IMPLEMENTATION OF HISTORY DEPENDENT PROPERTIES IN A MODEL OF HUMAN SKELETAL MUSCLE

by

ANTHONY J. LEYLAND

B.Ed. Hons. (Physical Education) Exeter, England, 1979.

THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in the school

of

Kinesiology

SIMON FRASER UNIVERSITY

April 1986

APPROVAL

Name: ANTHONY J. LEYLAND

Degree: Master of Science

Title of THESIS:

IMPLEMENTATION OF HISTORY DEPENDENT PROPERTIES IN A MODEL OF HUMAN SKELETAL MUSCLE

Examining Committee:

Chairman: M. Savage

Dr. A.E. Chapman

Senior Supervisor

Dr. T.W. Calvert

Vice-President

Research & information Systems

Dr. P. Vaughan

External Examiner

Dept. of Physiology U.B.C.

Date Approved: 12 March 1986

PARTIAL COPYRIGHT LICENSE

I hereby grant to Simon Fraser University the right to lend my thesis or dissertation (the title of which is shown below) to users of the Simon Fraser University Library, and to make partial or single copies only for such users or in response to a request from the library of any other university, or other educational institution, on its own behalf or for one of its users. I further agree that permission for multiple copying of this thesis for scholarly purposes may be granted by me or the Dean of Graduate Studies. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Title of Thesis/Dissertation:

IMPLEMENTATION OF HISTORY DEPENDENT PROPERTIES IN A MODEL OF HUMAN SKELETAL MUSCLE.

Author:

(signature)

ANTHONY LEYLAND

(name)

15/4/86

(date)

ABSTRACT

This thesis was designed to build upon an existing two-component model of muscle. The model has been used to simulate human muscular contraction. It was written in APL and performed on a digital computer using an iterative process.

Some modification of mechanical output is observed in human skeletal muscle which has undergone stretch. This phenomenon, called force enhancement (FE), was not included in the existing model. Consequently FE was investigated using existing data and those collected from additional experiments. The experiments were performed using one subject. Maximally contracting supinator muscles were stretched at various velocities by a free gravitational load.

Mechanical relationships for the contractile component (CC) and the series elastic component (SEC) of a two-element model were derived. These represented mechanical properties of a single equivalent muscle performing forearm supination for this subject. The phenomenon of FE was then incorporated into the model. Hotelling's t^2 test for matched pairs revealed no significant difference ($p<0.01$) between experimental and model output.

The analyses performed in investigating human eccentric contraction centred on the role of the CC and SEC in a two-element model. Simulations of muscle stretch showed that no CC stretch occurred in the model despite the presence of FE in the experimental results. Because only SEC stretch occurred and the SEC is considered to be a passive element not subject to FE, a criticism of the two-element model is presented. As most of the elasticity within whole muscle resides in the cross-bridges, they are considered to be central in a 'geometric' theory of FE presented in this thesis.

The model is statistically accurate as a predictor of the mechanical response of human skeletal muscle during and after eccentric contractions but this application is purely practical. Despite being able to simulate experimental output, it is not possible to explain theoretically the phenomenon of FE within the framework of the two-element model.

ACKNOWLEDGEMNTS

I would like to thank Dr. Arthur Chapman for his continual guidance, support and good humour (he needed it!!) throughout the course of this work. Thanks also go to Dr. T.W. Calvert and Graham Caldwell for their advice and helpful suggestions.

DEDICATION

To my parents without whom I could not have completed this work.

Both physically and financially!

Also to my wife Anne.

"Isn't it amazing how much "mature wisdom" resembles being too tired?"

Robert Heinlein

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

PART A

INTRODUCTION

The mechanical relationships which are required to describe the mechanical output from human skeletal muscle have received much attention. These relationships are discussed in Chapter 1 of the literature review. Chapter 2 is concerned with the history dependent nature of these mechanical relationships which were at one time considered dependent only on instantaneous values.

The research into these mechanical properties has led to the development of many models of human skeletal muscle. Some models have used the observed non-linear mechanical relationships exclusively, others have attempted to linearise these relationships to ease the amount of computations required, while others have attempted to model the muscle incorporating cross-bridge kinetics and Ca^{++} release from the sarcoplasm. These models are discussed in Chapter 3.

Baildon and Chapman (1983a,b) have shown the applicability of using a two-element model of forearm supination based on Hill's two element model (1938). The model uses relatively small amounts of computer time to complete simulation of experiments on whole muscle. The contractile component is defined by four parameters, force, velocity, activation, and length. The series elastic element is defined by its torque-extension relationship. One criticsim of this model is that it calculates the mechanical output on the instantaneous values of the essential parameters and no account is taken of the way these instantaneous velocities and lengths are approached.

Experiments on isolated muscle preparations have identified the phenomenon of force enhancement (Sugi, 1972; Edman *et al.*, 1978a,b; Cavangna *et al.*, 1968). Briefly this means that the isometric force produced when a muscle is stretched eccentrically to a given length is greater than that obtained without stretch. The phenomenon of force enhancement has also been shown to be present in human muscle (Cavagna *et al.*, 1968; Chapman *et al.*, 1985; Thomson, 1983).

While the exact mechanism of force enhancement is unknown, it appears that the concentric work (when the muscle shortens) which a muscle can produce following an eccentric contraction (muscle stretch) increases due to:

1. the storage of mechanical energy in passive elastic structures,
2. changes in the contractile properties of the actomyosin cross-bridges,
3. the increased level of activation at the start of the concentric phase, and
4. if the neural connections are intact, reflex activity.

The phenomenon of force enhancement may be utilised in human sporting events where it has long been noted that skilled performers in almost all striking and throwing activities utilize a 'counter-movement' prior to the explosive forward motion. This phenomenon is known as a 'back-swing' or 'wind-up' and has received considerable attention (Asmussen and Sorenson, 1971; Asmussen and Bonde-Peterson, 1974; Bosco *et al.*, 1982; Chapman, 1980).

The use of a back-swing movement in so many sporting activities has led to much speculation as to its exact role. Research into the biomechanical and physiological aspects of stretch often focuses on the cross-bridges and the results of such studies may not explain all of the benefits of the back-swing seen in human activity. The human may benefit from a back-swing in such areas as timing (optimal performance), initial speed (as the contractile mechanism is fully activated by the prior stretch), and range of movement. Care must be taken therefore not to apply results from isolated muscle fibres to human skeletal muscle *in situ* without further research.

PART B

LITERATURE REVIEW

CHAPTER 1

MUSCLE PROPERTIES

When modelling human muscle it is imperative that the properties of the muscle are understood. The following sections include a discussion of the mechanical characteristics of human skeletal muscle.

1.1 Theories of Muscular Contraction

The fundamental property of muscle is that it can produce force. Pollack (1983) reviewed the large body of research work which concerns the most accepted theory of muscular contraction based on Huxley's (1957) sliding filament theory. This theory is often called the 'cross-bridge theory'.

Huxley (editors: Sugi and Pollack, 1979, p653) described the basics of the theory as follows:

> My ideas have been simply that myosin and actin are organised into separate filaments, and that these filaments move past each other during contraction, and that the force for this movement is produced by a projection on the myosin filament, and that this projection or cross-bridge interacts with actin, moves, and in some way pulls the actin along.

Pollack (1983) lists the structural and dynamic features of the theory and indicates the following four which are the essential framework.

1. Cross-bridges extending from thick filaments are structured so as to enable them to attach to complimentary actin sites on thin filaments.
2. Some bridges attach to actin while others continue to seek attachment sites.
3. Attached bridges undergo a structural-chemical transition so as to produce tension and then detach.
4. A contraction involves the buildup and subsequent decline of the number of such cycling bridges.

1.2 Force-Velocity Relationship

In isotonic (constant force) contractions the relationship between force applied and velocity of shortening was first investigated by Fenn and Marsh (1935). Using a viscosity hypothesis, a straight line relationship between these two variables was predicted. Instead they found that the relationship in isolated frog and cat muscles was clearly curved towards the origin (see Figure 1).

Hill (1938) conducted an investigation into heat production during isotonic contraction of frog muscles and showed that the shape of the force velocity curve was governed by the way in which energy was released during shortening. From thermal measurements he derived a simple equation (a rectangular hyperbola) relating the two variables. This equation is:

$$(P + a)(V + b) = (Po + a)b$$

where P = force of contraction; V = velocity of shortening; and a and b are constants. Po is the force of contraction at zero velocity (i.e. isometric force). This characteristic equation as Hill defined it fitted the mechanical data of Fenn and Marsh (1935) as well as the subsequent results of Katz (1939). Figure 1a depicts the relationship between force and velocity.

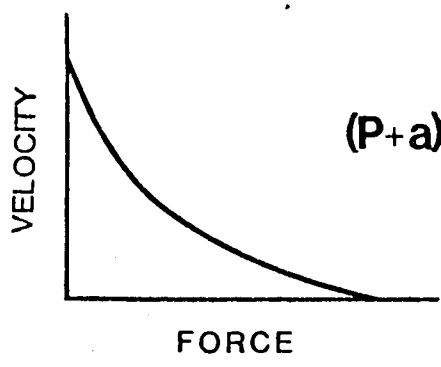
In 1950 Wilkie published the results of an extensive analysis of the force–velocity relationship in human forearm flexor muscles. He concluded that Hill's equation is very convenient for describing the force–velocity curves he obtained (Figure 1b), and made no attempt to identify the equation with any particular mechanism of contraction. This is an important point to make as throughout the experimental work of this thesis the relationships derived are descriptive and do not imply specific structures and mechanisms within human muscle. Wilkie (1950) accepted that the adoption of any other equation would not affect the predictions made by the force–velocity and tension–time curves since these are based strictly on the two hypotheses:

1) That the velocity of maximum contraction depends only on the tension. No particular mathematical function is specified.

2) That there is an elastic element in series with the contractile one.

It is clear that forces during eccentric contractions are much higher than isometric forces and that Hill's characteristic equation does not fit the eccentric portion of the force–velocity relationship (Katz, 1939; Joyce and Rack, 1969; Joyce *et al.*, 1969; Flitney and Hirst, 1978). This fact must be accounted for when modelling human muscle which can undergo stretching. Very little work has been done to define a standard form of the relationship between force and velocity when the CC is contracting eccentrically. Empirical curve fitting has been used to define the eccentric force–velocity relationship in the absence of a function which has a fundamental physiological basis. The analysis performed in this thesis will hopefully stimulate further study into defining this region of the force–velocity curve.

a) HILL'S EQUATION



$$(P+a)(v+b)=(Po+a)b$$

b)

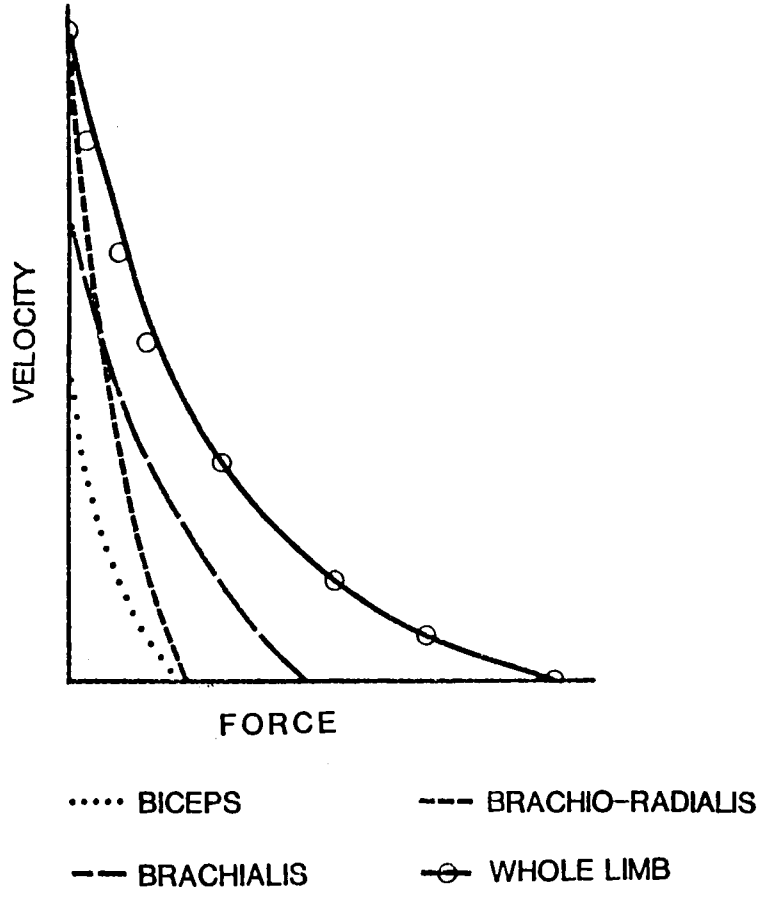


Figure 1. Force-velocity curves for:
a) Hill's characteristic equation, and b) human forearm flexion (Wilkie, 1950).

1.3 Series Elasticity

Wilkie (1950) showed that the rise in observed force during contraction of human muscle matched a theoretical curve which he calculated from an equation describing a model which included a CC in series with an elastic component. This elasticity can be considered to be 'lumped together' and is termed the series elastic component (SEC). It is important to note that the CC and SEC do not represent separate anatomical structures.

When a contraction is initiated the force produced by the CC stretches the SEC. The external load does not accelerate as quickly as the CC due to this extension of the SEC. Indeed the velocity of the CC is only the same as the external velocity of the muscle when the rate of change of SEC extension is zero. When the CC force drops as its velocity increases the SEC will recoil. During this period the external velocity is greater than the CC velocity .

It is important when visualising the SEC to realise that unlike a regular spring the SEC does not always experience a recoil after it has been stretched. As most of the series elasticity within a muscle resides in the cross-bridges, a muscle that relaxes after stretch will not experience SEC recoil. This is because despite having stored elastic potential energy the cross-bridges will simple detach when deactivated (Hill, 1950).

1.4 Parallel Elasticity

Resting muscle is elastic and can only be stretched by the application of force. Wilkie (1976) reviewed studies that show the presence of an elasticity in parallel with the CC. This is known as the parallel elastic component (PEC). The site of the PEC has been considered as the connecting tissue surrounding and invading whole muscle and the sarcolemma in single fibres. Wilkie (1976) reports that the PEC can develop considerable forces when isolated muscles undergo large changes in length.

To what extent the PEC is significant in human muscle is an important consideration, and Chapman (1975) and Chapman *et al.* (1985) provided experimental evidence which suggests that the PEC is an insignificant contributor to force throughout almost the entire range of human elbow flexion and forearm supination. Wilkie (1950) demonstrated that the PEC was not a significant factor in elbow flexion between 40 to 120 degrees.

1.5 Force-Length Relationship

The isometric tension which a muscle can exert varies with its length. Isometric tension (Po) appears in the characteristic equation as a constant, so this equation can be expected to apply over only a limited range of muscle length.

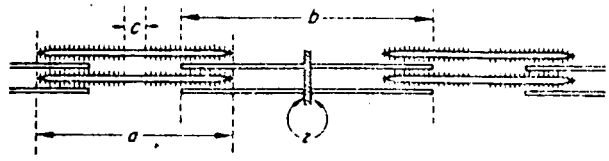
Experiments on isolated fibres give a clear picture of the relationship between length and tension (Gordon *et al.*, 1966). Tension is zero at extremely long sarcomere lengths ($\geq 3.65\mu m$) because no cross-bridges can attach to sites on the actin helix. At very short sarcomere lengths ($\leq 1.65\mu m$) there is an overlap of actin filaments causing some cross-bridges to become attached to inappropriate actin filaments with consequent lack of tension. Further, since the myosin filaments are squashed against the z-bands, cross-bridges at the ends of the filament become ineffective. Figure 2 shows this relationship.

In human skeletal muscle *in situ* the relationship between isometric tension and length is more complicated. The physiological range over which the muscle can shorten or lengthen is limited by the joint range of movement. Furthermore, every movement involves more than one muscle, and each muscle used would probably be on a different portion of its force-length curve. Figure 3a shows results from Wilkie (1950) for human forearm flexion and Figure 3b graphs results from the data of Chapman *et al.* (1985) for human forearm supination. As expected, these relationships do not resemble the data obtained from isolated preparations (Figure 2).

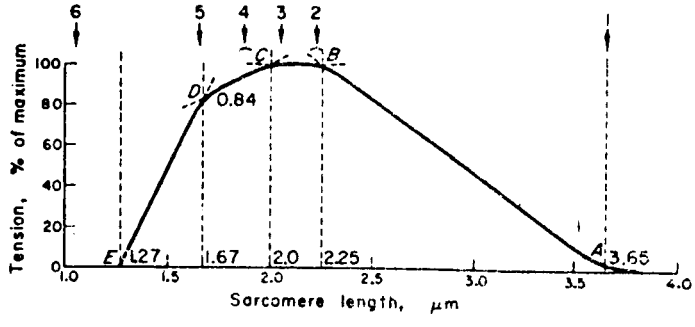
1.6 Activation

In response to a stimulus the CC of a muscle is activated. It is important that the terms activation and stimulation are defined in any discussion of this nature as confusion is possible. Stimulation of a muscle is the input. In isolated preparations it is the level and frequency of the applied current and can be measured directly and accurately. In human skeletal muscle *in situ* it is the subjective level of the input, i.e. the fraction of maximum effort which the subject used. During experiments for this thesis the effort, and therefore the stimulation, were maximal. 'Activation' refers to the number of motor units activated and is the result of stimulation. Activation is not directly measurable in either isolated preparations or muscle *in situ*.

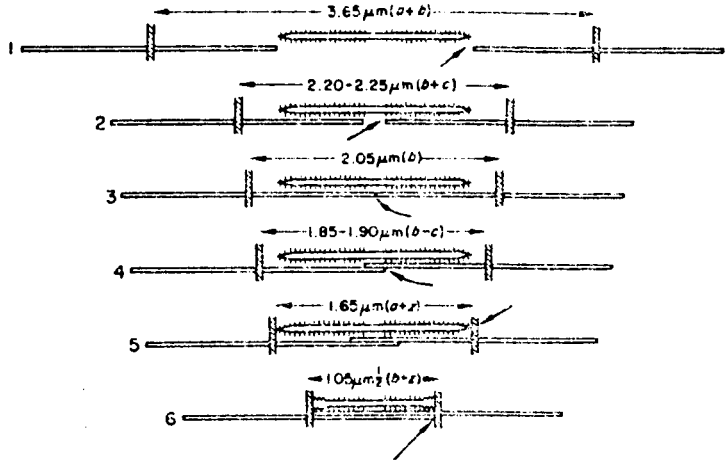
The above studies on the force-velocity, and force-length relationships of the muscle used a constant activation (usually maximal). This was so because it was realised that there was a relationship



a) Standard filament length



b) Tension-length curve



c) Critical stages of overlap

(#'s correspond to graph b)

Figure 2. Force-length relationship in isolated muscle fibres. Adapted from Gordon, Huxley and Julian, (1966).

