ANTAGONIST COCONTRACTION OF THE KNEE FLEXORS AND EXTENSORS DURING CONTROLLED MUSCLE SHORTENING AND LENGTHENING.

by

CHRISTOPHER JOHN SNOW

A Thesis
Submitted to the Faculty of Graduate Studies
in Partial Fulfilment of the Requirements
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University of Manitoba
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ABSTRACT

The technique of electromyography was used to analyze the antagonist activity of the quadriceps and hamstring muscles of 24 normal male and female subjects during constant velocity muscle performance tests performed in the sitting position on a KIN/COM™ robotic dynamometer.

A pilot study was carried out to determine the most appropriate location for the surface electrodes on the thigh musculature for the measurement of antagonist muscle activity without cross-talk, establish the test velocities to be used, and determine the chart speeds and resolvable angle for optimal measurement of EMG activity.

Each subject performed four maximal effort dynamic tests of knee flexion and extension through a 65° range of knee motion under two constant velocity conditions in a single session. Each test consisted of four repetitions of agonist muscle shortening followed by agonist muscle lengthening. Two tests were performed with the left knee extensors as agonists, and two tests were performed with the left knee flexors as agonists. For each muscle group, one test was performed at 30°/s, and another at 90°/s. The sequence of muscle group testing was randomized.

The normalized values of the peak amplitude EMG of the knee flexors and extensors at every 5° of the constant velocity phase of each test was measured and compared in a paired manner between each angle, contraction type, velocity, and muscle group by split-plot 2-way ANOVA.

Antagonist hamstrings cocontraction was found to be contraction type (ml>ms:$p<.003$), and velocity ($90°>30°$/s:$p<.001$) dependent, with significant interactions between angle and contraction type ($p<.001$). Antagonist quadriceps cocontraction was also greater at the faster velocity ($p<.02$) but was always less then the hamstrings, and was not significantly affected by angle or contraction type.

It is concluded that antagonist hamstrings and quadriceps respond differently under typical constant velocity test conditions. It is also concluded that the levels and temporal pattern of antagonist hamstring and quadriceps cocontraction does not support the hypothesis that such activity is a function of muscle moment-arm. Results do support the idea of control by muscle spindles and perhaps Golgi tendon organs. It appears that antagonist cocontraction occurs more consistently and at greater levels than previously thought in normal healthy individuals. This has implications for clinical muscle performance testing and interpretation.

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LIST OF ABBREVIATIONS

ACL	anterior cruciate ligament
AG1	agonist burst #1
AG2	agonist burst #2
AGml	agonist muscle lengthening
AGms	agonist muscle shortening
ANOVA	analysis of variance
ANT	antagonist burst
ANT1	antagonist burst #1
ANT2	antagonist burst #2
ANTml	antagonist muscle lengthening
ANTms	antagonist muscle shortening
ATPase	adenosine triphosphatase
EMG	electromyogram/electromyographic
GTO	Golgi tendon organ
iEMG	integrated EMG
MAV	mean absolute value
MVC	maximum voluntary contraction
%Max	percent maximum
PCL	posterior cruciate ligament

1.0 INTRODUCTION

Muscle performance tests performed under constant velocity conditions are becoming a common clinical procedure in the evaluation of musculoskeletal and neurological conditions. Although the dynamometers used to perform such tests are expensive, their cost is often justified by their objectivity and high reliability. As well, dynamic tests using a dynamometer allow the study of force-velocity and length-tension relationships during movements of muscle shortening and lengthening. However, while most major commercial dynamometers are intrinsically accurate and have good to excellent test-retest reliability for measuring torque, velocity and displacement, data from different instruments are not comparable because of differences in design.

The torque recorded by these dynamometers is generally assumed by clinicians to accurately reflect the torque produced by agonist muscles and test results are invariably reported as such. However, questions have been raised regarding what is actually being measured and whether the high level of confidence needed for clinical judgment is justified based on the lack of scientific confidence in those measurements (Rothstein et al, 1987). It is therefore important to identify and study the factors which contribute to the torque measured by the dynamometer and to establish their relative importance in normal and abnormal conditions. From a biomechanists point of view, the dynamics of muscles contracting about an articular joint are not well understood (Catani et al, 1988). Every joint in the body is mobilized by muscles that generate forces in opposing directions (DeLuca and

Mambrito, 1987). Therefore it is possible to control separately both the torque and the stiffness at a joint. The net torque at a joint is the difference between the torques of the agonist and antagonists. The net stiffness is the sum of the individual stiffnesses of the agonists and antagonists. Thus the value of the two variables may range from high torque and low stiffness (reciprocal contraction of agonist and antagonists) to zero torque and high stiffness (cocontraction of agonists and antagonists) (DeLuca and Mambrito, 1987).

Therefore, it is important to understand the relationship between agonist and antagonist activation during constant velocity testing. The factors which affect this relationship must also be outlined to include the likely mechanisms of control, and its importance in clinical interpretation of torque data. Few investigators have studied antagonist muscle activity during constant velocity muscle testing. Fewer still have studied the effects of velocity and contraction type on antagonist activity. There are no studies of antagonist activity where the effects of angle, velocity, and contraction type have been determined using a typical clinical test protocol.

The present study was conducted to measure the level of antagonist cocontraction in the knee flexors and extensors of normal subjects using a typical clinical test protocol for the KIN/COM™ robotic dynamometer. The electromyographic activities of the m. vastus lateralis and the hamstrings muscle group were recorded while subjects maximally contracted knee flexors and extensors through a preset range of motion at two constant velocities. The parameters of antagonist muscle activity used in the analysis were the amount of rectified

electrical activity expressed as a percentage of that same muscle's maximum agonist activity normalized to velocity and moment-arm angle.

The data from this study on antagonist muscle activity in normal individuals may result in (1) a better understanding of torque-angle curves and their interpretation, and so (2) may necessitate the need to modify clinical test protocols accordingly, (3) enable comparison of such antagonist activity to that found in various joint deficiencies and neurological disorders, (3) provide a better understanding of joint mechanics and motor control, and (4) lead to the development of better therapeutic strategies to improve muscle function and movement.

2.0 REVIEW OF LITERATURE

2.1. Skeletal Muscle

Muscle, which is responsible for animal motility, transforms chemical energy into mechanical energy. The general working principles are well known, but the intimate molecular mechanisms by which the chemical energy of adenosine triphosphate salts are converted into mechanical energy are not completely understood. There are three types of muscle identified in man: smooth muscle, striated cardiac muscle, and striated skeletal muscle. Approximately 43% of human body mass is contributed by skeletal muscle. Skeletal muscle contractility and its intricate control by the nervous system, enable the body to adapt to the need for fine motor control, short intense effort, or prolonged activity. Such adaptability reveals the plastic nature of muscle tissue.

2.1.1 The Muscle Fibre

The movement produced by skeletal muscle is carried out by specialized cells called muscle fibres. A muscle fibre appears in the form of a long filament of 10 - 100 μm in diameter, extending between the two tendons or attachments of the muscle. Each muscle fibre contains from several hundred to several thousand myofibrils, each 1-2 μm in diameter extending throughout the length of the fibre (Lehninger, 1975). Although comparable from one skeletal muscle to another, the characteristic dimensions of the myofibrils vary slightly according to the type of muscle and the species from which it is obtained (Carlson and Wilkie, 1974). When observed under the optical phase contrast microscope, the myofibrils show the

presence of a succession of transverse bands, termed A and I bands. Under the electron microscope, these bands can be subdivided into filaments of actin and myosin. These are large polymerized protein molecules which are responsible for muscle contraction and constitute approximately 65% of muscle protein. The actin filaments are attached to Z discs from which they extend on either side. Myosin filaments, aligned at the M line interdigitate with the actin filaments. The section of myofibril between the two Z discs is called a sarcomere, and sarcomere length varies such that actin filaments can overlap the myosin filaments. The interaction of actin and myosin under control by troponin, tropomyosin, [Ca^{2+}], and other factors cause the sarcomere to shorten, the muscle to contract and generate tension, as originally described by the sliding filament theory (Huxley and Huxley, 1964).

2.1.2 Fibre Types

Muscle does not constitute a functionally homogeneous tissue. Most muscles are composed of fibres with different mechanical properties, which are correlated to their histochemical and morphological characteristics (Green, 1986). As early as 1678, Lorenzini had classified muscle into two types, red and white (Close, 1972). They were also classified by speed of contraction and fatigability in 1873 by Ranvier (Close, 1972). Today, however, fibre types are usually identified primarily by the metabolic pathways by which they generate adenosine triphosphate and the rate that energy is made available to produce contraction. In 1962, Engel suggested the use of a stain for myofibrillar ATPase after alkaline incubation (Engel, 1962). This method separates muscle fibres into two well-defined groups. He proposed the

names type I and type II fibres for those fibres staining light and dark respectively.

Type I fibres are relatively small in area (3000 - 6000μm^2 in untrained men) and so produce relatively little tension. They have a relatively poorly developed glycolytic enzyme system, but there are many mitochondria which provide a high oxidative enzyme activity for aerobic metabolism. These fibres are therefore well adapted for prolonged work at low levels of force production. The time course of an isometric twitch by the type I fibres is also relatively slow (90 - 110ms in humans). These type I fibres, often referred to as slow-twitch, or slow oxidative fibres (Green, 1986), predominate in muscles which require sustained contraction such as the postural muscles in man (e.g. m. soleus, m. biceps femoris) (Monster et al, 1978).

Type II fibres differ in many respects from Type I fibres. They have a relatively low oxidative capacity, but high glycolytic capacity. Type II fibres have faster contraction times (40 - 85ms in humans) than type I, and are often referred to as fast-twitch fibres (Green, 1986). These fast fibres predominate in muscles requiring strong, rapid and transient contractions.

In addition to the histochemical analysis described above for ATPases, a mitochondrial enzyme stain was subsequently found to further differentiate type II fibres into those with moderate oxidative potential from fibres with very little oxidative activity (Barnard et al, 1971). They are typically referred to as type IIa or fast oxidative-glycolytic fibres, and type IIb or fast glycolytic fibres respectively (Brooke and Kaiser, 1970a, 1970b). This subdivision can also be made using the

lability of fast fibre ATPase activity to acid pH. Type IIb fibres will begin to stain positive for myosin ATPase at more alkaline pH (4.6-4.8) than type IIa fibres, which stain at pH 4.5 (Brooke and Kaiser, 1970a, 1970b). However, many different regimes of staining and fibre typing have been described due to technical pitfalls of the staining procedure. Recent immunohistochemistry has updated our understanding of distinctions between fast and slow fibres with developmental and physiological correlates (Hoh et al, 1988).

While it has been suggested that the proportions of type I and type II fibres in a muscle are genetically determined (Åstrand and Rodahl, 1977; Komi et al, 1977), it has been well demonstrated that the nerve innervating the muscle fibre can also influence its contractile characteristics and fibre type (Burke et al, 1971; Munsat et al, 1976). This has been dramatically illustrated in animal studies where nerves innervating slow-twitch and fast-twitch fibres were transected and cross-innervated causing the muscle fibres to alter and partially reverse their fibre types (Buller et al, 1960; Dubowitz, 1967). More recent evidence indicates that the expression of various proteins (Tm, Tnc, Tni, myosin heavy chains, myosin light chains) may not all be nerve-dependent (Gauthier and Hobbs, 1982).

Within a human muscle, fibre type distribution appears mosaic in that type I and II fibres are not grouped together within the muscle (Johnson et al, 1973). Typical fibre type composition of human muscles have been reported to be 50 to 55% type I, 30 to 35% type IIa, and about 15% type IIb, but these proportions vary considerably in different muscles, among individuals in general (Pitman and

Peterson, 1989) and to a lesser extent between males and females (Komi and Karlsson, 1978). Differences in fibre composition can also be seen in different athlete populations. For instance, in endurance athletes, type I fibres predominate, whereas in sprinters or jumpers, type II fibres are more plentiful (Costill et al, 1976; Komi et al, 1977; Thorstensson et al, 1977). Different muscles in normal individuals typically have different proportions of fibre types. For example, m. biceps femoris fibre composition includes about two-thirds type I fibres whereas m. vastus lateralis is typically composed of two-thirds type II fibres (Johnson et al, 1973; Monster et al, 1978).

2.1.3 Muscle Contraction

Muscle fibres are organised into functional units by virtue of their nerve supply. An alpha motor neuron and the muscle fibres it supplies constitute a motor unit. For fine motor control, alpha motor neurons may supply only 2 - 3 muscle fibres, such as in the extraocular or intraocular muscles. Alternatively, a single nerve axon can innervate 1000 - 2000 fibres in muscles where large forces are required, such as in the m. gastrocnemius (Pitman and Peterson, 1989). Therefore, both the number of muscle fibres supplied by a single motor neuron and the number of motor units making up a whole muscle vary, depending on the type and size of the muscle and its function.

All muscle fibres within a motor unit have the same properties, i.e. they are all of one fibre type. When a motor unit is excited, the whole population of muscle fibres within that motor unit contracts. Thus, the functional unit of contraction force

is not the single muscle fibre, but the muscle fibres within a single motor unit. One muscle thus has a mixture of fibre types, and therefore is composed of multiple motor units. In many cases, one fibre type predominates in a muscle, determined in part by the normal functional role of and demands on that muscle. Motor units consisting of Type I muscle fibres are innervated by small, low threshold alpha motor neurons, whereas motor units incorporating type II muscle fibres are innervated by larger, high threshold alpha motor neurons. This "size principle" was first described by Henneman et al (1965). Therefore, postural muscles such as m. soleus are typically innervated by low threshold motor units, while the innervation of the m. gastrocnemius is dominated by the high threshold motor units.

2.1.3.1 Gradation of Tension

Gradation of muscle force is accomplished by the regulation of the rate of motor unit firing and the recruitment of additional motor units by the excitation of more alpha motor neurons.

In normal physiological activity, the first motor units excited are the small motor neurons which innervate the type I muscle fibres. The greater the tension required, the more motor units are excited. The excited motor units fire asynchronously causing them to contract and relax at different times. In addition, the scattering of individual fibres comprising a single motor unit throughout the whole muscle produces a total muscle response which is a smooth contraction with a relatively even distribution of force across the muscle.

As well as producing a smooth contraction, asynchronous motor unit firing

also facilitates a maintained, steady force contraction. If the activated motor units consist of type I muscle fibres, the contraction will also be resistant to fatigue. When a muscle is required to produce greater force, additional, higher threshold motor units are recruited. Larger forces therefore require the recruitment of more and more motor units and the recruitment of the larger, faster and more powerful units. However, because these units consist of Type II muscle fibres, they rapidly fatigue. The maximum contractile force of a muscle can therefore only be maintained for short periods of time. While this "rate order" size principle of recruitment can be reversed by artificially stimulating a motor nerve from an external electric current source, reverse order recruitment under normal physiological conditions has only rarely been seen.

2.1.3.2 Types of Muscle Contraction

When muscle contracts in situ, it exerts a force on its attachments which exerts a turning effect, or torque on the joint spanned by that muscle. Torque is the product of the muscle force and the perpendicular distance (or moment-arm) between the point of applied force and the axis of joint rotation and is expressed in newton meters (N·m). The greater the external resistance, or load is, the greater the muscle force (and therefore the torque) must be in order to produce movement at the joint (Soderberg, 1986).

A muscle contraction which generates force but does not produce movement at a joint is called isometric (iso - constant; metric - length). During an isometric contraction, although no movement is produced and no mechanical work is

performed, muscle work is performed and energy is expended (mainly as heat) (Komi, 1986).

A muscle contraction which produces movement at a joint by generating a constant force, is called isotonic (iso - constant; tonic - force) (Rogers and Cavenagh, 1984). However, isotonic muscle contraction in its truest sense does not exist in the production of joint motion because the moment-arm changes throughout the range of motion and therefore, muscle force must also change (Pitman and Peterson, 1989). A more operational definition of isotonic muscle contraction in kinesiology is that in which the internal force produced by a muscle produces movement against a constant external load (Åstrand and Rodahl, 1977). When the muscle force exceeds the external load, muscle shortening results. This is referred to as a concentric contraction. When the muscle force is exceeded by the external load, muscle lengthening results. This is called an eccentric contraction (Pitman and Peterson, 1989).

The total force that a muscle can produce is influenced primarily by mechanical properties such as its force-velocity, length-tension, and force-time relationships. Factors such as muscle temperature, muscle fatigue, and precontraction stretch also affect force generating capacity (Pitman and Peterson, 1989).

2.1.3.3 Force-velocity relationship

Levin and Wyman reported in 1927 that force production in skeletal muscle is dependent on the speed of shortening and lengthening in animal muscle. The

early work of A.V. Hill (1938), subsequently described what has become the classic hyperbolic force-velocity curve, and its equation:

$$(P + a)(V + b) = \text{constant} = (Po + a)b$$

where P = force of contraction, Po = isometric tension, V = velocity, and a and b are constants.

The force-velocity relationship defines the intrinsic mechanical character of muscle. The shape of the curve for isolated muscle in vitro (Hill, 1938) is similar to that in human movements when a muscle group is involved in variable velocity (Wilkie, 1950) or constant velocity (Komi, 1973; Thorstensson, 1976) movement. Complex movements involving several body joints can also be described by force-velocity relationships (Bosco and Komi, 1979). Briefly, the velocity of shortening is greatest when the external load is zero, and as the load increases, the muscle shortens more and more slowly. When the external load equals the generating force of the muscle, the muscle contracts isometrically. As the load rises, the muscle lengthens while contracting. With increasing loads, the muscle lengthens more quickly (Pitman and Peterson, 1989). Therefore, the greatest force and power capabilities of skeletal muscle occur during muscle lengthening.

2.1.3.4 Length-tension relationship

The tension a muscle exerts also varies with muscle length during activation. This length-tension relationship is determined by the overlapping of myosin and actin filaments, and cross-bridge formation between the two within the sarcomere units of striated muscle (Gordon et al, 1966). At shorter and longer sarcomere

lengths, fewer cross-bridges are possible and force production is also less than maximal. Maximal tension is produced when the sarcomere is at its resting length (2.0 - 2.25μm) where overlap permits the maximum number of cross-bridges to form. In whole muscle-tendon-joint systems, the passive length-tension relation (Carlson and Wilkie, 1974) needs to be added to the active length-tension curve. The passive tissues include tendon and connective tissues which are considered as elastic components in series and in parallel with the active component respectively. The total length-tension curve demonstrates that as a muscle is progressively lengthened beyond its resting length, the passive tension rises and the active tension decreases (Carlson and Wilkie, 1974). Active and passive components exist in every muscle, and their interaction determines the length-tension relationship for a given muscle.

Clinical implications of the length-tension relationship include the use of precontraction stretch to modify muscle tension. As described above, if a muscle contracts isometrically, it will generate a given level of tension. However, when the muscle is lengthened before or during contraction, the tension output is increased. Thus by applying a stretch (lengthening) during the course of a voluntary contraction, a greater tension can be applied to the required function.

2.1.3.5 Force-time relationship

The force generated by a muscle is also proportional to the speed of contraction. The relationship is best represented by the measurement of a maximal voluntary isometric contraction plotted against contraction time. As contraction

begins, the contractile elements of muscle stretch the elastic components within the muscle, producing a nonlinear increase in force. This phase is followed by a linear increase in force. Maximum force is reached when no further stretching of the series elastic component is possible. The resulting force-time curve is also dependent on the joint angle at the time of contraction (Soderberg, 1986).

2.1.4 Muscle Actions

Muscles are often classified by their function in the performance of specific movements. Among the most common terms are agonist, antagonist, synergist, stabilizer, and fixators, but their definition varies considerably in the literature (MacConaill and Basmajian, 1977; Gowitzke and Milner, 1981; Hay and Reid, 1982; Basmajian and DeLuca, 1985; Soderberg, 1986) This variability requires definition of each term.

A muscle primarily responsible for performing a specified movement by producing the most important force generating a torque is called an agonist (or prime mover) (Soderberg, 1986). Other muscles which contribute significantly to the movement, but are not primary to it are classified as assistant movers by some authors (Gowitzke and Milner, 1981; Hay and Reid, 1982; Soderberg, 1986), but not others (MacConaill and Basmajian, 1977; Basmajian and DeLuca, 1985). Muscles which oppose a given movement, and whose action on a joint is opposite to that of the agonist (MacConaill and Basmajian, 1977; Gowitzke and Milner, 1981; Hay and Reid, 1982; Soderberg, 1986), or negatively contribute to that particular movement (Basmajian and DeLuca, 1985) are called antagonists.

Knee movement can illustrate these definitions. Active knee extension is produced by the quadriceps muscle group (agonists), and this movement is opposed (if they are active) by the hamstring muscle group (antagonists). During active knee flexion, the hamstrings become the agonists and the quadriceps are antagonists. The relationship between agonist and antagonist is straightforward when muscle shortening occurs in the direction of joint movement.

However, a large proportion of muscle activity during activities of daily living occurs during muscle lengthening against an external load such as occurs in the quadriceps when walking downstairs. In such activity, the muscle producing the force actually resists the direction of joint motion while it acts as an agonist. During this complex type of joint movement, when active shortening and lengthening can resist joint movement, agonists are practically defined as those muscles which generate the greatest force during the movement. Clinical muscle testing during a cycle of resisted quadriceps shortening which is followed by resisted quadriceps lengthening, can be used to illustrate this type of agonist activity. The joint movements which result from such a sequence are knee extension followed by flexion. Since the quadriceps produce the primary force during both phases of the cycle, they are considered the agonists for the whole cycle. Consequently, the hamstrings are by definition, the antagonists for the whole cycle of testing resisted quadriceps torque.

2.2 Clinical Muscle Performance Testing

The practice of physiotherapy commonly measures the force produced by

muscle contractions. In order to evaluate strength or its limitation, complex muscle testing must account for many components and the wide variability in a normal population. "Performance" and variability are affected by many factors such as age, weight, sex, physical development, previous level of activity, test position, and type of contraction, as reviewed by Mayhew and Rothstein (1985). The ability to detect reproducible changes in muscle performance and their comparison with expected functional norms is essential to all clinicians involved in rehabilitating the physically disabled.

Clinical muscle performance tests ultimately aim to measure the functional ability of a muscle or muscle group. Performance is used to evaluate fitness and athletic ability in trained individuals, and to assess injury and disability in a medical rehabilitation setting. Muscle performance can be quantified by its primary components of force, power, work, and endurance (Mayhew and Rothstein, 1985). Some or all of these parameters can be measured using the three forms of muscle contraction used clinically: isometric, isotonic, and isokinetic (constant velocity) contractions. Each mode has specific advantages and disadvantages in evaluating muscle performance.

2.2.1 Isometric Muscle Testing

The traditional clinical method of measuring muscle performance has been the manual isometric muscle test, originally described as gravity tests by Lovett and Martin (1916). A numerical system was added to the tests by Lawson (1922) to provide a more quantitative measure of strength. This type of manual muscle testing

became a vital part of the evaluation of agents designed to combat paralytic poliomyelitis in 1951 (Daniels and Worthington, 1986). Although still in common clinical use today, manual muscle testing has limited application in scientific studies because it is a subjective rather than an objective measurement tool. There can also be problems with intra- and inter- tester reliability (if tester training is not sufficient), and poor sensitivity (i.e. significant weakness can go undetected) (Beasley, 1961; Nicholas et al, 1978). However, such tools have been used successfully in recent trials (Brooke et al, 1983; Florence et al, 1984; Mendell et al, 1989).

There are more quantitative measures of isometric force which are in clinical use. The cable tensiometer and various portable myometers have been found to be valid and reliable (Hosking et al, 1979; Marino et al, 1982; Scott et al, 1982; Hyde et al, 1983a, 1983b; Mathiowetz et al, 1984; Mayhew and Rothstein, 1985; Bohannon, 1986; Finucane et al, 1988; Bäckman et al, 1989). The advantage of isometric testing is its relative simplicity and the minimal equipment required to do the tests. However, it cannot measure dynamic muscle performance.

2.2.2 Isotonic Muscle Testing

Force, work and power are not easily measured in human muscle testing when velocity is not controlled. The difficulty arises because the changing mechanical advantage of the limb-lever system alters the forces applied by and to the muscles through their range of motion. During isotonic contractions, the load applied to the contracting muscles is maximal at points where the mechanical

advantage of the muscles is minimal (e.g. the limits of the range of movement in knee flexion-extension) (Osternig, 1986; Baltzopoulos and Brodie, 1989). Therefore, while this form of muscle testing measures a muscle's performance during dynamic contractions, all that can be measured is the resistance force (the maximum that can be overcome by the muscles at their lowest mechanical advantage) and the completed number of repetitions (Hettinger, 1961).

2.2.3 Isokinetic Muscle Testing

Muscle performance tests under constant velocity conditions are becoming common clinical procedures and the practice has been recently reviewed (Watkins and Harris, 1983; Osternig, 1986; Baltzopoulos and Brodie, 1989). The advantage of constant velocity testing over isometric testing is that constant velocity is a dynamic test which allows the behaviour of muscle to be measured during shortening and lengthening. The advantage of isokinetic over isotonic testing is that during constant velocity, the muscle can be maximally activated throughout the test range of motion. This allows the dynamic tension-generating capacity of the muscle to be measured. Depending on the testing apparatus, evaluation can be made within a pre-defined range of motion over a range of constant velocities while the muscle group is either shortening or lengthening. The length-tension and force-velocity characteristics of the muscle group can therefore be studied in humans.

The force or torque recorded by the KIN/COM™ and similar dynamometers is generally assumed by clinicians to accurately reflect the tension produced by agonist muscles, and test results are invariably reported as such. However, questions

have been raised regarding what is actually being measured by these machines (Herzog, 1988) and whether the high level of confidence with which data are used to make clinical judgments is justified based on the lack of scientific evidence to support such use (Rothstein et al, 1987).

2.2.3.1 Pre-clinical machines

Cable tensiometers and similar equipment have been in common use for many years for the measurement of static muscle force. However, until the late sixties, devices used to measure dynamic qualities of human muscle were confined to the laboratory and not readily available for clinical use (Dern et al, 1947; Wilkie, 1950; Abbott and Aubert, 1951; Abbott et al, 1952; Abbott and Bigland, 1953; Bigland and Lippold, 1954). Pre-commercial constant velocity dynamometers were usually designed only to study a certain muscle group under specific loading conditions (Doss and Karpovich, 1965; Cavagna et al, 1968). Doss and Karpovich (1965) constructed a dynamometer specifically to measure the dynamic strength of elbow flexors. It consisted of pulleys, a strain gauge bolted to the floor, and a windlass operated by the tester. Since the tester had a mechanical advantage of 9:1 over the subject, he was able to easily control the movement which was allowed to occur at 5°/s. Analysis of the torque-angle curves suggests that the velocity was held reasonably constant considering it was under manual control. Differences predicted by Hill's equations (Hill, 1938) were demonstrated in elbow flexor torque between concentric, eccentric and isometric contractions (Doss and Karpovich, 1965).

Cavagna et al (1968) developed a machine for dynamic human muscle testing based on a design by Levin and Wyman in 1927. However, that device was not driven by weight (Levin and Wyman, 1927), or by springs (Hill, 1950), but by a piston fed by compressed air and operated by an electrovalve. Using this device, Cavagna et al (1968) were able to show that during muscle shortening after an initial stretch, the force developed by the contractile component is greater than that developed when it shortens from an initial isometric contraction at the same velocity and length. This phenomenon has since been named the 'stretch-shortening cycle' (Norman and Komi, 1979).

2.2.3.2 The Cybex II® and similar machines

The first machines designed for clinical use to measure maximal effort dynamic muscle contractions of several different muscle groups were conceived and developed by Perrine in the late sixties (Hislop and Perrine, 1967; Thistle et al, 1967; Moffroid et al, 1969). Perrine (1969) defined the term "isokinetic exercise process" as:

> "The process and apparatus for isokinetic muscular exercise provides for exercise movements which are initiated and maintained by active muscular forces continuously provided by a person exercising. The speed of an exercise movement is allowed to accelerate essentially unopposed by resistance forces from zero to a preset or pre-determined rate of speed, and any magnitude of muscular force tending to accelerate the exercise movement beyond the pre-determined rate of speed is counteracted by the system, thereby establishing the pre-determined rate of speed as the maximum rate of speed attainable and loading the particular muscles in proportion to the force they can develop at such speed."

An isokinetic contraction was also more concisely defined as "a dynamic

muscular contraction when the velocity of movement is controlled and maintained constant by a special device" (Thistle et al, 1967). There was a great deal of interest in the development of the Cybex I within the Physical Medicine, Rehabilitation and Sports Science communities because it was the first commercially available device that measured muscle performance during limb movement. It was anticipated that muscle strength and endurance training on such a device would be more effective and efficient than with either isometric or isotonic exercise, because isokinetic contractions would allow muscles to contract maximally through a range of motion.

Technical modifications and improvements to the original design led to the introduction of the Cybex II® which is probably still the most popular muscle performance test and exercise machine. The Cybex II® dynamometer consists of an electric motor that rotates an axle. The speed of rotation of the axle can be preset to various levels and is kept constant by a generator-servo control feedback unit (Perrine, 1968). The axle of the motor is connected to a rotational lever arm by a clutch coupling that allows free movement of the lever arm at all preset angular velocities about that of the motor driven axle. When the lever arm is moved by an external force and its angular velocity reaches that of the rotating axle, the coupling engages. A braking action, produced by a system of gear reductions between the motor and rotational axle, inhibits the lever arm from rotating faster than the motor driven axle. Therefore, when maximum force is applied to the dynamometer over a range of movement, dynamometer resistance will be proportionally applied at different joint angles.

