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Canada

Coordination of Multiple Muscles in Two Degree of Freedom Elbow
Movements

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ABSTRACT

The present study quantifies electromyographic variables in one and two degree of freedom elbow movements involving flexion / extension and pronation / supination, in order to understand the associated central commands. Agonist burst magnitude varied with motion in a second degree of freedom for some muscles but not for others. In movements for which a biarticular muscle acted as agonist in two degrees of freedom, agonist burst magnitudes were approximately the sum of the magnitudes in the component movements. Agonist burst magnitude varied with motion in a second degree of freedom for some, but not all, monoarticular muscles. When biarticular muscles acted as agonist in one degree of freedom and antagonist in the other, the muscle often displayed both components simultaneously. The additivity of EMG burst magnitudes in two degree of freedom movements and the presence of both agonist and antagonist bursts in a muscle suggest that central commands associated with motion in individual degrees of freedom are superimposed in producing two degree of freedom movements.

RÉSUMÉ

L'objet de cette étude est de quantifier des mesures électromyographiques des mouvements du coude afin d'expliciter les commandes centrales associées. Les mouvements étudiés sont à un et deux degrés de liberté et mettent en oeuvre la flexion ou l'extension et la pronation ou la supination. Les résultats montrent que pour certains muscles, l'amplitude des impulsions EMG agonistes varie avec le mouvement dans un autre degré de liberté. Dans le cadre des mouvements pour lesquels un muscle bi-articulaire se comporte comme agoniste dans les deux degrés de liberté, les amplitudes des impulsions agonistes correspondent approximativement à la somme des amplitudes des composantes du mouvement. De même, l'amplitude des impulsions agonistes varie avec le mouvement dans un second degré de liberté pour certains muscles mono-articulaires. Lorsque les muscles bi-articulaires se comportent comme agonistes dans un degré de liberté et antagonistes dans l'autre, les muscles présentent souvent les deux composantes simultanément. L'additivité des amplitudes des impulsions EMG pour les mouvements à deux degrés de liberté et la présence simultanée des impulsions agoniste et antagoniste dans un même muscle suggèrent que les commandes centrales associées à chaque mouvement à un degré de liberté se superposent pour produire des mouvements à deux degrés de liberté.

CONTRIBUTIONS TO ORIGINAL KNOWLEDGE

The methodology and results of the experiments which comprise the present thesis are, in a number of ways, unique. First, a thorough search of the literature indicates that studies involving anisometric movements in two degrees of freedom about the elbow, and their associated EMG patterns, have not been previously reported. Prior studies examining multiple degree of freedom movements and the associated electromyographic activity have focussed only on movements involving rotation about more than one joint, and isometric force production about a single joint. Second, the finding that a single muscle may display both an agonist and antagonist burst within the same movement is unique. That is, during movements in which a muscle acts as agonist to motion in one degree of freedom and antagonist to the other, both agonist and antagonist EMG components were often displayed. Previously, muscles have been reported as showing only one or the other. This may be due to the fact that two degree of freedom movements in which a muscle acts as both agonist and antagonist have not been previously examined in detail. Lastly, results of the present studies suggest that central commands for motion in individual degrees of freedom may be superimposed in the production of two degree of freedom motion. The description of empirical evidence supporting this notion is original.

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1. INTRODUCTION

The performance of arm movements requires the coordination of a number of muscles, both mono- and multi-articular, acting across a number of joints. The electromyographic (EMG) correlates of these movements have been studied extensively in the context of single joint or single degree of freedom arm motion. However, only recently have these lines of work been extended to multi-joint or multi-degree of freedom movements. The experiments which comprise this dissertation extend this line of work by examining both the magnitude and timing of electromyographic activity during elbow movements involving flexion/extension, pronation/supination, and combinations of the two. The relationships between movement kinematics and the associated EMG activity parameters are quantified under a number of different conditions. The aim is to assess the organization of commands to the elbow muscles that subserve motion in individual degrees of freedom and their combination.

In this introduction, the first section will review studies examining single joint arm movements and their associated EMG activation patterns. The second section will review findings in the analogous multiple degree of freedom situation. The goal is to provide a summary of the current state of knowledge concerning the relationships between movement kinematics and related EMG patterns. The third section will discuss ways in which central commands may

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be organized for motion in multiple degree of freedom movements and review evidence supporting these ideas. The final section will provide an overview, and discuss the aims of, the set of experiments described in this thesis.

RELATIONSHIPS BETWEEN KINEMATIC VARIABLES AND ELECTROMYOGRAPHIC ACTIVITY PATTERNS IN SINGLE JOINT HUMAN ARM MOVEMENTS

A number of studies have investigated the relationships between movement related variables (amplitude, movement time, velocity, inertial load) and parameters of the associated electromyographic signal in single joint arm movements. The goal has been to find consistent relationships between kinematic variables and associated EMG activity. In elucidating what the central nervous system views as important variables in planning goal directed movements, these relationships may allow an understanding of the underlying central commands.

Discrete, "fast", movements about a single joint are generally associated with an alternating pattern of activity in the muscles surrounding that joint. Prior to limb movement onset, agonist muscles display a burst of activity - defined as a rapid increase in activity followed by a rapid return to baseline - followed by a silent period and then a second burst of activity. During this period of inactivity in the agonist muscles there is a burst of activity in the

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antagonist muscles. The precise timing and overlap of these signals depends on, among other things, factors such as speed of movement. This "tri-phasic" burst pattern was first documented by Wachholder and Altenburger in 1926 and more recently by Hallett et al. (1975). Since then, many investigations have examined each component of the tri-phasic burst pattern and their relationship to various kinematic parameters. This section will review a number of these studies in order to provide an overview of these relationships.

Relationships between agonist burst properties and movement kinematic parameters

Early studies examined the magnitude, duration, and onset time of the first agonist burst (AG1) for horizontal plane movements involving different movement amplitudes, speeds, and inertial loads (Brown and Cooke 1981, Hallett and Marsden 1979, Lestienne 1979, Marsden et al. 1983). Small amplitude movements in the range of 65° or less were initially described. It was found that the onset time of the first agonist burst, relative to movement onset, did not vary over different movement amplitudes, durations, or inertial loads. The onset of activity generally preceded movement onset by approximately 50 milliseconds. Similarly, burst duration did not vary, lasting nearly 75 milliseconds over the range of velocities and amplitudes used in these studies (but see below). What was found to vary in a number of studies was the

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magnitude of the first agonist burst. Brown and Cooke (1981) reported a linear relationship between burst magnitude and movement amplitude in visually guided step tracking movements about the elbow. The slope of this relationship varied, however, for different "intended speeds": the slope was greatest when subjects were instructed to "move as fast as possible", less when instructed to "move fast and accurately", and less still when instructed to "move as accurately as possible". Lestienne (1979) described comparable findings in which, for elbow movements of constant amplitude and speed, faster movements were achieved through an increase in the magnitude of the first agonist burst rather than an increase in burst duration. This was also the case for constant amplitude movements performed against greater inertial loads. These findings were also reported for thumb flexions by Hallett and Marsden (1979).

Later studies, which have explored larger amplitude movements, have reported conditions under which the agonist burst duration varied (Berardelli et al. 1984, Brown and Cooke 1984). For example, when 105° elbow movements where examined, the first agonist burst duration was roughly double that seen for smaller movements. In addition, agonist burst duration increased when a large extensor load was added prior to movement onset (Berardelli et al. 1984, Benecke et al. 1985). Brown and Cooke (1984) found that EMG durations increased when elbow flexion or extension amplitudes were greater than 50°.

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Interestingly, first agonist burst durations did not increase in a continuous fashion. Instead, a second initial agonist burst component was added, roughly doubling the total burst duration from 70 ms to 140 ms. Lastly, Mustard and Lee (1987) reported prolonged (e.g. greater than 150 ms) first agonist bursts for low velocity wrist flexion movements.

While prior studies focussed on EMG variables such as burst magnitude or onset time, recently the initial rate of change in EMG activity has been examined during elbow flexions in the horizontal plane (Corcos et al. 1989; Gottlieb et al. 1989a,b). In one set of experiments, movement speed was explicitly controlled (Corcos et al. 1989) while in a second set it was not (Gottlieb et al. 1989a). Movement speeds were controlled by either instructing the subjects to move at a particular rate, or by changing the size of the target (Fitt's Law stipulates that greater accuracy constraints will lead to slower movement speeds). In both situations movements were made under different conditions of load and distance. The aim was to formulate rules relating regularities among task, kinematic, and EMG variables.

It was observed that the initial rise in EMG activity did not vary over different distances or loads when movement speed was not controlled. Similarly, the rate of torque development during the accelerative phase of the movement did not vary for the different movements when speed was not

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controlled. However, a number of variables such as peak inertial torque and integrated EMG activity were correlated with distance and load. In movements in which movement speed was controlled, the rates of both joint torque development and rise in agonist EMG activity were greater for faster movements.

The authors suggested that a control signal consisting of a pulse of fixed amplitude (but variable duration) could lead to uniform excitation of the motoneuronal pool thereby causing the observed invariant rise in torque and EMG activity. A pulse of variable intensity would be associated with a variable increase in EMG activity and, subsequently, joint torque. It was proposed that movements, in which speed is not a controlled variable, are executed by a pulse of fixed intensity and variable duration. For movements performed at controlled speeds the amplitude of the excitation pulses to motoneurons would be modulated while it's duration would be held constant. One drawback to such a control scheme is the implicit assumption that EMG parameters are controlled variables. Descending systems, however, cannot unambiguously specify EMG activity due to, among other things, the influence of the peripheral system on motoneuron pools (Adamovich et al. 1989). A number of other issues that arise with such a control scheme have been discussed elsewhere (Gottlieb et al. 1989b).

Considerably fewer findings have been reported for the second agonist burst (AG2). This is because this burst is small, variable and thus difficult to systematically quantify. Studies which were able tu examine properties of the second agonist burst, both in movements about the elbow (Brown and Cooke 1981) and the wrist (Mustard and Lee 1987), are in agreement, however. It was found that the magnitude of AG2 increased with greater movement amplitudes. The slope of this relationship varied with movement speed such that higher velocities led to greater burst magnitudes. These findings are comparable to those described for the first agonist burst. Unlike the first agonist burst, however, the onset of the second agonist burst was found to be later for larger amplitude movements and earlier for higher velocity movements. In addition, while the duration of this burst was constant within a given movement speed condition, the burst duration was shorter for faster movements (Brown and Cooke 1981).

In summary, first agonist burst was found to vary with respect to both magnitude and duration. For a given movement duration, AG1 scaled in magnitude with changes in both inertial load and movement amplitude. Intended movement speed affected the slope of the relationship between movement amplitude and burst magnitude. The duration of AG1 also increased for large movements. Only onset time with respect to movement onset remained relatively constant over different conditions of movement amplitude,

load, and peak velocity. The second agonist burst likewise displayed a positive linear relationship between movement amplitude and burst magnitude. However, its onset varied over different velocity and amplitude conditions while its duration varied with movement speed.

Antagonist burst properties in one degree of freedom movements

The kinematic parameters associated with aspects of the antagonist burst (ANT) such as its onset, magnitude, duration, and even its very appearance have also been studied at great length.

In experiments in which subjects performed movements against a mechanical stop, the antagonist burst was not present (Marsden et al. 1983, Benecke et al. 1985, Mustard and Lee 1987). Nor was it present during low velocity (0.5 rad/s for a loaded limb, 3.5 rad/s for an unloaded limb) movements (Lestienne 1979, Maton et al. 1980). However, an increase in an opposing inertial load at low velocity could induce the appearance of the burst. On the basis of this finding, it was suggested that the force at which the antagonist burst would appear was equal to the estimated passive force in the antagonist elbow muscles (approximately 35 Newtons). Thus if the force required to brake the movement was less than the visco-elastic force provided by the antagonist muscle, the antagonist burst would not be displayed

(Lestienne 1979).

It has been shown that the magnitude of the antagonist burst increased with movement peak velocity in horizontal movements about the elbow (Lestienne 1979, Brown and Cooke 1981, Marsden et al. 1983, Karst and Hasan 1987) and wrist (Mustard and Lee 1987). The slope of this relationship increased with greater inertial loads (Brown and Cooke 1981, Karst and Hasan 1987).

Studies relating the magnitude of the antagonist burst to movement amplitude have been more contradictory. Earlier studies suggested that the antagonist burst magnitude was invariant over different movement amplitudes · for the elbow (Brown and Cooke 1981) and thumb (Hallett and Marsden 1979). Others report an increase in magnitude with a greater elbow flexion amplitude (Gielen et al. 1985, Mustard and Lee 1987). Still others report a decrease in antagonist burst magnitude with a greater amplitude thumb flexion, elbow extension (Marsden et al. 1983), and elbow flexion (Benecke et al. 1985). It should be noted that because of the interdependence of movement amplitude, peak velocity, and movement time, some of these contradictory findings may be due to variations in actual testing conditions under which antagonist burst magnitude was examined. The use of different initial joint configurations in different studies may also have affected the variables examined.

Karst and Hasan (1987) examined the magnitude of the antagonist burst over a variety of elbow flexion amplitudes, speeds, and inertial loads. The aim was, in light of previous contradictory findings, to relate ANT1 magnitude to kinematic and loading parameters valid over a wide range of conditions. It was found that the ANT1 magnitude could be related to a simple algebraic expression,

$$ANT1 = f(IV^2 / A) \qquad\qquad (1.1)$$

This expression contained terms for total moment of inertia (I), peak velocity (V) and movement amplitude (A). Antagonist burst magnitude was related linearly to expression 1.1 over different conditions of load, velocity, and amplitude and remained linear over a 500-fold range of EMG magnitudes. Note that expression 1.1 has the same dimensional units as torque, thus suggesting that it could represent the net torque necessary to brake the movement. To test this hypothesis, Karst and Hasan applied an external torque to certain flexion movements to assist in braking. It was found that the decrease in antagonist activity was less than would be expected had the sole function of this burst been the provision of a braking torque for the movement. It was suggested that the excess torque may play a role in increasing joint stiffness. This would presumably assist in the precise control of the termination phase of the movement.