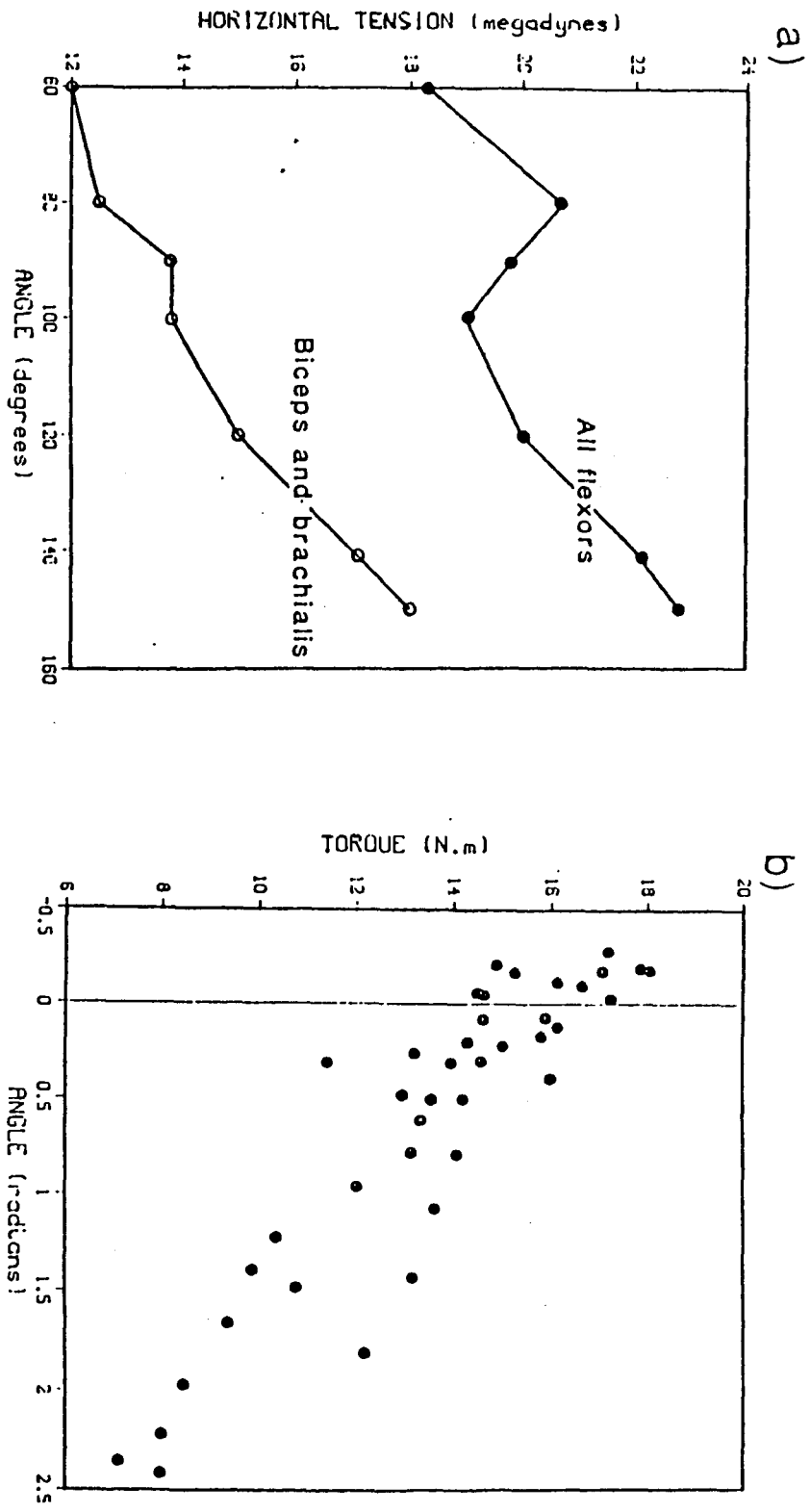


Figure 3. Force length relationship in human muscle.
a) Elbow flexion (Wilkie, 1950), and b) Forearm supination (Chapman *et al.* 1985).

between force produced and activation. Parmley *et al.* (1970) investigated force–velocity relationships for soleus muscle of the rat and showed that in the early stages of isometric contractions from rest, low forces were produced with a low velocity of shortening. They attributed this result to the fact that the muscle takes a finite time to become fully active. Fenn (1938) calculated that the activating input to the muscle took about 40 msec. to reach its peak.

In isolated preparations and in intact muscle *in situ* the force produced is known to be related sigmoidally to the rate of stimulation (Rack and Westbury, 1969; Julian and Sollins, 1973). Activation also appears to be dependent on the muscle length (Rack and Westbury, 1969; Lambert *et al.* , 1979). Muscle fibres are better activated at longer muscle lengths with the same level of stimulation.

Chapman (1985), in reviewing the mechanical properties of human muscle, notes that small motor units of the slow–twitch variety are recruited first followed by those of increasing diameter (fast–twitch), and deactivation occurs in the reverse order. Thus he points out that any measure of activation which is based upon numbers of active motor units and their frequencies of firing will exhibit a complex relationship to force produced. Similar complex effects of activation will be seen on the force–velocity relationship.

Attempts to monitor activation in human muscular contraction have relied heavily upon recordings of the electromyogram (EMG). Bigland and Lippold (1954a) demonstrated that a family of force–velocity relationships may be plotted for different levels of the EMG. These curves all met at the point of zero force where the maximum velocity of shortening was considered to be constant irrespective of activation level. Work by Petrofsky and Phillips (1980) has suggested that the maximum velocity (V_{max}) decreases with decreasing numbers of motor units being recruited. As it is impossible to achieve zero inertial load against a muscle, the muscle itself providing some inertial resistance, Petrofski and Philips (1980) have extrapolated their force–velocity curves to calculate V_{max} at zero force. It would therefore be difficult to model any variation in V_{max} as the error in their results is difficult to evaluate.

In eccentric contractions the forces produced are greater than those obtained during concentric contractions. Bigland and Lippold (1954b) further illustrated that greater forces were obtained in eccentric compared with concentric contraction at a given velocity and level of EMG. These results are supported by the work of Komi (1971, 1973) and Chapman and Calvert (1979). Thomson (1983) found no significant difference in EMG levels in forearm supinators at different velocities and amplitudes of

stretch.

Activation of a muscle spreads inwards from its surface, probably along the system of transverse tubules (Huxley and Taylor, 1958). Rack and Westbury (1969) suggest that the geometry of the tubules may be altered at different muscle lengths thus affecting the spread of the depolarising current. They suggest that at shorter fibre lengths the tubules may become thicker and offer higher resistance. This could account for the decline in activation at short muscle lengths shown by Rack and Westbury (1969) and Lambert *et al.* (1979). Whatever the mechanism of activation it is clear that the surface electromyogram could provide a misleading picture. Invasive EMG probes may affect the muscles ability to contract, and can cause pain when used in muscle *in situ* (Thomson 1983).

Efforts to reveal the state of activation of the muscle by appropriate treatment of the EMG continue, but many researchers consider that any estimate of this kind will be very approximate (Calvert and Chapman, 1977; Chapman and Calvert, 1979; Grieve and Cavanagh, 1974). To overcome this problem a straight line relationship between activation and force ouput is used in the present model. This is not meant to represent the actual mechanism of activation, but unless activation of a muscle can be obtained from some independent physiological recording (other than either force output or EMG) the problem of defining the relationship will not be solved.

CHAPTER 2

HISTORY DEPENDENT PROPERTIES OF MUSCLE

Pollack (1983) states that one assumption inherent in the cross-bridge theory is that the probability of attachment of a bridge should be independent of its previous cycling history. Attachment should depend only on local instantaneous environmental conditions, not on the pathway by which those conditions have been approached. This condition of the theory was examined in the prior discussion where the mechanical output of the muscle was discussed in terms of instantaneous parameters such as CC length, velocity and activation.

This 'history independence' requires that steady state isometric tension generated at a given sarcomere length should not be altered by the manner in which the muscle arrived at that sarcomere length. A classic experiment by Abbott and Aubert (1952) shows that this prediction is not valid. When muscle is stretched to a given length during contraction, the steady tension is greater than if the muscle had remained at that length throughout the contraction. This was later termed 'force enhancement'. Conversely, if the muscle is allowed to shorten to the prescribed length during contraction, it develops less tension than if it had been at the final length throughout contraction. This is now known as 'mechanical deactivation' and has been confirmed by Edman (1975).

Further evidence of the history dependent nature of the CC is shown when after stretch (eccentric contraction) at a constant velocity the force output of a muscle, which has previously been higher than the isometric value at that length, decays to values below the isometric value at that length. Griffiths *et al.* (1983) did work to suggest that this phenomenon was due to cross-bridge slippage where the myosin bridges detached and quickly reattached further back on the actin helix thus causing a reduction in tension. Flitney and Hirst (1978) showed that the force-length graph of a muscle undergoing stretch was clearly discontinuous. They identified a point where the rise in tension, due to stretch and longer muscle lengths, decreased markedly due to cross-bridge slippage. It is not clear how applicable this work is to human skeletal muscle but it does indicate that the CC output appears to be dependent on more than just instantaneous parameters.

The results of Abbott and Aubert (1952) have been supported by the literature (Edman, 1975, 1980; Edman *et al.*, 1978a,b; Sugi, 1972; Cavagna *et al.*, 1968) and it is clear that isometric tension depends on mechanical history, and that the dependence is systematic and predictable in isolated muscle

preparations.

Although force enhancement has been demonstrated to be present in human skeletal muscle *in situ* (Cavagna *et al.*, 1968; Thompson, 1983; Chapman *et al.*, 1985), the exact effect of amplitude and velocity of stretch, and the rate of decay of the 'excess tension' is not clear. It is this problem and the modelling of its solution which are addressed in this thesis.

2.1 Evidence of increased concentric work following stretch.

As early as 1885 Marey and Demeny noted that when two vertical jumps are performed in succession the second jump was consistently higher than the first. Anderson (1967), Cavagna *et al.* (1971), Assmussen and Bonde-Peterson (1974), and Komi and Bosco (1978) all report more favourable results in standing vertical jumps when the jump is preceded by a counter movement.

Skilled performers use a wind-up before striking or throwing movements and evidence suggests that they do this to increase the work done in the concentric phase of the movement. Asmussen and Sorenson (1971) showed an increase in concentric work through prior eccentric activity in tasks involving flexion and extention of the elbow. Similar results have been obtained by Chapman (1980), and Chapman *et al.* (1985) using supination of the forearm.

Chapman *et al.* (1985) have shown that after stretch of the forearm supinators, it is possible to obtain values of force greater than the isometric value at that length despite the fact the CC is shortening. Chapman suggests that the effect of prior stretch on a muscle is to 'shift' the force -velocity curve of the muscle towards higher force values for the same velocity. Whether the point of maximal velocity of shortening is affected as well is not clear at present.

2.2 Mechanisms of Force Enhancement

The mechanisms by which a muscle is able to produce higher tensions after stretch is still the subject of research. Studies on both isolated muscle preparations and whole muscle *in situ* suggest that a variety of factors may be responsible. These factors are discussed below.

2.2.1 Elastic energy storage within muscle

Cavagna (1977) reviewed the work concerning elastic energy in skeletal muscle. The basic premise is:

> During negative work, when the muscle is forcibly stretched, mechanical energy is absorbed by the muscle; this energy can be dissipated into heat or it can be stored within the muscle. In turn, the mechanical energy stored can be subsequently degraded into heat (e.g., if the muscle relaxes) or reutilised during a subsequent active shortening of the muscle. In this last case the positive work done by the muscles during shortening would derive from (a) the transformation of the chemical energy into mechanical work by the contractile machinery and (b) the mechanical energy previously stored within the muscle and tendon during the phase of negative work.

Cavagna *et al.* (1976) showed that the mechanism of running is more suitable than that of walking for the storage and utilisation of mechanical energy by muscles. This storage of energy in the muscle's passive components, first discussed by Hill (1950), was found to increase linearly with speed in level running. Cavagna and Kaneko (1977) and Cavagna *et al.* (1976b) suggest that since efficiency of concentric work increases with speed, the excess concentric work is primarily the result of stored mechanical energy rather than the transformation of chemical energy by the CC.

Only certain acitivities, where contracted muscles are forcibly stretched before shortening, will benefit from a storage of mechanical energy. For example, cycling, rowing and other cyclic activities where the concentric phase is preceded by a relaxation of the muscles, do not benefit from stored mechanical energy. As the site of much of the SEC is located in the cross-bridges any prior stretch stores mechanical energy in these bridges. Therefore, any subsequent relaxation of the muscle would prevent this mechanical energy from being recovered in any subsequent concentric contraction.

Earlier studies by Fenn (1930) and by Elftman (1966) argued that the usefulness of muscle elasticity was negligible because the muscle cannot retain this store of energy without continuous contraction. The muscle therefore performs work to maintain the potential energy stored by the stretch. Cavagna (1977) suggests that the recovery of mechanical energy, from this stored potential energy, more than compensates for the chemical energy spent to maintain tension during stretch. He argues that active stretch temporarily modifys muscle compliance enabling a greater amount of mechanical energy to be recovered for a given drop in force during shortening (Cavagna and Cittero, 1974).

The recovery phase is of limited duration however, since as Fenn (1930) suggested, the elastic energy is continually being dissipated as heat. Cavagna (1977) reports that during successive deep knee bends, as the length of the interval was decreased, the efficiency, measured by oxygen uptake,

increased. This they concluded was due to more of the elastic energy stored during stretch being recovered during subsequent shortening. Since both the storage and recovery of this mechanical energy are time dependent, Asmussen and Sorenson (1971) recommend that in athletic activities with a wind-up, the delay between the eccentric and concentric phases be minimized.

2.2.2 Re-synthesis of ATP during negative work

The theories put forward by Fenn (1930) suggested that the prior eccentric contraction produced a shift in the creatine kinase pathway favouring the synthesis and accumulation of adenosine triphosphate (ATP). However, Cavagna *et al.* (1968) questioned this hypothesis indicating that only when energy stores fall below a critical level, as in fatigue, does potential chemical energy become a limiting factor. It therefore seems unlikely that force enhancement is due to any increase in ATP synthesis.

Curtin and Woledge (1979) showed that ATP was consumed at similar rates for both eccentric and concentric contractions, but the work done in concentric contractions following an eccentric contraction was greater for a given amount of ATP consumed than in a concentric contraction from rest. As the splitting of ATP is the source of energy for the contractile process and is proportional to the number of conventional cross-bridge cycles, the increase in tension after active stretch cannot be explained by an increase in the number of cycling cross-bridges.

2.2.3 Potentiation of the contractile component

Any suggestion that the benefits of a wind-up are based entirely on storage of elastic energy within the series elastic structures of the muscle would be misleading. The exact role played by the eccentric contraction in arresting the counter-movement of a back-swing is complex. Following stretch, the recoil of passive elastic structures after maximum torque has been reached allows the CC to shorten more slowly at a lower velocity than that of the load. As previously discussed, if the CC can shorten more slowly it will be able to produce more force. Also the back-swing will fully activate the muscle before the concentric phase occurs and this is beneficial to the athlete in terms of acceleration. Undoubtedly the above factors combine to allow the athlete to 'fine tune' his muscular response to achieve optimal performance.

While all the above observations remain plausible there is no doubt that at the sarcomere level there is a portion of the work output after stretch which is due to a potentiation of the CC (Cavagna *et*

al., 1968; Chapman *et al.*, 1985). Cavagna and his colleagues argue that the passive elastic elements within muscle are unable to store enough mechanical energy, even when coupled with changes in compliance, to account for all of the increased positive work. Single muscle fibres, lacking much of their connective tissue, also showed a similar shift in the force–velocity curve (Edman *et al.*, 1978a,b). It would appear therefore that in addition to the storage of mechanical energy in the SEC some other mechanism is involved. The work presented in this thesis also supports this conclusion. Guth *et al.* (1977) showed that there was some altered cross–bridge activity present with excess tension after stretch. A possible explanation for this 'potentiation of the CC' is presented in the discussion section of this thesis.

CHAPTER 3

MUSCLE MODELLING

Many models of human skeletal muscle have been proved useful within limited application. Some models have approximated the fundamental mechanical relationships discussed in Chapter 1 by assuming them to be linear. Despite some success (Bawa *et al.*, 1976; Houk, 1963), these models have been criticised as inaccurate in general applications (Chapman and Harrower, 1977; Baildon and Chapman, 1983a,b).

After A.F. Huxley's (1957) mathematical formulation of the sliding filament theory a number of increasingly sophisticated kinetic models have appeared. These models attempt to predict the mechanics and energetics of muscle on the basis of chemical interactions between actin and myosin at the cross-bridge level (T.L. Hill *et al.*, 1975; Hatze, 1981; Zheng *et al.*, 1984). Unfortunately these kinetic models are too complex to be used directly for macroscopic descriptions of whole muscle. They perform a large number of calculations even for the simplest of experiments and require numerous muscle parameters to be specified as input, some of which are not directly measureable experimentally. Zheng *et al.* (1984) proposed a model of skeletal muscle activation which incorporated the size recruitment principle and muscle spindle reflex activity. However, the stimulus rate from the CNS, the number of motor units, and the amount of free calcium in the muscle are required as inputs.

Zahalak (1981) points out that:

> Such elaborate computations are justified for the purpose of interpreting precise experiments on isolated muscle fibres in order to test hypothises concerning molecular contraction mechanisms, but would be unwarranted and prohibitive for studies of limb motion where several muscles, length-dependent behaviour and time-varying stimulation, as well as passive visco-elastic properties play a role.

The classic two-element model of muscle introduced by A.V. Hill in 1938 has proved very useful in predicting mechanical output from whole muscle. Both elements, the CC and SEC, were assumed to display non-linear mechanical properties. Figure 4 shows Hill's model and the relationships needed to define each component.

Using a two-element model similar to Hill's, the procedure outlined by Baildon and Chapman (1983a) steers a middle course between over-simplifying the model by using linear relationships and over-complicating the model by basing it on molecular kinetics at the cross-bridge level. This model, which has been rewritten and expanded in this thesis, although computationally easier and intuitively

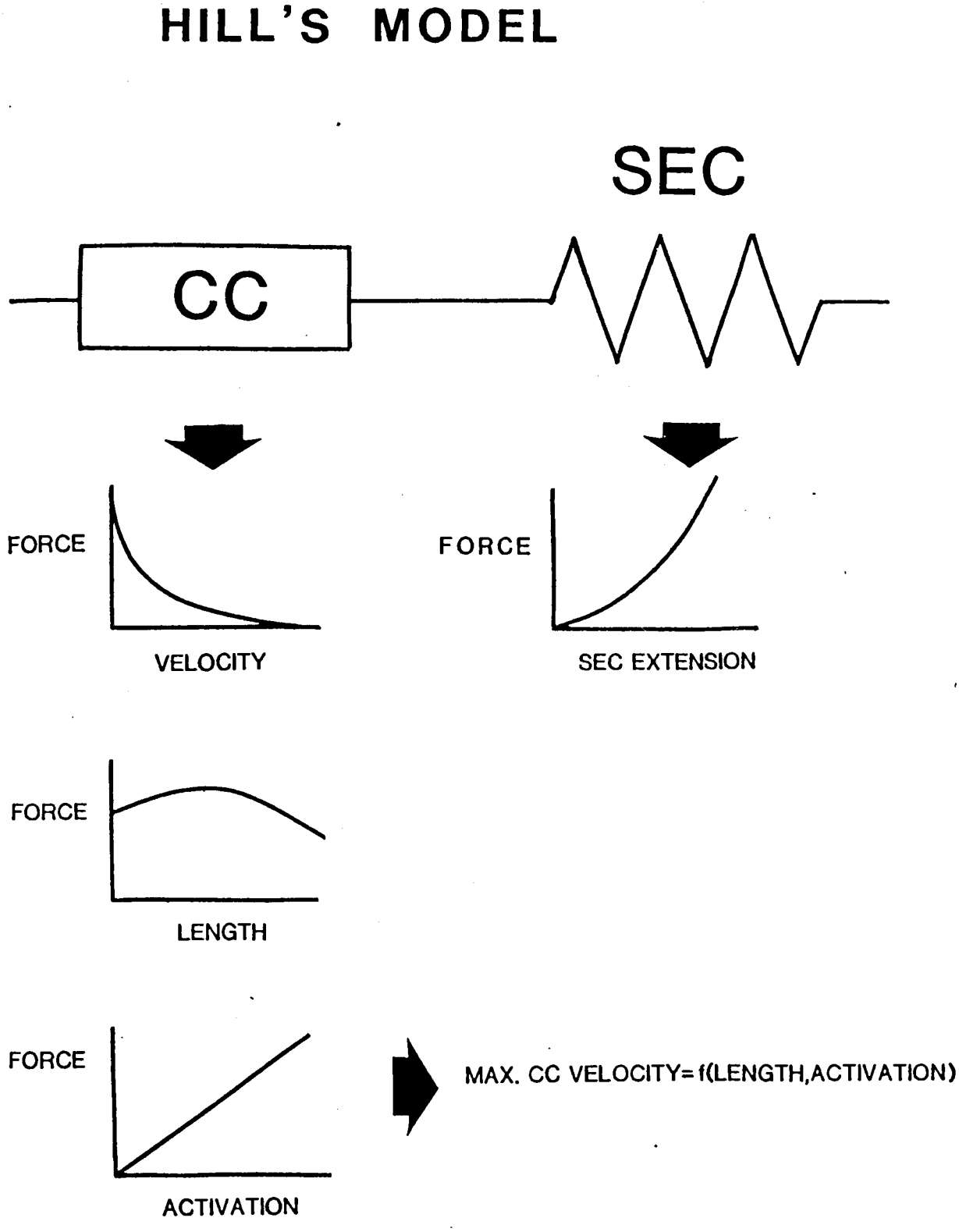


Figure 4. Hill's model of muscle. Parameters used to describe each component are included.

more attractive than many others, provides an accurate prediction of muscle behaviour. The model has been validated by comparing its output with the output from a variety of experiments. Although the CC and SEC do not represent actual anatomical structures within muscle, accurate prediction of mechanical output can be considered a strong claim to validity. As an educational tool it would be better to keep the model as simple as possible. This is a further advantage of the two–element model.

