered and continued to climb until the stretch was completed (Figure 7). This pattern was most evident in medium and fast stretches from 170°-10° or 170°-90°.

When submaximal preloads were used subjects AC, CT and AP also showed a similar drop in torque and then a secondary rise. As the isometric preloads were increased the height of both peaks increased and the trough between peaks became smaller. With maximal preloads the two peaks had more or less fused together (Figure 8).

On several occasions subject BC showed a different sort of response when medium and fast stretch velocities were used. In these trials torque rose sharply at first then suddenly levelled off. This plateau persisted until the movement stopped and then torque began to drop away (Figure 9).

P/Po

On successive experimental sessions fluctuations were observed in the isometric values (Po) as well as day to day variations in the peak eccentric values (P). Despite the fact that isometric torques prior to stretch were kept constant there was considerable intra-subject and inter-subject variability in the values of P/Po (peak eccentric/isometric torque) obtained for each stretch condition. An example of the intra-subject variability is given in Appendix D. In any event the effect which changes in muscle length, velocity and amplitude of stretch had on P/Po tended to follow the same pattern on each

Figure 8: Rectified:averaged EMG and the torque response during stretch with different isometric preloads. The preloads were determined through visual feedback from an oscilloscope and represent minimal effort (0%), half maximal effort (50%) and maximal voluntary effort (100%). Subject: AP.

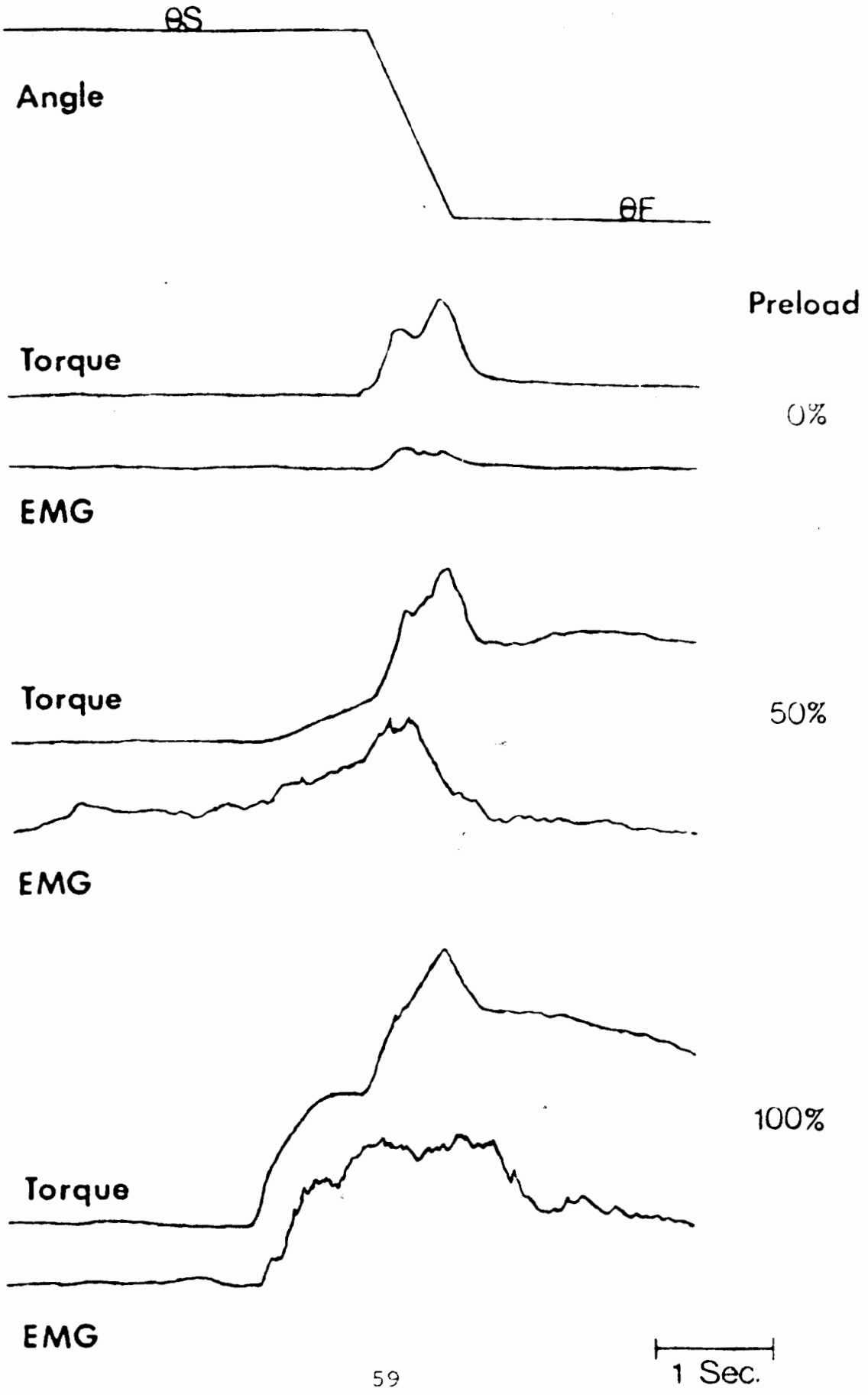
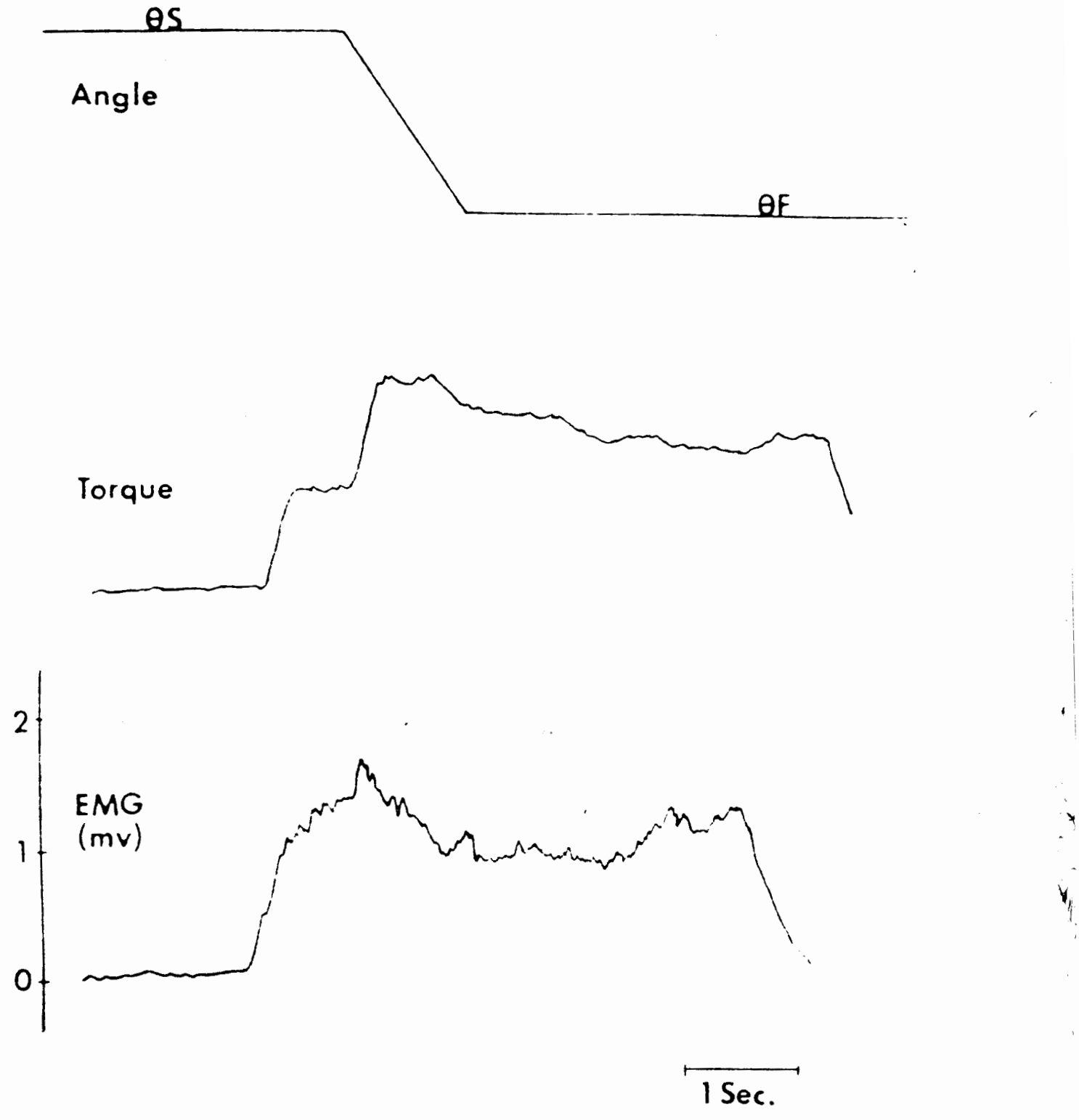