Antagonist burst onset (relative to AG1 onset) was found to vary under

different experimental conditions. Some studies found that the time of antagonist burst onset was related to peak velocity, but was not load or amplitude dependent (Lestienne 1979, Maton et al. 1980). Others report a complex relationship between burst onset and both movement amplitude and velocity such that onset was later for large slow movements and earlier for small fast movements (Marsden et al. 1983, Brown and Cooke 1981, Benecke et al. 1985, Mustard and Lee 1987). When movement time was explicitly controlled, the antagonist burst onset time remained constant with respect to movement onset over different amplitudes and velocities. This suggested that a major role for the antagonist burst was regulating movement time, a notion supported by simulation studies (Wierzbicka et al. 1986, Wierzbicka and Wiegner 1992).

The antagonist burst duration was seen to remain constant for a given movement speed instruction across movement amplitudes for the elbow (Benecke et al. 1985, Gielen et al. 1985), wrist (Mustard and Lee 1987), and thumb (Hallett and Marsden 1979). The duration of the antagonist burst decreased when subjects were instructed to move more quickly, however (Brown and Cooke 1981).

In summary, the antagonist burst demonstrated a variability in its magnitude, onset, and duration over variations in kinematic parameters. The

magnitude was found to be linearly related to an expression incorporating load, velocity, and amplitude terms and having units of torque. The burst onset was constant for a specific movement time over movements differing in amplitude and inertial load. Antagonist burst duration was constant over different amplitudes for a particular speed instruction, and decreased when subjects were instructed to move more quickly.

The relationship between components of the triphasic burst pattern and the temporal profile of the movement

Recent studies have consolidated previous findings by relating properties of EMG activity to movement acceleration and deceleration (Hoffman and Strick 1990, Brown and Cooke 1990, Cooke and Brown 1990).

Hoffman and Strick (1990) examined the first agonist and antagonist bursts in step-tracking wrist flexions over different conditions of movement amplitude and intended speed. It was found that, over the different movement conditions, the magnitude of the first agonist burst varied directly with the amplitude of initial peak acceleration. The magnitude of the antagonist burst was highly correlated with the reciprocal of movement duration. It was suggested that the antagonist burst magnitude might therefore also be correlated with the reciprocal of acceleration duration (although there was no

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direct evidence provided for this expectation). The authors propose that the nervous system modulates the magnitude of the first agonist burst to control the *amplitude* of a derivative of displacement (e.g., acceleration, jerk, etc.). They further proposed that nervous system modulated the magnitude of the antagonist burst to control the *duration* of a derivative of displacement.

Brown and Cooke (1990) have used a movement paradigm which allowed control over the temporal structure of a step-tracking elbow movement. Through use of a velocity-position tracking procedure, subjects were able to simultaneously maintain a constant movement duration, amplitude, and peak velocity while varying the amount of time spent in accelerative and decelerative phases of a movement. This allowed an examination of the behaviour of the triphasic burst pattern components with respect to movement acceleration and deceleration. It was seen that characteristics of the triphasic burst pattern were modified for movements having different temporal profiles. Magnitude and duration of agonist and antagonist bursts varied with the ratio of acceleration duration to deceleration duration (termed the "symmetry ratio"). For greater symmetry ratios, the first agonist burst increased in duration and decreased in magnitude. The opposite pattern was observed for the second agonist burst. For the antagonist burst, larger symmetry ratios were associated with later activity onset and a greater burst magnitude. The duration of the antagonist burst remained constant. Thus it was the desired acceleration / deceleration

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characteristics of the movement that were associated with the EMG activity pattern, independent of parameters such as movement amplitude, duration, or velocity.

In a companion study, Cooke and Brown (1990) again utilized the velocity-position tracking paradigm to alter the temporal characteristics of elbow movements. In this study, subjects performed elbow flexions and extensions in which they accelerated, maintained a constant velocity, then decelerated. It was found that the acceleration phase was associated with an initial agonist burst and a later antagonist burst. EMG activity during the constant velocity phase consisted of tonic activity in the agonist muscle. During the deceleration phase there was an initial antagonist burst followed by a second agonist burst. That is, subjects displayed a quadraphasic burst pattern. The first agonist burst accelerated the limb, the first antagonist burst slowed the acceleration, the second antagonist burst decelerated the limb, and the second agonist burst stopped the deceleration.

Normally, goal directed movements are performed with symmetrical, bell-shaped velocity profiles such that the reversal of limb acceleration and the initiation of limb deceleration are combined smoothly (Soechting and Laquaniti 1981). Findings from the study by Cooke and Brown (1990) illustrate how the antagonist burst seen in the "classic" triphasic burst pattern may actually be

separable into two components which normally are merged. In fact, when the temporal profile of the movement was altered such that the amount of time spent moving at a constant velocity was systematically reduced to zero, the time between the two antagonist bursts decreased until they finally became one.

These results support the idea that the triphasic burst pattern is not a reflection of the basic unit of movement control. Rather, agonist / antagonist burst pairs more closely reflect the underlying control signals (Cooke and Brown 1990).

In summary, electromyographic activity associated with motion about a single joint is often in the form of a triphasic burst pattern. A great deal of research has examined correlations between various movement related variables and EMG parameters. While many findings were consistent over a large number of experimental manipulations (for example, the timing of the agonist burst onset), many other relationships remained unclear. Recent studies suggest that the basic form of EMG activity associated with single joint movements may in fact be an agonist / antagonist burst pair. Temporal aspects of the movement, such as the amount of time spent accelerating or decelerating the limb, appear to be related to the timing and magnitude of these burst pair components.

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Relationships Between Kinematic Variables and Electromyographic Activity Patterns in Multiple Degree of Freedom Human Arm Movements

In shifting from a single joint to a multi-joint system, a number of complexities arise with respect to movement control. First, there is the issue of the mechanical redundancy of the human arm. The number of axes about which a joint can rotate is referred to as the number of degrees of freedom at that joint. The human shoulder, elbow, and wrist joints possess seven degrees of freedom (three at the shoulder, two at the elbow, two at the wrist). However, the location and orientation of any object in space can be fully specified with only six coordinates: three positions and three orientations. Thus, the arm has one "extra" degree of freedom. While this is advantageous in that it provides flexibility, from a control aspect it is problematic as there are many possible trajectories for reaching the object.

A similar situation occurs at the muscular level. There are thirty-three muscles acting across the shoulder, elbow, and wrist (Hasan et al. 1985). Theoretically, only fourteen are required (an agonist and antagonist about each degree of freedom). Again, while this arrangement provides flexibility, a given level of force at a single joint may be achieved through various combinations of muscle activation. In addition, a number of muscles are multi-articular and consequently produce force in more than one degree of freedom. Therefore,

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there is no one-to-one mapping between individual muscle actions and kinematic degrees of freedom. In dealing with redundancy at both the joint and muscular level, the nervous system is thus faced with the task of reducing indeterminancy without compromising adaptability.

One final problem has to do with the mechanics of multi-joint movement. Motion of any one segment, through inter-segmental dynamics, will lead to motion in other segments. That is, motion at more than one joint also produces interaction torques not found in single joint movements. Thus in planning a multi-joint movement the nervous system may have to account for the mechanical properties of the limb.

The following section will review a number of studies which have extended the single joint experiments by examining motion in more than one degree of freedom. Many of these studies have investigated the EMG correlates of motion involving rotation about more than one joint. Typically, hand movements in different directions (or isometric force production, in some cases) are made in a horizontal or vertical plane. Similar to the analyses performed in studies of motion about a single joint, relationships between EMG parameters and movement related variables are explored. In addition, the effect of varying direction of hand motion is examined.

As with single joint movements, examination of EMG activity in a multi-joint or multi-degree of freedom system may shed light onto the underlying control signals. Comparison of EMG activity between single and multi-joint arm movements may provide insight into the ways that the nervous system deals with the additional control problems mentioned above.

Movements involving rotations about more than one joint

Movements in a horizontal plane

One of the earliest descriptions of EMG patterns associated with movements about the shoulder and elbow was provided by Wadman and colleagues (1980). In their study, subjects performed rapid reaching movements of different amplitudes in the horizontal plane to targets in eight different directions. It was found that, for a particular movement direction, the muscles surrounding the joint whose amplitude of movement was greatest produced a triphasic burst pattern comparable to that seen in single joint studies. Muscles at the other joint produced what was termed "support", or tonic, activity. When a muscle displayed phasic activity, the magnitude of that activity depended more upon the direction of hand motion rather than movement amplitude in a given direction. This was true both for muscles displaying agonist bursts and those displaying antagonist bursts. Burst timing varied,

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however, with different movement amplitudes within a given hand direction: agonist burst duration increased and antagonist burst onset was delayed for larger movements. To summarize, in planar shoulder-elbow movements, muscles acting about both joints displayed activity over the whole range of directions tested. Muscles surrounding those joints with the largest movement amplitudes displayed an agonist / antagonist burst pairing like that observed for single joint movements. The remaining muscles displayed a non-phasic increase in activity. Both single and double joint muscles showed similar patterns.

Karst and Hasan (1991b) examined the timing and magnitude of EMG activity associated with shoulder / elbow movements having different initial and final positions. The goal was to characterize features of the EMG signal, such as relative timing of activity and burst magnitude, and to relate them to positional variables.

Qualitatively, EMG patterns at the individual shoulder and elbow joints displayed phasic agonist / antagonist burst pairings for some movement directions, while other hand movement directions were associated with only tonic activity at a particular joint. When phasic activity was present at both the shoulder and elbow joints, the relative timing and magnitude of EMG activity were examined in relation to a single positional variable: the difference between initial forearm orientation and the direction of hand movement, ψ. This variable

was chosen based on the findings of a previous study (Karst and Hasan 1991a) in which the selection of muscles used to initiate movement at a joint were found to be related to ψ.

It was observed that the relative agonist burst magnitude between shoulder and elbow muscles was systematically modulated as a function of ψ. Specifically, the ratio between shoulder and elbow agonist burst magnitudes was greatest when the direction of hand motion was the same as the initial forearm orientation ($\psi = 0°$ or 180°), and was smallest when the direction of hand motion was 90° away from the initial forearm orientation ($\psi = 90°$ or 270°). Between these two extremes the agonist burst magnitude ratio increased or decreased in a linear fashion with respect to ψ.

Similarly, onset of agonist activity in shoulder muscles relative to activity onset in elbow muscles varied as a function of ψ. Shoulder and elbow agonist activity onsets were nearly synchronous when ψ was either 90° or 270°, while shoulder activity preceded elbow EMG by, on average, 80 ms when hand motion direction and initial forearm orientation were the same. Again, the relative timing of agonist burst onsets between the two joints varied in linear fashion between these extremes. The onset of antagonist activity at the elbow joint was found to precede antagonist shoulder activity by approximately 40 ms. This difference did not vary with the positional variable ψ.

In summary, the appearance of paired agonist / antagonist burst activity at the shoulder and elbow for those movement directions for which joint rotations were sufficiently large (Karst and Hasan 1991b) is comparable to the results obtained by Wadman et al. (1980). This bolsters the notion of the agonist / antagonist burst pair as a fundamental feature in controlling limb movements. In addition, the relative timing and magnitude of EMG signals at the shoulder and elbow were both modulated with hand motion in different directions. Because the direction of motion was related to the position of a limb segment, the reference system for central commands coordinating muscles acting in different degrees of freedom may, to some extent, be a body centred one.

Movements in a vertical plane

Control of arm movements performed in the vertical plane is more complex than movements performed in a horizontal plane. In vertical movements, the force of gravity adds a load that varies as the arm moves throughout the workspace. Thus the nervous system may compensate for an additional force. Only a limited number of studies have examined systematically the relationship between kinematic and electromyographic variables in vertical plane multi-joint arm movements.

Flanders and Soechting (1990) examined the activation of nine muscles around the shoulder and elbow during isometric force production at the wrist in several different directions. Subjects held the arm in one of six static postures while a constant force was applied at the wrist in twenty different directions in a sagittal plane, and again in a frontal plane. In each plane the applied forces covered a full 360° range. The aim was two-fold: to provide a description of EMG activity for isometric forces in various directions, and to compare this empirical data with that predicted by a mathematical model. The model was based on the assumption that the relationship between muscle activity and force magnitude over different directions may be characterized by one or more cosine-shaped tuning curves having a maximum in one "preferred direction".

It was found that the activity levels for most muscles examined could be fit by a broadly tuned cosine function, having two peaks, which related the magnitude of EMG activity to the direction of the applied force. An exception was the single-joint elbow flexors, whose activity was characterized by a tuning curve having only a single peak. The direction of the larger peak (or only peak for those muscles displaying only one) was close to the estimated mechanical pulling direction for a given muscle, where it would be maximally effective as an agonist. The smaller peak was usually in the opposite direction, corresponding to the direction of maximally effective antagonist activity.

Activation of a muscle during force production in a direction opposite to that muscle's direction of maximal effectiveness would produce coactivation of antagonist muscles and thereby stiffen the joint. Figure 1.1 presents an example of a cosine tuning function containing two peaks. The axes correspond to force direction while the distance from the centre represents the magnitude of EMG activity. For the hypothetical muscle depicted, an upward force produced at the wrist (counteracting a downward applied force) is associated with maximal muscle activation. A force of the same magnitude produced in an upward and forward direction is associated with less EMG activity, a force in a forward direction is related to no activity, etc. A second, smaller peak in EMG magnitude can be seen for the same magnitude of isometric force produced in a downward direction.

