EFFECTS OF VELOCITY ON WORK PRODUCTION ABOUT THE HUMAN ELBOW JOINT DURING STRETCH-SHORTENING AND NON-STRETCH-SHORTENING TASKS

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DANIEL BENOIT

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AUTHOR: Daniel Leonard Benoit, B.Sc. (L'université d'Ottawa)

SUPERVISOR: Dr. James J. Dowling

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Abstract

The performance enhancement of stretch shortening cycle (SSC) contractions has been well documented in the literature. However, the majority of these studies have been performed either on gross human systems for multi-jointed movements, or in isolated animal muscle studies using in-vitro preparations. This study was designed to apply the principles used for these in-vitro animal studies to the human system, under conditions that would allow results to be directly associated with a specific muscle or muscle group. Previous investigations by Lynch (1992) and Benoit and Dowling (1995) have supported the use of muscle models to predict elbow flexor torque and SSC performance enhancement. The purpose of this study was to use an EMG based muscle model to investigate the possible relationship between SSC tasks at different frequencies of elbow flexion-extension and performance enhancement of the elbow flexor muscles.

A Hill based muscle model was used to predict elbow flexor torque of seven healthy male subjects (23-40 years of age) under voluntary and stimulated contraction conditions. EMG of the elbow flexors and extensors was recorded from the biceps brachii and triceps respectively. Elbow flexor stimulation was done transcutaneously with a voltage equivalent to a 60% MVC torque; stimulation lasted four seconds at a frequency of 50 Hz. A simulated constant muscle activation torque was also derived from the muscle model for all trials. Externally measured torque was measured using a strain gauge located on a

shaft situated along the axis of rotation of the elbow joint. A torque motor was used to drive the forearm (fastened to a manipulandum) at four frequencies of elbow flexion-extension (.58, 1.5, 2.4, and 3.3 Hz) over a range of 162 to 105 degrees of elbow extension. Non-SSC trials were performed at these same velocities and over the same range of motion. Torque was then integrated as a function of joint angle displacement to yield the work produced about the elbow. Passive work was subtracted from all trials.

The results indicate that a significant increase in muscle work followed SSC tasks as opposed to non-SSC tasks and this increased work was relatively highest at 2.4 Hz. Work about the elbow decreased with increasing frequency of movement for both SSC and non-SSC conditions. The simulated constant activation muscle model predicted work well for all trials and conditions, indicating muscle model accuracy. The EMG driven model predicted well for all non-SSC trials but significantly underestimated the work for SSC tasks, suggesting a decrease in myoelectric activity. This decrease was evidenced by a decrease in average M-wave amplitude with increasing SSC velocity. This study indicates that the contractile component is directly involved in optimizing muscle work during SSC tasks and that the performance enhancement of SSC tasks may take place at the myofilament and cross-bridge level.

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CHAPTER I

1.0 Introduction and Review of Literature:

The storage of energy within muscle has been under investigation for some time. The early work of Marey and Demeny in 1885 investigated its impact on vertical jumping and indicated that a tensed muscle that had been stretched by a vertical jump added a "very high elastic force" when a second jump immediately followed (cited in Cavagna, 1977). The actual mechanism that causes the force enhancement is not clearly defined, however, its influence on force generation has been widely reported (Hill, 1953; Cavagna, 1965; Asmussen and Bonde-Peterson, 1974a,b; Cavagna, 1977; Bosco et al., 1982; Komi, 1984a,b; Hudson and Owen, 1985; Ettema et al., 1990a,b, 1992; Kyröläinen et al., 1990; Cook and McDonagh, 1995). This increased performance benefit has since been associated with muscle contractions which take place during a stretch-shortening cycle. A stretch shortening cycle (SSC) is defined as an active stretch (eccentric contraction) of a muscle followed by an immediate shortening (concentric contraction) of that same muscle. This occurs in many activities of daily living and sports such as walking and throwing. Numerous studies have been performed using muscle specimens and isolated muscles stimulated supramaximally under differing contraction conditions in order to quantify this phenomenon (Rack and Westbury, 1974a,b; Ettema and Huijing, 1989; Ettema, 1996a). While there are many investigations performed in-situ, only a limited number of studies have been performed

on humans to investigate this phenomena (Joyce et al., 1974; Bach et al., 1983). At present, there is some debate as to where and how this performance enhancement takes place. It has been postulated that elastic structures in series with the contractile component can store energy like a spring after being forcibly stretched (Alexander, 1987). Since the length of the tendon increases due to the active stretch phase, if the series elastic component acts as a spring, it would therefore be storing more potential energy. This energy would be released as the spring shortened. Thus, the recoil of the tendon during the shortening phase of the movement would result in a more efficient movement than one in which no energy had been stored (Hof and van den Berg, 1986). However, other studies have found that removing portions of these series-elastic components (by way of tendon length reduction) had little effect on muscle performance (Baratta and Solomonow, 1991). This work is further supported by recent work on turkeys by Roberts et al. (1997). Since the tendons of turkey gastrocnemius are almost entirely calcified, they are unable to store large amounts of elastic energy. Studies on turkeys have, nevertheless, shown that during SSC, a performance enhancement still takes place but it is thought that the aponeurosis (or internal tendon) could be a major source of energy storage (Roleveld et al., 1994). The contractile component itself has been associated with the ability to increase contractile performance through muscle potentiation (Cavagna, 1977) while other studies have found that this ability is quite limited and unable to account for such enhancements (Lensel and Goubel, 1987, Lensel-Corbeil and Goubel, 1990; Ettema and Huijing, 1989). The

results of these often contradictory studies have been associated with improved efficiencies for human or animal movements such as counter-movement jumps and running (Asmussen and Bonde-Peterson, 1974b; Cavagna, 1977).

In the human system movement about a single joint is accomplished only through the combined efforts of many muscles, both agonist and antagonist, that must function together to control limb displacement. It is therefore unrealistic to assume that results of such isolated contraction situations would be representative of muscle contraction under physiological conditions. As such, it is logical to perform related studies in-vivo on human subjects to get a clearer picture of how skeletal muscle regulates a potential performance enhancement following a SSC contraction in the human system. To accomplish this task, it is necessary to limit the observations and conclusions to joints about which relatively few muscles act and about which predictable movement and muscle activity patterns will occur. Furthermore, the muscles to be observed must be accessible so that information regarding their function and activation may be recorded (Wilkie, 1950). This study will make these observations and investigate how the human system reacts to SSC tasks. This study has been designed so that the results may be compared with some of the more isolated animal experiments. In order to discuss the contractile component's role in energy storage, a brief description of its elements will be provided, as well as the active state of muscle. The following sections will review the basic anatomy of muscle contraction, current concepts and results of enhanced performance following SSC

effects of muscle mechanics on movement outcome, as well as muscle modelling approaches necessary to relate the results of this study to the human system.

1.1 Anatomy and physiology of muscle contraction:

Excitation-contraction coupling refers to all the events occurring between the propagation of an action potential by a motoneuron into the sarcolemma and the actual mechanical response of the contractile machinery. The action potential, moving along the sarcolemma, spreads inwards via the transverse tubules. This brings the action potential into close proximity with the fibrils over the areas of the A and I band junctions. This electrical activity in the transverse tubules causes the release of calcium ions from the terminal cisterna of the sarcoplasmic reticulum. The increased calcium concentration establishes the conditions necessary for the interaction of the protein filaments actin (thin filament) and myosin (thick filament). The muscle is then considered to be in an active state and the outcome of the actin and myosin interaction is a bound site between these two proteins (called actomyosin) and is referred to as a bound cross-bridge (Poland et al.,1977). The formation of actomyosin increases the myosin filament's ability to hydrolyze adenosine triphosphate (ATP), an energy providing molecule located in the sarcoplasm. ATP is hydrolyzed and the result is adenosine diphosphate (ADP) + inorganic phosphate + energy. This reaction either causes the myosin cross-bridge to alter its angle of pull or for the globular myosin head to rotate (both interpretations are accepted, although the latter is a more modern

interpretation), resulting in the "power stroke" (action responsible for filament sliding) that allows actin to slide past the myosin attachment site. (Van de Graph and Fox, 1995; McComas, 1996) The summation of "power strokes" from all active muscle fibres is responsible for muscle contraction and produces an externally measurable force.

1.2 Enhancement of Muscle Force Production:

This section is devoted to describing current concepts of energy storage and utilization during SSC contractions. It will be shown that many different theories have been proposed and the results of studies in the area are not always conclusive or easily adaptable to the human system.

1.2.1 Muscle Potentiation:

Cavagna (1977) has proposed that energy can be stored in the actomyosin bond of an attached cross-bridge. He applies this concept in his description of muscle potentiation. Cavagna concluded that stretching a contracted muscle had two effects:

1) an increase in the efficiency with which positive work is done during a subsequent shortening contraction derived from the passive recoil of elastic structures and not the active transformation of chemical energy,

2) previous stretching reduces the time in which positive work must be done therefore the muscle is actively contracting for a shorter period of time, the net result being an increase in muscle power (this results from a higher acceleration which leads to a shorter time to peak velocity).

It was proposed that by forcibly moving the bonded myosin head it could be placed in a position which maintains greater potential energy. This could be described as an enhancement of the contractile machinery's ability to produce work. This "attached cross-bridge" state would be time dependent such that the stress of this new position could not be indefinitely maintained. Therefore increased time between pre-stretch and the release of pre-stretch (i.e.: muscle shortening) would cause a decrease in the amount of potential energy (due to cross-bridge detachment) that could be released as kinetic energy. This approach has been used to describe the apparent increases in muscular efficiency in running as compared to walking (Asmussen and Bonde-Peterson, 1974b; Cavagna, 1977). This theory has not been without its critics, however. Lensel and Goubel (1987) stated that the usefulness of the "Cavagna effect" was questionable because of the amount of cross-bridge pre-stretch necessary for the potentiation effect to be mechanically significant.

1.2.2 Storage of Elastic Energy:

A great deal of work has been done to explain the potential storage of elastic energy in the passive structures of the muscle tendon unit. Many of the studies in this area have involved isolated in-situ experiments performed on rat gastrocnemius and cat soleus muscles (Rack and Westbury, 1974a,b; Ettema and Huijing, 1989; Ettema et al., 1990 a,b; Huijing, 1992; Ettema, 1996a,b) as well as frog sartorius (Lensel-Corbeil and Goubel, 1990) and other animal experiments (Roberts et al., 1997). On this subject, Huijing (1992) stated that:

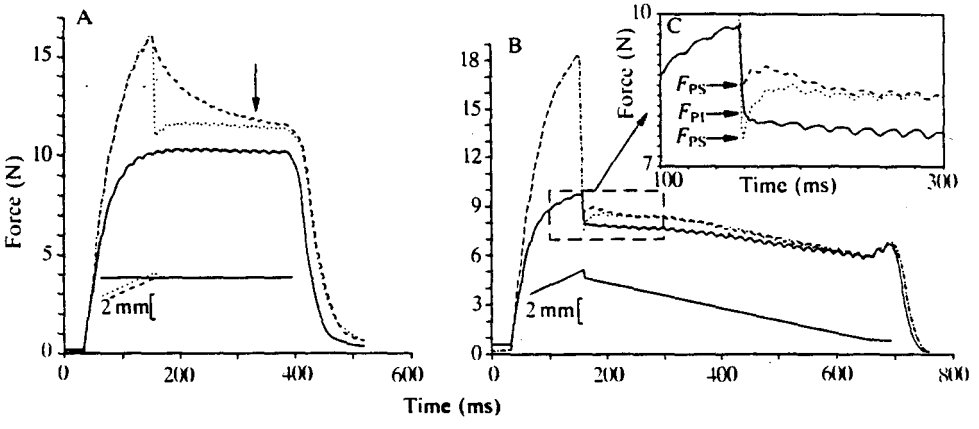
> "an important effect of elastic elements is that muscle fibres can store energy in the tendon at a relatively low rate, allowing the occurrence of high levels of force because of force-velocity characteristics. Subsequently, the tendon acts as an energy pool, which can be applied to obtain high velocities of movement and thus high power output without imposing these high velocities on the muscle fibres".

This "energy pool" could, therefore, be responsible for the increased efficiency following an active pre-stretch. Ettema and Huijing (1989), Ettema et al. (1990a,b; 1992) proposed that contractile element work plays a minor role in work enhancement and that all of the differences could be explained by series elastic component enhancement. To investigate this, Ettema et al. (1992) used a methodology that ensured that the onset of stretch preceded the onset of muscle stimulation, in contrast to the experiments by Cavagna that used a previously active muscle. It was shown that although 90% of the work done during the shortening phase of the movement was a result of the contractile element, the actual increase in work for the stretch shortening cycle (over that of a pre-activated isometric muscle released at an equivalent muscle length) was mostly (80%) a result of elastic energy release (see figure 1). Another observation of the study was that the total work done during a stretch shortening cycle increased with pre-stretch amplitude and was actually greater than for the isometric quick release trial (muscle is active but not pre-stretched before the movement begins) at all but the shortest pre-stretch situations, possibly due to short-range stiffness. This

increase in work was associated with the need for cross-bridge cycling with increased range of movement and seems in agreement with the previous work of Rack and Westbury (1974a).

Figure 1: Force-time figures(Ettema et al., 1992):

Figure 1A: Effect of quick-release after a pre-stretch on muscle force. Solid line, isometric control; dotted line, pre-stretch with a quick release; dashed line, pre-stretch without a quick release.
Figure 1B: Effect of pre-stretch amplitude on quick-release and isometric force levels with a pre-stretch velocity of $10mmS^{-1}$. Solid line, pre-isometric; dashed and dotted lines, pre-stretch.
Figure 1C: Enlargement of the outlined part of 1B. Fps and Fpi indicate the force level at the onset of the pre-stretch for pre-stretch and pre-isometric contractions respectively.



Other studies have been performed in order to relate muscle-tendon unit stiffness with SSC force enhancement (Hill, 1950; Rack and Westbury, 1974a,b; Cnockaert et al., 1978; Niku and Henderson, 1989; Amis et al., 1987; Bach et al., 1983). The concept of short range stiffness and its effects on muscle mechanics was investigated by Rack and Westbury (1974a,b) using tetanized cat soleus and

gastrocnemius muscles. They observed that over very small changes in muscle length, muscle tension rose steeply during the initial muscle lengthening phase. As the movement amplitude increased, the stiffness (measured as the slope of a length-tension graph) tended to decrease so that the change in tension became more gradual. As a result, when muscle shortening was confined to a short distance little work was expended in driving the muscle and the muscle exhibited fundamentally elastic characteristics of energy storage and return (length-tension refers to muscle length and the externally measured tension the muscle is providing, such as force or torque, depending on the experiment). This short range stiffness showed change with varying levels of muscle tension: as muscle tension increased, so did the stiffness. The explanation given by Rack and Westbury (1974a) for this phenomenon was that muscle stiffness was due to attached cross-bridges in the sarcomere and that the tension would regulate the number of cross-bridge attachments formed. As the amplitude of the movement increased, the cross-bridges would be broken and have to cyclically reform, thus lowering the overall stiffness of the myofibril. The effects of short range stiffness were associated with a 35 to 50% increase in force per 1% extension of the cat soleus muscle, over a physiological range of 3-4%.

