UNIVERSITY OF CALGARY

# Force Depression in Human Quadriceps Femoris During Voluntary Contractions

by

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# ABSTRACT

Force depression following muscle shortening has been studied extensively using artificial electrical stimulation, but has not been studied at all during voluntary contractions. There is the possibility that force depression properties may differ conceptually between muscle stimulated artificially and muscle contracted voluntarily. Therefore, the purpose of this thesis was to investigate force depression following muscle shortening and its properties related to speed and distance of shortening for human *voluntary contraction*.

The isometric force produced immediately following muscle shortening was depressed compared to the corresponding force during purely isometric contractions for voluntary contractions and was typically accompanied by constant muscle activation. Therefore, it was concluded that force depression is an actual property of skeletal muscle rather than an artefact of electrical stimulation. However, the amount of force depression was independent of the speed and distance of shortening; a result that was in direct contrast to the findings reported on artificially stimulated muscle.

# PREFACE

Chapters 3 and 4 of this thesis are based on the following manuscripts:

Lee, H.-D., Suter, E. and Herzog, W. (1998) Force depression in human quadriceps femoris following voluntary shortening contractions. Accepted with revisions *Journal of Applied Physiology*.

Lee, H.-D., Suter, E. and Herzog, W. (1999) Effects of speed and distance of muscle shortening on force depression during voluntary contractions. Submitted to *Journal of Biomechanics*.

Chapters 3 and 4 are stand alone papers. Consequently, there is some repetition in the introduction and methods sections.

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## DEDICATION

*To Jung Min and Catherine*

*Mom and Dad*

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# CHPATER 1: INTRODUCTION

The maximal isometric force produced by a muscle or muscle fibre subsequent to shortening is smaller than the force when the muscle or muscle fibre contracts purely isometrically. This phenomenon is termed force depression following muscle shortening (Herzog and Leonard, 1997), and has been demonstrated consistently in a variety of preparations (e.g. isolated muscle fibres [Edman *et al.*, 1993; Granzier and Pollack, 1989; Julian and Morgan, 1979; Sugi and Tsuchiya, 1988]; in-situ muscles [Abbott and Aubert, 1952; Maréchal and Plaghki, 1979; Herzog and Leonard, 1997]; in-vivo human muscle [de Ruiter *et al.*, 1998]). It has been well accepted that the amount of force depression increases with increasing distance of shortening, and decreases with increasing speed of shortening (de Ruiter *et al.*, 1998; Herzog and Leonard, 1997; Maréchal and Plaghki, 1979; Sugi and Tsuchiya, 1988). In an effort to explain the mechanism of force depression following muscle shortening, three basic hypotheses have been proposed; (i) sarcomere non-uniformity, (ii) stress-induced cross-bridge inhibition, and (iii) increase in proton and inorganic phosphate concentrations. However, these hypotheses are still under debate, and no consensus as to the correct mechanism causing force depression following muscle shortening has been reached.

What has drawn so many scientists to investigate the phenomenon of force depression following muscle shortening? First, force depression following muscle shortening is in conflict with the generally accepted idea that the force-length relationship of muscle depends exclusively on the instantaneous contractile conditions and does not depend on

the time history of contraction (Ramsey and Street, 1940; Gordon *et al.*, 1966). Second, force depression following muscle shortening is not contained as an intrinsic property in the primary theory of muscle contraction; the cross-bridge theory (Huxley, 1957; Huxley and Simmons, 1971).

In spite of the abundance of studies demonstrating force depression following muscle shortening, none of the previous investigations was performed for in-vivo human *voluntary contractions*. Although a recent study by de Ruiter *et al.* (1998) was the first to demonstrate force depression following muscle shortening in *human skeletal muscle*, muscle contractions were induced by artificial, electrical stimulation, as it had been in previous studies. Electrical stimulation of the muscle or nerve is non-physiological in the sense that all motor units are recruited almost simultaneously, at the same frequency, and at a nearly constant current. During voluntary contractions, motor units are recruited asynchronously, non-periodically, and at different frequencies (Basmajian and de Luca, 1985; Bellemare *et al.*, 1983; Bigland-Ritchie *et al.*, 1983).

Therefore, the question arose: "Does force depression following muscle shortening occur during in-vivo human *voluntary contractions*?" It was speculated that if force depression following muscle shortening does not occur during human voluntary contractions, it might be an artefact of electrical stimulation rather than a true property of skeletal muscle.

As a first attempt to investigate force depression for human muscle during *voluntary contractions*, this thesis was aimed at:

1) Testing if force depression following muscle shortening does indeed occur during human voluntary contractions, and if so,
2) To test if force depression is related to the speed and distance of muscle shortening during human voluntary contractions, as it is during artificial electrical stimulation.

It was hypothesised that force depression following muscle shortening would be different during human voluntary contractions than it is during electrically stimulated contractions.

The thesis continues with a review of the literature in chapter 2, providing information on relevant issues related to force depression following muscle shortening. Chapters 3 and 4, based on two stand-alone manuscripts, describe the experiments that were performed to show the existence of force depression following muscle shortening for human voluntary contractions, and how force depression relates to the speed and distance of shortening, respectively. Chapter 5 contains a summary of results and the general conclusions of the thesis.

# CHAPTER 2: LITERATURE REVIEW

The literature review presented here will provide background and rationale for the work done in the present study. Sections 2.1 and 2.2 describe the primary mechanism of muscle contraction, the cross-bridge theory and a primary property of skeletal muscle, the force-length relationship, respectively. These two sections provide some rationale of why force depression following muscle shortening has been an interesting issue to researchers in the area of muscle mechanics. Sections 2.3 and 2.4 provide an overview of relevant previous work, and some proposed mechanisms of force depression following muscle shortening, respectively. Section 2.5 presents some of the differences associated with voluntary and electrically elicited muscle contractions

## 2.1 Current Mechanism of Muscle Contraction

Force depression following muscle shortening has been consistently observed experimentally and has been well accepted in the scientific community. However, it is not explained by any mechanism of muscle contraction proposed to date. In spite of the fact that the primary mechanism of muscle contraction, the cross-bridge theory (Huxley, 1957; Huxley and Simmons, 1971), has universally been used in the field of muscle mechanics, the cross-bridge theory does not account for the effects of force depression following muscle shortening.

Prior to the 1950's, the continuous-filament theory was widely accepted as the basic mechanism for muscle contraction. The continuous-filament theory was based on the idea that the contractile material, which was in the form of continuous filaments, shortened by some kind of folding or coiling (Huxley, 1974). In 1954, two microscopic

studies (Huxley and Hanson, 1954; Huxley and Niedergerke, 1954) proposed a new concept of muscle shortening. The idea was that, upon activation, thin (actin) and thick (myosin) filaments, which are arranged in an interdigitating structural format in a sarcomere (Figure 2-1), slide past each other, and so, produce muscle shortening and force (the sliding filament theory).

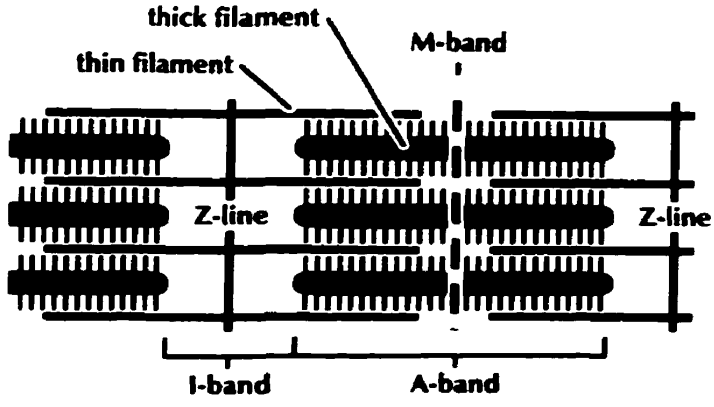


Figure 2- 1. Schematic illustration of the basic contractile element of skeletal muscle, the sarcomere (from Pollack, 1990).

In 1957, Huxley proposed a mechanism of muscle contraction (the cross-bridge theory) aimed at explaining "what makes the filaments slide?" The cross-bridge theory can be described in a simple conceptual way as follows: side projections (cross-bridges) arising from the thick (myosin) filaments spontaneously attach to specialised sites on the thin (actin) filaments, and when attached, the cross-bridges pull the thin past the thick filaments, thereby producing force and contraction (Epstein and Herzog, 1998).

There are several basic assumptions underlying the cross-bridge theory. Cross-bridges project laterally from the thick filaments and they are arranged in a regular pattern. These cross-bridges are assumed to be individual and independent force generators.

Because of the regular arrangement of the cross-bridges on the thick filament, the number of cross-bridges available in the thin-thick filament overlap zone is proportional to the length of the overlap zone. Upon activation, the total force in a muscle is thought to depend on the proportion of attached cross-bridges and on the average force per cross-bridge. The proportion of attached cross-bridges, in turn, depends on the activation, length, rate of change in length, and the attachment/detachment constants of the muscle. If the cross-bridge theory was correct, the isometric muscle force would depend exclusively on the length of the sarcomeres, and therefore, the muscle or fibre length, but not on the time history of contraction. The concept of force depression following muscle shortening is not part of the mathematical description of the cross-bridge theory, and to date has no molecular or structural explanation.

### 2.2 Force-Length Relation of Skeletal Muscle

The force-length relationship of muscle is defined by the maximal isometric force a muscle, muscle fibre, or sarcomere can exert as a function of its length (Herzog and ter Keurs, 1988).

Ramsey and Street (1940) demonstrated experimentally that the isometric force in isolated frog muscle fibres depends on the length at which it is held, declining steeply on either side of some specific length, typically referred to nowadays as the optimum length. Gordon *et al.* (1966) investigated the force-length relationship in isolated frog muscle fibres using a photo-electric spot follower. The photo-electric spot follower was used to ensure uniformity and isometricity of sarcomere length during contraction. The general results of Gordon *et al.* (1966) agreed with those of Ramsey and Street (1940). The peak

of the force-length relation curve was found to consist of a plateau between sarcomere lengths of 2.05 and 2.25 $\mu$m. The decline of force above the plateau was close to a straight line from maximum tension at a sarcomere length of 2.25 $\mu$m to zero at 3.65 $\mu$m. The decline in force at length below the plateau became steeper at a sarcomere length of 1.65 $\mu$m, and the tension approached zero at about 1.3 $\mu$m. This result was explained with critical stages of overlap of the thick-thin filaments (Figure 2-2), which is associated with the cross-bridge theory (Huxley, 1957; Huxley and Simmons, 1971).