Similar findings were reported for EMG activity recorded during isometric force production in a horizontal plane (Miller et al. 1992). Subjects held the arm horizontally while counteracting a given amount of force applied in different directions about the wrist. Both mono- and bi-articular muscles displayed a variation in muscle activity with force direction that could be fit by a single circular function passing through an origin corresponding to the hand position. The direction of maximal activity (corresponding to the point on the circle opposite to the hand position) varied for the different muscles examined.
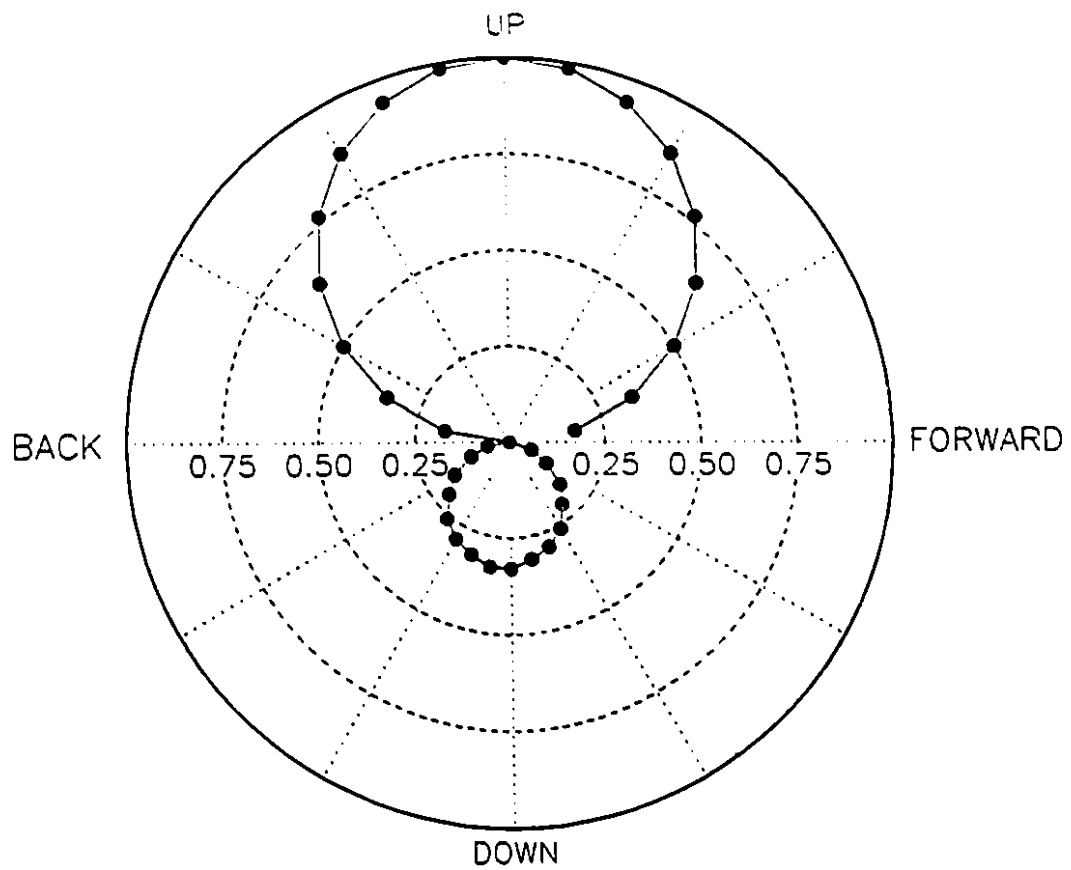
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Figure 1.1. Hypothetical cosine tuning function displaying two peaks. Distance from the centre corresponds to the magnitude of muscle activity (see text). Axes represent direction of an applied force in a sagittal plane.

Flanders and Soechting (1990) also showed that the force direction associated with maximal EMG activity (i.e., the peak of the cosine function) did not vary with force amplitude, but did change with the different static arm postures. The change in peak activity direction paralleled the change in the mechanical line of action for a given muscle. This was cited as evidence that the nervous system takes limb position into account when producing motor commands for a single movement. That is, depending on arm posture, different levels of muscle activity must be used to produce a desired force vector.

The authors noted that a given direction of force at the wrist could have been achieved through different combinations of activation in each of the muscles examined. However, little variability in the pattern of activation was observed both within and between subjects. These patterns may represent some of the constraints employed by the nervous system in contending with the problem of muscle redundancy.

A series of studies have examined activity in multiple muscles about the shoulder and elbow during vertical plane movements in different directions (Flanders 1991, 1994), at different speeds (Flanders and Herrmann 1992, Flanders 1994, Buneo et al. 1994), and of different amplitudes (Buneo et al. 1994). In all of these experiments, a principal components (PC) analysis was employed in order to quantify the EMG signal. This analysis is useful in

quantifying the shape of a waveform. In brief, a PC analysis is similar to a Fourier analysis in that the original arbitrary signal is transformed into a smaller set of orthogonal functions, or PC waveforms, which can be ordered by degree of importance. Unlike Fourier analysis, however, the transformed components are not restricted to sinusoids but depend on the data themselves.

In all of the studies using the PC analysis to quantify the EMG signal, it was found that the muscle activity could be transformed into two separate, additive waveforms. One of the waveforms was found to be related to the phasic component of the EMG signal and comprised a curve of activation-inactivation-activation. The other was related to a tonic EMG signal and consisted of a sustained curve of activation. A number of aspects of these phasic and tonic EMG signal components were observed to vary under different conditions of movement direction, speed, and amplitude.

In each muscle examined, the phasic EMG waveform occurred in hand movements to different directions, although for certain directions it was inverted, or negated (Flanders 1991). The directions in which a particular muscle produced an inverted waveform corresponded to that region in which the muscle was mechanically an antagonist. Other directions displaying the "upright" phasic waveform were in regions of the workspace where a given muscle mechanically acted as an agonist. This is in agreement with a previous

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study (Flanders and Soechting 1990) which found regions of agonist and antagonist activity based on static force direction tuning curves. The finding of reciprocal patterns of EMG activation for movements in opposite directions is also reminiscent of Wadman et al. (1980) in which, depending on movement direction, muscles displayed agonist or antagonist activity.

It was observed that certain muscles exhibited a phasic EMG component that shifted temporally in a continuous manner as a function of movement direction (Flanders 1991, 1994). For example, anterior deltoid - a shoulder flexor - displayed phasic activity which began later and later in time relative to movement onset as direction of hand motion shifted from downward / forward to downward / backward. For other muscles, a continuous change in movement direction would be associated with agonist activity in one region and antagonist activity in the opposite region. That is, as movement direction was gradually shifted, the muscle would display phasic activity which switched abruptly from the timing of an agonist to the timing of an antagonist (Flanders 1994). These different temporal patterns were observed across muscles, even those spanning the same joint. Thus it was demonstrated that EMG activity associated with multi-joint reaching movements are not simply reciprocal alternating burst patterns that are fixed with respect to movement onset.

The magnitude of the phasic EMG signal component was observed to

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increase with an increase in velocity when movement distance and direction were held constant (Flanders and Herrmann 1992). However, movements of a greater distance, for a given velocity and direction, were associated with smaller phasic component magnitudes (Buneo et al. 1994). In both situations, the magnitude of phasic EMG activity increased with shorter movement times, suggesting that the magnitude of activation may be related to movement time rather than movement speed.

Buneo et al. (1994) developed a technique which allowed the duration of the phasic EMG component to be quantified. It was found that the duration of the phasic EMG component increased for greater movement distances. This relationship was constant across subjects for a given muscle. However, the increase in duration for a given increase in movement distance varied for the different muscles examined. The authors suggested that this differential scaling may be related to differences in the line of action of the various muscles involved. Indeed, for the movement direction studied, the muscle which scaled the duration of its phasic EMG component the most was that muscle in an anatomical position to generate the most torque.

The tonic waveform of the EMG signal was found to remain constant in magnitude for different movement times in a single direction (Flanders and Herrmann 1992). However, an increase in magnitude was observed for greater

movement distances (Buneo et al. 1994). It was demonstrated that the level of activity in the tonic waveform was comparable to the EMG levels observed when the arm was held in a series of corresponding static postures against gravity. The authors concluded that the tonic waveform approximated the postural, or gravitational, component of the arm movements (Flanders and Herrmann 1992, Buneo et al. 1994).

To summarize, EMG activity associated with multi-joint movements in different directions could be characterized by two additive waveforms: a phasic component and a tonic component. The onset of the phasic waveform relative to movement onset varies for movements to different directions. The magnitude of this phasic component was greater for shorter movement times, while its duration increased for greater movement distances. The tonic EMG waveform did not vary with movements of varying speeds but did increase with movement distance. This tonic component was associated with the EMG activity necessary to counteract gravity.

A direct comparison of findings between two-joint studies made in the horizonal and vertical planes is difficult. This is due to the variation in both the methods of EMG quantification and the kinematic parameters examined. Nevertheless, a number of similarities and differences are worth noting. When isometric force production in different directions was studied, the magnitude

of EMG activity varied in a systematic fashion, with each muscle displaying a particular direction of maximal activation. In the horizontal plane, the variation in magnitude was fit by a circular function relating the magnitude of EMG activity to direction of isometric force production (Miller et al, 1992), while in the vertical plane magnitude was fit with a cosine function often having two peaks in opposite directions (Flanders and Soechting 1990). EMG magnitude also varied for movements of different directions in non-isometric horizontal movements (Wadman et al, 1980, Karst and Hasan 1991b), but a search of the literature has been unable to reveal an equivalent study of EMG magnitudes in the vertical plane. Temporal variations across muscles, for movements in different directions, were observed in both the horizontal (Karst and Hasan 1991b) and vertical planes (Flanders 1991). In addition, temporal relations were also found to vary for different speeds (Flanders and Herrmann 1992) and distances (Buneo et al. 1994). (Analogous studies for movements in the horizontal plane have not been done.)

Because aspects of the different classes of studies varied, it is difficult to ascribe differences in findings to the effects of changing from a horizontal to a vertical plane. It is reasonable to assume certain differences, such as the coactivation observed in vertical plane isometric contractions (manifested as the second cosine function peak-Flanders and Soechting 1990) and partitioning of the EMG signal into phasic and tonic components (Flanders and Herrmann

1992) may result from the addition of gravity and its accompanying dynamic effects on a multi-segmented system.

Isometric contractions in two degrees of freedom about the elbow

A number of studies have investigated movements involving isometric force production in two degrees of freedom at the elbow. These studies have examined the overall EMG magnitude for torques exerted in a number of directions (Buchanan et al. 1986), and the behaviour of both individual motor units (van Zuylen et al. 1988, Riek and Bawa 1992, Jones et al. 1993) and overall EMG activity (Jamison and Caldwell 1993, Caldwell et al. 1993) during force production in two degrees of freedom about a single joint. The findings in general are three-fold. Distinct motor unit sub-populations whose recruitment thresholds depend on motion in two degrees of freedom have been reported. Torques in one degree of freedom have been found to affect the magnitude of the EMG signal during simultaneous torques in a second degree of freedom. In addition, synergistic relationships between muscles also change with motion in a second degree of freedom.

Van Zuylen et al. (1988) recorded the activity of individual motor units in a number of elbow muscles while subjects produced flexion/extension and pronation/supination torques, both separately and in combination. They found

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that, on the basis of motor unit recruitment thresholds, individual muscles were not activated homogeneously. Rather, subpopulations of motor units were identified whose recruitment thresholds for the torque in one degree of freedom varied with the torque exerted in a second. As an example, for biceps brachii, recruitment thresholds for flexion torques decreased during the simultaneous production of a supination torque. There were also motor units in these muscles whose activity was not modulated by torque exerted in a second degree of freedom. For example, biceps brachii also contained single degree of freedom units. Motor unit sub-populations defined on the basis of motion in two degrees of freedom were reported in biceps brachii, triceps brachii (all three heads), brachialis, brachioradialis and pronator teres. It is interesting to note that some of these muscles are monoarticular (e.g., brachioradialis, brachialis, triceps lateral head), and thus biomechanically only contribute to motion in a single degree of freedom. Nevertheless, they have been shown to contain motor unit subpopulations affected by torque exerted in a degree of freedom for which they have no mechanical action.

Van Zuylen et al. (1988) also observed that the recruitment threshold of motor units was affected by the elbow angle at which isometric contractions were performed. For all of the elbow flexor muscles, it was found that motor unit recruitment threshold decreased as elbow angle, and therefore muscle length, increased. Since the mechanical advantage of these muscles is less as

the elbow is more extended (due to moment arm changes), a greater amount of muscle activation would be required to produce a given amount of torque. The authors suggest that the observed lowering of recruitment threshold acts as the mechanism to compensate for this decrease in mechanical advantage. It should be noted that a length-dependent facilitation to the motoneuron pool may account for this as well.

While the existence of task-specific motor unit subpopulations was observed in a majority of the muscles surrounding the elbow, studies examining wrist muscles have not found these subpopulations. Riek and Bawa (1992) examined extensor carpi radialis, a muscle active in the two degrees of freedom at the wrist. Subjects performed wrist extension (pitch) and radial deviation (yaw) while motor unit activity was recorded from 15 to 30 different sites in the muscle. It was found that every motor unit that fired during extension also fired during radial deviation. It was concluded that motion in one degree of freedom was associated with the activation of the same group of motoneurons as motion in the second degree of freedom.

Similarly, Jones et al. (1993) examined the behaviour of flexor carpi ulnaris during isometric wrist flexion and ulnar deviation. All 238 motor units examined (from five subjects) were recruited for both tasks. Thus there was no evidence for subpopulations active for movements in one degree of freedom

only. It should be noted however, that motor unit thresholds were not obtained during simultaneous wrist flexion and ulnar deviation. Thus the existence of subpopulations whose recruitment thresholds vary with torque in a second degree of freedom cannot be ruled out by these studies.

In addition to studies which have examined individual motor units, studies have been reported in which isometric torque production is related to overall EMG activity.

Buchanan et al. (1986) examined the behaviour of a number of muscles about the elbow during isometric contractions in the flexion / extension and varus / valgus (the motion resulting from humeral rotation) directions. Subjects held their upper arms horizontally with the elbow flexed 90°. Torques of varying amounts were exerted in eight to ten different directions in a sagittal plane while intramuscular EMG activity was recorded from seven muscles.

Individual muscles' activation patterns displayed a number of characteristics. With an increase in exerted torque there was a linear increase in EMG magnitude within a given direction. However, the maximum EMG magnitude varied with direction. The range of directions a particular muscle was active was often approximately 180°, indicating it was only active for those directions for which it could contribute to joint-torque production. As an

example, triceps brachii (medial head) displayed a large EMG magnitude in an extension / valgus direction which steadily decreased for torques in other directions. This muscle displayed activity over a 225° range. It was noted that the direction of maximum EMG activation corresponded to the direction in which the moment arm, and hence the mechanical advantage, was greatest for that muscle. However, it is unclear to what extent the observed changes in torque are due to changes in moment arm alone. These findings are similar to those of Flanders and Soechting (1990) for isometric force production in different directions involving both the elbow and the shoulder. That is, muscle activity could be described by a tuning function relating the level of activity to the direction of force production. The direction of maximal EMG activity corresponded to the direction in which a muscle had the greatest mechanical advantage.