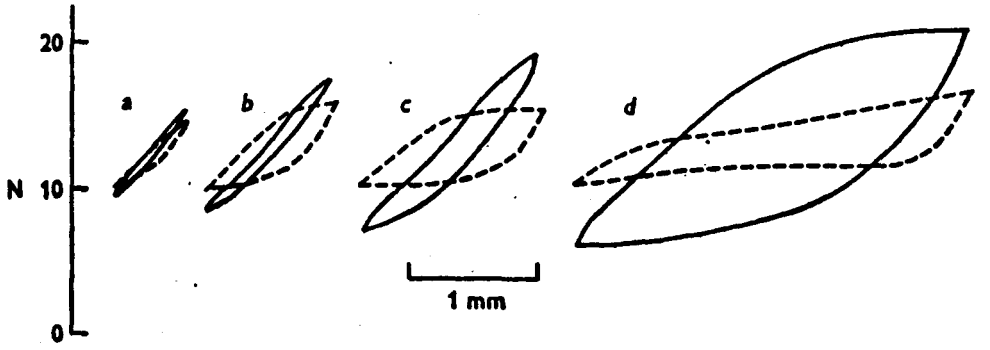
Since the short range stiffness seemed to demonstrate spring like properties, it was hypothesized that if the muscle was to shorten over this short stiff range, the net work absorbed by the muscle would theoretically be zero. It was also proposed that the amount of work required to drive the muscle through a complete cycle would indicate

the muscle's ability to absorb and dissipate mechanical work. The effectiveness of the muscle was therefore evaluated by measuring the work required by the muscle to produce the movement. This was accomplished by taking the area within the length-tension graph. It was shown that as the amplitude of movement increased (increasing muscle length), so too did the amount of work absorbed by the muscle. However, over very small changes in length, Rack and Westbury found that the work absorbed was minimal. The ability of the muscle to return energy to the system was attributed to the elastic-like characteristics of the short-range stiffness of skeletal muscle. The short range stiffness demonstrated by Rack and Westbury (1974a) has been seen in both isolated muscle (Lensel-Corbeil and Goubel , 1990) and in-situ muscle studies (Hufschmidt and Schwaller, 1987). The length-tension, or "work loop", graphs were also used to investigate the effects of contraction velocity on muscle effectiveness. The results of these experiments can be seen in figures 2A and 2B and describe a velocity effect for increased torque during muscle lengthening which produces greater muscle work at higher SSC contraction velocities. Figure 2A describes the work produced when the change from lengthening to shortening is immediate, as opposed to figure 2B where the movement is sinusoidal in nature. The work increase in figure 2A associated with increased movement velocity is exponential in nature whereas the sinusoidal movement describes an optimum work production frequency of 4.5 Hz before work begins to decrease. The reasons for the differences between the two experiments are not well defined, however, it is clear that during the sinusoidal movement, maximum

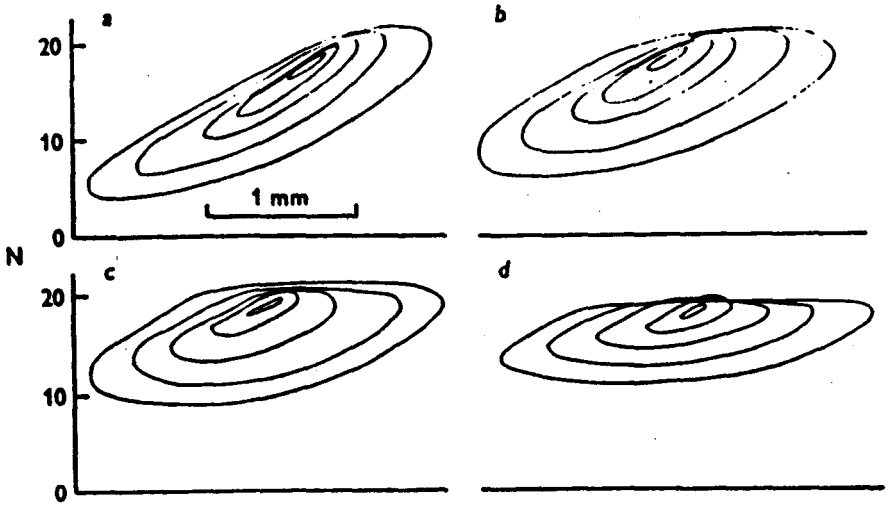
eccentric torque is maintained for a shorter period of time as velocity increased, thus decreasing the amount of work produced.

Figure 2: Work Loops (taken from Rack and Westbury, 1974a)

2A: Length-tension figures of cat soleus through four different amplitudes at two different velocities during constant velocity changes in length. Continuous line, 20mmS^{-1}; interrupted line, 5 mmS^{-1}; a, b, c, d, describe different muscle lengths.



2B:Length-tension figures of cat soleus through four different amplitudes at four different velocities during sinusoidal changes in length. a, 11 Hz; b, 4.5 Hz; c,2.1 Hz; d, 0.9 Hz



Another means of evaluating the elastic characteristics of the muscle-tendon unit is by investigating the resonant frequency of a given joint in the body (Berthoz and Metral, 1970; Joyce, Rack and Ross, 1974; Bach, Chapman and Calvert, 1983). In these studies, movement is evaluated about a given joint. Frequencies of movement, or oscillations, are monitored while force data are recorded as the structures surrounding the joint resist the movement. This resistance is caused by the limb's inertia, passive muscle-tendon structures, the possible activation of an antagonist muscle via the stretch reflex, and a visco-elastic resistance caused by the musculo-tendinous unit. As the frequency of movement approaches the optimum frequency, less force will be required to maintain the movement. Berthoz and Metral (1970) found resonant frequencies for the human elbow joint between 3 and 4.5 Hz while Joyce et al. (1974) reported frequencies for the same joint of about 5 Hz (the differences were associated with varying degrees of muscle tension at the joint and different experimental setups). In these studies, movement was limited to small flexion-extension oscillations of up to 8.3 degrees. In the latter study, the elastic stiffness of the elbow flexors was calculated by subtracting the calculated (negative) force required to move the mass (of the forearm and apparatus) from the measured in-phase force. This stiffness was found to be 2.6 Nmm^{-1} measured at the wrist. Assuming that this stiffness value is due to the innate elastic properties of the elbow flexors (and not reflex activity), comparisons were made with the results found by Rack and Westbury (1974b) for the short range stiffness of cat soleus. The equivalent increase in force production associated with the elbow

flexor stiffness was 17% compared to the 25-35% increase in force found due to the short range stiffness of the cat soleus. The lower increase in force was associated with the lack of reflex activity for the human study and a relatively low force production at the elbow during the stiffness measurements, assuming that short range stiffness increases with greater force production.

1.2.3 Muscle Mechanics:

This section will introduce concepts of muscle mechanics which are widely accepted and well documented. The interaction of these known mechanical properties with SSC conditions will be discussed and their potential ability to account for performance enhancement will be introduced.

1.2.3.1 Force-length Relationship:

For a given amount of muscle activation, muscles produce different levels of force at different muscle lengths (Wilkie, 1950; 1953). A fundamental active length-tension curve was derived from these isolated tissue studies, however, attempts to reproduce this relationship in the human system were not as successful (Wilkie, 1950). Gordon et al. (1966) has shown that the amount of force that an isolated muscle is able to produce is dependent on the number of myosin cross-bridges that are able to bind with actin. For in-vivo preparations, the relationship between torque and joint angle will be dependent upon many factors inherent in the human system. These factors may include the fact that the mechanical advantage of the muscle moment arm length changes with changing joint angles. There may also be more than one muscle crossing

the joint being investigated that is contributing to the measured force. Tendon series elasticity will also affect the force output for different tendon lengths and voluntary muscle activation will not be as constant or controllable as a stimulated contraction. Based on a Hill-type muscle model, any increase in muscle-tendon unit length is a sum of the contractile element and series elastic component length changes (Hof and van den Berg, 1986). During a stretch shortening cycle, the contractile component is actively producing force against the tendon. As a result, a stretch shortening cycle may provide a shorter contractile element length due to a pre-stretch of the series elastic component at the beginning of the shortening phase of the movement (Hof and van den Berg, 1986, Ettema et al., 1990a,b). The outcome of this process of pre-stretch could allow the muscle to contract over a more favorable range of the force length curve and result in greater force production (Edman et al. 1978).

1.2.3.2 Force-velocity Relationship:

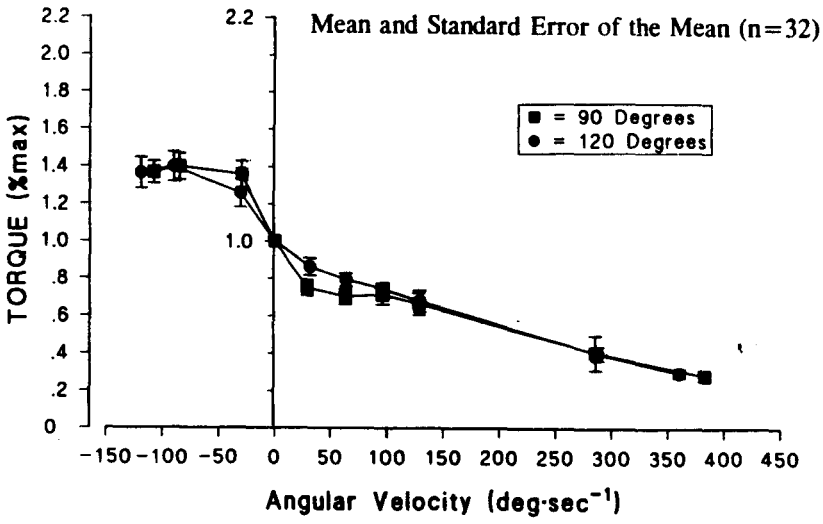
Muscle tissue is also known to be "activation time" dependent as well as shortening velocity dependent. As the velocity of muscle shortening increases, the amount of force produced by the muscle decreases following a hyperbolic curve described by the Hill equation (Kojima, 1991). This force-velocity relation was defined by the following function:

$$(F + a)(V + b) = b(Fo + a) \qquad (1)$$

Where:
F = the muscular force
V = the shortening velocity of the contractile component
Fo = the maximum isometric force
a and b = constants ($0.1 < a < 0.5$ and $0.4 < b < 1.6$)

During eccentric contractions, however, muscle force has been shown to be higher than the total force produced during an isometric contraction. This increase in force during eccentric contractions has not been as thoroughly investigated as the concentric force curve and the literature describes varying results ranging from 1.2 to 1.6 times isometric force levels for the elbow flexors (Jorgensen, 1976; Dowling, 1997). This force has been shown to increase following a hyperbolic function before reaching a plateau at its maximum eccentric torque level (Faulkner et al., 1980). Dowling et al.(1995) analyzed the data of 32 young male and female adult subjects and derived a torque-angular velocity graph which can be seen in figure 3. Applying the principles of muscle mechanics, Dowling (1992) used computer simulation to assess their influence on SSC force production. He showed that the effects of muscle mechanics could be altered to reproduce enhancement of mechanical outputs during stretch-shortening tasks without the added benefits of reflex activity or storage of elastic energy. It was found that by taking advantage of the non-linear force-time, force-length and force-velocity relationships, higher final movement velocities could be achieved following an active pre-stretch.

Figure 3: Elbow Flexor Torque-Angular Velocity Curve (Dowling et al.,1995): Average data of 32 male and female subjects, taken at two joint angle (90 and 120 degrees of elbow extension)



1.2.4 Other Explanations of Force Enhancement:

The tendon has been described as an energy storing spring, but it has also been likened to a rope which goes slack when no force is exerted upon it (van Ingen Schenau, 1984, 1986). The benefit of a SSC contraction has been associated with the active stretch of the muscle taking up the "slack" in the muscle-tendon unit prior to the shortening phase of contraction. This so called "in-waste" theory proposes that the amount of energy which can be stored in the muscle-tendon unit is too small to account for the associated performance enhancement and, as such, an alternative view was described. The increased muscle force following an active stretch is associated with the increased potentiation of an eccentric contraction which, since the cross-bridges are already bound, allows for a shorter rise-time in concentric force production (less time spent for cross-bridge binding) and a contraction from a higher force level. This

theory was refuted by Hof and van den Berg (1986) as they proposed that van Ingen Schenau's estimates of elastic energy storage did not take into account the potential for energy storage within both the internal and external elastic muscle structures.

1.3 Muscle Models and Modelling principles:

The concept of muscle modelling is to combine known phenomenological results of muscle contractions with the theoretical principles of how muscle contractions take place. The fact is that the actual process of muscle contraction is still unclear (Alter, 1996; McComas, 1996; Pollack, 1996). This has led to models that both support and contradict modern theories of muscle contraction. Certain models employ a neural network approach which uses a "black box" to predict muscle force output (Liu and Herzog, 1996). These models have their strength in the mathematical prediction of the contraction properties, but often fail to account for the physical and chemical processes which must take place in order to transform chemical energy to mechanical energy.

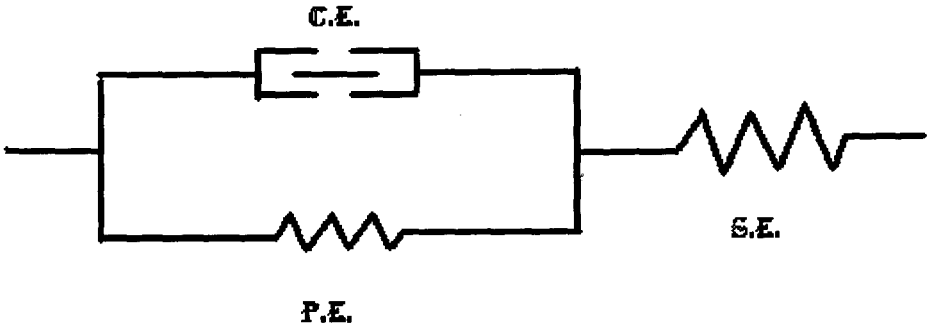
Zahalak (1990) pointed out two major guidelines that must be taken into account when creating a muscle model. First, he proposed that the model should be sufficiently simple so that studies involving large amounts of data can be analytically and computationally manageable. This stems from the fact that the muscle model, though it may be an excellent predictor of muscle output, could require so much optimization and computation that it becomes too time intensive to apply over a wide range of experimental conditions. As such, these models are difficult to verify and are not "user friendly". His second consideration is that structures being mathematically represented

should embody as closely as possible the known chemical and physiological character of the modeled muscles, so that theoretical predictions are credible. This would allow present theories of muscle contraction to be verified by models that produce acceptable mechanical outputs. This also ensures that the elements used in the muscle model are realistic computations and not simply a means to a well predicted end. Keeping these considerations in mind, certain muscle models will now be presented.

1.3.1 Phenomenological Models:

In 1938, A.V. Hill proposed a phenomenological model that contained a contractile component surrounded both in series and in parallel by passive connective tissues. He then formally described the muscle-tendon unit as a lightly damped elastic element in series with a contractile element (Hill, 1938, Winters, 1990). It was proposed that the parallel component corresponded to the passive connective tissue around, and within, the contractile element while the series elastic component was a representation of the tendon complex attached to the muscle (figure 4) .

Figure 4: Classical structures for the Hill muscle model, with contractile element (CE), parallel element (PE) and series element (SE):



Although the model itself is a gross oversimplification of a complex system, it does seem to produce consistent reproductions of the mechanical force output of the muscle tendon complex under many experimental conditions. Furthermore, the model operates within the confines of the sliding filament theory. In addition, the model was not merely a mechanical representation of a mass-spring-damper system: Hill's original research was, in fact, based on heat and energy fluctuations of the contracting muscle. The classic Hill equation was derived from the relationship between changes in muscle temperature as a function of muscle length and time. For these reasons, Hill-based models have been the most popular basis for model building (Winters, 1990; Cole et al., 1996). With the basic concept of the Hill model, one has a building block which can lead to sophisticated muscle models that can predict force output of a muscle-tendon complex under various experimental conditions. The contractile element is characterized by the force-length and force-velocity relationships described earlier that govern the behavior of a Hill-based model. This portion of the muscle can be

described as the actual motor that produces the tension that will eventually be transmitted to the adjoining connective tissue and finally to the bones that make up the joint. This simplification of the musculo-tendinous unit has been widely used in muscle models to predict muscle force.