θS
Angle
θF
Preload
Torque
0%
EMG
Torque
50%
EMG
100%
Torque
EMG
1 Sec.

Figure 9: A sample record showing a plateau in torque part way through the stretch. This sort of response was most frequently observed in medium and fast stretch traces from subject BC.


θS
Angle
θF
Torque
2
EMG
(mv)
1
0
1 Sec.

test day. The mean values of P/Po for each subject are given in Appendix B.

When the mean P/Po values for the group were plotted against velocity for each experimental condition several trends were apparent (Table 2 and Figure 10). At each of the velocities tested the largest values for P/Po were generally obtained (20/25 trials) with stretches from 90°-10°. Stretches from 170°-90° produced intermediate values of P/Po and stretches from 170°-10° the smallest P/Po. When the shorter stretch amplitudes (90°-10° & 170°-90°) were used, P/Po increased with each increase in velocity. This was not true of the longer stretches (170°-10°). In these trials P/Po tended to be greatest when medium stretch velocities were used. Subject AC consistantly produced results which were different from those of the other subjects. At any given velocity his values of P/Po tended to be greatest for the 170°- 90° stretch condition.

A two way analysis of variance (BMDP2V) with velocity (slow, medium , fast) and $\theta S-\theta F$ (90°-10°, 170°-10°, 170°-90°) as the grouping variables yielded $F(3,36) = 6.72$ and $F(3,36) = 6.63$ respectively at $p<0.01$. Since there was no significant interaction between the two grouping variables the mean P/Po values for each velocity and $\theta S-\theta F$ could be examined independently. The results of Tukey's pairwise comparison of means at $p<0.05$ are given in Table 3. The increase in P/Po was found to be statistically significant when comparing the values for velocity 2 and 3 with that of velocity 1. The value of P/Po

Figure 10: Peak eccentric/isometric torques (P/Po) for the three stretch conditions. Each value is the mean (±SE) of 25 trials (5 subjects).