EMG activity recorded using surface electrodes was examined for elbow flexion and pronation / supination. In one study (Caldwell et al. 1993), the magnitude and median power frequency of the EMG signals from two muscles were compared in different tasks: flexion alone, supination alone, and flexion combined with supination. It was observed that biceps brachii magnitude (both heads) during a maximum voluntary contraction was greater for the combined flexion / supination compared to either flexion or supination alone. This indicated that the task specificity observed in motor units at low torque level

(van Zuylen et al. 1988) was also present at much higher torque level. The median power frequency of the EMG signal did not change over the different tasks. This frequency has been shown to increase upon recruitment of larger motor units having greater conduction velocities. The authors suggest that lack of any shift between force tasks provides evidence that two degree of freedom contractions arise from the activation of separate subpopulations of motor units, rather than the recruitment of larger units in an already active motoneuron pool.

In a similar study, Jamison and Caldwell (1993) examined the magnitude of EMG activity during a maximal flexion contraction while subjects simultaneously exerted torque in a pronation or supination direction. Torques in the pronation and supination direction were found to affect the magnitude of the EMG signal during a maximum isometric flexion torque for biceps brachii and brachioradialis, but not for triceps brachii. In both heads of biceps brachii, combined flexion / supination torque enhanced EMG magnitude relative to flexion alone, while flexion / pronation reduced it. The enhanced bicep activity during flexion / supination is consistent with the findings of van Zuylen et al. (1988) concerning task-specific motor unit subpopulations.

While individual muscles' activation patterns varied with motion in a second degree of freedom, synergistic relationships between muscles also

changed. Jamison and Caldwell report that biceps brachii activity increased during a combined flexion / supination torque and decreased during a flexion / pronation torque. Brachioradialis displayed the opposite pattern; its activity increased during a flexion / pronation torque. This is presumably to compensate for the reduced biceps brachii contribution. It is interesting to note that the magnitude of brachioradialis activity was affected by a pronation / supination torque, since it is a monoarticular muscle which exerts torque primarily in the flexion / extension direction. The finding that synergistic action varies with torque direction has also been reported by Buchanan et al. (1986) in the context of isometric torques produced simultaneously in the flexion / extension and varus / valgus directions, and Miller et al. (1992) during isometric shoulder-elbow movements.

In summary, Buchanan et al. (1986) described patterns of muscle activation for isometric contractions in two degrees of freedom about the elbow that are similar to those seen in shoulder-elbow studies, both in the vertical (Flanders and Soechting 1990) and horizontal planes (Wadman et al. 1980, Miller et al. 1992). That is, EMG activity varied systematically with a continuous change in force or movement direction. Van Zuylen et. al. (1988) have observed motor unit subpopulations whose recruitment thresholds varied during torque production in two degrees of freedom about the elbow. Other motor unit subpopulations had thresholds which were dependent on torques in

one degree of freedom only. With respect to the generalizability of this phenomenon, these subpopulations were not observed in certain muscles acting about the wrist (Riek and Bawa 1992, Jones et al. 1993). Studies examining motor unit properties in muscles surrounding other joints with multiple mechanical degrees of freedom cannot be found. Nevertheless, dependence of EMG activity, measured using surface electrodes, on motion in two degrees of freedom has been reported (Caldwell et al. 1993, Jamison and Caldwell 1993). If motor unit subpopulations are found to be a general feature of neuromuscular architecture, the activation of these different subpopulations of motor units may provide a neuroanatomical mechanism subserving motion in multiple degrees of freedom.

ORGANIZATION OF CONTROL IN MULTIPLE DEGREE OF FREEDOM TASKS

Performance of a complex task using a multiple degree of freedom limb involves the coordination of a number of segments. For example, when catching an object one must move the hand to the proper position, orient the hand to align it with the objects' surface, and time the grasp to coincide with object contact. All of these actions must follow a strict spatio-temporal sequence for the catch to be successful. The commands to the degrees of freedom associated with, in the catching example, reaching, orienting, and grasping may be specified through parallel neural channels. A number of lines

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of evidence suggest that commands for motion in different degrees of freedom may be specified independently and issued in parallel. That is, control may be at the level of the systems' degrees of freedom. In this thesis, the phrase "control at the level of degrees of freedom" indicates that the nervous system is controlling motion in individual mechanical degrees of freedom. This may be contrasted with, for example, control of motion at the hand level or at the individual muscle level.

The first part of this section will review studies supporting the existence of central commands organized in parallel for multiple degree of freedom movements. A majority of the studies will be concerned with various aspects of arm movements, although control of motion in the multi-degree of freedom jaw will be considered as well. In the second part of this section, the issue of coordinating control signals for multi-degree of freedom motion will be discussed. Specifically, the notion that parallel central commands for individual degrees of freedom may be superimposed will be explored.

Evidence for the existence of independently specified central commands in the control of multiple degree of freedom movements

The performance of a complex task requires the superposition of a number of motor acts, as discussed above. Independent specification of the

control signals dealing with different aspects of a movement may be beneficial. In general, any system operating in a parallel fashion will process information in a more efficient manner. In addition, damage to any one area will not provoke a complete breakdown of the system. From a control system point of view, parallel processing would impart greater efficiency and adaptability.

One approach to investigating whether control signals for motion in individual degrees of freedom are specified separately is to examine the kinematics of movements in a variety of movement conditions. A change in kinematics for motion in one degree of freedom achieved independently from motion in a second degree of freedom implies associated control signals may themselves be independent. Studies investigating the possibility of parallel control signals for jaw orientation and position, reaching and grasping, and reaching and hand orientation have all taken this approach.

Ostry and Munhall (1994) examined the kinematics of jaw motion during speech and mastication. Like the arm, the jaw is an example of a multi-muscle system with multiple degrees of freedom. During speech and mastication, the jaw both rotates and translates in a sagittal plane. Because many of the muscles acting about the jaw have multiple mechanical actions, central commands to individual muscles must be coordinated so that translation or rotation may be produced alone. An analysis of jaw motion paths in the joint

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coordinates of rotation and horizontal translation was performed in order to gain insight into the organization and coordination of control signals for jaw movements.

When examining motion in joint coordinates (each coordinate representing an individual kinematic degree of freedom), straight line paths were observed. These straight line paths were maintained over different conditions of speech (e.g. different utterances, volumes) and mastication (e.g. bolus size, chewing rate). Different speech sounds resulted in changes in the slope of the motion paths. In addition, paths associated with speech of varying volumes displayed different intercepts. Alterations in the slopes of these motion paths indicated that there was a change in the rate of motion in one degree of freedom relative to the other. Changes in intercept, regardless of slope, suggested that position in each degree of freedom could be specified separately. That is, these results suggest that the central nervous system specifies control signals to jaw orientation and horizontal position separately.

Different components of goal directed arm movements may similarly be organized independently and executed in parallel. In a pair of experiments, Paulignan et al. (1991a,b) examined the kinematics of movements having reach and grasp components. In one study (1991a), the position of the object was altered after movement onset. In this situation, the subject would have to reach

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to the new target, but the grasp would remain the same. In a second study (1991b), the size of the object was altered once movement had begun. For this manipulation, the grasp component would have to be altered but not the reach component. The authors proposed that, if control of reaching and prehension were mediated by separate channels, one would see the kinematics of the grasp component unaffected by a change in object position, and the kinematics of the reach component unaffected by a change in object size.

It was observed that the kinematics of the transport phase of the movement, represented by the wrist trajectory, were indeed unaffected by a shift in object size. However, the kinematics of the grasp phase, represented by the thumb and index finger trajectories, were altered by a shift in object location. In control trials (where neither object size nor position were altered) both the aperture between thumb and index finger and the wrist trajectory increased and then decreased in a unimodal fashion. In trials where the size of the object changed, a corrective grasp movement began only after the wrist trajectory was nearly completed. In trials where object position was altered, corrective wrist movement began almost immediately and the grasp aperture decreased then increased again. That is, the timing between the two components was such that an altered reaching movement affected the grasp portion, but an altered grasp component did not affect the reaching movement. It was suggested that channels coordinating reach and grasp were separate and

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operated using different time constants. It was further suggested that the visuomotor channel coordinating the reach movement component may process information more quickly than the channel processing grasp movements. The slower "grasp channel" would constrain the timing of the faster "reach channel" during a perturbation to object size but not vice versa.

In a similar manner, Lacquaniti and Soechting (1982) examined the kinematics of motion at the wrist, elbow, and shoulder during reaching movements involving a rotation of the forearm (or wrist). Motion paths in joint coordinates were examined to see if motion of the distal arm segment affected the more proximal joints.

Motion in the shoulder and elbow joints was tightly coupled for reaching movements in which the forearm did not rotate (i.e. pronate or supinate). Note that in the task used, forearm rotation and wrist motion are equivalent and the two terms will be used interchangeably. This coupling manifested itself as a straight line in velocity space during movement deceleration. That is, when the velocity of shoulder motion was plotted against the velocity of elbow motion, the trajectories converged onto a straight line during the deceleration phase of the movement. This relationship held for reaches to different directions and at various speeds. When reaching movements involving forearm rotation were performed, this invariant characteristic of shoulder and elbow motion persisted.

In addition, fixed relations observed for shoulder and elbow motion were not found between either shoulder and wrist or elbow and wrist. Lastly, unlike shoulder and elbow motions, wrist motion was more variable in timing and duration, and there was a greater amount of inter-trial variability. These data imply that wrist motion is functionally decoupled from elbow and shoulder motion, which are coupled to each other. Forearm rotation is inertially decoupled from the elbow and shoulder. That is, a flexion torque produced at the elbow joint will not rotate the forearm (although it will produce a torque at the shoulder joint). The argument was made that wrist motion could be controlled separately from elbow and shoulder motion given that there is no need for synchronous timing between reaching and rotation. The variable duration and timing seem to support this. It was noted, however, that while they may be controlled separately, the existence of biarticular muscles acting in both degrees of freedom precludes their independence.

Clinical evidence for a dissociation between reaching and hand orientation was reported in patients with unilateral posterior parietal lesions (Perenin and Vighetto 1988). Patients were asked to reach forward and move their hand through a slot cut out of a board placed in front of them. Thus the task required reaching accurately for a target while rotating the forearm to a specific position so that the hand could slide through the slot.

When reaching for the slotted board, three types of errors were observed in all ten patients tested. For the first type of error, the patient would reach correctly for the hole, but the orientation of the hand would not be appropriate to allow passage through the slot. In the second, patients would orient the hand correctly, but the reach would not be to the slot. The third type of error consisted of both a spatial and orientation error. These spatial errors were usually fairly small and corrected, while the orientation errors could be large and went uncorrected. Patients did not display any global motor, proprioceptive or visual field deficits, indicating that this lesion was affecting a junction between visual and motor domains. Moreover, it could affect reaching and orientation separately, supporting the existence of distinct processing channels for these two movement components.

In summary, studies examining the control signals for multi-degree of freedom movements about jaw and arm joints have supported the notion that control is organized in terms of individual degrees of freedom and that the central commands for motion in these degrees of freedom are specified in parallel. While the laboratory studies have utilized a comparison of kinematics for each component degree of freedom, a clinical study has revealed deficits in actual movement performance specific for orientation versus reaching motion.

Superposition of commands for motion in different degrees of freedom

In light of evidence supporting separate specification of commands for individual degrees of freedom, it is of interest to consider how these control signals may be organized to produce the desired movements. Because, as mentioned previously, a number of muscles are multi-articular, production of motion in individual degrees of freedom cannot be achieved by simply activating individual muscles. Therefore one must consider how the nervous system coordinates central commands in light of this rather complex mapping.

One possibility is that commands for individual degrees of freedom are simply superimposed. In this situation, multi-articular muscles may display activity appropriate for motion in each component degree of freedom. In addition, characteristics of the overall activity patterns seen for one degree of freedom movements would be retained in composite multi-degree of freedom movements. (The patterns, of course, may not directly superimpose due to the non-linear relations between control signals, EMG activity, and force production).

Alternatively, the control signals may interact in a highly non-linear manner. That is, a larger signal associated with motion in one degree of freedom could cancel out a lesser signal associated with motion in a second. Multi-articular muscles would not reflect activity displayed for motion in individual degrees of freedom. Moreover, control signals could be combined in

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a more complex weighted manner. When going from motion in one degree of freedom to motion in multiple degrees of freedom, relationships between EMG and kinematic patterns would again be difficult to quantify in any straightforward manner.

If, as suggested, control is organized at the level of degrees of freedom there must exist a mapping between control signals at that level and control signals at the level of commands to individual muscles. Previous work on isometric two degree of freedom elbow movements may provide insight into the nature of this mapping (Van Zuylen et al. 1988). Control at the level of individual degrees of freedom may be mapped onto control at the level of individual muscles using these sub-populations of motor units as a neuro-anatomical substrate. To date, the only evidence of additivity of control signals at an electromyographic level has come from a study by Caldwell et al. (1992). In their study, evidence was presented that a larger magnitude EMG signal associated with torques exerted simultaneously in two degrees of freedom resulted from the activation of separate motor unit subpopulations, themselves active in the component individual degrees of freedom.

Although the goal of this thesis is to infer control on the basis of kinematics and EMG parameters, it should be kept in mind that movement kinematic patterns and their associated EMG activity provide indirect measures

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of descending control signals. These patterns, which are readily measurable experimentally, offer a window onto control, but presumably neither are themselves controlled variables: EMG varies with load and with the position of the limb while kinematic patterns reflect a combination of dynamics, muscle mechanical properties, reflexes and central commands. Nonetheless, by elucidating the relationships between EMG patterns and the resulting movement kinematics, inferences may be drawn concerning the form of central commands underlying these EMG patterns. That is, with an appropriate consideration given to the segmental and neuromuscular systems, and their relation to measured EMG signals, one may gain insight into the underlying control signals.