1.3.2 Biophysical Models:

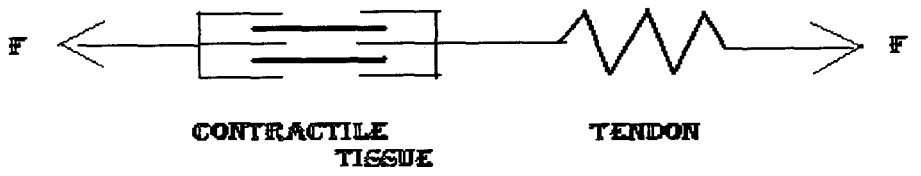
Shortly after the introduction of the Hill model, Huxley (1957) introduced a biophysical cross-bridge model which represented a more realistic and detailed interpretation of cross-bridge mechanics. In this model, the cross-bridges were considered to be either attached to an actin site or detached from actin. In the attached state, the cross-bridges are assumed to be bound by an elastic linkage which is in random thermal motion about a neutral equilibrium position. As such, the cross-bridges are considered to act independently of each other. These actomyosin links may cause a force in either direction of pull. Therefore, the sum of the forces produced by the bound cross-bridges is the cause of muscle force production. Regulation of force is based on the displacement of a cross-bridge from its neutral (resting) equilibrium position to a position of higher potential energy. The model portrays each cross-bridge as a ball attached by a spring to a myosin backbone. The ball is the globular head which tends to attach to actin sites. The globular head is assumed to have a higher probability of binding for a situation in which the spring is allowed to shorten before attaching to an actin site. The mathematical functions describing force production are based on a bond distribution function that incorporates cross-bridge displacement and

velocity, as well as chemical kinetic descriptors that are representative of ATP hydrolysis. Although the original model was able to predict the broad features of muscle behavior known at the time (force-velocity and heat production during shortening contractions) it has since been elaborated upon to include different cross-bridge states. This has improved the model's ability to represent different types of contractions and recent developments in cross-bridge theories, but it has also made it impractical and labor intensive to use (Zahalak, 1990). Although Huxley's model was much more descriptive of cross-bridge mechanics than the original Hill model, scientists interested in the basic principles of myofilamentary interaction still consider it to be too rudimentary to explain cross-bridge theory. As a result, more rigorous models have been developed, such as Hatze's "charge transfer model", that are based on inter-molecular force production. However, this model requires extensive calibration and is unlikely to be representative of a contracting muscle (Hatze, 1990).

1.3.3 Hybrid Models:

The "distributed moment" model was developed by Zahalak (1981) as a compromise between the highly complex biophysical models and the phenomenological Hill-type models.(see figure 5)

Figure 5: Structures of the Zahalak muscle model (displays the contractile component attached in series with the series elastic structures):



This model is based on the myosin-actin-troponin complex (contractile tissue) which is regulated by calcium concentrations in the sarcomere (Zahalak, 1990). The model is mathematically formulated in terms of three state variables representing the cross-bridge distribution function: a state proportional to the stiffness of the contractile element, a state proportional to force, and a state which is proportional to the elastic energy in the cross-bridges. However, recent studies have shown that the "distributed moment" model is not rigorous enough to describe muscle function under different velocities of shortening using one set of parameters (Cole et al., 1996). The parameters must be modified in order to maintain good predictions for varying shortening situations. In fact, when dealing with biophysical muscle models, van den Bogert et al. (1996) went so far as to conclude that "present theories of cross-bridge dynamics and muscle architecture are not a suitable basis for the development for force production.".

1.3.4 EMG Driven Model:

The chemical processes which lead to muscle contraction produce an electrical current in the muscle that can be registered externally through the use of an electromyogram (EMG). The EMG signal is said to be a representation of the muscle's activity level (Basmajian, 1985). As such, it has been used in muscle modelling to predict the force output generated by the active muscle (Hof, 1984, van Ruijven and Weijs, 1990; Dowling, 1997). Certain assumptions must be made in order for EMG of a dynamic movement trial to be used as a determinant of muscle force:

1) the recorded EMG is a suitable representation of muscle activation

2) the recorded joint angles are an adequate method of representing muscle length

3) the muscle model used allows for an adequate representation of the force produced by the active muscle groups.

The prediction of work using EMG for muscles crossing the elbow joint requires a number of processing procedures to take place and because of this, the use of EMG for dynamic force calibration is in itself an area of research that is far from perfected. However, it has been shown that EMG can be related to force by manipulating the processed EMG gain in order to create a "signal match" with the resultant force output signal (Hof, 1984). This technique is based on the linear relationship displayed between isometric muscle force and mean rectified EMG:

$$F_{system} = (Emg * g) = F_{muscle} \qquad (2)$$

where: F_{system} = the resultant external force output signal

Emg = the recorded electromyography signal (in mV) of the muscle responsible for the movement (rectified and optimally smoothed)
g = gain value derived from optimization (NmV^{-1})
F*muscle* = the equivalent muscle force as a function of activation

However, a non-linear relationship has been seen in several studies and a modified version of equation (2) was used by Lynch (1992) to predict isometric force output of the elbow flexors:

$$F_{muscle} = g * EMG^{n} \qquad (3)$$

where: n = exponent factor determined by optimization (varies between .2 and .9 for stimulated trials)

In dynamic movement trials, the EMG alone does not provide sufficient information to represent muscle force. In these situations, the time course of the muscle length should also be known and a force-length relationship included in the force calculations (Hof, 1984). The concept is, therefore, to choose a procedure that reliably predicts muscle force in an isometric situation. With these gain modifications, a muscle model must then be chosen from previous studies or developed for the specific study to be performed. A model with these factors will allow the mechanical characteristics of the observed joints and muscles to modify the collected EMG signal in a way that will accurately depict muscle force output for the contraction protocol. Force-velocity and force-length relationships must be devised that contribute to the non-linearities of the dynamic muscle force to EMG relationship. This model, however,

will not take into account the effects of muscle fatigue or any increase in muscle force caused by SSC performance enhancement. As such, any experiments performed must ensure that muscle fatigue does not occur. If this is accomplished and if the muscle model is assumed to represent muscle force during a non-SSC task, then the deviation of the predicted force from the actual measured force could be attributed to the storage of elastic energy. Lynch (1992) used an EMG driven Hill-type model to assess the influence of elastic recovery during a stretch-shortening task. The model used predicted elbow flexor torque output very well in non-stretch shortening cycle tasks (average error of -0.83 +/- 2.00 Nm over eight subjects, or less than 27% RMS error). It was hypothesized that any deterioration of model accuracy from a non-SSC task to a SSC task would be due to elements unaccounted for by the muscle model. A protocol was designed to assess muscle function under three conditions: concentric action from rest, concentric action from an active state (quick release) and a concentric contraction immediately following an eccentric contraction (SSC). Lynch's model was able to predict elbow joint angular impulse under all conditions but the SSC condition, which showed an increase in performance not accounted for by the model. The increased impulse was attributed to a storage of elastic energy in the muscle-tendon unit, as this was considered the only factor which the model and protocol did not account for.

1.5 Relating In-Vivo Results and Isolated Muscle Performance:

In order to study the mechanical characteristics of human muscle in-vivo, certain criteria must be met that will allow the conclusions rendered to be related to the muscles investigated. Wilkie (1950) proposed the following guidelines:

1) the joint to be analyzed must be geometrically simple,

2) the movement to be analyzed must involve relatively few muscles ,

3) the movement does not disturb the rigid fixation of the rest of the body,

4) the movement is accurately reproducible.

According to Wilkie (1950), the movement of elbow flexion satisfies these conditions. Wilkie's observations have been substantiated by the numerous studies performed on the elbow joint for various muscle modelling and in-situ muscle preparation studies (Joyce, Rack and Ross, 1974; Zahalak et al., 1976; Cnockaert et al., 1978; Nygaard et al., 1983; Chapman and Caldwell, 1985; Fellows and Rack, 1987; Bouisset, 1987; Niku and Henderson, 1989; Dowling, 1992). In the present study, observations relating muscle activity (internal responses) and mechanical (external) responses have been investigated. Therefore, access to the muscles responsible for these responses was critical. Analysis of the elbow joint flexors and extensors meets this requirement. The biceps brachii is the primary elbow flexor (van de Graff and Fox, 1995) and is also the most superficial of the important elbow flexors (Nygaard et al, 1983). Therefore, muscle activity can be recorded from the muscle using surface EMG electrodes (Basmajian, 1985). Studies have also shown that biceps brachii muscle activity is

highly correlated with elbow flexor muscle activity when the forearm is kept supinated (Crowninshield, 1978), and can be used to represent the muscle activity of the elbow flexor muscle group (Bouisset, 1987). Based on these assumptions, the results that will be discussed in this study will be representative of all the primary muscles used in flexion of the elbow joint. These muscles are the biceps brachii, brachioradialis, and brachialis, which account for more than 80% of the potential flexor moment, as well as the pronator teres and extensor carpi radialis longus (Dowling, 1987). Another advantage of using the elbow joint when investigating issues related to storage of elastic energy is that the primary flexor (biceps brachii) is a fusiform muscle whose fibers are oriented along the axis of the humerus and which has relatively short tendons (van de Graph and Fox, 1995) which minimize the effects of pennation angle during muscle shortening. Furthermore, the elbow joint can be considered a pure hinge joint with one degree of freedom about the center of the joint that allows movement only in flexion and extension (Dowling, 1987).

1.6 Purpose and Rationale:

The purpose of this paper is to use EMG modelling techniques to determine the role of the contractile component based on the possible relationship between stretch-shortening cycle tasks, increased frequency of movement and changes in work produced by the flexor muscles of the human forearm. The first hypothesis is that the muscle will be more effective during stretch-shortening tasks due to an increase of stored energy in the muscle-tendon complex, resulting in increased torque production.

It is also hypothesised that as the frequency of movement about the elbow joint approached the optimum frequency of the elbow joint, a greater amount of negative work done on the segment would be stored in elastic structures and returned to the arm as positive work because of series elastic element and contractile component interaction. This work would not be accounted for by the EMG driven model but would be present when dealing with externally recorded work values. It was therefore hypothesised that as the frequency of movement increased, the amount of muscle activity (derived from the recorded EMG) required to produce the measured work would decrease due to increased stored elastic energy. The protocol for data collection and analysis was devised based on previous studies investigating the elbow joint (Lynch, 1992; Benoit and Dowling, 1995; Dowling et al., 1995) and previous isolated animal studies (Rack and Westbury, 1974a,b; Ettema et al., 1990a,b, 1992). It was designed to allow conclusions to be made based on the relevance of using data from stimulated muscle protocols and in-vitro animal studies to explain physiological situations in humans.

CHAPTER II

2.0 Methods and procedures:

A total of seven healthy males, 23-40 years of age, took part in this study after giving informed consent. Care was taken to ensure that all subjects were from a relatively homogenous group. This ensured that the apparatus used in the study was well suited to all subjects and that the 60% MVC values would be relatively equal and allow subjects to be grouped together.

2.1 Methods:

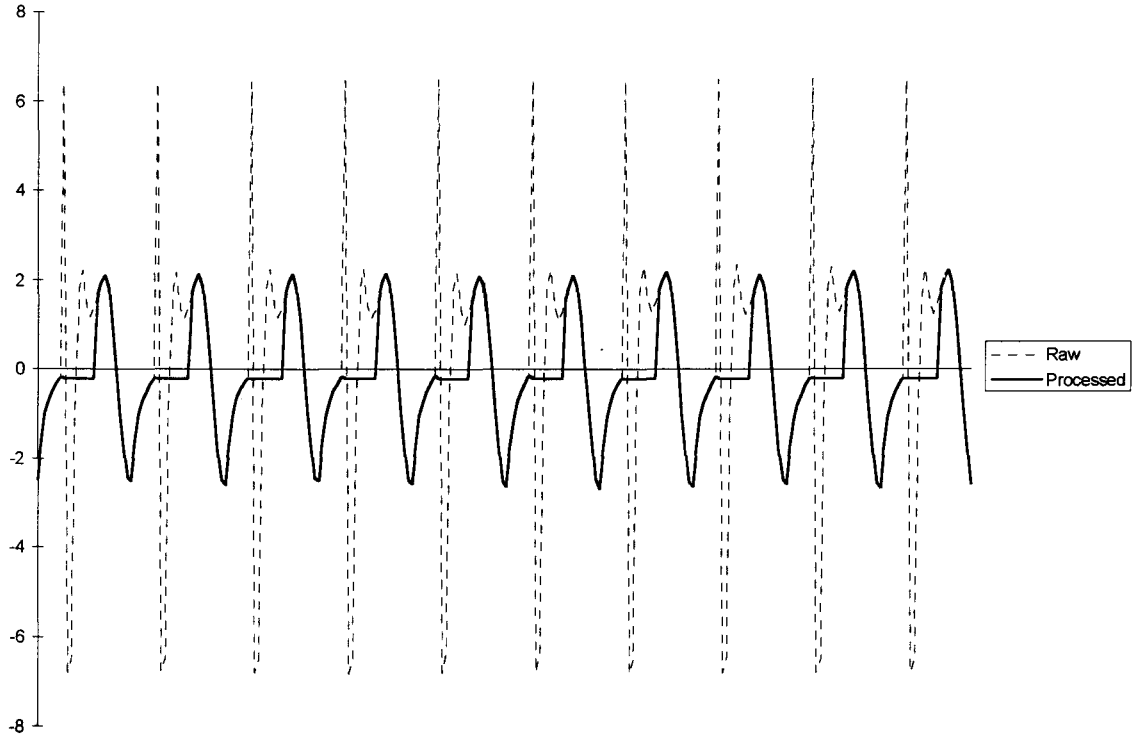
2.1.1 Electromyography:

In order to collect acceptable EMG data to meet the assumptions reviewed in the introduction, a number of guidelines were to be followed. The electrodes were designed and placed so as to collect sufficient information from the desired muscle group while minimizing voltage readings from neighboring muscles (crosstalk). The most important factor was not necessarily eliminating the unwanted noise, but increasing the signal to noise ratio when the EMG signal was used in a processed form (Hof, 1984). Bipolar surface electrodes were used to collect EMG from the biceps brachii and triceps brachii muscles which are easily palpable beneath the skin. This type of electrode provided an acceptable compromise between ease of use and data collection quality. Due to the very small voltage being read at the skin surface (up to 5 mV for a maximum contraction using surface electrodes), cable sway can add unwanted noise to the signal. This was reduced by pre-amplification of the at the electrode level.

The signal was then externally amplified, full-wave rectified and low-passed filtered. This processing allowed the EMG signal to be used on a comparative basis with recorded isometric force (Hof , 1984). In this study, EMG signals were recorded from the elbow flexors and extensors and digitally converted at a frequency of 1250 Hz using high impedance dry bipolar surface electrodes (designed by C. J. Deluca). Skin preparation involved removing dead skin and debris with an emery pad, followed by a thorough cleansing with an alcohol swab. The electrodes had a constant separation of 1 cm, and were preamplified at the electrode level with a differential gain of 1000. The frequency bandwidth was 5.5 to 450 Hz with an input impedance of $10^{12}\ \Omega$ and a common mode rejection ratio (CMRR) of >80dB. The elbow extensor electrode was located over the lateral head of the triceps brachii. The flexor electrode was located over the biceps brachii and its specific location was determined by verifying the M-wave amplitude and clarity during stimulated twitch protocols. The electrode was, however, located over the muscle belly for all subjects. The digitally converted EMG signal was saved in CODAS and then converted to ASCII format and saved on computer disk. EMG signal bias was removed by calculating the mean of the digitized EMG during a quiet portion of the signal. The EMG was then full-wave rectified and low-pass filtered (using a single pass critically damped filter) with a cutoff of 3.5 Hz for the voluntary activation trial and 3 Hz for the stimulated muscle trials. These cutoff frequencies were chosen by optimization using a root mean squares method, comparing the shape of the recorded signal with the shape of the corresponding torque

output during an isometric trial. The signals recorded from the stimulated muscle activation trials contained not only the M-wave produced by the contractile component, but also the stimulation artifact which caused the activation. This artifact was removed by analyzing the frequency with which the artifact occurred and the magnitude of its voltage. A computer algorithm was written that reduced the unwanted signal to zero while passing over the important M-wave. All processed M-wave signals were verified to ensure that only the unwanted noise was removed and the software was modified to accommodate each subject. Figure 6 shows the recorded signal and the modified signal of a representative subject.