The torque from the resulting movement is sensed by an internal load cell within the motor housing. The load cell uses hydraulic pressure to actuate a pressure gauge. As well, a transducer converts the pressure signal to an electrical one, which in turn controls the mechanical circuit (Perrine, 1968; Nelson et al, 1973). The muscle torque analogue signal is then transmitted to a dual channel strip chart recorder. One channel records the torque of the active muscles and the second channel simultaneously records the joint angle or range of motion. More recently, computers have been linked to the Cybex II®, including the Apple II+ (Apple Computer Inc., Cupertino, California), and a dedicated microprocessor from Lumex Corporation (Ronkonkoma, New York).

Although the Cybex II® is the most used dynamometer in the isokinetic research literature, there are a growing number of reports which question its validity and reliability (Winter et al, 1981; Sapega et al, 1982; Watkins and Harris, 1983; Murray, 1986; Murray and Harrison, 1986; Rothstein et al, 1987; Bemben et al, 1988; Herzog, 1988). The first major drawback of the Cybex II® dynamometer to be recognised was the interpretation of the measured torques as the resultant joint torques (Winter et al, 1981). This was the implicit assumption of several early investigators (Thorstenson et al, 1976; Perrine and Edgerton, 1978; Osternig et al, 1983) and is still not recognised as a source of measurement error in many studies (Dibrezzo et al, 1985; Falkel et al, 1985; Lankhorst et al, 1985; Burnie and Brodie, 1986; Agre et al, 1987; Hsieh et al, 1987; Whipple et al, 1987; Johansson et al, 1989). When the dynamometer's torque values are not corrected for gravity, errors

of up to 79% in resultant joint torque, and over 500% in work measurements have been reported (Winter et al, 1981). This error increases at high velocities whenever torque values are small and large limb segments are being measured (Winter et al, 1981; Fillyaw et al, 1986). While this kind of measurement error is not necessarily restricted to Cybex II® type dynamometers, gravity correction on dynamometers such as the KIN/COM™ dynamometer is technically much easier to measure and incorporate into data analysis. The continued use of uncorrected torque values has been described as unwarranted and inappropriate (Rothstein et al, 1987).

A major problem with electric motor controlled velocity devices such as the Cybex II® is the torque "overshoot" phenomenon (Sapega et al, 1982). The overshoot is seen in the strip chart recordings as a prominent spike at the beginning of the torque angle curve, followed by a series of progressively diminishing oscillations. Apparently this results from the design of the dynamometer, as the moment-arm is not connected directly to the electric motor that drives it. This indirect connection allows some mechanical play, or backlash in the arm movement, and because the Cybex II® uses a gear mechanism in the drive, there is a "catch-up" motion required at the start of a movement before the preset velocity is reached. This results in an early phase of uncontrolled acceleration when torque is not measured or recorded, even though muscles are contracting to produce the movement. Once reached, the preset velocity is maintained by a resistance applied to the moment-arm by the dynamometer. By this time, the limb has accelerated to velocities up to 200% above the preset velocity (Sapega et al, 1982) and an

overshoot spike is recorded reflecting the reaction of the dynamometer to the speeding limb moment-arm. This overshoot also occurs at the beginning of the deceleration phase, but to a much smaller degree (Sapega et al, 1982). If the overshoot is interpreted as the peak muscle torque (as is often the case in clinical practice), the true maximum muscle torque will be overestimated with consequent misinterpretation of data. Subsequent design modifications to the equipment added an adjustable damping mechanism to the system, which could decrease the overshoot. Such analog filters, while convenient, also have limitations which can significantly affect the whole torque-angle curve (Osternig et al, 1982; Sapega et al, 1982; Sinacore et al, 1983). A study of these important shortcomings concluded that (1) the Cybex II® dynamic calibration protocol should not be used; and (2) use of the Cybex II® damping filter can produce false estimates of joint torque. Furthermore, (3) inertial corrections need to be applied to Cybex data in order to minimise serious errors due to uncontrolled angular acceleration; and (4) the use of the Cybex damping settings should be discouraged (Murray and Harrison, 1986; Murray, 1986).

2.2.3.3 The Kinetic Communicator

The Kinetic Communicator or KIN/COM™ (MED-EX Diagnostics of Canada, Inc., Coquitlam, BC) was designed to measure human muscle performance during muscle shortening and muscle lengthening, and later developed for manufacture on a commercial scale. The system is computer controlled, incorporating software programs to produce isometric, isotonic, isokinetic, or passive test modes. All major

muscle groups can be tested in isolation by use of various exercise and positioning attachments fitted to the machine. The following design details are excerpts from the KIN/COM™ operating manual and accompanying reference (Short et al, 1986).

The essential components of the constant flow hydraulic resistance system include the moment-arm and actuator, a motor-pump unit, a servo valve, an optical shaft encoder, a load cell, and a computer. These elements comprise a closed feedback loop whereby the force, velocity, and displacement of the moment-arm are constantly monitored by the computer, and the computer output controls the motion of the arm accordingly.

When a force is exerted against the moment-arm, it will rotate at the velocity dictated by the test mode selected (isometric, isotonic, constant velocity, or passive). The arm is made to rotate by the hydraulic actuator, which in turn receives its power from the motor-pump unit via hydraulic lines. The servo valve in the hydraulic power circuit controls the angular velocity and direction of the actuator. A ±5 V DC signal generated by the computer controls the servo valve, and therefore ultimately controls the moment-arm motion. The feedback parameters to the computer are the level of force exerted by the subject, the moment-arm angle, and the moment-arm velocity.

The applied force is detected by a strain gauge bridge on the moment-arm. Arm displacement is measured by a digital optical shaft encoder. Arm velocity is calculated in the software as a function of arm displacement per unit time, using a sampling rate of 100 Hz.

With this feedback loop, any change in force or velocity will initiate a response by the computer to adjust the servo valve accordingly. For example, in the constant velocity mode, a change in force initiates an increase in hydraulic pressure to maintain a constant arm velocity, regardless of the applied force. An IMS 5000 computer (IMS International, Carson City, NE) controls the dynamometer and collects, stores, and processes the data. One of the two 5¼" disk drives handles the system software disk, and the other is used for data capture.

The advantages of the KIN/COM™ over other dynamometers such as the Cybex II® and Orthotron® include (Short et al, 1986):

1. A high dynamic frequency response of 20 Hz.
2. Force measurement is completely independent of velocity. A strain gauge bridge acts as the load sensor, which is located directly where the subject applies force. This eliminates any inertial artifacts arising from the moment-arm itself.
3. There is no mechanical play or backlash in the arm movement because the moment-arm is directly connected to the hydraulic actuator that drives it. Without a gear or clutch mechanism in the drive, there is no "catch-up" motion required during a test to attain the desired velocity. The torque overshoot phenomenon and high velocity artifacts which occur with other dynamometers (see section 2.2.3.2) are thus eliminated in the KIN/COM™ design.
4. Velocity feedback is provided both on-line and in hard copy. Thus, it

is possible to see whether a test is proceeding at constant velocity in real time and over which part of the range of motion constant velocity is maintained.

5. Motion of the moment-arm can be controlled in either direction, and so both concentric (muscle shortening) and eccentric (muscle lengthening) muscle contractions can be tested. The ability to measure muscle performance during lengthening allows for the effect of the series elastic component of muscle to be studied.

6. The moment-arm rotates only when force is applied to the force transducer; a useful safety feature.

7. A sub-program which compensates for limb weight is available. Limb weight is resolved by vectors over the entire ROM and subtracted from the raw force values.

The validity and reliability of the KIN/COM™ dynamometer for measuring force, angle, and velocity have been comprehensively assessed and reported (Farrell and Richards, 1986). Reliable test protocols have been developed for muscle groups crossing large joints including the knee flexors and extensors (Harding et al, 1988; Highgenboten et al, 1988; Snow and Johnson, 1988a, 1988b; Tredinnick and Duncan, 1988; Kramer, 1990); the elbow flexors (Griffin, 1987); and the shoulder flexors (Snow and Weare, 1988).

2.3 Measurement of Muscle Activity

2.3.1 The EMG signal

An alpha motor neuron and all the muscle fibres it supplies make up one motor unit. When a nerve impulse (or action potential) propagates down the axon of an alpha motor neuron, it will excite all the muscle fibres in that motor unit. This excitation produces a change in the membrane potential of a muscle cell from negative to positive resulting in the depolarization of the whole sarcolemmal membrane. The depolarization is accompanied by the movement of ions which in turn generates an electromagnetic field. It is this electrical manifestation of the neuromuscular excitation that is detected by electrodes and is called the electromyogram or EMG signal (Basmajian and DeLuca, 1985).

In order to sustain a whole muscle contraction, many motor units must be activated repeatedly. The resulting EMG signal comprises of a series (or train) of motor unit action potentials consisting of positive and negative spikes of varying amplitude which represent the superimposed activity of a number of motor units. In order to increase the tension of a muscle contraction, additional motor units must be activated, resulting in an EMG signal of increased magnitude and complexity.

There are many variables that can influence the EMG signal at any given time such as velocity of muscle shortening or lengthening, rate of tension rise, muscle fatigue, and reflex activity. Therefore, it is not surprising to find variations in the amplitude and duration of the EMG signal among muscles and between subjects. In normal muscle, a typical peak-to-peak EMG signal amplitude (detected with

indwelling electrodes) is 500μV with a range from a few microvolts to 5mV (Basmajian and DeLuca, 1985).

2.3.2 Detection and processing methods

Due to its small amplitude, the EMG signal from contracting muscle must be detected, amplified, processed, and recorded before it can be analyzed. The signal is detected by either indwelling or surface electrodes. Each type has advantages and disadvantages. While indwelling electrodes are relatively easy to implant and withdraw, and detect clear EMG signals, they are subject to kinking and displacement during contraction, and have poor day-to-day reliability (Komi and Buskirk, 1970; Jonsson and Komi, 1973). There is also some controversy over whether their pick-up is broad, or is limited to a few myofibres (cells) within a muscle and therefore not representative of the muscle activity as a whole. However, they are the method of choice when precision measurement is needed, or when studying deep or adjacent muscles (Basmajian and DeLuca, 1985).

Surface electrodes such as the commercially available Beckman (Beckman Instruments, Palo Alto, CA) silver-silver chloride electrodes have the advantage of being easy to apply with no discomfort for the subject. The positioning and orientation of the surface electrodes can be easily standardized so repeatability of measurements is higher than with indwelling electrodes (Merletti and DeLuca, 1989). Their light mass, small size, high reliability and durability have made surface electrodes very popular for kinesiological studies, and they are commonly used to detect gross EMG signals consisting of electrical activity from numerous motor units

(Basmajian and DeLuca, 1985).

The amount of EMG signal detected by surface electrodes is generally representative of the superficial activity of the underlying muscle (Bouisset and Maton, 1973), but the precise determinants include several factors such as size of the electrodes, location over the muscle, inter-electrode spacing, electrode-to-muscle fibre distance, orientation with respect to the underlying muscle fibres, the magnitude of the depolarization, and the conductance of the tissues between the fibre and the electrodes (Basmajian and DeLuca, 1985). The interplay of these and other factors produces a complex waveform made up of components that vary in frequency from one to 1000 Hz (Soderberg and Cook, 1984). Although there is consensus that electrode distance, size, and orientation to the muscle fibres will influence the recorded EMG signal, no prescribed methods for locating surface electrodes exist (Soderberg and Cook, 1984).

Since electrodes will detect all electrical signals in their vicinity, including unwanted electrical noise from sources other than the muscle under investigation, a bipolar electrode configuration can be used over the muscle site. The two electrical potential signals detected by these electrodes with respect to the reference electrode are subtracted one from the other in a differential amplifier, and the difference amplified. In this way, signals (muscle or otherwise) received simultaneously and with equal magnitude at both electrodes will automatically be cancelled. The ability of EMG equipment to remove these common signals is given by its Common Mode Rejection Ratio.

Electrodes of about 8mm diameter are appropriate for recording activity from the large muscles of the thigh (Gilmore and Meyers, 1983). Generally, the larger electrodes monitor greater activity of the muscle volume. The greatest signal is recorded when the electrodes are placed on a longitudinal axis in the direction of the muscle fibres (Ahlgren et al, 1980), and significant reduction in signal voltage is seen with a transverse siting. There also appears to be no advantage in sensitivity gained by selecting the motor point for electrode sites (Zuniga et al, 1970). The optimal inter-electrode spacing varies according to the size of the muscle being monitored and its proximity to other muscles. Generally, the further the distance between electrodes, the broader the pick-up area of muscle activity.

The EMG amplifiers are needed to enlarge the muscle EMG signal, and for observation their output must be linked to a visual display such as an oscilloscope or chart recorder. Amplifiers serve several functions including, isolation between the signal source and the recording instrumentation, distortionless reproduction of the bioelectric signal, voltage gain, and noise reduction (Soderberg and Cook, 1984). The sensitivity of the amplifier should be such that it can detect signal amplitudes from 100μV to 5mV peak-to-peak, and accommodate input gain ranges from 100 to 10,000 (Cooper, 1982). Independent of the amplifier gain, the amplitude of the signal (in mV) should be reported as it appears at the electrodes. In order to avoid attenuation of the signal due to voltage drop across resistances, amplifier input impedance should be at least 100 times greater than the electrode/skin impedance or at least 1Mohm for surface electrodes. In addition, EMG amplifiers should have

high and low frequency filters which establish a bandwidth within which signals are monitored. The bandwidth is a compromise between preserving as much signal information as possible while eliminating frequencies which contain interference and typically range from 10 to 1000 Hz for surface EMG with most of the signal concentrated between 20 and 200 Hz (Cooper, 1982).

In order to simplify quantification of EMG activity, processing of the EMG signal is required. Full wave rectification inverts the polarity of the negative portion of the signal and superimposes it on the positive portion, and is often useful for peak amplitude analysis. Additional processing of the signal with a low pass filter limits the higher frequency components and "smooths" the signal to provide easier definition of the amplitude changes that occur. This form of processing tends to connect the peaks of the signal and forms what is called a linear envelope, denoting the shape of the signal trace on an oscilloscope (Soderberg and Cook, 1984).

2.3.3 EMG Cross-talk

In kinesiological research, surface electrodes are often used to detect muscle activity. With this type of electrode, convenience for subject and investigator are combined with the advantage of a large "pick-up" area, so that the electrical activity over a large muscle mass can be detected. This large pick-up area can be a disadvantage however, because muscle action potentials spread by volume conduction through adjacent tissues, producing potentials that may be recorded at considerable distances from their source (Gydikov et al, 1982). Therefore, because EMG signals are not restricted to anatomical borders, signals can be attributed to

one muscle which might originate from a second muscle. This phenomenon is known as cross-talk (O'Connell and Gardner, 1963; Hof and v.d.Berg, 1977).

Therefore, in studies of cocontraction where the muscles under study are usually in close proximity, it is important to establish that the recording of the antagonist EMG signal is not contaminated by the EMG signal from the agonist muscle.

2.4 Studies of cocontraction

Since Sherrington's 14 classic papers in the early 1900's on reciprocal innervation of antagonist muscles (Sherrington, 1909), it has generally been thought that antagonist muscles are inactive during most voluntary movements. Sherrington demonstrated a fundamental activity pattern of reflex activity and called it "reciprocal innervation". The definition can be paraphrased to state that when a muscle or muscle group contracts, its antagonist must relax in order for movement to occur. While Sherrington was not the first to put forward this hypothesis (Bell, 1823; Hering, 1895 & 1898; Beevor, 1891), he was the first one who named the phenomenon and demonstrated that it occurred during flexor reflex activity (Sherrington, 1906). Sherrington studied three examples of simple reciprocal innervation. Two were segmental reflexes (the limb flexion and knee jerk reflexes), which were studied in spinal or decerebrate preparations. The third was reciprocal inhibition elicited in the elbow flexors by electrical stimulation of the motor cortex (Sherrington and Hering, 1897). These studies provided the impetus in the fields of neuroanatomy and neurophysiology to explain in considerable detail how and

where these fundamental reflexes were mediated. However, that same impetus stifled any research which might suggest anything other than reciprocal inhibition as a significant factor in motor control. Reports by some of Sherrington's contemporaries (Beaunis, 1889; Demeny, 1890) and the evidence provided by Tilney and Pike (1925) that "muscular coordination depends primarily on the synchronous cocontraction relation in the antagonist muscle groups", were strongly opposed by the scientific community (Holmes, 1927; Pollock and Davis, 1927; Walshe, 1927; Wilson, 1928). Since then, the ability of the nervous system to control both reciprocal muscle activation and cocontraction has either been denied, neglected or ignored (Smith, 1981). For the next 50 years, the study of antagonist cocontraction received only sporadic attention (Wachholder and Altenberger, 1926; Gellhorn, 1947; Wilkie, 1950; Levine and Kabat, 1952; Barnett and Harding, 1955; Duchenne, 1959; Person, 1960; Travill and Basmajian, 1961; Patton and Mortensen, 1971; Bouisset and Lestienne, 1974; Polit and Bizzi, 1979).

From a detailed review of these and other studies, Smith (1981) compiled a list of motor states which he thought were controlled by invoking either reciprocal contraction or cocontraction:

Conditions favouring reciprocal contraction:

1. When external resistance prevents displacement or muscle shortening by the prime movers (exception -isometric prehension).
2. In rhythmic motor processes (locomotion, mastication, respiration) where the activity of the agonist-antagonist muscles alternates.

3. Low velocity limb displacements without load.

Conditions favouring cocontraction:

1. When muscle tension or limb position must be precisely monitored without load (e.g. the initial phase of learning a new skill).
2. In high velocity limb displacements or under loaded conditions when a joint is required to be stiff (e.g. a strong cocontraction to decelerate limb).
3. Isometric prehension or power grip (for stabilizing the wrist and adding stiffness to the carpal and metacarpal phalangeal joints).

These latter conditions have been supported in part, and modified by Basmajian and DeLuca (1985). They included additional movements in infants and children, and patients who had spasticity to the cocontraction grouping; but considered that high velocity movements involved reciprocal inhibition (Basmajian and DeLuca, 1985).

From these possibilities, it is difficult to determine whether reciprocal contraction or simultaneous cocontraction of agonist and antagonist muscles should occur during constant velocity testing. Constant velocity testing usually involves several rhythmic contractions through a range which would favour reciprocal contraction. But these rhythmic contractions are also performed under loaded conditions, and sometimes at high velocity, which may favour cocontraction (Smith, 1981).

Much of the recent research on antagonist cocontraction can be divided into

3 movement categories: (1) static, (2) slow, and (3) fast limb movements.

2.4.1 Static Conditions

One of the best examples of antagonist cocontraction is prehension (Smith, 1981; Basmajian and DeLuca, 1985). Many studies have shown that during the isometric contractions of the power and pinch grips, almost all the extrinsic and intrinsic muscles of the hand and forearm are active (Long et al, 1970; Rasch and Burke, 1974). Many of the muscles are active to provide postural stabilization so as to increase the mechanical advantage of the long finger flexors, whereas other muscles provide stiffness at the carpal, metacarpal, and phalangeal joints (Smith, 1981). Such complex activity has been called the "synergic coactivation of antagonist muscles" (Smith, 1981).

Eloranta (1989) studied isometric knee extension torque and the EMG activity of the m. rectus femoris, m. vastus medialis, m. vastus lateralis, and m. semimembranosus of five male human subjects at various knee joint angles, in three postures. In a sitting posture, knee extensor EMG activity was found to decrease in greater degrees of extension. This was particularly observed in m. rectus femoris, which became silent in full knee extension. In the sitting posture, antagonist m. semimembranosus activity was present throughout the test range, ranging from a relatively low level in knee flexion which increased in more extended positions to equal knee extensor EMG activity in full extension.

A more elaborate study involved the EMG measurement of 12 knee muscles in four male subjects under several isometric loading conditions (48-72 N·m

tending to flex the knee; and 8-32 N·m tending to extend the knee) (Andriacchi et al, 1984). They found that individual muscle responses were not only dependent on knee flexion angle, but also on the direction, magnitude and combination of external torques. While little or no antagonist activity was reported in either the quadriceps or hamstrings muscle groups at any knee angle or joint load tested, actual antagonist quadriceps and hamstring data were not presented. As well, any definition of "little or no activity" was omitted.

Insignificant antagonist hamstring activity during isometric knee extension was also reported by Grabiner et al (1989). In that study, nine healthy male subjects produced knee extension torques ranging from 10% to 100% of each subject's MVC at a knee flexion angle of approximately 20°. Hamstring cocontraction was shown to increase only marginally from 9.6% of maximal agonist activity at 10% MVC, up to 13% at 100% MVC (Grabiner et al, 1989). They concluded that such low levels of antagonist activity could not have a significant effect on reducing anterior cruciate ligament (ACL) strain as suggested by others (Solomonow et al, 1987). However, a close look at their data reveals the subject variability for hamstring cocontraction to be high, particularly when compared to the variability of the quadriceps. For example, at a quadriceps muscle tension of 100% MVC, the coefficients of variation for the quadriceps and hamstrings normalised EMG values were 18% and 85% respectively. This indicates that hamstring cocontraction is quite variable, and therefore could be of clinical significance.

The maximal possible cocontraction of quadriceps and hamstring and whether such cocontraction has a clinically important effect are not yet known. These questions have recently been studied by two groups (Yasuda and Sasaki, 1987a and b; Draganich and Vahey, 1990). After calculating the forces exerted on the tibia by separate isometric contractions of the quadriceps and hamstrings, Yasuda and Sasaki (1987a and b), studied the effects of maximal voluntary contractions of those muscles alone and in combination, on the anterior and posterior shear forces in the knee at various joint angles in 20 healthy adult males (Yasuda and Sasaki, 1987b). After practising for 5 minutes, subjects were asked to cocontract their quadriceps and hamstrings maximally while maintaining a specified angle of knee flexion. Levels of cocontraction ranged from 30% to 60% of a muscle's isolated maximum voluntary contraction with no statistically significant angle dependence between 5° and 90° of knee flexion (Yasuda and Sasaki, 1987b). This level of voluntary isometric cocontraction however, produced a significant shift in the resultant shear force to a posterior shear for all but the last 5° to 10° of knee extension. This contrasted with an anterior shear force from 45° to 5° knee flexion when only the quadriceps were contracting. It was concluded that maximal antagonist cocontraction of the knee flexors significantly decreased anterior shear and therefore decreased strain on the ACL (Yasuda and Sasaki, 1987b).

The reduction in ACL strain by cocontraction of knee flexors is supported in a more recent study of six cadaver knees (Draganich and Vahey, 1990). It was found that, when compared to ACL strain at 0° knee flexion, isometric quadriceps

muscle forces alone produced significantly greater strain on the ACL between 0° and 40° of knee flexion but not between 50° and 90° knee flexion. As well, simultaneously applied quadriceps and hamstrings forces significantly reduced ACL strain at 10°, 20°, and 90° of knee flexion (Draganich and Vahey, 1990).

2.4.2 Slow Movements

Antagonist cocontraction has been studied during slow voluntary isotonic movements (Patton and Mortensen, 1971; Carlsöö et al, 1973; Draganich et al, 1989). Cocontraction was found to be greater during extension than during flexion when the antagonists were acting in synergy with gravity (Patton and Mortensen, 1971; Draganich et al, 1989). Antagonist cocontraction was only seen at the end range of movement in unloaded conditions, but with increasing load (Patton and Mortensen, 1971; Draganich et al, 1989), and velocity (Carlsöö et al, 1973), the levels of antagonist activity increased, and these muscles became active earlier in the range of movement (Patton and Mortensen, 1971; Carlsöö et al, 1973; Draganich et al, 1989). Maximum antagonist hamstring cocontraction was reported to be between 10% and 20% of activity recorded during a maximal voluntary contraction (Draganich et al, 1989). Overall, cocontraction levels were considered to be less in skilled and strong subjects (Patton and Mortensen, 1971).

2.4.3 Fast Movements

While relatively few studies have examined agonist-antagonist interactions under the above contraction conditions (DeLuca and Mambrito, 1987), there have been many studies in recent years on agonist-antagonist interactions in the control

of rapid movements (Hallett et al, 1975; Angel, 1977; Hallett and Marsden, 1979; Jacobs et al, 1980; Brown and Cooke, 1981; Ghez and Martin, 1982; Marsden et al, 1983; Benecke et al, 1985; Mustard and Lee, 1987; Brown and Cooke, 1990; Cooke and Brown, 1990, and others). In fast movements, a regular three burst electromyographic pattern of phasic muscle activity, called the "triphasic pattern", has been described as a basic unit of movement control (Wachholder and Altenburger, 1926; Hallet et al, 1975; Brown and Cooke, 1981). It consists of an initial agonist burst (AG1), followed immediately by an antagonist burst (ANT) which in turn is followed by another agonist burst of activity (AG2). It is thought that AG1 produces the torque which acts as the driving force to initiate movement and accelerate the limb; ANT decelerates the limb and stops the movement; and AG2 acts to provide any necessary changes to final limb position (Cooke and Brown, 1990).

The various parameters which define the triphasic pattern have been studied in some detail. There is general agreement that (1) the magnitude of AG1 increases with movement amplitude and speed (Hallet and Marsden, 1979; Brown and Cooke, 1981; Marsden et al, 1983; Benecke et al, 1985); (2) the magnitude of AG1 is dependent on the inertia of the load being moved (Lestienne, 1979); and (3) the duration of AG1 increases with movement amplitude (Benecke et al 1985; Mustard and Lee, 1987). The details of ANT are less established, but evidence suggests that the timing of ANT is related to the time of peak velocity of the movement (Cooke and Brown, 1990), and ANT is often associated with a transient silent period in

AG1 (Angel et al, 1965). Even less is known about AG2, probably because it is difficult to determine the timing of the burst (Brown and Cooke, 1990). However, the recent studies of Brown and Cooke have provided convincing evidence which suggests that this triphasic pattern is really a merging of two separate and independent actively controlled functions, namely acceleration and deceleration. In an elegant series of experiments (Brown and Cooke, 1990; Cooke and Brown, 1990), normal human subjects were asked to perform a phase plane tracking task (Cooke and Brown, 1986). This involved making constant velocity voluntary horizontal flexion/ extension movements at the subject's supported elbow while grasping a vertical rod attached to what was described as a manipulandum. Constant velocity movements were achieved by requiring the subjects to track a target presented on an oscilloscope over a template. The template consisted of a phase-plane representation of a constant velocity movement. Therefore, if the subject could reproduce the template on the oscilloscope screen by moving the rod, the subject would be making the desired constant velocity movement. It was noted that it took several minutes of training for subjects to become proficient at producing consistent constant velocity tracking movements, and not all subjects could achieve such proficiency.

In proficient subjects, the two underlying properties of the triphasic pattern (i.e. acceleration and deceleration) could be studied by changing the properties of the target template. In this way, movement amplitude, duration and velocity could be varied. First, it was shown that phase-plane tracking movements could simulate

step tracking movements, (the method used in previous studies of the triphasic pattern in which only endpoint and/or duration of movement were specified) (Brown and Cooke, 1990). The subsequent experiments involved the addition of a constant velocity movement phase of approximately 800 ms between the acceleration and deceleration phases. This separation revealed a single agonist/antagonist burst during acceleration (AG1/ANT1) and a single antagonist/agonist burst during deceleration (ANT2/AG2). By shortening the constant velocity phase sequentially until it reached zero (i.e. incrementally returning to the equivalent of a step tracking movement), it was demonstrated that ANT1 and ANT2 merged, and the triphasic pattern was reestablished. It was concluded that (1) acceleration, deceleration, and their related muscular drives can be treated independently by the motor system and therefore paired agonist/antagonist activation is the basic unit of movement control; (2) AG1 and ANT1 burst were associated with acceleration, and ANT2 and AG2 were associated with deceleration; and that (3) the triphasic pattern is really an "accidental phenomenon" and is in fact a "quadriphasic pattern... produced by the smooth merging of the paired muscular activations related to acceleration and deceleration" (Cooke and Brown, 1990).

2.4.4 Neurological models of hardwiring

Since there is now substantial evidence to support the hypothesis that antagonist cocontraction, in addition to reciprocal contraction, is an integral part of normal human movement, it is important to consider what parts of the nervous

system are involved, what neurological circuitry is used and, what determines whether cocontraction or reciprocal contraction predominates.

It has been suggested that a wide range of motor activities may be generated by a combination of control signals from two partly independent central systems: one organised for reciprocal contractions and one organised for cocontractions (Feldman, 1980a & 1980b). Which control strategy is used depends on the speed and frequency of the movement (Humphrey and Reed, 1983). Well-trained monkeys were presented with a torque tracking task which required the monkey to maintain a fixed wrist position despite various torque perturbations. It was found that reciprocal contractions around the joint were used during slow perturbations, but for rapid perturbations the joint was "tonically stiffened" by antagonist cocontractions (Humphrey and Reed, 1983). It was argued that in order to maintain wrist position over a wide range of perturbation frequencies, the monkey must have to maximise the mechanical wrist impedance. While some of this impedance would be due to passive factors such as the inertia of the system and the viscoelastic properties of the wrist joint structures and surrounding muscles, a major component would involve active muscle contraction and therefore, neural input. A series of experiments led them to conclude that (1) wrist stiffness contributes 90% to 95% to total joint impedance in both active and passive conditions; (2) only 15% to 20% of this stiffness is due to passive factors; (3) the major contribution to wrist stiffness comes from neural activation of the flexor and extensor muscles; and (4) the increase in stiffness is paralleled by an increased response speed (Humphrey and

Reed, 1983).