Even when more extensive relationships are used to derive a model it is unlikely that the author will claim that his model describes the exact anatomical structure of the muscle. Hatze (1981) uses 51 equations to define his muscle model. Despite this detailed analysis he himself admits that the hypothesis (outlined in Hatze, 1973) is "highly speculative and that it is unlikely that the model describes an actual contractile mechanism". Chapman (1985) further explains that even when accurate descriptions of the CC at the sarcomere level are utilized, as in some models based on kinetic theories, the exact function of the structures linking the CC and its external environment is unclear.

However, the two component model is not without its critics. Zahalak (1981) proposes an approximation for kinetic theories of muscular contraction. He claims that his approach avoids the problems inherent in the detailed kinetic theories while exhibiting more realistic behaviour than the classic two–element model. He criticizes Hill's model in part due to experimental evidence that a muscles properties are dependent on its prior mechanical history. These properties have been discussed in Chapter 2.

The two–element model critized by Zahalak (1981) has a CC component whose velocity is dependent upon force and activation. This does not take into account the force length relationship of the CC which has been clearly demonstrated. In addition history dependent properties have now been incorporated into the model in this thesis.

3.1 Modelling the Phenomenon of Force Enhancement

Many models of whole muscle do not include FE. Perrynowski (1982) produced a physiological model which simulated normal human walking. He approximated the eccentric portion of each muscles force–velocity relationship from the work of Fitzhugh (1977). It is accepted that the relationship includes a plateau where any further increase in stretch velocity does not result in an increase in force. This according to Perrynowski is approximately 1.25 Po, where Po is the maximal isometric force the

muscle can produce at that same length. Values for this maximum force level in the eccentric region are dependent on muscle temperature and fibre composition. Flitney and Hirst (1978) recorded this plateau to be between 1.58 to 1.19 Po for frog sartorius at 0 to 30° Celsius respectively. Mouse soleus at 23° Celsius had a value of 1.7 Po compared with 1.25 Po for frog sartorius at the same temperature.

Whenever the eccentric portion of the force-velocity relationship is studied the phenomenon of FE is included in the results. This is so because by definition it is not possible to observe directly an 'unenhanced' force-velocity curve in the eccentric region. Therefore, models like Perrynowski's (1982) are taking FE into account to some extent. In his simulations the velocity of stretch during any eccentric contraction was not high and any error due to the FE being underestimated would be small.

Using one force-velocity curve does not present problems when observing forces during stretch (as FE is present in the experimental data used for the simulations). Indeed it has been argued that the elevation of this portion of the force-velocity curve can be modelled with a force-velocity relationship which is discontinuous when moving from concentric velocities to eccentric velocities. This discontinuity is due to the F-V curve in the eccentric portion including FE and in the concentric portion being affected by mechanical deactivation (Edman, 1975). This hypothetical curve would not take into account the fact that force is elevated in the concentric region if the contraction was preceded by an eccentric phase. It is this situation that not directly modelling for FE could cause larger errors.

Crowe (1970) used a three-component model where the elastic components (SEC and PEC) had variable moduli of elasticity. The third component was a viscous component rather than a force generator. The effect of having a force generator, which is obviously a fundamental property of muscle, is "produced by postulating that the unstretched lengths of the elastic components are reduced as a result of stimulation" (Crowe 1970). This approach to predicting tension during stretch of the active and passive muscle was only successful at a qualitative level.

Despite not being applicable to human muscle *in situ* the option of having a variable modulus of elasticity for the SEC was investigated in the present adaptation of Chapman and Baildon's model. This is discussed in more detail in following sections.

An approach by Hatze (1981) used Ca^{++} release into the sarcoplasum as a trigger for cross-bridge interaction. Despite accurate results these attempts are not transferable to a model of human skeletal muscle *in situ* without prohibitive computer costs. Again data was obtained from experiments *in vivo*

and the validity of applying this work to model muscle *in situ* would be questionable. An attempt to study FE *in situ* in this work was undertaken and further work in this area is needed if the exact role FE plays in normal human activities is to be understood.

Modelling to fit experimental results can provide a model which is empirically accurate and often versatile. It does not however indicate the mechanism responsible for the observed behaviour. Despite this limitation the model can be used as a tool to test the applicability and accuracy of a theory. Certainly the attempt to include FE into a simple two-element model has highlighted some theoretical problems of such a model.

PART C

THEORETICAL AND EXPERIMENTAL WORK

Prior to the present work a mathematical model based on Hill's two element model was in existence. The model was able to calculate mechanical output from human skeletal muscle during eccentric contractions but did not incorporate force enhancement. This led to a systematic error in calculating muscle output under such conditions. As the model is used extensively in the teaching of an undergraduate course and in the analysis of human muscle mechanics it required updating.

The first Chapter in this section describes the experimental work undertaken as well as a brief description of the methods of Chapman *et al.* (1985) and Thomson (1983). This will aid the reader to gain a fuller understanding of the theoretical analysis. The remainder of the study is presented in chronological order.

CHAPTER 1

EXPERIMENTAL WORK

As discussed previously the literature does not indicate any standard equation which describes the eccentric portion of the force–velocity curve. The following experimental procedures were designed to obtain peak and trough torques ($d\tau/dt=0$) during an eccentric contraction against a free weight. At these peaks the velocity of the CC is the same as that measured externally and yields points on the CC force–velocity relationship.

1.1 Subjects and methods

Data using AC as a subject was obtained. All trials required supination of the right (dominant) forearm in a standing posture with the upper arm vertical, the forearm horizontal and a broad strap around the shoulders to prevent lateral motion of the torso. Position of the elbow was monitored by means of a horseshoe–shaped cup surrounding but not touching the elbow, the movement of which discounted a trial. Supination was performed with maximal voluntary effort under either isometric, or dynamic conditions against a gravitational load.

1.2 Apparatus

The subject had to grasp a handle which was instrumented with strain gauges to measure torque (τ) and which was fixed to an axle colinear with the forearm. Angular displacement (θ) of the forearm was monitored by means of a POLGON light goniometer (Grieve 1969) with a moving sensor fixed to the distal end of the axle. The distal end of the axle carried a metal flywheel to which various masses could be attached by means of a chain to produce a variety of torques in the direction of forearm pronation. Underneath the weights were a series of foam pads which gradually decelerated the masses. Another chain linked to a mechanical trigger allowed the gravitational load to be fixed in position so that no torque in the direction of pronation would stretch the subjects muscles before isometric torque had been reached. Release of the load was obtained by movement of the trigger. An acclerometer was mounted on the handle to register tangential acceleration. Angular velocity (ω) was calculated from differentiation of the displacement trace. Outputs from each transducer were amplified and sampled at 200 Hertz by means of a 12–bit A/D converter interfaced with an Apple II+ computer. The experimental apparatus is dipicted in Figure 5.

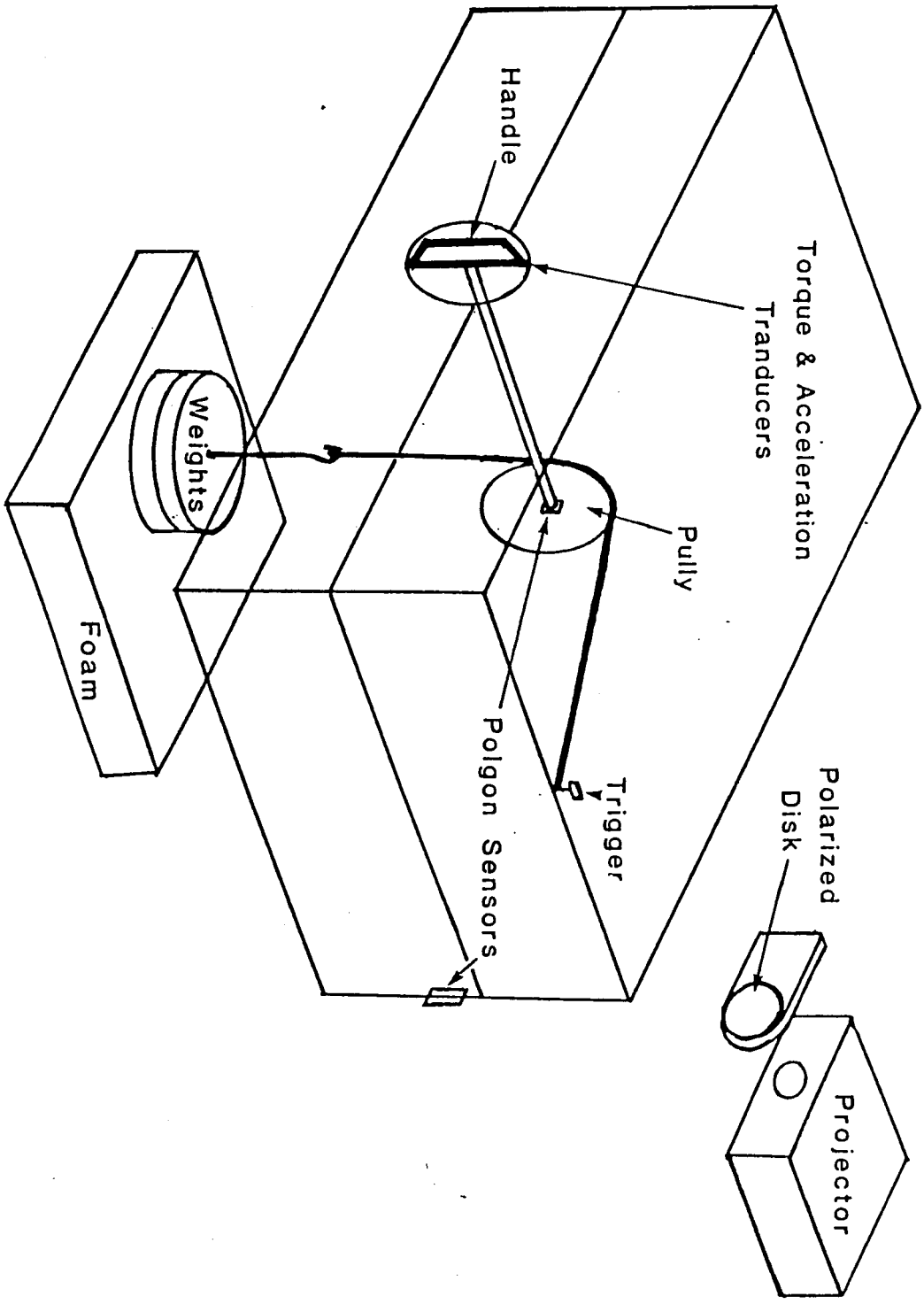


Figure 5. Experimental apparatus.

1.3 Calibration

By fixing the handle horizontally and then vertically the tranducers of angle and acceleration were given calibration inputs of 90 and 0 degrees and 0 $m.s^{-2}$ and 9.81 $m.s^{-2}$ respectively. A 1-meter bar fixed with its centre on that of the horizontal handle and a mass of 1 Kg. applied at one end gave a calibration input to the torque transducer of 4.905 Nm. No applied mass gave an input of 0 Nm. Calibration was performed before and after an experimental session. No large descrepencies ($\leq$3%) between these two calibrations occurred. Any small discrepencies were averaged to reduce errors.

1.4 Data Treatment

Angle of the handle was computed in radians with reference zero occuring with the handle horizontal (palm facing downwards). Output from the accelerometer was corrected for the gravitational effect and the resulting tangental acceleration was divided by radial distance of the accelerometer to yield angular acceleration. The latter was multiplied by moment of inertia of the forearm and added to the transducer torque to yield absolute torque produced by the muscles involved in supination. Both angle and torque were smoothed using a dual pass 3-point moving average. Angular velocity was subsequently obtained by finite difference differentiation of angle over two time intervals (dt= 10ms). The array of torque was scanned to identify peaks and troughs of torque (where its rate of change was zero) and their times of occurrence. Angular velocity and angle were obtained at the onset of the trial and at all peak and trough torques.

1.5 Procedures

Isometric contractions were performed by fixing the flywheel with a mechanical stop at various handle angles. Dynamic contractions began with an isometric contraction at a given starting angle whenever the subject was ready. When the subject felt that he had reached maximal isometric torque he signalled this fact to the experimenter who simultaneously triggered the release of the load and started the A/D sampling.

The load created too high an opposing torque for the subject to 'hold' resulting in a stretch of his maximally contracting supinator muscles. When the load came into contact with the foam the opposing torque was reduced causing the velocity of stretch to decrease. This initial rise and

subsequent decline in torque produced a peak torque during the stretch.

Each trial produced two or more points on the torque–time curve where $d\tau/dt=0$. This was because the load actually 'bounced' off the foam allowing the subject to move the handle in the direction of supination. The load subsequently fell again stretching the muscles once more. This produced a trough in the torque–time curve as the torque dropped during the brief concentric phase only to rise again when stretch reoccurred. This oscillation in the load displacement led to as many as 5 points being obtained per trial where $d\tau/dt=0$. As activation was maintained at a voluntary maximal level all these points are valid points on a $\tau-\omega$ relationship curve. Figure 6 is a ultra–violet recorder trace from a typical trial. This data will be referred to as the 'stretch data' in the remaining text.

1.6 Additional Data

Chapman *et al.* (1985) conducted an experiment into the mechanical output produced during forearm supination following stretch. The apparatus used was almost identical to that of the present study except that a purely inertial load was used (load supported). A series of isometric contractions was performed to determine the torque–angular displacement relationship for each of the subjects. Two more conditions were used; one, a concentric contraction from rest, and two, an eccentric contraction immediately followed by a concentric contraction. In the second condition the load was rotated in the direction of pronation and on a given command the subject had to arrest the loads momentum (eccentric work) and move it back in the opposite direction (concentric work). Both these sets of trials used maximal activation with various inertial loads and starting positions. Data from the first condition will be referred to as the 'rest data', and from the second condition as the 'back–swing data'.

Chapman and his colleagues studied the work performed over various time intervals. They had recorded the peak torques and it was therefore possible, without duplication of work, to obtain data that allowed calculation of the mechanical properties of a single equivalent muscle performing forearm supination for subject AC.

Many studies have controlled the force and/or velocity of contraction in human muscle. The very different force–velocity curves reported by Joyce and Rack (1969) and Joyce *et al.* (1969) could be due to an artifact of these controlled contractions. As the experiments of Chapman *et al.* (1985) are neither isotonic nor isokinetic it was considered important that the eccentric contractions in the

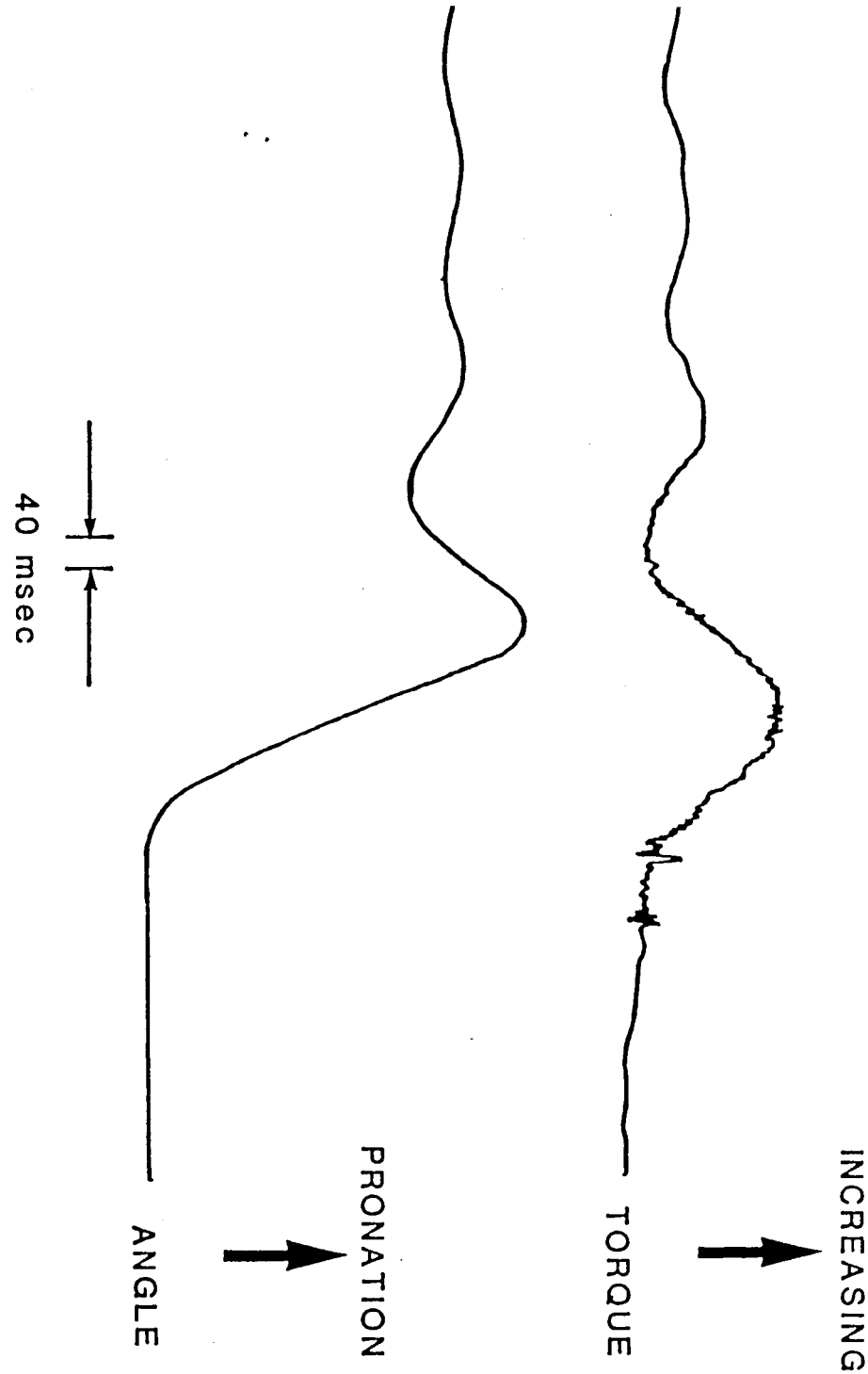


Figure 6. Ultra-violet paper recording of a typical experimental trial. A 40 msec time delay is present in the angle trace.

present study be similar.

In the dynamic contractions of the above experiments it is impossible to calculate decay time-constants of the torque due to the varying lengths of the muscle. Thomson (1983) conducted research into the stretch response of human skeletal muscle *in situ* using forearm supination. He controlled the velocity of stretch using a constant velocity device and his protocol had each subject perform contractions at a constant velocity. After stretch had ceased the subject continued to produce a maximal contraction so that the decay time-constant of the excess torque due to the stretch could be calculated. These were used in the present modelling application.

The remaining work is presented in chronological order. The results and analysis of the data from the experiment described above are included in a subsequent chapter.

CHAPTER 2

REWRITING AND REDOCUMENTING THE MODEL

The model is written in APL and is implemented on an IBM 4341 computer at Simon Fraser University, Burnaby, B.C., Canada. When a study of the original model was completed with a view to incorporating new features, it became clear that the program was poorly documented. Further scrutiny of the APL code showed the functions within the model were cumbersome, occasionally erroneous and without programming comments (see Gilman and Rose, 1976 for an introduction to APL).