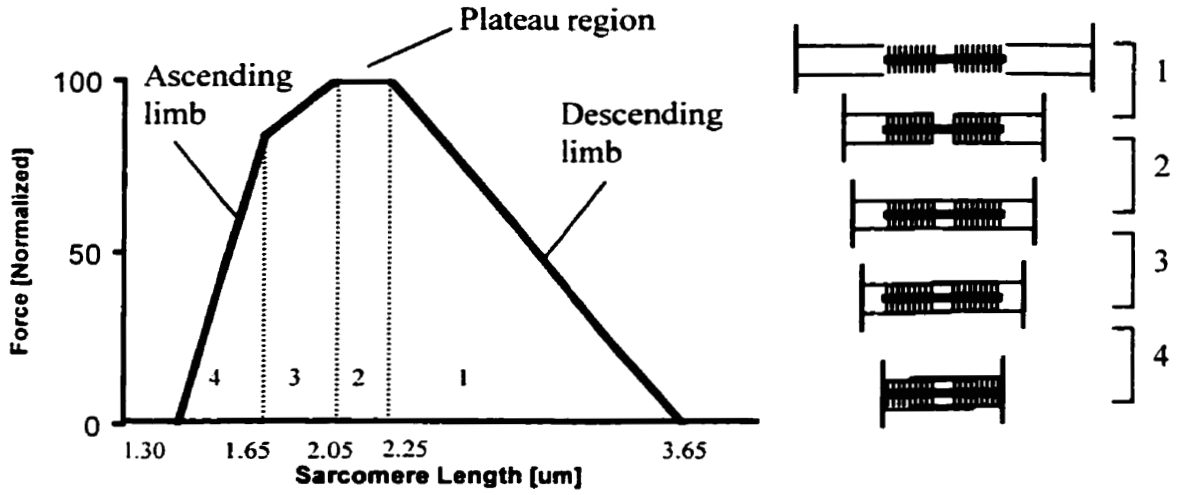


Figure 2- 2. The sarcomere force-length relation in isolated frog muscle fibre and critical stages in the degree of overlap of the thick-thin filaments. Thick filament – 1.60 $\mu$m; thin filament including the thickness of the Z-line (0.05 $\mu$m) – 2.05 $\mu$m; Middle region of the thick filament without the cross-bridges – 0.20 $\mu$m.

The isometric force is directly proportional to the number of attached cross-bridges in the thick-thin filament overlap zone for sarcomere lengths above 2.05 $\mu$m. At sarcomere lengths below 2.05 $\mu$m, the isometric force starts to decrease. Although the exact mechanisms for this loss in force are not known, several suggestions have been made.

These include (i) meeting of the thin filaments at the centre of the sarcomere, (ii) formation of "double overlap" of the thin filaments by sliding past each other at the centre of the sarcomere, and (iii) resistance to shortening created by crumpling or folding of the thick filament which might be caused by a collision of thick filaments with the Z-line.

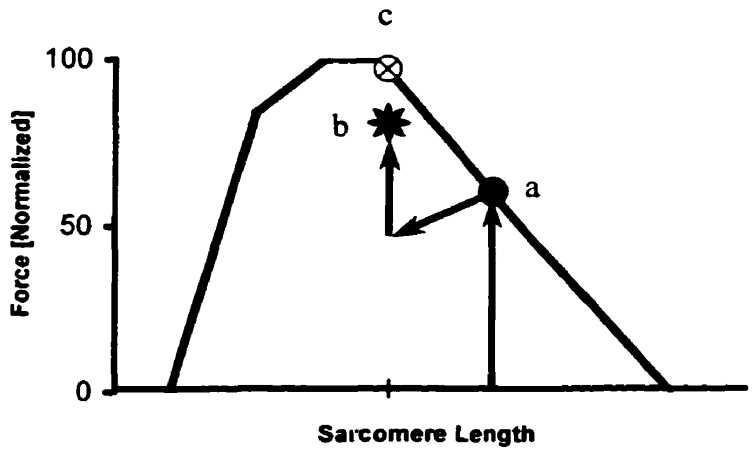


Figure 2- 3. Schematic illustration of force-length relationship, and the conceptual force trace for an isometric contraction following muscle shortening. The isometric force of a sarcomere generated at a given length (a) does not climb up to the new force level (c) along the slop of the force-length relationship, when the sarcomere shortens to a new length. The force decreases during shortening and reaches a new force level (b) following shortening, which is smaller than that predicted by the force-length relationship.

One important point in interpreting the force-length relationship is that the relationship is not a continuous function of its length. Rather, it is a static relationship composed of a series of independent force measurements at a series of different isometric lengths. Therefore, if a muscle or fibre is stimulated at a certain length and stretched or released to a new length, the isometric force of the muscle or fibre at the new length would be higher or lower, respectively, than the force predicted by the isometric force-length relationship (Figure2-3). The latter case is called force depression following muscle shortening. Not only is the isometric force following shortening/lengthening different than that predicted

by the force-length relationship, but also the force is different than the isometric force even when the muscle is shortening/lengthening at very slow speeds.

### 2.3 Previous Studies on Force Depression Following Muscle Shortening

Force depression following muscle shortening has been demonstrated in a variety of preparations from amphibian and mammalian skeletal muscle (Table 2-1).

Table 2-1. Summary of studies on force depression following muscle shortening with different preparations

| Researcher | Year | Specimen |
|---|---|---|
| Abbott and Aubert | 1952 | Sartorii of frog and toad<br>Coracomandibulars of dogfish |
| Julian and Morgan | 1979 | Single twitch fibre of frog |
| Maréchal and Plaghki | 1979 | Sartorii of frog |
| Sugi and Tsuchiya | 1988 | Fibres from tibialis anterior of frog |
| Granzier and Pollack | 1989 | Fibres from semitendinosus of frog |
| Edman *et al.* | 1993 | Fibres from anterior tibialis of frog |
| Herzog and Leonard | 1997 | Cat soleus |
| De Ruiter *et al.* | 1998 | Human adductor pollicis |

The study of Abbott and Aubert (1952) is thought of as the first one to investigate systematically how muscle force changes during and after changes in length. Using sartorii of frog and toad, and coracomandibulars of dogfish, they demonstrated experimentally that the isometric force developed following shortening was lower than that developed during purely isometric contractions at the corresponding length. The amount of force depression has been found to be directly related to the distance of shortening and inversely related to the speed of shortening (de Ruiter et al., 1998; Herzog and Leonard, 1997; Maréchal and Plaghki, 1979; Sugi and Tsuchiya, 1988: Figure 2-4).

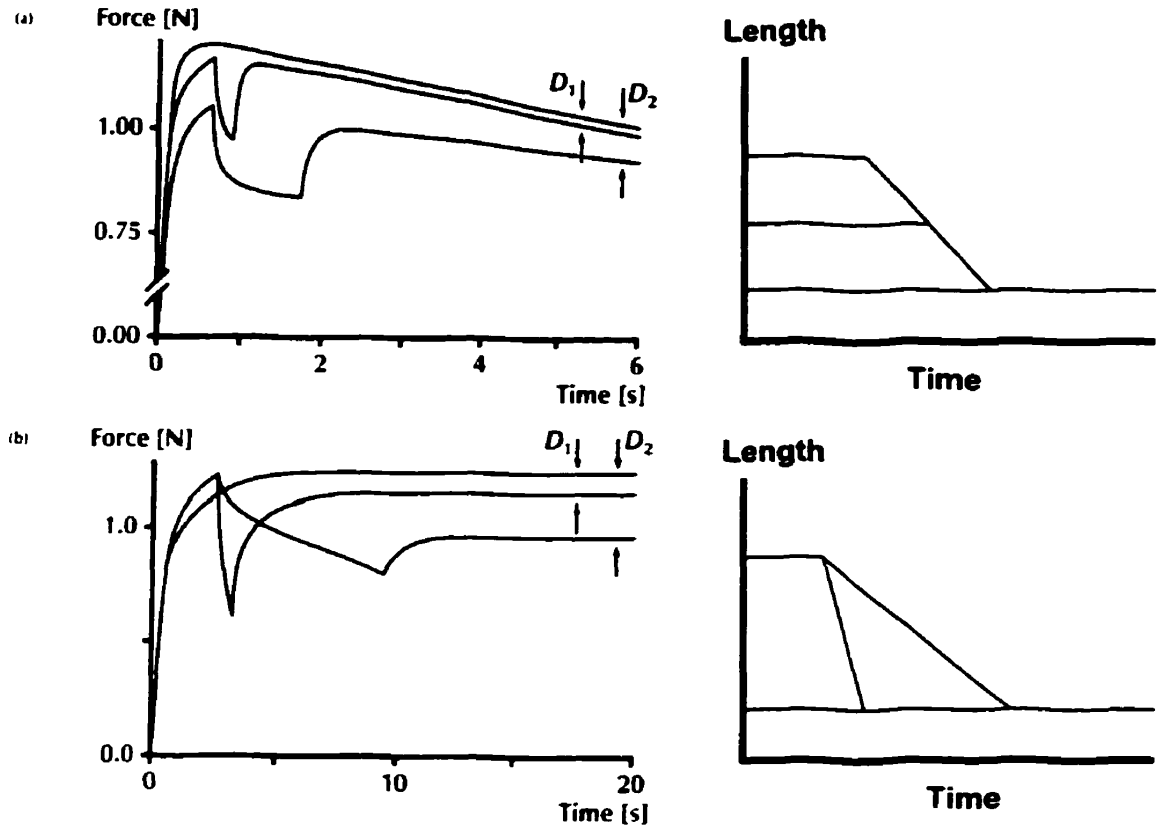


Figure 2- 4. Illustration of force depression following muscle shortening as a function of the distance (from Maréchal and Plaghki, 1979; (a) and the speed (from Abbott and Aubert, 1954; (b) of shortening.

Compared to studies on isolated muscles or fibres using electrical stimulation, the studies of Herzog and Leonard (1997) and de Ruiter *et al.* (1998) used an in-situ muscle preparation that was stimulated via the nerve.

Herzog and Leonard (1997) showed force depression following muscle shortening and its properties in in-situ cat soleus. The cat soleus muscle was activated through a nerve cuff implanted on the tibial nerve. For testing the influence of the distance of shortening on force depression, they used different distances of shortening ranging from 0.25 mm to 8 mm and a constant speed of shortening of 4 mm/s. For testing the influence of speed of

shortening on force depression, they used shortening speeds ranging from 2 to 256 mm/s and a constant distance of shortening (4 mm). The amount of force depression was increased with increasing amounts of shortening preceding the isometric contraction, and was decreased with increasing speeds of shortening. The results of this study agreed well with results obtained in previous studies.