At the same time, neuronal discharge patterns from the wrist-control area of the precentral motor cortex were monitored, and contour maps were drawn from both these experiments and those involving direct stimulation of the medullary pyramidal tract and red nucleus in the monkey (Humphrey and Reed, 1983). The principal findings showed that the observed cortical cells fell into two major functional groups, partially overlapping each other in spatial distribution. First, there was a low threshold wrist flexor and extensor area where microstimulation produced activity in either the wrist flexors or extensors. Conversely, these cells also responded to activity in either the flexors or extensors, but rarely both. Second, there was a second area surrounding the low threshold area, which, when stimulated, evoked cocontraction of the wrist flexor and extensor muscles. Lastly, cells in this cocontraction zone were generally more tonically active and their activity either increased or decreased progressively in parallel with the discharge of slow-twitch motor units (Humphrey and Reed, 1983). Evidence supporting the red nucleus as a coactivator in this control mechanism has been reported by Cheney and Mewes (1986) while others have implicated the cerebellum (Tilney and Pike, 1925; Brooks, 1979; Massion and Sasaki, 1979; Smith, 1981). It was concluded that the control mechanisms of antagonist cocontraction provided a wide range dynamic control of joint stiffness and hence, joint stability and control of movement (Humphrey and Reed, 1983).

Using a different approach, DeLuca and Mambrito (1987) also found

evidence for separate control of joint movement and stiffness. They rationalised that since joints are mobilised by muscles that generate forces in opposing directions, it must be possible to control separately both the torque and the stiffness at the joint. The net torque at a joint is the difference between the torques of the agonist and antagonist muscles. The net stiffness is the sum of the individual stiffnesses of the agonist and antagonist muscles. Therefore the values of these two variables could range from high torque and low stiffness when either the agonist or antagonist set is individually activated, to zero torque and high stiffness when both sets are maximally coactivated (DeLuca and Mambrito, 1987). Recent advances in EMG signal analysis techniques allowed the decomposition of gross signals into the individual constituent motor-unit action potential trains. The firing rates of motor units within the agonist/antagonist muscle pair flexor pollicis longus and extensor pollicis longus in three male human subjects were studied under a variety of movement conditions. These included (1) stiffening of the joint by voluntary cocontractions, and (2) force-varying isometric contractions. Preview tracking, for which the whole force trajectory was displayed to the subject before contraction began, or instantaneous tracking, where the trajectory was displayed in real time were also used. Cross-correlation analysis of the motor unit activity in the muscle pair pointed to the presence of three central commands for motor control: flex, extend, and coactivation commands. Support for the latter came from the considerable cross-correlation observed with no time shift among the firing rates of the motor units of the two muscles during voluntary joint stiffening. Such

coordinated behaviour in firing rates implies that they are produced simultaneously (DeLuca and Mambrito, 1987). This has been called the "common drive" (DeLuca et al, 1982). It was concluded that (1) the nervous system controls the motor neuron pool rather than individual motor units by a common drive which includes a component of central, supra-segmental origin; (2) cocontraction is used when there is task uncertainty or when compensatory force corrections are required; and (3) reciprocal contractions are used when both of these conditions are present (DeLuca and Mambrito, 1987).

In summary, there is a growing amount of evidence which shows that cocontraction of antagonist muscles commonly occurs in both static and dynamic conditions. In static conditions, the level of cocontraction is generally thought to be small and insignificant, even though it has been shown to reach as much as 60% of the muscle's MVC (at least in the quadriceps and hamstring muscles), and the variability of levels of cocontraction can vary considerably among individuals. During movement, cocontraction is most striking during the acceleration and deceleration phases. It can also be seen to a lesser extent between these phases, where it appears to be load and velocity dependent. Neural circuitry for such a motor control system has been proposed, and parts of it have been identified.

In trying to extrapolate the above findings to clinical constant velocity testing, several questions arise. (1) While several investigators considered antagonist cocontraction during agonist isometric contractions to be small and insignificant, their own data revealed significant individual variation, particulary in antagonist

hamstring activity. Voluntary cocontractions have been shown to reach up to 60% of the muscles' MVC. Such levels of cocontraction, if present during constant velocity testing, may have significant impact on the torque-angle curves and their subsequent interpretation, therefore levels of antagonist cocontraction seen during clinical constant velocity testing should be examined. (2) An increasing number of constant velocity tests now include maximal effort agonist muscle lengthening (AGml) tests. None of the above studies looked at antagonist cocontraction during AGml, so those levels of antagonist cocontractions should be determined. (3) Since constant velocity testing occurs over a range of motion at different velocities, it is important to know if such antagonist cocontractions are angle and/or velocity dependent.

2.5 Cocontraction during Constant Velocity Testing

2.5.1 Ankle

The first study of antagonist cocontraction during constant velocity testing on an isokinetic dynamometer was that of Nelson et al (1973). In 15 "normal young people" they measured the EMG activity of m. tibialis anterior and m. soleus during maximal dorsiflexion and plantarflexion at 6 constant velocities from 24°/s to 216°/s on a Cybex I dynamometer. Greater agonist EMG activity was seen in the m. tibialis anterior than m. soleus, but antagonist activity was low and similar for both muscles. Antagonist activity was reported to be inversely proportional to velocity, with decreases of 26% and 46% of the agonist:antagonist ratios for m. tibialis anterior and m. soleus respectively, in the constant velocity range of 24°/s

to 216°/s.

However, there were several important weaknesses in that study. For example, it was not clear whether maximal or submaximal effort contractions were used, no details of the EMG equipment or signal conditioning were given, the EMG was not normalised, tests were performed over two sessions but no mention is made of measurement of inter-session reliability, and there were inconsistencies between the reported data and the text.

2.5.2 Elbow

Solomonow et al (1988) studied the electromyographic cocontraction patterns of the elbow antagonist muscles during slow constant velocity movement. Maximum effort elbow flexion and extension were studied in 22 healthy, normal individuals (12 males, 10 females) using a Cybex II® dynamometer. The arm was positioned to eliminate any reaction forces due to gravity. Antagonist EMG was normalised to angle specific agonist EMG. The absence of cross-talk was confirmed by calculating the coherence of the two signals throughout each trial. The pattern of antagonist EMG was thought to be more likely related to the varying moment-arm joint angle interaction than to muscle spindle activity, suggesting that pre-programmed neural circuits rather than simple random afferent flow is involved.

A surprising finding was that antagonist cocontraction in females was significantly higher than in males. However, the two groups were dissimilar in terms of how they usually used their upper limbs. The females were university students who reported no notable physical arm activities, whereas the males were skilled

individuals who used their upper limbs in regular highly skilled activities (e.g. orthodontists, orthopaedic surgeons, engineers, and athletes). The authors argued therefore that the level of physical activity and upper limb training history rather than sex of a subject that produced the significant differences in antagonist cocontraction.

2.5.3 Knee

Considering the voluminous literature on "isokinetics", and the clinical importance given to torque-angle data as objective information on muscle and joint performance, relatively little attention has been given to the activity of muscles under constant velocity, maximal voluntary joint loading. Including the first two independent reports (Eloranta and Komi, 1980; Knutsson and Martensson, 1980), reports in the literature on cocontraction of knee muscles during constant velocity testing are few (Osternig et al, 1984 & 1986; Knutsson and Martensson, 1985; Knutsson, 1987; Solomonow et al, 1987; Baratta et al, 1988). Subjects studied have included normal healthy individuals (Eloranta and Komi, 1980; Solomonow et al, 1987; Baratta et al, 1988), athletes (Osternig et al, 1986; Baratta et al, 1988), patients with ACL insufficiency (Solomonow et al, 1987), and patients with spastic (Knutsson and Martensson, 1980; Knutsson, 1987) or hysterical (Knutsson and Martensson, 1985) paresis. One other study reported using five adult male subjects but their physical or medical condition was not described (Osternig et al, 1984).

2.5.3.1 Normal Subjects

Eloranta and Komi (1980) used a unique dynamometer design with the force

transducer embedded in a footplate. This allowed force to be transmitted through the feet, and to be measured while both legs extended and flexed at the hip and knee, with the subject in a long sitting position. Such an instrument allows the measurement of linear velocities, but the device also requires simultaneous movement at both hip and knee joints. Since the hamstring muscle group crosses both the hip and knee joint, relating its activity to only the knee joint is difficult. In any case, such activity is probably different from that found when using the more common clinical dynamometers.

However, the activation patterns of the quadriceps and hamstring muscle groups during maximum effort quadriceps AGms and AGml were tested at eight different linear velocities in six male volunteers. Antagonist hamstrings activity was found to be angle dependent during AGms (i.e. antagonist cocontraction activity increased as the legs extended). This was not the case during AGml, when the antagonist hamstrings cocontraction returned to relatively low levels of EMG activity. Antagonist hamstring cocontraction was also reported to be velocity dependent during AGms between constant linear velocities of 0.12 to 0.97 m/s. This coactivation was again generally low, but the EMG activity in m. semimembranosus increased linearly with increased movement speed. However, no velocity dependence was seen during AGml.

The force-velocity curve depicting the agonist quadriceps data showed that peak force decreased with increasing velocity during AGms, but did not increase during AGml as expected, based on known muscle mechanics (Carlson and Wilkie,

1974) and previous human studies (Bigland and Lippold, 1954; Komi, 1973). The overall EMG activities of the agonists m. vastus medialis and m. vastus lateralis were higher during AGms than AGml. Under both contraction conditions, EMG activity was highest in mid-range and lowest at both extremes of range. While there was no velocity dependence to the agonist quadriceps activity during AGml, the was a trend towards increased EMG levels with greater velocities during AGms (Eloranta and Komi, 1980).

Osternig et al (1984), studied antagonist cocontractions of the knee flexors and extensors during AGms, using a modified Orthotron® (Cybex Division of Lumex, NY) which is commonly used in clinical facilities. Five young adult male subjects performed maximal alternate knee flexion and extension through an approximate range of motion of 115°, at four angular velocities (100°/s to 400°/s), while EMG activity over the m. vastus lateralis and m. biceps femoris were recorded. It was reasoned that if antagonist cocontraction was present, its probable purpose would be to decelerate the limb towards the end of the range of movement. Therefore, Osternig et al (1984) compared the average antagonist EMG activity for the first 100° of movement (the early phase), to the last 25° of movement (the late phase).

It was found that the overall antagonist cocontraction (defined as the %maximum activity of the same muscle group during its muscle shortening agonist phase) over all conditions was low, but hamstrings were considerably more active during knee extension than the quadriceps were during knee flexion (3.6% for quadriceps versus 14.9% for hamstrings). Antagonist cocontraction was not velocity

dependent, nor was there a difference between the early phase and late phase in either hamstring or quadriceps antagonist cocontraction. This appeared to refute a braking action by antagonist muscles at the end of either flexion or extension. It was concluded that other factors such as gravity and inertia of the limb/lever system were probably the major factors responsible for limb deceleration under these conditions.

Solomonow et al (1987) studied six normal healthy young men using a Cybex II® dynamometer. EMG was sampled over the mid-line of the quadriceps and mid-line of the hamstring muscle groups while maximal effort knee flexion or extension was performed through a range of 90° at a constant velocity of 15°/s. This was considered to be a quasi-isometric test condition. Levels of antagonist cocontraction were defined as the percent of the muscle's maximal value during its agonist phase for all subjects (i.e. %Max). With the exception of a "short initial peak" as the extension movement began, antagonist hamstring cocontraction remained steady throughout the test range of motion at 7%Max (±1.5%). Antagonist quadriceps cocontraction remained at 6%Max (±2%) during knee flexion.

Baratta et al (1988), used a similar protocol to Solomonow et al (1987), and investigated antagonist cocontractions during AGms in four young healthy males and females. However, in that study, tests were performed with the subjects in a side lying position to eliminate the effects of the gravity vector. As well, in the EMG analysis, the mean absolute value (MAV) of the EMG was normalized at each joint

angle with respect to its MAV at the same angle when acting as an agonist during maximal effort. This technique eliminated the possible dependence of the EMG on changes in muscle length (joint angle) and contraction rate changes during constant velocity loading over the test range of motion (Heckathorne and Childress, 1981; Perry and Bekey, 1981). In contrast to the previous study (Solomonow et al, 1987), antagonist cocontraction did vary significantly with joint angle, and relatively larger variations were seen in the knee flexors than the knee extensors. They reasoned that joint angle dependence was inversely related to the muscle moment-arm variations over the test range of motion (using muscle moment-arm values reported by Haxton, 1945a & 1945b; Kaufer, 1971; Smidt, 1973). When that relationship was taken into account, it was concluded that in normal subjects, antagonists exerted nearly constant opposing torque throughout joint range of motion, and therefore did not have a primary joint restraining function as suggested by Osternig et al (1986). In fact, it was concluded that the results supported the view that antagonist coactivation was necessary to 1) aid the ligaments in maintaining joint stability; 2) equalise the articular surface pressure distribution; and 3) regulate the joint's mechanical impedance. They also found that these patterns of cocontraction were different in certain athletic subjects (see section 2.5.3.2).

The only other report found concerning antagonist cocontraction in normal subjects was that of Knutsson and Martensson (1980) who briefly stated that they did not find cocontractions exceeding 100μV in their 12 healthy male and female volunteers under constant velocity testing (see section 2.5.3.3 for details of the main

part of the study). Knutsson and Martensson (1980) concluded that cocontraction of antagonist muscle in normal subjects was infrequent and insignificant.

2.5.3.2 Athletes

Osternig et al (1986) studied nine female track athletes (four sprinters, five distance runners) using a protocol reported earlier (Osternig et al, 1984). The antagonist integrated EMG (iEMG) data was computed as a percentage of the mean iEMG of the same muscle group during its maximal agonist contraction test (Knutsson and Martensson, 1980). Hamstrings were found to be considerably more active as antagonists (mean 33%Max) than were quadriceps (mean 6%Max). In addition, antagonist hamstring activity was angle dependent, with cocontraction increasing up to 58% in the last quarter of knee extension. Osternig et al (1986) also found differences in cocontraction patterns between sprinters and distance runners. Sprinters were found to have much higher hamstring cocontraction levels than distance runners (57% versus 14% respectively). However, at the fastest velocity tested (400°/s), distance runners increased their level of cocontraction at a greater rate than sprinters, during the final quarter of the test range of motion.

Osternig et al (1986), concluded that the hamstrings were used to a much greater extent that the quadriceps to decelerate the limb. Based on these conclusions, it was implied that this cocontraction was undesirable, in that such high levels of cocontraction in the hamstring muscle group would adversely affect knee extension constant velocity torque values, and possibly induce hamstring soreness or strain in vulnerable individuals. Curiously, there was no discussion of

the contradictory findings between this study and their previous one (Osternig et al, 1984) concerning the role of antagonist cocontraction during limb deceleration.

Based on a previous study by Solomonow et al. (1987) which suggested that the function of hamstring cocontraction was that of a dynamic stabilizer of the knee, cocontraction of the knee flexors and extensors was studied in athletes by Baratta et al (1988). Subjects were sub-divided according to whether or not they used hamstring curls in their individual training programs. Athletes who regularly exercised their hamstrings were found to have a cocontraction pattern similar to that observed in normal subjects, whereas the athletes who concentrated on quadriceps hypertrophy demonstrated very low hamstring cocontraction, similar to that of quadriceps. Two athletes who initially had low hamstring antagonist cocontraction were then given a three week daily hamstring exercise program, during which a gradual increase towards "normal" cocontraction levels was seen. Based on these findings, it was concluded that 1) cocontraction of the muscle which is antagonist to hypertrophied quadriceps is significantly inhibited, increasing the risk of joint injury, and 2) antagonist hamstring coactivation can be modified with training.

2.5.3.3 Patients

2.5.3.3.1 Spastic Paresis

Dynamometers have also been used to study antagonist cocontraction under constant velocity conditions in some patient populations (Knutsson and Martensson, 1980; Knutsson, 1983; Knutsson and Martensson, 1985; Knutsson, 1987;

Solomonow et al, 1987). In one study, Knutsson and Martensson (1980) reported significant cocontractions in both antagonist hamstrings and quadriceps muscle groups during constant velocity muscle shortening at 30°/s, 90°/s, and 180°/s in 24 subjects with spastic paresis. EMG levels of antagonist cocontraction of both the knee flexors and extensors increased with increasing constant velocity from 30 to 180°/s. To quantify the cocontractions, a "coactivation index" was used to compare the relative levels of cocontraction at different velocities. That index was the ratio between the mean EMG activity (sampled in mid range) of the muscle as an antagonist, and its mean activity as an agonist at maximal effort.

For the quadriceps muscle group, the coactivation index increased from 0.25 at 30°/s, to 0.46 at 90°/s, to 0.62 at 180°/s. For the hamstrings muscle group, the coactivation index was 0.32, 0.39, and 0.64 at 30°/s, 90°/s, and 180°/s respectively. No such relationship was seen in normal subjects (see section 2.5.3.1). At the faster velocities, Knutsson and Martensson (1980) reported that the cocontractions were sometimes so large as to significantly diminish the recorded resultant torque values. It was also concluded that the antagonist coactivation of both muscle groups was similar in magnitude, that the mean antagonist restraint was relatively larger in flexion movements than in extension movements as knee extensors are stronger than knee flexors. In seven limbs, they estimated this quadriceps antagonist restraint to be 70 N·m, which equalled the average torque generated by the knee flexors in healthy subjects. This led Knutsson and Martensson (1980) to conclude that, in the presence of such marked quadriceps

antagonist cocontraction, the paretic patient was incapable of flexing the leg at fast speed even when the force in the knee flexors was well preserved.

In a subsequent study, Knutsson (1987) confirmed the velocity dependence of antagonist cocontraction in spastic paresis during maximal effort AGms. The same effect (although to a lesser degree) was also observed during passive constant velocity movements (e.g. cocontraction of hamstrings while the leg was passively extended at constant velocity). From the relative change in antagonist EMG activity Knutsson reported that spastic restraint is two to four times larger in maximal effort voluntary movement compared to passive movement under constant velocity conditions even though stretch of the antagonist muscles was precisely the same under both conditions. This finding, together with the observed decrease in antagonist activity during cooling (compared to increased agonist activity), pointed to two different motor control mechanisms which might contribute to impaired movement in spastic paresis. However, there were exceptions to this relationship, so Knutsson cautioned against the use of passive movements alone, or examination of spastic restraint at rest, to predict the restraint of spastic muscles under active conditions.

Knutsson (1987) also reported on the effect of contraction type (i.e. muscle shortening versus muscle lengthening) on agonist and antagonist activation in spastic paresis. A KIN/COM™ dynamometer was used to study maximal effort voluntary knee extensor contraction at a constant velocity of 180°/s. It was found that agonist activity was greater during AGml than AGms while antagonist

cocontraction was less during AGml. The resultant knee extension torque increased dramatically from minimal torque during AGms to torque values in the normal range during AGml. This suggested that antagonist stretch reflexes can significantly affect both antagonist activity and the ability of the agonist to be activated, and therefore can profoundly influence the resultant torque output.

2.5.3.3.2 Hysterical Paresis

Excessive antagonist quadriceps activity has been seen in patients with hysterical paresis (Knutsson and Martensson, 1985). Using a Cybex II® dynamometer to test muscle performance at constant velocities of 30/90/120 and 180°/s while monitoring the EMG of the knee flexors and extensors, some subjects were found to produce a negative knee flexion torque, suggesting that the agonist hamstring muscle force was less than the gravitational force. However, the EMG recording showed that it was due to antagonist quadriceps cocontraction. This led Knutsson and Martensson to suggest that this kind of testing can by used to recognise inconsistent and contradictory motor performance that is not compatible with a genuine paresis, and therefore could help in the identification of feigned weakness.

2.5.3.3.3 Anterior Cruciate Ligament Deficiency

The role of the hamstring muscle group as a joint stabilizer in the ACL deficient knee has been reported (Solomonow et al, 1987). Using the protocol described earlier (section 2.5.3.1) the torque and EMG activity of the quadriceps and hamstrings were monitored in a group of 12 adult patients with ACL deficiency.

Solomonow et al (1987) reported that anterior subluxation of the tibia occurred at an angle of 37° to 46° of flexion, during maximal effort voluntary knee extension performed at a constant velocity of 15°/s on a Cybex II® dynamometer. In patients who had not received any muscle exercise rehabilitation, or in patients with poor muscle tone and bulk, this resulted in what was described as a "large torque failure". At the same time as the sudden decrease in resultant torque output at 37° to 46°, a simultaneous decrease in quadriceps EMG and increase in hamstrings EMG was observed, suggesting the existence of a ligament-muscle reflex arc. The existence of this reflex arc was confirmed in anaesthetized cats by applying various traction forces to the ACL and recording the EMG response of the hamstrings muscle group. The confirmation of a reflex arc between the ACL and hamstrings suggests that knee joint stability in extension is not an exclusive function of the ligaments. The hamstring muscles can therefore be viewed as dynamic stabilisers or torque regulators of the joint, which are activated "on demand" during ligament overloading. Solomonow et al (1987) concluded that hamstring muscle strengthening has significant potential for improving knee stability in patients with ACL deficiency. However, Solomonow's conclusions have recently been questioned (Pope et al, 1990).

2.5.3.4 Summary

In spite of the strong points of these reviewed studies, there are major criticisms which detract from their value and prompt the need for the present study. There is also a need to simulate a clinical testing regimen in order to determine the

relevance of antagonist cocontraction under such conditions. Such knowledge will result in a better understanding of torque-angle curves and their interpretation, and may necessitate the need to modify clinical test protocols accordingly.

When comparing and interpreting the significance of the results and conclusions of the above studies, it is important to take into account any differences regarding methodology, equipment, controls, subjects, and statistical analysis. For example, the constant velocity dynamometer used in the Eloranta and Komi study (1980) was a unique design, allowing simultaneous movement at both the hip and knee joints. All the other studies used clinical equipment such as the Orthotron (Osternig et al, 1984,1986), the Cybex II® (Baratta et al, 1988; Knutsson and Martensson, 1980; Knutsson, 1983; Solomonow et al, 1987 & 1988), and the KIN/COM™ (Knutsson and Martensson, 1985; Knutsson, 1987) which only allowed movement at the knee joint during tests. Since the validity and reliability of the Cybex II® has been questioned (Gransberg and Knutsson, 1983; Murray, 1986; Murray and Harrison, 1986), and the ability to reach a constant velocity at the faster velocities used in the Orthotron studies is in doubt (based on the results of the present pilot study, section 3.3), the above results are brought into question.

While the EMG equipment and processing were generally well described in these previous reports, details of how the EMG data were analyzed were lacking in many studies. The importance of measuring cross-talk was only discussed and controlled for by the Baratta/Solomonow group. Since monitoring for cross-talk is a critical step before meaningful interpretation of this kind of EMG data can be

carried out (Solomonow et al, 1988; Baratta et al, 1988), the conclusions of most of the studies discussed are open to challenge.

While Knutsson has reported interesting results in cocontraction in spastic paresis, it would seem useful to be able to compare such activity to that in normal subjects. Knutsson considers normal cocontraction under constant velocity conditions to be small and insignificant. However, while some support that idea (Nelson et al, 1973; Osternig et al, 1984), others argue that normal cocontraction can still be clinically significant (Osternig et al, 1986; Solomonow et al, 1987; Baratta et al, 1988; Draganich et al, 1989). Also, the level of cocontraction may be changed by adopting certain training methods (Osternig et al, 1986; Baratta et al, 1988).

The overall level of antagonist cocontraction in normal subjects during constant velocity AGms has varied in reports between 3.6% to 6% for quadriceps and 7% to 14.9% for hamstrings (Osternig et al, 1984; Solomonow et al, 1987). In the hamstrings at least, this level of cocontraction may increase significantly (Eloranta and Komi, 1980; Osternig et al, 1986) or remain constant (Osternig et al, 1984) as the knee extends. This increase may (Eloranta and Komi, 1980; Osternig et al, 1986) or may not (Osternig et al, 1984) be velocity dependent.

The effect of contraction type (i.e. muscle shortening vs muscle lengthening) on cocontraction has been the least studied in patients (Knutsson, 1987) and has not been tested at all in normal subjects on a clinical dynamometer. If hamstring cocontraction is a function of the muscle stretch reflex as suggested by Knutsson, then it would be expected to decrease during quadriceps AGml. However, if the

function of hamstring cocontraction is to provide stability to the knee, it might be expected to increase during AGml to compensate for the increased torque produced at the joint by such a muscle contraction. The former mechanism would seem to be supported by Eloranta and Komi (1980), but their study did not isolate the movement to one joint, and the EMG analysis did not control for muscle activity dependence on muscle length or contraction rate changes.

In considering the conflicting results and questionable technique used in some studies, it is desirable to correct some of the weaknesses and omissions, and establish more reliable normative data using a clinical dynamometer. Gathered under more typical clinical conditions, these data would better describe normal antagonist cocontraction under controlled angle, velocity and contraction type conditions, and enlarge our understanding of normal motor control at the knee joint. Valid secondary evaluation of abnormal clinical conditions could then be made accurate and meaningful in comparison to these norms of joint function.

3.0 MATERIALS AND METHODS

3.1 Purpose

The purpose of this study was two-fold: first, to investigate, using surface electrodes, the electrical activity of antagonist quadriceps and hamstring muscle groups during maximal effort constant velocity AGms and AGml using a typical clinical test protocol. This would establish normal baseline data for subsequent studies of males and females with various orthopaedic and neurological conditions affecting the knee joint and surrounding musculature. Second, the present study was designed to determine the relative effects of joint angle, limb velocity, and contraction type on the level of antagonist activity using the above test protocol.

3.2 Experimental Paradigm

After a familiarisation period with the equipment and a warm-up of sub-maximal and maximal AGms and AGml repetitions, each subject performed four maximal effort dynamic tests of the knee flexors and extensors through a defined range of motion under two constant velocity conditions in a single session. Each test consisted of four repetitions. A repetition was defined as an AGms phase followed by an AGml phase, through a preset range of motion of 65°. Two tests were performed with the left knee extensors as agonists, and two tests were performed with the left knee flexors as agonists. For each muscle group, one test was performed at 30°/s, and another at 90°/s. To control for possible order effects, the sequence of muscle group testing (extensors/flexors or flexors/extensors) and velocity (30/90 or 90/30) were randomized in a balanced design. While the tests

were performed, the electromyographic activity of the knee flexors and extensors was recorded simultaneously with KIN/COM™ force and angle data on a chart recorder. KIN/COM™ force, angle, and velocity data were also collected by the machine's data acquisition system.

Before the study began, the test protocol was approved by the University of Manitoba Faculty Committee on the Use of Human Subjects in Research (Appendix A).

3.3 Pilot Study

A total of 5 subjects were used in a pilot study to:

1. Determine optimal electrode placement sites for measuring EMG activity of the hamstring and quadriceps muscle groups.

Placements from other studies were used for detecting the electrical activity of the hamstrings muscles as a group and the m. vastus lateralis as representative of the quadriceps muscle group. The electrode placements are described in section 3.6.2. and shown in figure 3.1A&B.

The electrode sites were chosen for optimal sensitivity to hamstring muscle activity while minimizing any possibility of cross-talk from other muscle groups. In the present study, considerable attention was given to the elimination of cross-talk. Two methods of demonstrating absence of cross-talk were employed in the pre-test procedures (Gottlieb et al, 1982; Basmajian and DeLuca, 1985; Flanders and Cordo, 1987; Solomonow, 1989). First, to detect any gross cross-talk, the agonist and antagonist EMG were monitored on a 2-channel oscilloscope during reciprocal

contractions. Subjects were asked to slowly extend and flex their leg while positioned for testing on the KIN/COM™ dynamometer. While they pushed and pulled against the shin pad on the moment-arm, the machine offered moderate resistance to the movement, and the agonist and antagonist EMG activity were monitored on the oscilloscope. Antagonist EMG recordings were considered valid if no simultaneous agonist/antagonist activity could be seen on the tracings (figure 3.2A-B).

Second, in a more sensitive check, the phase of the two signals from the agonist and antagonist muscle pairs was monitored using an oscilloscope according to Solomonow (1989). The agonist EMG signal was input to the x-axis of the oscilloscope and the antagonist EMG signal was input to the y-axis. Randomly changing phase of the signals in all quadrants of the oscilloscope would indicate no correlation between agonist and antagonist EMG (see figure 3.2C-D). Alternatively, a fixed phase (combined signals in less than 4 quadrants) would indicate cross-talk was present (Baratta et al, 1988). This latter cross-talk check was considered to be more sensitive than the former method of observing the raw EMG signals, and was therefore the method chosen for the main study.

2. Establish test velocities to be used.

Typical velocities used for clinical testing range from 30°/s to 300°/s. However, as faster velocities are used, more of the test range of motion is needed to accelerate and decelerate the limb. Consequently the range available for constant velocity is decreased (Osternig et al, 1983). Since the objective of the study was to

investigate the effects of angle, velocity, and contraction type during isokinetic muscle testing might have on antagonist EMG activity, it was necessary to determine the relationship between a selected velocity and constant velocity angular range within a preset range of motion. Therefore, the left knee flexors and extensors of 5 subjects were tested using the KIN/COM™ test protocol described below. Seven different angular velocities (30, 60, 90, 120, 150, 180, and 210°/s) were tested. A digital printout was obtained from the KIN/COM™, which listed the velocity and angle of the moment-arm for every 10 ms of the movement duration. From this information, the angle change required for acceleration at the start of the movement and that required for deceleration at the end of the movement could be determined. The constant velocity phase of the movement was therefore delineated between the acceleration and deceleration phases.