To facilitate the updating of the model it became clear that the programme would have to be rewritten and redocumented. This required four major changes.

1. Rewriting the APL code in an efficient manner.
2. Writing intuitively separate functions which will help any future programmer to follow the sequential nature of the model and add further functions more easily.
3. Including an introduction and descriptive lines in every function to allow future programmers to follow each step involved.
4. Writing a detailed programmers guide to provide full information for anyone wishing to adapt the model in the future, and providing a brief user's guide to help students operate the model without encountering problems.

From the user's point of view the revised model appears basically similar to the initial version. Computer time required for an equivalent simulation is decreased however, and the addition of further mechanical properties and/or experimental options is much easier. No experimental option existed which allowed the muscle to work against a gravitational load. New functions were included which facilitated this option in combination with an inertial load.

CHAPTER 3

PRELIMINARY ANALYSIS

Prior to the investigation of the force enhancement in human skeletal muscle and the role it is likely to play in everyday sports activities, a series of studies were conducted to obtain the mechanical properties of the subject AC's forearm supinator muscles. Statistical analysis was also performed to validate that the assumptions being made in this investigation were correct, and that the inclusion of a back-swing significantly altered the torque output in the subjects trials.

As the experimental apparatus displayed in Figure 5 is designed to study forearm rotation, all units used in the following results and descriptions are rotational. These units could easily be replaced by equivalent linear units and the results of this thesis would bear comparison with other studies.

3.1 Torque-angular velocity relationship

Using the isometric contraction data from Chapman *et al.* (1985) the torque-angular displacement ($\tau-\theta$) relationship was calculated for the CC from isometric contraction trials for subject AC using a polynomial regression program (BMDP5R, University of California, 1981) to identify the best fitting polynomial. This data is shown in Figure 3b. This relationship was calculated to be a straight line, which does not represent a surprising result. Figure 7 shows the experimental data of Chapman *et al.* (1985), all points represent a datum from each trial where the rate of change of torque was zero. Positive values of angular velocity represent supination (concentric contraction) and negative values represent pronation (eccentric contraction). Figure 7a is data from rest and Figure 7b the back-swing data. All the data from contractions from rest (Figure 7a) were then used to calculate a relationship between P/Po vs CC velocity, where P is the observed peak torque and Po was the calculated isometric torque at the angle where this peak torque occurred. This normalising procedure is crucial so that the $\tau-\theta$ relationship does not alter the relationship between torque and CC angular velocity ($\tau-\omega$). This analysis was also conducted using polynomial regression.

It is important to realise that this normalising procedure, while reducing the errors possible in obtaining the $\tau-\omega$ relationship, is in itself only an approximation. This is because the $\tau-\theta$ curve obtained is a measure of the 'isometric torque-whole muscle length relationship' , not the torque-CC length relationship. For any muscle length measured externally (by handle angle in these experiments)

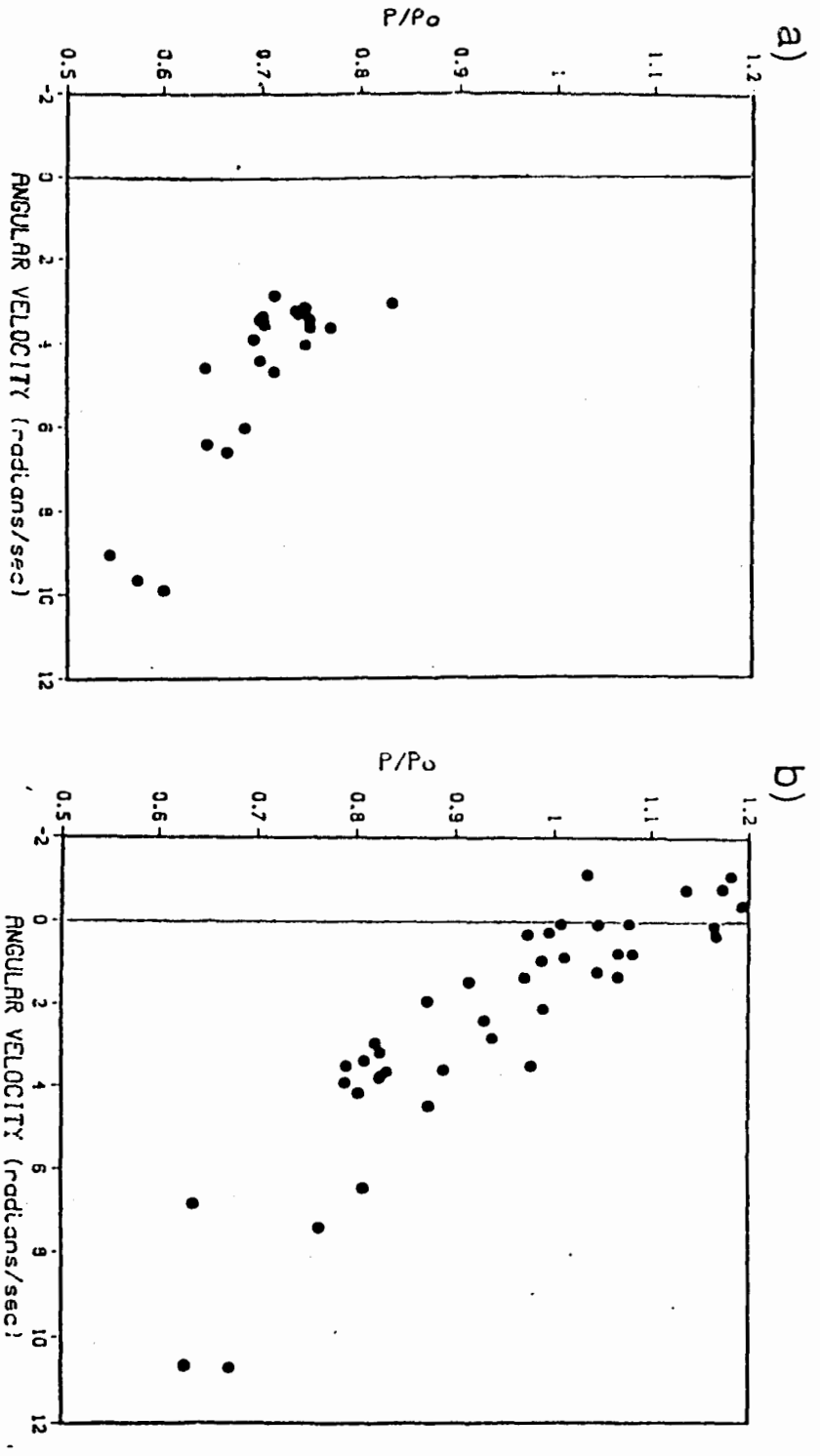


Figure 7. P/Po versus angular velocity for subject AC. a) Rest contractions, and b) backswing contractions.

the CC could be at various lengths due to the type of contraction. With the back-swing contractions the peak torques were higher than the isometric torques at similar lengths. If the torques are higher the SEC (which transmits the torque externally) must be at a greater length thus the CC would be shorter. To obtain the most accurate $\tau-\omega$ curve for a subject, each peak torque should be divided by the isometric torque at that CC length. This is impossible as the CC length can only be calculated after the $\tau-\omega$ curve has been obtained.

3.2 Series elastic component characteristics

Using the $\tau-\theta$ and $\tau-\omega$ relationships already calculated and the empirical torque-time and load velocity-time curves for each trial, the torque-SEC extension relationship was investigated. A torque -SEC extension curve for each data trial was calculated and despite being qualitatively similar there was too much variation to be able to obtain a statistically significant polynomial relationship (BMDP5R). Fifty-two torque-SEC extension curves were generated which precludes displaying them all on one graph. A sample of six curves is shown in Figure 8. These curves represent the extreme ranges observed.

One explanation for this variation is that the onset of each trial was identified, by the data collection program, as being after three consecutive torque readings had a rate of torque increase of 0.5 Nm./second. The problem with this method, in the back-swing trials, is that the subject often produces enough passive resistance to the stretch to initiate data collection early. In the rest trials the subject could anticipate the signal to go early and not continue with an immediate maximal contraction despite having produced enough torque to trigger collection. In these events the torque remains low, because the subject has not yet started his maximal contraction, and the angular velocity is calculated as being near maximal (from the fitted $\tau-\omega$ relationship). The SEC velocity is obtained by subtracting the velocity of the load from the CC velocity and is consequently also too high. As seen from Figure 8, this problem can produce some very varied results and is probably present to some degree in all trials.

The few trials that displayed the greatest SEC extension had no common parameters other than being back-swing trials, e.g. they were not all at extreme ranges of length. In the absence of any systematic variation these trials were discarded as outliers. The remaining trials still showed a considerable variation but 10 trials representing the 'mode' of the results were almost identical. As these trials lay in the middle of the range of results, near the 'mean curve', they were used to fit a

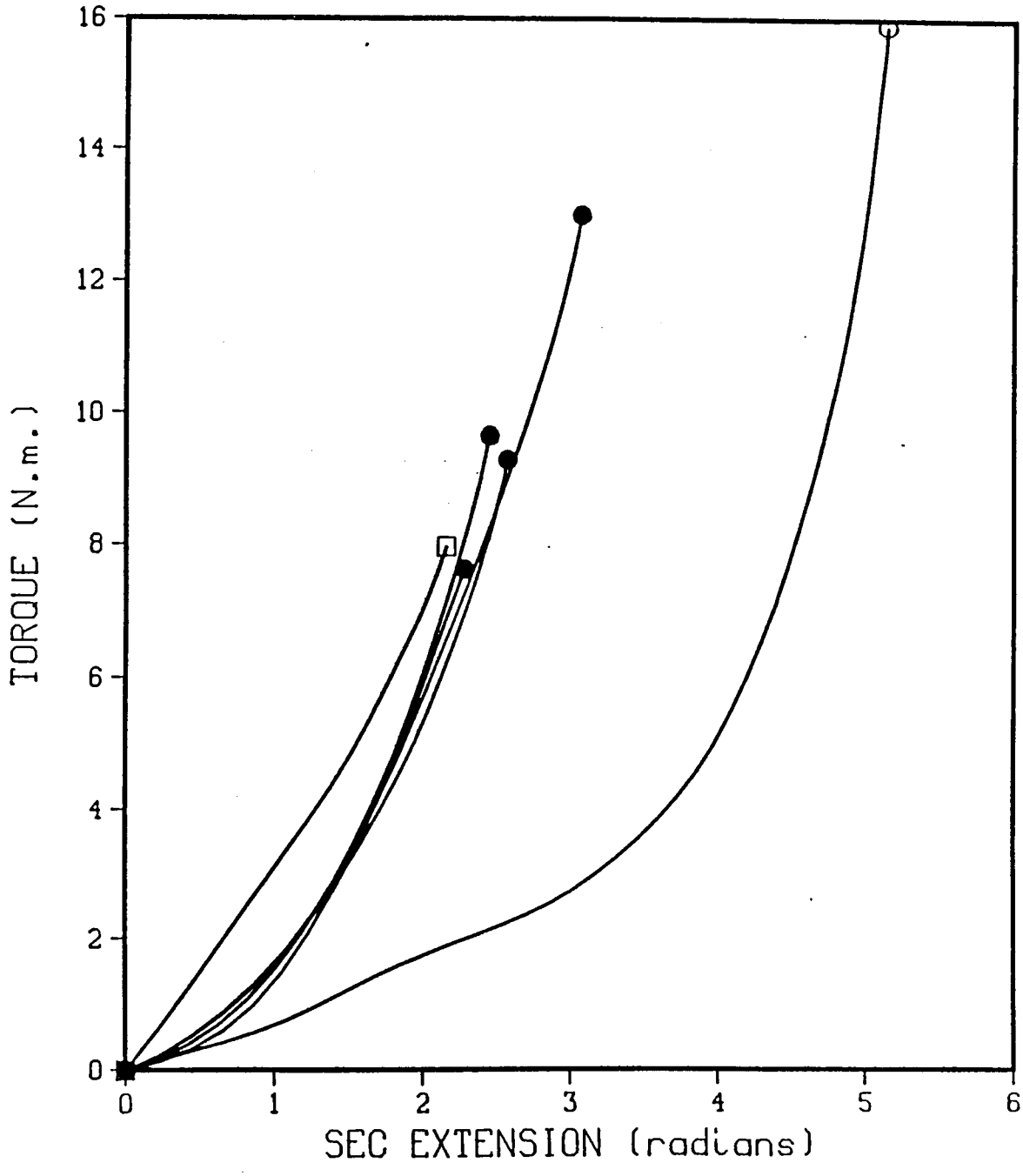


Figure 8. Calculated torque–SEC extension relationships.

polynomial regression equation that described the torque–SEC extension curve (BMDP5R, $p>0.05$).

Results showed that the SEC was slightly more compliant during back–swing contractions. This increased compliance is probably due to an increased rate of cross–bridge cycling (Joyce *et al.*, 1969) or cross–bridge slippage (Flitney and Hirst, 1978). However, as this small variation could be due to changes in activation it was decided not to model with variable moduli of elasticity for the SEC as suggested by Crowe (1970). This observation is discussed in detail in the final discussion.

3.3 Activation profiles

As discussed in the literature review, it is extremly difficult to quantify the stimulation and subsequent activation of a muscle *in situ*. Fenn (1930) suggested that a muscle takes 40 msec. to become fully active which would correspond to an exponential rise–time–constant of 0.012 sec.. Baildon and Chapman (1983a) considered any value over 0.01 sec. as high and the filtered integrated EMG obtained by Thomson (1983) took 200–300 msec. to reach maximal values. It is clear therefore that this problem requires further research.

Baildon and Chapman (1983b) showed that a step (immediate increase to the maximum) in activation produced very similar results to an exponential increase with a rise–time–constant of 0.01 sec. or less. As an instantaneous increase is unrealistic a value of 0.01 sec. was chosen for the simulation of the experimental data. Trial runs of the model showed that any variation of this value between 0.005–0.015 seconds produced negligible differences in model output.

Running the model with different SEC stiffnesses within the variation (and possible error) calculated for the SEC above produced variations in the rise–time of torque greater than those due to the approximation in the activation rise–time–constant. Until further work can accurately evaluate the time course of increasing activation in human skeletal muscle *in situ* this value will be one more small approximation inherent in such models.

3.4 Variations in mechanical output

As mentioned in the literature, attempts to measure the level of activation in a muscle have so far proved problematic. To overcome this problem maximal voluntary stimulation was required of the subject AC in this study and those of Chapman *et al.* (1985) and Thomson (1983). It was hoped that

this would produce maximal activation in every trial and therefore reduce one source of error in the data. Despite this stipulation some variation in activation will occur even in skilled subjects such as AC. Therefore even under identical experimental conditions some variation in torque is to be expected. One possible way of analysing such data would be to set up series of intervals for the independent variable and only consider the maximal values of torque obtained between these interval points. The rationale for such a technique would be that the higher torque values represented trials where the activation was nearer maximal.

Using polynomial regression (BMDP5R) a curve was fitted to both the maximal data, and all the data, for each of the three experimental conditions. Statistical analysis revealed no significant difference between any of these pairs of curves at the 0.05 level. These results suggest that the variation in AC's mechanical output from trial to trial and day to day, is not so large that the data cannot be considered as the same population group. Therefore all the data from the isometric, rest and back-swing contractions can be used in the estimation of mechanical properties of the supinator muscles of subject AC.

3.5 Identification of force enhancement

The polynomial regressions gave the relationships between P/Po and CC velocity for the data from the rest (Figure 7a) and back-swing trials (Figure 7b). The torques obtained for any given velocity were clearly higher for the back-swing trials and a statistical comparison of the curves of both relationships showed that they were statistically different at the .01 level. Nearly all peak torques from the back-swing data (96%) occurred at positive values of CC velocity so the relationships fitted to both sets of data cover common CC velocity ranges. It is clear therefore that the inclusion of a back-swing does in some way modify the $\tau-\omega$ relationship of the CC in human muscle.

CHAPTER 4

SIMULATION OF PREVIOUS RESULTS

An initial analysis was carried out to see if the model was accurate in its prediction of the rest data trials of Chapman *et al.* (1985). Failure of the model to do so would indicate errors in either the model itself or in the calculated values of AC's muscle properties. After these muscle properties are entered into the model, the moments of inertia of the muscle system and the load, the starting position of the muscle and its initial velocity are entered. These are all obtainable from the original contractions from which relationships were obtained.

Due to the number of trials, comparison for each trial was restricted to the following four output variables, peak torque (P_T), time to P_T, angular velocity at P_T, and angle at which P_T occurred. All four variables carry the same importance as the model would not be accurate if, for example, it predicted P_T exactly but the rise-time of this torque was faster and its P_T occurred significantly earlier than the P_T in the experimental condition. This preliminary simulation of the rest data showed that the model predicted these variables to within 10% accuracy. This initial analysis was performed only as a 'check' to see if the calculated relationships were quantitatively accurate before proceeding. As the results of the simulations are presented later they will not be duplicated here.

As mentioned, the literature does not indicate a fixed relationship between force and eccentric CC velocity. Many such curves have been reported and the approximate shape of this portion of the force-velocity curve, at least at maximal stimulation (Joyce *et al.*, 1969), is well known (Flitney and Hirst, 1978; Joyce and Rack, 1969). The calculated $\tau-\omega$ relationship for AC in this study was extended into the eccentric region using the maximum torques obtained in the study as the saturation level, and by simply 'drawing' the rest of the curve to conform with the curves reported in the literature. Although this method is rather crude it did allow the model to be used to simulate the back-swing data. Despite the approximation it was hoped that some idea of the internal interaction between the CC and SEC in a two-element model undergoing stretch could be gained.

In the majority of model simulations of the back-swing trials no CC stretch occurred. The CC shortened slowly allowing the torque to rise to high levels and this produced large SEC extensions. As the SEC extended more than the CC shortened the whole muscle underwent stretch. The rise time in the torque obtained from the model was generally in close agreement with experimental data. This

supports the use of the value obtained for the fitted SEC characteristic. Peak torques were too low as the $\tau-\omega$ relationship used did not fit the back-swing data. Nevertheless, time of zero angular velocity, the angle at which this occurs, time to $P\tau$, and angle at $P\tau$ were calculated relatively accurately (within 15% of the experimental results). The errors tended to be systematic with angular displacement at $P\tau$ being low which was probably due to the fact that the model predicted a lower force which could not 'turn the load around' quite as quickly. At this point in the study it could not be determined if an increase in force due to FE would adversely affect the other variables or erase the systematic errors.

Despite the limitations of this second series of simulations it did present an indication of the CC and SEC length changes. In simulations using the lower moment of inertias as loads, the model showed that the CC could still shorten relatively quickly throughout the contraction. To model these contractions so that the CC is stretched would require drastic changes in either the calculated properties of AC's supinator muscles or the model. The accuracy of the model in predicting the rest data suggests that such changes are not warranted.

Although there is a close agreement between the calculated stiffness of the SEC for AC and the estimate of this value presented by Baildon and Chapman (1983a), the model was run again with various higher SEC stiffnesses. Although this produced a CC stretch in more cases than was previously recorded, many trials still indicated no CC stretch even with an SEC component twice as stiff as that which was originally used. The time-courses of the torques were adversely affected when high stiffness values were entered so the evidence suggested that the original value should not be altered.

4.1 Theoretical problems of the two-element model

The above simulations, while not providing the exact magnitudes of the internal length changes in the SEC and CC, did highlight many problems with the two-element model. These problems occur when history dependent properties are to be included in such models. The SEC is considered to be a passive spring and yet it is the only element being stretched in many situations where the mechanical output of the whole muscle is enhanced by a prior back-swing. If SEC stretch is the cause of 'enhancement' then the SEC can no longer be considered to be a passive element. However the SEC is stretched in all contractions, and as already discussed the model simulations of rest contractions produced accurate results without any alterations. Any attempt to make the SEC active and 'enhanced' by stretch would therefore reduce its accuracy in predicting rest contractions.