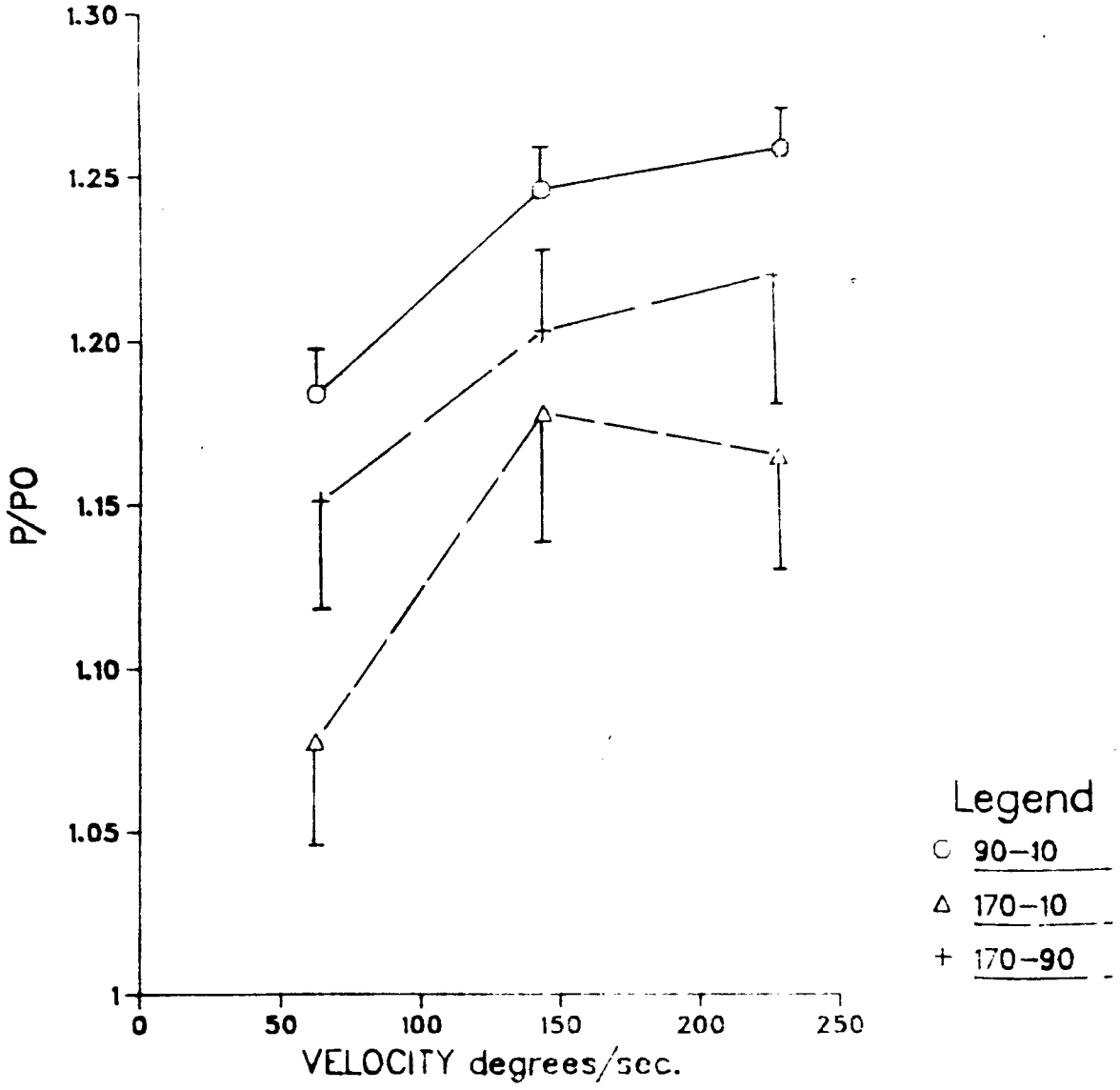

1.30
1.25
1.20
1.15
1.10
1.05
1
P/PO
0
50
100
150
200
250
VELOCITY degrees/sec.
Legend
90–10
170–10
170–90

Table 2: Peak eccentric/isometric torques (P/Po) at the same final position. Each value is the mean (±SD) of 25 trials (5 subjects).

	θS-θF		
	90°-10°	170°-10°	170°-90°
Vel 1.	1.109±.030	1.080±.079	1.166±.074
Vel 2.	1.250±.034	1.180±.060	1.200±.087
Vel 3.	1.260±.026	1.165±.070	1.223±.076

for velocity 3 was not however significantly different from that for velocity 2. A change in final muscle length (90°-10° vs. 170°-90°) failed to have a statistically significant effect on P/Po while an increase in stretch amplitude (170°-10° vs 90°-10°) produced significantly smaller values of P/Po.

Torque Changes After Stretch

As soon as the movement stopped torque began to drop. It fell sharply at first and then more slowly as it approached the isometric level for that finishing position. This decline continued and eventually fell below isometric for that muscle length. Isometric levels were generally reached less than 1.0 second after the stretch had been completed. While many of the traces had slight oscillations in torque during the hold phase, the general response can be described as an exponential decay. The shape of the torque records during the hold phase were similar to the tension changes after stretch reported by Sugi (1972) and Edman et al. (1978a; 1978b).

While the immediate drop in torque following stretch was greatest when the highest velocity and smaller amplitudes were used the time required for torque to drop below isometric was the longest under these conditions. With an increase in amplitude of the stretch or a reduction in velocity torque decayed more slowly when the movement stopped. Yet when the large amplitude (170°-10°) and slow velocity were used the excess torque was short-lived. The reason for this was that in

Table 3: Results of Tukey's pairwise comparison of group P/Po values with angular velocity and θS-θ F as the grouping variables (HSD= 0.066, $p<0.05$). Fractions in parentheses represent the number of subjects who showed significant differences in P/Po between stretch conditions. S and NS represent statistically significant and non-significant differences respectively.

$\theta S-\theta F$	Vel 1.	Vel 2.	Vel 3.	Mean
90-10	1.169	1.250	1.260	1.226
170-10	1.080	1.180	1.165	1.142
170-90	1.166	1.200	1.223	1.200
Mean	1.140	1.210	1.216	

S (5/5)

NS (2/5)

S (4/5)

NS (1/5)

S (4/5)

these trials torque started to decay before the end of the stretch and P/Po was not that large to begin with.

Final muscle length by itself (90°-10° vs. 170° -90°) had little effect on the rate at which torque decayed after stretch. There were several trials however when torque never dropped to isometric levels during the 4-5 second hold phase. This was most frequently observed in traces from subject AC with stretches from 170°-90°.

Decay Constants

The decay time constants ( ) for each subject are presented in Appendix C. While the shape of the decay phase seems to be dependent on the stretch parameters this dependence is not so evident when the decay in torque is expressed as a time constant. As with P/Po there was a large amount of intra-subject and inter-subject variability.

Most of the subjects showed a slight increase in decay time constants with increased velocity. At each velocity torque tended to decay most slowly following stretches from 170°-90°. With stretches from (170°-10°) the decay in torque was slightly faster and stretches from 90°-10° produced the most rapid decay.

The mean decay time constants for the group are presented numerically in Table 4 and graphically in Figure 11. Using velocity and θS-θF as the grouping variables a two way ANOVA failed to suggest any statistically significant trends at $p<0.05$.

Figure 11: Time constants (τ) for the decay in torque after stretch. Each value is the mean (±SE) of 25 trials (5 subjects).