As can be seen from figure 3.3 the constant velocity range during muscle shortening decreased gradually from 60° - 62° at 30°/s, to 38° - 42° at 210°/s, with small variation (±2°) between subjects. However, the constant velocity range during muscle lengthening was much more variable between subjects and decreased much more quickly from 58° - 60° at 30°/s to 0° - 32° at 210°/s (figure 3.3). In fact, one subject could not generate constant velocity at all during muscle lengthening at 120°/s or above (figure 3.3). It was therefore decided that in order to generate a useful constant velocity range in all subjects, the highest velocity to be used in the study would be 90°/s. Similarly, in order to have the maximum possible separation between chosen fast and slow velocities within typical clinical

test ranges, and still address the question of the velocity dependence of cocontraction, the lower velocity to be used would be 30°/s, which has been used in many previous muscle performance studies (Thorstensson et al, 1976; Goslin and Charteris, 1979; Murray et al, 1980 and 1985; Richards, 1981; Sale and MacDougall, 1984; Falkel et al, 1985; Knutsson and Martensson, 1985; Lankhorst et al, 1985; Danneskiold-Samsoe and Grimby, 1986a and 1986b; Lennox et al, 1986; Weltman et al, 1986 and 1988; Agre and Baxter, 1987; Jacobsen and Danneskiold-Samsoe, 1987; Knutsson, 1987; Johansson et al, 1989).

3. Determination of the angle resolvable on the chart recording of EMG.

Three subjects were studied using the test protocols derived for velocity and electrode placement while agonist and antagonist EMG was recorded on a Honeywell chart recorder. From the trials, it was decided that a 50 mm/s chart speed for a 30°/s test and a 125 mm/s chart speed for a 90°/s test produced a recording which would allow determination of EMG activity resolved at 5° and 10° intervals. Subsequent measurement and plotting of the EMG for 5° and 10° intervals showed that measuring EMG activity every 10° of movement was not as representative of the actual recording as measuring every 5° (figure 3.4A&B). 5° was therefore chosen as the EMG measurement resolution. Previous reports have used resolutions from as large as 25° (Osternig et al, 1986), to as small as 1° when computer data capture and analysis was used (Baratta et al, 1988). While accuracy is important, the number of intervals were balanced by practicality, time, clinical significance, and statistical usefulness of such information.

3.4 Subjects

Healthy male and female volunteer subjects between the ages of 21 and 31 who had no history of knee injury or pathology and who were moderately active in a range of recreational activities, but were not engaged in any regular training program, were eligible for admission into the study. Twenty-four male and female volunteers were sought from staff of the Department of Rehabilitation Therapies, St. Boniface General Hospital, and students from the Faculty of Medicine and School of Medical Rehabilitation, University of Manitoba. Assurance of good health was documented by the administration of an interview questionnaire (Appendix B) designed to screen out subjects with cardiovascular, neurological, or orthopaedic medical problems, and specifically, any pathology related to the left lower limb. Following satisfactory completion of the questionnaire, informed consent was obtained from each subject (Appendix C). Study subjects were then grouped in a balanced design to cover two velocities (30°/s and 90°/s) and two exercise types (agonist hamstrings and agonist quadriceps) during a single session.

3.5 Equipment

3.5.1 Muscle Testing Equipment

The muscle tests were performed on a Kinetic Communicator (KIN/COM™) dynamometer details of which are described in section 2.2.3.3)

3.5.2 EMG Equipment

The EMG equipment employed in this study has been reported previously (Peat and Grahame, 1977a & 1977b; Tata and Grahame, 1978 & 1979; Tata, 1980)

and consisted of signal amplifiers which were developed, designed, constructed, adjusted and modified, in the electronics shop of the Bioengineering Department of the Rehabilitation Centre for Children, Winnipeg. The input impedance was 20 megohms and the common mode impedance 10 megohms. The band width was from 20 Hz to in excess of 5 kHz, and the common mode rejection ratio extended from 80 to 100 dB at 60 Hz. For the two channels used for EMG in this study, the signals were processed by means of full wave rectification and a first order low pass filter with a 3 dB cut off frequency of 10 Hz. This combination produces a linear envelope which is the most suitable method for demonstrating peak activity because the amplitude of the envelope closely follows the amplitude of the peaks of the raw EMG, as opposed to an integrated signal which is a measure of the product of the EMG amplitude and time (Tata, 1980).

A series 2500, eight channel Honeywell pen recorder (model 1500) was used for all recordings. The signals were attenuated by a Honeywell Accudata 125 attenuator which has nine ranges from 0.5 to 200 V/cm.

The input into the amplifiers was from bipolar silver/silver chloride commercial surface electrodes (Beckman) via shielded cables. The overall size of each electrode was 16 mm diameter with an active electrode size of 9 mm. The electrodes were filled with a saline contact paste (Redux Cream, Hewlett-Packard, Waltham, MA) to increase the conductivity and reduce electrode/skin resistance.

3.6 Procedures

3.6.1 Muscle Test Procedure

3.6.1.1 Pre-Warm up

Each subject was positioned on the dynamometer in the sitting position, leaning against a backrest. The backrest was placed in its most upright position which was inclined 16° from vertical, and the backrest formed an angle of 100° to the seat. The axis of each subject's left knee was aligned with the axis of the dynamometer's moment-arm by visual inspection and with the aid of the shin pad on the moment-arm acting as a marker approximately 2 cm above the medial malleolus of the tibia. The alignment was checked by allowing the subject to extend the leg while pushing against the shin pad. If the pad did not move up or down the leg over the test range of motion, the knee axis was considered to be aligned with the axis of the moment-arm.

It has been reported that only minimal stabilization (by the subject grasping the sides of the testing table) is required for reliable torque measurements to be made during AGms and AGml constant velocity testing (Hanten and Ramberg, 1988). However in this study, active stabilization (Appendix E) was combined with passive stabilization, which included one strap fastened over the lower half of the thigh and another around the subject's waist to prevent unwanted body movement and minimise the possibility of shifts in the axial alignment (figure 3.5A).

In order for the KIN/COM™ computer to calculate torque, the radius of rotation (the distance between the point of application of the generated force and

the axis of rotation of the moment-arm) was measured to the nearest 5 mm using the moment-arm scale, and entered into each subject's data file. The radius was kept constant for the four tests of each subject.

The test leg of each subject was then weighed using the dynamometer's force transducer. This was done by positioning the moment-arm 30° down from the horizontal position, and the subject's left leg was placed at rest on top of the shin pad. The KIN/COM™ computer then recorded the resting weight measurement from the force transducer. To ensure an accurate measurement, the weight of the leg measured at rest was confirmed by monitoring the EMG signals from the knee flexors and extensors. Complete relaxation of the knee musculature (i.e. no EMG activity), and easy passive medial/lateral deflections of the patella were confirmed prior to recording leg weight. Once this was established for each subject, all subsequent force measurements were automatically adjusted for the calculated effect of gravity at each angle in the test range. To ensure that all torque measurements were accurate, the KIN/COM™ load cell calibration was checked at the start of the study and periodically thereafter (Appendix D).

The dynamometer's exercise arm was used to set the test range of knee motion, previously determined to be 65°. This included the range from 10° to 75° down from horizontal with reference to the moment-arm (figure 3.5B&C). Therefore, for the knee flexion tests, the angle at which muscle shortening began (i.e. START ANGLE) was 10° down from horizontal; and the angle at which muscle lengthening began (i.e. RETURN ANGLE) was 75° down from horizontal. For the

knee extension tests, START ANGLE was 75° and RETURN ANGLE was 10°.

Moment-arm velocity was set to either 30°/s or 90°/s depending on the test to be performed. The accuracy of the velocity was checked by reading the printout of the KIN/COM™ torque report which included the velocity of the moment-arm every 10 ms during a test. At both 30°/s and 90°/s, the coefficient of variation was 3%.

Computer control was also set so that the moment-arm would not move unless a minimum force of 20 N (for the knee extensors) or 50 N (for the knee flexors) was applied to the force transducer. In the KIN/COM™ protocol, this rules out spurious passive force transmission to the moment-arm.

A pause of 100 ms was set between the AGms and AGml phases of each repetition in order to clearly delineate the two phases.

Before the initial warm-up, subjects were asked to perform slow submaximal muscle shortening and lengthening contractions of the left knee flexors and extensors while the leg was attached to the moment-arm but without the dynamometer under computer control. In this mode, the dynamometer offered moderate resistance, and the reciprocal EMG activity of the knee flexors and extensors could be monitored for cross-talk. If cross-talk was present, adjustments of electrode position were made. This was followed by maximal isometric contractions to set the chart attenuator gains (see section 3.6.2), and oscilloscope checks for cross-talk were repeated. None of the recordings indicated fixed phase was present, confirming the independence of the two EMG signals. Typical

recordings confirming absence of cross-talk during both isometric and constant velocity tests are shown in figure 3.6A-D.

A passive constant velocity test was then performed at 30°/s through the test range in order to confirm that no EMG activity occurred in the knee flexors or extensors due to passive stretch. For this procedure, the subject was asked to completely relax while the dynamometer moved the leg through four repetitions of the test range. Following the passive test, warm-up trials were performed.

3.6.1.2 Warm-up

To minimize the risk of muscle injury and to ensure optimal test performance, submaximal and maximal warm-up and familiarization trials were completed by each subject before every test. The first warm-up was designed to familiarise the subject with the "feeling" of the constant velocity test, which can be confusing. The subject was told to push his/her leg against the shin pad and feel that the harder they pushed, the stronger the KIN/COM™ resisted the movement. When the leg reached the return angle, they felt how the dynamometer overcame their resistance, and forced the leg back to the start angle. By repeating this cycle several times the subjects became accustomed to the test movement. After any four repetitions, a two minute rest period was strictly enforced to minimise muscle fatigue.

Submaximal trials were repeated until the subject could produce four smooth and uninterrupted repetitions at the set velocity. Once this was achieved, the subsequent trial involved producing progressively stronger contractions such that the

final repetition involved a maximum dynamic voluntary contraction. If this was completed satisfactorily, the subsequent two minute rest was then followed by the actual test.

3.6.1.3 Muscle Test

Prior to each test, each subject was given precise instructions on active stabilisation, unwanted body movements and the effort required during the test (Appendix E).

Subjects then performed four maximal effort muscle shortening and lengthening repetitions, with verbal encouragement from the tester. After a two minute rest, a warm-up for the next test was performed and the test cycle repeated until all four tests were completed.

3.6.2 Electromyographic Procedure

M. vastus lateralis was chosen as representative of the quadriceps muscle group. One pair of electrodes was applied over the m. vastus lateralis of the left thigh as shown in figure 3.1A. They were positioned to maximise the m. vastus lateralis signal while minimising signal detection from m. rectus femoris, m. vastus medialis, and m. vastus intermedius as predicted from normal human anatomy (Moore, 1985).

The second pair of electrodes was applied over the left hamstrings muscle group along their mid-line as shown in figure 3.1B, to ensure a representative EMG from all flexors. All measurements were made with an anthropometric tape measure and the inter-electrode distance was 4cm for each pair of electrodes. Also, a

reference electrode was attached over an area of relative inactivity, the proximal antero-medial surface of the tibia.

Skin resistance is not a critical factor when using amplifiers with high input impedance (i.e. > 7 Mohm) (Quanbury, 1989). However, care was taken to ensure that hair was removed if necessary (with a safety razor), and that the skin was cleaned of oils and lightly abraded by rubbing with alcohol swabs in order to provide consistent electrode contact with the skin surface.

The electrodes were filled with the contact paste and attached to the skin with double-sided adhesive collars to restrict relative motion between the electrodes and the skin, and to provide a seal which prevents the paste from drying out (Quanbury 1972). Adhesive tape was applied over all the electrodes to further ensure good electrode/skin contact and decrease the chance of movement artifact.

Before each test series, the chart recording of the EMG amplifier outputs were calibrated to a 1 mV signal with the chart attenuator ranges set at 2 V/cm, 5 V/cm, and 10 V/cm. This allowed the subsequent measurement of the EMG recordings to be converted to actual mV values. Before each test, the chart attenuator ranges were set separately for agonist and antagonist while the subject performed maximal isometric voluntary contractions described above. Each attenuator range was set to achieve maximum amplification of the signal without saturation.

Channels 1 and 2 of the chart recorder were used to collect the EMG data (channel 1, agonist EMG; channel 2, antagonist EMG), and channels 5 and 6

recorded the analog force and angle signals respectively from the KIN/COM™ dynamometer. The EMG and KIN/COM™ signals were synchronised by the use of a START and STOP signal from the KIN/COM™ which was recorded on channel 4. This signal identified the point at which the subject's leg began to move the moment-arm and also signalled the end of the last repetition.

For tests at the slower velocity (30°/s), the chart speed was run at 50 mm/s. For the faster velocity (90°/s), the chart speed was increased to 125 mm/s so that a similar resolution of the EMG, force and angle signals could be attained. Samples of the strip chart recordings are shown in figure 3.7(A&B).

3.7 Analysis

3.7.1 EMG Data Collection and Analysis

As outlined above, collected data included the simultaneous recordings of the net resultant joint torque compensated for gravity, moment-arm angle in the set range, moment-arm velocity variation around the preset value, and the EMG activity of each muscle or muscle group under study. The torque, angle, and velocity data were stored on computer disk as well as in analogue form by the chart recorder in synchrony with the EMG data.

From the chart recording of the temporal sequence of events from the start of the first repetition, the peak amplitude EMG at every 5° to the end of the test was calculated. The calibrated chart speed and test velocity allowed the chart distance in mm between each 5° change in range of motion to be calculated.

Since only the muscle activity that occurred during the constant velocity

phases of muscle contraction was of interest, the activity during the acceleration and deceleration components of each respective AGms and AGml contraction were excluded from the analysis. At the slower velocity (30°/s), the first and last 5° of muscle shortening and muscle lengthening were excluded, leaving a net ROM of 55° (between 15° and 70° down from horizontal). At the faster velocity (90°/s) the first 10° and last 5° of AGms and the first 15° and last 5° of AGml were excluded as acceleration and deceleration phases, leaving a net ROM of 50° for AGms and 45° for AGml. This allowed EMG activity in each muscle group to be measured at exactly the same angles for each contraction type and velocity. Therefore the effects of velocity and contraction type could be evaluated in a paired manner while controlling for joint angle.

To improve the accuracy and precision of manually measuring 5° intervals (over 20,000 in this study) two templates were made (figure 3.7A&B): one for measuring 5° intervals on the 90°/s tests, and the other for measuring the same intervals on the 30°/s tests. The relevant template was aligned with each phase of the EMG tracing and a dot was placed on the chart with a fine fibre-tipped pen at 5° intervals. At each of these intervals, the distance from baseline to the peak EMG amplitude was measured to the nearest 0.5 mm and recorded on a data capture sheet. For each test and both muscle groups, the DC offset and the EMG calibration factor (to convert the chart measurement in mm to EMG activity in mV) were also recorded. These EMG data were then entered into a Lotus123 data file on a Mind XT PC for later analysis.

The EMG analysis was based on the method described by Baratta et al (1989). To quantify the antagonist muscle activity, the peak EMG amplitude was normalised at each joint angle with respect to its peak EMG amplitude at the same angle when that muscle group was acting as agonist at maximum effort. For example, the antagonist hamstring peak EMG amplitude at 60° during maximal effort knee extension at 30°/s was divided by its agonist peak EMG amplitude at 60° during maximal effort knee flexion at the same velocity. In this way, two variables known to influence muscle activation profiles, muscle length (Haffajee et al, 1972) and velocity (Bigland and Lippold, 1954), were controlled (Baratta et al, 1988). However, the present study had the additional variable of contraction type (muscle shortening and muscle lengthening) to consider. If Baratta's method of normalization was applied, it would not have been possible to compare ANTms EMG activity to ANTml EMG activity, or slow velocity activity to faster velocity activity, because antagonist activity would have been normalized to different agonist velocities and contraction types, (e.g. ANTms would have been normalized to AGms and ANTml to AGml). Since it was desirable to compare antagonist activity between contraction types and velocities as well as between joint angles, it was decided that for a given muscle group, the same normalisation factor would be used for both contraction types and velocities. Therefore, for this analysis, the antagonist peak EMG amplitude was normalised at each joint angle with respect to its peak EMG amplitude at the same angle when that muscle group was acting as agonist at maximum effort during muscle shortening at 30°/s.

3.7.2 Peak Torque and Demographic Data

A different Lotus123 file was set-up to store and analyze data from the KIN/COM™, and to calculate peak torques of the knee flexors and extensors. To allow direct comparison of torque values within the whole sample and between other studies, peak torque data were reported separately for males and females, and included means of absolute peak torque, and mean peak torque normalized for body weight (Table 4.2). The data collected on the health questionnaire and data form (Appendix B) including age, height, and weight were also compiled in the same file (Table 4.1).

3.7.3 Statistical Tests

The present design used comparison of the muscle antagonist EMG activity normalized to the %Max of its agonist activity in a paired fashion for two angular velocities or for two muscle contraction types. Application of a series of 2-way ANOVA split plot (repeated measures) tests enabled this comparison in order to (1) take advantage of the paired data available for each subject, and (2) strongly detect variation in the paired data without normalizing against an unnecessary parameter which would be presumed to be less variable between subjects. The NWA STATPAC software program version 4.1 was used to perform all statistical tests in consultation with a University of Manitoba statistician. A probability of $p<.05$ was used to reject the null hypothesis.

4.0 RESULTS

4.1 Demographics

A total of 24 healthy subjects were tested using the protocol described above. Collected data on sex, age, height, and weight are given in Table 4.1. In two subjects, antagonist hamstring activity exceeded the gain limits set in the amplifiers in some tests. The peak EMG amplitudes could therefore not be measured in these subjects. Therefore, in all cases antagonist hamstring data includes 22 subjects and the antagonist quadriceps data includes 24 subjects.

4.2 Torque data

The mean peak torque data separated by sex, muscle group, velocity, and contraction type, corrected and uncorrected for body weight, are reported in Table 4.2. The data were analyzed by 3-way ANOVA, and as expected, there was a significantly greater output of absolute peak torque generated by males than by females for AGms ($p < 0.001$) and AGml ($p < 0.001$). Absolute peak torque for females varied from 65% to 72% of that for males in all tests and contraction types (Table 4.2). When peak torque was normalized for body weight, the male:female torque ratio narrowed to 82% to 91% but remained significantly different ($p < 0.001$). In addition, AGml peak torque was significantly greater than AGms peak torque for both quadriceps ($p < 0.001$) and hamstrings ($p < 0.001$). Peak knee extension torque was significantly greater than peak knee flexion torque ($p < 0.001$) and significantly more torque was produced at 30°/s than at 90°/s during 8AGms ($p < 0.01$), but not during AGml ($p > 0.3$).

The corrected and uncorrected peak torques are comparable to those found in previous studies (Snow and Johnson, 1988a & 1988b; Snow and Blacklin, 1991) and to values reported by others (Hanten and Ramberg, 1988; Rizzardo et al, 1988; Kramer, 1990). Since the torque-angle data follow a typical force-velocity relationship, they are consistent with a maximal effort contraction by subjects.

4.3 EMG Data

Antagonist EMG activity was observed in all subjects in all tests and contraction types. However, much higher antagonist EMG activity was observed in the hamstrings (Table 4.3) than the quadriceps (Table 4.5), so the ranges of antagonist EMG activity are reported separately. Overall, mean antagonist hamstring activity ranged from 15.6 - 31.7 percent of maximum (%Max). Mean antagonist quadriceps activity ranged from 5.8%Max - 8.2%Max.

4.3.1 Effect on antagonist hamstring activity of contraction type

ANTml displayed quite different activity to ANTms at both fast and slow velocities (compare Tables 4.3 and 4.4; figures 4.1 and 4.2). At the slower velocity, overall ANTml activity was significantly higher than ANTms activity ($p = 0.003$).

There was also a significant ($p < 0.001$) interaction between angle and contraction type. EMG activity decreased with angle during extension (ANTml) whereas there was no change in activity with angle during flexion (ANTms). As depicted graphically (figure 4.1) for the constant velocity phase of the continuous sequence from full knee flexion to extension and back into full flexion, the following pattern of activity can be described. Antagonist hamstring activity was greatest

(approx. 30%Max) during the first 10° to 15° of AGms. Then the level of antagonist EMG activity declined rapidly and levelled off at about 17%Max during the last 20° to 25° of AGms. During AGml, the antagonist hamstring activity did not change significantly, and remained at about the same level (17%Max to 22%Max) throughout the AGml phase (figure 4.1).

At the faster velocity, the differences in antagonist EMG activity between the two types of contraction were essentially the same as at the slower velocity (compare figures 4.1 and 4.2). There was a significant ($p < 0.001$) difference between contraction types, in that antagonist hamstring EMG activity was greater during AGms than AGml. There was no significant effect of changes in angle (2-way ANOVA: Table 4.4). However, a significant ($p < 0.001$) interaction between angle and contraction type was shown which was similar to that seen at the slower velocity. This interaction can be observed as antagonist EMG activity remained relatively constant through the range of motion during AGml, while during AGms, antagonist EMG activity started high at 65°, but declined as angle decreased (to 25°) as seen in figure 4.2. Therefore, antagonist hamstring activity was greatest (24%Max to 28%Max) during the first 10° of constant velocity AGms and declined rapidly to about 18%Max for the last 10° to 15° of constant velocity AGms. During the subsequent AGml, the antagonist hamstring activity was relatively unchanged (17%Max to 22%Max) throughout the constant velocity range.

4.3.2 Effect on antagonist quadriceps activity of contraction type

In contrast to the hamstring antagonist activity, there was no significant

difference in antagonist quadriceps activity between contraction types at either velocity. Data are presented for the slow velocity in Table 4.5 and figure 4.3, and for the fast velocity in Table 4.6 and figure 4.4. There was no significant difference in the overall antagonist EMG activity during either AGms or AGml at either the slow ($p > 0.2$, Table 4.5) or fast ($p > 0.8$, Table 4.6) velocities. There was no significant effect of angle on antagonist quadriceps activity during AGms or AGml at either the slow ($p > 0.8$, Table 4.5) or fast ($p > 0.3$, Table 4.6) velocities. There was also no significant interaction between angle and contraction type at either velocity. These findings are also depicted graphically and when viewed in sequence, the following activity pattern can be described. During the constant velocity phase of AGms, antagonist quadriceps activity averaged 6 - 8%Max. During the subsequent AGml phase, antagonist quadriceps activity remained relatively unchanged (6 - 8%Max) (figures 4.3 and 4.4).

4.3.3 Effect on antagonist hamstring activity of moment-arm velocity

Antagonist hamstring activity during the constant velocity phases of movement was significantly ($p < 0.001$) higher at the fast velocity than at the slow velocity during AGms (Table 4.7 and figure 4.5). There was also a significant change ($p < 0.01$) in antagonist activity with change in angle during AGms (Table 4.7). There was no significant ($p > 0.9$) interaction between angle and velocity (Table 4.7). Early in the constant velocity phase of AGms, antagonist hamstring activity averaged 26%Max and 28%Max for the slow and fast velocities respectively and declined as the movement continued, to end at 17%Max and 19%Max

respectively (figure 4.6). The statistical significance of this difference is likely the result of consistently low variation between subjects and within groups.

During AGml, antagonist hamstrings activity, was again significantly ($p < 0.04$) higher at the fast velocity (Table 4.8 and figure 4.6), although the difference was less than that during AGms. There was no significant ($p > 0.8$) change in antagonist hamstrings activity with change in angle, and there was no significant ($p > 0.8$) interaction between angle and velocity during AGml (Table 4.8).

4.3.4 Effect on antagonist quadriceps activity of moment-arm velocity

During AGms, antagonist quadriceps activity was significantly ($p = 0.02$) greater at the fast velocity than at the slow velocity (Table 4.9 and figure 4.7), as observed for antagonist hamstrings. The difference was small (<2%Max) and its statistical significance was probably the result of low variability between subjects and within groups. However, there was no significant ($p > 0.1$) difference in antagonist quadriceps activity between the fast and slow velocities during AGml (Table 4.10 and figure 4.8).

There was no significant change in antagonist quadriceps activity with change in angle during either AGms ($p > 0.2$, Table 4.9) or AGml ($p > 0.7$, Table 4.10). There was also no interaction between angle and velocity during AGms ($p > 0.4$, Table 4.9) or AGml ($p > 0.8$, Table 4.10). Therefore, as demonstrated graphically in figures 4.7 and 4.8, antagonist quadriceps activity remained relatively constant with a mean of 6 - 8%Max during AGml and AGms.

4.3.5 Summary of Results

Antagonist EMG activity (cocontraction) was measured in both quadriceps and hamstrings during the constant velocity phase of movement produced by maximal effort voluntary contractions of agonist muscles on a KIN/COM™ dynamometer. However, hamstrings coactivity was significantly greater than quadriceps level of cocontraction.

Hamstrings antagonist activity was dependent on angle, contraction type, and on velocity. During AGml, antagonist hamstring activity was constant. However, during AGms, activity began high and declined rapidly to the level maintained during AGml. Increased contraction velocity produced significantly greater hamstring antagonist activity during both AGms and AGml.

For the quadriceps, overall antagonist activity was low compared to that in hamstrings. In contrast with hamstrings, cocontraction activity was not significantly affected by either angle or contraction type. However, increased contraction velocity produced increased quadriceps antagonist activity similar to the effect in hamstrings, although this was only observed during AGms.

5.0 DISCUSSION

While other studies have reported antagonist cocontraction under certain muscle test conditions (see section 2.5), this is the first report to quantify and describe the antagonist activity in normal subjects under typical clinical test conditions, and to report the relationship of this activity to angle, velocity and contraction type. In addition, the results also support some of the previously reported findings, and refute others. The similarities and differences with previous studies will be discussed together with a review of the specific findings of this study and their clinical implications.

5.1 Angle, Velocity and Contraction Type Dependence

ANTms hamstrings were found to be consistently more active than ANTms quadriceps (compare figures 4.6 and 4.8), which is in agreement with the relationship found between antagonist hamstrings and quadriceps activities during AGms. It is concluded therefore, that under these test conditions, hamstring cocontraction was consistently greater than quadriceps cocontraction irrespective of whether the muscles were shortening or lengthening.

The finding that antagonist hamstring EMG activity was also in part angle, velocity and contraction type dependent is in partial agreement with the findings of Eloranta and Komi (1980). However, there are notable differences in the details between the two studies. Eloranta and Komi (1980) reported that during quadriceps AGms, antagonist hamstrings EMG activity increased, reaching a maximum towards knee extension. The opposite was found in the present study (figure 4.1) in which

antagonist hamstring activity was greatest (mean EMG activity of approximately 30%Max) during the first 10° to 15° of AGms. The level of antagonist EMG activity then declined rapidly and levelled off at about 17%Max during the last 20° to 25° of AGms. Also, Eloranta and Komi's velocity dependence was limited to hamstrings ANTml whereas results from the present study showed velocity dependence during both ANTml and ANTms (see section 4.3.3). The apparent disagreement could be due to methodological differences. Eloranta and Komi used a dynamometer which allowed simultaneous bilateral hip and knee movements, with resistance offered through a foot plate (see section 2.5.3.1). The dynamometer used in the present study restricted movement to the knee joint and resistance was offered perpendicular to the axis of rotation on the distal one third of the tibia. Therefore hamstring muscle action on the two dynamometers is not directly comparable.

While the findings of the present study agree in general with those of Osternig et al (1984) that overall, antagonist hamstrings were more active than antagonist quadriceps, those authors did not observe any angle or velocity dependence in either muscle group (see section 2.5.3.1). Differences in the velocities tested and EMG analysis might account for the divergent opinions. Specifically, velocities used were 100°/s to 400°/s, and standardized procedures were not reported. In addition, EMG activity was not normalized, and levels of EMG activity were divided into initial and final phases, and results compared with numerous t-tests (Osternig et al, 1984).

Angle dependence for levels of antagonist cocontraction was reported by the

Solomonow group (Baratta et al, 1988; Hagood et al, 1990). Antagonist hamstring activity increased during the last 20° of AGms in one study (Baratta et al, 1988) and in the other study increased further from the end of range as velocity increased (Hagood et al, 1990), but this was not observed in the present study. There are two possible explanations for the different patterns of activity. First, Baratta et al (1988) and Hagood et al (1990) included all movement phases in their analysis including the acceleration and deceleration phases whereas in the present study, only the constant velocity phase was included in the analysis. Therefore the increase in antagonist hamstring activity reported by Baratta et al (1988) and Hagood et al (1990) at the end of extension could be due to limb deceleration. In addition, the limit of knee extension was defined as either 10° (Baratta et al, 1988) or 0° (Hagood et al, 1990) short of full extension, whereas in the present study the reference limit was a moment-arm angle of 10° from horizontal. From this measurement, the anatomical knee angle is estimated to be between 10° and 20°. Therefore, the increased activity reported by Baratta et al (1988) at the limits of extension may not have been seen in the present study due to possibility of having studied different ranges of motion.

The shape of the quadriceps ANTml curves reported by Baratta et al (1988) appear similar to those seen in the present study. However, while there was a tendency for mean quadriceps ANTml activity in the present study to decrease from 8%Max to 6%Max during the knee flexion, Baratta et al reported the opposite, with significant increases from 5%Max at the beginning of flexion to 8%Max at the end

of flexion. This pattern of activity was also reported by Hagood et al (1990). The difference between these studies and the present investigation may be due to the fact that the moment-arm of the Cybex II® dynamometer was actively decelerated by the subject, and there was no separation between constant velocity and deceleration phases of knee flexion. With the Cybex II® dynamometer, deceleration can only be achieved by decreasing agonist torque, increasing antagonist torque, or both. The increase in antagonist quadriceps activity noted by Baratta could therefore be due largely to active limb deceleration. In comparison the KIN/COM™ dynamometer actively controls moment-arm (limb) deceleration, removing the need for quadriceps to increase its antagonist cocontraction towards the end of the deceleration phase of knee flexion. Thus the strict selection of constant velocity phase and dynamometer control of limb deceleration likely resulted in different antagonist patterns of activity between the present study and that of Baratta et al (1988).