OVERVIEW AND AIM OF THE PRESENT STUDIES

The electromyographic correlates of arm movements have been studied extensively both in the context of single joint and, more recently, multi-joint or multi-degree of freedom movements. Studies of the relationship between muscle activity and movement kinematics in multi-degree of freedom movements have examined muscle activity during isometric force production in two degree of freedom movements about a single joint and during isotonic movements involving rotations about more than one joint. Both types of studies have shown that parameters associated with EMG activity may be dependent on motion in more than one degree of freedom. The present set of experiments

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extends this line of work by examining both the magnitude and timing of electromyographic activity during elbow movements involving flexion / extension, pronation / supination, and combinations of the two. The relationships between movement kinematics and the associated EMG activity parameters are quantified.

In the first three experiments, subjects held the arm in a sagittal plane while performing movements about the elbow in two degrees of freedom. Movements of systematically varying amplitude in each degree of freedom were performed both individually and in combination. In the first experiment, movement time was not explicitly controlled, and movements were performed with the arm in two different static postures. In the second and third experiments, movement time was explicitly controlled through use of an audio metronome. In addition, a larger range of movement amplitudes was employed. Different static arm postures were used in the second and third experiments.

For all three experiments, muscles were observed to fall into two categories with respect to the magnitude of the first agonist burst. Some displayed graded activity that depended on motion in two degrees of freedom. Others displayed activity that did not depend on motion in a second degree of freedom. The timing of agonist and antagonist activity did not differ significantly, both across muscles within a movement condition, and across

movement conditions for an individual muscle.

In the final experiment presented, subjects again performed elbow movements involving various combinations of flexion / extension and pronation / supination. Patterns of muscle activity in biarticular muscles were assessed for movements in which those muscles acted as agonist in one degree of freedom and antagonist in the other. This created a situation where the biarticular muscle acted simultaneously as an agonist and an antagonist. Under these circumstances, a number of possibilities could exist concerning the activation patterns of these muscles.

One possibility was that, in cases where a biarticular muscle could be either agonist or antagonist, the muscle acts solely as one or the other. An alternate possibility, and one which was supported in this experiment, was that a biarticular muscle may display both agonist and antagonist components in its EMG pattern even within a single movement. The determinants of the magnitude of agonist and antagonist activity were explored in this situation. The movement parameters which determine the relative magnitudes of agonist or antagonist activity presumably reflect the organization of central commands determining muscle coordination. Whether the relative magnitude of the agonist or the antagonist component in these muscles was determined by the amplitude of motion in each degree of freedom was assessed. It was found, in biarticular

muscles displaying both agonist and antagonist activity in a single movement, the burst that was greater in magnitude was not directly related to the amplitude of movement in the two degrees of freedom.

The movements examined in this thesis involve articulations about the elbow joint. While two of the muscles examined (biceps brachii and triceps brachii) also contribute to motion about the shoulder joint, muscles will be designated mono- or biarticular based on their contribution to motion at the elbow joint. Specifically, biceps brachii will be referred to as biarticular because it produces both flexion / extension and pronation / supination torque about the elbow, although in the strict sense it is a triarticular muscle since it also flexes the shoulder. Similarly, triceps brachii will be referred to as a monoarticular muscle since mechanically it produces extension torque at the elbow, even though it also produces extension at the shoulder.

In brief, the present study quantified various EMG signal parameters - burst onset, magnitude, and duration - affiliated with movements in one and two degrees of freedom about the elbow. There were two main goals for these experiments: to describe the patterns of EMG activity associated with non-isometric two joint elbow movements, and to use this description in order to understand the associated neural commands subserving these movements.

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2. METHODS

Four experiments tested a variety of conditions involving motion in two degrees of freedom about the elbow. In total, nine different subjects were used, six of them participating in more than one experiment. Because each experiment varied in a number of ways, a categorization of movement conditions, subjects used, and the pertinent variables manipulated for each study is provided in Table 2.1.

SUBJECTS

Four female and five male subjects between the ages of 19 and 28 were used in the four experiments. All subjects were right handed, in good physical condition, and had no known neurological impairments. In addition, none had suffered any serious injury to the right arm or shoulder which might have affected EMG or kinematic recordings. The procedure was explained and informed consent was given prior to commencement of each experiment. All but two subjects (Subject A and F) were naïve to the purposes of the experiment.

Experiment	Description	# conditions	# subjects
1	- no explicit timing constraints - upper arm held vertically and horizontally	22	4: A - D
2	- explicit timing constraints - upper arm held vertically	32	5: A - E
3	- explicit movement timing - upper arm held horizontally	32	3: A, F, G
4	- explicit timing constraints - movements where biarticular muscles act as agonist in one degree of freedom and antagonist in the second	16	5: A, F - I

Table 2.1

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Arm position was recorded using an Optotrak optoelectronic position sensing system (Northern Digital, Inc). The pre-calibrated system consists of three linear sensors with intersecting planes of view. The system monitors the position of Infra Red Emitting Diodes (IREDs) placed on the subject. The three-dimensional marker locations are reconstructed from the recorded IRED positions, using vendor supplied software. The three dimensional coordinates were accurate to within one millimetre (mm) in each dimension. Figure 2.1 displays the distribution of IRED positions during a 1000 ms stationary marker recording. It can be seen that the variability in marker position is less than one mm in each dimension. IRED position data were stored onto a computer hard disk for off-line processing.

Electromyographic (EMG) activity patterns were recorded from muscles about the elbow using bipolar surface electrodes (Neuromuscular Research Center). The electrodes consisted of two 1 by 10 mm parallel silver bars placed 10 mm apart. The silver bars were housed in a compact, lightweight, polyurethane case which also contained a x10 pre-amplifier. The electrode output was connected to an eight channel myoelectric preamplifier which could be clipped onto the subject. The electromyographic signals were then fed into an analogue-to-digital converter (Optrotrak Data Acquisition Unit, Northern

Digital, Inc.) and stored on a computer hard disk for processing.

PROCEDURE

Experiment 1

Subjects made forearm movements in a sagittal plane which involved flexion or extension alone, pronation or supination alone, and combinations of the two. In movements involving flexion or extension alone the forearm was either fully pronated, semi-pronated, or fully supinated. The flexions started from a position 45° below the horizontal plane and ended at targets located 45° above the horizontal plane. Start and end positions were reversed for extension movements. Thus, in total there were six flexion / extension movement conditions (three initial forearm orientations x flexion or extension). The movements were performed with the upper arm vertical and then repeated with the upper arm in a horizontal plane (the arm itself remained in the sagittal plane). When the upper arm was horizontal the initial forearm orientation was either 45° out from a vertical plane or 45° behind it.

In movements involving pronation or supination alone the forearm was held either in an extended position 45° below the horizontal or in a flexed position 45° above the horizontal. The movements consisted of full and half (to

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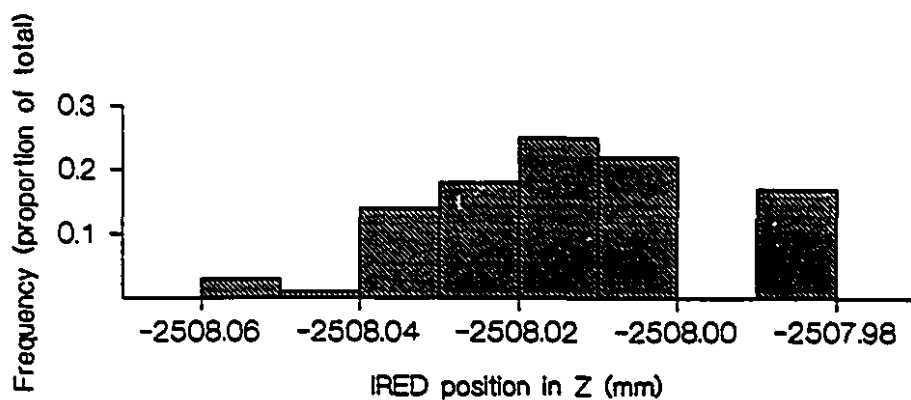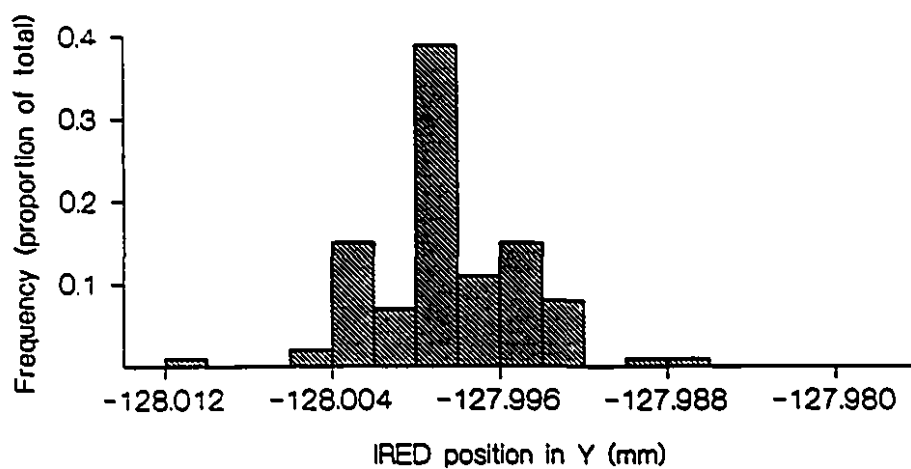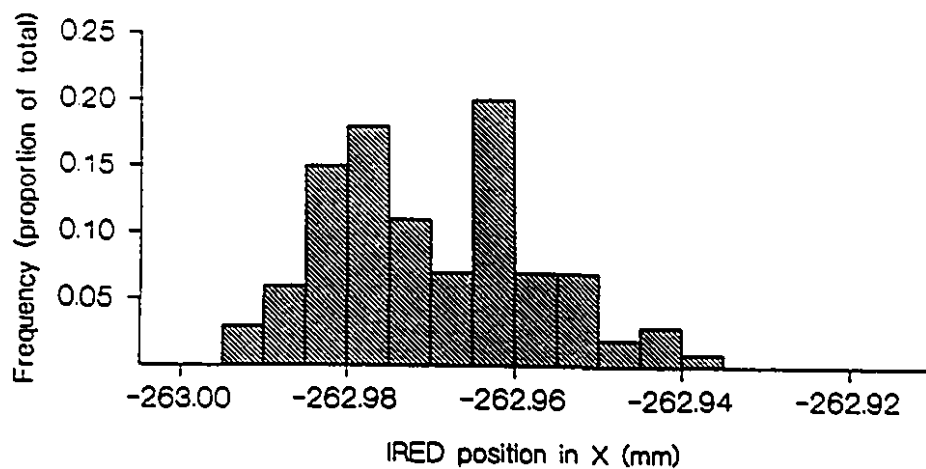
Figure 2.1. Each histogram indicates the distribution of an IREDs location in each dimension during a one second recording. Note that the location varies less than 0.1 mm in each dimension. Positions are given in Optotrak coordinates.

the semiprone position) pronations and full and half supinations. In total there were eight pronation / supination movement conditions. The movements were performed both with the upper arm vertical and with the upper arm horizontal.

In movements combining flexion / extension with pronation / supination, subjects started from 45° below the horizontal plane (flexion movements) with the forearm either fully pronated or fully supinated. Subjects flexed the arm while simultaneously supinating or pronating, and ended at a target located 45° above the horizontal plane. Both full and half pronations, and full and half supinations were collected. Start and end were reversed for extensions. There were, therefore, a total of eight combination movement conditions (flexion or extension x pronation or supination x full or half movement amplitude). Vertical and horizontal upper arm positions were used .

In order to ensure that movements were limited to the two degrees of freedom about the elbow, a brace was used to restrict wrist motion. Subjects were instructed to keep the upper arm stationary. The upper arm position was monitored visually during the experiment. Trials were repeated if there was discernable upper arm movement. During data analysis, trials in which forearm yaw angle exceeded 30° were excluded (yaw angles typically varied over a 20° range for these movements). This procedure was followed in this and all other experiments performed.

Five trials were collected in each of the 22 movement conditions with the upper arm held both vertically and horizontally. Thus, in total, there were 220 (5 X 22 X 2) trials collected for each subject. Subjects were instructed to move quickly and were allowed rest periods between conditions, but timing was not explicitly controlled. Four subjects (Subjects A - D) were tested.

Experiment 2

As in experiment 1, subjects made forearm movements to targets in a sagittal plane, which involved flexion or extension alone, pronation or supination alone and combinations of the two. Whereas in Experiment 1 subjects were not given any specific instructions concerning movement timing, in Experiment 2 an audio metronome was employed to explicitly control movement timing. In addition, movement amplitude was varied in the flexion / extension degree of freedom as well as in pronation / supination.

Figure 2.2 shows the experimental setup and the arm position conventions used. The setup was similar to that used in Experiment 1 with the exception that targets were presented on a video monitor in Experiment 2 (see section on Movement Targets).