Figure 6: Isometric Elbow Flexor EMG: Raw signal and artifact suppressed (processed) signal of a representative subject under constant voltage 50Hz stimulation at 105 degrees of elbow extension for 0.2 seconds:



2.1.2 Muscle stimulation:

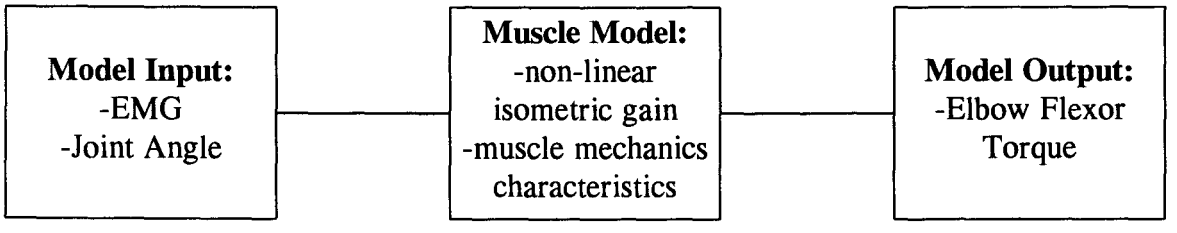
To investigate the storage and utilization of elastic energy at a constant level of muscle activation, contraction levels must remain constant for the entire trial duration. In order to achieve this, submaximal tetanic muscle stimulation was used to activate the elbow flexors. Transcutaneous muscle stimulation using two lead surface stimulation electrodes was used to achieve constant activation (Merletti et al., 1992) The anode was rectangular, measured approximately 5 by 3 cm, and was placed over the motor point of the biceps brachii. The motor point was considered to be located where the stimulating electrode elicited the highest torque value for a stimulated muscle twitch at

a given submaximal voltage. The cathode was also rectangular, measuring approximately 7 by 3 cm. This was placed 4 cm below the axis of the elbow joint on the anterior aspect of the forearm. This location was chosen so as to include as much elbow flexor muscle activity as possible. A stimulation frequency of 50 Hz was used as 50 Hz provided a tetanic stimulation while minimising subject pain (Merletti et al., 1992). The stimulation lasted for 4 seconds, allowing numerous SSCs to take place while minimising the effects of fatigue. A pre-test session was used to determine the maximal amount of stimulation to be used for each subject and to familiarise subjects with the stimulation protocol. Subjects that could not comfortably tolerate a stimulation voltage that produced a torque level equivalent to 60% of their maximal voluntary isometric contraction were excluded from the study. This pre-test session was also used to ensure that the subjects were comfortable with the experimental apparatus and informed consent was received from all subjects to continue with the testing sessions. The levels of stimulation were associated with the amount of torque output so that on subsequent testing sessions, the voltage could be adjusted to match the torque values previously determined. Since the levels of stimulation used provided such high torque outputs (60% MVC), it was concluded that the stimulation electrodes were activating the elbow flexor group, and not simply the biceps brachii (Crowninshield, 1978). For this reason, the M-waves recorded from the stimulation trials were considered to be representative of the elbow flexor muscle group.

2.1.3 Muscle model:

The model used in this study was a Hill-based EMG model which included a torque-velocity relationship, a torque-joint angle relationship, and a passive component which included the effects of gravity, joint viscosity, passive joint stiffness and possibly stretch reflex activity (see figure 7). The elbow flexor EMG-torque gain was determined by a computer algorithm using equation 3 (p.23). The gain factor and non-linear exponent were determined by optimisation using the parameter values which yielded the smallest RMS value between the actual torque and the predicted torque for isometric calibration trials. This non-linear function transferred mV to Nm. For all trials, visual inspection of the triceps EMG signal during data collection revealed insignificant muscular activity. Trials which were suspect were repeated at the time of data collection.

Figure 7: Schematic of muscle model used in this study:

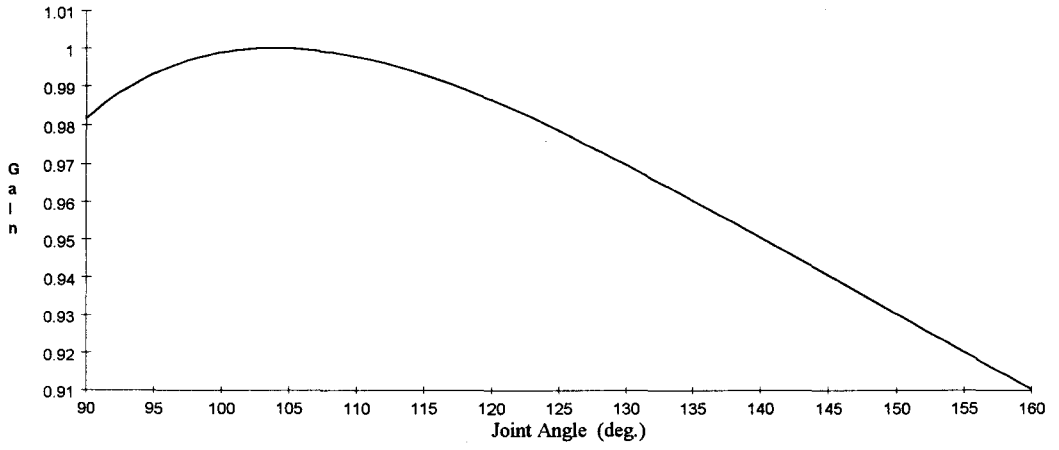


In this study, the muscle model deals with joint angle displacement, as opposed to muscle lengths and moment arms. This approach takes all contributing factors into account and relates the mechanical parameters of the elbow flexors into an easily measured kinematic variable. The strength curve for the elbow flexor has been shown to be quadratic in nature (Jorgensen, 1976; Kulig, Andrews and Hay, 1984) and this has also found by Dowling et al. (1995) when using the same apparatus as the present study. The parameters used in the torque-joint angle relationship was obtained for each subject by using values recorded during the testing session. Each subject was asked to perform a maximal isometric contraction at 105, 120, 140, and 160 degrees of elbow extension (180 degrees corresponding to full elbow extension). These values covered the full range of movement provided by the experimental apparatus. The full-wave rectified, low-pass filtered EMG signals were then gain adjusted in order to get a best match with the corresponding trial's isometric force measurements. This was done by using a torque-joint angle function described by the following equation and can be seen in figure 7:

$$Lfac = 1/(e^{(((1-Lo)\,/\,\theta)\,/\,sk)\;2})$$

Where:
Lfac = moment arm length factor (0 > Lfac > 1)
Lo = optimal joint angle for isometric force production (105 degrees)
θ = joint angle
sk = skew of the function (0.9-1.5)

Figure 8: Representative unitless "EMG gain" to joint angle curve for the elbow joint:



The torque-velocity relationship has a concentric and an eccentric component for the elbow flexors. This relationship was determined based on the work of Dowling (1987) and Dowling et al. (1995). The range of values used can be seen in figure 3.

Concentric Component:

$$V_{fac} = A + (B \times \omega)$$

Where: V_{fac} = a unitless velocity gain value
$A = (0.8 < A < .999)$
$B = (.001 < B < .004)$
ω = angular velocity (rad/sec)

Eccentric Component:

$$V_{fac} = 1 + (B \times \omega)$$

Where: V_{fac} = a unitless velocity gain value, $V_{fac} <= 1.35$
$B = (.005 < B < .009)$

The passive element of the model was obtained by calculating the torque generated for each different stretch-shortening trial. The inertial, passive, viscous , and gravitational torque produced by the arm and experimental apparatus were thus obtained for all of the movement velocities within the experimental protocol. Since the velocity was kept constant by the torque motor for the active muscle trials, the passive torque values could be used to measure the amount of passive work done by the passive elastic component, inertia of the arm and apparatus, and the friction of the system. Both positive and negative passive work were measured. This passive work could then be removed from the active muscle trials. Visual inspection of the biceps and triceps EMG showed no considerable activation for these trials.

2.2 Experimental Procedures:

Seated in a custom built apparatus, the right arm of each subject was fastened to a support allowing flexion and extension about the elbow joint in the sagital plane. The forearm and wrist were secured using "Velcro" fasteners in a supinated position. The shoulder position was maintained at 90 degrees forward flexion throughout the testing period. This was accomplished by adjusting seat height to the individual. The apparatus used in these experiments can be seen in appendix A. In order to minimize the effects of fatigue on the results, a resting period of no less than two minutes was also given between each trial (Tesch et al., 1990). Isometric, quick release and stretch-shortening cycles were randomised to minimise the effects of fatigue. Muscle activation protocol was also fully randomised.

2.3 Experimental Protocol:

2.3.1 Isometric Trials:

The subjects performed a series of four ramped maximum voluntary and stimulated isometric contractions followed by a fast submaximal effort, for calibration purposes. The elbow flexor torque was recorded at 105, 120, 140 and 160 degrees of elbow flexion. The torque, EMG and joint angle data were recorded for the eight trials and saved on computer disk. Joint angle data were collected by an electrogoniometer (JDK model 6209-2001) situated on the rotating shaft of the arm support using a DATAQ data acquisition unit and Codas software. Isometric torque was measured using a strain gauge (custom mounted Micro Measurements gauge, sensitivity optimized for a range of 0 to 120 Nm) mounted on the shaft of the arm support and recorded using Codas software. Isometric force was also displayed for the subject on a computer screen so that they could monitor their force production during voluntary trials. Encouragement was given during these trials to ensure a maximal effort.

2.3.2 Stretch Shortening Trials:

Four sets of trials were performed by each subject under SSC conditions. During these trials, an active muscle stretch (eccentric contraction) preceded an active muscle shortening (concentric contraction). For all trials the arm support was driven by an external torque motor (Reliance Electric model E728) that provided a range of motion of 105 to 160 degrees of elbow extension. The torque motor was controlled to

produce movements at four different constant frequencies of elbow flexion extension cycles (0.58, 1.5, 2.4, and 3.3 Hz). The three different muscle activation protocols consisted of a 60% stimulated muscle activation trial, a passive trial and a voluntary contraction trial. During all trials torque, joint angle, biceps muscle activity and triceps muscle activity were collected using the DATAQ data acquisition unit and Codas software, then analog to digital converted and stored on computer disk. Joint angle and torque data were low-pass filtered at 13Hz with ten dual passes using a critically damped filter. Elbow joint velocity was calculated by differentiating the elbow joint displacement data and used as an input to the muscle model. The duration of each trial was six seconds, of which 4 seconds were under either voluntary or stimulated contraction of the elbow flexors (except for the passive trial which had no muscle contraction).

2.3.3 Non-Stretch-Shortening Trials:

Non-stretch-shortening (quick-release) trials were performed for passive, stimulated and voluntary conditions. These trials were performed at all four different velocities (.58, 1.5, 2.4, and 3.3 Hz) under all three activation protocols (stimulated, passive, and voluntary). The starting position for the quick-release trials was at 160 degrees of extension. The motor was locked in this position for the beginning of the trial, then released and elbow flexion continued down to 105 degrees before the motor was de-activated. Stimulated and voluntary trials initiated isometric muscle activation for 1 to 1.5 seconds prior to concentric muscle shortening, while the passive trial was

performed with no muscle activity. During all trials torque, joint angle, biceps muscle activity and triceps muscle activity were collected using the DATAQ data acquisition unit and Codas software. Joint angle and torque data were low-pass filtered at 13Hz with ten dual passes using a critically damped filter. Elbow joint velocity was calculated as the differentiated elbow joint displacement data.

2.4 Work Calculations:

The work calculations reported for the SSC were measured on all seven subjects. When, however, comparisons are made with the non-SSC trials, the data of only four subjects was available and only positive work is reported (no negative work is performed during the non-SSC trials). In order to perform repeated measures on these trials, the SSC data of only these four subjects was used and reported in the text and graphs. Because the apparatus was unable to maintain the full range of motion as the frequency of movement increased (see figure 9), positive work calculations were done for all subjects during the concentric phase of movement over a range of 157 to 130 degrees of elbow extension. This results in diminished positive work values at the slower velocities.

2.4.1 Externally Measured Work:

The externally measured work (as calculated by the torque transducer and electrogoniometer outputs) was measured by taking the integral of the torque and angular displacement data. The total work (TWRK) was taken as the algebraic sum of the positive work (PWRK: produced during the concentric phase of movement) and the

negative work (NWRK: produced during the eccentric phase of movement). During non-stretch-shortening trials, only positive work was recorded as the movement included only a concentric movement phase.

Measured Work: $TWRK = \sum Tor \bullet (Jt_i - Jt_{i-1})$

$PWRK = \sum Tor \bullet (Jt_i - Jt_{i-1}),\ \Delta Jt < 0$

$NWRK = \sum Tor \bullet (Jt_i - Jt_{i-1}),\ \Delta Jt > 0$

Where:
$\sum$ = sum of all samples during the recorded trial
Tor = torque measured by the strain gauge (Nm)
TWRK = total work performed on the system (J)
PWRK = positive work performed on the system (J)
NWRK = negative work performed on the system (J)
Jt_{i-1} = initial joint angle (rad)
Jt_i = initial joint angle plus one sample (rad)

The work done by the passive components (inertia, friction, viscosity) was calculated during a passive trial for each subject at each speed under both SSC and non-SSC protocols. These values could then be subtracted from the active muscle contraction trials. This subtraction would therefore yield the amount of work produced by the elbow flexors.

2.4.2 Internally Measured (muscle) Work:

Once the muscle activity had been calibrated using the muscle model to obtain a muscle torque output, this data was then integrated with respect to the angular displacement over time. This gave the amount of work produced for each SSC trial (total muscle work or TMWRK). The TMWRK was then separated into positive muscle work (PMWRK) which was the work produced during concentric contractions

and negative muscle work (NMWRK) or the work done for the eccentric phase of the movement. For the non-SSC trials, only positive muscle work was produced (since only a concentric contraction took place), therefore, the positive muscle work from the non-SSC trials could be compared to the positive muscle work of the SSC trials.

Muscle Work:

$$TMWRK = \sum Mtor \bullet (Jt_i - Jt_{i-1})$$

$$PMWRK = \sum Mtor \bullet (Jt_i - Jt_{i-1}),\ \Delta Jt < 0$$

$$NMWRK = \sum Mtor \bullet (Jt_i - Jt_{i-1}),\ \Delta Jt > 0$$

Where:

$\sum$ = sum of all samples during the recorded trial
MTor = torque derived from the EMG muscle model (Nm)
TMWRK = total work produced by the muscle (J)
PMWRK = positive work produced by the muscle (J)
NMWRK = negative work produced by the muscle (J)
Jt_{i-1} = initial joint angle (rad)
Jt_i = initial joint angle plus one data point (rad)

The predicted muscle work could then be compared to the measured work and an indication of the muscle's effectiveness could thus be obtained. Simulated muscle work was also calculated using the muscle model and a constant muscle activation input. This simulated work could be used to indicate the muscle model's accuracy and the consistency of stimulated muscle activation. It was assumed that the elbow flexors of all subjects would react in a similar fashion with respect to their ability to convert muscle activity to mechanical work.