Baratta et al (1988) assumed that the afferent source of antagonist cocontraction consisted of central reflexes, direct common drive, and peripheral reflexes (primarily kinaesthetic, and possibly proprioceptive). They ruled-out muscle spindles and Golgi tendon organs (GTO) as likely contributors to cocontraction. Based on the pattern of antagonist muscle activity, thought to be "nearly" inversely proportional to its moment-arm, it was concluded that kinaesthetic joint capsule receptors were "the major source to dictate..." such antagonist cocontraction activity. Three aspects of the results of the present study suggest otherwise. First, no

statistically significant changes in EMG activity were observed in antagonist quadriceps during either AGms or AGml. In fact, the trend observed in such activity was opposite to that reported by Baratta et al (1988). Second, while angle dependent changes in antagonist hamstrings in the early phase of AGms were similar to those reported by Baratta et al (1988), that activity during the terminal phase of AGms did not increase as would be expected according to the moment-arm hypothesis of Baratta et al (1988). Third, it would be predicted from the moment-arm hypothesis that the pattern of antagonist activity during AGml should also be inversely related to the muscle moment-arm. The results of the current study demonstrated distinctly different antagonist hamstring responses between AGms and AGml. While antagonist quadriceps did show similar responses between AGms and AGml, the pattern of activity was not inversely related to muscle moment-arm. Therefore, the conclusion that normalized antagonist EMG plotted against joint angle for each muscle group relates inversely to the muscle moment-arm (Baratta et al, 1988) cannot be supported by the findings of this study.

The results of the present study point to muscle spindles as the major source of the pattern of antagonist cocontraction. While the velocity dependence during hamstring ANTms was significant, it was not as clearly apparent as during hamstring ANTml (compare figures 4.6 and 4.5). The difference in response points to the velocity-sensitive component of the muscle spindle afferents as a possible source of these variations. Primary afferents from muscle spindles are very sensitive to rate of change of stretch (Carew & Ghez, 1985). Therefore the faster the

antagonist hamstrings are stretched during ANTml, the greater the hamstrings muscle spindle response, and the greater the resulting reflex contraction of the hamstring extrafusal fibres. However, during ANTms the spindles will be biased towards being unloaded, and their sensitivity to the rate of change of shortening will be less than during ANTml. This explanation of the source of antagonist cocontraction is also supported by the antagonist quadriceps activities (compare figures 4.7 and 4.8). Antagonist quadriceps were significantly more active at 90°/s than at 30°/s during AGms, and while not significant, the trend was in the same direction during AGml.

Other evidence which points to muscle spindles as the source of the observed pattern of antagonist activity is the work of Cooke and Brown (1990) and Knutsson (1987) described in previous sections. The former demonstrated a burst of activity from an agonist/antagonist pair during limb acceleration. While the EMG activity in the acceleration phase was not examined in the present investigation, it is possible that the non-linear decrease in antagonist hamstring cocontraction during AGms is the decaying slope of such a phasic burst. Knutsson (1987) demonstrated very high levels of antagonist cocontraction in patients with spastic paresis during constant velocity muscle testing and that excessive activity was significantly reduced by the application of ice to the antagonists prior to testing.

It can therefore be concluded that (1) the results of this study support the hypothesis that the pattern of antagonist activity is controlled primarily by antagonist muscle spindle afferents, and (2) the pattern of antagonist activity seen

in this study does not support the hypothesis that such activity is inversely related to the muscle moment-arm.

5.2 Overall Levels of Antagonist Activity

The overall low levels antagonist quadriceps EMG activity during AGms observed in the present study are consistent with the findings of others (Osternig et al, 1984; Solomonow et al, 1987; Baratta et al, 1988). The finding in the present study that there was no significant change in antagonist quadriceps EMG activity between AGms and AGml has not been reported previously (figures 4.3 and 4.4). However, the relatively high levels of antagonist hamstrings EMG activity during AGms found in the present study are consistent with the findings of Osternig, et al (1984), but not with those of Solomonow's group (Solomonow et al, 1987; Baratta, et al, 1988; Hagood et al, 1990). In addition, the finding in the present study that antagonist hamstring EMG activity remained high during AGml but with angle dependence which was different than that during AGms has not been reported previously and will be discussed in detail following this section on overall levels of antagonist activity. Obvious differences between the studies which might explain the apparent contradictions in antagonist hamstring EMG activity during AGms will be discussed in terms of positioning, stabilization, test velocities, and subject populations sampled. The physiologic and anatomical differences between the quadriceps and hamstrings might also account for the different levels of antagonist activities between the two muscle groups found in the present study and will also be discussed.

5.2.1 Positioning

Baratta et al (1988) tested all subjects in side lying with the hip flexed to 30°. In contrast, the current investigation as well as Osternig et al (1984) and Solomonow et al (1987) tested subjects in the sitting position with the hip flexed to 90°. The difference in hip angles between studies would affect the hamstrings length-tension curve, and could affect the activity of the hamstrings to a greater degree than the quadriceps (Felder, 1978; Lunnen et al, 1981; Bohannon et al, 1986). However, positioning is unlikely to be the main reason for different antagonist hamstring cocontraction levels because Baratta et al (1988) and Solomonow et al (1987) used the side lying and sitting positions respectively but reported similar low antagonist hamstring cocontraction levels.

The effect of gravity could also be a factor which influences effects of position on hamstring and quadriceps muscle activity. In the side lying position, gravity has minimal effect on knee flexion/extension. However in sitting, gravity opposes the extension moment of a quadriceps muscle shortening action. Thus antagonist hamstrings would not need to be as active to maintain a given flexion moment. Hamstring antagonist activity would therefore be expected to be lower in sitting than in side lying. However, when comparing the results of the present study with those of Baratta et al (1988), the opposite was found. Therefore, gravity cannot account for the disparity in overall antagonist hamstring cocontraction between Baratta et al (1988) and the present study.

5.2.2 Stabilization

Inadequate stabilization is known to increase the compliance of the system making it unlikely that axial alignment between the dynamometer and knee can be maintained during maximal effort. This will produce large errors in the measurement of torque output (Nosse, 1982; Herzog, 1988), and decrease the maximal torque that can be generated during muscle testing on a Cybex II® dynamometer (Nosse, 1982; Smidt and Rogers, 1982; Cybex, 1983; Hart et al, 1984). The manufacturer's instruction manual states that Cybex II® protocols require maximum stabilization during testing and rehabilitation (Cybex, 1983).

In the study of Baratta et al (1988) the trunk was stabilized to the Cybex frame with the knee and shank "resting on the Cybex crank". It was also specifically stated that no other restraints were used because they wanted to obtain data under conditions as close to normal as possible. Since no straps were applied to the thigh or pelvis, the degree of stabilization would be low. In the present study and in others (Osternig et al, 1984; Solomonow et al, 1987; Hagood et al, 1990), subjects were well stabilized by straps applied to the trunk, pelvis and thigh. Although different levels of stabilization used by the Solomonow group (Solomonow et al, 1987; Baratta et al, 1988; Hagood et al, 1990) did not affect the levels of antagonist hamstring cocontraction, it seems unlikely that differences in stabilization alone could explain the differences in antagonist hamstring cocontraction levels between the present study and those of the Solomonow group (Solomonow et al, 1987; Baratta et al, 1988; Hagood et al, 1990).

However the importance of stabilization as a factor in the present study cannot be ruled out completely because both active bracing and passive stabilization straps were used. Subjects were asked to lean back against the back rest, grasp the sides of the table and hold themselves down while performing an MVC. With the addition of active stabilization, it may be possible for subjects to exert greater agonist torque. Since it has been postulated that hamstrings cocontract with quadriceps in order to generate enough posteriorly directed force on the tibia to produce a mechanically significant decrease in the load on the ACL (Solomonow et al, 1987; Draganich et al, 1989) more agonist quadriceps torque would result in a high anterior shear force on the ACL, and therefore more antagonist hamstring cocontraction would be needed to counteract the shear force.

Therefore stabilization may be an indirect factor in the different levels of antagonist hamstring cocontractions found between the present study and the Solomonow group (Solomonow et al, 1987; Baratta et al, 1988; Hagood et al, 1990).

5.2.3 Velocity

It has been shown in this study and in others (Eloranta and Komi, 1980; Osternig et al 1984, 1986) that higher test velocities result in higher levels of hamstring antagonist cocontraction during AGms. The velocity used by Solomonow's group (Solomonow et al, 1987; Baratta et al, 1988) was 15°/s, half of the slowest velocity used in the present study, and also less than those used by Osternig et al (1984, 1986). However, Hagood et al (1990) studied a velocity range from 15°/s

to 240°/s and reported that while both antagonist hamstrings and quadriceps demonstrated increased levels of cocontraction during the final 40° of movement at increased velocities, decreases in coactivation levels were observed during the initial phases of movement with increased velocity. In addition, levels of antagonist hamstri ng cocontraction were still lower than reported in the present study at similar velocities. Therefore it is unlikely that test velocity alone can account for the different levels of antagonist cocontraction between the present study and the Solomonow group (Solomonow et al, 1987; Baratta et al, 1988; Hagood et al, 1990).

5.2.4 Subject Populations

Different subject populations have different levels of antagonist cocontraction, and this is particularly evident in antagonist hamstrings (Osternig et al, 1986; Baratta et al, 1988). Baratta et al (1988) demonstrated that normal subjects and high performance athletes have low levels of antagonist hamstring cocontraction similar to the level of antagonist quadriceps cocontraction, but two athletes showed significantly greater antagonist hamstring cocontraction which approximated levels found in the present study and those reported after 2-3 weeks of hamstring training (Baratta et al, 1988). Osternig et al (1986) also studied athletes (sprinters and endurance runners) and reported mean antagonist cocontraction levels as high as 59% with a very wide variability (SD = ±37%) in sprinters, but as low as 8% (SD = ±2.7%) in endurance runners. Based on these results, it could be argued that the normal subjects in the current study may be equivalent to the hamstring trained

high performance athletes studied by Baratta et al (1988), and somewhere between the sprinters and endurance runners in the Osternig et al (1986) study. In the present study, each subject was screened for fitness and activity levels prior to entry into the study. Level of fitness and activity level were assessed. Only two subjects described themselves as 'very fit' while 17 described themselves as 'fit' and reported exercising three times a week. None of the subjects considered themselves or were considered by the investigator to be athletes. Osternig et al (1984) described their subjects as five young male adults. Solomonow et al (1987) reported on six normal healthy men. Baratta et al (1988) tested seven nonathletic normal subjects (four female and three males), and Hagood et al (1990) sampled six adult males and two adult females. The present study tested twelve young adult males and twelve young adult females who were generally active. Clearly, subject sample differences are a possible explanation for the different findings, although it is known that the Solomonow group also used volunteers from the local Physiotherapy and Occupational Therapy School (Solomonow, 1989) as was done in the present study.

The most obvious difference between the present study and the others is study sample size. The present study had more than three times the subject numbers than any other study of normal subjects to date, and they were tested in a balanced design by rigorous statistical analysis. It is possible therefore that the studies which used small samples were biased towards subjects with low levels of cocontraction by the large standard deviations in their groups.

Therefore, possible differences in subject sampling together with significant

differences in numbers of subjects tested is the most likely explanation for the differences in antagonist hamstrings cocontraction reported for normal subjects in the studies discussed above. However the differences in positioning, stabilization, and test velocities cannot be ruled out completely, as contributors to the different levels of antagonist hamstring cocontraction reported.

5.3 Differential Levels of Antagonist Activity

Having concluded that the relatively high level of antagonist hamstring cocontraction in the present study is plausible, it is now necessary to discuss why a difference in levels of antagonist cocontraction between quadriceps and hamstring muscle groups was found and what controlling mechanisms might be involved.

5.3.1 Biomechanical Differences

One of the functions of cocontraction is thought to be active regulation of joint stiffness and therefore joint stability, which compliments the passive roles of the ligaments and contoured joint surfaces (Solomonow et al, 1987; Baratta et al, 1988; Solomonow et al, 1989). For this function, hamstrings must be able to control anterior shear of the tibia against the femur, while quadriceps should control posterior shear (as well as various rotatory and torsional stresses). From a biomechanical point of view, hamstrings are well positioned to control anterior shear between 85° and 25° of flexion (O'Connor et al, 1990). Computer models of the knee joint have shown that between these flexion angles, antagonist hamstring cocontraction can substitute completely for the load carrying functions of the ACL in the sagittal plane under certain test conditions (O'Connor et al, 1990). Hamstring

cocontraction has also been shown to reduce total torsional knee laxity at all flexion angles with the largest reduction (76%) occurring at 90° of flexion (Louie and Mote, 1987). These results correspond closely with clinical studies which show that hamstring cocontraction can decrease the anterior tibial shift during the classical anterior drawer test (knee flexion at 90°) significantly more than during the Lachman test (knee flexion at 15°) (Iversen et al, 1989).

Since anterior shear stress would be predicted to be greater during AGml than during AGms, antagonist hamstring activity should be greater during quadriceps AGml than during AGms, particularly at the beginning of knee flexion where ACL anterior shear stress is greatest (Renstrom et al, 1986). This hypothesis appears to be supported by the data recorded at 30°/s (figure 4.1) but not by the data recorded at 90°/s (figure 4.2). On closer examination of these data, the differences between hamstring ANTms and ANTml in the early part of flexion appear due to lower cocontraction during ANTml rather than to higher cocontraction during ANTms. If ACL protection or support was the primary function of antagonist hamstrings, hamstring ANTms activity would have to be greater than ANTml. More specifically, hamstring ANTms activity would have to be greater at the beginning of AGml than at the end. These ideas are also supported by an examination of ACL mechanics. The ACL applies tension between the tibia and femur only between 60° and 0° of full extension whereas little or no tension is developed for angles between 120° to 60° (Renstrom et al, 1986). Such a pattern of cocontraction was not found (see figure 4.5). Indeed, although not statistically

significant, the trend was just the opposite. Therefore while antagonist hamstring cocontraction may contribute toward joint stiffness or stability during AGms, such control appears limited since it did not increase during AGml. In fact, the pattern of antagonist hamstring cocontraction in the present study suggests that in the normal knee, the hamstring muscle group controls anterior shear in flexion where the ACL cannot provide adequate tension, and as the knee extends, hamstrings group decreases its activity as the ACL increases its control of anterior shear.

Applying the same concept of muscles relieving or controlling stress on ligaments, O'Connor et al (1990) used the same computer models to study the quadriceps antagonist activity. He found that quadriceps could only act as an antagonist to hamstrings in controlling posterior shear over the very narrow range of 10° to 25° of knee flexion. Within that range, very large muscle and contact forces were predicted to be necessary to relieve stress on the PCL. For most of the flexion range, the geometry of the patellar tendon is such that the major force vector is directed upwards towards the femur rather than forwards in the direction of limb movement. This would increase joint contact forces but provide minimal assistance to the PCL in controlling posterior shear. Therefore the minimal antagonist quadriceps cocontraction in comparison to hamstring cocontraction observed in this and other studies can be at least partly explained by the fact that the quadriceps via a taut patellar tendon cannot control the posterior shear stress on the PCL and is therefore not required to be activated to any significant degree.

5.3.2 Morphological Differences

As discussed in section 2.1.2, muscles contain two types of fibres, fast- and slow-twitch, which can be distinguished both morphologically and physiologically. If levels of excitation to antagonist quadriceps and hamstrings are the same, then for fibre type composition to explain greater antagonist cocontraction in hamstrings than in quadriceps, hamstrings would have to be more excitable. Since slow muscle fibres have a lower threshold of excitation, and are recruited before fast fibres during low level muscle contractions (Guyton, 1981), the proportion of slow:fast fibres in a muscle may contribute to the final amount of cocontraction.

The fibre composition of m. vastus lateralis has been studied extensively, but results are conflicting (Johnson et al, 1973; Edgerton et al, 1975; Monster et al, 1978; Lexell et al, 1983). The percentage of slow fibres in the normal human m. vastus lateralis has been found to range from 37% (Monster et al, 1978) to 52% (Lexell et al, 1983). While some of this variability is due to the sampling error of the biopsy technique (Lexell and Taylor, 1989), there is also general agreement that considerable inter- and intramuscular heterogeneity exists (Johnson et al, 1973; Edgerton et al, 1975; Lexell et al, 1983 & 1988). An example of the extremes of such variability is the comparison of fibre composition in elite athletes. The proportion of slow fibres in the m. vastus lateralis have been reported to be as low as 20% in sprinters and as high as 95% in marathon runners (Gollnick et al, 1972; Thorstensson et al, 1977). On the whole, overall m. vastus lateralis has between 1:2 - 1:1 slow:fast fibre ratio.

The fibre composition of human hamstring muscles is less well documented, however 65% to 67% slow fibres in m. biceps femoris has been reported (Johnson et al, 1973; Monster et al, 1978). While inter-subject variability is as high in hamstrings as it is in quadriceps, hamstrings show less intramuscular variation judging by the standard errors reported in studies to date.

Therefore, based on the evidence for normal subjects, quadriceps is proportionally faster than hamstrings based on fibre type distribution. So for a given level of excitation, hamstrings would be expected to produce a greater level of cocontraction than quadriceps. The findings of the present investigation are consistent with this supposition.

Furthermore, since inter-subject variability of fibre type composition is high, large variations in levels of cocontraction could also be predicted between subjects in this type of study. Such variability was clearly seen in antagonist hamstrings data, although it was less apparent in antagonist quadriceps (compare standard errors in figures 4.1 and 4.2 with figures 4.3 and 4.4. Another possibility for the cause of the inter-subject variability in antagonist hamstring activity is also examined in APPENDIX F). Therefore the differences in fibre type distribution between the two muscle groups could explain, at least in part, the gross difference in levels of antagonist cocontraction between quadriceps and hamstrings.

5.3.3 Motor Control Differences

The motor control of quadriceps and hamstrings may also have an impact upon their cocontraction as antagonists. It is known that increased intra-articular

pressure and joint capsule tension inhibit muscle activity. In the knee joint, the quadriceps are much more sensitive to these inhibitory influences than are the hamstrings (Young et al, 1987). For example, knee joint distension in normal subjects has been shown to lower quadriceps motor neuron excitability via reflex joint afferent stimulation (Kennedy et al, 1982; Spencer et al, 1984; Iles et al, 1990). In patients with knee ligament injury, such inhibition leads to relatively greater muscle atrophy in the quadriceps than in the hamstrings (Ingermann-Hansen and Halkjaer-Kristensen, 1980; Gerber et al, 1985; Lorentzon et al, 1989). The joint distension need only be small (20-30ml) to produce profound quadriceps inhibition (Young et al, 1987), and the level of the inhibition is independent of pain (Shakespeare et al, 1985). Infusion of only 10ml of saline (a clinically undetectable volume) into the knee joint has been shown to inhibit the quadriceps H-reflex particularly during voluntary muscle contractions (Iles et al, 1985). Presumably the inhibitory effects arise due to stimulation of proprioceptors in the joint capsule (Iles et al, 1990).

Other sensory receptors such as GTO Ib afferents could also be involved in differentially regulating levels of antagonist cocontraction between quadriceps and hamstring muscle groups. The rate of firing of GTO's is proportional to the tension developed in the tendon of the muscle innervated by the GTO, and that firing rate in turn increases both the level of excitation of the muscle's antagonist, and the level of inhibition to itself (Carew, 1985). Since Ib afferents make relatively weak connections to flexor muscles in comparison to the extensors (Carew, 1985), and

the quadriceps can produce much greater absolute tension than hamstrings, quadriceps GTO's must provide proportionately more excitation to antagonist hamstrings (and more auto-inhibition) than the hamstrings do to antagonist quadriceps.

The function of these spinal reflex pathways together with supraspinal influences (see section 2.4.4) would be reflected in the relative cocontraction between quadriceps and hamstrings observed during functional activities such as locomotion. Typical EMG patterns of knee flexor and extensor activity during gait show cocontraction of these muscles just before and after heel strike, and these levels of activity tend to increase with increased walking speed (Murray et al, 1984; Shiavi et al, 1987).

5.4 Summary and Implications

Under typical clinical muscle test conditions, antagonist hamstring cocontraction was found to be angle, velocity, and contraction type dependent, being more active at the faster velocity, during ANTml, and at the beginning of ANTml. No such relationships were seen for antagonist quadriceps except for the velocity dependence during ANTml. It is proposed that the differences in levels of antagonist hamstring and quadriceps cocontractions between previous studies and the present investigation are due to a combination of differences in positioning, stabilization, preset velocities, and subject sampling. The differential levels of antagonist cocontraction between quadriceps and hamstrings found in the present investigation can be explained by knee joint biomechanics, different muscle fibre

composition, and different sensitivities to motor control regulation. It is proposed that the patterns of activity observed in the present study are the result of known functions of Ia and Ib afferents, rather than the result of varying muscle moment-arm as suggested by Baratta et al (1988).

It is also proposed that the variability in overall levels of antagonist hamstring cocontraction between subjects is in some way due to motor control bias within each subject towards either joint stability and protection of ligaments (high cocontraction), or the production of joint torque (low cocontraction). The use of high cocontraction could be used as a strategy for unloading and protection of ligaments but the price to be paid for such protection would be increased joint contact forces. This may lead to increased wear and tear on the joint surfaces which over time may not be wholly benign.

Finally, constant velocity muscle performance tests are considered an important clinical procedure in the fields of orthopaedic medicine, rehabilitation medicine, and physiotherapy. The results of the present study suggest that knowledge of the muscle activity that produces the torque-angle data is necessary for an accurate interpretation of such data. Comparing such activity to that in patients with motor control problems (e.g. spasticity, rigidity), or musculoskeletal pathology (e.g. ACL insufficiency) could lead to new noninvasive tests of muscle and joint dysfunction and new rehabilitation treatment strategies.

6.0 CONCLUSIONS

The purpose of this study was to investigate the electrical activity of antagonist quadriceps and hamstring muscle groups during maximal effort constant velocity AGms and AGml, and to determine the relative effects of joint angle, limb velocity, and contraction type on the level of antagonist activity using a typical clinical test protocol. This was done to establish normal baseline data for the KIN/COM™ and similar robotic dynamometers for subsequent studies of males and females with various orthopaedic and neurological conditions affecting the knee joint and surrounding musculature. In addition, it is also important for constant velocity muscle testing to understand the relationship between agonist and antagonist activation, the factors which affect this relationship, the likely mechanisms of control, and whether the measurement of such activity is important for clinical interpretation of patient torque data.

The results of a pilot study confirmed the appropriateness of the EMG procedure for electrode placement and measurement of antagonist muscle activity without cross-talk contamination, established the test velocities and range of motion to be used, and determined the chart speeds and resolvable angle for optimal measurement of EMG parameters.

To facilitate evaluation, the raw EMG signal was converted into a linear envelope by full wave rectification and low pass filter processing. The dynamometer torque and angle tracing and EMG signal were recorded simultaneously on a moving

chart. Digital recordings of the dynamometer data were also recorded onto computer discs.

Twelve male and twelve female healthy volunteers were required to perform four maximal effort dynamic tests of the knee flexors and extensors through a defined range of motion under two constant velocity conditions in a single session. Each test consisted of four repetitions. A repetition was defined as an AGms phase followed by an AGml phase, through a preset range of motion of 65°. Two tests were performed with the left knee extensors as agonists, and two tests were performed with the left knee flexors as agonists. For each muscle group, one test was performed at 30°/s, and another at 90°/s. The sequence of muscle group (extensors/flexors or flexors/extensors) and velocity (30/90 or 90/30) testing was randomized.

The electromyographic activity of the knee flexors and extensors was recorded while the tests were being performed. From the chart recording the peak amplitude EMG at every 5° of the constant velocity phase of each test was measured. For each contraction type and velocity of contraction in each muscle group, EMG activity was measured at exactly the same moment-arm angles so that the effects of velocity and contraction type could be evaluated in a paired manner while controlling for joint angle. To quantify the antagonist muscle activity, the peak EMG amplitude was normalised at each joint angle with respect to its peak EMG amplitude at the same angle when that muscle group was acting as agonist at maximum effort during muscle shortening at 30°/s.

Hamstrings antagonist activity was dependent on angle, contraction type, and velocity. Antagonist hamstring activity began high (mean EMG activity 25%Max to 28%Max) during AGms, and as the knee extended, declined rapidly to approximately 18%Max to 20%Max (mean EMG activity). This level of cocontraction was maintained during AGml. Increased contraction velocity produced significantly greater hamstring antagonist activity during both AGms and AGml. Overall hamstring antagonist activity was significantly higher than antagonist quadriceps activity in all tests.

Overall antagonist quadriceps EMG activity was low (5%Max to 8%Max mean EMG activity) and was not significantly affected by either angle or contraction type. However, increased contraction velocity produced increased quadriceps antagonist activity during AGms but not during AGml.

The results demonstrate antagonist hamstrings and quadriceps respond differently under typical constant velocity test conditions. While levels of antagonist activity, particularly hamstrings, vary among individuals, the influence of that pattern by the angle, velocity, and contraction type of that activity is the same between individuals. These findings suggest that mechanisms regulating cocontraction are the same between individuals and probably involve the interaction of muscle spindles and GTO's. The variability in overall levels of antagonist hamstring cocontraction between subjects suggests that motor control strategies are used to bias the system towards either joint stability and protection of ligaments (high cocontraction), or the production of joint torque (low cocontraction). The use

of high levels of cocontraction could be a strategy for unloading and protection of ligaments, but the price to be paid for such protection would be an increase in joint contact forces. This would increase wear and tear on the joint surfaces and over time may not be wholly benign. Further study of cocontraction is warranted.

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Figure 3.1. Electrode placement sites for (A) M. Vastus Lateralis, and (B) Hamstrings muscle group


B

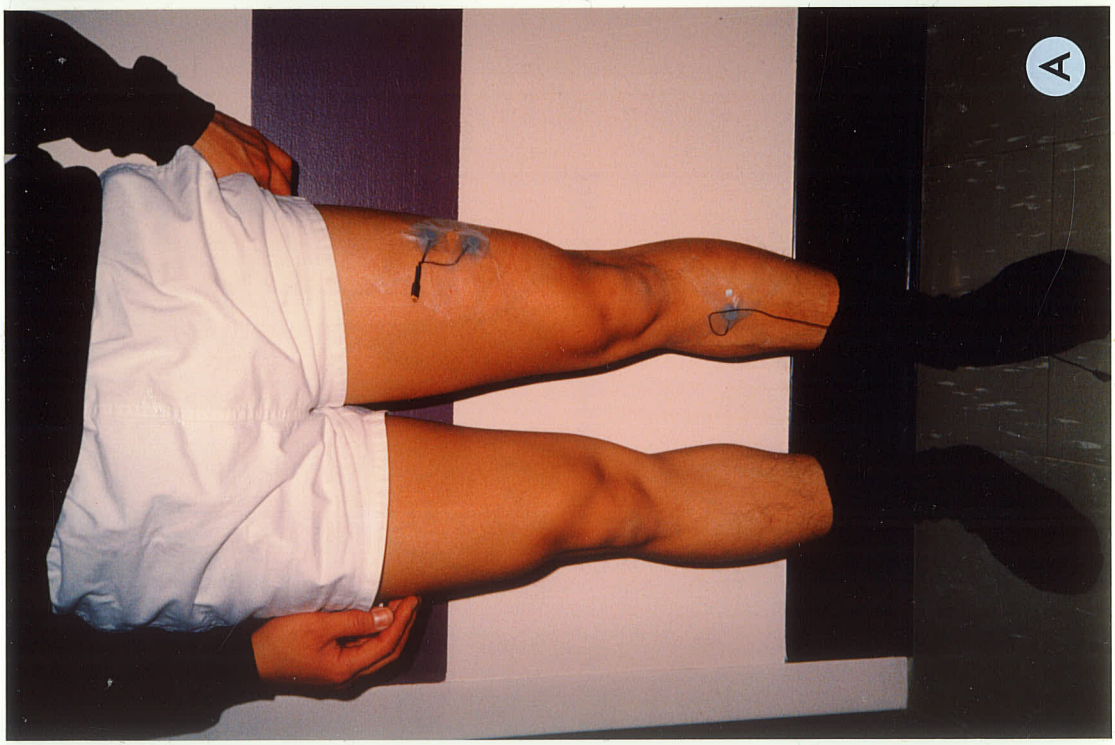

A

Figure 3.2. Oscilloscope recordings for cross-talk in the pilot study. The dual trace recordings in (A) and (B) demonstrate submaximal reciprocal contractions of the quadriceps and hamstrings during repeated active knee flexion and extension. (C) and (D) demonstrate randomly changing phase of the signals in all quadrants during quadriceps maximal muscle tests. The signal from the agonist is input to the X-axis and the signal from the antagonist is input to the Y-axis.