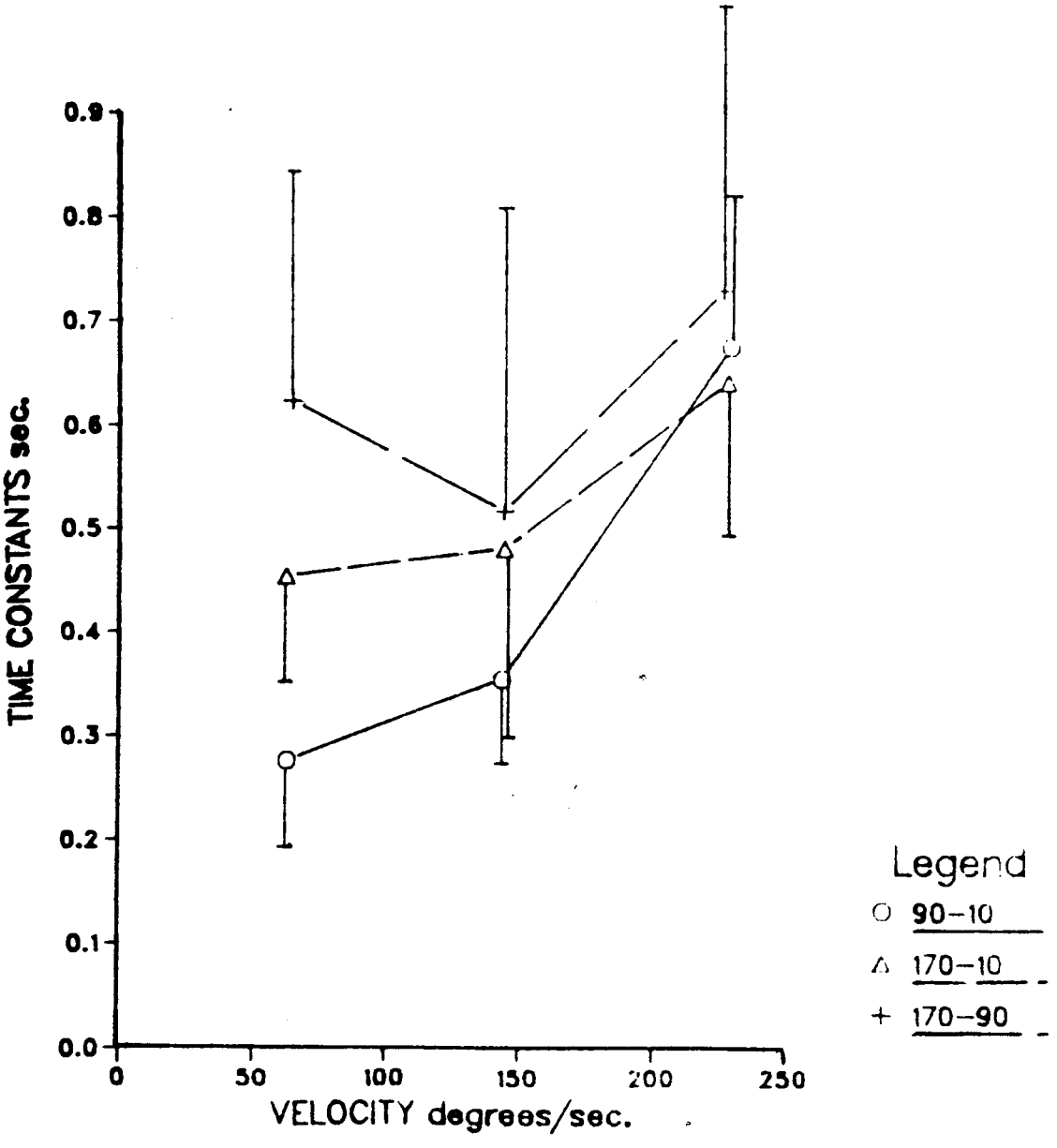

TIME CONSTANTS sec.
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
0
50
100
150
200
250
VELOCITY degrees/sec.
Legend
90–10
170–10
170–90

Table 4: Time constants for both the decay in torque after stretch and the rise to Po during isometric contractions at $\theta F = 10^{\circ}$ and 90°. Each value is the mean (±SD) of 25 trials (5 subjects).

	θS-θF		
	90°-10°	170°-10°	170°-90°
Vel 1.	0.277±.191	0.455±.179	0.625±.324
Vel 2.	0.355±.232	0.481±.418	0.518±.340
Vel 3.	0.641±.668	0.641±.668	0.729±.670

Isometric	10°	90°
	0.073±.019	0.070±.021

Time constants were also calculated for the rise in torque during isometric contractions at both θF= 90° and 10°. Assuming that lengthening and recoil of the SEC follows a similar time course, these time constants can also be used to describe the decay in stored elastic energy during the hold phase. These rate constants and decay constants at the same final position are presented in Table 4.

EMG

Subjects AC, AP and CT rarely showed a considerable increase in EMG during stretch (Figure 6). On the other hand RB and BC consistantly showed a definite reflex component in both their torque and EMG traces (Figure 7). A similar stretch response has been observed in the EMG from human wrist flexors (Bawa, 1981b).

With the onset of stretch EMG increased sharply reaching a peak of 1.5-2.0 mv after approximately 200-300 msec. The EMG then began to drop and continued to fall throughout the rest of the stretch. When the movement stopped EMG increased slightly and then stayed more or less the same for the remainder of the hold phase. Based on the results from these two subjects there was no obvious relationship between the magnitude of this EMG response and total muscle length, stretch velocity or amplitude. Since peak eccentric torques were generally recorded at the end of stretch, EMG levels at that time were compared with those from an isometric contraction at the same angle (Table 5). A two

way ANOVA using velocity as the grouping variable failed to indicate any statistically significant differences in mean EMG levels at $p<0.05$.

Table 5: Rectified and averaged EMG (mv) measured at the end of stretch and during an isometric contraction at $\theta F = 10°$ and 90°. Each value is the mean (±SD) of 25 trials (5 subjects).

	θS-θF		
	90°-10°	170°-10°	170°-90°
Vel 1.	0.92±.19	1.08±.13	0.90±.14
Vel 2.	1.14±.29	1.13±.17	0.91±.12
Vel 3.	1.12±.18	1.25±.38	0.97±.14

Isometric	10°	90°
	1.12±.10	0.92±.22

V. Discussion

When actively contracting single muscle fibres (Hill, 1977; Edman et al., 1976, 1978, 1981) or bundles of fibres (Sugi, 1972) are subjected to a ramp and hold displacement series the tension response is as follows. Tension rises during stretch reaching a peak when the movement stops. This excess tension then drops away eventually settling at the isometric level for the new muscle length. While the shape of the tension response is much the same in isolated whole muscle, the increase in tension during stretch tends to be smaller and the decay phase slower (van Atteveldt and Crowe, 1980; Flitney and Hirst, 1978).

The results from this study suggest that the response of human forearm supinator muscles to ramp and hold stretches is also similar to that of single fibres. Torque increases during stretch and then decays when the movement stops. Again there are some quantitative differences. The rise in torque during stretch is smaller and the rate of decay in torque much faster than with either single fibres or isolated whole muscle. It appears that these discrepancies may be the result of differences in the muscle preparation ie. the amount of series elasticity, temperature, fibre composition, etc.

The amount of series elasticity (SEC) is much less in single fibres, approximately 20% of that in isolated whole muscle (Flitney and Hirst, 1978, van Atteveldt and Crowe, 1980, Guth et al., 1979). During stretch these elastic elements will

be extended by an amount (x) and resist the movement with a force which is equal but opposite to the force generated by the contractile machinery, $F=k1(x)$ where k1= SEC stiffness. At the same time much of the negative work done in stretching the muscle will be stored in the SEC as elastic energy. After stretch the SEC will start to recoil as the tension generated by the contractile machinery gradually decreases. This will result in an internal lengthening of the contractile component (eccentric phase) which in turn slows down the decay process.

With whole muscle *in vitro* the total SEC stiffness is reduced to $(k1k2)/(k1+k2)$ where k2 represents the additional series elasticity of tendon and associated connective tissue. For a given amplitude of external displacement the SEC will be extended to longer lengths and the percentage change in contractile component length will be smaller. Since there is also a certain amount of elasticity within the cross-bridges the forces generated by the contractile component will be less. With this reduction in SEC stiffness the decay in excess tension after stretch will also be slower.

It is reasonable to assume that the amount of series elasticity in muscle *in situ* is even greater than isolated whole muscle. Because of the greater extensibility of the SEC and therefore the smaller length excursion of the contractile tissue one might expect the stretch response to be smaller and the decay phase even slower in muscle *in situ* than whole muscle *in vitro*. This was not the case with human forearm supinator

muscles. While P/Po did tend to be smaller the decay in torque was much more rapid.