The most likey cause of this problem with the SEC is that it is a theoretical construct which represents both the tendon of a muscle and the cross-bridges within the sarcomere. While it is reasonable to say that the tendon is passive, it is certain that the cross-bridges are not passive elastic structures. Furthermore a large proportion of the elasticity of a muscle occurs in the cross-bridges because, as the literature indicates, the tendon is so stiff that it cannot account for much of the SEC extention (Joyce and Rack, 1969; Morgan, 1977; Morgan *et al*, 1978). Therefore constructing a model that has a passive SEC appears to be an oversimplification that seriously affects the adaptability of the model to general applications.

Another possibility which could explain why no CC stretch was observed is concerned with the concept of a 'single equivalent muscle'. Forearm supination is the result of a group of muscles contracting. During eccentric contraction one or more of these muscles may experience a CC stretch (if each were modelled separately) while others do not. The effect of the single equivalent muscle model may 'average out' the mechanical response of each individual muscel and as such may present a misleading picture.

A detailed discussion on the possible mechanism of force enhancement and the role of the cross-bridges is included in the discussion section. Despite the theoretical ambiguities, practical implementation of force enhancement into a model of human skeletal muscle based on a two-element model was possible. Rather than modelling the enhancement of torque on the amount of CC stretch as originally envisaged, it was necessary to calculate a relationship between whole muscle stretch and the augmented torque. This overcomes the problem of the SEC being stretched in rest as well as back-swing contractions. As the model is used as a teaching aid, accuracy of the predicted mechanical output can be used as a claim of validity for the model. There is no doubt however that as a theoretical construct it appears that the two-element model is incapable of explaining the phenomenon of force enhancement.

CHAPTER 5

RESULTS AND ANALYSIS

After the analyses described above it was necessary to obtain more data on the phenomenon of force enhancement. In addition, calculation of the torque-angular velocity curve in the eccentric region was needed for a more accurate investigation. The experiments described in Chapter 1 were performed to obtain data in this region.

Work by Kaneko *et al.* (1983) suggests that a training effect may be present over the course of the trials. As the data collected by Chapman *et al.* (1985) and Thomson (1983) are also to be used in this thesis it seems unlikely that this training effect would be as large a problem as the possibility that the mechanical properties of AC's supinator muscles have changed over time. These two sets of experiments and the present set span three years. The series of isometric contractions were conducted to check whether there was any change. They indicated that these changes, if any, are of no greater magnitude than the inherent variability present in any experiment using human skeletal muscle *in situ*.

There were 30 dynamic trials that produced 30 peak torques at relatively high stretch velocities (2 to 5 radians per second). Also calculated were 65 troughs and peaks in torque from the oscillatory phase of the contractions. These oscillation points were characterised by lower torques and low velocities of shortening and lengthening. All 95 points are shown in Figure 9.

The data fitted the basic shape of the eccentric portion of the force-velocity curves reported in the literature without any allowances being made for stretch amplitude and time after or during stretch. An examination was conducted to see if there was a relationship between peak torque and amplitude of stretch. Only the initial peak torque in each trial was analysed due to difficulty of obtaining values for variables during the periods of oscillation. During this period a series of smaller stretches occur making it impossible to know the 'true' amplitude of stretch and the time of the onset of stretch is no longer a meaningful variable.

A slight decrease in P/Po with increased amplitude of stretch was indicated ($r=-0.562$), but when this data was combined with the back-swing data (Chapman *et al.*, 1985) no relationship was discernable ($r=-0.127$). Time from the onset of stretch and peak torque were also tested for a correlation but showed no significant result ($r=-0.398$). Again, combination of these results with the back-swing data reduced the r value ($r=-0.14$). Some high peak P/Po values were recorded long after

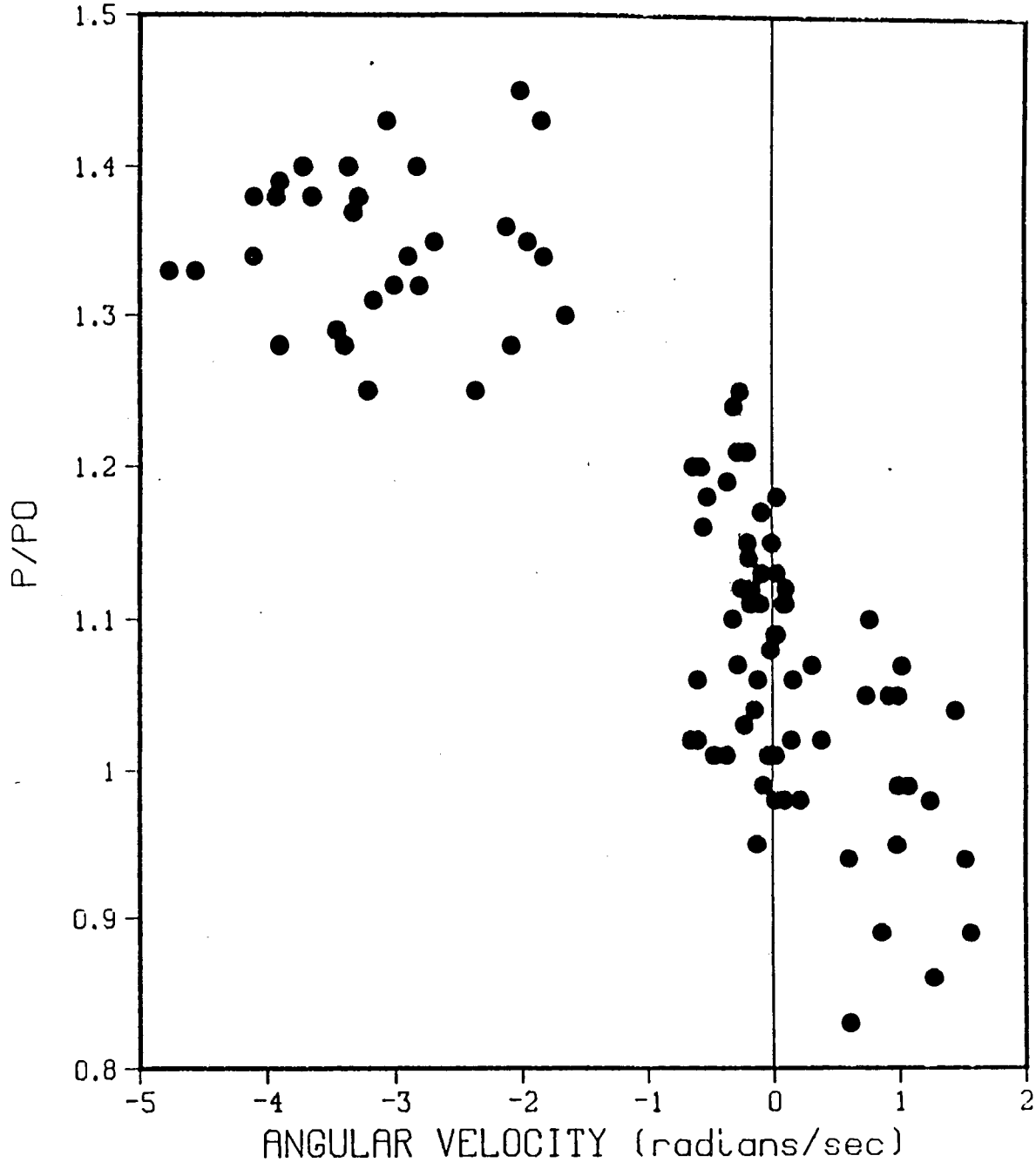


Figure 9. Complete experimental results.
All points represent a time when the rate of change of torque was zero.

stretch had ceased and other trials showed low peak P/Po values during stretch and soon after.

The lack of any set relationships was not surprising due to the results of previous studies. Edman *et al.* (1978a,b) showed that "at sarcomere lengths between 1.9 and 2.3μm the force enhancement after stretch decayed rapidly" and was "independent of amplitude of stretch". And yet at sarcomere lengths above 2.3μm they states that "force enhancement after stretch decayed very slowly" and "the force enhancement after stretch increased with amplitude of stretch and increased for any given stretch amplitude with sarcomere length.". Gordon *et al.* (1966) plotted the ascending limb of the isometric tension-length relationship up to sarcomere lengths of 2.25μm (see Figure 2). As the relationship of isometric torque-angular displacement for AC indicates that he is still on the ascending limb of the typical force-length curve it is likely that in the contractions studied the sarcomere lengths were below, or very close to, 2.3μm. Thomson (1983) also failed to define any relationship between amplitude of stretch and force enhancement in 5 males performing forearm supination.

Despite the lack of relationships discussed above the data did lie close together in the shape of a typical force-velocity curve. It is interesting to note that the peak torques from the back-swing data fitted a typical force-velocity curve (higher correlation) far better than did the results from the rest data. One reason athletes use a back-swing could possibly be because they are able to obtain more consistent results than if they start from rest with the muscle 'unactivated'.

It was hypothesied that the results indicated that the inclusion of a stretch phase in human muscle contractions does enhance the contractile mechanism and that this force is shifted towards higher values of the force-velocity curve. However, there seems to be an upper limit to this shift and a maximum 'enhanced' force-velocity curve is reached in the results of this study and that of Chapman *et al.* (1985). It is likely that small, and very slow stretches result in a smaller shift of the F-V curve, but the data points of these studies appear to lie on, or very close to one curve. Greater amplitudes of stretch did not produce a significant number of torques which lay on higher $\tau-\omega$ curves. Both studies show that the greater amplitudes of stretch often produce lower P/Po values. This could be due to the plateau of the $\tau-\omega$ curve being reached (P=constant) and the muscle being stretched to lengths where Po is increasing.

Figure 10 shows the curves fitted to the back-swing data and the stretch data (curves 1 and 2). These curves intersect the ordinate at very similar values and produce the shape of a typical force -velocity curve. This is remarkable when one considers that these sets of data were obtained from

different types of contractions two years apart. It is felt that the 'link' between these two curves supports the hypothesis of the existence of a maximum $\tau-\omega$ presented above.

An 'unenhanced' force-eccentric velocity relationship cannot be obtained by direct experimentation as it is a theoretical construct. Curve 3 in Figure 10 shows the fitted curve from the rest data. The pattern of these three curves make it easy to envisage the shape a non-enhanced $\tau-\omega$ curve would take in the eccentric region.

Figure 11 shows the oscillation data with a line of best fit. This line intersects the ordinate (at zero velocity) at a value of 1.09 P/Po. This line is not meant to represent a predicted relationship but was calculated purely to investigate the intersection point with the y-axis (if no enhancement was in effect this intersection value would be 1.00 P/Po). The back-swing data also intersects at 1.09 P/Po. The constant value of the shift in P/Po at the ordinate indicate that the values of the enhanced curve (curve 2, Figure 10) could be reduced by subtracting 0.09 P/Po to obtain an estimate of the un-enhanced curve at eccentric velocities. This section of the curve fitted well with the concentric curve as can be seen in Figure 12 which shows both the un-enhanced and enhanced curves. The apparent 'link' between the eccentric and concentric regions of both curves indicates the repeatability it is possible to obtain using a trained subject.

This procedure is not meant to provide a set relationship between the two variables but rather is a practical tool used in the curve fitting procedure. Numerous other curves, using hyperbolic and exponential functions for example, could describe the eccentric or concentric portions of the $\tau-\omega$ curve. However, to define the curve over the entire range of velocities requires a function which can fit a curve with numerous points of inflexion. A high order polynomial can easily handle such a curve although care must be taken not to use such a high order that the basic shape of the $\tau-\omega$ curve is destroyed. It must also be understood that the equation will not be able to calculate the value of the dependent variable for values of the independent variable outside the range of the original values.

In the initial stages of a simulated contraction the exerted torque is extremely low. Therefore, the angular velocity of the CC is near to maximum. It is therefore neccessary to fit the $\tau-\omega$ relationships for velocities up to the maximum velocity possible in the concentric region (V_{max}). In the eccentric region, as velocity increases there is no further increase in P/Po. This plateau is seen in the literature (Joyce *et al.*, 1969) and in the data from this thesis (Figure 9). It was therefore necessary to add to the experimental data values for V_{max} and for the plateau of torque so that the $\tau-\omega$

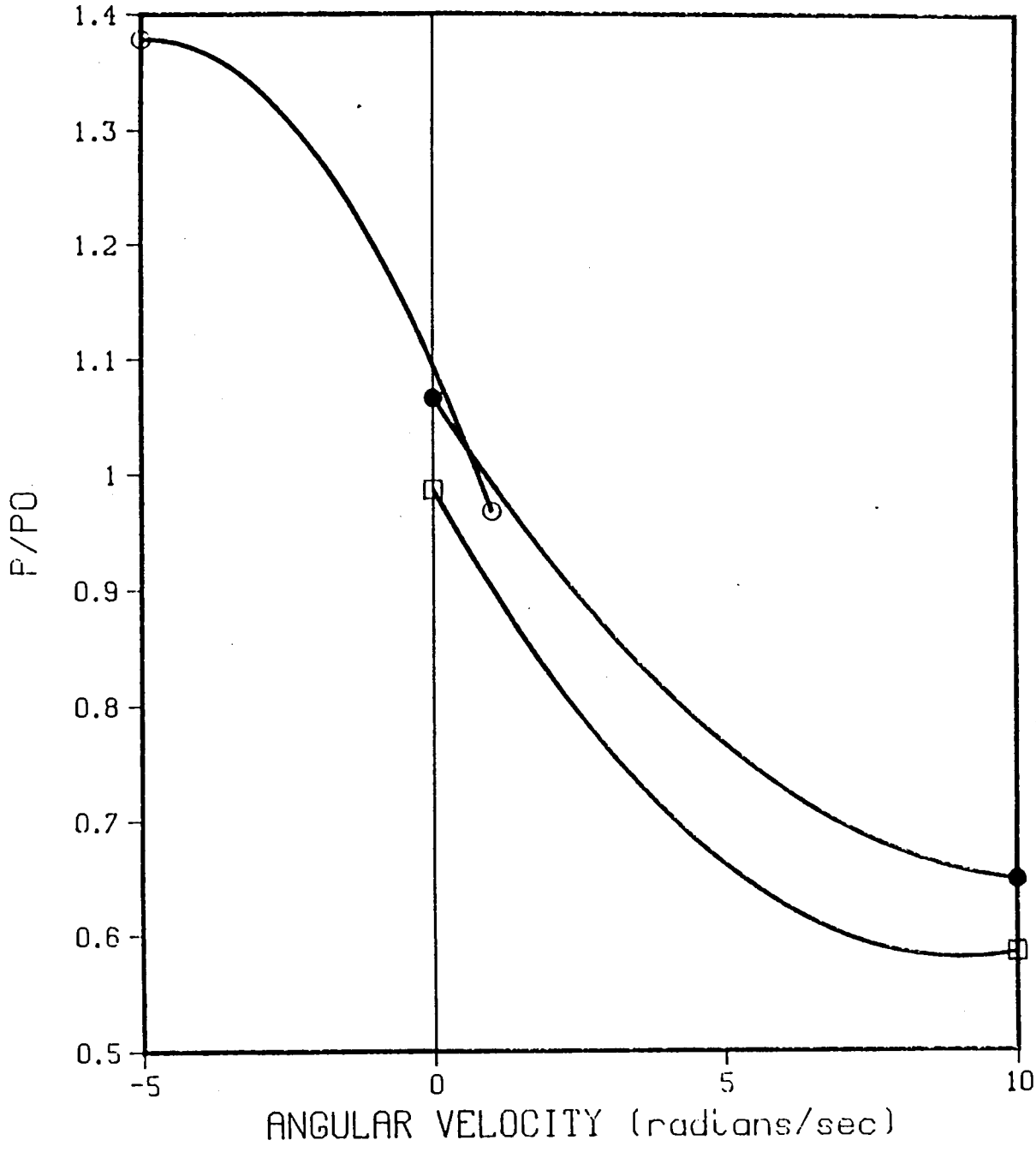


Figure 10. Fitted torque-angular velocity curves from experimental data.
Curve 1: Backswing data of Chapman *et al.* (1985), •.
Curve 2: Stretch trials from this thesis, o.
Curve 3: Rest contraction trials from Chapman *et al.* (1985), □.

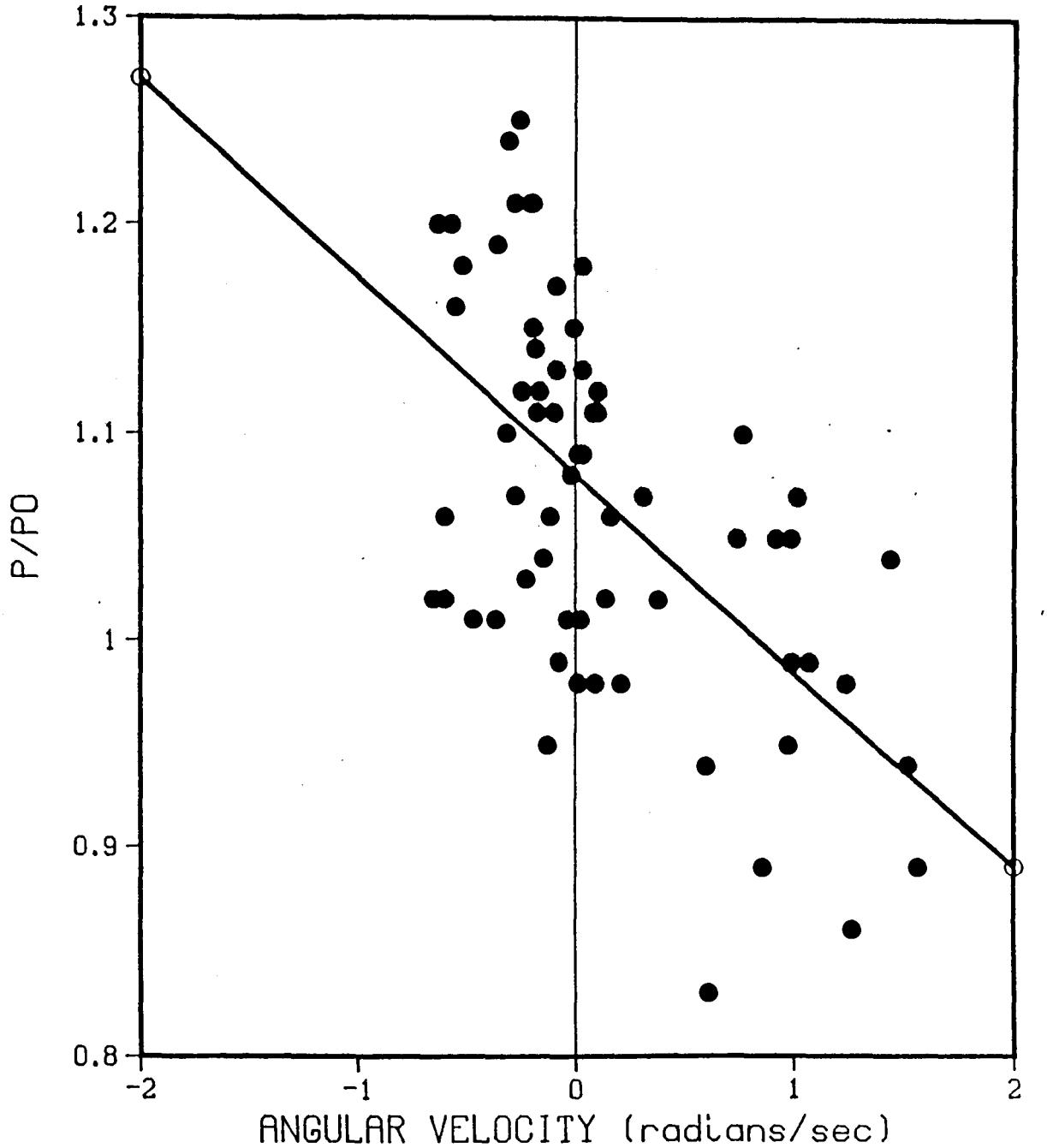


Figure 11. Peak and trough torque values during oscillation, •, and fitted relationship, o.