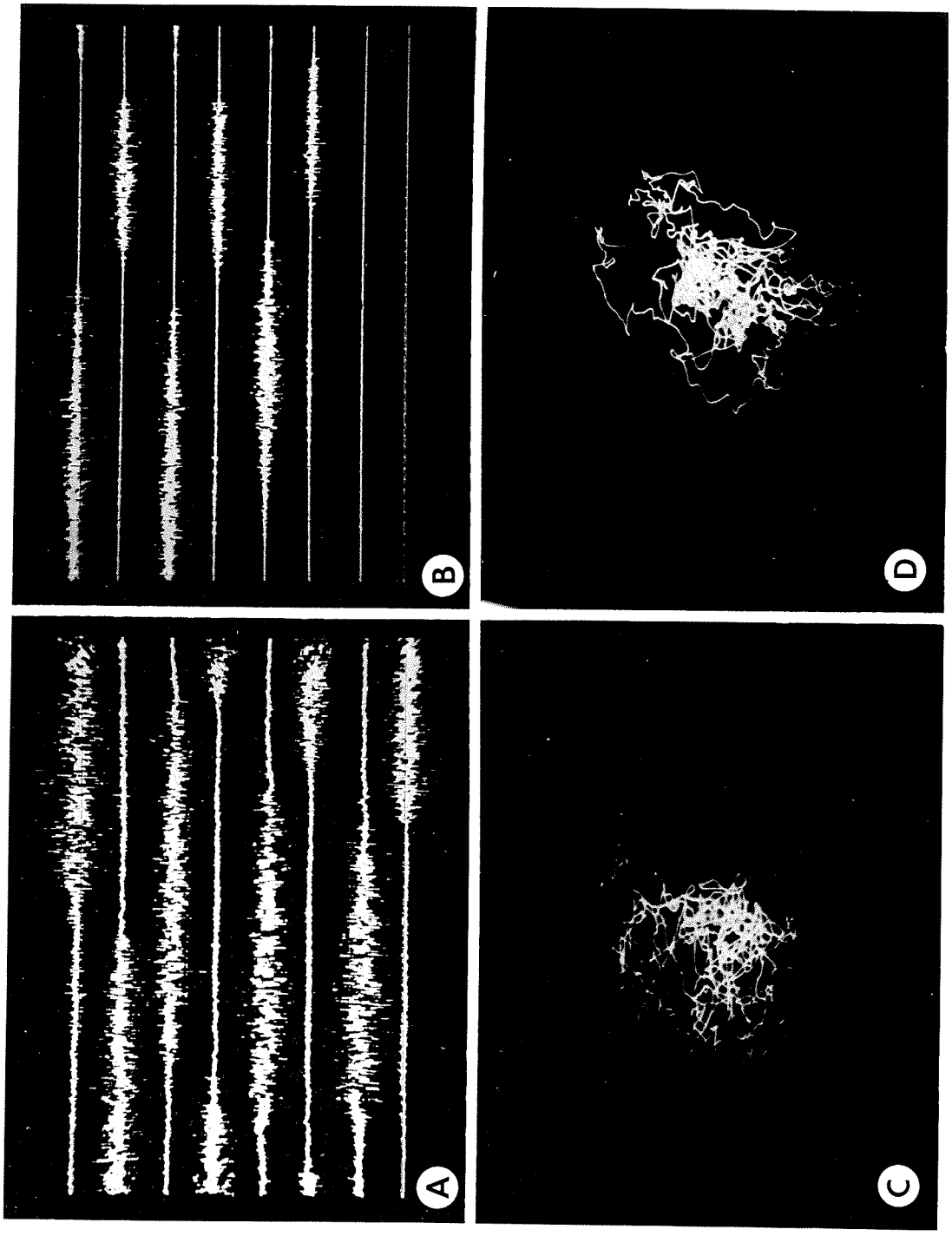

A
B
C
D

Figure 3.3. Constant velocity range for agonist quadriceps muscle shortening (ms) and lengthening (ml) measured between 5 subjects for preset velocities of 30 - 210°/s. Small differences between subjects were seen during ms together with a decline in constant velocity range at higher preset velocities. At 30°/s, the constant velocity range for ml was the same as that during ms. At higher velocities however, the variability between subjects increased. One subject did not achieve the preset constant velocity at 120°/s or higher.

Constant Velocity Range

Quadriceps

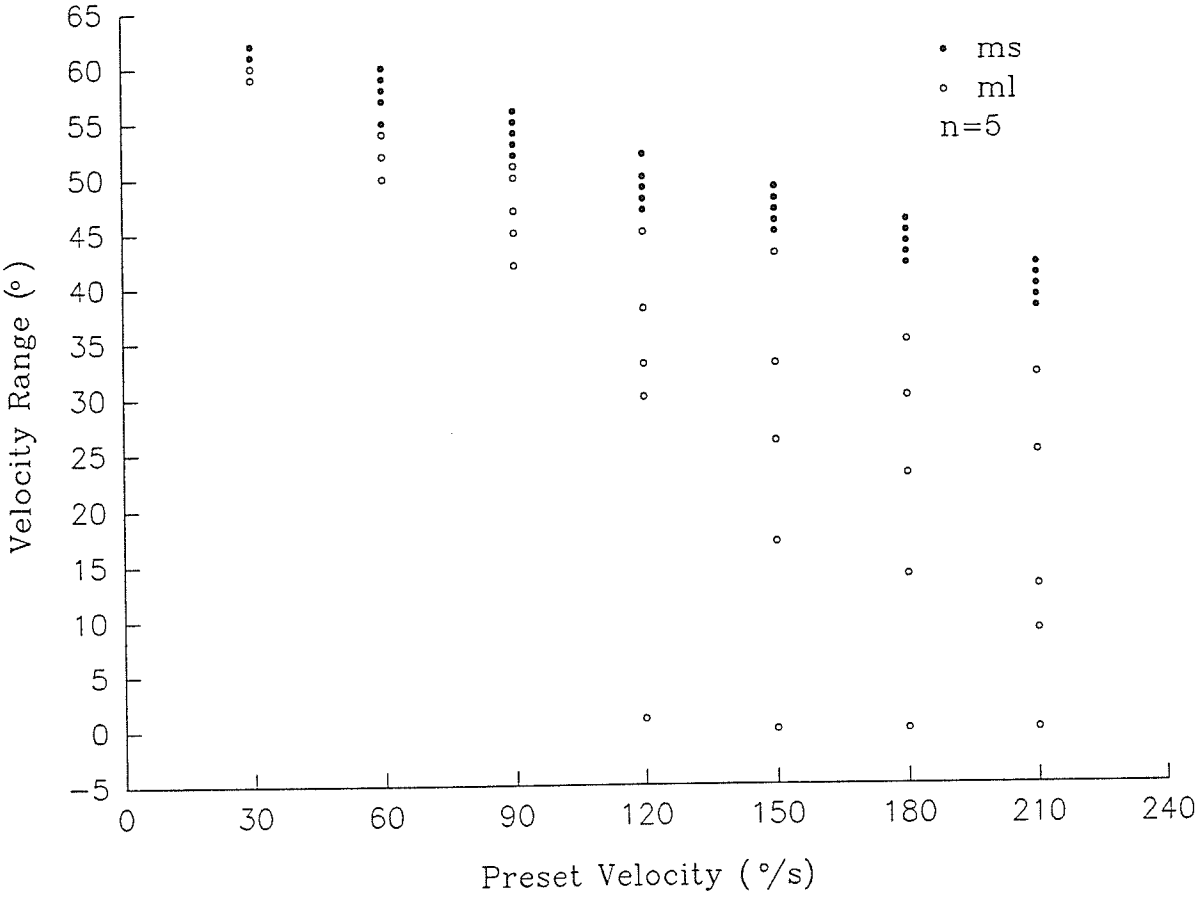
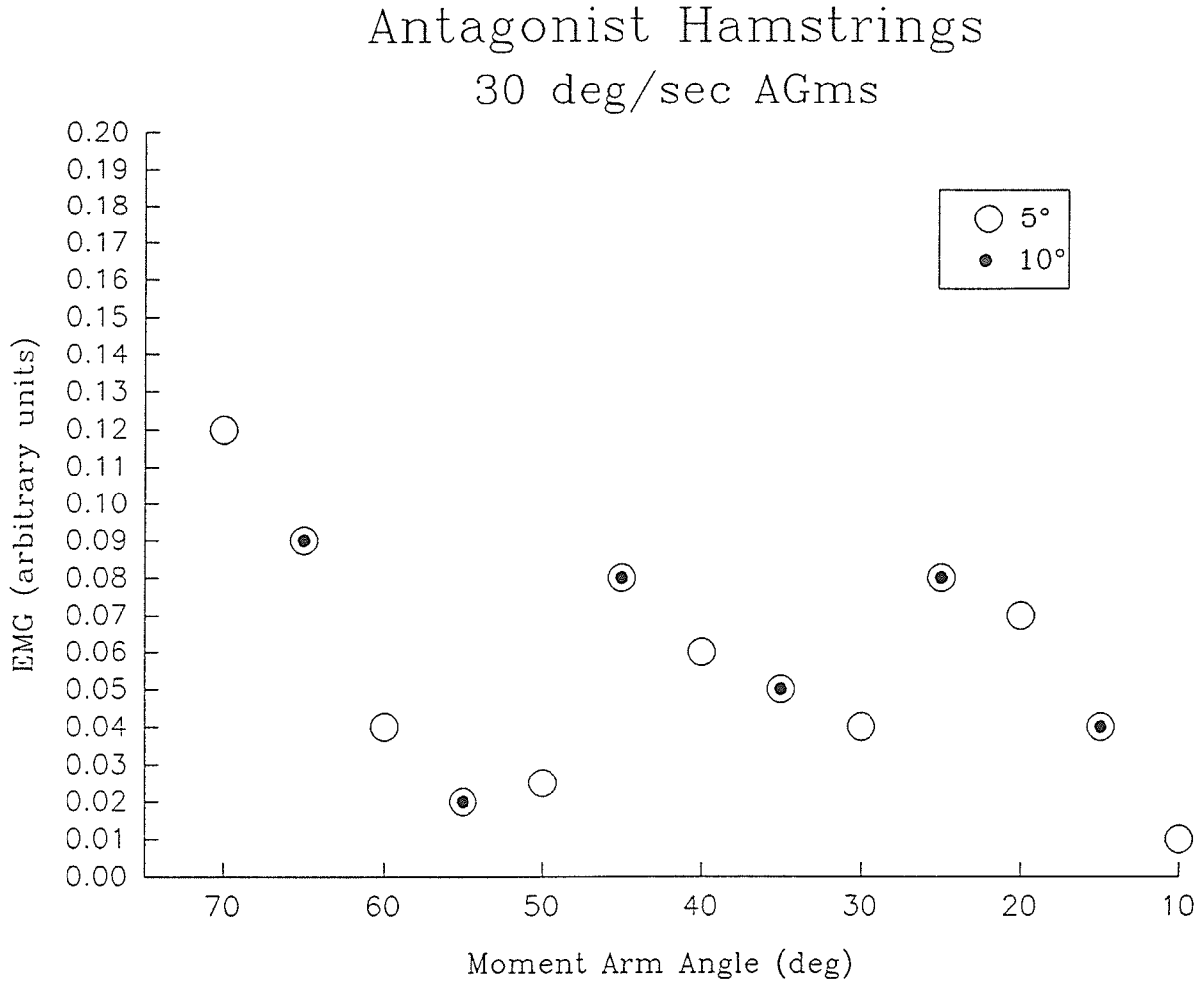


Figure 3.4. Examples of measurement of antagonist EMG activity every 5° vs 10° during (A) agonist muscle shortening (AGms), and (B) agonist muscle lengthening (AGml). It was found that measuring EMG activity every 5° by connecting each point was more discriminating than measuring every 10° by connecting alternate points over a given range.


Antagonist Hamstrings
30 deg/sec AGms
EMG (arbitrary units)
0.20
0.19
0.18
0.17
0.16
0.15
0.14
0.13
0.12
0.11
0.10
0.09
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0.00
70
60
50
40
30
20
10
Moment Arm Angle (deg)
5°
10°

Antagonist Hamstrings

30 deg/sec AGml

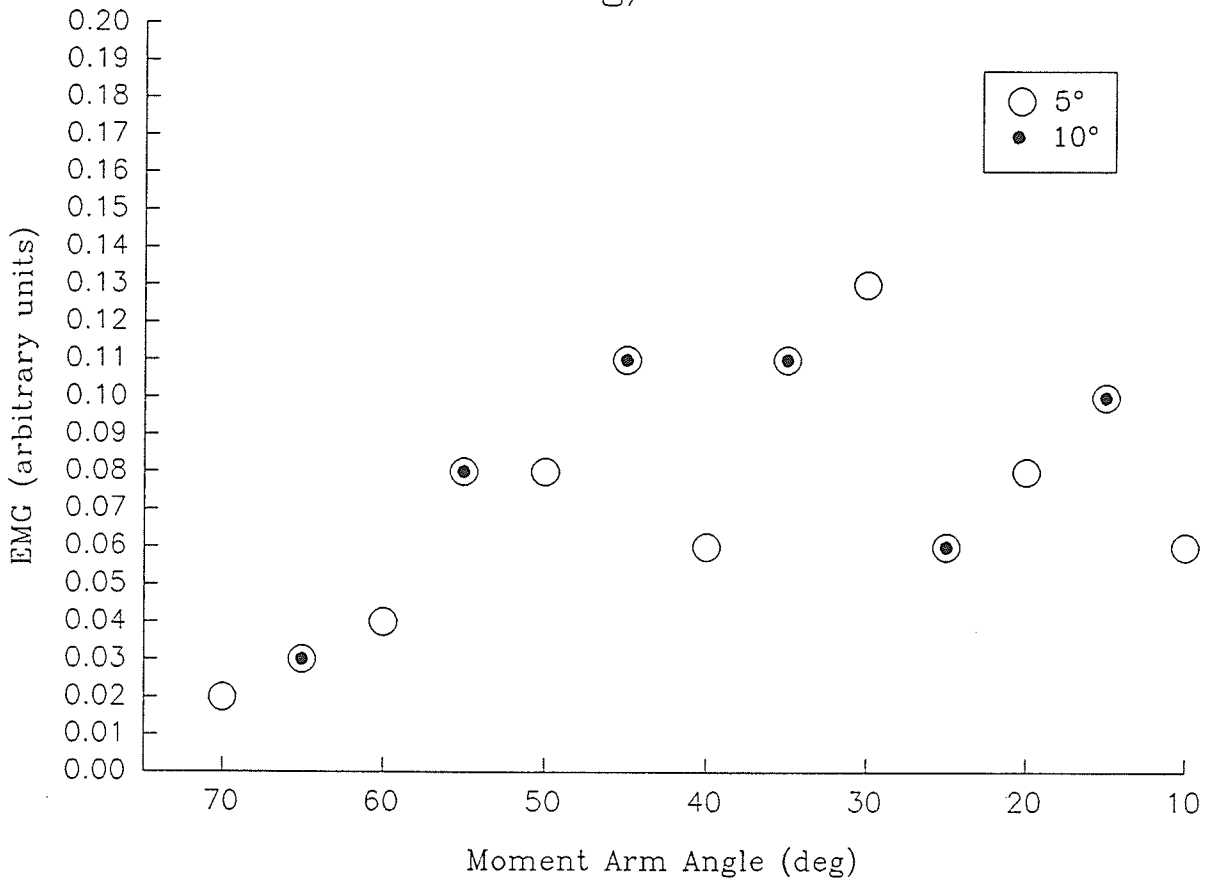
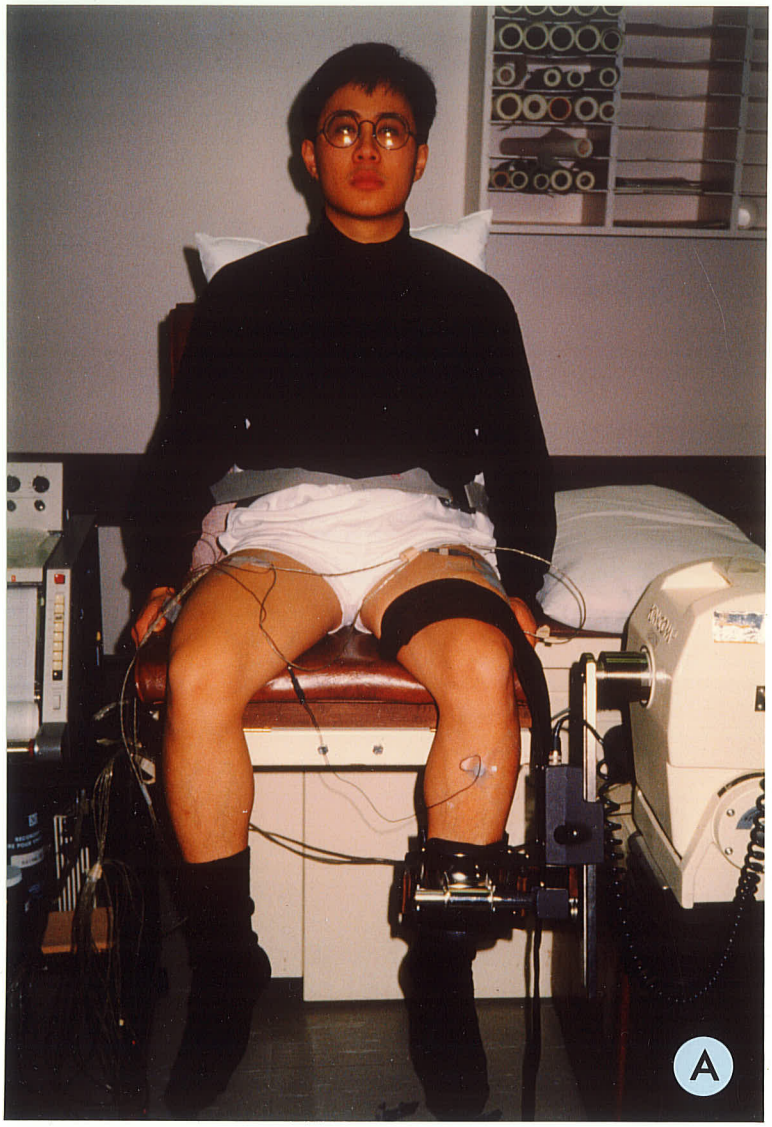


Figure 3.5. (A) Frontal view of the position of a subject on the dynamometer. Note the stabilization straps securing the pelvis and the lower one third of the left thigh. (B & C) Side views of the test range of motion showing (B) the limit of knee extension, corresponding to a moment-arm angle of 10°, and (C) the limit of knee flexion, corresponding to a moment-arm angle of 75°.


A


B


C

Figure 3.6. Examples of oscilloscope recordings demonstrating the absence of cross-talk during maximal isometric contractions (A & B) and maximal constant velocity contractions (C & D). The hamstring electrodes are input to the Y-axis and the m. vastus lateralis is input to the X-axis. In (A) the signal phase is restricted to m. vastus lateralis EMG activity. In (B) the signal phase is restricted to hamstring EMG activity. In (C) and (D) randomly changing phase of the signals indicates both muscle groups to be active with no cross-talk between electrodes.

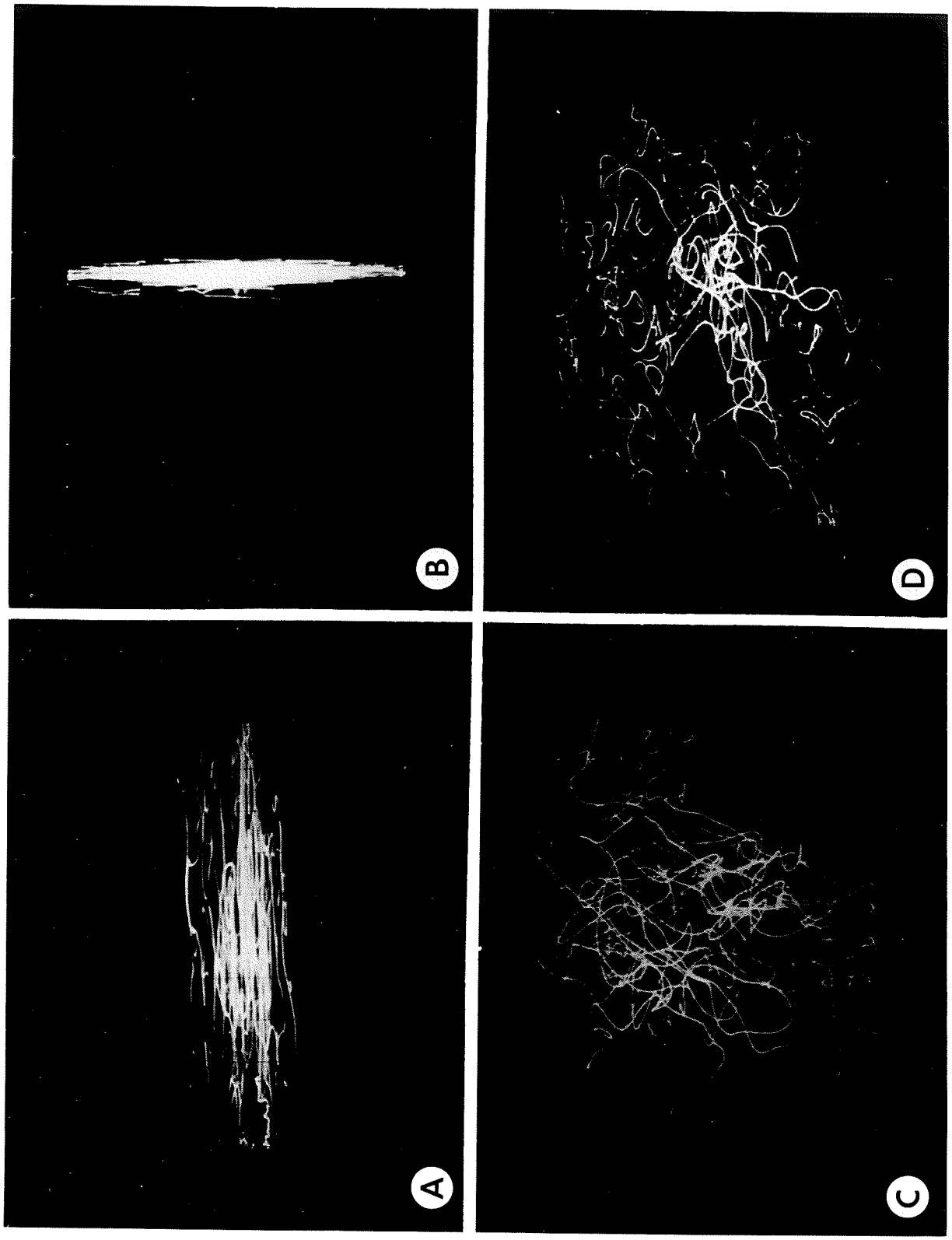
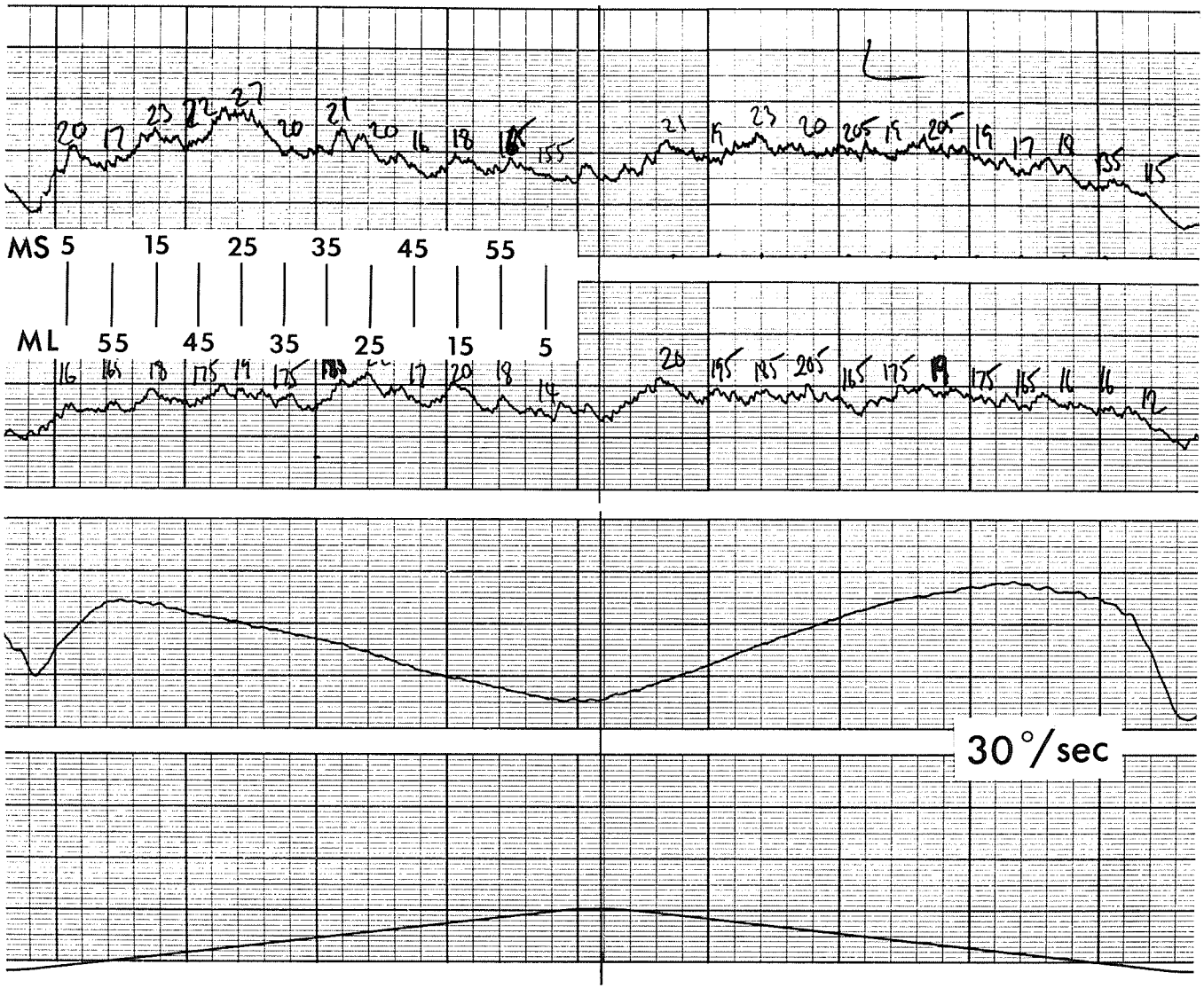

B
D
A
C

Figure 3.7. Typical chart recordings of one complete repetition showing EMG activity for both muscle groups, force, and angle (A) for agonist quadriceps (channel 1; 1mm deflection = 33.2μV) and antagonist hamstrings (channel 2; 1mm deflection = 8.5μV) for a test at 30°/s. (B) for agonist hamstrings (channel 1; 1mm deflection = 83.3μV) and antagonist quadriceps (channel 2; 1mm deflection = 4.6μV) for a test at 90°/s. Channels 3 and 4 in both A & B recorded force and angle respectively. Templates used to identify the angles at which EMG activity measurements were taken are also shown.


MS 5 15 25 35 45 55
ML 55 45 35 25 15 5
30°/sec

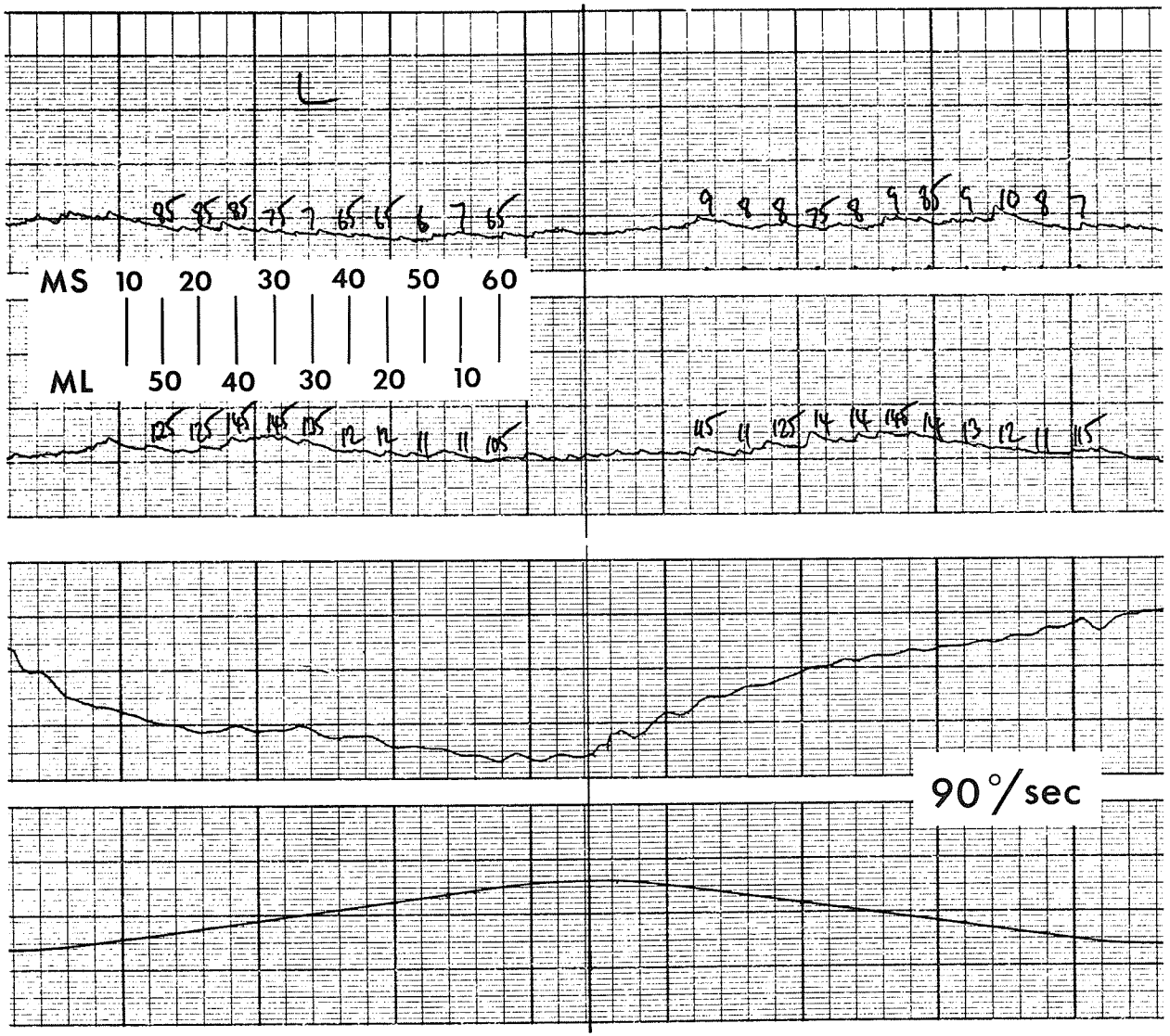

MS 10 20 30 40 50 60
ML 50 40 30 20 10
90°/sec

Figure 4.1 A graph of %Max EMG activity for antagonist hamstrings shortening (closed circles) and lengthening (open circles) at 30 degrees/second measured within a constant velocity range of 15-70° (moment-arm angle). The significant effects of contraction type (ms versus ml: $p=.003$) on activity, and significant interaction between angle and contraction type ($p<.001$) are shown.