In Experiment 2, for movements involving flexion or extension alone the
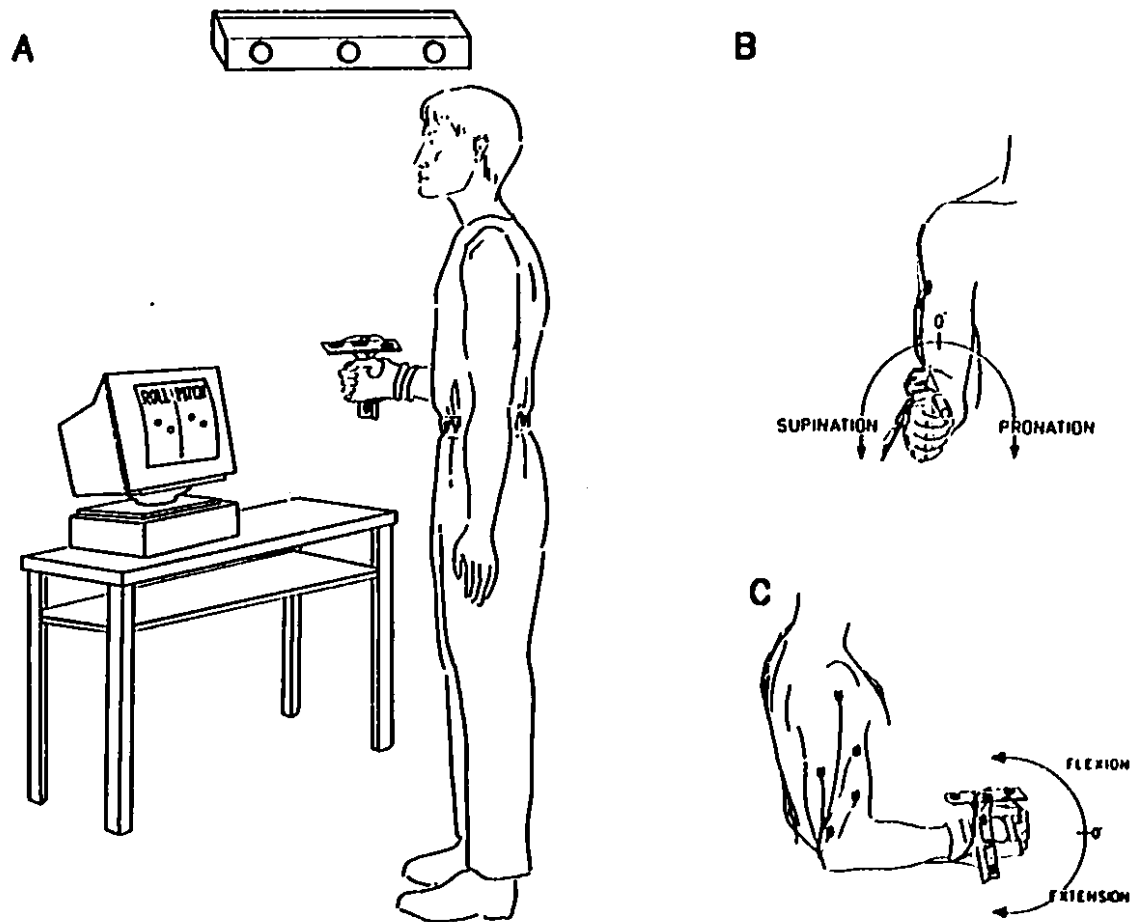
Figure 2.2. A: Schematic of the experimental setup. Subjects face a video monitor displaying targets in each degree of freedom. A plexiglass apparatus with attached IREDs allows Optotrak to monitor forearm orientation and position. B: Reference position for forearm rotation. 0° corresponds to the arm held in a semiprone position. C: IRED placement on the upper arm and on the forearm apparatus; reference position for forearm flexion / extension. 0° corresponds to the forearm held horizontal, and the upper arm vertical and in a parasagittal plane.

forearm was either fully pronated or fully supinated. The flexions started with the elbow fully extended and were either 70° or 140° in amplitude. Start and end positions were reversed for extension movements. Thus there were a total of 8 movement conditions involving flexion or extension alone: (2 directions x 2 magnitudes x forearm prone or supine).

In movements involving pronation or supination alone the forearm was held either with the elbow fully extended (-90°), or flexed 50°. The movements consisted of 70° and 140° pronations and 70° and 140° supinations (starting positions were forearm fully supinated and fully pronated respectively). Thus there were eight pronation / supination movement conditions. As in Experiment 1, procedures were followed to ensure that motion was restricted to the two degrees of freedom about the elbow. These included use of a wrist brace and visual monitoring of upper arm motion. Trials in which there was discernable upper arm motion were repeated; trials in which there was significant yaw motion (> 30°) were discarded.

In movements combining flexion / extension with pronation / supination, subjects started with the elbow fully extended (flexion movements) with the forearm either fully pronated or fully supinated. Subjects flexed the arm either 70° or 140° while simultaneously supinating or pronating either 70° or 140°. Start and end positions were reversed for extension movements. All

combinations of the two magnitudes in each of the two degrees of freedom and in both directions were performed for a total of 16 combination movement conditions. The upper arm was held vertically for all movements.

An audio metronome was used to maintain movement durations at 350 milliseconds (ms). This movement duration was chosen based on the results of a control study in which a subject performed one and two degree of freedom movements ranging in duration from 250 to 400 ms. The time that was chosen satisfied two criteria: it was well below the subjects maximal effort and produced discernable muscle bursts in all muscles in almost all movement conditions. Ten trials were collected in each of the 32 movement conditions for a total of 320 trials per subject. Between five and ten practice trials were allowed before each movement condition until the subjects could perform the movements smoothly within the required movement time to the targets. Subjects were given rest periods between each movement condition. Five subjects (Subjects A - E) participated in Experiment 2.

Experiment 3

Three subjects (Subjects A, F, and G) repeated a subset of Experiment 2 with the upper arm held in a horizontal position, while movement continued to be restricted to a sagittal plane. This data was collected in a separate

experimental session.

The number of combined flexion / extension with pronation / supination movement conditions were reduced such that only 140° movements in each degree of freedom were performed. Thus there were a total of four combined movement conditions: 140° flexion with 140° supination or pronation, and 140° extension with 140° supination or pronation.

Movements involving flexion alone or extension alone were performed with the forearm held supine, and were 140° in amplitude. Similarly, movements involving pronation alone or supination alone were performed at an elbow angle of 0° and were 140° in amplitude. Hence there were four single degree of freedom movement conditions (one flexion, one extension, one pronation, one supination).

Again, in order to ensure that movements were limited to the two degrees of freedom about the elbow, a brace was used to restrict wrist motion. Subjects were instructed to keep the upper arm stationary. Upper arm position was monitored visually and trials where were repeated if the upper arm dropped out of a horizontal plane. Because maintaining the upper arm in a horizontal position required a large amount of exertion, subjects were given extended rest breaks following each movement condition.

As in experiment 2, an audio metronome was used to maintain movement durations at 350 milliseconds.

In total, 80 trials (10 trials for each of 8 conditions) were collected for each subject. Subjects practised each movement until the movement could be performed smoothly while starting and ending within the targets for each degree of freedom.

Experiment 4

During movements involving both degrees of freedom at the elbow, biarticular muscles may act as agonist in one degree of freedom and as antagonist in the second. A further study was run to assess the conditions under which biarticular muscles display either agonist or antagonist activity. Five subjects (Subjects A, F, G, H, and I) performed four different sets of discrete movements. In each movement condition the amplitude of the movement in one degree of freedom was fixed while the amplitude in the other degree of freedom was gradually increased. The four movements were: fixed amplitude flexion (90°) with a continually increasing supination (10° - 130°), fixed amplitude flexion with a continually increasing pronation, fixed amplitude pronation (120°) with a continually increasing flexion, and fixed amplitude supination with a continually increasing flexion. Twenty discrete movements

were collec٬٬d in each condition.

The manipulation was repeated using a continuous change in movement amplitude in one degree of freedom while holding the amplitude of the movement in the second degree of freedom constant. For example, subjects would flex 90° and supinate, then extend and pronate back to the original starting position, then flex 90° and supinate a little more, then extend and pronate back to the original position again, etc. The audio metronome would sound continuously at 350 msec intervals so that a seven second trial would contain twenty movements - ten flexing supinations alternated with ten extending pronations. Subjects performed two repetitions of continuous movements for each of the four movement conditions used in the discrete manipulation, for a total of eight continuous movement trials.

For both discrete and continuous movements, the forearm was held at an initial flexion angle of -70°. For two of the subjects, all movements were repeated with the forearm held at an initial flexion angle of -40°.

MUSCLE ACTIVITY RECORDING

Experiment 1

Recordings were made from the following eight muscles in subjects A, B and C: triceps brachii (lateral head), biceps brachii (long head), biceps brachii (short head), brachialis, brachioradialis (except in Subject A), anconeus, pronator teres, and pronator quadratus. In subject D recordings were made from the muscles listed above, but brachioradialis was replaced by the medial head of the triceps. Electrode placement was verified by having subjects perform test manoeuvres (Delagi, 1980). Table 2.2 lists the muscle, placement, and test manoeuvre used for the ten muscles used in all four Experiments. EMG signals were sampled at 1200 Hz, digitally band-pass filtered between 20 and 300 Hz, rectified, and integrated off-line. Trials for which movement artifact was evident were discarded.

A number of points should be addressed concerning the use of surface electrodes. One concern during anisometric motion is that the motor unit population sampled during the course of the movement may change due to uncontrolled motion of the electrode with respect to the underlying muscle. The magnitude of error introduced by the use of surface electrodes in these studies has not been assessed. However, the patterns of activity which are reported in this thesis are similar for muscles with different electrode placements and different mechanical actions. In addition, the data reported here are consistent across all subjects despite differences in subject anthropometries as well as differences in electrode placement between subjects. This suggests that the

error due to electrode movement does not significantly alter the conclusions of the present studies.

A second concern is whether surface electrodes are suitable for recording muscles such as pronator quadratus, pronator teres, and brachialis. Pronator quadratus is situated underneath wrist tendons while pronator teres and brachialis are situated nearby large wrist and elbow flexor muscles. A number of steps were taken to ensure that only the desired muscle activity was recorded. The wrist and fingers were stabilized using a brace strapped securely onto the hand. This minimized wrist flexor activity which might have contaminated the pronator teres signal. In addition, it was observed that any motion of the fingers produced a saturation of the EMG signal for the pronator quadratus, and thus was readily detectable. Brachialis could be distinguished from biceps brachii as a muscle which produced activity during flexion only and not during supination. This was confirmed during electrode placement. Other tests for electrode placement are shown in Table 2.2.

Experiment 2 and Experiment 3

Recordings were made from the following eight muscles: triceps brachii (long head), triceps brachii (lateral head), biceps brachii (long head), biceps brachii (short head), brachialis, brachioradialis, pronator teres, and pronator

Muscle	Mechanical action	Electrode placement test
Triceps Brachii, long head	extends elbow/shoulder	1.pre-movement activity during elbow or shoulder extension
Triceps Brachii, lateral head	extends elbow	1. pre-movement activity during elbow extension 2. no activity during shoulder extension
Triceps Brachii, medial head	extends elbow	1. pre-movement activity during elbow extension 2. no activity during shoulder extension
Anconeus	extends elbow	1. pre-movement activity during elbow extension 2. post-movement activity during elbow flexion
Biceps Brachii, long head	supinates forearm flexes elbow flexes shoulder	1. pre-movement activity during elbow or shoulder flexion 2. pre-movement activity during supination
Biceps Brachii, short head	supinates forearm flexes elbow	1. pre-movement activity during elbow flexion or supination 2. no activity during shoulder flexion
Brachialis	flexes elbow	1. pre-movement activity during elbow flexion 2. no activity during forearm supination
Brachioradialis	flexes elbow	1. pre-movement activity during elbow flexion
Pronator teres	pronates forearm flexes elbow	1. pre-movement activity during elbow flexion 2. pre-movement activity during forearm pronation
Pronator quadratus*	pronates forearm	1. pre-movement activity during forearm pronation

* wrist and fingers were braced to prevent the effects of tendon movement on pronator quadratus activity measurement

Table 2.2

quadratus. Again, electrode placement was verified by having subjects perform test manoeuvres (Delagi 1980, Table 2.2). EMG signals were sampled at 1200 Hz, digitally band-pass filtered between 20 and 300 Hz, rectified, and integrated off-line.

Experiment 4

Because activity in the biarticular muscles and their single-joint counterparts was the focus of Experiment 4, recordings were made from only five muscles in Subjects H and I: triceps brachii (lateral head), biceps brachii (long head), brachialis, pronator teres, and pronator quadratus. Because Experiments 3 and 4 were run consecutively for Subjects A, F, and G (who participated in both), recordings were made from eight muscles (as in Experiments 2 and 3) for these subjects. Electrode placement was verified and EMG signals were processed as in the previous three experiments.

MOVEMENT RECORDING

In all four experiments, the position of the arm was recorded in three dimensions using an Optotrak system. IREDs were placed on the subject's upper arm and on a lightweight plexiglas apparatus strapped to the wrist (Figure

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2.2). The wrist apparatus was used to obtain the motion of the forearm. In Experiment 1, IREDs were also placed on the subject's torso. Five to six IREDs were used to define each structure. IRED positions were sampled at 100 Hz.

Orientation angles of the upper and lower arms were calculated from raw data using a vendor-supplied rigid body algorithm based on the method of quaternions (Horn, 1987). Lower arm motion was specified relative to the upper arm. An angle of 0° pitch corresponded to a right angle at the elbow (Figure 2.2c); an angle of 0° roll corresponded to the forearm in a semi-prone position (Figure 2.2b). In Experiment 1, upper arm position was specified relative to the torso. An angle of 0° corresponded to the upper arm aligned with the frontal plane.

Prior to the start of each experiment, the static positions of IREDs relative to anatomical landmarks were recorded for later calculation of the orientation angles of the upper and lower arm. Specifically, 3D distances between the acromion and upper arm markers, and between the oleocranon and lower arm markers were measured. Using these known distances, forearm orientation were calculated in an elbow centred coordinate system. In Experiment 1, these distances were also used to calculate upper arm orientation in a shoulder-centred coordinate system.

Experiment 1

Target positions for flexions and extensions were specified using plastic washers hung from the ceiling. Physical targets were not used for pronation / supination movements. Rather, subjects were instructed to either "fully pronate (or supinate)", or "pronate (or supinate) to the semiprone position". All subjects were able to produce accurate "full" movements ranging from 135° to 155°, and "half" movements ranging from 80° to 110°. During subsequent data analysis, trials which did not fall into these ranges (less than 20% of all trials) were discarded.

Experiments 2 and 3

Subjects made movements to circular targets which were displayed on a video monitor (Figure 2.2a). The target positions were calculated individually for each subject in a recording session a day before the actual experiment. Subjects wore the wrist brace and plexiglass apparatus with six markers used in the experiment, while a seventh marker was placed on the lateral epicondyle to mark the elbow location. The subject then held the arm in each of the desired start and end configurations while the Optotrak recorded the IRED

positions. The position of the subject in the recording area and the position of the forearm apparatus on the wrist brace was marked so that the subject would be in the same position during the actual experiment. During testing, pronation / supination targets, as well as the roll angle, were displayed in a frontal plane relative to the subject. On the other half of a split screen, flexion / extension targets, as well as the current pitch angle, were displayed in a sagittal plane. The diameter of the target circles corresponded to 15° in either the pitch or roll orientation. The target zones were displayed separately on a video monitor for each degree of freedom (Figure 2.2a).