Joyce et al. (1969) have examined the eccentric forces developed by cat soleus in situ using stimulus rates of 3, 7 and 35 pps. When peak eccentric tension at 35 pps is compared with isometric tension at the same ankle angle their values are similar to those obtained in the present study.

A stimulus rate of 35 pps might be considered to be quite high for cat soleus. However Zajac (1981) reports that during treadmill running the range of discharge rates in a population of extensor motor units was 15-55/s with a mean of 33/s. Similar firing rates (15-54/s) have been recorded by Severin et al. (1967).

Unlike animal studies where force transducers can be attached directly to the muscle, external strain gauges must be used to examine the stretch response of human skeletal muscle in situ. In the present study torques were measured at the handle as shown in Figure 2.

Travill and Basmajian (1961) have shown that when the elbow is flexed to 90° the biceps is the primary supinator during forceful supination. Under these conditions they also report little activity in antagonist muscles (pronators teres and quadratus). Torque is therefore primarily a function of biceps muscle length and the biceps muscle moment ie. the perpindicular distance from the axis of forearm rotation to the line of force action of the biceps.

Since the strain gauges were located on the handle the torque response to stretch will also be influenced by the mechanical properties of those visco-elastic tissues located in series between the insertion of the biceps on the radius and the handle ie. the interosseus membrane, ligaments and bones of the wrist and hand, soft tissues of the hand and to a certain extent the radius as well.

Several of the ligaments which help to stabilize the elbow joint are also stretched during forearm rotation. However these ligaments which act in parallel with the biceps tend to have the greatest effect on torque as the forearm approaches full supination or full pronation.

It must also be remembered that muscle studies *in vitro* are usually carried out at temperatures of 0°-4°C while muscle temperatures *in situ* may range from 35° to 39°C. With temperatures at or near 0° the rate of cross-bridge cycling is much slower and there is a substantial increase in muscle stiffness. With this increased resistance to lengthening the forces generated during stretch are increased and more mechanical energy is stored in the SEC. Increases of 30%-50% in peak eccentric tension have been reported when the temperature of single fibres is reduced from 20° to 0°C. (Sugi, 1972, Buchtal and Kaiser, 1951). Flitney and Hirst (1978) observed a similar increase in peak eccentric/isometric tension at the same muscle length with a decrease in temperature.

At these cooler temperatures the increase in muscle viscosity and reduction in the rate of cross-bridge cycling allows the SEC to recoil more slowly thereby prolonging the decay in excess tension after stretch.

Although muscle temperature was not monitored in the present study an attempt has since been made to measure biceps muscle temperature under similar experimental conditions. Unfortunately even a slight contraction of the biceps produced considerable pain in the region of the temperature probe. Therefore it can not be assumed that muscle temperature at rest, during prior isometric contractions and active stretch remained more or less the same.

These differences in the amount of series elasticity and temperature will not only influence the magnitude and duration of the stretch response but also how the muscle responds to changes in the stretch conditions ie. total muscle length, the amplitude of stretch and stretch velocity.

Flitney and Hirst (1978) report that in whole muscle <u>in vitro</u> excess tension increases with speed of stretch up to a critical velocity (Vc). Beyond this point velocity had little effect on peak eccentric tension.

At velocities greater than Vc strained actomyosin linkages are mechanically broken faster than they can reform. The contractile machinery is unable to resist the tension stored in the series elastic elements and the sarcomeres give. Tension is maintained at levels higher than isometric for the new muscle

length since the rate of cross-bridge reattachment is fast enough to prevent the SEC from recoiling completely (Joyce et al., 1969, Rack and Westbury, 1974, Flitney and Hirst, 1978).

In the present study there was a significant increase in P/Po going from slow to medium stretch velocities with all three experimental conditions ($\theta S-\theta F$= 90°-10°, 170°-10°, 170°-90 °). A further increase in velocity produced a small additional increase in P/Po with stretches from 90°-10° and 170°-90° while P/Po for stretches from 170° -10° decreased slightly. However these changes in P/Po were not found to be statistically significant. The fact that P/Po did not continue to increase with velocity suggests that cross-bridge slip may also be present in muscle in situ.

The shape of several of the torque traces from medium and fast stretch trials also suggests the presence of cross-bridge slip. In these trials torque rose sharply at the onset of stretch and then suddenly leveled off, remaining more or less constant for the rest of the movement (Figure 9). At the same time there is nothing in the EMG traces from these trials to indicate that this plateau in torque is the result of some form of reflex inhibition. While this sort of response was most frequently seen in traces from subjects BC, an abrupt though less dramatic decrease in slope was also shown by the other subjects. With stretch velocities exceeding Vc the response which Flitney and Hirst (1978) observed in isolated whole muscle is almost identical to that shown in Figure 9.

In isolated whole muscle the critical velocity (Vc) has a large positive temperature coeffecient ie. a 5-6 fold increase over a temperature range of 0°-30°C. It also tends to be greater in muscle with greater intrinsic speeds of shortening ie. a higher Vmax (Flitney and Hirst, 1978). For a given muscle Vmax will itself be a function of temperature. In the medial gastrocnemius of the cat for example Vmax is reduced by approximately 50% when muscle temperature is lowered from 38° to 28°C (Petrofsky and Phillips, 1980).

It should be noted that van Atteveldt and Crowe (1980) report that the slip effect occurs in isolated whole muscle at much slower stretch velocities than in single fibres at the same temperature. Again this can probably be attributed to differences in the amount of series elasticity since isolated whole muscle is often dissected out with the tendons intact. Still it may be argued that with muscle *in situ* temperatures of 36°±1°C, Vc is raised to a level where cross-bridge slip is unlikely under normal physiological conditions.

It is likely that the stretch velocities used in this study are vastly different from those used *in vitro*. With single fibres, isolated whole muscle or even cat soleus *in situ* stretch velocities can be measured directly and are generally expressed as %L/sec. or mm/sec. When studying human muscle *in situ* the change in muscle length per unit time must be calculated based on externally obtained measurements.

In the present study an attempt was made to equate a given angular velocity of the handle with the rate of change in biceps muscle length (Appendix A). When this conversion from °/sec to %L/sec is carried out it is apparent that the stretch velocities used in this study were considerably slower than those used *in vitro*. Still it is perhaps more meaningful to compare the range of stretch velocities used with the maximum speed of unloaded shortening since Vmax has been shown to be one of the determinents of cross-bridge slip.