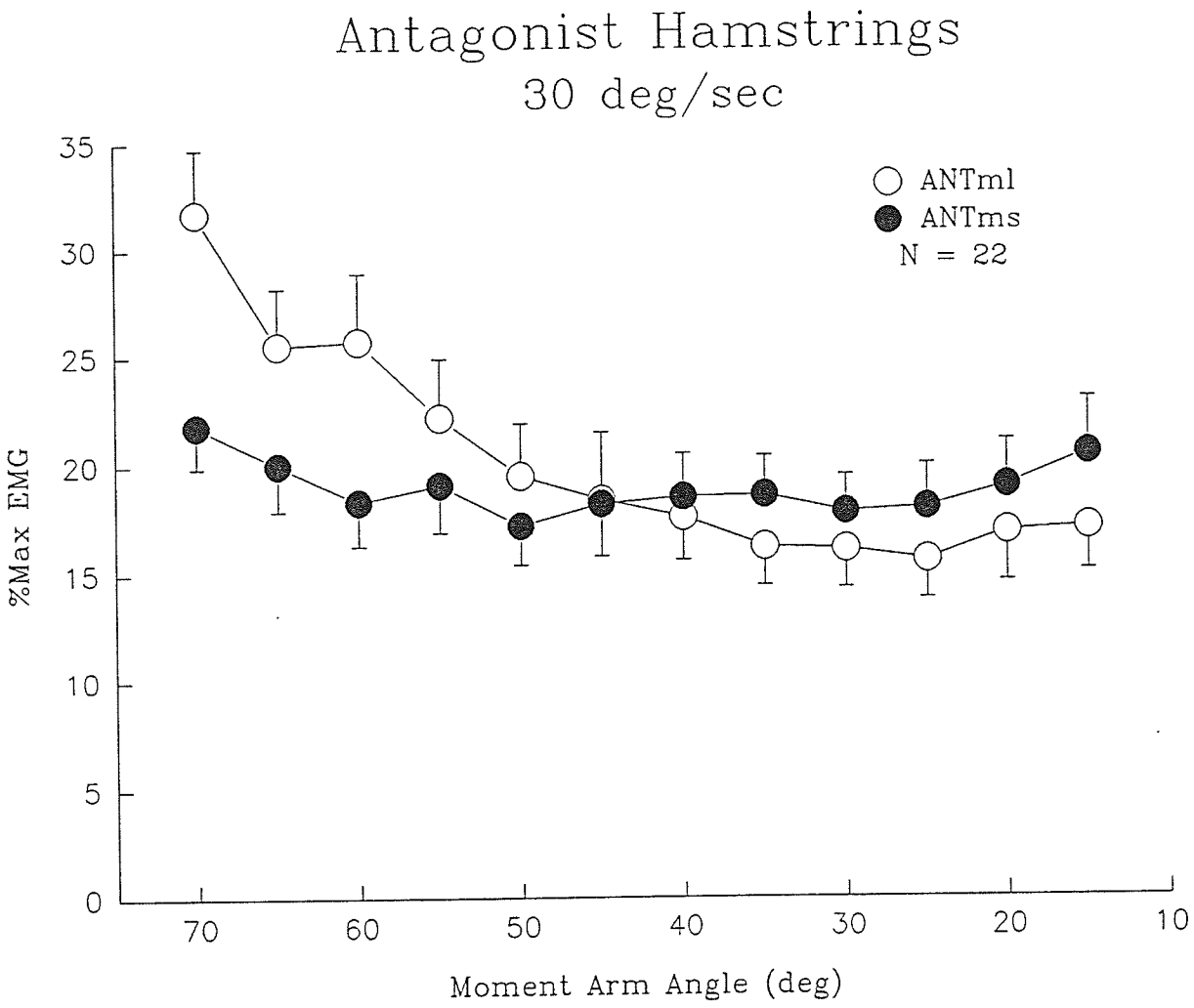

Antagonist Hamstrings
30 deg/sec
ANTml
ANTms
N = 22
%Max EMG
35
30
25
20
15
10
5
0
70
60
50
40
30
20
10
Moment Arm Angle (deg)

Figure 4.2. A graph of %Max EMG activity for antagonist hamstrings shortening (closed circles) and lengthening (open circles) at 90 degrees/second measured within a constant velocity range of 25-65° (moment-arm angle). The significant effects of contraction type (ms versus ml: $p<.001$) on activity, and significant interaction between angle and contraction type ($p<.001$) are shown.

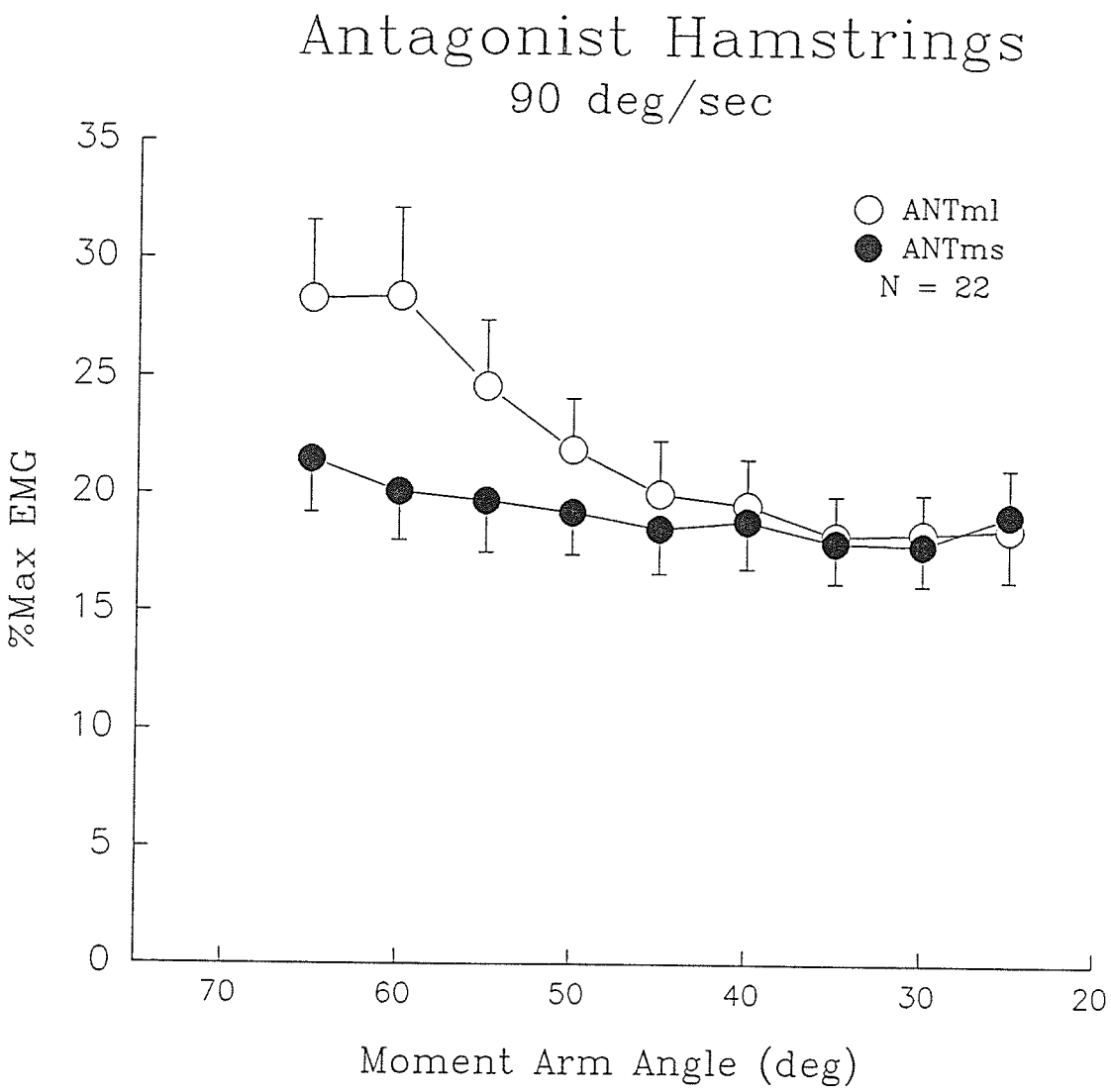

Antagonist Hamstrings
90 deg/sec
ANTml
ANTms
N = 22
%Max EMG
35
30
25
20
15
10
5
0
70
60
50
40
30
20
Moment Arm Angle (deg)

Figure 4.3. A graph of %Max EMG activity for antagonist quadriceps shortening (closed triangles) and lengthening (open triangles) at 30 degrees/second measured within a constant velocity range of 15-70° (moment-arm angle). No significant effects of angle ($p>.8$), or contraction type (ms versus ml: $p>.2$) on activity, and no significant interaction between angle and contraction type ($p=.8$) are shown.

Antagonist Quadriceps

30 deg/sec

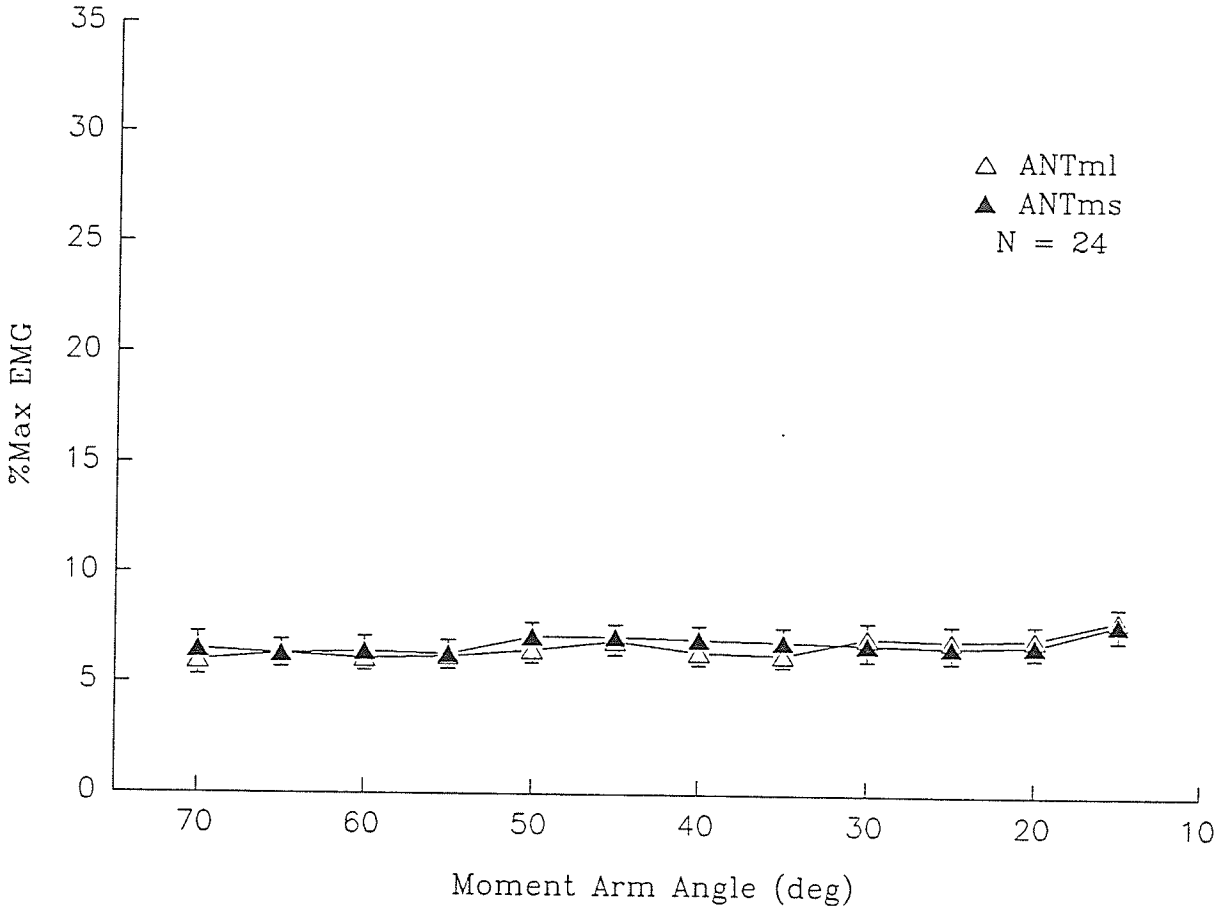


Figure 4.4. A graph of %Max EMG activity for antagonist quadriceps shortening (closed triangles) and lengthening (open triangles) at 90 degrees/second measured within a constant velocity range of 20-60° (moment-arm angle). No significant effects of angle ($p>.3$), or contraction type (ms versus ml: $p>.8$) on activity, and no significant interaction between angle and contraction type ($p>.9$) are shown.

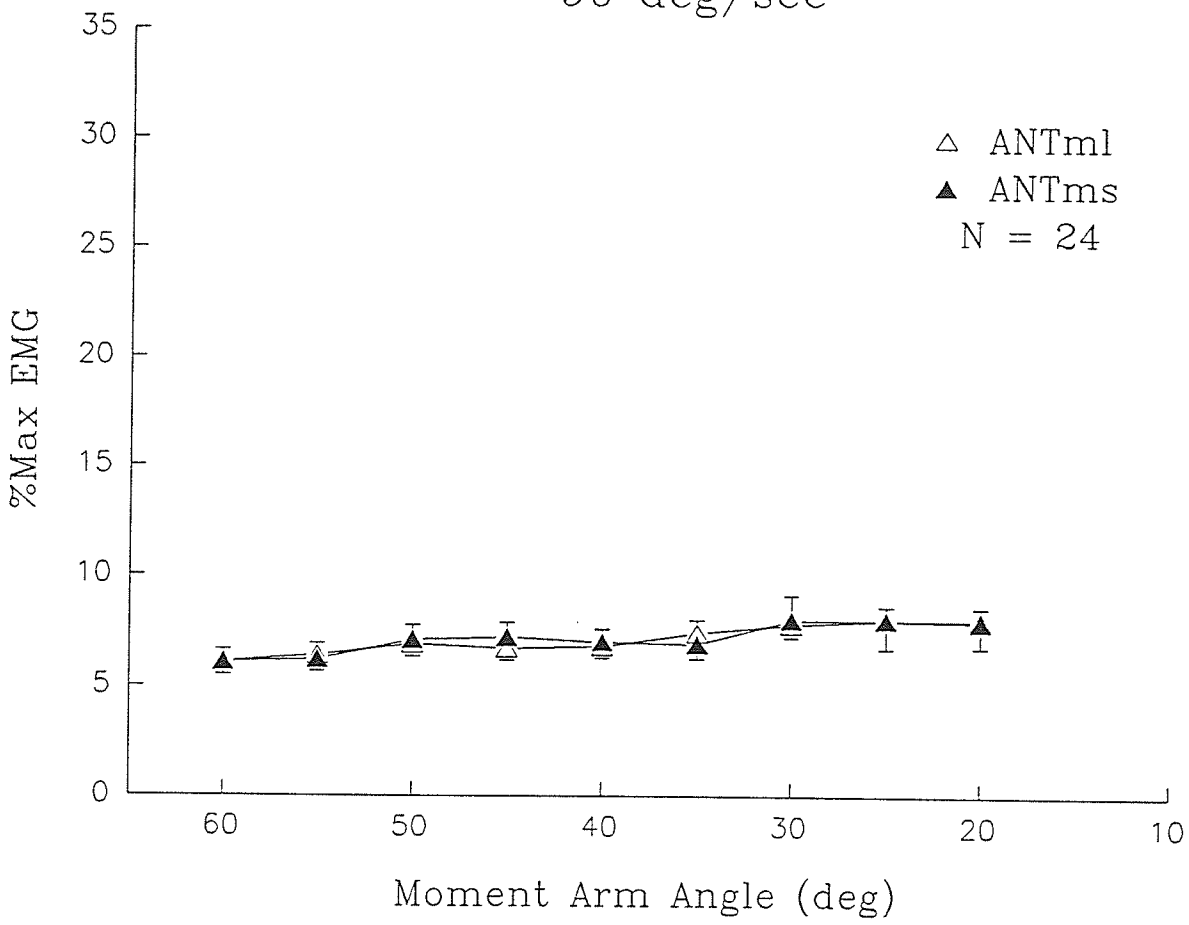

Antagonist Quadriceps
90 deg/sec
ANTml
ANTms
N = 24
%Max EMG
35
30
25
20
15
10
5
0
60
50
40
30
20
10
Moment Arm Angle (deg)

Figure 4.5. A graph of %Max EMG activity for antagonist hamstrings at 30 degrees/second (open circles) and 90 degrees/second (closed circles) during antagonist muscle lengthening measured within a constant velocity range of 15-65° (moment-arm angle). The significant effects of angle ($p<.01$) and velocity (30 versus 90: $p<.001$) on activity are shown. There is no significant interaction between angle and velocity ($p>.9$).

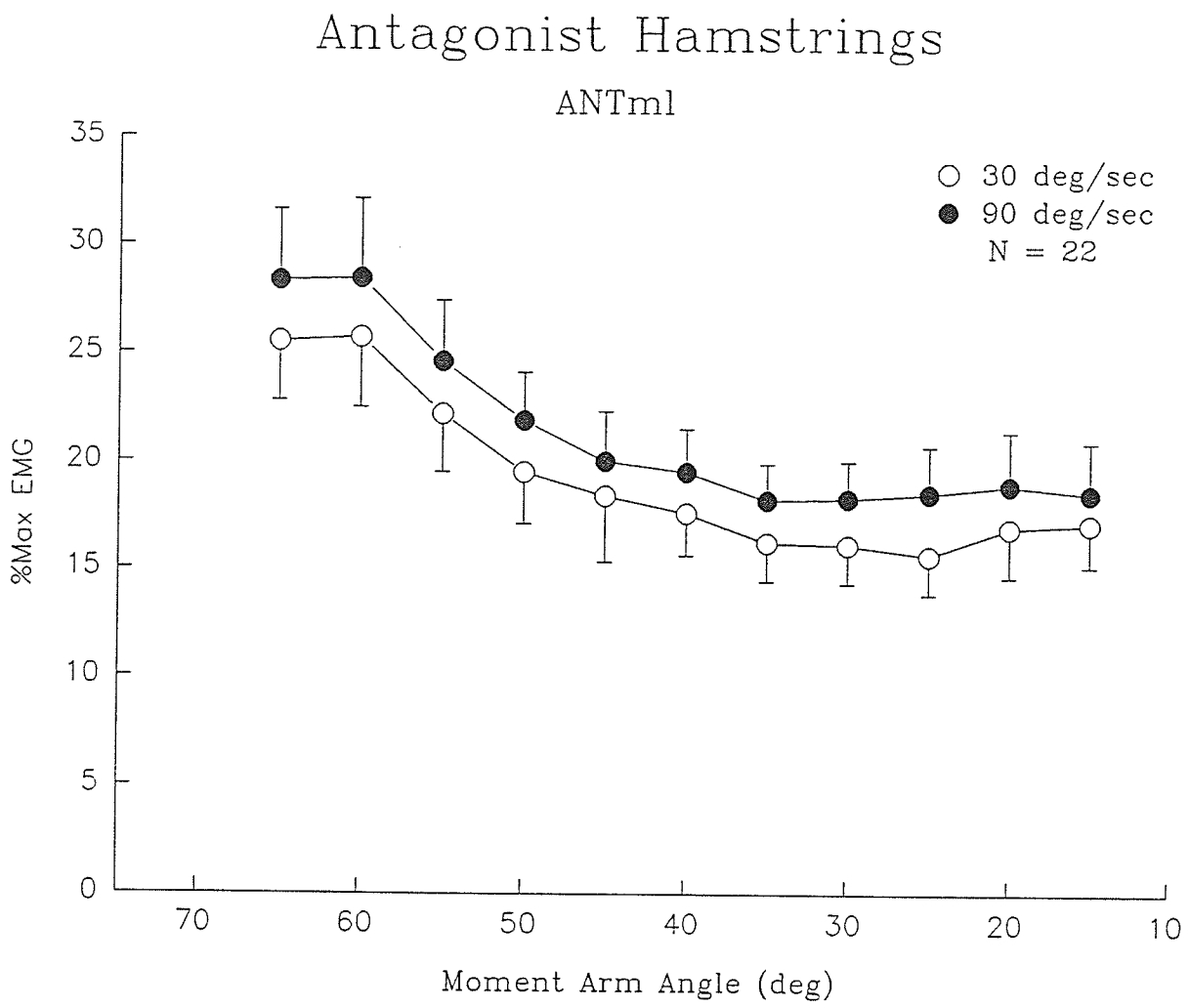

Antagonist Hamstrings
ANTml
30 deg/sec
90 deg/sec
N = 22
%Max EMG
35
30
25
20
15
10
5
0
70
60
50
40
30
20
10
Moment Arm Angle (deg)

Figure 4.6. A graph of %Max EMG activity for antagonist hamstrings at 30 degrees/second (open circles) and 90 degrees/second (closed circles) during antagonist muscle shortening measured within a constant velocity range of 25-70° (moment-arm angle). The significant effects of velocity (30 versus 90: $p<.04$) on activity are shown. There is no significant effect on angle ($p>.8$) and no significant interaction between angle and velocity ($p>.8$).

Antagonist Hamstrings

ANTms

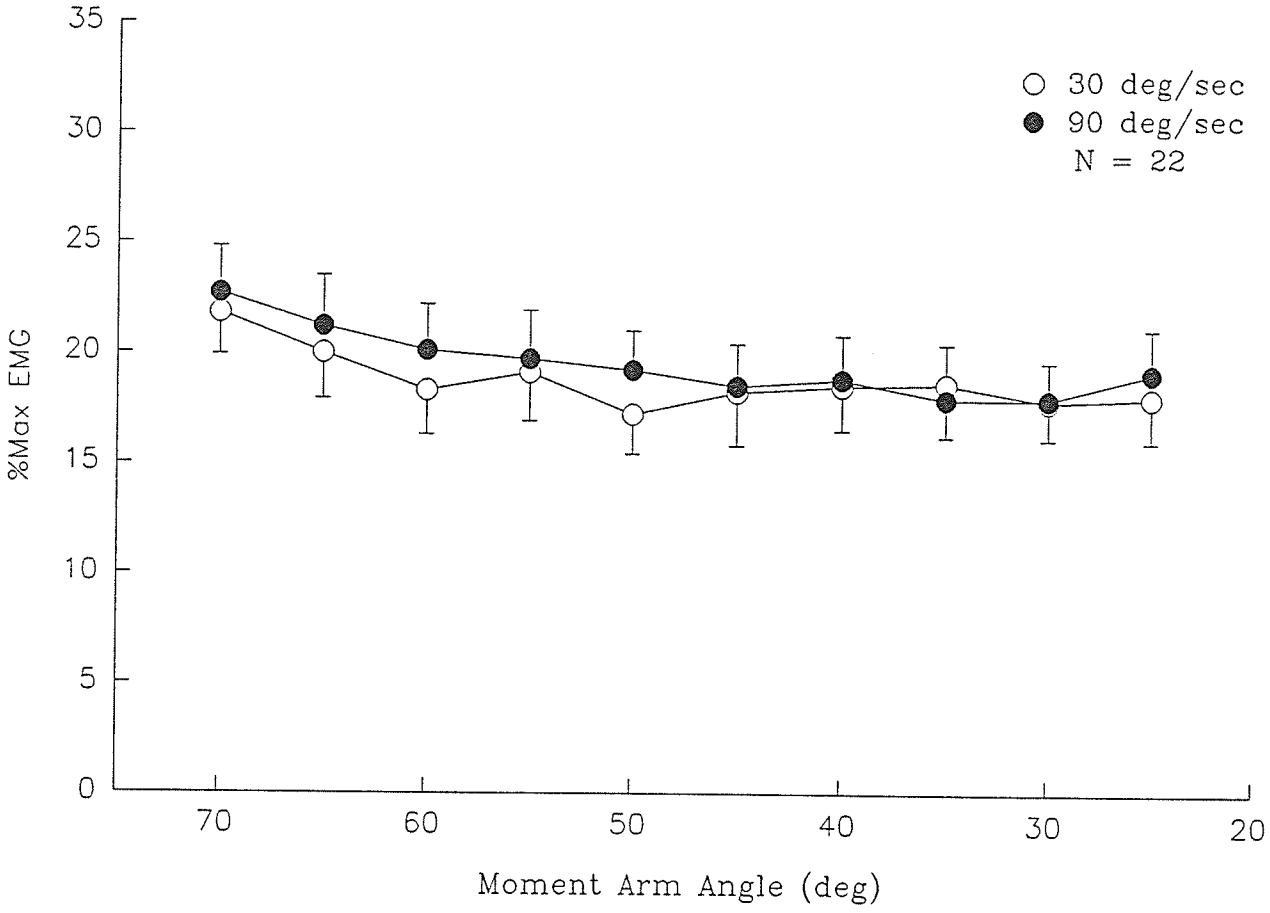


Figure 4.7. A graph of %Max EMG activity for antagonist quadriceps at 30 degrees/second (open triangles) and 90 degrees/second (closed triangles) during antagonist muscle lengthening measured within a constant velocity range of 20-70° (moment-arm angle). The significant effects of velocity (30 versus 90: $p<.02$) on activity are shown. There is no significant effect on angle ($p>.2$) and no significant interaction between angle and velocity ($p>.4$).

Antagonist Quadriceps

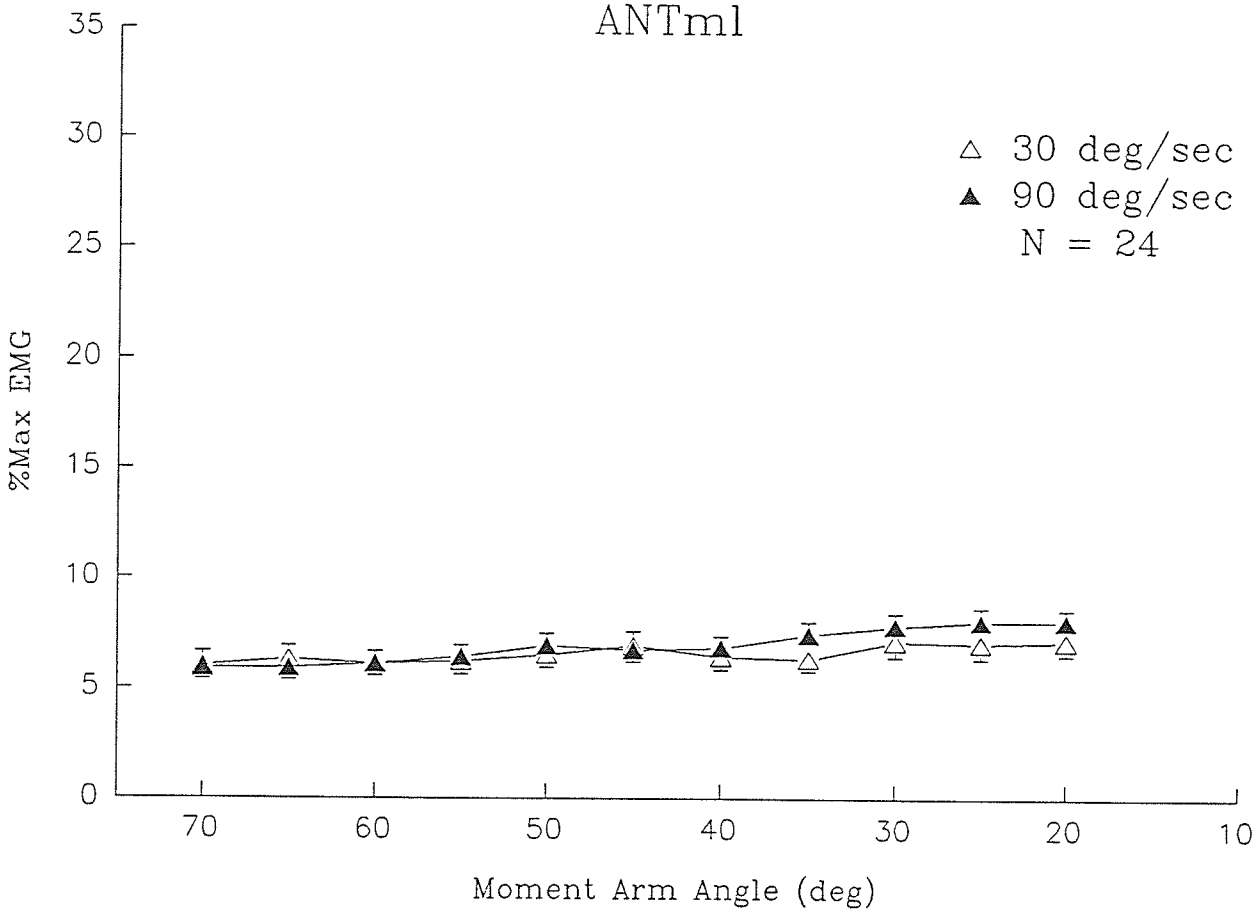


Figure 4.8. A graph of %Max EMG activity for antagonist quadriceps at 30 degrees/second (open triangles) and 90 degrees/second (closed triangles) during antagonist muscle shortening measured within a constant velocity range of 15-60° (moment-arm angle). No significant effects of angle ($p>.7$), or velocity (30 versus 90: $p>.1$) on activity, and no significant interaction between angle and velocity ($p>.8$) are shown.