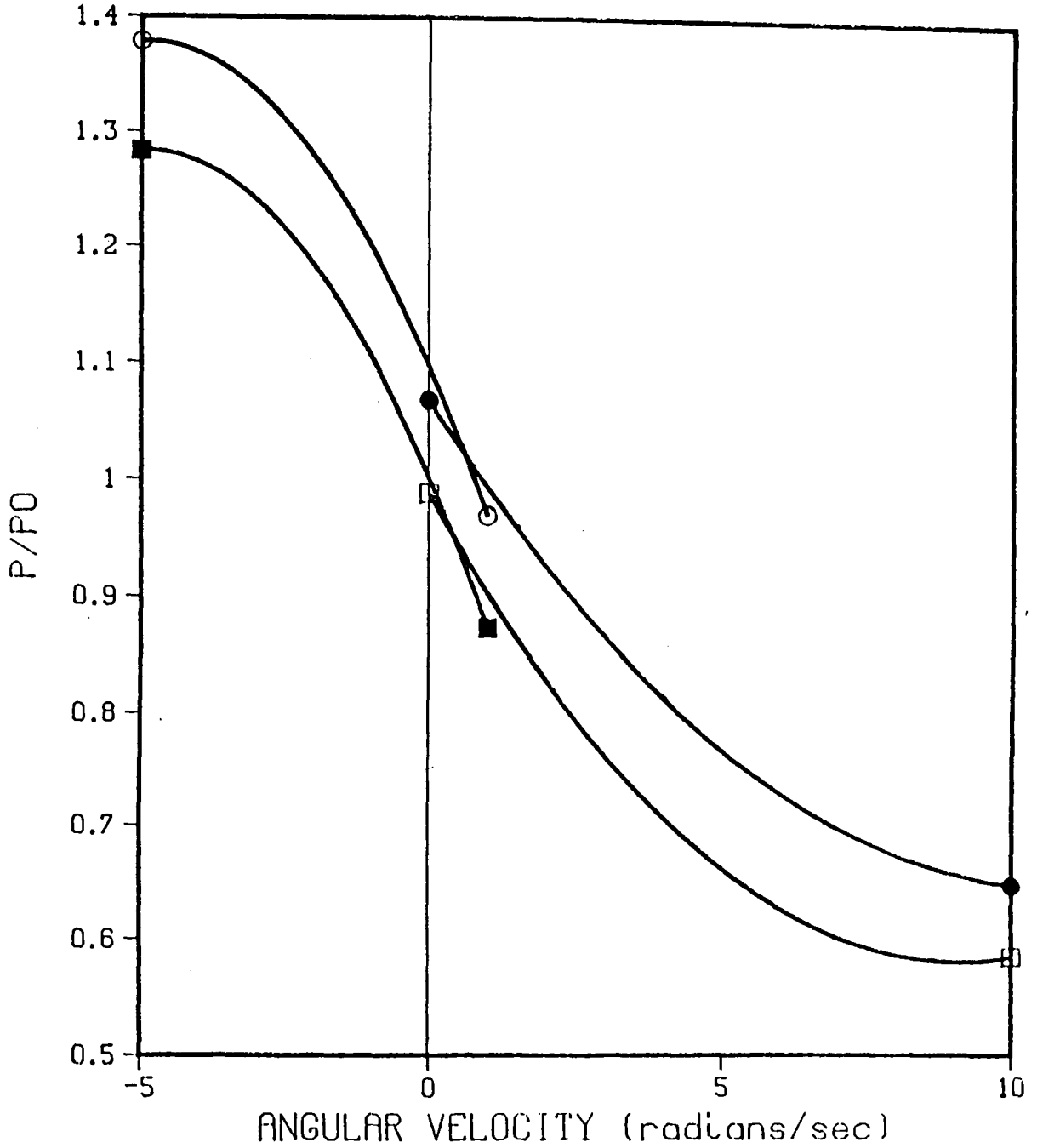


Figure 12. Estimated unenhanced $\tau-\omega$ (eccentric) relationship.
Curves from Figure 10 with hypothetical unenhanced force-velocity (eccentric) curve, ■.

relationships could be used to calculate velocities for all values of P/Po encountered in the model. Chapman (1980) showed that subject AC was capable of producing forearm rotations of 40 radians per second during unloaded contractions. Despite the fact that the forearm itself is a small inertial load (0.004 Kg.m^2 for AC) and V_{max} is considered to be the velocity of shortening when the load is zero, V_{max} was considered to be 40 rad/sec. In any simulated contraction, Baildon and Chapman (1983b) showed that the muscle spends so little time in this region that any errors encountered by the adoption of this value would be negligible.

Petrofski and Phillips (1980) have indicated that V_{max} changes with changes in activation and this option was already included within the model. There is the possibility that force enhancement in human muscle *in situ* is at least partly due to an increase in activation for a similar level of stimulation and V_{max} could therefore be different in the enhanced $\tau-\omega$ relationship. Nevertheless, the literature is not entirely in support of Petrofski and Phillips' hypothesis and Edman (1978) showed no alteration in V_{max} following stretch in isolated preparations. It was therefore decided not to alter V_{max} in the enhanced $\tau-\omega$ relationship because of the lack of firm evidence of its variablility.

After the cessation of stretch the excess force takes a finite time to decay back to pre-stretch levels. This decay time has been observed in isolated preparations (Edman *et al.*, 1978a,b; Sugi, 1972) and human muscle *in situ* (Thomson, 1983). Thomson calculated the exponential decay time constant of torque for AC after a ramp stretch. As it is not possible to calculate the decay time constant in a dynamic contraction, Thomson's value was used in the modelling application. The rate at which the enhanced $\tau-\omega$ relationship is obeyed is more problematic. The experimental results indicated several points where the peak torques were recorded very quickly after the start of the stretch. This indicates that the muscle responds very rapidly to stretch but it does not provide a quantitative verifiable value of the rise-time-constant.

CHAPTER 6

INCLUSION OF FORCE ENHANCEMENT INTO THE MODEL

6.1 Adaptation of Iterative Loop Algorithm

The approach to the modelling of human muscle used in this thesis has been outlined in detail by Baildon and Chapman (1983a). The 'heart' of the model is an iterative loop which uses entered muscle properties and experimental conditions to calculate the mechanical output from the muscle. The relationships used in this application were obtained from the empirical data discussed previously. All curve were fitted using BMDP polynomial regression.

The CC $\tau-\omega$ relationship was caculated as a 5^{th} order polynomial from the rest data of Chapman *et al.* (1985) and the hypothetical unenhanced $\tau-\omega$ relationship proposed above (lower two curves, Figure 12). The CC $\tau-\theta$ relationship is a 1^{st} order polynomial fitted to data from a series of isometric trials by Chapman *et al.* (1985), (Figure 3b), and some further isometric torque data obtained in the present study. The torque–activation relationship is represented as a straight line for convenience due to problems of quantifying EMG output.

The torque–time and angular velocity (external)–time data from each trial of Chapman *et al.* (1985) used in conjunction with the calculated $\tau-\omega$ relationship allowed an angular velocity (CC)–time array to be calculated. Subtraction of these angular velocities (CC – external) gave the angular velocity of the SEC. Integration with respect to time produces SEC stretch and thus torque–SEC extension curves were obtained (Figure 8 shows some of these curves).

Not all of the equations which describe the system can be solved simultaneously so a stepwise iterative routine (Figure 13) is used to provide an approximation. Initially torque (T) is evaluated as a function of SEC stretch (δ), limb angular displacement (α) and activation (A). Once torque is known CC angular velocity ($\dot{\beta}$) can be evaluated as a function of torque, activation and limb angular displacement. Subsequently CC angular displacement (β) is obtained by integration of its angular velocity. Limb angular acceleration ($\ddot{\alpha}$) is calculated by dividing torque by moment of inertia (I), and limb angular velocity ($\dot{\alpha}$) and displacement (α) are obtained by integration. Finally the new SEC angular stretch is obtained by subtracting α from β.

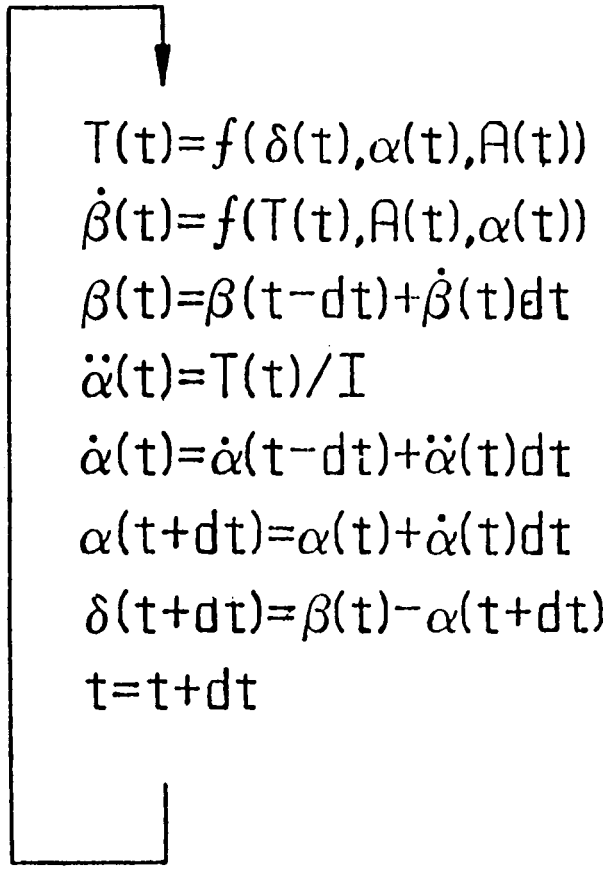


Figure 13. Diagram of iterative loop used to calculate model output.
T is exerted torque; A is activation; α is limb angular displacement, $\dot{\alpha}$ and $\ddot{\alpha}$ are the first and second derivatives; β is CC angular displacement, $\dot{\beta}$ is CC angular velocity; δ is SEC angular stretch; I is the external moment of inertia; t is time, dt is time increment.

Due to the integration steps α and β are known at different times only (t+dt and t respectively) and a small error is introduced. Baildon and Chapman (1983b) show in their analysis that for appropriately small time intervals (dt), this error is negligible.

To model the phenomenon of force enhancement a check is made during this iterative loop to determine if whole muscle stretch has or is occuring. That is if $\alpha(t-dt)$ is greater than $\alpha(t)$. If this is the case CC angular velocity is now calculated from an enhanced relationship between torque and CC angular velocity. This relationship is a 5^{th} order polynomial fitted to the backswing data of Chapman *et al.* (1985) and the stretch data of this thesis (top two curves in Figure 12). For a given torque, assuming that activation and limb angular displacement are the same, this results in a higher $\dot{\beta}$, which when integrated, produces a greater value for β than would be the case without FE. Because β is larger each iterative step produces a larger SEC stretch [$\delta\ (t+dt) = \beta(t) - \alpha(t+dt)$] which subsequently results in a larger torque being calculated at the beginning of the next iteration. This excess torque is the force enhancement. This value is intially adjusted to allow the muscle to take a finite time to move from the un-enhanced $\tau-\omega$ curve up to the enhanced $\tau-\omega$ curve. After stretch has stopped this excess in $\dot{\beta}$ is gradually reduced so that the torque output decays back to un-enhanced levels at the same rate as Thomson (1983) calculated for AC. Both these adjustments are performed using exponential equations. Figure 14 is a flow diagram of the iterative routine displayed in Figure 13 with the force enhancement option included.

6.2 Validation of the Force Enhancement option

The model was run using the calculated properties of AC's supinator muscles. Starting angle and initial velocity were entered and the outputs of peak torque, time to peak torque, angle and angular velocity at peak torque were recorded. Two arrays were produced, one containing these four variables from each of the experimental trials of Chapman *et al.* (1985), and the other containing the model output simulating these trials.

Hotelling's t^2 test for matched pairs was performed as a multivariate statistic to test if the difference between the model and experimental data was significant. The results showed a significant difference between model and experimental data at the 0.01 level (BMDP3D). Analysis of individual variables by the program showed that only torque was significantly different ($P>0.01$). Separate Hotelling's t^2 tests for matched pairs were performed on the rest data and back-swing data. The results

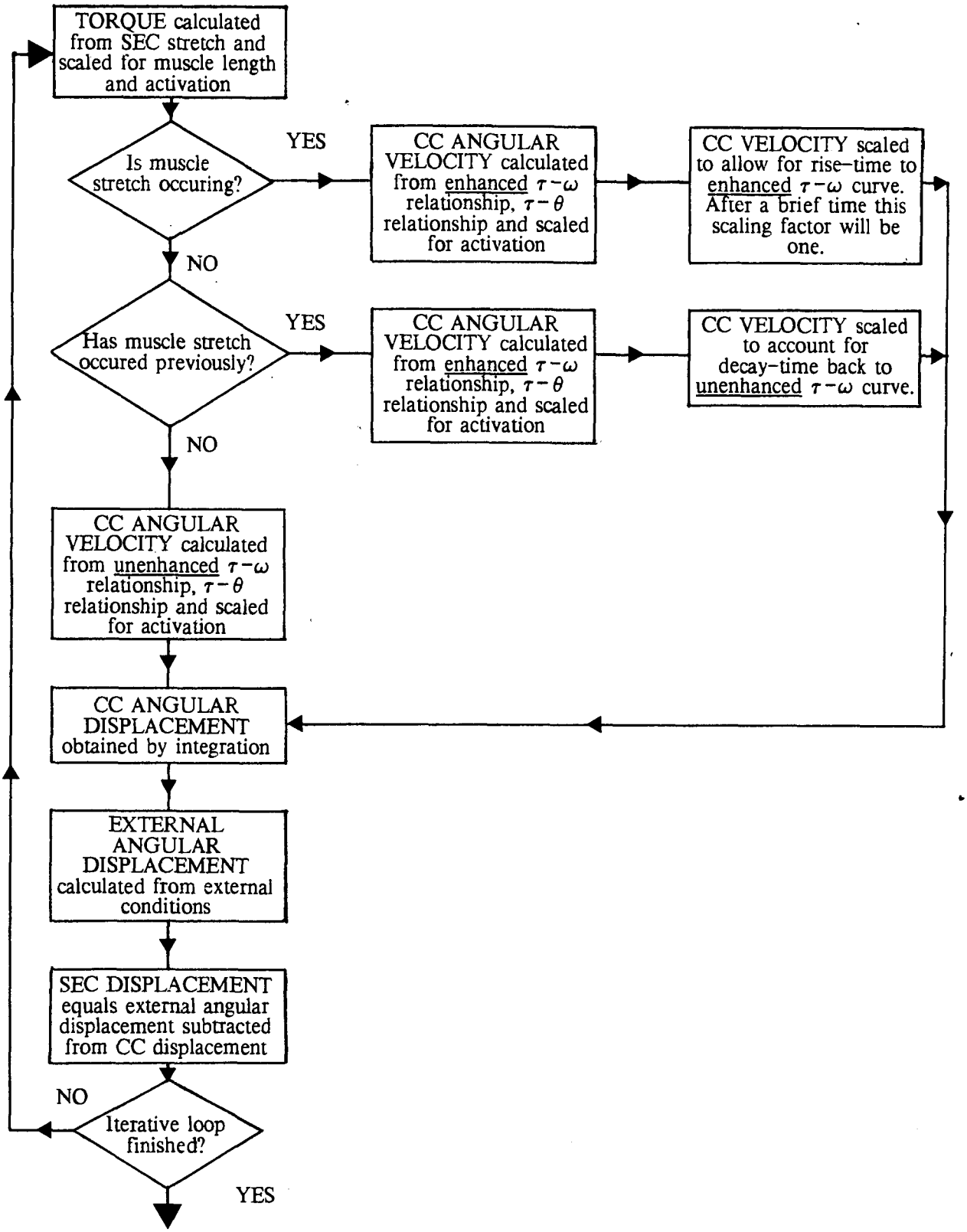


Figure 14. Flow diagram of force enhancement option in iterative routine.

for the rest data indicated no significant difference while the back-swing data produced a significant difference, again with torque being the reason.

These differences may be due to the nature of the experimental protocol. Despite holding the handle loosely, when the mass is rotated in the direction of pronation some resistance is present. Also the subject can anticipate that soon he will be required to produce maximal supination and as such his activation response may be quicker due to mechanisms as yet not investigated. He can not anticipate in this manner during the rest contractions. Another consideration is that the A/D sampling does not start until the rate of change of torque is greater than 0.5 Nm./second for three consecutive recordings. If the torque is greater than 1 Nm. at the start of each trial the trial is discarded. Therefore in the experimental data when time (t) is zero seconds the torque is greater than 0 Nm.. In the model simulation, when t=0 seconds, torque=0 Nm. This coupled with the approximation made to calculate activation could explain the torque being higher in the experimental results.

As mentioned earlier in the calculation of the torque-SEC stiffness it was noted that the time-course of some trials was in error due to an early initiation of data collection. These trials obviously affected the time variable although not enough to produce a significant statistic on that variable. Examination of the data showed that these trials plus a few others had torques up to 2 Nm. higher than predicted by the model. If these trials were removed from the analysis on the back-swing data the results of Hotelling's t^2 test was not significant at the 0.05 level. This indicates that the data collection problem could well be the cause of any discrepancy in the two sets of data.

Table 1 and Table 2 show the arrays of experimental and model outputs for the rest and back-swing data respectively. Table 3 shows the results of Hotelling's t^2 test on the rest and back-swing data, and both sets of data together.

Comparisons of this type are difficult as the model is designed to have physiological significance due to its use in research and teaching. It would be possible to have a 'black-box' system where the equations describing the system are arranged so that the sum of the errors for each variable is zero. This is statistically accurate, but the equations describing the CC and SEC have been calculated and statistically validated separately. If when put together the model is inaccurate, then the theory is wrong and altering the equations is pointless. If the model is accurate then the CC-SEC model is appropriate.

Experimental Output				Model Output			
t	τ	ω	θ	t	τ	ω	θ
0.16	10.86	3.35	0.367	0.174	10.1978	3.7705	0.4333
0.16	10.86	3.42	0.342	0.175	10.2722	3.7689	0.4023
0.165	10.7	3.29	0.343	0.1745	10.2281	3.8064	0.4102
0.155	10.79	3.14	0.332	0.175	10.2327	3.7809	0.4142
0.15	10.36	2.85	0.325	0.175	10.2028	3.7741	0.4285
0.14	11.32	3.04	0.611	0.178	10.7669	3.9116	0.7134
0.165	11.95	3.61	-0.104	0.177	11.1408	4.1049	-0.0521
0.155	10.48	3.16	0.469	0.1745	9.9906	3.6799	0.54
0.18	10.63	3.89	0.077	0.1685	10.6385	4.412	0.0922
0.14	11.04	3.208	0.175	0.1705	10.3858	4.0914	0.2763
0.16	10.99	3.61	0.411	0.1695	10.0219	3.9504	0.4647
0.15	11.09	3.43	-0.075	0.1715	10.8982	4.2972	0.006
0.155	10.66	3.55	0.141	0.172	10.5509	4.0645	0.2136
0.155	9.64	4.02	0.807	0.1575	9.1242	4.1234	0.8128
0.155	10.36	4.41	0.239	0.159	10.076	4.7027	0.2751
0.165	10.15	4.57	-0.05	0.161	10.705	4.914	-0.0521
0.165	10.43	4.66	0.302	0.1595	10.0767	4.6219	0.2923
0.115	8.216	9.64	0.392	0.1175	8.4928	8.4222	0.3467
0.135	7.801	9.89	0.796	0.116	8.0305	7.9491	0.6599
0.115	8.25	9.03	0.136	0.1185	8.8252	8.8437	0.1024
0.14	9.698	6.398	0.173	0.141	9.5918	6.3855	0.1589
0.16	9.203	6.594	0.538	0.14	9.1383	5.8806	0.4683
0.135	10.56	6.02	0.045	0.141	9.8889	6.247	0.044

Table 1. Arrays of data from rest trials.
Time = t, Torque = τ, Angular velocity = ω, and Angular displacement = θ.