Experiment 4

Targets were produced as in Experiments 2 and 3. Experiment 4 consisted of combined movements where motion in one degree of freedom was constant in amplitude while motion amplitude in the second degree of freedom was systematically varied. Thus, targets were displayed only for that degree of freedom whose amplitude was held constant in a given movement condition. Subjects were instructed to first increase the amplitude of motion in the second degree of freedom (so that the largest movement possible was attained by the fifth trial), and then decrease the amplitude back to a value of nearly zero by the tenth trial. In this way a large variety of amplitudes was collected for motion in the second degree of freedom while keeping movement amplitudes

in the first degree of freedom constant. The setup was similar for both continuous and discrete movement conditions.

DATA ANALYSIS

Kinematic analyses were carried out on the orientation angles of the lower arm. The orientation angles of the lower arm were calculated from raw data using rigid body reconstruction techniques based on the method of quaternions (Horn, 1987). Lower arm motion was specified relative to the upper arm. In Experiment 1, the orientation angles of the upper arm relative to the torso were also calculated and were used to verify that movements were initiated from either 0° or 90°, as instructed, and changed little during the trial. The orientation angles were numerically differentiated by the use of the least squares method (Dahlquist & Björck, 1969, Eq. 7.2.8). Kinematic records were scored for movement onset and offset using 10 % of the maximum velocity of the lowest velocity movement in each condition. That is, for each movement condition, velocity traces in each degree of freedom were examined. The 10 % maximum velocity value for the slowest movement was used as an absolute threshold to score movement onset and offset in both degrees of freedom for all trials in that movement condition. An absolute threshold was used when scoring movement onset in order to capture accurately the smallest amplitude movement.

Because movement time was fixed, the slowest movement corresponded to the degree of freedom having the smallest amplitude. In general, subjects were good at reaching the movement targets and thus movement amplitudes did not vary greatly within a movement condition. Trials were discarded if the movement amplitude deviated more than 15% from the target amplitude in either degree of freedom. For example, the acceptable range for a 140° movement was 129.5° to 150°. Less than 5% of all trials were discarded for this reason for any one subject.

EMG ANALYSIS

Qualitative and Quantitative analyses

In all experiments, EMG signals were initially scored qualitatively, as displaying either a phasic agonist burst, a phasic antagonist burst, a tonic increase in activity, or little to no activity.

EMG signals were scored for the onset and offset of the first burst of activity displayed by a muscle. Burst onset was scored as the point on the EMG record two standard deviations above the baseline level prior to movement. Burst offset was the point at which the EMG signal returned to baseline. A numerical estimate of the burst magnitude was obtained by calculating the

integrated area under the rectified EMG signal between the point of burst onset and offset.

In 10 to 20% of trials in which flexion / extension was combined with pronation / supination, muscles which acted as agonist in both degrees of freedom (e.g., biceps in combined flexion / supination) displayed an EMG activity pattern that did not return to baseline until the end of the movement. In other similar trials, where EMG did return to baseline, the offset of the first agonist burst corresponded closely to both the onset of antagonist activity and the peak velocity of movement in either the flexion or supination degree of freedom. Thus, for purposes of data analysis, the burst offset was scored at the point of peak velocity in trials which displayed an extended agonist burst (see section on EMG scoring verification study).

Figure 2.3 displays position, velocity, and EMG activity records for a flexion / supination movement. The two agonist bursts are well defined for the long head of biceps brachii but less so for the short head. A comparison between using standard deviations and peak velocities to score first agonist burst offset are shown. The solid lines indicate the burst onset and offset scored by measuring the point two standard deviations above the EMG baseline level. The dashed line indicates where burst offset would be scored using the point of peak roll velocity. It can be seen there is only an approximately 10 ms
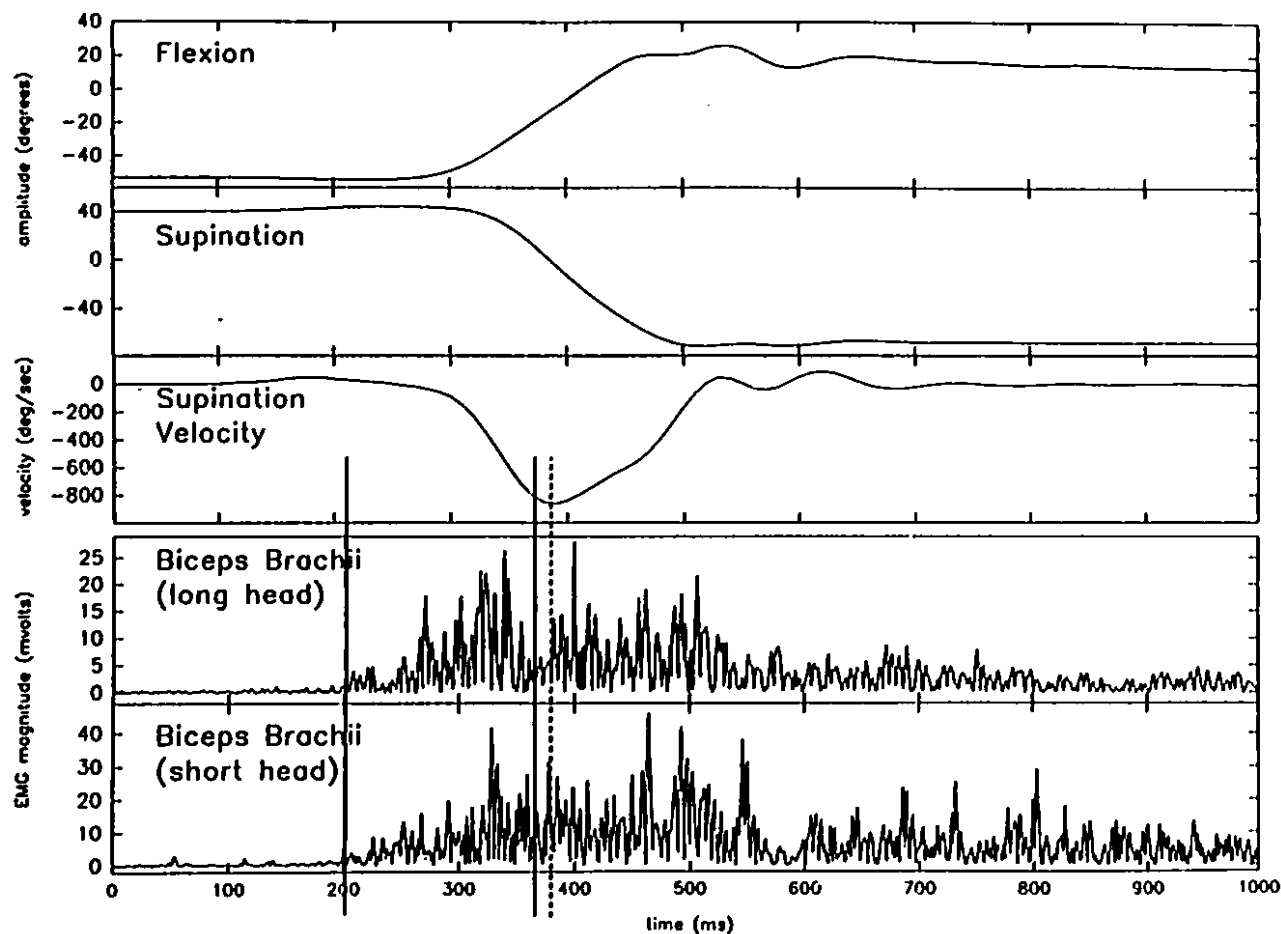
Figure 2.3. Kinematic and EMG activity records to illustrate scoring procedures. Vertical solid lines indicate where biceps brachii (long head) increases and then decreases at a point two standard deviations above the baseline EMG level. Vertical dashed line is the point of burst offset scored by using the peak supination velocity. The two methods differ in scoring burst offset by 11 ms. Data are shown for Subject E.

difference between the two offset points.

EMG scoring verification study

A study was undertaken to determine the variability in calculated EMG burst magnitudes for different scoring methods. It was assumed that the ideal way to determine an EMG burst onset and offset was by finding the point where the activity increased from its baseline level and then returned to its baseline level. Because it was observed that occasionally the EMG signal did not return to baseline until the end of the movement (see above), this study set out to determine which points on the kinematic record could serve as an accurate reflection of burst offset. Two points on the kinematic record were examined: peak tangential velocity of the hand, and peak velocity in either the pitch or roll degrees of freedom. To assess which point provided a good estimate of burst offset, a comparison was made between the burst magnitude calculated from direct scoring of the EMG signal and the magnitude calculated from use of points on the kinematic record. It was found that a good estimate of EMG burst offset could be provided by scoring the point of peak pitch or roll velocity.

EMG traces from the biceps long head recorded from Subject A were scored in three different ways. In all three methods, burst onset was scored as

the point where activity increased two standard deviations above baseline. In Method 1, burst offset was scored at the point where the EMG signal returned to baseline. In Method 2, burst offset was scored at the point of peak pitch or roll velocity. In Method 3, burst offset was scored at the point of peak tangential velocity, determined from a marker placed on the hand. All trials from three different movement conditions (140° flexion, 140° supination, combined flexion / supination) were scored. In the second scoring method, the peak velocity used depended on the movement. Thus, for flexion trials the point of peak flexion velocity was scored. For supination trials the point of peak supination velocity was scored. For combination trials, it was observed that the peak velocities in each degree of freedom roughly coincided (within 20 ms of each other). Hence, peak supination velocity was arbitrarily chosen for scoring in this movement condition.

Figure 2.4 displays burst magnitudes calculated from the three different scoring methods for nine flexion movements. It can be seen that when burst offset was determined by use of flexion peak velocity, the burst magnitudes accurately reflected the actual burst magnitude. When burst offset was determined as the point of peak tangential velocity, however, burst magnitudes were different in magnitude. Results for the two other movement conditions were similar. A statistical analysis revealed a significant difference in flexion burst magnitudes between Method 1, burst offset scored as two standard

deviations from baseline, and Method 3, burst offset scored at peak tangential velocity for all movement conditions ($p < 0.001$). There was no significant difference in burst magnitudes between Method 1 and Method 2 (burst offset scored at the point of flexion or supination peak velocity).

These results show that the peak pitch or roll velocity may act as an accurate kinematic marker to be used in determining burst offset in those situations where the actual burst offset is not readily identifiable.
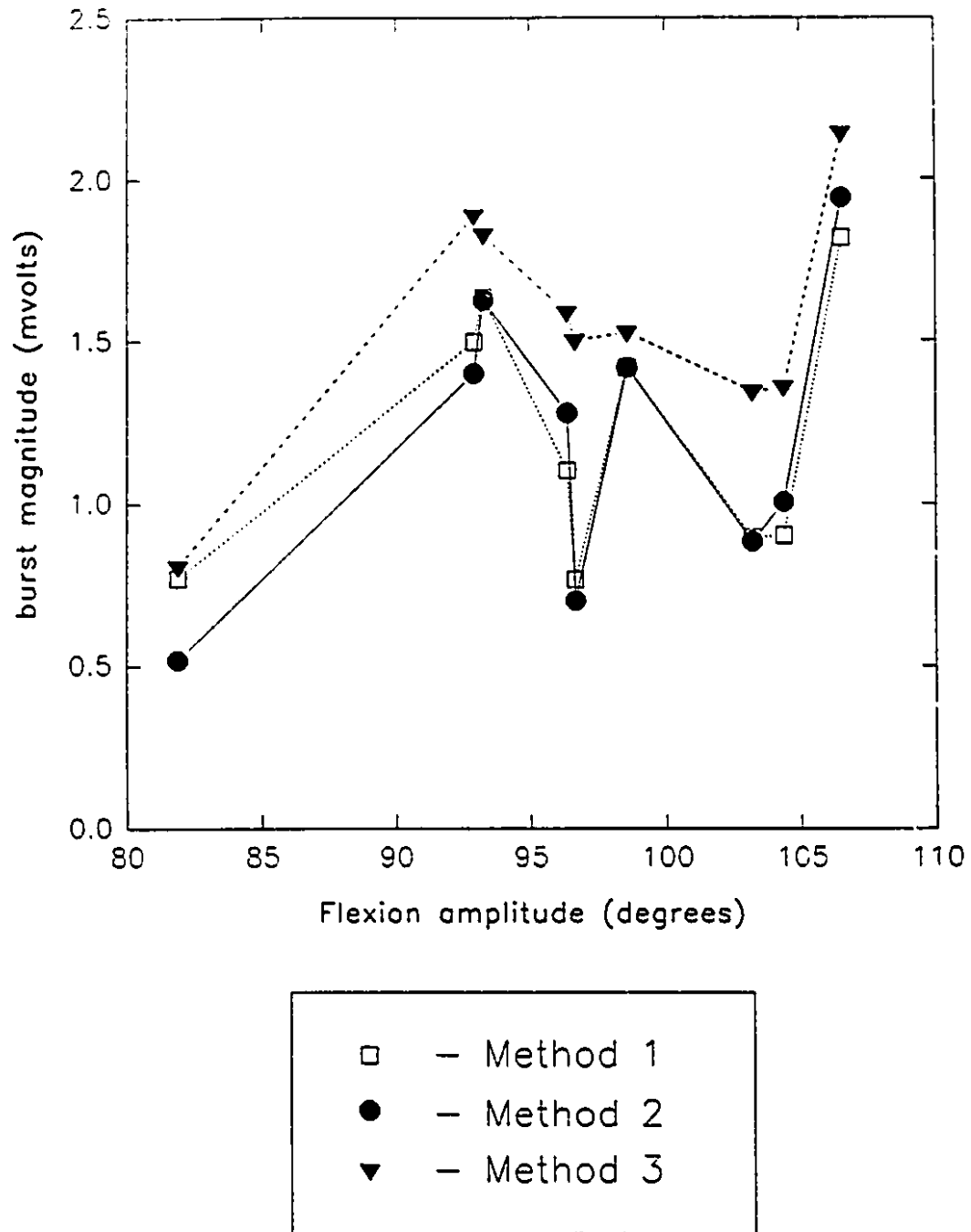
Figure 2.4. Flexion amplitude vs burst magnitude for three different burst offset scoring criteria. Note that Method 1 (return to baseline) is more closely approximated by Method 2 (peak flexion velocity) compared to Method 3 (peak endpoint tangential velocity).