De Ruiter *et al.* (1998) demonstrated for the first time that there is force depression following muscle shortening in *human skeletal muscle* (adductor pollicis). They activated the adductor pollicis by electrical stimulation (30 % above the stimulus which produced maximal isometric tetanic force) of the ulnar nerve at the wrist. A maximal force depression of 37% was reported after 38° of shortening at 6.1 °/s. In addition, de Ruiter *et al.* (1998) showed that force depression following muscle shortening was directly related with angular displacement ($r^2$ > .98), i.e. the amount of muscle shortening, and was inversely related to the speed of muscle shortening.

### 2.4 Proposed Mechanisms of Force Depression Following Muscle Shortening

In spite of the fact that several mechanisms of force depression following muscle shortening have been proposed, no mechanism has been universally accepted in the scientific community. Sarcomere length non-uniformity is a commonly proposed mechanism of force depression following muscle shortening. Julian and Morgan (1979) suggested that sarcomere length non-uniformity might be the primary cause of force depression following muscle shortening. In subsequent studies by Edman *et al.* (1993), and Sugi and Tsuchiya (1988), this mechanism was further investigated. Edman *et al.* (1993) showed that shortening from a sarcomere length of 2.55 $\mu$m to slack length (2.04

$\mu$m) depressed the subsequent isometric force by about 13 %, and caused a marked non-uniformity of sarcomere length. They showed a positive correlation between the amount of force depression and the degree of non-uniformity of sarcomere length. Sugi and Tsuchiya (1988) reported that sarcomere length non-uniformity was more pronounced following slow compared to fast speed of shortening.

The idea underlying sarcomere length non-uniformity was as follows: there exist inherent differences in the force-generating capability along a muscle fibre. During shortening, segments reach different lengths because of the differences in shortening speed along the fibre. Such differences in segment length along the fibre last for the subsequent isometric contraction. Because all segments produce the same amount of force at different segment lengths during the isometric phase due to the fact that they are in series, some stronger segments generate force lower than their true isometric force in order to match the force of the weakest segment(s). Therefore, the measured isometric force following shortening will be lower than the true isometric force of purely isometric contraction in which sarcomere non-uniformity is much less produced.

There is evidence that sarcomere length non-uniformity cannot be the primary mechanism for force depression. The strongest evidence comes from the results of a study by Granzier and Pollack (1989). These investigators studied force depression on single fibres of frog semitendinosus in two separate experiments. In one experiment sarcomere lengths were kept uniform, while in the other experiment sarcomere length uniformity was not enforced. The results showed that force depression was similar following shortening independent of the amount of sarcomere length non-uniformity, indicating that sarcomere non-uniformity cannot be the main cause for force depression.

Maréchal and Plaghki (1979) proposed a mechanism for force depression following muscle shortening based on the idea of stress-induced cross-bridge inhibition. The idea of this mechanism is that the actin filaments which enter the overlap region during muscle shortening, are strained by muscle force. The strain on the thin filaments entering the overlap zone is thought to cause a reorientation of cross-bridge attachment sites, which in turn, is thought to cause a decrease in the probability of cross-bridge attachment in these strained filaments. This hypothesis was supported by the study of Herzog and Leonard (1997). These investigators showed a direct relationship between the amount of force depression and the amount of mechanical work done by the muscle during shortening. Since the amount of mechanical work is directly dependent on the force of the muscle during shortening and the amount of shortening, the close relationship between work and force depression was interpreted to support Maréchal and Plaghki's (1979) hypothesis. Although the study of Herzog and Leonard (1997) provided experimental evidence supporting the idea of stress-induced cross-bridge inhibition, no effort has been made to prove the mechanism itself.

Granzier and Pollack (1989) investigated the effects of pre-shortening on isometric force and isotonic velocity in single intact fibres from frog semitendinosus using a laser-diffraction method and a segment-clamp method that can be used to control the lengths of small segments within a fibre preparation. They showed that the isometric force is depressed following shortening and full recovery of force production required 5-10 minutes of rest. They proposed that an increase in proton ($H^+$) and inorganic phosphate ($P_i$) concentrations during shortening might cause force depression following muscle shortening. This mechanism of force depression requires that full isometric force can

only be achieved minutes after a shortening contraction. This idea was contradicted by observations of Abbott and Aubert (1952) and Herzog and Leonard (1997) who demonstrated that the isometric force following shortening was fully recovered after a time period of relaxation just long enough for the force to drop to zero.

### 2.5 Motor Unit Activity during Voluntary Contraction

In all published studies on force depression following muscle shortening, experiments were performed using artificial contractions elicited by electrical stimulation to the muscle or nerve. Electrically induced contractions are non-physiological because all motor units are recruited almost simultaneously and supramaximally, at a nearly constant current and frequency.

In the study of de Ruiter *et al.* (1998) on human adductor pollicis, stimulation of the muscle was produced with unidirectional square wave pulses of 0.1 ms duration and a constant current of 30 % higher than the stimulus that produced maximal isometric tetanic force. For in-situ cat soleus, Herzog and Leonard (1997) stimulated the soleus nerve at 100 Hz and 0.1 ms pulse duration, and with a current of 2-3 times the amount of the $\alpha$-motoneuron threshold.

During human voluntary contractions, motor units are recruited in an orderly fashion according to the motoneuron size (the size principle, Henneman, 1965): the small, slow-twitch motoneurons are recruited first, followed by the fast-twitch oxidative, and finally, the large, fast-twitch glycolytic motor units (Burke, 1986). Once a motor unit is recruited, it will remain active until the force declines, and when the force is lowered, the motor units are deactivated in the reverse order of activation (Enoka, 1994). For most

muscles, all motor units are recruited at around 50 - 85 % of the maximum force; subsequent increases in force are accomplished exclusively by increases in the motor unit discharge rates. During sustained maximal voluntary contractions, motor unit discharge rates change (Bigland-Ritchie *et al.*, 1983; Bellemare *et al.*, 1983) and different motor units fire at vastly different frequencies (Bellemare et al. 1983).

There is a possibility that muscle activation during maximal voluntary contractions might regulate muscle force by changing its discharge rates, and therefore might compensate for any loss of force caused by preceding shortening contraction, whereas muscle activation is fixed during artificial contractions by supramaximal electrical stimulation does not seem to allow such changes.

# CHAPTER 3: Force Depression in Human Quadriceps Femoris Following Voluntary Shortening Contractions

## 3.1 Introduction

The observation that the isometric muscle force redeveloped immediately after shortening contractions is smaller than the muscle force during purely isometric contractions at the corresponding length has been made in isolated skeletal muscle fibres (Edman *et al.*, 1993; Granzier and Pollack, 1989; Sugi and Tsuchiya, 1988) and in in-situ skeletal muscles (Abbott and Aubert, 1954; Herzog and Leonard, 1997; Maréchal and Plaghki, 1979). This phenomenon is referred to as force depression following shortening contractions (Herzog and Leonard, 1997). Its existence has been well accepted in the scientific community. Force depression is directly related to the amount of muscle shortening (Herzog and Leonard, 1997; Maréchal and Plaghki, 1979) and the force during the shortening phase (Abbott and Aubert, 1954; Herzog and Leonard, 1997), and is inversely related to the speed of shortening (Abbott and Aubert, 1954; Herzog and Leonard, 1997; Maréchal and Plaghki, 1979). Furthermore, force depression following shortening contractions is long lasting (Abbott and Aubert, 1954; Herzog and Leonard, 1997) and can be abolished instantaneously by deactivating the muscle for a period long enough so that the force drops to zero (Abbott and Aubert, 1954; Granzier and Pollack, 1989; Herzog and Leonard, 1997). Despite these consistent experimental observations in muscle fibres and whole muscle, the mechanism causing force depression following muscle shortening is a matter of intense debate.

Recently, force depression following muscle shortening was demonstrated for the first time in human skeletal muscles. De Ruiter *et al.* (1998) reproduced many of the results

obtained from *in situ* animal skeletal muscles in human adductor pollicis. Most importantly, they found a direct linear relationship between force depression and the magnitude of shortening ($r^2 > 0.98$) as well as force depression and force during shortening ($r^2 > 0.89$). De Ruiter *et al.* (1998) found a maximum force depression in the human adductor pollicis of 37.2 % with a shortening velocity of 6.1 °/s in thumb adduction, and concluded that these results had profound practical implications for intact human skeletal muscles.

De Ruiter *et al.* (1998) used artificial electrical stimulation of the ulnar nerve (20 and 50 Hz) to produce adductor pollicis contractions. Similarly, in all previous studies on entire muscles or muscle fibres, artificial stimulation of the preparations had been used. For entire muscles, electrical stimulation means that all (stimulated) motor units are recruited almost simultaneously, at the same frequency and at a nearly constant current.

During voluntary contractions, motor units are recruited asynchronously, and non-periodically, at different firing frequencies, and the magnitude of activation may change (Basmajian and de Luca, 1985; Bigland-Ritchie *et al.*, 1983). Therefore, it is not clear whether force depression following shortening occurs during *voluntary* contractions as it does during *electrically stimulated* contractions. The force depressions observed in the past could have been an artefact of electrical stimulation rather than represent an actual muscle property.

During voluntary contractions, force depression might be masked by an upregulation of muscle activation following muscle shortening. Such upregulation is possible during maximal voluntary contractions because motor unit pools are (typically) not recruited to their full extent in these situations (Basmajian and de Luca, 1985; Suter and Herzog,

1997; Suter *et al.*, 1996). Observations of increased muscle activation during maximal voluntary efforts have been made when the contractile conditions became unfavourable for large force production (e.g. at very short muscle length, Hasler *et al.*, 1994).

The purpose of this study was to investigate whether force depression was observed following maximal effort, voluntary shortening contractions in human skeletal muscles. It was hypothesized that there would be no observable force depression during maximal, voluntary contractions because any possible loss of force associated with muscle shortening would be compensated for by increased muscle activation.

## 3.2 Methods

### 3.2.1 Subjects

Ten healthy volunteers (7 males and 3 females; Age: 29.6 ± 7.4 yr.; Body height: 1.79 ± 0.13 m; Body weight: 77 ± 14 kg) participated in this study. The subjects were informed about all testing procedures and gave free informed consent to participate in this study. The study was approved by the Conjoint Ethics Committee of the University of Calgary.

### 3.2.2 Testing machine set-up

The subjects were seated on a strength-testing machine (CYBEX NORM™ Testing & Rehabilitation System, Lumex, INC., New York, U.S.A.), which was set up for left leg flexion/extension exercises. The subjects wore a double shoulder seat belt to stabilize the upper body. The distal ends of the thigh and shank were strapped to the test seat and the dynamometer arm, respectively. The rotational axis of the dynamometer was aligned

with the knee joint axis (i.e. the most prominent point on the lateral epicondyle of the femur). A zero knee angle was defined as the knee fully extended.