Experimental Output				Model Output			
t	τ	ω	θ	t	τ	ω	θ
0.245	14.14	1.99	-0.199	0.204	14.0486	2.2801	-0.0223
0.255	12.76	1.91	0.296	0.199	12.5245	2.3216	0.4824
0.215	16.41	0.253	-0.257	0.226	16.1975	0.99	-0.2134
0.23	15.4	1.33	-0.069	0.2115	14.291	1.7496	0.0743
0.205	12.99	1.44	0.434	0.2015	12.4674	2.1727	0.543
0.2	17.00	0.056	-0.181	0.2255	15.972	1.0326	-0.1652
0.28	15.08	0.046	0.197	0.245	15.3544	0.1985	0.446
0.2	12.5	2.94	0.114	0.184	12.418	3.5896	0.1878
0.225	14.52	0.932	0.284	0.218	14.1471	1.1452	0.35
0.185	12.49	3.5	-0.054	0.184	12.644	4.0242	-0.0045
0.22	11.9	3.9	0.165	0.1745	12.2327	3.7858	0.2104
0.125	10.86	3.365	0.669	0.132	11.3361	3.4447	0.6271
0.14	8.87	10.72	0.737	0.123	9.1675	8.4799	0.575
0.13	9.04	10.665	0.368	0.1245	9.7219	8.9091	0.2488
0.145	12.68	3.78	0.068	0.156	12.1219	4.5006	0.0922
0.275	12.24	3.63	0.27	0.164	11.8724	2.5543	0.6469
0.17	14.49	0.292	0.228	0.1755	14.2688	1.2951	0.2533
0.175	16.44	1.209	-0.26	0.178	15.627	1.0613	-0.0708
0.22	11.42	6.43	0.45	0.1565	11.9593	3.8348	0.3059
0.225	11.105	4.445	0.875	0.1545	10.3823	3.3666	0.9857
0.115	11.45	3.74	0.529	0.131	11.7466	3.6258	0.4365
0.185	16.58	-1.12	-0.119	0.2125	16.0504	0.8027	-0.0759
0.125	15.2	2.393	-0.223	0.135	14.9213	2.2621	-0.3062

Table 2. Arrays of data from back-swing trials.
Time = t, Torque = τ, Angular velocity = ω, and Angular displacement = θ.

BMDP3D UNIVERSITY OF CALIFORNIA.

Hotelling's T^2 analysis is used for the two independent groups (model and experimental output) each with 4 dependent variables (time, torque, angular velocity and displacement). Hotelling's T^2 is a multivariate analogue to the single t-test for independent means. It tests the overall null hypothesis that the two populations from which the two vectors of means were sampled do not differ on any of the measures (Schutz *et al.*, 1985; Huck *et al.*, 1974).

BMDP3D Hotelling's T^2 fit for REST DATA.

MAHALANOBIS D SQUARE	0.6232		
HOTELLING T SQUARE	14.3341		
F VALUE	3.0949	P-VALUE	0.0404
DEGREES OF FREEDOM	4, 19.0		

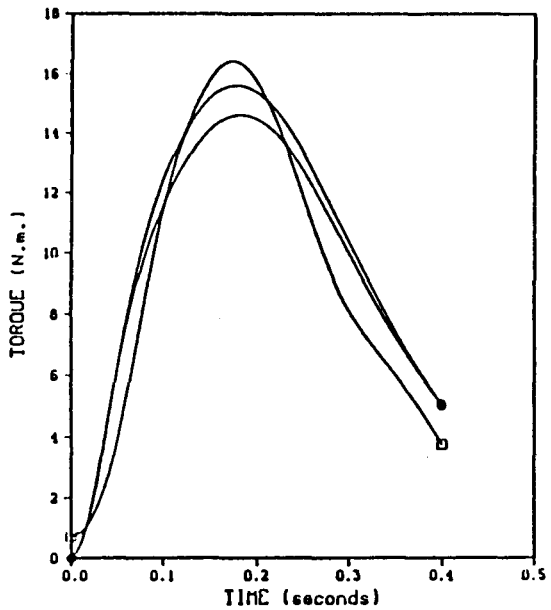
BMDP3D Hotelling's T^2 fit for BACKSWING DATA.

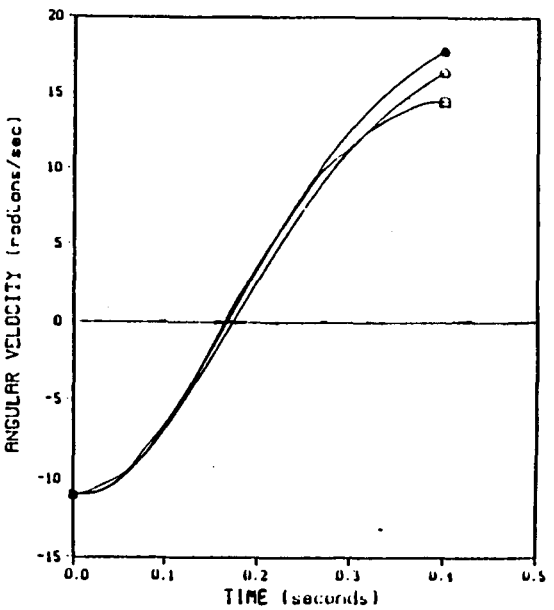
MAHALANOBIS D SQUARE	0.5331		
HOTELLING T SQUARE	12.2611		
F VALUE	2.6473	P-VALUE	0.0654
DEGREES OF FREEDOM	4, 19.0		

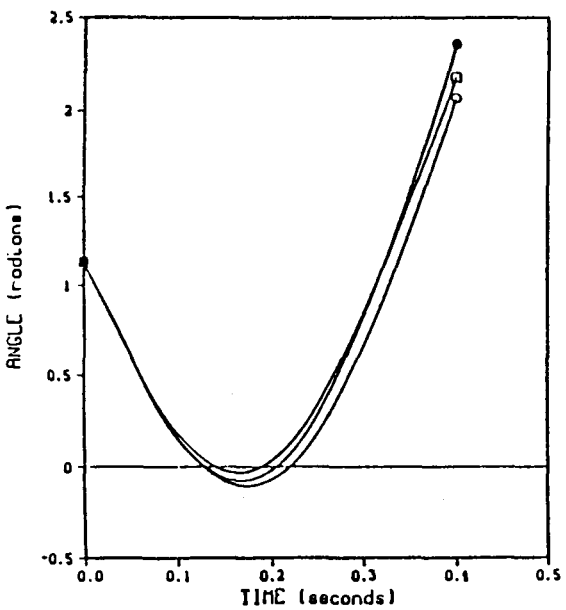
BMDP3D Hotelling's T^2 fit for BOTH SETS OF DATA.

MAHALANOBIS D SQUARE	0.3494		
HOTELLING T SQUARE	16.0702		
F VALUE	3.7497	P-VALUE	0.0107
DEGREES OF FREEDOM	4, 42.0		

Table 3. Results of Hotelling's t^2 test for rest, back-swing, and combined data.





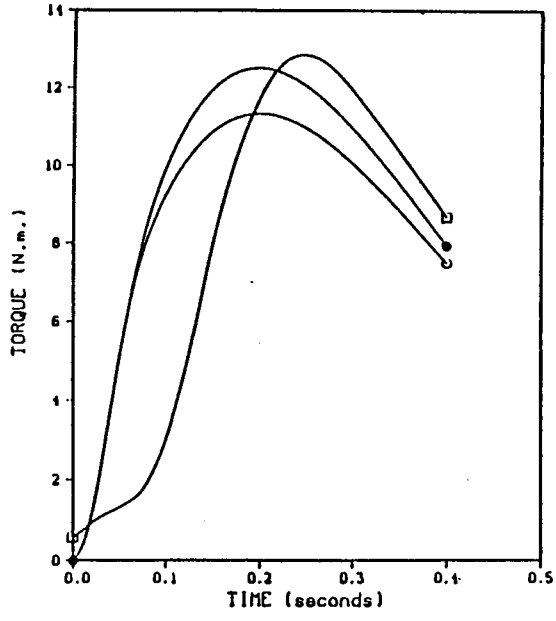


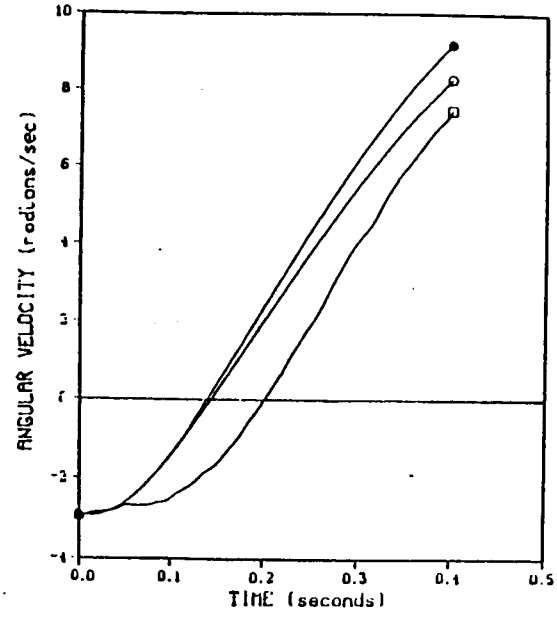
Moment of Inertia 0.141 kg.m^2

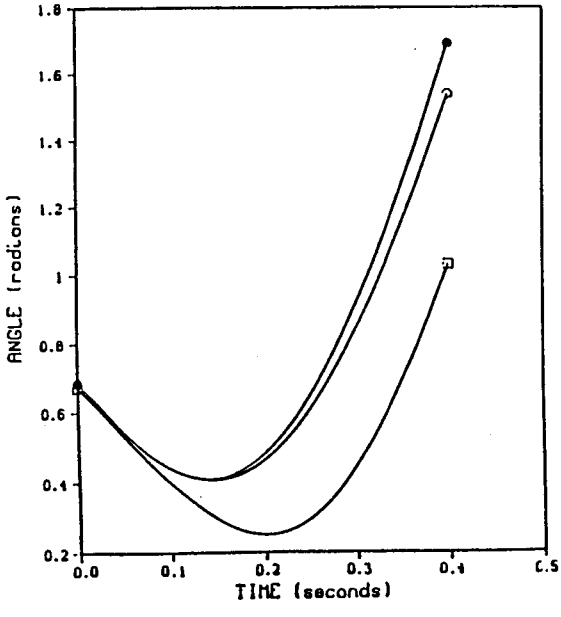
Legend

- enhanced curve
- un-enhanced curve
- experimental data

Figure 15. Curves representing typical experimental and model output. Torque, angle and angular velocity versus time.







Moment of Inertia 0.3095 kg.m^2

Legend

- ● enhanced curve
- ○ un-enhanced curve
- □ experimental data

Figure 16. Model and experimental ouput for a trial with large discrepancies.

The original model was intended to simulate conditions involving stretch but with only one force-velocity curve and thus no force enhancement. It is not the purpose of this work to criticise this original model. For better illustration however, Figure 15 includes an experimental trial and simulations from the original and new model. As can be seen the torque is too low when calculated without the force enhancement option. Figure 16 shows one of the worst fits obtained, which appears to be due to the problem of identifying the trials 'starting point'. When compared with the trial in Figure 15 it is apparent that the time-rise of torque (in Figure 16) is much slower and suggests that the subject is not producing maximum stimulation to his supinator muscles.

Even in Figure 15, in which the enhanced curves produce values close to the experimental data near peak torque, the time-course of these variables is often quite different. Initially the torque does rise as fast as the model outputs. Once it starts to rise more quickly the slope of this curve is very close to that predicted by the model. After peak torque has been reached the torque from the experimental trial decreases rapidly. This is not the case in the model simulation. However, the model continues to predict maximal activation and any drop in stimulation to the subjects muscles, as the velocity of the load starts to increase, could explain this discrepancy. If an accurate measure of the muscles activation could be obtained it is possible that the model would fit experimental output far more accurately.

Both model outputs for the rest trials are obviously identical as no force enhancement (FE) is present. To test the applicability of the FE option, rather than to criticise the original model, Hotelling's t^2 test was calculated for the back-swing trials simulated by the model without the FE option. The same four dependent variables were used (see Table's 1 and 2). The results are shown below and indicate a significant difference between model and experimental output. This is not surprising as the peak torque output from the model is too low in every trial.

MAHALANOBIS D SQUARE	9.5375		
HOTELLING T SQUARE	219.3637		
F VALUE	47.3626	P-VALUE	0.0000
DEGREES OF FREEDOM	4, 19.0		

CHAPTER 7

DISCUSSION

The fact that we can jump down safely from a greater height than we can jump up to indicates the protective nature of our skeletal muscle. The realisation of this fact can be observed in the eccentric velocity region of the F–V relationship where we can produce higher forces than when our muscles contract and shorten.

The force–length relationship of AC's supinator muscles, and those of the other 5 subjects tested by Chapman *et al.* (1985), show that isometric maximum torque increases as the supinator muscles lengths increase. Although these torques are the result of at least 3 muscles (Thomson 1983) it does indicate the protective nature of the human skeletal muscle system once again. If we examine the force–length relationship presented in Figure 2 we can see that the physiological limits of the muscles are not being reached even at full pronation. This arrangement of muscles in the forearm guards against hyper–pronation and possible injury. As will be discussed later this arrangement suggests that force–enhancement may not be as important in muscle *in situ* as it appears to be in isolated preparations where the muscle can be stretched nearer to its anatomical limits.

Further demonstration of this 'protection theory' can be found in the literature where a relationship between muscle length and activation has been reported (Rack and Westbury, 1969; Lambert *et al.*, 1979). At identical stimulus rates, as muscle length increases so does the activating input to the muscle. Rack and Westbury suggest that this may be due to the wave of depolarization encountering less resistance at longer muscle lengths because the tubules of the muscle fibre are stretched and reduced in thickness. As this depolarizing current spreads inwards from the surface of the muscle fibre, it would not be possible to monitor changes in activation due to this mechanism with EMG surface electrodes.

When discussing the function of human skeletal muscle and presenting theorems to explain its phenomenological behaviour, an understanding of its role in preventing joint and tendon damage by the mechanisms outlined above may prove helpful.

Despite being able to model the phenomenon of force enhancement and to use the model to examine the function of the CC and SEC as theoretical components of muscle, the mechanism of force–enhancement in whole muscle has not been isolated. The work using isolated preparations

(Edman *et al.*, 1978a,b; Sugi, 1972; Cavagna *et al.*, 1968) suggests that FE is significant at muscle lengths that may not be attained within normal ranges of movement for human muscle. However, with more than one muscle being responsible for any movement of the human skeleton it is possible that any stretch of a muscle group is being resisted, at least in part, by one or more muscles that have been stretched to a length were FE is significant.

It is possible that the inclusion of a back-swing enhances the activating input to a muscle rather than enhancing the contractile machinery at the sarcomere level. The muscle is being stretched towards the extremes of joint movement and one response could be to activate more motor units so as to protect the tendon, muscle and joint. This theory is compatible with the modelling application whereby muscle spindles could produce not only reflex activity but also initiate a more efficient activation response. The enhanced $\tau-\omega$ curve discussed previously could be due to this increased level of activation rather than some mechanical enhancement at the cross-bridges.

In isolated preparations the level of activation can be set very high and is easier to control than in human muscle *in situ*. The stories of the increased strength displayed by humans due to emotional derangement or drugs indicate that 'maximal voluntary effort' is likely to be far less than the true maximum. It is proposed that the inclusion of a back-swing in a movement requiring maximal force (e.g. discus, hammer, baseball pitching etc.) does elicit a protective increase in the activating input to the muscle, the advantage of which is not immediately lost in the subsequent shortening phase. The family of force-velocity curves at different levels of activation (Bigland and Lippold, 1954a) indicate that the observation of an enhanced $\tau-\omega$ curve in the present study is not an unexpected result, and that the hypothesis above is feasible.

Nevertheless, when activation is controlled in isolated preparations FE is still present and increases with increased amplitude of stretch. Furthermore, the results of this study indicate that the enhanced $\tau-\omega$ curve is reached even in stretches which would not appear to threaten the stability of the joint. Relatively slow stretches at medium muscle lengths resulted in P/Po values that lay on the enhanced $\tau-\omega$ curve. It seems unlikely that any 'protective mechanism' is responsible for this enhanced torque and it is concluded that more than one mechanism is responsible for the advantage gained by the inclusion of a back-swing.

The SEC and CC components of the two-element model were never meant to represent anatomical structures, but a model should be applicable to the general case without any theoretical

ambiguities. It appears the two-element model cannot simulate whole muscle stretch without abandoning the role of the SEC as a passive element. For any given muscle length after stretch the SEC of a two-element model is longer due to the higher forces produced. This results in a shorter CC. The CC force-length relationship would indicate that the muscle is capable of producing less force in this situation than during an isometric or concentric contraction at the same external muscle length. So the two-element model requires a considerable enhancement of the CC during stretch and yet the CC itself is not stretched in the majority of simulations performed with the model. No site for the phenomenon of force enhancement has been agreed upon in the literature although force is believed to be generated by cross-bridge heads at their point of attachment on the actin filaments. It is reasonable to assume therefore that any mechanical FE occurs in the cross-bridges. As the elasticity of a muscle consists largely of cross-bridge elasticity the concept of a passive SEC appears to be an oversimplification which seriously affects the applicability of the two-element model.

The elasticity of the cross-bridges could explain the phenomenon of FE by means of a 'geometrical' model. During stretch the cross-bridges could be extended so that they are attached to points on the actin filament which are further away than would be the case in an isometric or concentric contraction. As discussed SEC stiffness increases with SEC extension and this would result in greater forces being transmitted by the cross-bridges. This theory supports the observation that a muscle produces less force when shortening to a given length, than when lengthening to that length (Abbott and Aubert, 1952), arguing that this is due to differences in cross-bridge length. Another advantage of this geometry is the angle of pull of the cross-bridges. During stretch the filaments are extended and reduced in thickness, thus the cross-bridges are also extended placing them in a more mechanically advantageous position to produce force parallel to the filament alignment. Figure 17 displays this 'geometrical' theory.

This 'enhanced alignment' of the cross-bridges would explain the increased efficiency of eccentric contractions. Early work by Abbott *et al.* (1952) and Abbott and Bigland (1953) showed that the physiological cost of negative work is less than the physiological cost for an identical level of positive work. As the energy cost to the body is the same when each cross-bridge attaches to a site and generates force, a better angle of pull will be more efficient. Therefore fewer cross-bridges will be required to resist an opposing force eccentrically (negative work) than to generate that force concentrically (positive work).

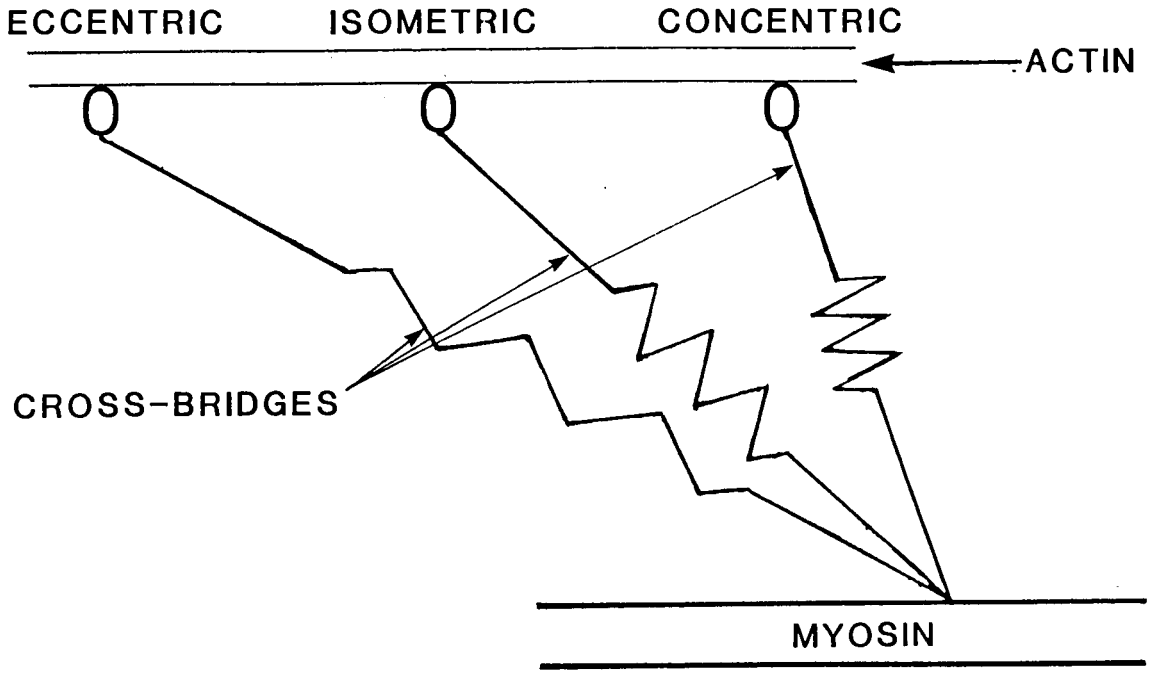


Figure 17. Possible cross-bridge alignments at the same sarcomere length obtained during concentric, isometric, and eccentric contractions.