At 0°C., Vmax for frog sartorius muscle is approximately 1.3 L/sec (Abbot and Richie, 1951). Flitney and Hirst (1978) examined the stretch response of frog sartorius (0°C.) at velocities up to 65 mm/sec (2.4 L/sec) and found that the ratio of peak eccentric/isometric tension at the same muscle length reached a maximum value at 3.8 mm/sec or .14 L/sec (Vc/Vmax= 0.108). With cat soleus *in situ* Joyce *et al.* (1969) used stretch velocities up to 40 mm/sec. While excess tension did continue to increase with velocity over this range it had more or less plateaued, reaching 95% of its maximum value at a stretch velocity of 5 mm/sec. Work by Petrofsky and Phillips (1980) indicates that the maximum velocity of unloaded shortening for cat soleus (38°C. and full activation) is about 60 mm/sec (Vc/Vmax= 0.083).

In the present study the eccentric angular velocities ranged from 63.1±.88°/sec to 227±1.6°/sec. During unloaded supination it is possible to reach angular velocities of

2300°/sec (Chapman, 1981). If one takes Vc to be the medium stretch velocity (144.5° ±.81°/sec.) then Vc/Vmax = 0.063. While this value is less than that for frog sartorius (*in vitro*) or cat soleus (*in situ*) all three values are of the same order of magnitude. It is also possible that Vc for forearm supinator muscles lies somewhere between the medium and fast stretch velocities. If Vc is set to 227°±1.6°/sec. then Vc/Vmax increases to 0.098.

Unlike most studies *in vitro* P/Po did not increase with stretch amplitude ie. 170°-10° versus 90°-10°. In fact an increase in amplitude had just the opposite effect (Figure 10). This may simply be due to the fact that the subject was unable to maintain a maximal contraction throughout the entire stretch when the amplitude was 160°. While effort is difficult to measure there was no drastic reduction in EMG to suggest that the subjects were not giving a maximal effort through out the stretch and hold phases.

Although the slip phenomenon is generally expressed as a function of stretch velocity (Vc) a certain amplitude must also be exceeded before cross-bridges will be mechanically broken and the sarcomeres give. This critical amplitude is thought to represent the working range of the myosin heads or short range stiffness (Rack and Westbury, 1974). Edman *et al.* (1978a) found that the critical amplitude required to reach a plateau in the force record during stretch was approximately 15 nm. Flitney and Hirst (1978) suggest that slip occurs when the actomyosin

filaments have been displaced by more than 11-12 nm.

With the elbow flexed to 90° the stretch amplitudes of 80° and 160° used in this study represent a change in muscle length relative to resting biceps length of approximately 4% and 8% respectively (Appendix A). These values are similar to the percentage length changes (2%-10%) used in most muscle studies *in vitro*.

With single fibres Hill (1977) suggests that each sarcomere is extended by the same amount during active stretch. However it seems unlikely that a given amount of handle rotation produced a uniform change in sarcomere length through out the supinator muscles. It is possible that with the smaller amplitude (80°) most of the cross-bridges remained intact since the supinator muscles had not been extended beyond their short range stiffness. With the larger stretch amplitude (160°) the short range stiffness accounts for the initial steep rise in torque. However during the stretch a greater proportion of the cross-bridges will be displaced by more than the critical amount ie. 12-15 nm. The result will be a reduction in peak eccentric torque (P). Since Po for 90°-10° and 170°-10° stretches are the same P/Po will also decrease.

The amount of excess tension present during stretch has been shown to be dependent on total muscle length (Hill, 1977; van Atteveldt and Crowe, 1980). This relationship is most apparent at lengths greater than Lo, the length at which maximum isometric tension is observed. Peak eccentric tension (P)

increases while isometric tension (Po) decreases over this range of muscle lengths ie. the descending portion of the force-length curve. The end result is a substantial increase in P/Po.

With forearm supination maximal torques are recorded in the fully pronated position. As the forearm is supinated torque drops away slowly at first. Once the handle has passed beyond approximately 120° (full supination = 170°) isometric torque starts to decrease much more rapidly. Up to 120° the decrease in torque can be attributed to changes in muscle length. Beyond 120° the muscle moment arm suddenly starts to decrease as well (Figure A1).

It is unlikely that the supinator muscles, biceps in particular were stretched to lengths greater than Lo. In going from a handle position of 10° to one of 90° the supinator muscles shorten and Po decreases moving to the left along the ascending limb of the force-length (torque-angle) relationship. However P will also increase with muscle length over this range. Although P and Po will be greater for stretches from 90°-10° than with stretches from 170°-90° the ratio P/Po may not necessarily be significantly different.

The fact that muscle lengths less than Lo were probably used in the present study also helps to explain why the stretch response of the forearm supinator muscles was considerably smaller and the decay back to isometric much faster than with muscle <u>in vitro</u>.

With single amphibian fibres (0°C) at sarcomere lengths less than Lo (2.4 um) Edman et al. (1978a, 1981) report that force enhancement after stretch decays quite rapidly, reaching isometric levels in about 1.0 second. The amount of tension present at a given time after stretch is independent of velocity and amplitude. They suggest that the decrease in excess tension represents the decay of force built up in visco-elastic tissues during stretch.

At lengths less than Lo parallel elastic elements do not generally have a great deal of influence on tension. It should therefore be possible to describe the decay phase using the stress relaxation function for a Maxwell body (spring and dashpot in series).

$$F(t)=A\exp|(B/K)t|$$

B=coeffecient of viscosity

K=spring constant

A=torque present at end of stretch

t=time

If the above equation accurately describes the decay in supinator torque during the hold phase then the decay time constant shouild be a function of muscle stiffness and viscosity (=B/K) but not the prior stretch conditions. In the present study changes in total muscle length, stretch velocity or amplitude of stretch did not have a statistically significant ($p<0.05$) effect on the rate of decay in torque after stretch. While the rate of decay may not change with the stretch

condition the absolute torque present at a given instant after stretch will still vary depending on the peak eccentric value.

Since the forces produced by a visco-elastic element are the same for lengthening and shortening the decay time constants at $\theta F=90^{\circ}$ and 10° should theoretically be the same as the time constant for the rise in isometric torque at those positions. The fact that the isometric rate constants were much faster than the decay constants for all stretch conditions (Table 5) indicates that the storage of mechanical energy in the SEC can not by itself account for the changes in torque after stretch.

It is possible that the reflex recruitment of additional motor units and/or increase in the rate of firing of previously active motor units may have influenced the stretch response of human muscle *in situ*. In decerebrate cats, the additional tension recruited by reflexes (net reflex contribution) is greatest with medium preloads and decreases towards zero when high opeating forces are used (Hoffer and Andreasson, 1981). With minimal preload (0%) or half maximal effort (50%) all of the subjects in this study showed a secondary, reflexly mediated rise in torque. When a maximal isometric contraction (100%) preceeded stretch this reflex component was no longer present in the traces from subjects CT, AC and AP (Figure 8). It was however still evident in the torque records from subjects BC and RB. Unfortunately with a filter time constant of 200 msec it is impossible to determine accurately the origin of the reflex response.