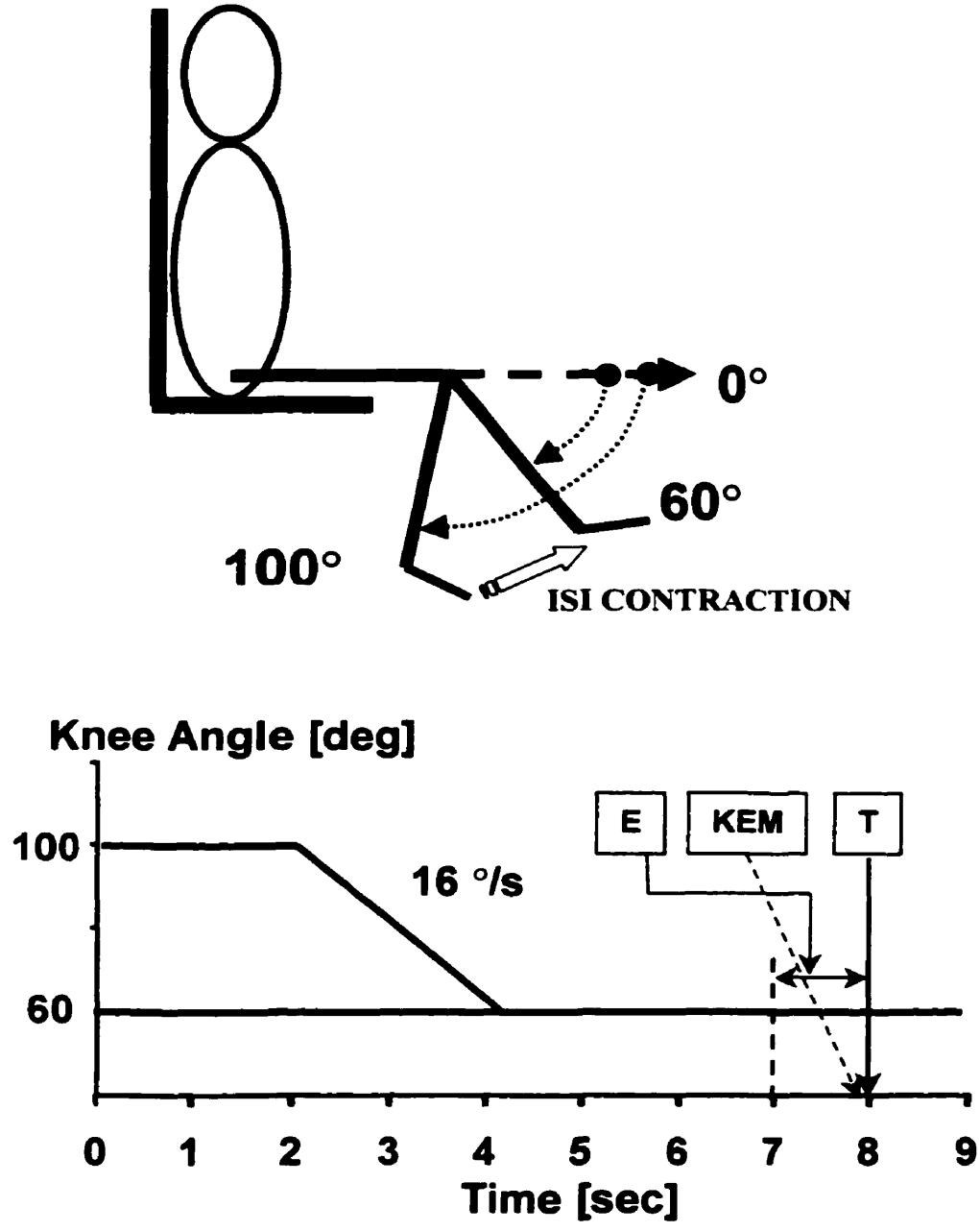


Figure 3- 1. Definition of the knee angles, and schematic illustration of the test contractions. KEM: Knee extensor moment; T: interpolated twitch; E: EMG analysis.

### 3.2.3 Test contractions

Subjects performed two different sets of voluntary contractions; isometric (ISO) and isometric-shortening-isometric contractions (ISI). For the isometric contractions, the subjects produced a maximal voluntary effort, isometric knee extension contraction at a 60° knee angle for approximately 9 s. For the isometric-shortening-isometric contractions, the subjects produced maximal, voluntary effort, isometric contractions for 2 s at a knee angle of 100°, followed immediately by a maximal effort shortening of the knee extensors from a knee angle of 100° to a knee angle of 60° at an angular speed of 16 °/s, and finally the subjects continued the maximal effort, isometric contractions for another 4-5 s at the 60° knee angle (Fig. 3-1). The duration of the ISI contractions was approximately 9 s.

### 3.2.4 Electromyography (EMG)

In order to assess the degree of muscle activation, bipolar Ag-AgCl surface electrodes were placed on the distal third of the vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) muscles. A ground electrode was placed on the patella. EMG signals for each muscle were passed through amplifiers located no further than 10 cm from the recording electrodes. Amplification was between 500 – 5000 and was kept constant for a given muscle within a subject throughout testing. All signals were band-pass filtered using cut off frequencies of 10 Hz and 1 kHz.

### 3.2.5 Interpolated twitch technique (ITT)

In order to evaluate the extent of motor unit recruitment, the twitch interpolation technique was used (Belanger and McComas, 1981; Hales and Gandevia, 1988; Suter and Herzog, 1997). Two carbon-impregnated rubber electrodes (4.5 × 10 cm) were thinly covered with a layer of conductive gel. One electrode was placed over the femoral nerve just distal to the inguinal ligament; the other electrode was placed at the distal end of the quadriceps muscle. During the knee extensor contractions, a doublet electrical stimulus (110 V, 8 ms interpulse interval, 0.8 ms pulse duration) was applied to the femoral nerve using a Grass S88 Muscle Stimulator with an isolation unit approved for human use (Quincy, MA, U.S.A.). Resting twitch moment (RTM) was measured by applying a doublet twitch to the relaxed muscle and recording the corresponding twitch moment. The interpolated twitch moment (ITM) was determined by superimposing a doublet twitch to the voluntarily contracting muscle and recording the additional moment produced by the electrical stimulation on top of the maximal voluntary moment. The extent of motor unit recruitment during the maximal voluntary contraction was calculated as (ITM/RTM)×100 %.

### 3.2.6 Experimental protocol

The protocol consisted of two parts; the measurement of test variables during the maximal voluntary contractions and the resting twitch moment.

Subjects performed 3-4 ISO contractions and 6-8 ISI contractions in a random order. For each contraction, EMG signals of VL, VM and RF were measured and an electrical twitch was applied approximately 8s following the start of the ISO or ISI contractions

(Figure 3-1). Following the ISO and ISI contractions, the resting twitch moment was measured three times at a 60°-knee angle. The average of the peak values of these three measurements was taken as the resting twitch moment used for calculating the extent of motor unit recruitment.

Since each voluntary contraction was performed at maximum effort, care was taken that the subjects had enough rest; i.e. at least 3 min., between contractions.

### 3.2.7 Data collection and Analysis

During the ISO and ISI contractions, knee extensor moment, angular displacement, interpolated twitch moment, and EMG signals were collected simultaneously at a sampling frequency of 2000 Hz on a microcomputer using an analogue-to-digital board with a resolution of 12 bits.

The moment values for each contraction were extracted 100 ms prior to applying the interpolated twitch. EMG signals for each contraction were extracted over a 1s period preceding the interpolated twitch. Root mean square (RMS) values from the combined EMG data of VL, VM and RF were calculated for the ISO and ISI contractions.

Prior to performing any statistical analysis, trials in which the peak moment did not reach at least 95 % of the absolute peak moment of the corresponding test contractions were excluded because they were deemed to not represent a maximal voluntary effort. Following the exclusion of trials, a one-tailed Student's *t*-test ($\alpha = 0.05$) for paired data was used for comparison of the mean values of the knee extensor moment, RMS of the EMG, and (ITM/RTM)×100 % from the ISO and ISI contractions. For the comparison of individual test trials, all possible comparisons among the accepted ISO and ISI

contractions were made within a subject. For example, if one subject had two accepted ISO ($ISO_1$ and $ISO_2$) and three accepted ISI ($ISI_1$, $ISI_2$ and $ISI_3$) contractions, six comparisons were made ($ISO_1$ vs. $ISI_{1,2,3}$ and $ISO_2$ vs. $ISI_{1,2,3}$).

### 3.3 Results

#### 3.3.1 Isometric knee extensor moments following shortening contractions

The mean isometric knee extensor moments for the entire group (n = 10) were found to be significantly smaller than following shortening contractions the corresponding moments during the purely isometric contractions (Tables 3-1 and 2, Figure 3-2). Eight out of the ten subjects showed consistent force depressions i.e. force depressions in 61 of 62 individual test comparisons. The mean values of force depression following shortening contractions in these eight subjects ranged from 0.7 % to 11.8 % (Mean ± S.D. = 6.5 ± 3.7 %; Table 3-2). These force depressions were statistically significant. In the remaining two subjects, the mean isometric knee extensor moments following shortening contractions were consistently higher than the corresponding isometric moments in all individual test comparisons (n = 8); the force enhancement following shortening in these two subjects ranged from 6.3 % to 8.3 % (Mean ± S.D. = 7.2 ± 1.6 %; Table 3-2).

Table 3-1. One-tailed Student t-test for paired data ($\alpha = .05$) using the mean values of all accepted trials.

| | Moment(Nm) | RMS of EMGs | (ITM/RTM)×100% |
|---|---|---|---|
| ISO | 230 (57) | 1.4 (0.5) | 15.2 (7.4) |
| ISI | 220 (48)* | 1.5 (0.5) | 16.1 (9.5) |

Values are mean (S.D.)
* ISI was significantly smaller than ISO contractions ($p = .05$)
ISO: isometric contraction, ISI: isometric-shortening-isometric contraction

### 3.3.2 Muscle activation (EMG & Interpolated Twitch Technique)

Muscle activation evaluated by the RMS values of the combined EMGs from all instrumented muscles was the same for the ISO and ISI contractions (Table 3-1). In addition, the ratio of (ITM/RTM)×100 % determined by the interpolated twitch technique between the two types of contractions revealed no statistical significance (Table 3-1). Furthermore, for the eight subjects who showed consistent force depression, EMG and interpolated twitch moments were the same for the ISO and ISI contractions (Figure 3-3). For the two subjects who showed consistent force enhancement following muscle shortening, the EMG values were significantly larger in the ISI compared to the ISO contractions (Figure 3-3).