Antagonist Quadriceps

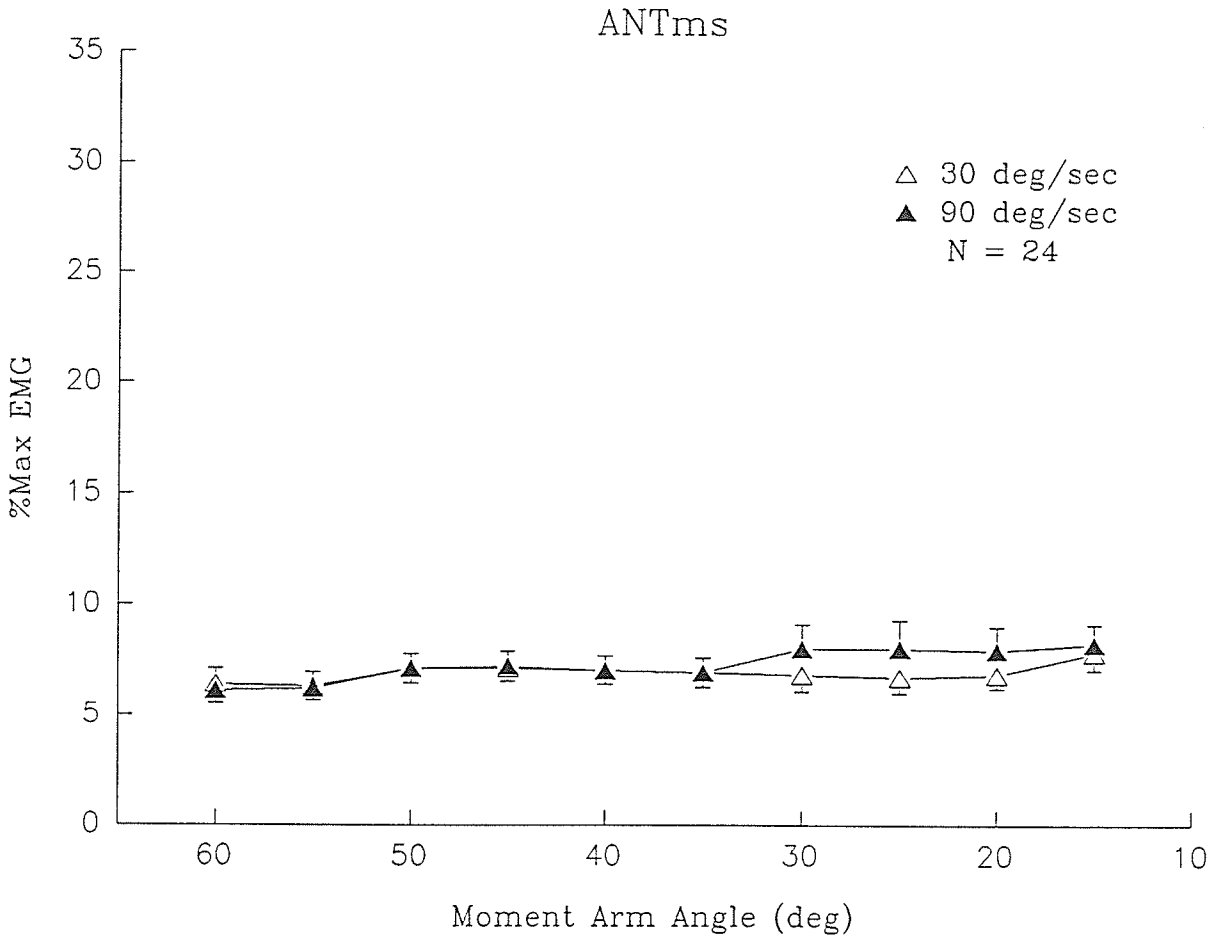


Table 4.1: Subject sample data of sex, and means and ranges of age, height and weight.

	Age(yrs)	Height(cm)	Weight(kg)
Males (n=12)	23.4 (21 - 31)	175.0 (165.0 - 188.0)	74.1 (58.5 - 99.0)
Females (n=12)	24.3 (21 - 31)	164.3 (151.1 - 182.9)	58.3 (47.8 - 68.0)

Table 4.2: Mean absolute peak torques (±SD) and mean peak torque (±SD) normalized to body weight (BW) separated by sex for quadriceps (Q) and hamstring (H) agonist muscle shortening (ms) and lengthening (ml) at each test velocity (30 and 90) °/s.

Males (n=12)	Mean Peak Torque (N·m)	Normalized Peak Torque (N·m/kgBW)
Q90ms	229.25 ± 34.91	3.11 ± 0.30
Q30ms	251.67 ± 39.06	3.42 ± 0.39
Q30ml	279.42 ± 40.39	3.84 ± 0.65
Q90ml	289.92 ± 37.32	3.95 ± 0.44
H90ms	111.50 ± 21.78	1.51 ± 0.23
H30ms	122.67 ± 18.80	1.67 ± 0.24
H30ml	132.33 ± 20.60	1.81 ± 0.27
H90ml	135.92 ± 19.78	1.86 ± 0.28
Females (n=12)		
Q90ms	148.17 ± 24.70	2.54 ± 0.29
Q30ms	167.67 ± 27.55	2.88 ± 0.37
Q30ml	199.25 ± 34.99	3.43 ± 0.54
Q90ml	207.92 ± 33.18	3.58 ± 0.48
H90ms	75.42 ± 11.78	1.30 ± 0.21
H30ms	82.17 ± 11.23	1.42 ± 0.19
H30ml	89.83 ± 12.60	1.55 ± 0.24
H90ml	91.92 ± 13.18	1.59 ± 0.24

Table 4.3: Data for %Max EMG activity of antagonist hamstrings (n=22) during constant velocity (30°/s) muscle lengthening (ml) and muscle shortening (ms).

Angle	Contraction Type	Mean ± SEM
70	ml	.317 ± .030
	ms	.217 ± .019
65	ml	.255 ± .027
	ms	.200 ± .021
60	ml	.257 ± .032
	ms	.183 ± .020
55	ml	.222 ± .027
	ms	.191 ± .022
50	ml	.195 ± .024
	ms	.172 ± .018
45	ml	.184 ± .031
	ms	.182 ± .024
40	ml	.175 ± .020
	ms	.184 ± .020
35	ml	.162 ± .018
	ms	.186 ± .018
30	ml	.161 ± .018
	ms	.178 ± .017
25	ml	.156 ± .018
	ms	.180 ± .020
20	ml	.169 ± .023
	ms	.190 ± .021
15	ml	.171 ± .020
	ms	.205 ± .025

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	11	1.916	.038
Error	252		
Contraction Type	1	8.859	.003
Interaction	11	8.389	<.0001
Error	252		

Table 4.4: Data for %Max EMG activity of antagonist hamstrings (n=22) during constant velocity (90°/s) muscle lengthening (ml) and muscle shortening (ms).

Angle	Contraction Type	Mean ± SEM
65	ml	.283 ± .033
	ms	.212 ± .023
60	ml	.284 ± .037
	ms	.201 ± .021
55	ml	.246 ± .028
	ms	.197 ± .022
50	ml	.219 ± .022
	ms	.192 ± .018
45	ml	.200 ± .023
	ms	.185 ± .019
40	ml	.195 ± .020
	ms	.188 ± .020
35	ml	.182 ± .017
	ms	.179 ± .017
30	ml	.183 ± .017
	ms	.179 ± .017
25	ml	.185 ± .022
	ms	.191 ± .020

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	8	1.45	.18
Error	189		
Contraction Type	1	33.51	<.0001
Interaction	8	4.93	<.0001
Error	189		

Table 4.5: Data for %Max EMG activity of antagonist quadriceps (n=24) during constant velocity (30°/s) muscle lengthening (ml) and muscle shortening (ms).

Angle	Contraction Type	Mean ± SEM
70	ml	.060 ± .007
	ms	.065 ± .008
65	ml	.063 ± .006
	ms	.063 ± .006
60	ml	.061 ± .006
	ms	.064 ± .007
55	ml	.062 ± .006
	ms	.063 ± .006
50	ml	.065 ± .006
	ms	.071 ± .007
45	ml	.070 ± .006
	ms	.071 ± .006
40	ml	.064 ± .006
	ms	.070 ± .006
35	ml	.063 ± .006
	ms	.070 ± .007
30	ml	.071 ± .007
	ms	.069 ± .008
25	ml	.070 ± .007
	ms	.067 ± .007
20	ml	.071 ± .006
	ms	.068 ± .006
15	ml	.080 ± .006
	ms	.078 ± .008

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	11	.608	.82
Error	276		
Contraction Type	1	1.232	.27
Interaction	11	.652	.78
Error	276		

Table 4.6: Data for %Max EMG activity of antagonist quadriceps (n=24) during constant velocity (90°/s) muscle lengthening (ml) and muscle shortening (ms).

Angle	Contraction Type	Mean ± SEM
60	ml	.061 ± .005
	ms	.061 ± .006
55	ml	.064 ± .005
	ms	.062 ± .005
50	ml	.069 ± .005
	ms	.071 ± .007
45	ml	.067 ± .005
	ms	.072 ± .007
40	ml	.068 ± .005
	ms	.070 ± .007
35	ml	.074 ± .006
	ms	.069 ± .007
30	ml	.078 ± .006
	ms	.080 ± .011
25	ml	.080 ± .006
	ms	.080 ± .013
20	ml	.080 ± .005
	ms	.079 ± .011

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	8	1.175	.32
Error	207		
Contraction Type	1	.050	.82
Interaction	8	.168	.99
Error	207		

Table 4.7: Data for %Max EMG activity of antagonist hamstrings (n=22) during muscle lengthening at two constant velocities (30°/s, 90°/s).

Angle	Contraction Velocity	Mean ± SEM
65	30	.255 ± .027
	90	.283 ± .033
60	30	.257 ± .032
	90	.284 ± .037
55	30	.222 ± .027
	90	.246 ± .028
	2	
50	30	.195 ± .024
	90	.219 ± .022
45	30	.184 ± .031
	90	.200 ± .023
40	30	.176 ± .020
	90	.195 ± .020
35	30	.162 ± .018
	90	.182 ± .017
30	30	.161 ± .018
	90	.183 ± .017
25	30	.156 ± .018
	90	.185 ± .022
20	30	.169 ± .023
	90	.189 ± .025
15	30	.171 ± .020
	90	.185 ± .024

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	10	2.54	.006
Error	231		
Contraction Velocity	1	39.83	<.0001
Interaction	10	.18	.99
Error	231		

Table 4.8: Data for %Max EMG activity of antagonist hamstrings (n=22) during muscle shortening at two constant velocities (30°/s, 90°/s).

Angle	Contraction Velocity	Mean ± SEM
70	30	.218 ± .019
	90	.227 ± .021
65	30	.200 ± .021
	90	.212 ± .023
60	30	.183 ± .020
	90	.201 ± .021
55	30	.191 ± .022
	90	.197 ± .022
50	30	.172 ± .018
	90	.192 ± .018
45	30	.182 ± .024
	90	.185 ± .019
40	30	.185 ± .020
	90	.188 ± .020
35	30	.186 ± .018
	90	.179 ± .017
30	30	.178 ± .017
	90	.179 ± .017
25	30	.180 ± .020
	90	.191 ± .020

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	9	.50	.87
Error	210		
Contraction Velocity	1	4.29	.04
Interaction	9	.47	.89
Error	210		

Table 4.9: Data for %Max EMG activity of antagonist quadriceps (n=24) during muscle lengthening at two constant velocities (30°/s, 90°/s).

Angle	Contraction Velocity	Mean ± SEM
70	30	.060 ± .007
	90	.059 ± .005
65	30	.063 ± .006
	90	.059 ± .005
60	30	.061 ± .006
	90	.061 ± .005
55	30	.062 ± .006
	90	.064 ± .005
50	30	.065 ± .006
	90	.069 ± .005
45	30	.069 ± .006
	90	.067 ± .005
40	30	.064 ± .006
	90	.068 ± .005
35	30	.063 ± .006
	90	.074 ± .006
30	30	.071 ± .007
	90	.078 ± .006
25	30	.070 ± .007
	90	.080 ± .006
20	30	.071 ± .006
	90	.080 ± .005

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	10	1.262	.25
Error	253		
Contraction Velocity	1	5.09	.02
Interaction	10	.99	.45
Error	253		

Table 4.10: Data for %Max EMG activity of antagonist quadriceps (n=24) during muscle shortening at two constant velocities (30°/s, 90°/s).

Angle	Contraction Velocity	Mean ± SEM
60	30	.064 ± .007
	90	.061 ± .006
55	30	.063 ± .006
	90	.062 ± .005
50	30	.071 ± .007
	90	.070 ± .007
45	30	.071 ± .006
	90	.072 ± .007
40	30	.070 ± .006
	90	.070 ± .007
35	30	.069 ± .007
	90	.069 ± .007
30	30	.068 ± .008
	90	.080 ± .011
25	30	.067 ± .007
	90	.080 ± .013
20	30	.068 ± .006
	90	.079 ± .011
15	30	.078 ± .008
	90	.082 ± .009

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	9	.659	.75
Error	230		
Contraction Velocity	1	1.879	.17
Interaction	9	.503	.87
Error	230		

APPENDIX A

UNIVERSITY OF MANITOBA
FACULTY COMMITTEE ON THE USE OF HUMAN SUBJECTS IN RESEARCH

NAME: Mr. Christopher Snow
Dr. Judith Anderson
Dr. A. Cooper

OUR REFERENCE: E89:16

DATE: January 31, 1989

YOUR PROJECT ENTITLED:

Temporal Patterns and Levels of Antagonist Co-Activation During Controlled-Velocity Resisted Muscle-Shortening and Resisted Muscle-Lengthening of the Knee Flexors and Extensors.

HAS BEEN APPROVED BY THE COMMITTEE AT THEIR MEETING OF:

January 23, 1989.

COMMITTEE PROVISOS OR LIMITATIONS:

Approved as per our letter dated January 31, 1989.

You will be asked at intervals for a status report. Any significant changes of the protocol should be reported to the Chairman for the Committee's consideration, in advance of implementation of such changes.

** This is for the ethics of human use only. For the logistics of performing the study, approval should be sought from the relevant institution, if required.

Sincerely yours,

J. P. Maclean, M.D.,
Chairman,
Faculty Committee on the Use of Human
Subjects in Research

JPM/ll

TELEPHONE ENQUIRIES:
788-6376 - Lorraine Lester

APPENDIX B

Subject Data Form and Health Questionnaire

Subjects's Name

Sex(M/F) Age(yrs) Height(cm) Weight(kg)

1. Do you have any neurological problems?

2. Do you have any cardiorespiratory problems?

3. Do you have any muscular problems?

4. Are you in general good health?

5. Do you have a history of injury/abnormality/surgery to your knees, hips or legs?

6. How would you describe yourself?

very fit/fit/not very fit/not fit

7. How often do you exercise?

Daily/about 3 times a week/once a week/<once a week/not at all

7a. If exercises, what kind of exercise?

8. Have you ever experienced isokinetic resistance before?

8a. If yes, what kinds? concentric/eccentric/both

9. Which leg do you prefer to kick a ball with?

10. What is your hand dominance?

APPENDIX C

Hôpital Général - St. Boniface - General Hospital
409 Tache Avenue,
WINNIPEG, MANITOBA R2H 2A6
(204) 233-8563

CONSENT FORM

The purpose of this study is to find out what thigh muscles work and to
at degree they work when a person performs maximum effort muscle contractions
flex and extend their knee under controlled-velocity conditions. The results
ll help us to better interpret routine muscle performance tests currently done
patients with musculoskeletal injuries, and they will also help in the
velopment of objective clinical tests for the measurement of spasticity and
gidity in patients with neurological disorders.

If you agree to participate in this study, you will be required to attend
e Muscle Performance Laboratory at St. Boniface General Hospital for one test
ssion which will last approximately two hours.

You will be asked to perform maximum effort contractions of your knee
exors and extensors on a machine that will control the speed of the
ntractions and also monitor the strength of your contractions while your leg
ves through a range of about 65 degrees.

Each test will require four maximum contractions of either the knee flexors
extensors. Some tests will be done at a relatively slow velocity while others
ll be done at a relatively fast velocity. A total of ~~eight~~ FOUR tests will be
quired to complete the session.

While you perform the maximum contractions, we will be monitoring the
tivity of some of your leg muscles. This is done by applying sensors to the
in which monitor the electrical signals from your contracting muscles. The
nsors are applied to the skin with tape. They are non-invasive (i.e. they do
t penetrate the skin).

Adequate warm-up procedures will be followed and instructions given to you
fore each test to minimize the risk of muscle or tendon injury during the
ximum muscle contractions. Hundreds of such tests have been performed in this
boratory over the last two years without incident. However, because these
sts are physically strenuous, you will probably experience some muscle
reness for two to three days following the tests.

You may voluntarily withdraw from the study at any time without prejudice.

bject's Signature Witness 8 March 90
Date

APPENDIX D

KIN/COM™ Load Cell Calibration

To ensure accurate recording of force, the Kin/Com™ load cell calibration was checked according to the manufacturers operating instructions before, and periodically during the course of the study. The procedure was as follows: Using the SERVICE program of the KIN/COM™ software, the exercise arm was maneuvered into a horizontal position (checked with a spirit level). The force transducer was then moved to the end of the arm and calibrated weights of 10 kg and 20 kg were hung from the transducer. The force reading on the computer was then checked with the calculated value to ensure accuracy to within ±1 N. No adjustments were necessary during the course of the study.

APPENDIX E

Subject Test Instructions

"Grasp the sides of the table, hold yourself down, lean against the backrest. When I say "push" you must push against the shin pad as hard and as fast as you can. When you reach the return angle, you will feel the machine push against your leg. Keep pushing against the pad as hard and as fast as you can while your leg returns to the start angle. During the test, I will be encouraging you to push as hard and as fast as you can by shouting "push hard, keep pushing". Are you ready?"

For the knee flexion tests the "push" was replaced with "pull".

APPENDIX F

Inter-subject variability of antagonist hamstring activity

Figure F.1. A graph of %Max EMG activity for antagonist hamstrings at 30 degrees/second for females (open circles) and males (closed circles) during antagonist muscle lengthening measured within a constant velocity range of 15-70° (moment-arm angle). The significant effects of angle ($p<.001$) and sex (male versus female: $p<.001$) on activity are shown. There is no significant interaction between angle and sex ($p>.9$).

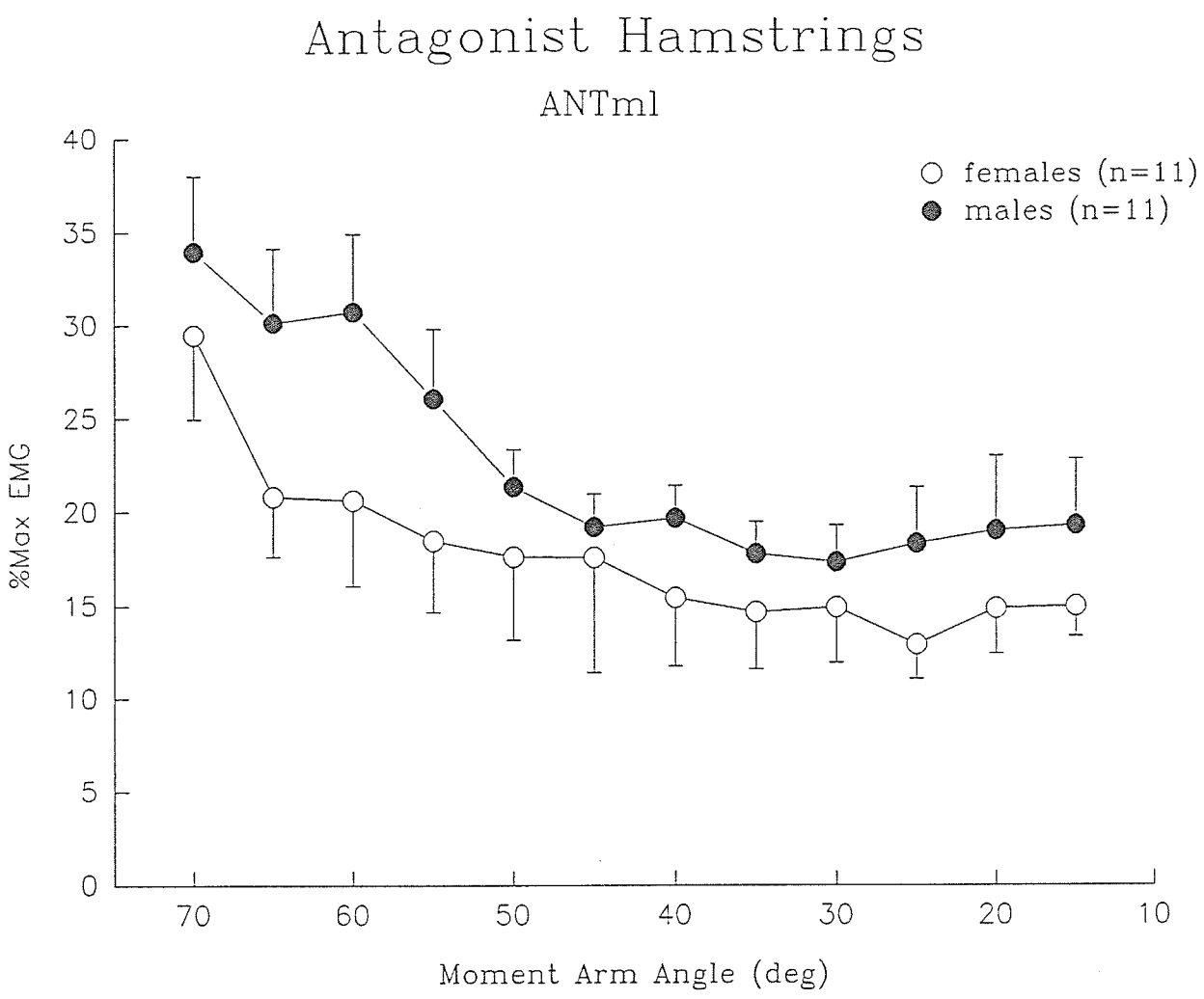

Antagonist Hamstrings
ANTml
females (n=11)
males (n=11)
%Max EMG
40
35
30
25
20
15
10
5
0
70
60
50
40
30
20
10
Moment Arm Angle (deg)

Figure F.2. A graph of %Max EMG activity for antagonist hamstrings at 30 degrees/second for females (open circles) and males (closed circles) normalized for body weight during antagonist muscle lengthening measured within a constant velocity range of 15-70° (moment-arm angle). Compared with figure F.1, the significant effect of angle remains ($p=.002$) but the normalization for body weight has eliminated the difference in activities between males and females ($p>.9$). There is no significant interaction between angle and sex ($p>.9$).

Antagonist Hamstrings

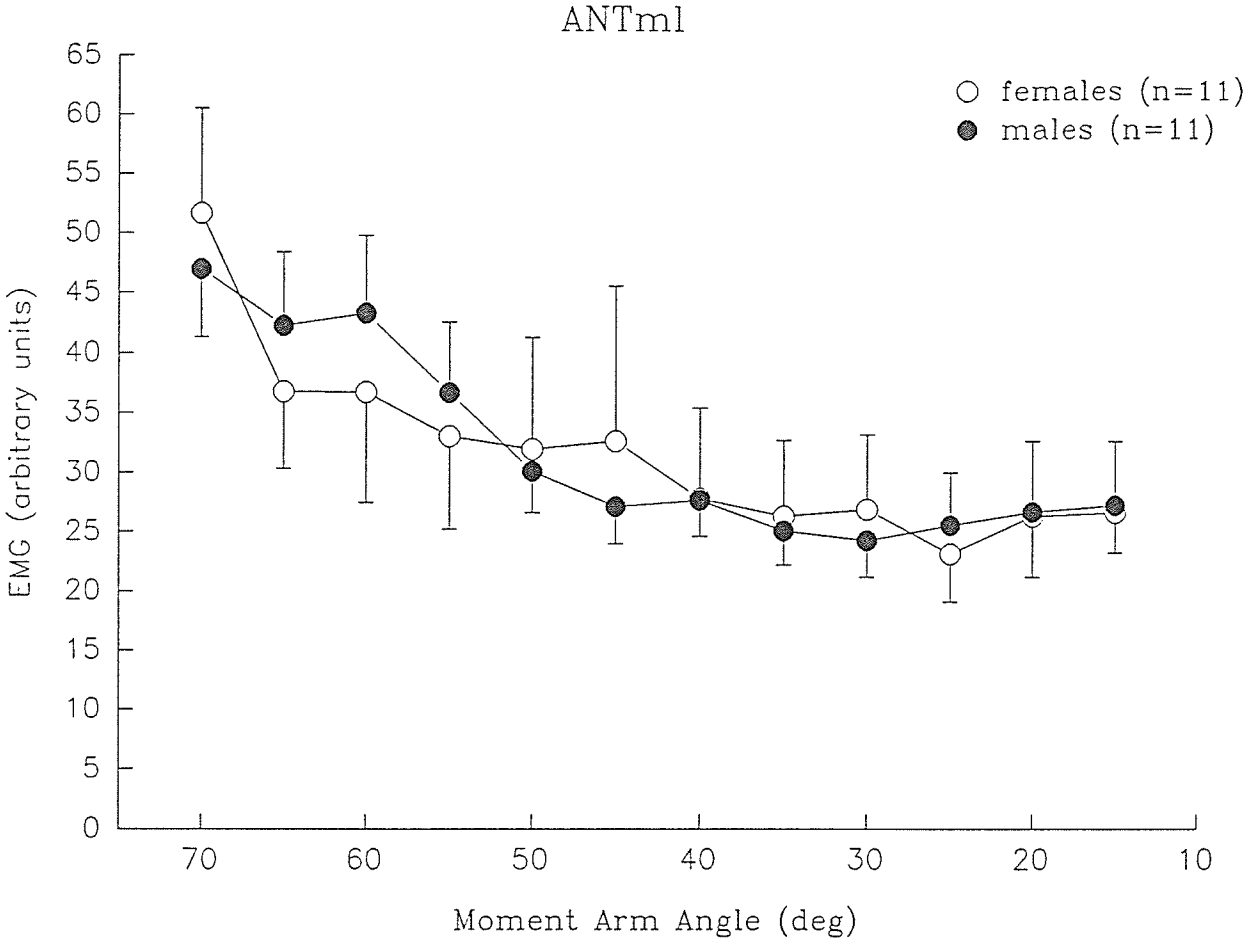


Table F.1: Data for %Max EMG activity of antagonist hamstrings during constant velocity (30°/s) muscle lengthening for males (n=11) and females (n=11).

Angle	Sex	Mean ± SEM
70	males	.340 ± .040
	females	.295 ± .045
65	males	.301 ± .040
	females	.208 ± .032
60	males	.307 ± .042
	females	.206 ± .046
55	males	.260 ± .038
	females	.185 ± .038
50	males	.213 ± .020
	females	.176 ± .044
45	males	.192 ± .018
	females	.176 ± .062
40	males	.197 ± .017
	females	.154 ± .037
35	males	.178 ± .017
	females	.147 ± .031
30	males	.173 ± .020
	females	.149 ± .030
25	males	.183 ± .030
	females	.129 ± .019
20	males	.190 ± .040
	females	.148 ± .024
15	males	.193 ± .035
	females	.150 ± .016

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	11	4.389	<.001
Error	263		
Sex	1	12.355	.001
Interaction	11	0.286	.987
Error	263		

Table F.2: Data for %Max EMG activity normalized for body weight of antagonist hamstrings during constant velocity (30°/s) muscle lengthening for males (n=11) and females (n=11).

Angle	Sex	Mean ± SEM
70	males	.469 ± .056
	females	.517 ± .088
65	males	.422 ± .062
	females	.367 ± .064
60	males	.433 ± .066
	females	.367 ± .092
55	males	.367 ± .059
	females	.330 ± .078
50	males	.300 ± .035
	females	.319 ± .094
45	males	.271 ± .031
	females	.326 ± .130
40	males	.276 ± .030
	females	.277 ± .076
35	males	.250 ± .029
	females	.263 ± .063
30	males	.242 ± .031
	females	.268 ± .063
25	males	.255 ± .045
	females	.231 ± .040
20	males	.266 ± .060
	females	.263 ± .052
15	males	.272 ± .054
	females	.265 ± .034

Summary of results from split-plot two-way Analysis of Variance on the above data.

Factor	df	F value	Significance
Angle	11	2.851	.002
Error	263		
Sex	1	0.010	.922
Interaction	11	0.172	.999
Error	263		