Both Grillner (1972) and Rack and Westbury (1974) feel that the initial response to stretch must be determined by the mechanical properties of muscle (short range stiffness) since even with spinal reflexes there will be a time delay before the reflex pathways can react. This has been confirmed by Hoffer and Andreasson (1981) who showed that for a given preload the initial rise in cat soleus tension is the same whether or not the soleus nerves had been cut.

In stretching the supinator muscles from θS to θF there was ample time for reflex pathways to react. During stretch there will be a decrease in EMG simply due to the change in muscle length. This can be shown by comparing the isometric EMG levels at various handle positions between 170° and 10°. Also Gottlieb et al. (1970) indicate that the excitability of the segmental reflex pathways may be momentarily reduced following a reflex burst. This rebound hyperpolarization lasts for 100-150 msec. At the same time many of the phasic motor neurons which were initially active suddenly stop firing. It is therefore possible for EMG to fall below isometric levels at the new muscle length (Ryall et al., 1972). Finally the stretch response may be reflexly inhibited by signals originating from the Golgi tendon organs (Houk and Henneman, 1967). In the present study the time taken for the small fast stretches to be completed was approximately 350 ms.

If reflex activity, regardless of its origin, is contributing to the enhancement of peak eccentric torques, one

might expect higher EMG levels at the end of stretch than with an isometric contraction at the same muscle length. These EMG values were not however found to be significantly different ($p<0.05$). This coupled with the fact that the EMG traces from three of the subjects (CT, AP and AC) rarely showed any sign of reflex activity suggests that with high initial operating forces (maximal voluntary contraction) it is the intrinsic mechanical properties of the supinator muscles and not reflex activity which is primarily responsible for the torque response during stretch. The above hypothesis is supported by Hoffer and Andreasson (1981) who have shown that in decerebrate cats the net reflex contribution during stretch is greatest with medium preloads and decreases quite dramatically when high preloads are used.

VI. Summary

Based on the results from this study it was found that excess torque , P/Po (peak eccentric/isometric torque), increases with stretch velocity until a certain critical velocity has been reached. It decreases with an increase in stretch amplitude and is not dependent on final muscle length. The rate of decay in torque after stretch was found to be independent of final muscle length, amplitude of stretch and stretch velocity. The effects of stretch velocity and amplitude on excess tension suggest that cross-bridge slip may be present in muscle *in situ* .

It would be wrong to suggest that these results can be applied directly to the contractile process at the molecular level. The change in torque during and after stretch is not simply the sum of the responses of fibres in a homogeneous population. There is no doubt considerable variability within a given muscle due to localized differences in activation and elasticity. The stretch response will also most certainly differ from muscle to muscle depending on fibre composition, orientation of the muscle relative to joints and the actions of agonistic/antagonistic muscle groups. Still these results may provide additional insight into the movement of intact joint systems and/or limb segments.

During many athletic activities, particularly those designed to produce a high terminal velocity, we take advantage

of prior stretch to improve performance. These tasks can generally be divided into two distinct alternating phases. Initially there is an eccentric countermovement or windup where the contracting muscle is stretched by antagonist groups, inertial forces and/or gravity. This is immediately followed by a concentric phase during which the activity is carried out using the previously stretched muscle.

This increase in work and/or power during the concentric phase has generally been attributed to the storage of mechanical energy in passive elastic elements within the muscle. While this interaction between series elastic and contractile components will undoubtedly enhance performance it can not by itself account for all of the increase in work/power.

With maximal voluntary activation it does not appear that reflexes play a major role in the torque response to stretch. While it is possible that the true EMG response was lost in processing the raw signal this additional enhancement seems to be the result of a temporary potentiation of the contractile machinery. Clearly with submaximal operating levels the relative contribution of each of the above mechanisms may be considerably different. With further research in this area it may be possible to develop optimal pre-stretch strategies for a variety of human activities.

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Appendix A: Biomechanics of Forearm Supination

As early as the mid 19th century, investigators were attaching metal rods and paint brushes to the radius and ulna of cadavers and recording the movements associated with pronation and supination. Development of more sophisticated techniques have simply made the kinematics more complex and confusing.

Ray et al. (1951) suggest that movement of the ulna during supination depends on both forearm position and the rotational axis chosen. In addition to this rotational motion Kapandji (1970) reports a certain amount of translational movement of the ulna at both the proximal and distal radioulnar joints. Little if any ulnar movement was observed by Morrey and Chao (1976) or Youm et al. (1979) with respect to the humerus while the distal end showed a combination of flexion:extension and abduction:adduction. These ulnar movements as well as the rotation of the radius appear to be impaired by abnormalities at either the radioulnar and/or humeroulnar joints (Ray et al., 1951).

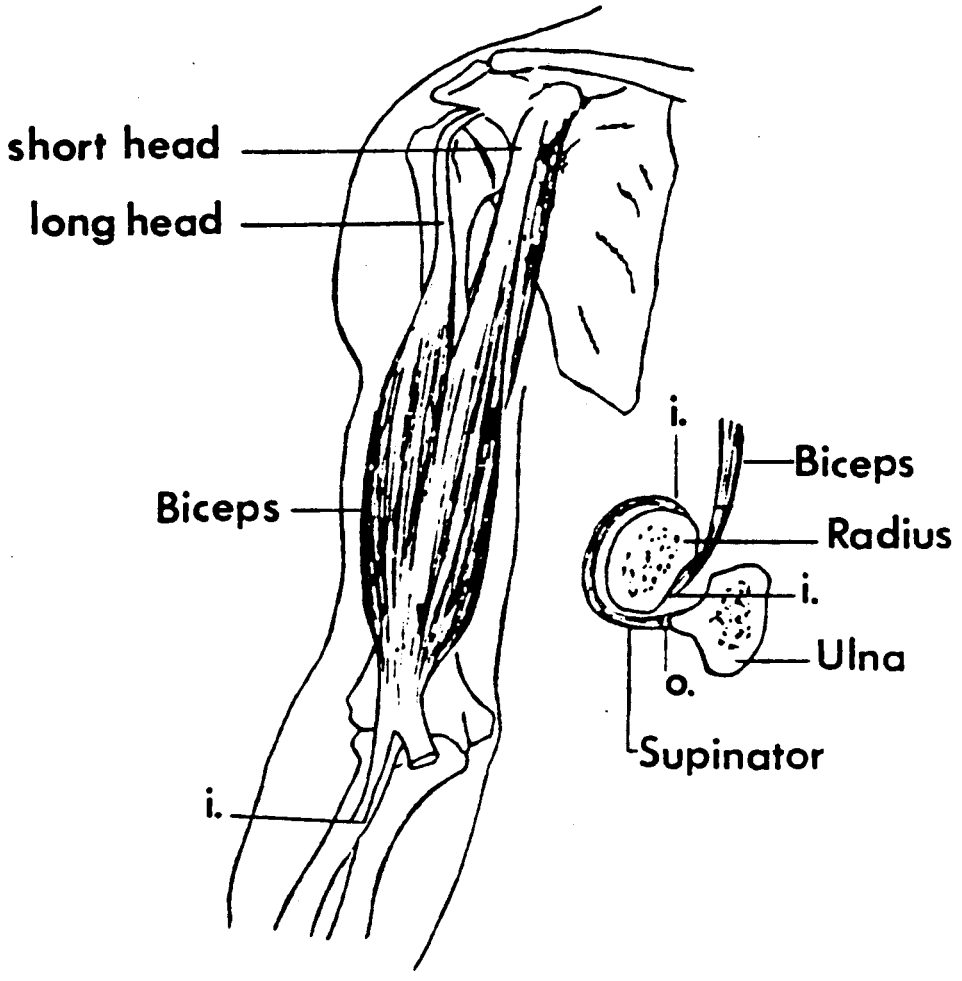
Youm et al. (1979) feel that forearm pronation:supination and flexion:extension of the elbow are independent motions which do not influence one another. Darcus and Salter (1953) present evidence from several studies which indicates that the amplitude of pronation:supination does indeed vary with both elbow and shoulder position. With the elbow flexed to 90° they report average values of 156.3° to 173.8° for the range of pronation:supination. Youm et al. (1979) give values of 70° from vertical for pronation and 85° for supination.