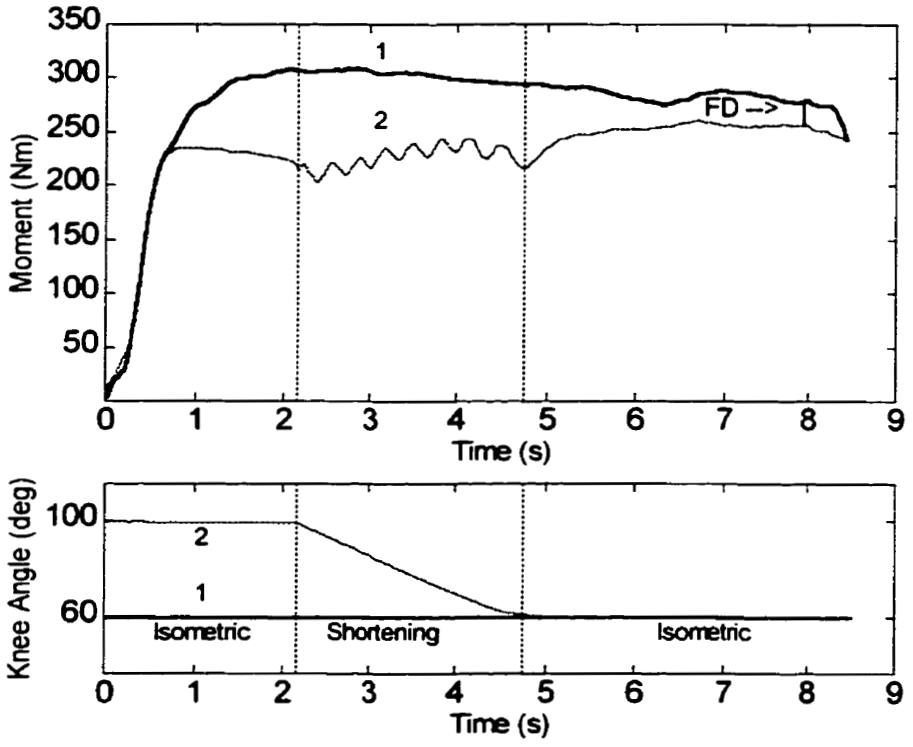


Figure 3- 2. Knee extensor moment-time and knee angle-time histories for an isometric (ISO) and an isometric-shortening-isometric (ISI) contraction from one representative subject which showed consistent force depression (FD) following muscle shortening. (1: ISO contraction, 2: ISI contraction)

Table 3-2. Individual test comparisons with the accepted trials for each subject and the mean force depressions of the ISI compared to the ISO contractions.

| | | Sub 1 | Sub 2 | Sub 3 | Sub 4 | Sub 5 | Sub 6 | Sub 7 | Sub 8 | Sub 9 | Sub 10 | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| # of accepted trials | ISO | 2 | 4 | 2 | 2 | 3 | 1 | 2 | 3 | 3 | 2 | 24 |
| | ISI | 4 | 4 | 1 | 4 | 1 | 2 | 3 | 3 | 4 | 2 | 28 |
| # of comparison | | 8 | 16 | 2 | 8 | 3 | 2 | 6 | 9 | 12 | 4 | 70 |
| Moment | #(ISI<ISO) | 8/8 | 16/16 | 0/2 | 8/8 | 2/3 | 2/2 | 0/6 | 9/9 | 12/12 | 4/4 | 61/70 |
| | FD (%)* | -3.6 | -4.4 | +8.3 | -11.8 | -0.7 | -7.0 | +6.1 | -10.8 | -5.9 | -7.7 | -3.83 (2.14)** |
| RMS of EMGs | #(ISI>ISO) | 0/8 | 14/16 | 0/2 | 2/8 | 0/3 | 0/2 | 0/6 | 7/9 | 12/12 | 0/4 | 35/70 |
| (ITM/RTM) ×100% | #(ISI>ISO) | 1/8 | - | 0/2 | 5/8 | 1/3 | 1/2 | 4/6 | 9/9 | 9/12 | 0/4 | 26/54 |

*FD: Force depression of the ISI compared to the ISO contractions (- represents force depression; + represent force enhancement); the values were calculated from the means of the accepted trials for each contraction type. The mean value for the subjects who showed force depression was 6.5 ± 3.6 % (mean ± S.D.) and the mean value for the subjects who showed force enhancement was 7.3 ± 1.4 % (mean ± S.D.).

**Value is mean (S.D.)

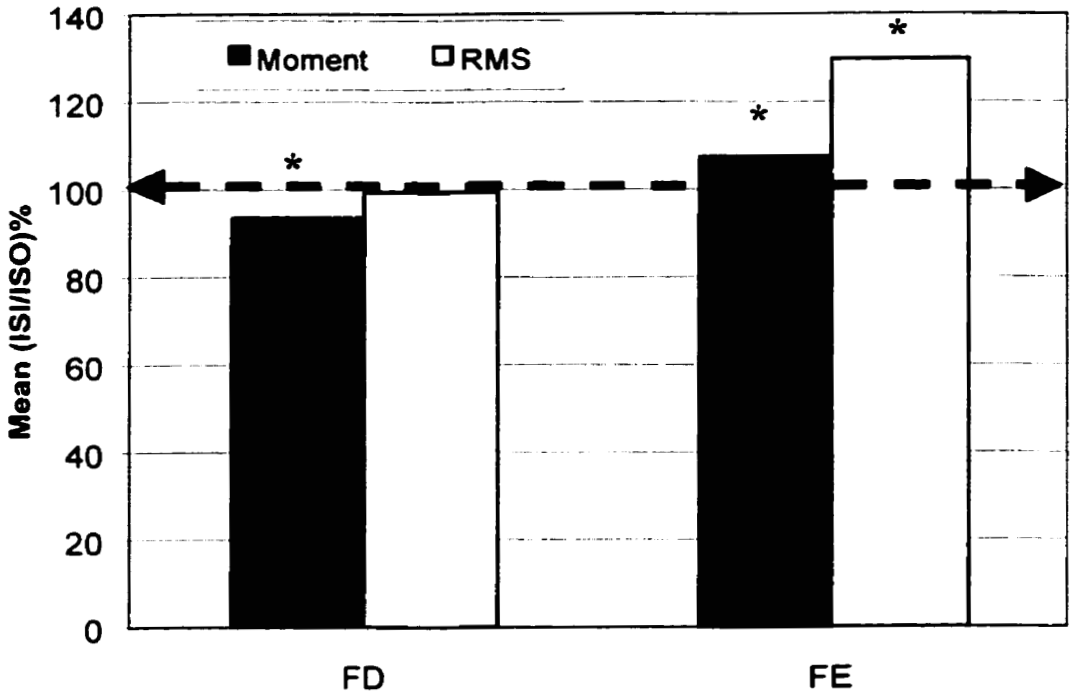


Figure 3- 3. Knee extensor moments and root mean square (RMS) values of the knee extensor EMG for the eight subjects who showed (in 61 out of 62 test comparisons) force depression (FD) following muscle shortening, and for the two subjects who showed force enhancement (FE) following muscle shortening. Values below 100% represent force depression, and values above 100% represent force enhancement following muscle shortening. Similarly, RMS values below and above 100% indicate a decrease and increase in muscle activation in the ISI compared to the ISO contractions. The increase in the EMG RMS values for the two subjects with force enhancement were statistically significant. (* $p < 0.05$)

### 3.4 Discussion

To our knowledge, this is the first time that force depressions following shortening contractions were demonstrated during voluntary muscle contractions. Force depression was observed consistently in eight of ten subjects and 61 of the 62 individual test comparisons from these subjects. In these eight subjects, muscular EMG and superimposed twitch moment values were, on average, the same for the ISO and ISI tests, indicating that muscular activation was similar in both tests (Figure 3-3). Under these

conditions, isometric knee extensor moments following muscular shortening were smaller than the corresponding moments obtained during purely isometric contractions. These results agree with those obtained using electrical stimulation of muscle or fibre preparations (Abbott and Aubert, 1954; de Ruiter *et al.*, 1998; Edman *et al.*, 1993; Granzier and Pollack, 1989; Herzog and Leonard, 1997; Maréchal and Plaghki, 1979; Sugi and Tsuchiya, 1988). Therefore, the results of this study lead to the conclusion that force depression following shortening is an actual property of skeletal muscle and not an artefact of artificial electrical stimulation.

The mean force depression in our study was about 4 %. The only other study in which force depressions in human skeletal muscle were investigated was the study by de Ruiter et al. (1998). These researchers found maximal force depressions for electrically stimulated adductor pollicis of about 37 % when shortening over a range of 38° (of thumb adduction) at a speed of 6.1 °/s. For other contractile conditions, force depression was lower, reaching a minimum value (about 4 %) for the smallest shortening range (8°) and the fastest shortening speed (458.4 °/s).

There are several reasons why the maximal force depressions found by de Ruiter et al. (1998) were three times the maximal values found in our study. First, it is conceivable that the supramaximal electrical stimulation and the choice of muscle might have influenced the results. Based on our results and those of de Ruiter at al. (1998), this point cannot be evaluated. Second, the contractile conditions (amount and speed of shortening) relative to the maximal range and speed of shortening might have been different for the two muscles tested. For lack of relevant information on the adductor pollicis, this point is also hard to evaluate. Third, and in the context of this study most importantly, the

differences in force depression in the two studies might be associated with the different definitions for force depression. In our study, force depression was evaluated approximately 3.5 s following the end of the shortening contraction. Therefore, it might be safely assumed that the recovery of force following shortening was complete (Fig. 2). De Ruiter et al. (1998) did not define at what instant in time following the shortening contraction force depression was determined. However, from their timing of the test in which maximal force depression was measured (total test period = 8.5 s, initial isometric contraction = 1 s, period required for shortening = 38 °/ 6.1 °/s = 6.2 s), it becomes apparent that force depressions must have been determined within 1.3 s of the end of muscle shortening. In Fig. 4 of de Ruiter et al. (1998), force depression measurements are shown after about 1.5 – 1.7 s. From their Figure 3-4, it is obvious that the top (isometric) and bottom traces (shortening of 38 ° at 38.2 °/s) are not parallel (i.e. they converge), showing that force depression values would decrease if force depressions were determined at a later instant in time. For the tests in which maximal force depression was obtained, the convergence of the isometric and the shortening traces would likely be much more pronounced than those shown in Figure 3-4 of de Ruiter et al. (1998) because force depression was evaluated earlier in time than shown in Figure 3-4. Moreover, the recovery of force following 6.1 °/s shortening depends directly on the shortening speed; i.e. the slower the shortening speed, the slower the force recovery (e.g. Herzog and Leonard, 1997). Therefore, the recovery after shortening at 6.1 °/s, which gave the maximal force depressions for de Ruiter et al. (1998), would likely be much less complete than those shown in Figure 3-4 of de Ruiter et al. (1998), indicating that their maximal force depression of 37.2 % is probably a vast overestimation of the actual force

depression that would be measured once force had been fully recovered following the shortening contraction.