2.5 Statistical Analysis:

For all statistical analysis alpha was set at .05 ($p < .05$). Repeated measures analysis of variance was used to assess differences between total, positive and negative work under SSC conditions. Trial conditions (SSC and non-SSC) were treated as independent variables while the repeated measures were performed for work type (externally measured, modeled and simulated) and movement velocity. Tukey HSD post-hoc test were performed to assess interactions and main effects and complete ANOVA tables can be found in appendix B.

CHAPTER III

3.0 Results :

The results section has been divided into two sections, the first dealing with the externally measured work (sections 3.1.1-3.1.2), the second dealing with internally measured muscle work and the accuracy of the muscle modelling procedures (sections 3.2.1-3.2.3). Due to processing issues which are insurmountable at this time, the voluntary contraction conditions are not discussed in this paper. The EMG data under voluntary conditions were not able to predict well under all non-SSC conditions and as such was unacceptable for the purposes of this paper (see section 5.1, Recommendations for Future Research).

3.1 Externally Measured Work:

3.1.1 SSC Trials:

Figure 9 illustrates the measured torque values as a function of angular displacement during SSC trials at all four velocities. These graphs represent the "work loops" for a representative subject (the results of the three additional subjects used in SSC and non-SSC comparisons can be found in appendix C). The area within the torque-displacement curve is the difference between the eccentric and concentric work and equals the amount of work performed by the limb and apparatus on the strain gauge. With the passive components removed, this work is equivalent to the amount of work performed by the elbow flexors. It should be noted that the direction of movement is clockwise, the eccentric contraction beginning at 105 degrees, with torque

rising to a maximum value, before descending to 160 degrees were the concentric contraction begins. Both end points correspond to an isometric contraction. The repeatable nature of the torque-joint angle curves suggests that the subject is producing consistent results for each cycle within the trial therefore lending confidence to the methodology.

The total work, positive work, and negative work derived from these loops can be seen in figure 10 and is expressed as work done per cycle while the muscle is being stimulated. Statistical analysis reveals that the total work does not change as a function of velocity. Positive work, however, showed a significant decrease from 0.58 Hz to all other velocities ($p < .008$), as well as a significant drop from 1.5 Hz to 3.3 Hz ($p < .01$). The negative work produced during SSC trials showed significant decreases from 0.58 to 3.3 Hz ($p < .006$) and from 1.5 Hz to 3.3 Hz ($p < .002$).

Figure 9: Representative work loop data for a representative subject at four different frequencies of elbow flexion-extension (.58, 1.5, 2.4, and 3.3 Hz) over four seconds of constant muscle stimulation. Note: square symbols represent isometric torque data under the same conditions of muscle stimulation, solid line represents SSC torque data.

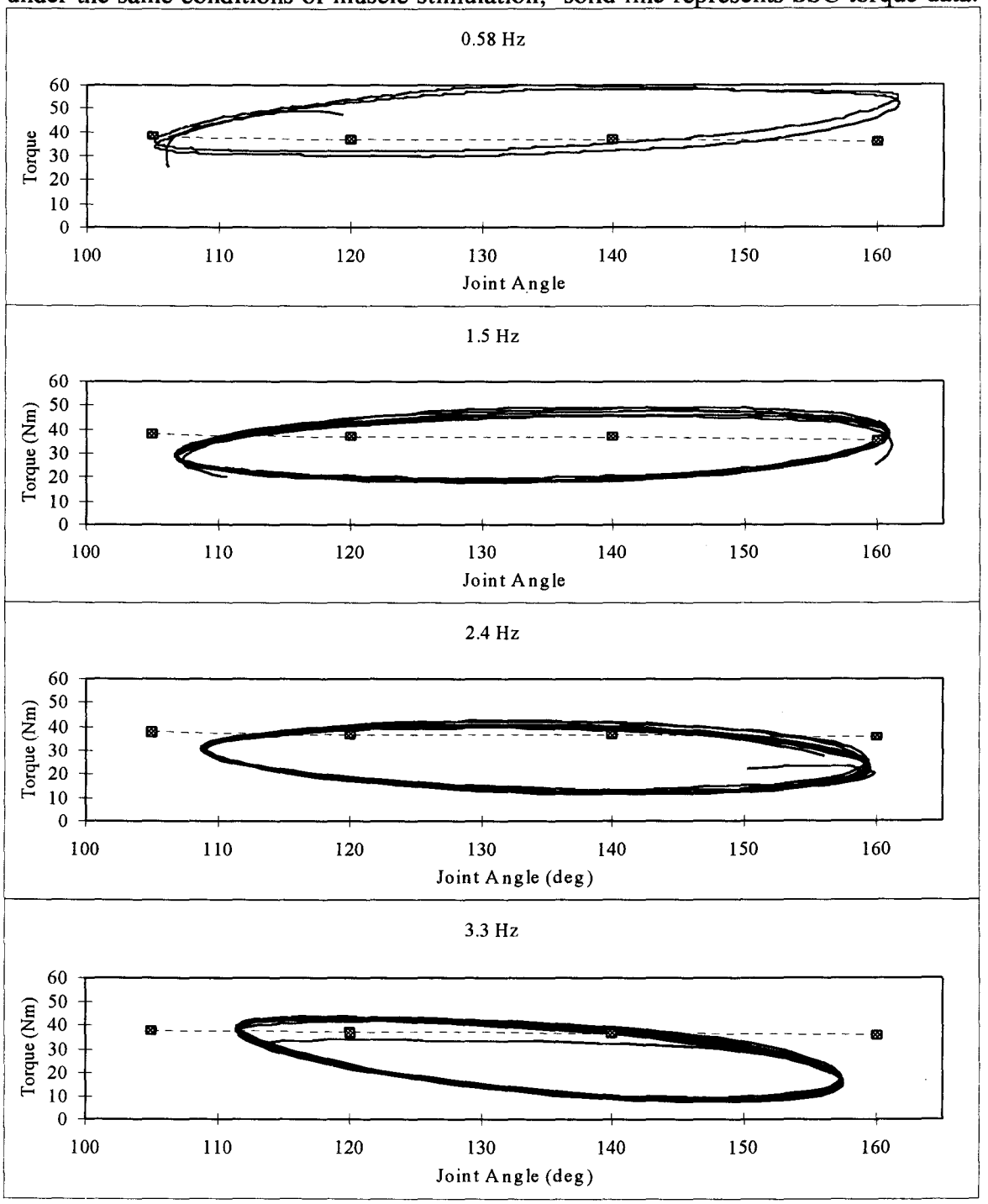
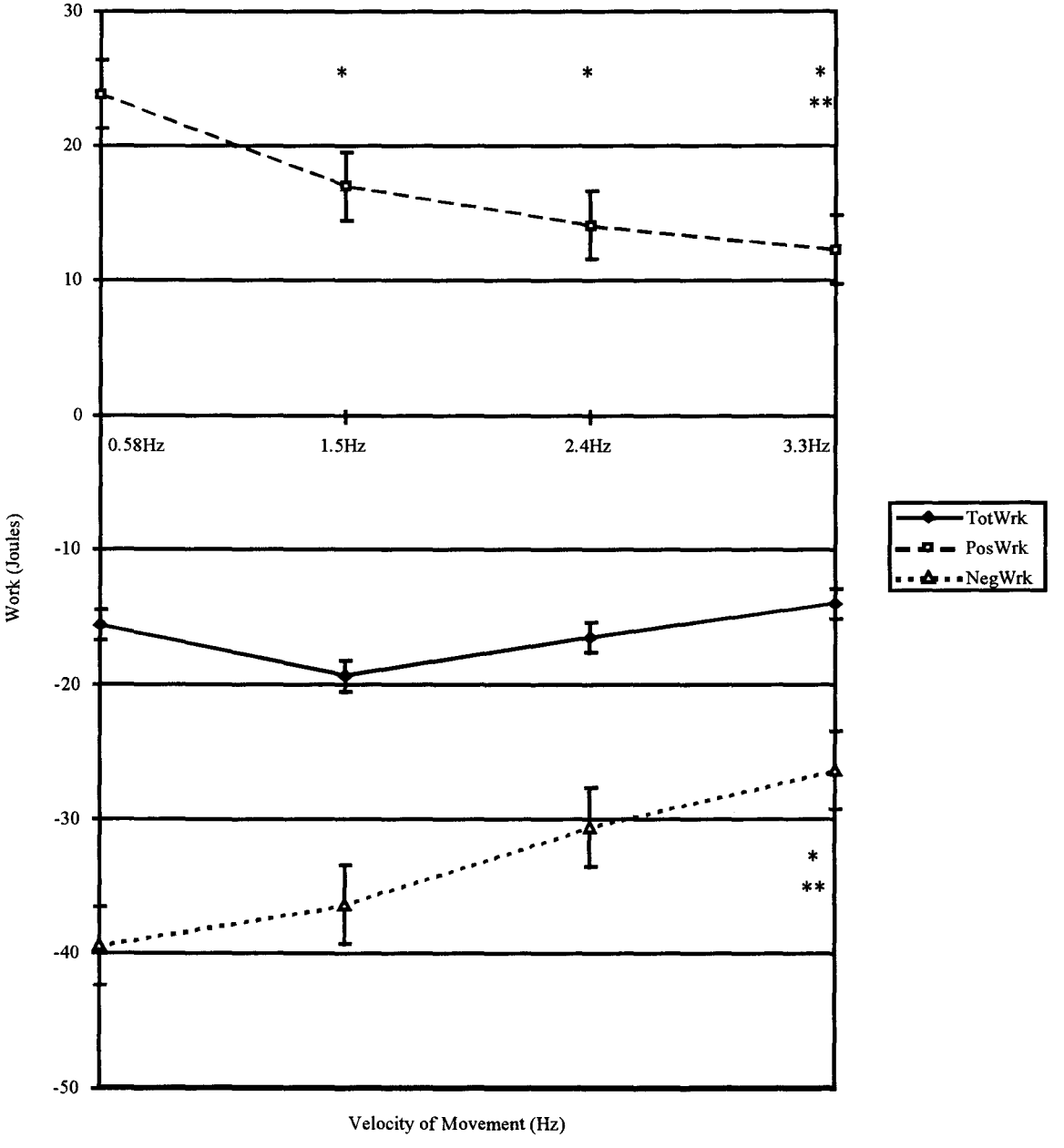


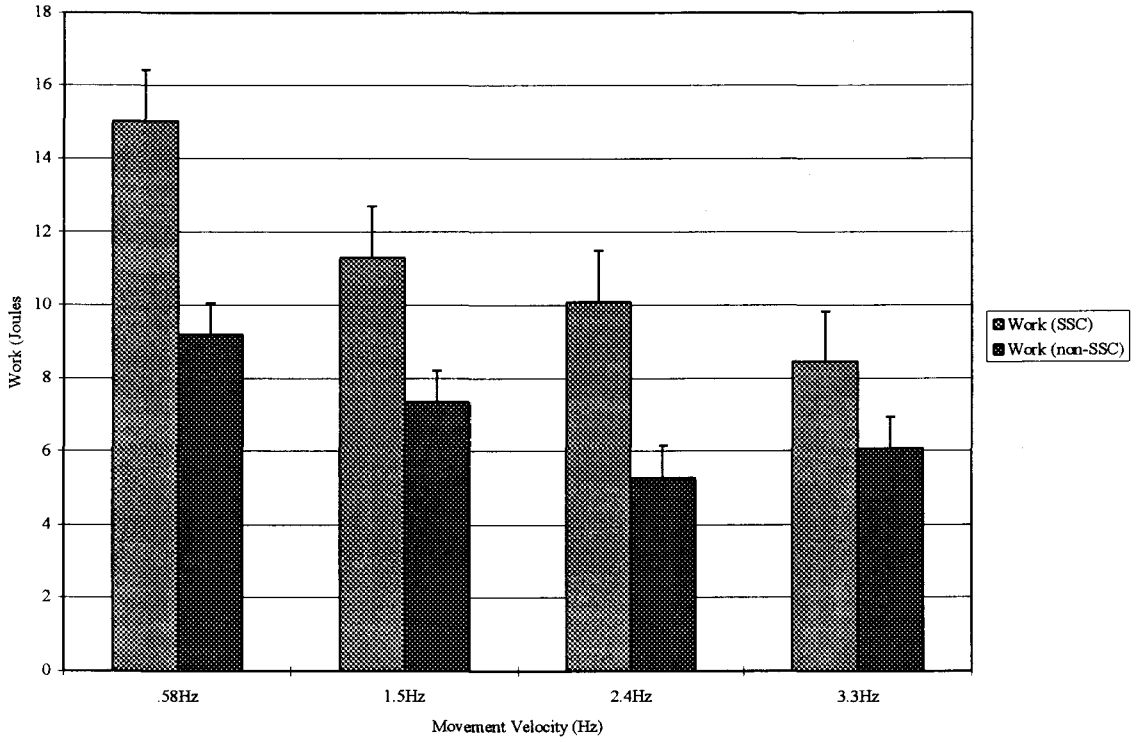
Figure 10: Average measured work per cycle for all seven subjects at all four frequencies of elbow flexion extension (.58, 1.5, 2.4, and 3.3 Hz), (±) standard error of the mean. *, significant decrease in work from .58 Hz; **, significant decrease in work from 1.5 Hz.:



3.1.2 SSC and non-SSC Trials:

The measured work of the SSC trials was compared to the work of the non-SSC trials over a concentric contraction from 157 to 130 degrees of elbow flexion. For these trials, the data of only four subjects were available. A significant increase in measured work was found during the SSC trials when compared with the non-SSC trials between 162 and 130 degrees of elbow flexion ($p < .05$) (see figure 11).

Figure 11: Average work measured per cycle of elbow flexion for four subjects under SSC and non-SSC (between 157 and 130 degrees of elbow extension) at all four frequencies of movement (.58, 1.5, 2.4, and 3.3 Hz), (±) standard error of the mean:



Figures 12 and 13 show the data of all four subjects for the SSC and non-SSC trials respectively. In these figures, there appears to be a trend for decreasing work with increasing frequency of movement. When work is collapsed across all subjects and conditions, there is a significant effect for decreased work with increased movement velocity ($p < .05$).