Joyce *et al.* (1969) suggest that at high stimulus rates cross-bridges are less likely to detach and will be stretched to greater lengths, thus increasing the force output. Edman *et al.* (1978a,b) adds that this force increase prevents dispersion of sarcomere lengths, and could be seen as a protective function. Some detachment of cross-bridges will occur during stretch and these will attach at a reduced length. If the velocity of stretch is too high and the force reaches a critical level then enough cross-bridges will detach causing a reduction in tension. This is the phenomenon of cross-bridge slippage (Flitney and Hirst, 1978; Griffiths *et al.*, 1980). Flitney and Hirst indicated that sarcomere stiffness reduces after slippage occurs and this would explain why the calculated SEC stiffness values are less in the back-swing condition. The appearance of a maximum enhanced $\tau - \omega$ curve from the analysis on the data may be the result of further increases in force by increased cross-bridge stretch being offset by increased rate of detachment.

Joyce *et al.* (1969) also found that at low stimulus rates lengthening of a muscle resulted in a reduction of force over the isometric force at that stimulus rate. They suggested that the reduced stimulus rates resulted in the cross-bridges being less likely to remain attached and therefore cycle more quickly. This would agree with the geometric picture of the sarcomere as the cross-bridges would be less likely to be stretched to long lengths and placed in advantageous alignments with these high rates of cycling.

The results of Joyce *et al.* (1969) indicate that isometric contractions at sub-maximal stimulus rates generate the highest forces. Joyce and his colleagues postulate that:

> "cross links are only slowly broken down as long as they remain in the position at which they were formed, but are more likely to be broken down when they are distorted by movement in either direction."

The idea that isometric contractions are more efficient than concentric, and even eccentric contractions at sub-maximal activation, is supported by work on the short range stiffness (SRS) of muscle. The SRS is the range of movement were the cross-bridges can exert force without having to detach and reattach at other sites. After this range of movement has been passed the stiffness of the muscle drops significantly (Morgan, 1977; Rack and Westbury, 1974) Morgan *et al.* (1978) show that kangaroos are more efficient, measured by oxygen uptake, than other bipeds at similar running speeds. They argue that this is due to the length of their Achilles tendon. The large extensions possible with this long tendon allows the kangaroos contractile tissue to shorten and lengthen within the range of SRS.

These studies indicate how the rate of cross-bridge cycling effects the stiffness and efficiency of a muscle. The greater the velocity of stretch the less stiff the muscle may become as reported by Flitney and Hirst (1978) who show how sarcomere stiffness reduces dramatically once cross-bridge slippage occurs. The efficiency of working within the SRS is analogous to the special nature of isometric contractions at sub-maximal stimulus rates reported by Joyce *et al.* (1969). Some internal movement may in fact be present in the muscle but as long as it is within the SRS the force will remain high as very little cross-bridge detachment will occur.

The above observations highlight the importance of cross-bridge cycling rate in the development of force and the stiffness of the sarcomere. The 'geometric' explanation of force enhancement is compatable with these results, adding that cross-bridge length and alignment also play an important role.

One possible reason that the generated force of a muscle reaches a maximum in the eccentric velocity range could be due to the cross-bridges reaching their maximum length and most efficient angle of pull. Another possibility is that the rate of cross-bridge cycling could increase to a level where it counteracts any further gains in favourable length and angle of pull. The results of Thomson (1983) showed that the longest amplitude of stretch exhibited the lowest FE and this may be due to a high cycling rate.

It is possible that a high velocity and amplitude of stretch causes a reduction in the stimulus rate in human muscle. On rare occasions subject AC mentioned that he could not 'hold' the stretch contraction, and in the results of Chapman *et al.* (1985) where 7 subjects were used, some felt that their muscles 'gave' when trying to arrest the back-swing of a large moment of inertia. While this reduced stimulation is a possibility, the nature of the muscle is to protect the joint and in the majority of cases it appeared that the subjects were able to resist maximally the stretch throughout the movement. In fact on occasion the subject could feel an excess force generated at the extreme range of movement which was large enough to cause concern over possible injury. While the problem of measuring the level of activation in human muscle *in situ* remains, much work in this area will remain speculative.

It has been proposed that the analyses conducted present a logical criticism of the two-element model and that the hypothesis presented is a viable explanation of the phenomenon of force enhancement. The existence of an upper limit to force enhancement appears defensible but why the

results from the back-swing and stretch experiments fitted a typical force-velocity relationship better than the rest data is open to speculation. One possibility is that the presence of a stretch results in faster activation which in turn allows the human subject to grade his response more efficiently. Similarly the favourable alignment of the cross-bridges to force production could allow for a more graded response.

Often a human performer will include a back-swing in his movement even when maximal force output is not required. It is often felt that one is better able to 'time' a movement this way. This may be supported by the improved repeatability of the back-swing trials observed in this research. The mechanism for this may be an increased activation rate, but is certainly unkown at present. It should be remembered however, that the data analysed was from maximal contractions and timing was not a factor in the required movement. It would be interesting to examine the response of human muscle to stretch during sub-maximal contractions. It is possible that subjects can better grade their responses to target force values after a back-swing.

While it is possible to model the 'average' mechanical output from human skeletal muscle qualitatively and often quantatively, there is no doubt that many approximations are being made. The exact relationship between length, velocity, activation, and enhancement (if any) in the whole muscle models developed is governed by cross-bridge dynamics and bio-chemical reactions. To model these variables becomes prohibitive in time and money in whole body movements. Nevertheless, the exact anatomical structure of muscle must not be forgotten when we teach and model with systems like the two-element model. To lose sight of the detailed knowledge we have gained about the 'micro', would result in little being gained from the knowledge 'macro' models supply.

REFERENCES

Abbott, B.C. & Aubert, X.M. (1952). The force exerted by active striated muscle during and after change of length. *J.Physiol.*, **117**, 77–86.

Abbott, B.C., Bigland, B., & Ritchie, J.M. (1952). The physiological cost of negative work. *J.Physiol.*, **117**, 380–390.

Abbott, B.C. & Bigland, B. (1953). The effects of force and speed changes on the rate of oxygen consumption during negative work. *J.Physiol.*, **120**, 319–325.

Anderson, F. (1967). Registration of the pressure power (the force) of the body on the floor during movements, especially vertical jumps. *Biomech.I, 1st Int.Seminar, Zurich,* 87–89.

Asmussen, E. & Bonde-Peterson, F. (1974). Storage of elastic energy in skeletal muscle in man. *Acta.Physiol.Scand.*, **91**, 385–392.

Asmussen, E. & Sorensen, N. (1971). The "wind-up" movement in athletics. *Le Travail Human,* **34**, 147–156.

Baildon, R.W.A. & Chapman, A.E. (1983a). A new approach to the human muscle model. *J.Biomech.*, **16–10**, 803–809.

Baildon, R.W.A. & Chapman, A.E. (1983b). Mechanical properties of a single equivalent muscle producing forearm supination. *J.Biomech.*, **16–10**, 811–819.

Bawa, P., Mannard, A. & Stein, R.B. (1976). Predictions and experimental tests of a visco-elastic muscle model using elastic and inertial loads. *Biol.Cybernetics,* **22**, 139–145.

Bigland, B. & Lippold, O.C.J. (1954a). Relation between force, velocity and integrated electrical activity in human muscle. *J.Physiol.*, **123**, 214–224.

Bigland, B. & Lippold O.C.J. (1954b). Motor unit activity in the voluntary contraction of human muscle. *J.Physiol.*, **125**, 322–335.

Bosco, C., Ito, A., Komi, P.V., Luhtanen, P., Rusko, H., & Viitasalo, J.T. (1982). Combined effect of elastic energy and myoelectrical potentiation during stretch-shortening cycle exercise. *Acta.Physiol.Scand.*, **114**, 557–565.

Bosco, C. & Komi, P.V. (1979). Potentiation of the mechanical behaviour of the human skeletal muscle through pre-stretching. *Acta.Physiol.Scand.*, **106**, 467–472.

Calvert, T.W. & Chapman A.E. (1977). The relationship between the surface EMG and force transients in muscle: simulation and experimental studies. *Proc.IEEE.*, **65–5**, 682–689.

Cavagna, G.A. (1977). Storage and utilization of elastic energy in skeletal muscle. *Exercise and Sports Sci.Rev.*, **5**, 89–129.

Cavagna, G.A. & Citterio, G. (1974). Effect of stretching on the elastic characteristics and the contractile component of frog striated muscle. *J.Physiol.*, **239**, 1–14.

Cavagna, G.A., Dusman, B. & Margaria, R. (1968). Positive work done by a previously stretched muscle. *J.Appl.Physiol.*, **24**, 21–32.

Cavanga, G.A. & Kaneko, M. (1977). Mechanical work and effieciency in level walking and running. *J.Physiol.*, **268**, 467–481.

Cavagna, G.A., Komarek, L., Citterio, G. & Margaria, R. (1971). Power output of the previously stretched muscle. *Med.and Sport.*, **262**, 639–657.

Cavagna, G.A., Thys, H. & Zamboni, A. (1976). The sources of external work in level walking and running. *J.Physiol.*, **262**, 639–657.

Chapman, A.E. (1975). The investigation of mechanical models of muscle based upon direct observations of voluntary dynamic human muscular contraction. Ph.D. thesis, University of London.

Chapman, A.E. (1980). The effect of 'Wind-Up' on forearm rotational velocity. *Can.J.Appl.Sports Sci.* 5, 215–219.

Chapman, A.E. (1985). The mechanical properties of human muscle. *Exercise and Sports Sci.Rev.*, **13**, 443–501.

Chapman, A.E., Caldwell, G.E. & Selbie, W.S. (1985). Mechanical output following muscle stretch in forearm supination against inertial loads. *J.Appl.Physiol.*, **59**, 78–86.

Chapman, A.E. & Calvert, T.W. (1979). Estimations of active-state from E.M.G. recordings of human muscular contraction. *Electromyogr.Clin.Neurophysiol.*, **19**, 199–222.

Chapman, A.E. & Harrower, P.T. (1977). Linear approximations of muscle mechanics in isometric contractions. *Biol.Cybernetics*, **27**, 1–8.

Crowe, A. (1970). A mechanical model of muscle and its application to the intrafusal fibres of the mammalian muscle spindle. *J.Biomech.*, **3**, 583–592.

Curtin, N.A. & Woledge, R.C. (1979). Chemical change, production of tension and energy following stretch of active muscle of frog. *J.Physiol.*, **297**, 539–550.

Edman, K.A.P. (1975). Mechanical deactivation induced by active shortening in isolated muscle fibres of the frog. *J.Physiol.*, **246**, 255–275.

Edman, K.A.P. (1978). Maximum velocity of shortening in relation to sarcomere length and degree of activation of frog muscle fibres. *J.Physiol.*, **278**, 9P–10P.

Edman, K.A.P. (1980). Depression of mechanical performance by active shortening during twitch and tetanus of vertebrate muscle fibres. *Acta.Physiol.Scand.*, **109**, 15–26.

Edman, K.A.P. Elzinga, G. & Noble, M.I.M. (1978a). Enhancement of mechanical performance by stretch during tetanic contractions of vertebrate skeletal muscle fibres. *J.Physiol.*, **281**, 139–155.

Edman, K.A.P., Elzinga, G. & Noble M.I.M. (1978b). Further characterization of the enhancement of force by stretching during activity in single fibres of the frog. *J.Physiol.*, **280**, 35p–36p.

Elftman, H. (1966). Biomechanics of muscel with particular application to studies of gait. *J.of Bone and Joint Surgery*, **48** A, 363–377.

Fenn, W.O. (1930). Frictional and kinetic factors in the work of sprint running. *Am.J.Physiol.*, **92**, 582–611.

Fenn, W.O. (1938). The mechanics of muscular contraction in man. *J.Appl.Physics*, **9**, 165–177.

Fenn, W.O. & Marsh, B.S. (1935). Muscular force at different speeds of shortening. *J.Physiol.*, **85**, 277–297.

Fitzhugh, R. (1977). A model of optimal voluntary muscular control. *J.Math.Biol.*, **4**, 203–236.

Flitney, F.W. & Hirst, D.G. (1978). Cross-bridge detachment and sarcomere 'give' during stretch of active frog's muscle. *J.Physiol.*, **276**, 449–465.

Gilman, L. & Rose, A.J. (1976). APL. An interactive approach (2nd edition). John Wiley & Sons, New York.

Gordon, A.M., Huxley, A.F. & Julian, F.J. (1966) The variation in isometric tension with sarcomere length in vertebrate muscle fibres. *J.Physiol.*, **184**, 170–192.

Grieve, D.W. (1969). A device called the POLGON for measurement of the orientation of parts of the body relative to a fixed external axis. *J.Physiol.*, **201**, 70P.

Grieve, D.W., & Cavanagh, P.R. (1974). The validity of quantitative statements about surface electromyograms recorded during locomotion. *Scand.J.Rehab.Med.Suppl.*, 3, 19–25.

Griffiths, P.J., Guth, K., Kuhn, H.J. & Ruegg, J.C. (1980). Cross-bridge slippage in skinned frog muscle fibres. *Biophys.Struct.Mech.*, 7, 107–124.

Guth, K., Kuhn, H.J., Herzig, J.W. & Rugg, J.C. (1977). Evidence for cross-bridge slippage in a stretched muscle fibre. *Experientia*, **34/9**, 1183–1184.

Hatze, H. (1973). A theory of contraction and a mathematical model of striated muscle. *J.Theor.Biol.*, **40**, 219–246.

Hatze, H. (1981). Analysis of stretch responses of a myocybernetic model of muscle fibre. *Biol.Cybernetics*, **39**, 165–170.

Hill, A.V. (1938). The heat of shortening and dynamic constants of muscle. *Proc.Roy.Soc.Lond.*, **B126**, 136–195.

Hill, A.V. (1950). The series elastic component of muscles. *Proc.Roy.Soc.Lond.*, **B137**, 273–280.

Hill, T.L., Eisenberg, E., Chen, Y.D. & Podolsky, R.J. (1975). Some self-consistent two-state sliding filament models of muscle contraction. *Biophys.J.*, **15**, 335–372.

Houk, J.C. (1963).A mathematical model of the stretch reflex in human muscle systems. M.S. Thesis, Massachussetts Institute of Technology, Cambridge. Reported in Milhourn, H.T. (1968). The application of control theory to physiological systems. W.B.Saunders Co., London.

Huck, S.W., Cormier W.H. & Bounds, W.G. (1974). Reading Satistics and Research. Harper & Row, New York.

Huxley, A.F. (1957). Muscle structures and theories of contraction. *Prog.Biophys.Chem.*, 7, 255–318.

Huxley, A.F. & Taylor, R.E. (1958). Local activation of striated muscle fibres. *J.Physiol.*, **144**, 426–441.

Joyce, G.C. & Rack, D.R. (1969). Isotonic lengthening and shortening movements of cat soleus muscle. *J.Physiol.*, **204**, 475–491.

Joyce, G.C., Rack, P.M.H. & Westbury, D.R. (1969). The mechanical properties of cat soleus muscle during controlled shortening movements. *J.Physiol.*, **204**, 461–474.

Julian, F.J. & Sollins, M.R. (1973). Regulation of force and speed of shortening in muscle contraction. *Cold Spring Harbour Symp.Quant.Biol.*, 37, 635–646.

Katz, B. (1939). The relation between force and speed in muscular contraction. *J.Physiol.*, **96**, 45–64.

Kaneko, M., Fuchimoto, T., Toji, H. & Suei, K. (1983). Training effect of different loads on the force –velocity relationship and mechanical power output in human muscle. *Scand.J.Sports Sci.*, **5**, 50–55.

Komi, P.V. (1971). Relationship between muscle length, muscle tension, EMG, and velocity of contraction under eccentric and concentric contractions. *IVth Int.Congress of Electromyography, Brussels.*

Komi, P.V. (1973). Measurement of the force-velocity relationship in human muscle under concentric and eccentric contractions. *Biomech.III, 3rd Int.Seminar, Rome*, 224–229.

Komi, P.V. & Bosco, C. (1978). Utilization of stored elastic energy in leg extensors by men and women. *Med.Sci.Sports* **10**, 261–265.

Lambert, C.R., Gladden, L.B. & Stainsby W.N. (1979). Length-dependent activation of *in situ* canine muscle. *Am.J.Physiol.*, **237**, 38-42.

Marey, M. & Demeny, M.G. (1885b). Locomotion humaine, mecanisme du saut. *Comptes Rendus Hebomadaires des Seances de l'Academie des Sciences (Paris)*, **101**, 489-494.

Morgan, D.L. (1977). Separation of active and passive components of short-range stiffness of muscle. *Am.J.Physiol.*, **232**, 45-49.

Morgan, D.L., Proske, U. & Warren, D. (1978). Measurements of muscle stiffness and the mechanism of elastic storage of energy in hopping kangaroos. *J.Physiol.*, **282**, 253-261.

Parmley, W.W., Yeatman, L.A. & Sonneblick, E.H. (1970). Differences between isotonic and isometric force-velocity relations in cardiac and skeletal muscle. *Am.J.Physiol.*, **219**, 546-550.

Petrofsky, J.S. & Phillips, C.A. (1980). The influence of recruitment order and fibre composition on the force-velocity relationship and fatiguability of skeletal muscle in cat. *Med.Biol.Eng.Comp.*, **18**, 381-390.

Pierrynowski, M.R. (1982). A physiological model for the solution of individual muscle forces during normal human walking. Ph.D. thesis, Simon Fraser University.

Pollack, G.H. (1983). The cross-bridge theory. *Physiol.Rev.*, **63**, 1049-1113.

Rack, P.M.H. & Westbury, D.R. (1969). The effects of length and stimulus rate on tension in the isometric cat soleus muscle. *J.Physiol.*, **204**, 433-460.

Rack, P.M.H. & Westbury, D.R. (1974). The short range stiffness of active mammalian muscle and its effect on mechanical properties. *J.Physiol.*, **240**, 331-350.

Schutz, R.W., Smoll, F.L. & Gessaroli, M.E. (1983). Multivariate Statistics: A self-test guide to their utilization. *Res.Quart.for Exercise and Sport*, **54-3**, 255-263.

Sugi, H. (1972). Tension changes during and after stretch in frog muscle fibres. *J.Physiol.*, **225**, 237-253.

Sugi, H. & Pollack, G.H. (editors), (1979). Cross-bridge mechanism in muscle contraction. University Park, Baltimore.

Thomson, D.B. (1983). The stretch response of human skeletal muscle *in situ*. M.Sc. thesis, Simon Fraser University.

University of California, Los Angeles, (1981). BMDP Statistical Software. University of California Press, Los Angeles.

Wilkie, D.R. (1950). The relationship between force and velocity in human muscle. *J.Physiol.*, **110**, 249-280.

Wilkie, D.R. (1976). Muscle (2nd Edition). Edward Arnold, London.

Zahalak, G.I. (1981). A distribution-moment approximation for kinetic theories of muscular contraction. *Math.Biosc.*, **55**, 89-114.

Zheng, Y.-F., Hemami, H. & Stokes B.T. (1984). Muscle dynamics, size principle, and stability. *IEEE Transactions Biomed.Eng.*, **31-7**, 489-497.