From the way the present study was performed and from the results of this study, it is not possible to elucidate a mechanism responsible for the observed force depression in voluntary human skeletal muscle contraction following shortening. However, the results of this study support the idea that force depression is an actual property of skeletal muscle rather than an artefact of artificial stimulation. The most common mechanism associated with force depression following muscle shortening is the idea that during the shortening phase sarcomeres become non-uniform in length. Therefore, some sarcomeres are weaker than others because of their local force-length properties. These 'weak' sarcomeres are thought to dictate the force capacity of the muscle, while the 'strong' sarcomeres are somehow limited in developing their full force potential. There is no evidence that sarcomere length non-uniformity is developed in the entire muscle during shortening under physiological stimulation. Furthermore, sarcomeres and fibres are connected in parallel in the whole muscle, therefore, it is hard to perceive how substantial sarcomere length non-uniformity may evolve in the whole muscle as they have been observed in single fibre preparations (e.g. Gordon *et al.*, 1966). Finally, large force depressions have been observed in single fibre preparations in which sarcomere length non-uniformity was prevented from occurring (Granzier and Pollack, 1989), therefore indicating that other mechanisms than sarcomere non-uniformity may be responsible for the force depressions observed here and elsewhere.

In two of the ten subjects, and in all eight individual comparisons between ISO and ISI contractions from these subjects, the knee extensor moments were larger in the ISI

compared to the corresponding ISO contractions. This result is in contrast to all published observations in which artificial muscle stimulation was used. If it is assumed, as we have done above, that force depression following shortening is an inherent property of skeletal muscle, the results of this two subjects can only be explained by an increased muscle activation in the isometric contractions following shortening compared to the corresponding purely isometric contractions. Inspection of the EMG values of the two subjects (Subjects 3 and 7, Table 3-1) revealed that the EMG values were always higher in the ISI than the ISO contractions, as expected (Figure 3-3). The percent increase in the EMG values of these two subjects from the ISO to the ISI contractions was larger than in any of the other subjects (Table 3-1), suggesting that indeed, muscular activation was increased from the ISO to the ISI contractions, thus possibly explaining the increased knee extensor moments following shortening contractions in these two subjects.

It is not clear from the results of this study why two subjects showed results opposite to those of the remaining eight subjects. These two subjects appeared to be able to compensate for the loss of muscular force potential following shortening with increased activation (Figure 3-3), and so were able to eliminate any force depression. The two subjects who were able to increase muscular activation following shortening were the only two within the subject group who lifted weights on a regular basis as part of their training. Therefore, one may speculate that it is possible to overcome the mechanical disadvantage of muscular force production following muscle shortening with systematic weight training. However, at present, the this idea remains speculation.

### 3.5 Summary and Conclusions:

To our knowledge, this is the first study in which force depressions following shortening contractions were demonstrated for voluntary contractions. From the results of this study, it can be concluded that force depression is a property of skeletal muscle contraction and not an artefact of artificial electrical stimulation. It appears that force depressions during voluntary contractions might be compensated for, partly or completely, by an increase in muscle activation following shortening beyond the levels obtained during purely isometric contractions.

# CHAPTER 4: Effect of Speed and Distance of Muscle Shortening on Force Depression During Voluntary Contractions

## 4.1 Introduction

It has been reported for a variety of skeletal muscle preparations that the maximal isometric force following muscle shortening is smaller than the corresponding force obtained during purely isometric contractions (e.g. isolated skeletal muscle fibres [Edman *et al.*, 1993; Granzier and Pollack, 1989; Sugi and Tsuchiya, 1988]; in-situ skeletal muscles [Abbott and Aubert, 1952; Maréchal and Plaghki, 1979; Herzog and Leonard, 1997]; in-vivo human skeletal muscle [de Ruiter *et al.*, 1998; Lee *et al.*, 1998]). This phenomenon is referred to as force depression (FD) following muscle shortening (Herzog and Leonard, 1997). Force depression following muscle shortening is inversely related to the speed of shortening and directly related to the distance of shortening. Furthermore, FD is long-lasting (Abbott and Aubert, 1952; Herzog and Leonard, 1997) and can be abolished by deactivating the muscle for a period of time long enough that force will go to zero (Abbott and Aubert, 1952; Maréchal and Plaghki, 1979; Herzog and Leonard, 1997).

Force depression following muscle shortening is not contained in the classical theory of muscle contraction; the cross-bridge theory (Huxley, 1957; Huxley and Simmons, 1971). Three basic hypotheses have been proposed to explain the mechanism underlying FD; i) sarcomere non-uniformity (Edman *et al.*, 1993), ii) increase in proton and inorganic phosphate concentration (Granzier and Pollack, 1989), iii) stress-induced cross-bridge inhibition (Maréchal and Plaghki, 1979). However, none of these hypotheses can explain all experimental observations.

De Ruiter *et al.* (1998) were the first to demonstrate that there is force depression following muscle shortening in *human skeletal muscle*. They reported maximal force depressions of 37 % in the adductor pollicis and found an inverse relation of FD with the speed of shortening and a direct relation with the distance of shortening ($r^2 > .98$). De Ruiter *et al.* (1998), like all previous researchers, used artificial electrical stimulation to activate the muscle. Since electrical stimulation is highly non-physiological, we began to examine force depression following muscle shortening during *voluntary contractions* (Lee *et al.*, 1998). We found that isometric force in human knee extensor muscles was depressed following shortening contractions. However, there are no data to show whether force depression following muscle shortening has the same conceptual relationship with the speed and distance of shortening during *voluntary* contractions as it has for contractions produced *artificially* using electrical stimulation of the muscle or the corresponding nerve.

Therefore, the purpose of this study was to determine the amount of force depression following *voluntary* muscle shortening at different speeds and for different shortening distances. It was hypothesized that for voluntary contractions, in contrast to *artificial* electrical contractions, force depression may not depend on the speed and the distance of shortening.

## 4.2 Methods

### 4.2.1 Subjects

Thirty subjects gave free informed consent to participate in this study (177.4 ± 10.3 cm in height; 72.6 ± 11.4 kg in weight; 19 – 45 years). 20 of the 30 subjects participated in the

first testing protocol, and 20 of the 30 subjects participated in the second protocol. Ten subjects participated in both testing protocols. Subjects had no recent history of knee injuries. All experimental tests were approved by the Conjoint Ethics Committee of the University of Calgary

### 4.2.2 Experimental Setup

Subjects were seated on a strength-testing machine (CYBEX NORM™ Testing and Rehabilitation System, Lumex, Inc., New York, U.S.A.), which was set up for left leg flexion/extension exercises. Subjects wore a double shoulder seat belt to stabilize the upper body. The distal ends of the thigh and shank were strapped to the seat and the dynamometer arm, respectively. The rotational axis of the strength-testing machine was aligned with the knee joint axis (i.e. the most prominent point of the lateral epicondyle of the femur). Anatomical zero was taken for the fully extended knee angle (Figure 4-1).

### 4.2.3 Moment measurement

During the test contractions, the resultant knee extensor moments were measured with the strength-testing machine and stored in a computer for off-line analysis.

### 4.2.4 Electromyography

In order to measure muscle activation, bipolar Ag-AgCl surface electrodes were used. After preparing the skin at the sites of electrode placements by shaving and rubbing with alcohol, one electrode pair each were placed on the distal third of the vastus lateralis (VL) and vastus medialis (VM), and the proximal third of the rectus femoris (RF). A ground

electrode was placed on the patella. EMG signals were pre-amplified (500 –5000) through an amplifier located no further than 10 cm from the recording electrodes. Signals were band-pass filtered using cut off frequencies of 10 Hz and 1 kHz.

### 4.2.5 Interpolated twitch technique

In order to assess the extent of motor unit (MU) recruitment, the interpolated twitch technique was used (Bélanger and McComas, 1981; Hales and Gandevia, 1988; Suter and Herzog, 1997). A pair of carbon-impregnated rubber electrodes (4.5 × 10 cm) was thinly covered with a layer of conductive gel. One electrode was placed over the femoral nerve just distal to the inguinal ligament; the other electrode was placed at the distal end of the quadriceps muscle. During the test contractions, a doublet electrical stimulus (220V, 8ms interpulse interval, 0.8ms duration) was applied to the femoral nerve using a Grass S88 Muscle Stimulator with an isolation unit approved for human use (Quincy, MA, U.S.A.). Resting twitch moment (RTM) was measured by applying a doublet twitch to the relaxed muscle and recording the corresponding twitch moment. The interpolated twitch moment (ITM) was determined by superimposing a doublet twitch to the voluntarily contracting muscle and recording the additional moment produced by the electrical stimulation on top of the maximal voluntary moment. The extent of motor unit recruitment during the maximal voluntary contraction was assessed by calculating the ratio; ITM/RTM.

### 4.2.6 Experimental protocols

For determining the possible effects of the distance of shortening on FD, subjects ($n$ = 20) produced three different isometric knee extensor contractions (Figure 4-1a): i)

isometric (ISO) at 50° knee flexion, ii) isometric following a small distance of shortening (ISI-S; shortening from a knee flexion angle 75° to 50°), and iii) isometric following a large distance of shortening (ISI-L; shortening from a knee flexion angle 100° to 50°) contractions. The speed of shortening was constant at 20°/s for all tests. All three types of contractions were repeated three times and the order of the tests was randomized in a balanced design.

For determining the possible effects of the speed of shortening on FD, subjects ($n$ = 20) performed three different isometric knee extensor contractions on the strength-testing machine (Figure 4-1b): i) isometric (ISO), ii) isometric following slow shortening (ISI20; shortening speed of 20°/s), and iii) isometric following fast shortening (ISI240; shortening speed of 240°/s). All isometric contractions were done at a knee flexion angle of 60°. Shortening of the knee extensor muscles was produced by extending the knee from 100° to 60°. All three types of contractions were repeated three times and the order of tests was randomized in a balanced design.

During all test contractions, subjects were verbally encouraged to produce maximal effort contractions. Each test contraction lasted for 6.5s. The interpolated twitch stimulation was applied 6s after the initiation of the contraction in all tests (Figure 4-1). Following all test contractions, the RTM was measured three times at a knee flexion angle of 50° for the first test and 60° for the second test.