Figure 12: Work measured per cycle of elbow flexion of four subjects for non-SSC conditions (between 157 and 130 degrees of elbow extension) at all four frequencies of movement (.58, 1.5, 2.4, and 3.3 Hz):

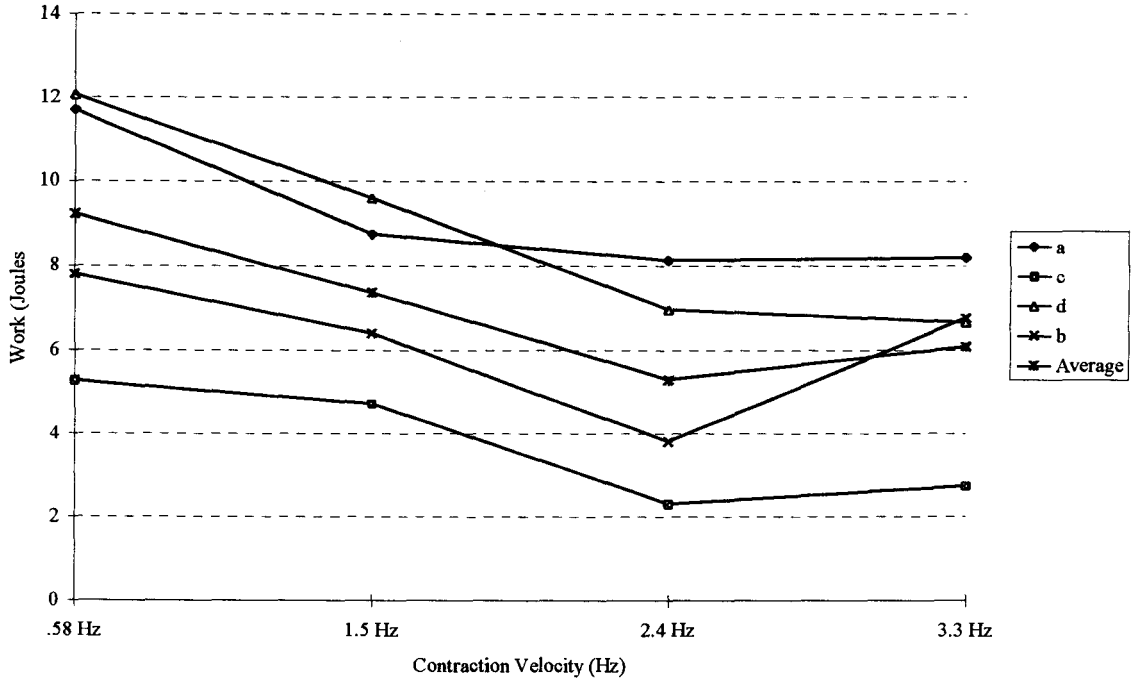
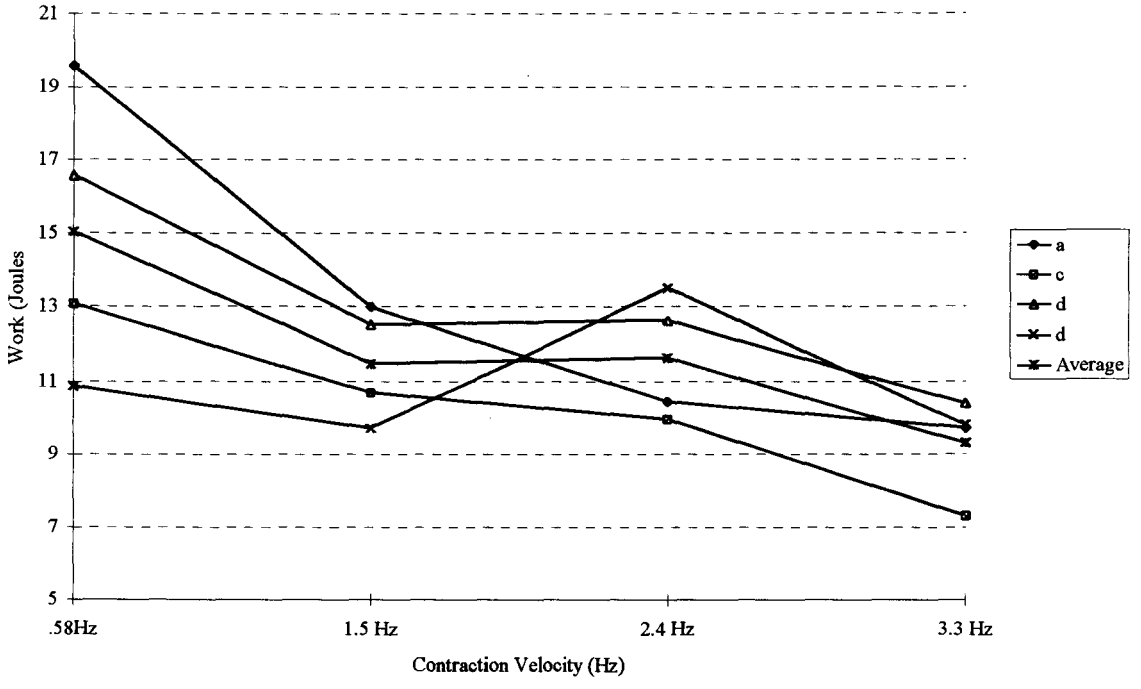


Figure 13: Work measured per cycle of elbow flexion of four subjects for SSC conditions (between 157 and 130 degrees of elbow extension) at all four frequencies of movement (.58, 1.5, 2.4, and 3.3 Hz):



3.2 Measured Muscle Work and Muscle Model Accuracy:

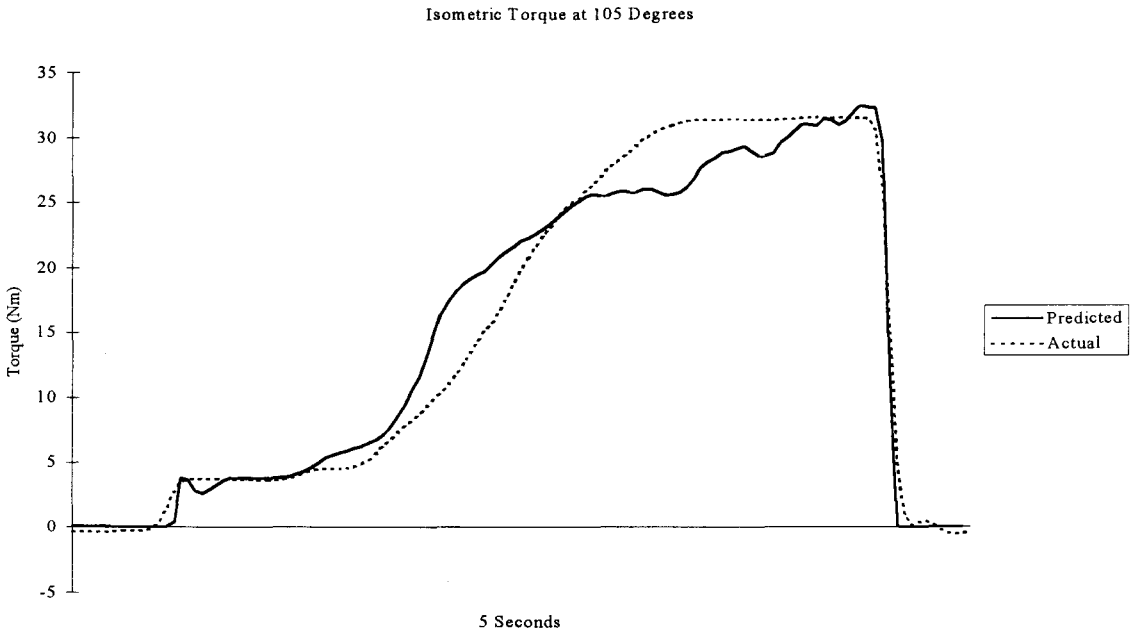
The nature of this study required that the EMG driven muscle model be an accurate tool that could be used to determine how muscle effectiveness is altered by changing kinematic variables and muscle activation characteristics. It was therefore important that the model accurately estimate the measured torque during contractile conditions which did not involve a SSC. If under non-SSC conditions the model accurately represents the measured torque, we can then assume that any deviation from model accuracy is due to series elastic component-contractile element interactions

induced by the SSC. To verify the model and determine the nature of the performance enhancement previously reported, constant activation simulation trials were performed. For these trials, muscle activation was determined for each subject based on their isometric stimulated torque output at optimal muscle length (105 degrees, Kulig et al., 1984). Constant activation muscle model simulation trials were then calculated for all dynamic trials. The muscle model was then applied to the actual EMG data. Accuracy of the simulations and EMG model was determined by statistical analysis using a repeated measures analysis of variance (see Table 1 for non-linear EMG gain model accuracy). An example of a typical predicted torque and measured torque under an isometric calibration trial can be seen in figure 14. It is important to note that the measured and predicted rise in torque were closely matched. This gave an indication that the EMG filter cutoff frequency used was appropriate for this type of modelling. This figure also indicates that the artifact suppression used to process the EMG signal does not interfere with muscle torque prediction.

Table 1.: Isometric Muscle Model Accuracy at 105 degrees Elbow Extension:

Subject	RMS error (Nm)	r-square
A	3.37	.949
B	3.0	.948
C	2.48	.964
D	4.224	.946
Average	3.2685	.9518
Std. Deviation	.7342	.0083

Figure 14: Isometric muscle model calibration trial for a representative subject recorded at 105 degrees of elbow extension:



In both SSC and non-SSC trials, simulated muscle work was a good indicator of externally measured work. This implied that the muscle model used in this study accurately reflected muscle performance under constant muscle activation conditions. Statistical analysis revealed that modelled muscle work was also a good indicator of measured work under non-SSC conditions (see figure 15). (note: the unusually high measured work value at 3.3 Hz may be associated with an outlying value) For SSC trials the work derived from the EMG data was lower than the externally measured work for all speeds, although not statistically significant ($p=.052$) (see figure 16).

Figure 15: Average measured, predicted (from EMG), and simulated (from constant activation model) work measured per cycle of elbow flexion of four subjects for non-SSC conditions (between 157 and 130 degrees of elbow extension) at all four frequencies of movement (.58, 1.5, 2.4, and 3.3 Hz). Work, measured work; Mwork, work predicted from EMG model; Pwork, work predicted from constant activation model:

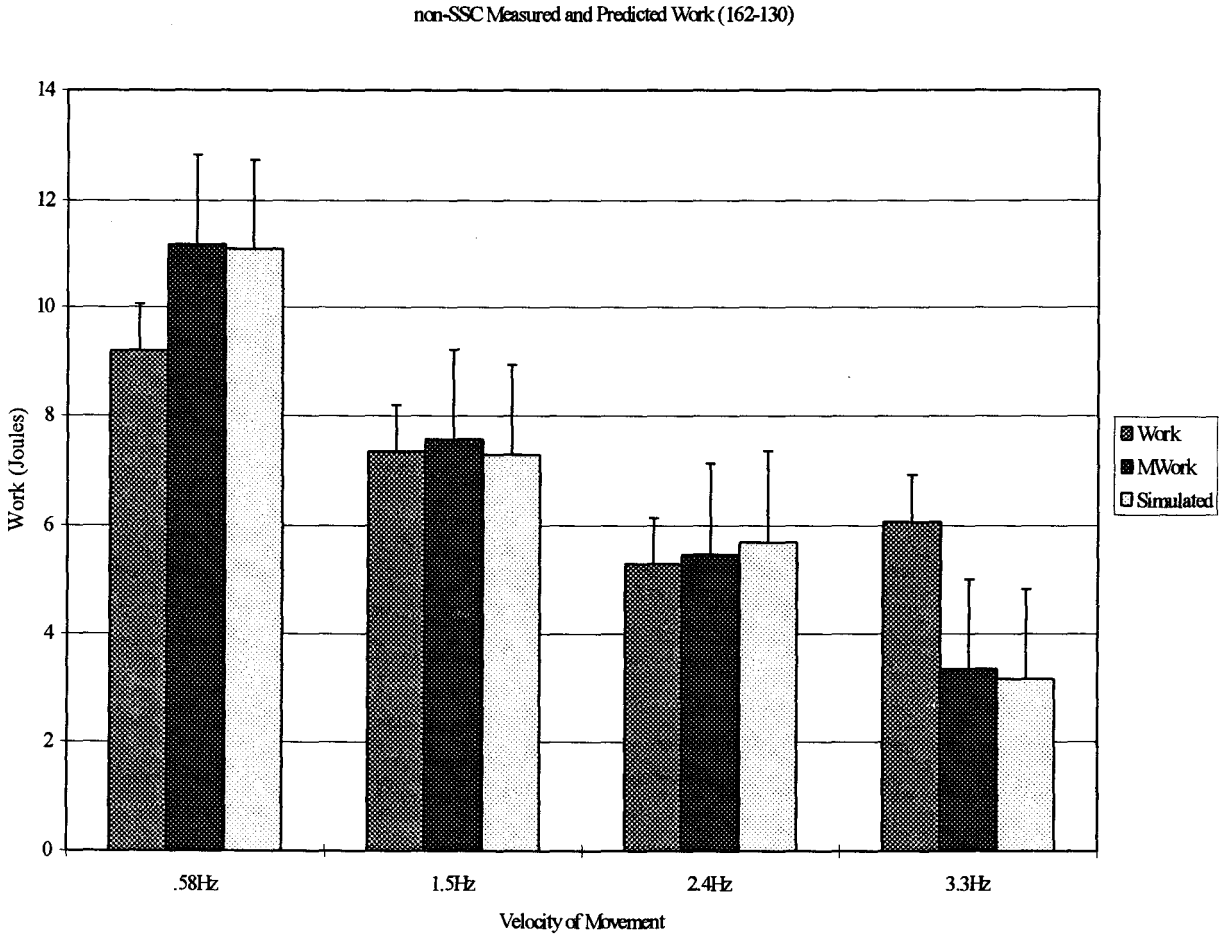
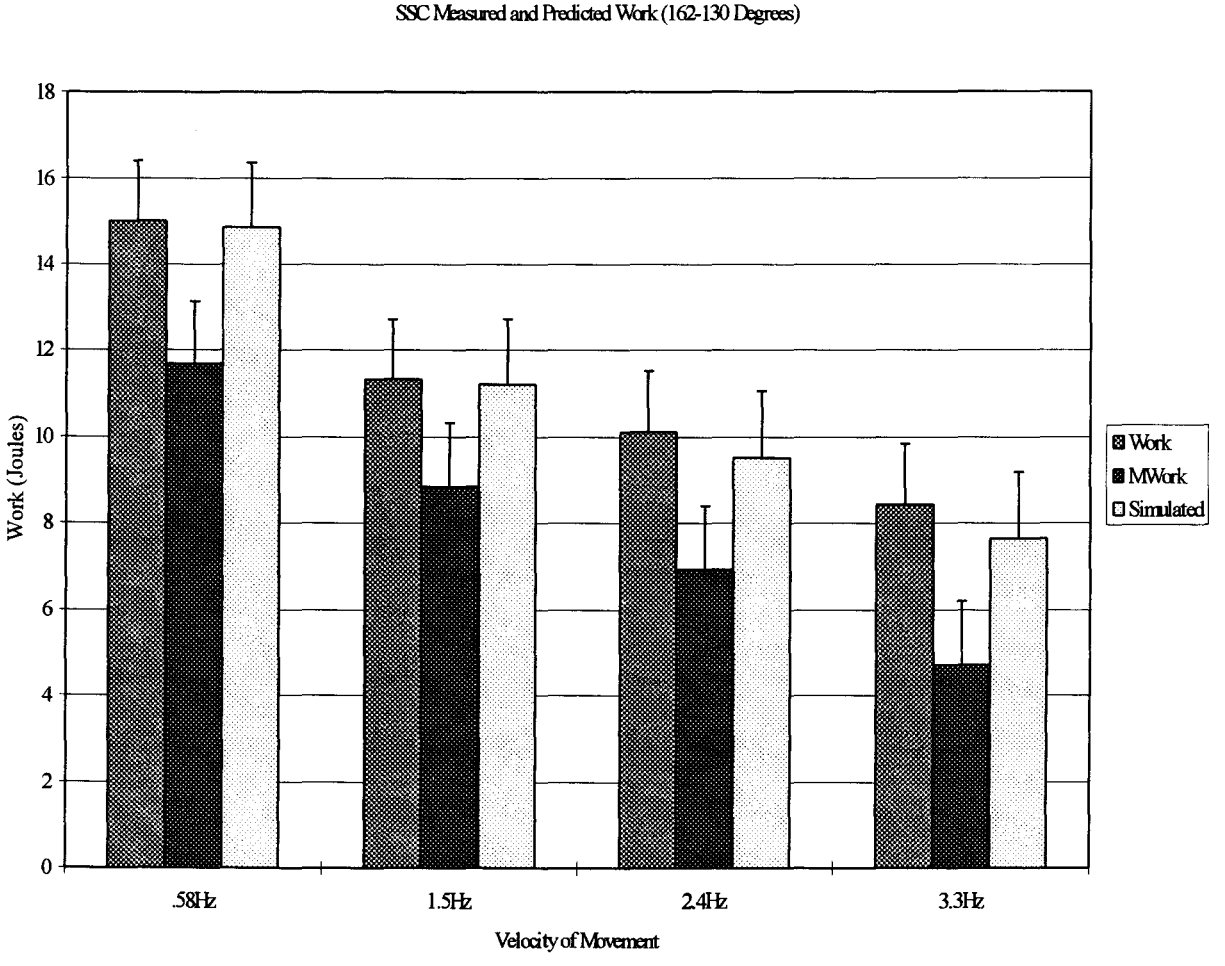


Figure 16: Average measured, predicted (from EMG), and simulated (from constant activation model) work measured per cycle of elbow flexion of four subjects for SSC conditions (between 157 and 130 degrees of elbow extension) at all four frequencies of movement (.58, 1.5, 2.4, and 3.3 Hz). Work, measured work; Mwork, work predicted from EMG model; Pwork, work predicted from constant activation model:



CHAPTER IV

4.0 Discussion:

4.1 Work Loops:

The area within the work loops in figure 9 and appendix B describes the work done by the elbow flexors to the system and therefore represents the external work. The shape of the work loops lends insight into how and when the elbow flexors are contributing this work. The work loops described by Rack and Westbury (1974) were used to describe the short-range stiffness of muscle and the effects of increased stretch-shortening velocity on work production. The work loops recorded in this study obviously differ from those in figures 2A and 2B from Rack and Westbury (1974). The sharp change in direction (from eccentric to concentric contraction) in Rack and Westbury's study were not achieved in this study, as the apparatus used in this study produced a more sinusoidal movement pattern. This would account for the more rounded loops in figure 9 and as such comparisons should be made with figure 2B. On an isolated cat soleus muscle, Rack and Westbury found that work increased up to a frequency of 4.5 Hz before decreasing at higher eccentric velocities. This study showed work decreasing from .58 Hz up to 3.3 Hz for the human elbow flexor muscle group. The reasons for the decreasing work in both studies, although taking place at different speeds, can be attributed to the same thing: muscle's inability to maintain high eccentric force values at relatively high eccentric velocities. It is obvious that in this study the eccentric torque values decreased at higher SSC movement velocities (see

figure 9 and appendix B), as did the average eccentric torque values of Rack and Westbury (see figure 2b). This decreased eccentric torque may be responsible for the decrease in work.