The maximum isometric torques generated during pronation:supination of the forearm also vary with shoulder, elbow and hand position (Darcus, 1951; Salter and Darcus, 1952). The reasons given for these differences include the degree of shortening and mechanical advantage of the prime movers, the extent of their contraction, the activation of synergistic and/or antagonistic muscle groups. With supination, the greatest torques were recorded when the shoulder was in the anatomical position, the elbow flexed to 90 degrees and the hand near full pronation.

Salter and Darcus (1952) report considerable variation in both amplitude and torque between successive recordings from the same subject. They feel this is due to slight changes in posture and/or handle grip. With a wrist-cuff arthrometer variations within a group were reduced by approximately 50%.

EMG studies conducted by Travill and Basmajian (1961) suggest that unresisted supination is initiated and generally maintained by the supinator muscle (figure A1). However during rapid movements with the elbow flexed or forceful supination regardless of elbow position, the biceps takes over as the principle supinator muscle. The above authors found little correlation between the various phases of supination and activity in the brachioradialis. Sometimes referred to as the supinator longus, it does not appear to be a true supinator. There was also little activity in antagonist groups, most notably pronator teres, during supination.

Figure A1: Origin, insertion and action of Biceps brachii and Supinator. (Redrawn from Crouch, 1976).


short head
long head
Biceps
i.
i.
Biceps
Radius
i.
Ulna
o.
Supinator

While it is impossible to partition out the actual changes in contractile component and SEC length an attempt was made to get an estimate of the overall changes in biceps muscle length during forearm rotation. Using isometric force-length data for biceps from Noble (1980) and simple skeletal measurements an increase in biceps muscle length of 0.016 cm per degree of pronation was calculated. Noble (1980) found the mean biceps length (short head) of 6 subjects to be 34.25 cm with the elbow flexed to 90°. Based on the above, stretch amplitudes of 80° and 160° produce a 4% and 8% change in biceps length respectively. The handle velocities used in the present study represent stretch velocities of 3%L/sec, 7%L/sec and 10%L/sec.

Appendix B: Average P/Po values for individual subjects.

	θS-θF		
	90°-10°	170°-10°	170°-90°
Subject:CT			
Vel 1.	1.209±.031	1.090±.039	1.188±.044
Vel 2.	1.249±.035	1.234±.027	1.205±.039
Vel 3.	1.264±.021	1.242±.065	1.233±.057
Subject:AP			
Vel 1.	1.197±.036	1.133±.098	1.175±.026
Vel 2.	1.288±.060	1.238±.114	1.225±.016
Vel 3.	1.290±.084	1.207±.115	1.186±.072
Subject:AC			
Vel 1.	1.185±.066	1.108±.063	1.232±.085
Vel 2.	1.269±.062	1.142±.075	1.186±.041
Vel 3.	1.268±.023	1.186±.041	1.349±.052
Subject:BC			
Vel 1.	1.198±.051	1.116±.049	1.122±.034
Vel 2.	1.219±.082	1.179±.016	1.158±.083
Vel 3.	1.237±.089	1.122±.044	1.214±.084
Subject:RB			
Vel 1.	1.132±.107	0.938±.082	1.038±.021
Vel 2.	1.206±.104	1.099±.092	1.085±.028
Vel 3.	1.233±.104	1.068±.079	1.137±.063

Appendix C: Time constants for individual subjects.

	θS-θF		
	90°-10°	170°-10°	170°-90°
Subject:RB			
Vel 1.	0.119±.038	0.538±.226	0.732±.244
Vel 2.	0.476±.103	1.182±.337	0.553±.247
Vel 3.	1.072±.297	0.986±.232	1.150±.310
Isometric	10°	90°	
	0.103±.018	0.101±.019	
Subject:CT			
Vel 1.	0.285±.055	0.648±.245	0.699±.253
Vel 2.	0.521±.141	0.610±.204	0.337±.127
Vel 3.	0.545±.356	0.579±.375	0.411±.2
Isometric	10°	90°	
	0.066±.004	0.071±.014	
Subject:BC			
Vel 1.	0.175±.158	0.242±.155	0.284±.265
Vel 2.	0.138±.019	0.140±.034	0.161±.062
Vel 3.	0.081±.029	0.166±.077	0.169±.085
Isometric	10°	90°	
	0.081±.010	0.086±.011	
Subject:AP			
Vel 1.	0.602±.154	0.312±.133	0.397±.176
Vel 2.	0.330±.144	0.430±.131	0.358±.215
Vel 3.	0.393±.105	0.200±.121	0.217±.142
Isometric	10°	90°	
	0.063±.016	0.051±.011	

	θS-θF		
	90°-10°	170°-10°	170°-90°
Subject: AC			
Vel 1.	0.205±.123	0.128±.058	1.470±.342
Vel 2.	0.705±.120	0.183±.065	1.811±.320
Vel 3.	0.852±.173	0.684±.194	1.700±.289
Isometric	10°	90°	
	0.054±.010	0.053±.013	

Appendix D: Data for repeated trials. Subject: BC.

	$\theta S-\theta F$		
	90°-10°	170°-10°	170°-90°
Vel 1.	1.220	1.108	1.105
	1.262	1.070	1.140
	1.152	1.108	1.148
	1.217	1.200	1.070
	1.139	1.095	1.149
Vel 2.	1.200	1.189	1.220
	1.349	1.162	1.077
	1.243	1.200	1.061
	1.149	1.167	1.188
	1.155	1.175	1.242
Vel 3.	1.260	1.283	1.315
	1.273	1.221	1.210
	1.262	1.315	1.158
	1.292	1.146	1.234
	1.233	1.244	1.248