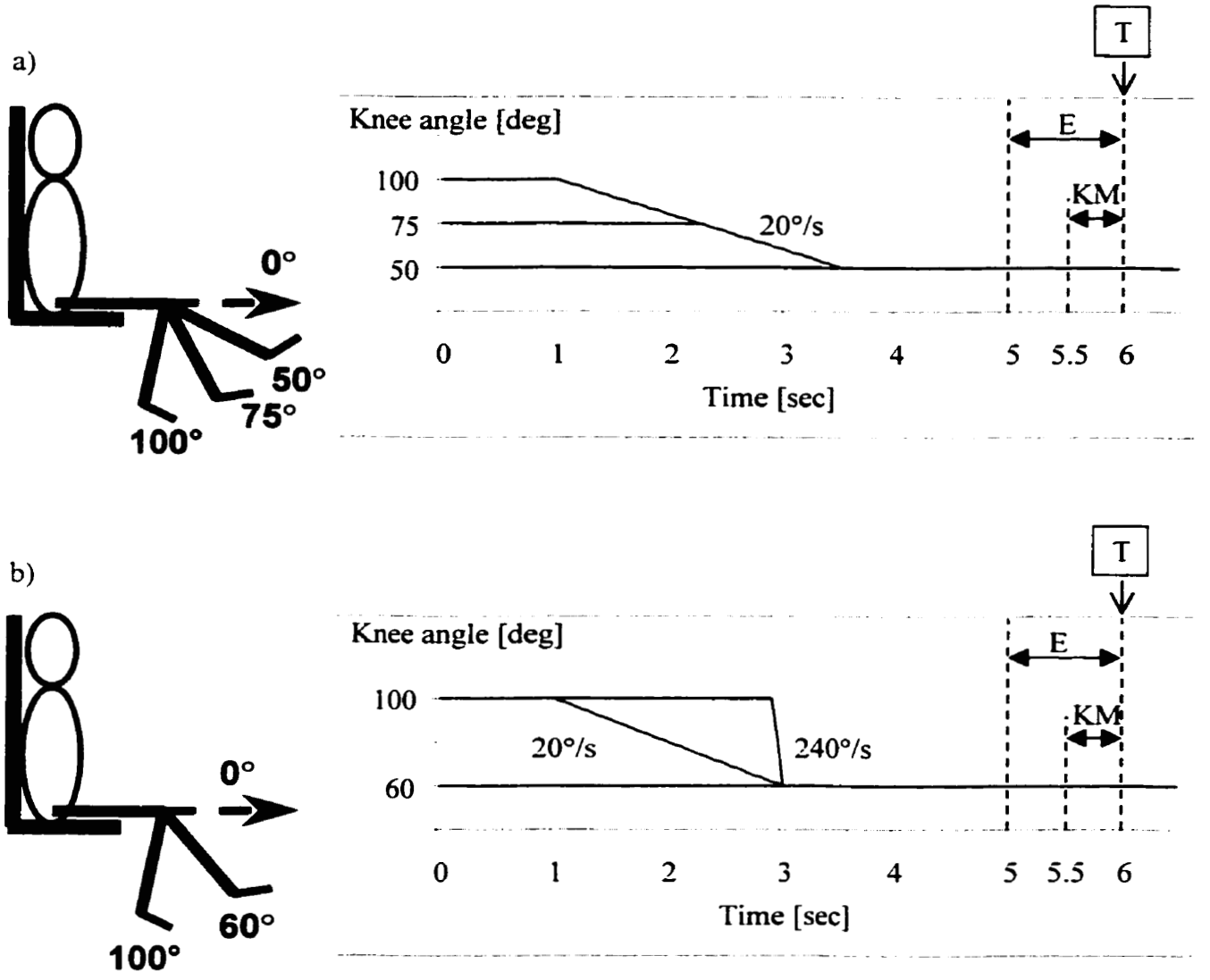


Figure 4- 1. Definition of the knee angles and schematic illustration of the test contractions. The fully extended knee was defined as 0°; a) Setup for testing the effects of shortening distance on FD; b) Setup for testing the effects of shortening speed on FD; T: interpolated twitch; E: EMG analysis; KM: Knee extensor moment analysis.

### 4.2.7 Data collection and Analysis

During the experiments, knee extensor moment, knee joint angle and EMG signal were simultaneously recorded at a sampling frequency of 2000 Hz and stored on a microcomputer using an analogue-digital board with a resolution of 12-bits.

The knee extensor moments for each test contraction were extracted over a 500 ms period prior to applying the interpolated twitch (Figure 4-1), and averaged for the subsequent analysis. EMG signals were extracted over a 1 s period preceding the interpolated twitch (Figure 4-1). Root mean square (RMS) values from the combined EMG of VL, VM and RF were calculated for all test contractions. The ratios of ITM/RTM were calculated to assess the extent of motor unit recruitment.

### 4.2.8 Statistics

Prior to performing any statistical analysis, trials in which the peak moment did not reach at least 95% of the absolute peak moment of the corresponding test contractions were excluded. These trials were not deemed to represent a maximal voluntary effort.

Using ANOVA with repeated measurements and Tukey's post hoc multiple comparisons, knee extensor moment, root mean square (RMS) of the combined EMGs, and the ratio of ITM to RTM were compared among the test conditions ($\alpha$ = .05). All results are presented as means ± 1 S.D.

## 4.3 Results

### 4.3.1 Effect of distance of shortening on force depression

The maximal isometric knee extensor moments following muscle shortening were smaller than the corresponding moments obtained during the purely isometric contractions for both distances of muscle shortening (small shortening distance; 95.5 % ± 8.2 %, and large shortening distance; 93.1 % ± 7.4 %: Figure 4-2). The RMS values of the combined EMGs and the ratios of ITM/RTM for assessing muscle activation and MU recruitment were the same for all contractions in this experiment (Figure 4-2).

### 4.3.2 Effect of speed of shortening on force depression

The maximal isometric knee extensor moments following muscle shortening were smaller than the corresponding moments obtained during the purely isometric contractions (93.1 % ± 8.4 % and 91.6 % ± 6.8 % for the ISI20 and ISI240 contractions, respectively; Figure 3). The isometric knee extensor moments following the two different speeds of muscle shortening were the same. The ratios of ITM/RTM were higher in the ISI20 and ISI240 contractions (135.2 % ± 44.8 % and 141.8 % ± 45.4 % for the ISI20 and ISI240 contractions, respectively; Figure 4-3) compared to the ISO contractions. The RMS values of the combined EMGs were the same for all contractions in this experiment (Figure 4-3).

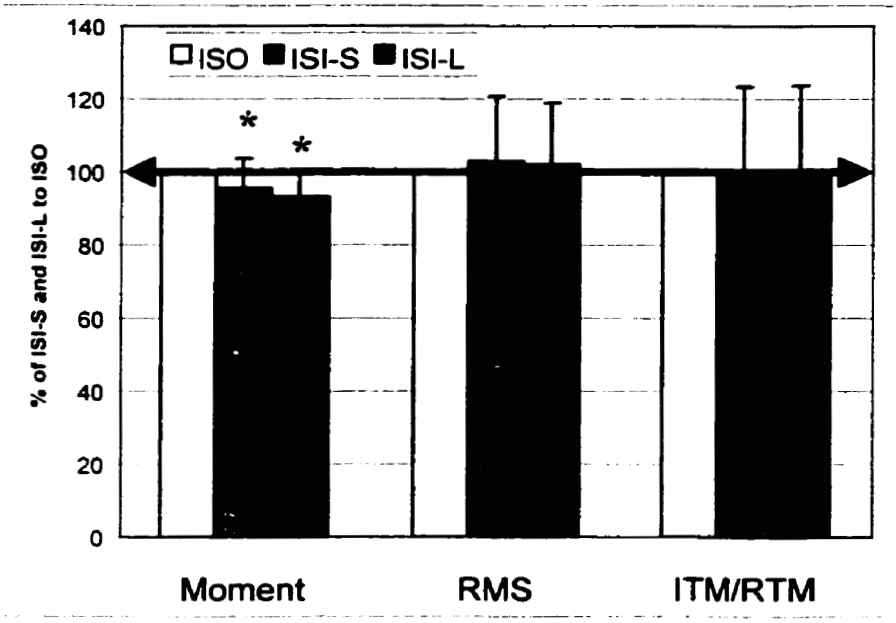


Figure 4- 2. Knee extensor moment, RMS and ITM/RTM of the ISI-S and ISI-L contractions compared to the ISO contractions. (The ISO contraction is represented as 100% in all cases: * $p < .05$ compared to the isometric reference contraction)

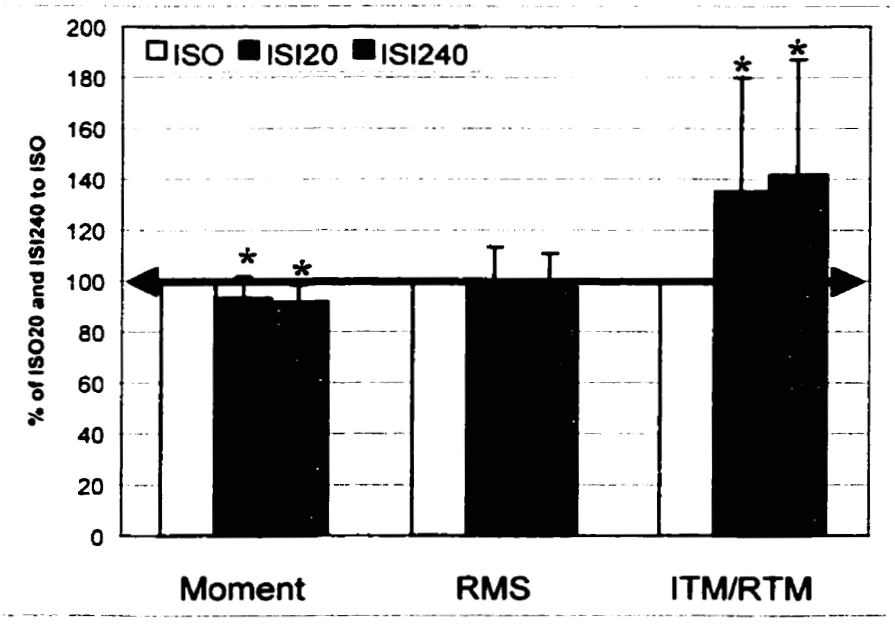


Figure 4- 3. Knee extensor moment, RMS and ITM/RTM of the ISI20 and ISI240 contractions compared to the ISO contractions. (The ISO contraction is represented as 100% in all cases: * $p < .05$ compared to the isometric reference contraction)

### 4.4 Discussion

It is well accepted that the isometric force following muscle shortening is reduced compared to the corresponding force obtained during purely isometric contractions (Abbott and Aubert, 1952; Edman *et al.*, 1993; Maréchal and Plaghki, 1979; Granzier and Pollack, 1989; Herzog and Leonard, 1997; Sugi and Tsuchiya, 1988). Furthermore, it is well accepted that force depression is increased for increasing distance of muscle shortening (and constant speed of shortening) and for decreasing speed of muscle shortening (and constant distance of shortening) (Maréchal and Plaghki, 1979; Herzog and Leonard, 1997). The above results have been found in isolated muscle fibre preparations (Edman *et al.*, 1993; Granzier and Pollack, 1989; Sugi and Tsuchiya, 1988), in whole muscle preparations (Abbott and Aubert, 1952; Maréchal and Plaghki, 1979; Herzog and Leonard, 1997), and in in-vivo human skeletal muscle preparations (de Ruiter *et al.*, 1998). However, in all previous studies, artificial electrical (muscle or nerve) stimulation was used. Lee *et al.* (1998) found, for a single speed and a single distance of muscle shortening, that isometric force during voluntary contractions were reduced following muscle shortening compared to the corresponding force during isometric contractions. The question of this study was whether or not force depression depends on the speed and distance of muscle shortening in the same way during *voluntary* contractions as published for *artificially* stimulated muscle and fibre preparations.