4.2: SSC Work:

Based on the force-velocity relationship, the total work done by the arm on the system should decrease with increasing shortening velocity. This would be expected since the concentric portion of the force-velocity curve indicates a decrease in torque of approximately 70% at 3.3 Hz while the increase in torque by the eccentric contraction should be approximately 40% over isometric values (see figure 3, Dowling et al., 1995). Since velocity maintained over all SSC cycles, and measurements were made over the same range of movement, torque should be directly correlated with work. The data indicates that while the positive work does decrease by over 50%, the negative work does not increase with velocity but in fact decreases. This decrease in negative work is therefore a function of a decreased muscle torque and is indicated in figure 8 since the maximal eccentric torque values did not increase with velocity. The trials performed in this study ranged from 66 to 378 $degS^{-1}$ of elbow flexion-extension while the most reliable data is limited to 110 $degS^{-1}$ for elbow extension (Dowling et al., 1995). Westing et al. (1991) indicated that under voluntary contraction conditions, subjects tended to reduce muscle activity during eccentric loading. This decrease was associated with a protective mechanism such as a reflex inhibition. In this study, however, the trials indicated are conducted with a constant muscle stimulation of 60%

MVC. For all trials, triceps muscle activity was negligible (as evidenced by the recorded EMG signal) and all trials were randomised to minimise the effects of muscle fatigue or muscle damage due to eccentric loading. Taking this into account, along with the fact that the muscle was stimulated and that the passive trial subtraction included possible torque changes due to a stretch reflex, it is unlikely that neural feedback is responsible for the decrease in muscle torque. The relative linear velocity of the biceps for the four trials is 29, 75, 120, and 165 mmS^{-1} based on a whole muscle length change for the SSC trials of 350 mm to 375 mm (Lynch, 1992, adapted from Frigo and Pedotti, 1978). At these velocities, over very short distances (1.49-3.95 mm) human muscle has been shown to produce significantly more force then concentric contractions during eccentric contractions (Cook and McDonagh, 1996). However, recent work by Ettema (1996a) has shown that :

> "mainly because of SEC-CE (series elastic component-contractile element) interaction, isokinetic and isotonic force-velocity curves are poor predictors of muscle-tendon performance ability during locomotor or sinusoidal movements".

It is therefore possible that under the conditions of this experiment (high load and high velocity over a large range of motion) the eccentric loading of the elbow flexors exceeds the muscle's capability to maintain cross-bridge integrity and that the force-velocity curve should have a descending slope. Since the eccentric portion of the force-velocity curve is not well predicted for in-vivo human studies (reliable high-velocity

constant activation eccentric torque values are not well represented in the literature), and non-existent for SSC movement tasks, it is possible that muscle torque does in fact decrease at high eccentric velocities under SSC conditions. Another possible explanation is that muscle activity under SSC conditions is actually decreasing.

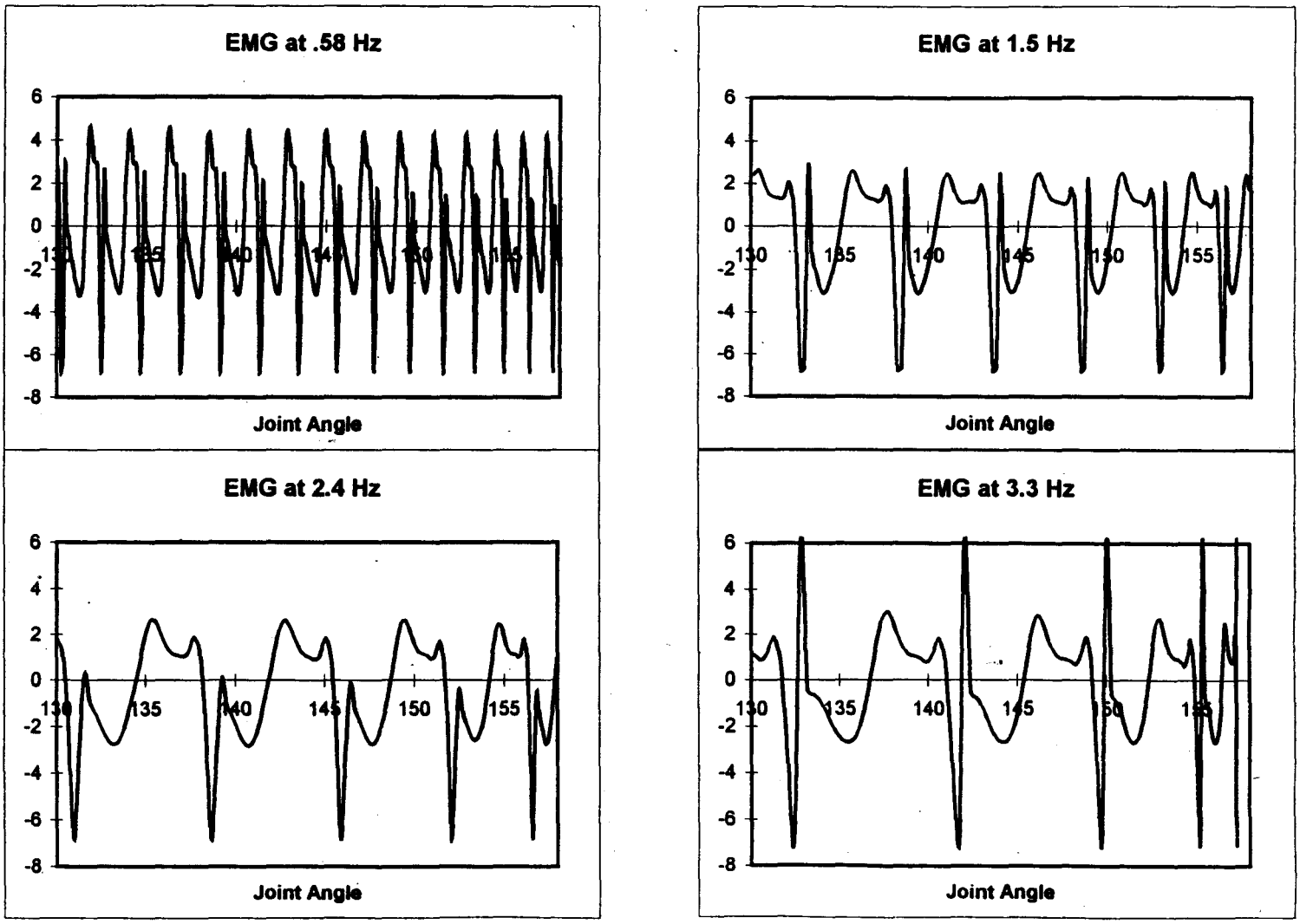
4.3 Changes in Myoelectric Activity:

Although the muscle was transcutaneously stimulated at a constant voltage and frequency (50 Hz), the results in figures 15 and 16 indicate that recorded muscle activity did in fact change. Since the simulated work (with a simulated constant muscle activation input) predicted the externally measured work well under all conditions whereas the modeled work (with an EMG based muscle activation input) predicted well for non-SSC conditions and under-estimated for SSC conditions, this implied that the EMG input was lower under SSC contractions. This was supported by the fact that the "average rectified value" of the M-wave (Merletti, Knaflitz and deLucca, 1992) decreased with increasing SSC movement velocity (see figure 17; note: in figure 17, signal artifact changes because the peak artifact signal may arrive between recorded samples due to the sampling rate of 1250 Hz and the high frequency of the artifact signal). The average rectified value corresponds to the average signal amplitude recorded over pre-determined period of time. In figure 17 these values decreased from 3.12 mV at .58 Hz to 1.855 mV at 3.3 Hz. It appears that the act of stretching the elbow flexors during an eccentric SSC contraction at a stimulation voltage which elicits a 60% MVC caused an inhibition of motor unit action potential conduction properties

which increased with velocity. It is worth noting that based on the force-velocity curve, the estimated eccentric torque under these conditions would meet or exceed maximum voluntary contraction torque (Dudley et al., 1992). The possible mechanisms for this alteration in membrane excitability are unclear and the available literature on changes in M-wave patterns under different movement velocities is virtually non-existent. Since the muscle was being transcutaneously stimulated, any change to the action potential must have taken place at or below the motor end plate. Electrode placement relative to the recorded M-wave has been shown to affect myoelectric signal amplitudes, however in this study, all recorded EMG used for the model was taken over the same joint angles (157-130 degrees of elbow extension). It is therefore unlikely that electrode placement was the cause of the M-wave discrepancies. The only apparent change between trials which affected the myoelectric signal seemed to be trial condition (SSC or non-SSC) and movement frequency. Although explaining this phenomenon goes beyond the scope of this paper, potential changes in membrane excitability could be induced by an unavailability of calcium (which has been linked to M-wave reduction due to changes in muscle length), an increase in extracellular potassium, a decreased M-wave conduction velocity, a decrease in extracellular pH, or a possible decrease in available acetylcoline receptor sites (possibly due to a lack of calcium or an increase of acetylcolinesterase from the basement membrane) (McComas, 1996; Brody et al., 1991, Broman et al., 1985). The presence of metaboreceptors has been linked with increased concentrations of extracellular hydrogen and potassium,

which would in turn decrease membrane excitability, and may be a possible cause for the decreased excitability in this study (McComas, 1996).

Figure 17: Raw EMG signal (in mV) of a representative subject recorded at all four frequencies of elbow flexion-extension (.58, 1.5, 2.4, and 3.3 Hz) under a constant voltage (representing 60% MVC) and stimulation frequency (50 Hz) over a concentric range of 157 to 130 degrees:



4.4 SSC Enhancement:

Although the recorded EMG signal was shown to decrease compared to the constant activation model (figure 15), the measured work output of the elbow flexors was still significantly higher under SSC conditions. This indicates that even with decreased muscle activation, the SSC still enhanced muscle work output. The mechanism for this appears to be affected by movement frequency, as evidenced by the decreased EMG activity with increased SSC velocity. There appears, therefore, to be an interaction between the recorded EMG signal form the contracting muscle and movement frequency. Ettema (1996b) has shown that series elastic component efficiency is highly correlated with mechanical efficiency and explains that the contractile element can temporarily store energy in the series elastic element when the contractile element is shortening and the series elastic element is lengthening. In his model, the series elastic element included both the extracellular structures (tendon elasticity) and intracellular structures (myofilament and cross-bridge elasticity). Since the biceps tendon is relatively short and stiff (Joyce et al., 1974), and in light of work by Barrata and Solomonow (1991), Roleveld et al. (1994), and Roberts et al. (1997), the potential energy stored in the tendon during a SSC may be somewhat limited. A more likely location of stored energy for the elbow flexors would therefore be in the intracellular structures and aponeurosis. If this in fact was the case, the increased strain within the contractile component induced by eccentric SSC contractions may have been

what caused the diminished M-wave response and, as a result, the decreased eccentric torque at high velocities.

CHAPTER V

5.1 Summary and Conclusions:

The results of this study indicate that a SSC enhances muscular work production of the elbow flexors during sinusoidal movements at and above 0.58 Hz of elbow flexion-extension. The protocol of this study was designed in order to relate the findings from certain isolated muscle studies (see chapter 2) to human experimental conditions. The non-SSC trials provided a basis for comparison of the SSC enhancement, while eliminating the possible performance enhancement generated by the "in-waste" theory (since the muscle-tendon unit was already pre-stressed). The non-SSC trials also provided a situation in which the contractile component would be potentiated (stimulation occurred for 1 to 1.5 seconds prior to the movement), thus providing similar contraction conditions for both SSC and non-SSC trials. Since the measured work values change with velocity, this suggests that storage of energy within the spring-like series elastic element is unlikely as series elastic element efficiency does not appear to be affected by movement frequency (Ettema, 1996b). Furthermore, the simulated constant activation muscle model is a good determinant of measured work for both the non-SSC and SSC contraction conditions, however the EMG based muscle model is only a good predictor under non-SSC conditions. Since the only difference between the simulated constant muscle activation model and the EMG based muscle model is the amount of recorded electrical output of the elbow flexors, the muscle work estimated by the EMG signal should also be a good predictor of SSC trial work. This,

however, does not appear to be the case, indicating decreased elbow flexor muscle activation, which was evidenced by a decreased M-wave during SSC contractions. Although this decreased muscle response would be expected under voluntary conditions, it was not expected under transcutaneous stimulation of 50 Hz at 60% MVC. It appears, therefore, that a mechanism is present below or at the motor end plate that reduces the depolarizing capacity of the muscle membrane. The mechanisms that may be responsible for this phenomena are however unclear. Research into the area of possible reflex inhibitions and factors affecting M-wave amplitude changes with velocity are not well documented and difficult to assess (McComas, 1996. Further investigation into this phenomenon is certainly needed. In conclusion, this study indicates that the contractile component is directly related to SSC work enhancement and that the mechanism for this energy storage and release may be taking place at the myofilament and cross-bridge level.

5.1 Recommendations for Future Research:

Further investigations of in-vivo SSC work enhancement are required and the focus of these studies should be on determining how muscle activation patterns and myoelectric signals change under different contraction conditions (specifically changes in velocity and SSC contractions), as well as how the contractile element interacts with the muscle-tendon unit to increase muscle effectiveness at certain frequencies. Determining maximum twitch M-wave amplitudes during the SSC may contribute to a better understanding of muscle membrane excitability during sinusoidal movement patterns.

Including a pre and post twitch protocol for each movement trial could also be beneficial in order to determine the effects of fatigue caused by the eccentric SSC contractions. Another important aspect that must be investigated is the comparison of voluntary and stimulated SSC contraction. Although this was initially a part of this study, the voluntary EMG signal proved to be extremely variable under SSC conditions and as such requires a great deal more processing then the stimulated EMG signal. This work is presently under way in the hopes of further improving the understanding of in-vivo muscle function in humans.

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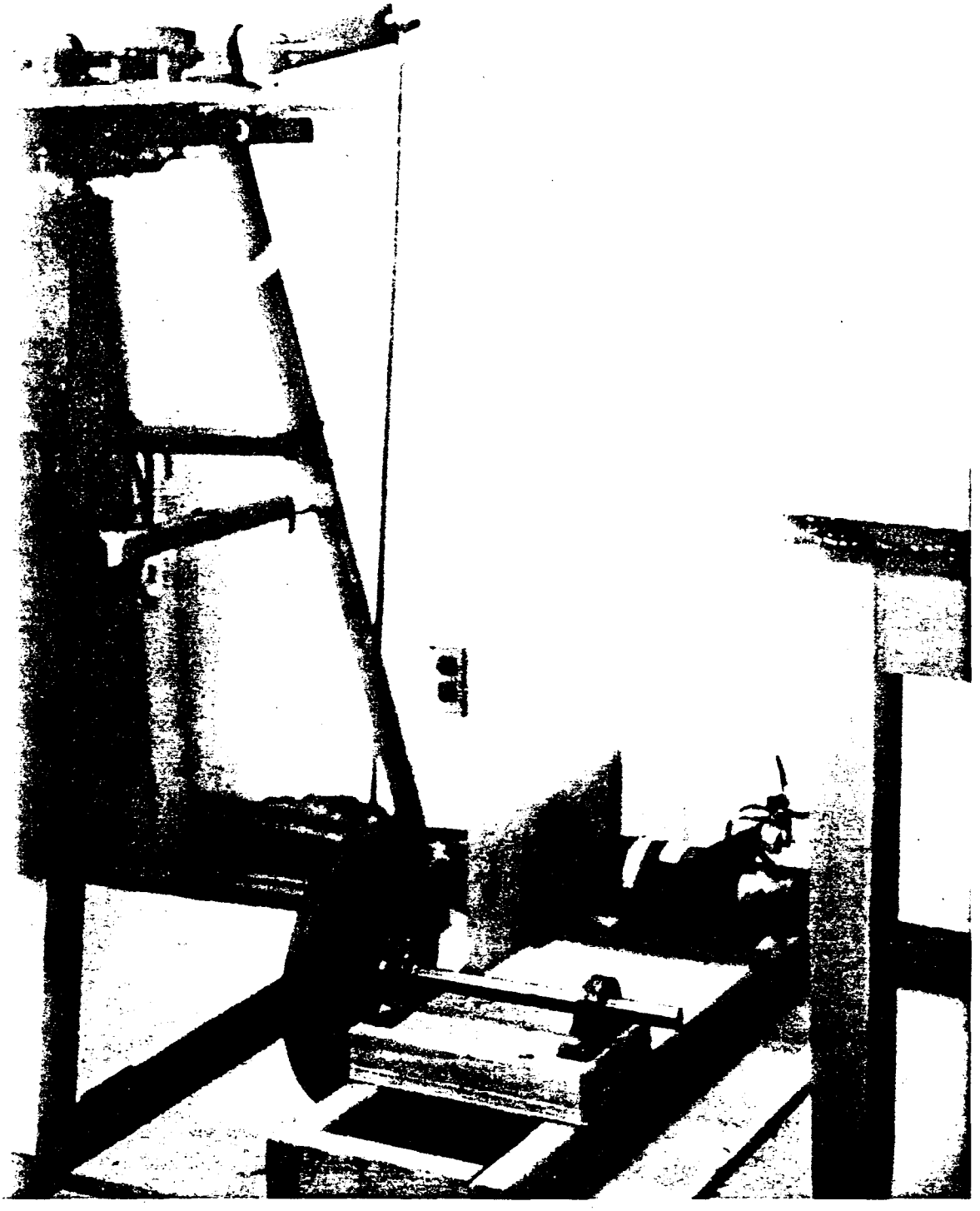
Appendix A

Testing apparatus: Arm Support (subject A)

Testing Apparatus: Drive System



Appendix B

i) SSC Statistical Analysis (7 subjects):

Total Work:

Summary of all Effects

1-SPEED effect on total work

Effect	df Effect	MS Error	df Error	MS	F	p-level
1	3	233680.8	15	233773.3	.999604	.420033

Positive Work:

Summary of all Effects

1-SPEED effect on positive work

Effect	df Effect	MS Error	df Error	MS	F	p-level
1	3	.083346	15	.004451	18.72637	*.000025*

Unequal N Tukey HSD

Probabilities for Post Hoc Tests

MAIN EFFECT: SPEED

		{.58 Hz}	{1.5 Hz}	{2.4 Hz}	{3.3 Hz}
	mean	.5187308	.3751667	.3092757	.2430216
1	{.58Hz}		*.009842*	*.000522*	*.000196*
2	{1.5 Hz}	*.009842*		.352575	*.017508*
3	{2.4 Hz}	*.000522*	.352575		.348072
4	{3.3 Hz}	*.000196*	*.017508*	.348072	

Negative Work:

Summary of all Effects

1-SPEED

Effect	df Effect	MS Error	df Error	MS	F	p-level
1	3	.133463	15	.015805	8.444382	*.001594*

Unequal N HSD

Probabilities for Post Hoc Tests

MAIN EFFECT: SPEED

		{.58 Hz}	{1.5 Hz}	{2.4 Hz}	{3.3 Hz}
	mean	-.854224	-.817691	-.669458	-.528576
1	{.58 Hz}		.957098	.092834	*.002320*
2	{1.5 Hz}	.957098		.216770	*.005997*
3	{2.4 Hz}	.092834	.216770		.253292
4	{3.3 Hz}	*.002320*	*.005997*	.253292	

ii) SSC and non-SSC Statistical Analysis (4 subjects):

Summary of all Effects

1-CONDITN (SSC, non-SSC), 2-TYPE (measured, EMG, simulated), 3-SPEED

Effect	df Effect	MS Error	df Error	MS	F	p-level
1	1	235.0659	6	37.76545	6.22436	*.046852*
2	2	24.3380	12	6.39544	3.80552	.052491
3	3	191.8362	18	3.52658	54.39726	*.000000*
12	2	23.4818	12	6.39544	3.67164	.057004
13	3	.5262 18	3.52658	.14920	.928854	
23	6	3.3923 36	1.34601	2.52027	.038606	
123	6	2.1861 36	1.34601	1.62416	.168830	

SSC vs non-SSC:

Tukey HSD test

Probabilities for Post Hoc Tests

MAIN EFFECT: CONDITION (SSC or non-SSC)

		{SSC}	{non-SSC}
(mean)		10.02122	6.891614
ssc	{1}		*.047035*
nonssc	{2}	*.047035*	

Work Type:

Tukey HSD test

Probabilities for Post Hoc Tests

MAIN EFFECT: TYPE (1=measured, 2=EMG, 3=simulated)

		{1}	{2}	{3}
	mean	9.098456	7.463531	8.807258
measured	{1}		.057845	.890749
muscle(EMG)	{2}	.057845		.126287
simulated	{3}	.890749	.126287	

Frequency (speed) of Movement:

Tukey HSD test

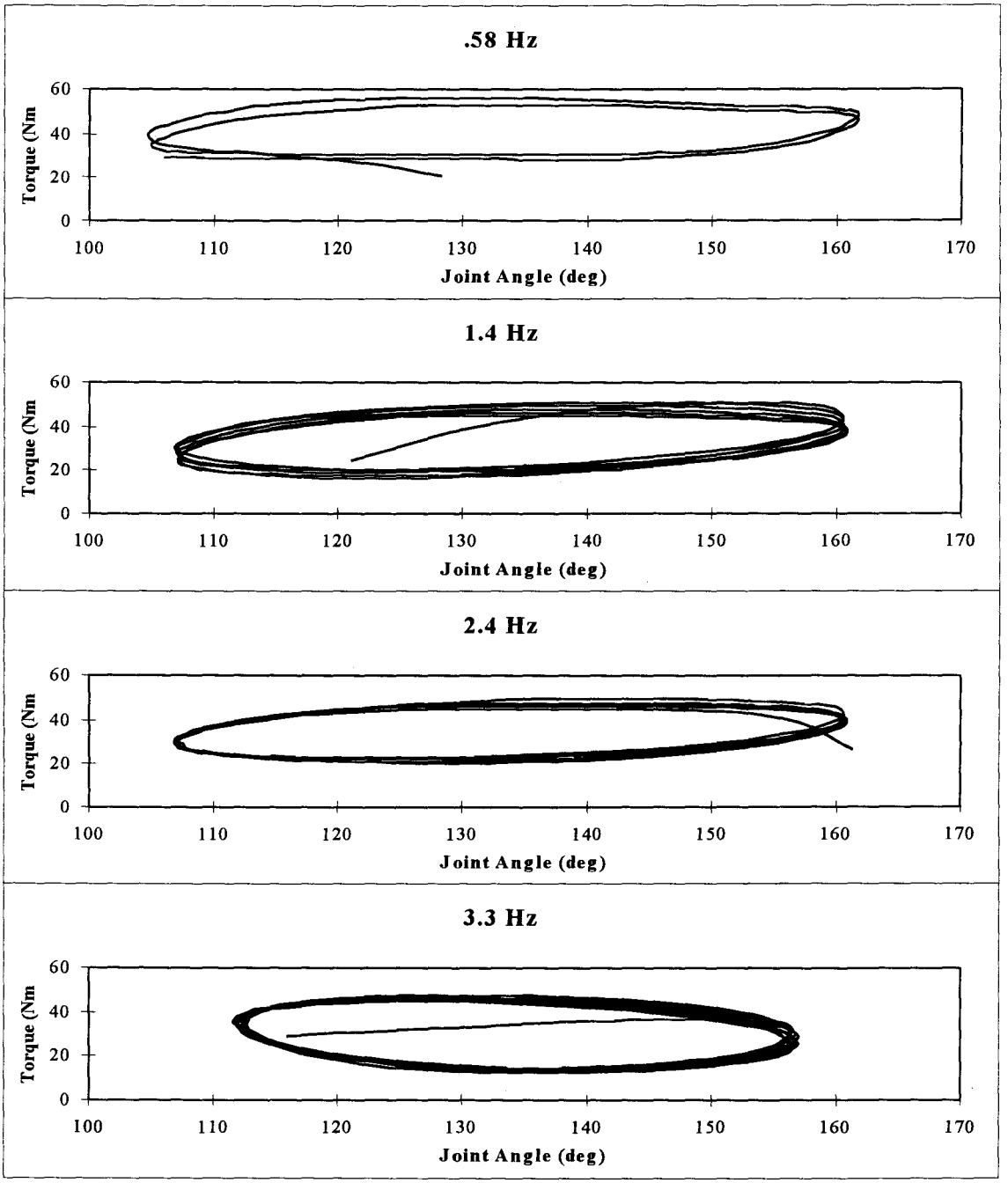
Probabilities for Post Hoc Tests

MAIN EFFECT: SPEED

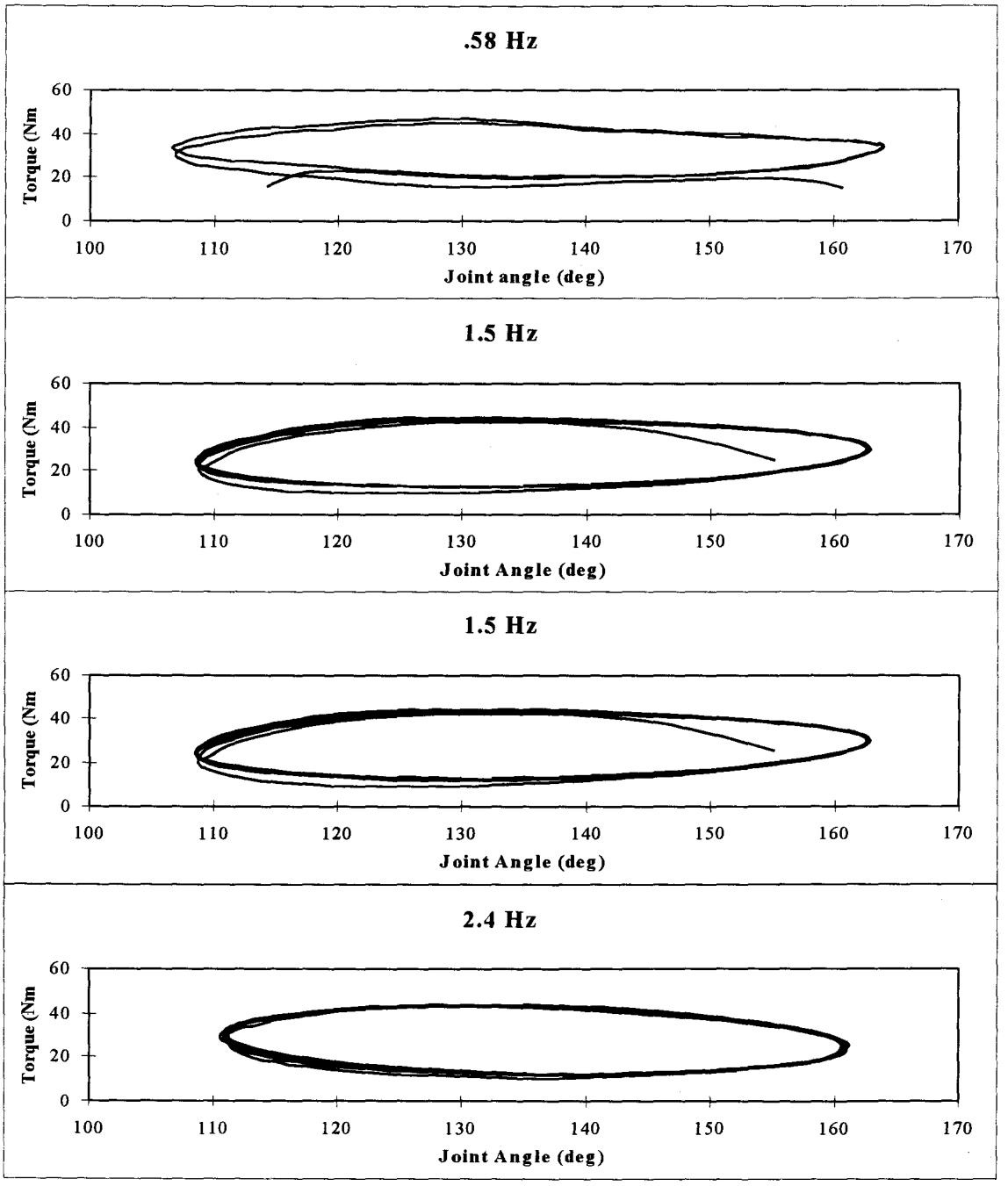
	{.58Hz}	{1.5 Hz}	{2.4 Hz}	{3.3Hz}
mean	12.16473	8.925596	7.168317	5.567019
{.58 Hz}		.000236	.000179	.000179
{1.5 Hz}	.000236		.021523	.000216
{2.4 Hz}	.000179	.021523		.038807
{3.3 Hz}	.000179	.000216	.038807	

Appendix C

Work loop data for subject B at four different frequencies of elbow flexion-extension (.58, 1.5, 2.4, and 3.3 Hz):



Work loop data for subject C at four different frequencies of elbow flexion-extension (.58, 1.5, 2.4, and 3.3 Hz):



Work loop data for subject D at four different frequencies of elbow flexion-extension (.58, 1.5, 2.4, and 3.3 Hz):