***Distance of shortening***: We found in this study that knee extensor moments were depressed by 4.5 % for the ISI-S and 6.9 % for the ISI-L contractions from the corresponding purely isometric force. Force depressions were statistically significant but did not increase significantly with increasing shortening distance, as we had expected

based on the results of all previous studies using artificial stimulation protocols. The EMG and interpolated twitch moment values for this experiment were the same for all experimental conditions, indicating that the voluntary muscle activation was about constant for the isometric and the shortening contractions, independent of the distance of shortening. Together, these results suggested that force depression following muscle shortening during voluntary contractions is independent of the distance of shortening. Furthermore, it appears that the difference in the findings for voluntary and artificially produced muscle contractions cannot be explained by changes in muscle activation associated with the voluntary contractions.

***Speed of shortening***: Knee extensor moments were reduced by 6.9 % and 8.4 % following muscle shortening at knee angular velocities of 20°/s and 240°/s, respectively. Forces were reduced significantly compared to the forces from the isometric reference contractions for both speeds of shortening. From previous experiments using artificial stimulation, it was expected that force depressions should decrease with increasing speeds of shortening (Maréchal and Plaghki, 1979; Herzog and Leonard, 1997). However, contrary to our expectation, the mean force depression was the same at the two speeds. The RMS values of the combined EMG signals were the same for all tests. The interpolated twitch results (i.e. ITM/RTM) were significantly higher for the tests involving the slow and fast speed of shortening compared to the corresponding isometric reference contractions (Figure 3).

The results of this study suggest that force depression following muscle shortening is independent of the speed and distance of shortening. This finding is in contrast to all previous studies on this subject which have been performed exclusively using artificial

electrical stimulation (Abbott and Aubert, 1952; de Ruiter *et al.*, 1998; Edman *et al.*, 1993; Granzier and Pollack, 1989; Herzog and Leonard, 1997; Maréchal and Plaghki, 1979; Sugi and Tsuchiya, 1988). Therefore, the question arises, how can one account for this conceptual difference between the current findings and those published earlier. Three possibilities come to mind: 1) the contractile conditions during the shortening phase were vastly different between the present study and those performed earlier; 2) the activation was not kept constant during the voluntary contractions; and 3) voluntary contractions and electrically induced contractions influence the history-dependent properties of skeletal muscle in different ways. We would like to discuss these possibilities below, but would like to point out that this study was not aimed at identifying the mechanism of force depression following muscle shortening during voluntary contractions. Therefore, a complete answer for the results obtained here will not be possible.

Regarding point (1), we estimated the distance and the speed of muscle shortening relative to the fibre lengths of the knee extensor muscles and compared these contractile conditions to those used in previous work on the cat soleus (Herzog and Leonard, 1997). Assuming an average moment arm of the knee extensors of about 50 mm (Herzog and Read, 1993) and average fibre lengths of 70-100 mm (Wickiewicz *et al.* 1983), one can estimate that the distances of muscle shortening in the present study ranged from about 1/4 to about 1/2 fibre length. The corresponding values in the cat soleus study were 1/20 to 1/4 fibre length. That means, muscle shortening in the present study exceeded that of our previous work. Since distance of shortening in the artificially stimulated muscle is directly related to force depression, the force depression effects should have been larger

and the difference in force depression between the two shortening distances should have been more pronounced in this study than the study using cat soleus.

The corresponding speeds of shortening were 0.2-2.5 fibre length/second (present study) and 0.1-6.0 fibre length/second (cat soleus study). Thus, the speeds of shortening used in the present study should have shown the same conceptual results since they are within the range of speeds tested using the cat soleus. From the above considerations, it appears that the contractile conditions during the shortening phase of the present study were similar to those used on cat soleus (Herzog and Leonard, 1997), and so, should have produced similar results.

Regarding point (2), we attempted to estimate whether activation was constant during the voluntary contractions. Analysis of the EMG and twitch interpolation results reveals that, on average, muscle activation was kept constant in the tests involving the two shortening distances (Figure 2). In the tests using the two speeds of shortening, the EMG values were the same for all three tests (isometric reference contractions, fast, and slow speed shortening contractions, Figure 3). However, the interpolated twitch values were found to be significantly higher for ISI20 and ISI240 compared to ISO (Figure 3), possibly indicating a decrease in activation following the shortening contractions at the two speeds. The significant increase in the interpolated twitch following the slow speed of shortening tests is insofar suspect as such an increase was not observed in the tests with the two shortening distances which were performed at the same speed (i.e. 20 °/s) as the slow speed test. In fact, combining the results of all slow speed tests performed in this study (n=60) reveals that the interpolated twitch following slow speed shortening was the same as that following the isometric reference contractions. This result is also in

agreement with our previous findings (Lee *et al.*, 1998) using a speed of shortening of 16 °/s.

From the above consideration, it appears that changes in muscle activation during the voluntary tests cannot account for the results observed in this study with the possible exception of the findings observed following the fast speed of shortening. The mean force depression following fast shortening was slightly larger than that following the slow speed of shortening. From previous studies, one would have expected force depression to be significantly smaller following the fast compared to the slow speed of shortening. The significant increase in the interpolated twitch compared to the reference contraction, as well as the slight (but non-significant) decrease in the EMG following the fast speed shortening contractions (Figure 3) may indicate that force depression was overestimated in the fast speed tests because of a decrease in activation. Such a decrease in the maximal voluntary activation may be triggered reflexively following fast voluntary muscle contractions. However, such a hypothesis will need further evaluation.

Finally, regarding point (3), it is well accepted that voluntary contractions differ considerably from typical artificial contractions in the way that the motor units are recruited. During maximal voluntary contractions, motor units are firing at different, non-periodical rates in a largely non-synchronized fashion. During artificial contractions, all motor units of a muscle are stimulated periodically, at the same rate and perfectly synchronized. It could well be that the stimulation characteristics may influence the history-dependent force properties of skeletal muscle. Although we have no direct proof of such a mechanism, future work on force depression during voluntary contractions should consider this possibility.

# CHAPTER 5: SUMMARY AND CONCLUSIONS

Force depression following muscle shortening has been demonstrated consistently in in-vitro and in-situ muscle preparations. Considering the non-physiological conditions caused by *electrical stimulation*, it was speculated that force depression following muscle shortening might be different for human *voluntary contractions* than for electrically stimulated contractions. The purpose of this research was;

1) To test if force depression following muscle shortening does indeed occur during human voluntary contractions, and
2) To test the relationship between force depression and the speed and distance of muscle shortening during human voluntary contractions.

## 5.1 Summary of Results

In chapter 3, the existence of force depression following muscle shortening during voluntary contractions was addressed. The isometric force produced immediately following muscle shortening was significantly decreased compared to the corresponding force during purely isometric contractions. The amount of force depression was, on average, 6.5 %. Muscle activation, as assessed by EMG and the interpolated twitch technique, was the same for all test contractions.

In chapter 4, the effect of speed and distance of shortening on force depression during voluntary contractions was addressed. The isometric forces following large (50°) and small (25°) amounts of shortening at a given speed (20 °/s) were significantly smaller

than the corresponding forces during the isometric reference contractions. However, force depression was independent of the amount of shortening, a result that is in contrast to previous findings obtained using electrically stimulated muscle preparations. Muscle activation was constant for the voluntary contractions, indicating that changes in activation are likely not responsible for the difference in the results between voluntary and artificially elicited contractions.

The isometric forces following fast (240 °/s) and slow (20 °/s) speeds of shortening over a given distance (40°) were significantly smaller than the corresponding forces during the isometric reference contractions. However, force depression was independent of the speed of shortening. Interestingly, the mean force depression following the fast shortening contractions was the largest among all tests performed, whereas, based on the results published in the literature, force depression following the fast speed of shortening should have been the smallest. Muscle inhibition might be a possible explanation for this result. The increased interpolated twitch following the fast speed contractions supports this idea.

### 5.2 General Conclusion

The results of the two experiments performed here lead to the conclusion that force depression following muscle shortening does indeed exist in human voluntary contractions. However, it does not seem safe to apply the results from in-vitro and in-situ experiments using electrical stimulation to human voluntary contractions because the amount of force depression was independent of the speed and distance of shortening.

During voluntary contractions, it is extremely difficult to produce a consistent maximal effort throughout all contractions. In order to test whether subjects produced maximal effort contractions, the interpolated twitch technique was used. However, the interpolated twitch technique gives large variations in the interpolated twitch magnitude for the same level of effort (i.e. the same knee extensor moment: Suter *et al.* 1996). Therefore, the results from the interpolated twitch technique must be considered with caution, and probably should be interpreted qualitatively rather than quantitatively. EMG measurement also showed a considerable amount of variation for given knee extensor moments, regardless of the care that was taken in the preparation and recording of the EMG signals. Therefore, one of the weaknesses of this study was that there was no way to measure muscle activation accurately and repeatably.

### 5.3 Significance

The present research was a first attempt at investigating force depression following muscle shortening during in-vivo human *voluntary contractions*. This research provides new information on in-vivo properties of human skeletal muscle. I hope that this new information contributes to fill, at least a small page in the history of muscle mechanics.

### 5.4 Future Directions

This thesis was aimed at describing force depression following muscle shortening and how force depression relates to the speed and the distance of muscle shortening during voluntary contractions of in-vivo human skeletal muscle. It is not easy to extrapolate possible mechanisms for the observed force depressions from the results of this thesis.

Future work needs to focus on why force depression following muscle shortening is different during voluntary contraction than it is for artificially induced electrical stimulation.

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