3. RESULTS

A number of characteristics of an electromyographic signal associated with discrete isotonic movements can be quantified. The following sections examine the relationship between arm movement kinematics and a number of these characteristics in turn. Specifically, the first section presents a qualitative assessment of muscle activity patterns over the different movements performed. The second section examines the magnitude of the first agonist burst as a function of movement amplitude in each degree of freedom. The third section presents data on the behaviour of biarticular muscles during movements for which the muscles act as agonist in one degree of freedom and antagonist in the second. Data showing the effects of varying movement amplitude in each degree of freedom on muscle burst onset and duration are presented in the fourth and fifth sections, respectively. Throughout these experiments, analyses were performed separately for each subject. The main findings in each section are consistent across all subjects tested in each paradigm.

QUALITATIVE ASSESSMENT OF ELECTROMYOGRAPHIC ACTIVITY PATTERNS

An individual muscles' activity pattern was initially categorized in one of four ways. Within a single movement condition, each trial was examined and

each muscle was classified on the basis of presence and timing of activity. Muscles which displayed a burst of activity (defined as an abrupt activity increase followed by an abrupt return to baseline) whose onset was prior to movement onset were classified as "agonists". Muscles which displayed a burst of activity whose onset was after movement onset were classified as "antagonists". Muscles which displayed an increase in activity which remained at a steady level throughout the movement and then decreased back to baseline were classified as "tonic". Lastly, muscles which displayed no appreciable change in activity from baseline were noted as showing "little to no activity". Table 3.1 shows the classification assigned to each muscle for all movements performed in Experiment 2. The classifications are shown for all five subjects. In a few instances, different subjects would display a different pattern of activity. Hence, in some of the boxes there are two classifications listed, along with the number of subjects displaying each type of activity. While the table illustrates the overall characteristics of muscle activation patterns for multi-degree of freedom arm movements, individual records will be used to provide more detailed examples of these properties.

Relationships between muscle activity patterns when movement time was not explicitly controlled

Relationships between muscle activity patterns were different in one and

	Tricep (Long)	Tricep (Lateral)	Bicep (Long)	Bicep (Short)	Brachialis	Brachio-radialis	Pronator Teres	Pronator Quadratus
Flexion Supination	T	ANT	AG	AG	T	T	X (1);AG(4)	ANT
Flexion Half Supination	T	ANT	AG	AG	AG	T	AG	ANT
Flexion Pronation	X	X	AG	AG	AG	AG	AG	AG
Flexion Half Pronation	ANT	ANT	AG	AG	AG	AG	AG	AG
Half Flexion Supination	ANT	ANT	AG	AG	AG	AG	AG	ANT
Half Flexion Half Supination	T	ANT	AG	AG	AG	AG	AG	X (2) ANT (3)
Half Flexion Pronation	X	ANT	AG	AG	AG	AG	AG	AG
Half Flexion Half Pronation	ANT	ANT	AG	AG	AG	AG	AG	AG
Extension Supination	AG	AG	ANT	ANT	ANT	T	X (2) ANT (3)	ANT
Extension Half Supination	AG	AG	ANT	ANT	ANT	ANT	ANT	ANT

Extension Pronation	AG	AG	ANT	ANT	ANT	X	AG (3) ANT (2)	AG
Extension Half Pronation	AG	AG	ANT	ANT	ANT	ANT	ANT (4) X (1)	AG
Half Extension Supination	AG	AG	ANT	ANT	ANT	T	ANT	ANT
Half Extension Half Supination	AG	AG	ANT	ANT	ANT	T	ANT	X
Half Extension Pronation	AG	AG	ANT	ANT	ANT	ANT	AG	AG
Half Extension Half Pronation	AG	AG	ANT	ANT	ANT	T	T	AG
Flexion	ANT	ANT	AG	AG	AG	AG	AG	T
Half Flexion	ANT	ANT	AG	AG	AG	AG	AG	T
Extension	AG	AG	ANT	ANT	ANT	ANT	ANT	X
Half Extension	AG	AG	ANT	ANT	ANT	ANT	ANT	T
Pronation	X	T	X	X	T	T	AG	AG
Half Pronation	X	T	X	X	T	T	AG	AG
Supination	T	T	AG	AG	X	X	ANT	ANT
Half Supination	X	T	AG	AG	X	X	ANT	X

T = TONIC ACTIVITY   X = LITTLE OR NO ACTIVITY   AG = AGONIST BURST   ANT = ANTAGONIST BURST

Table 3.1, cont.

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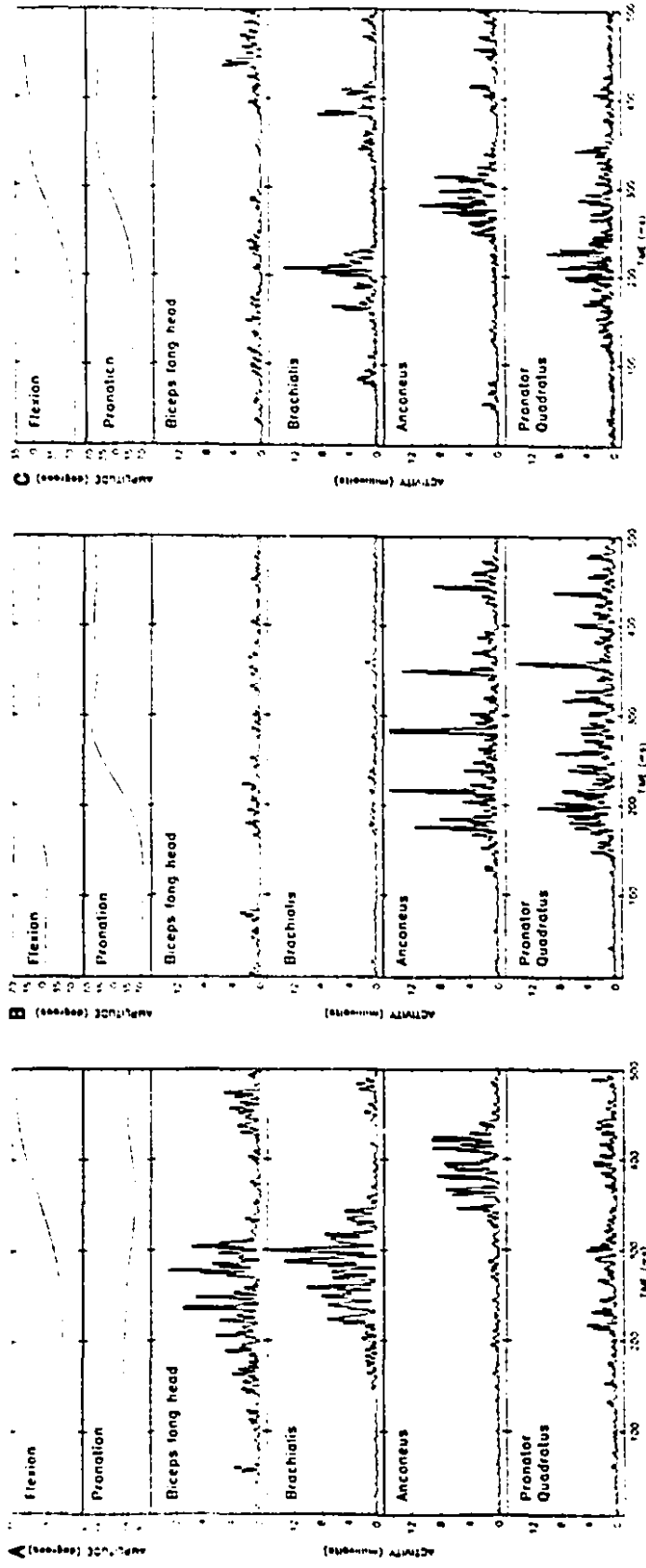
Figure 3.1 Kinematic patterns and EMG activity for elbow flexion alone (A), forearm pronation alone (B), flexion and pronation (C). Data are all from Subject A.

two degree of freedom elbow movements. Figure 3.1 shows activity patterns for four muscles involved in elbow flexion alone, pronation alone, and combined elbow flexion and pronation. The data are from Experiment 1 with the upper arm vertical. Similar patterns were observed in this experiment when the upper arm was horizontal. During a flexion movement (Figure 3.1a) biceps and brachialis act as agonists, anconeus acts as an antagonist and pronator quadratus shows little activity. During pronation (Figure 3.1b) anconeus and pronator quadratus are agonists while biceps and brachialis show little activity. Note that there is complete suppression of biceps activity prior to the onset of pronator quadratus and anconeus. During combined pronation and flexion (Figure 3.1c), the relationships differ from either flexion alone or pronation alone. Biceps activity is reduced relative to its activity in flexion alone whereas the magnitude of brachialis activity is unaffected. Thus, the relationship between biceps and brachialis may change depending on whether elbow flexion occurs alone or is accompanied by pronation. That is, the pattern of elbow flexor activity is affected by movement in the other degree of freedom.

During combined flexion and pronation, the patterns of anconeus and pronator quadratus activity also change relative to flexion or pronation alone. Anconeus acts as an antagonist during flexion alone whereas pronator quadratus shows little activity (Figure 3.1a). Anconeus and pronator quadratus are co-agonists during pronation alone and act antagonistically (i.e., one

displays an agonist burst while the other displays an antagonist burst) during combined pronation and flexion (Figures 3.1b and 3.1c). This pattern was also observed for pronator quadratus and pronator teres which acted as agonists during pronation and antagonistically during flexing supination. Thus, muscles which act as co-agonists in one movement may act antagonistically to each cther in another.

Relationships between muscle activity patterns when movement time was explicitly controlled

Relationships between EMG activity patterns also differed when timing was explicitly controlled. Figure 3.2 presents kinematics and associated EMG activity from a flexing supination (3.2a) and a flexing pronation (3.2b). As is the case when timing is not explicit, the pattern of activity across muscles is affected by movement in a second degree of freedom. For example, during a flexing supination, both heads of biceps brachii as well as brachialis display large bursts of activity, while brachioradialis shows only a small increase in activity above baseline. During a flexing pronation, however, brachioradialis exhibits a large agonist burst relative to the other three elbow flexors. Again, this is presumably to compensate for the reduced mechanical advantage of the biceps during a flexing pronation. A quantitative assessment of this relationship will be discussed below.
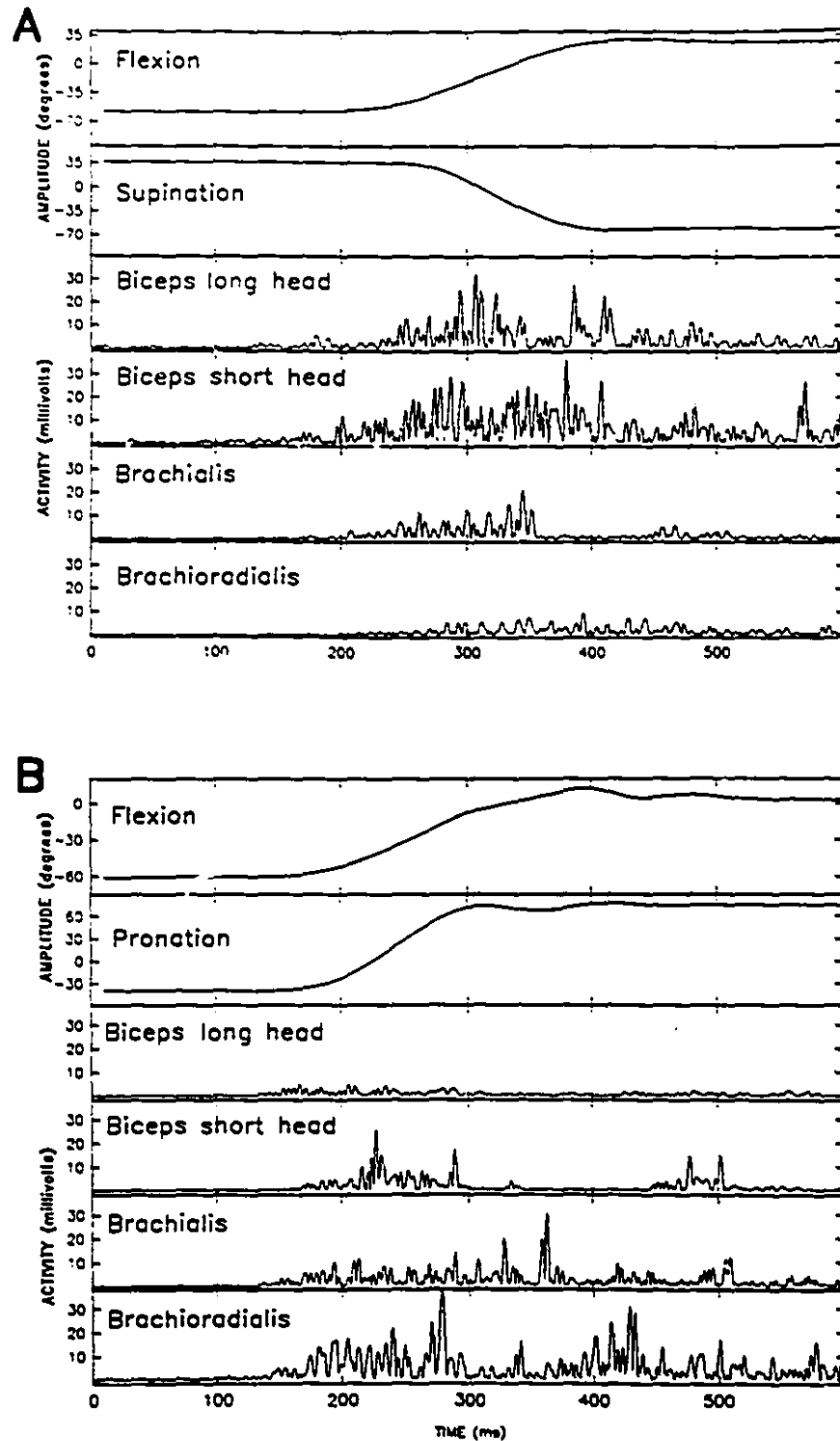
Figure 3.2. Kinematic patterns and EMG activity for a movement combining flexion and supination (A) and flexion and pronation (B). Note that the level of brachioradialis (a monoarticular elbow flexor) activity relative to the level of biceps (long head) activity changes with pronation / supination motion. Data are from Subject D.

Muscles fell into two categories with respect to the magnitude of the first agonist burst. Biarticular muscles such as biceps brachii (long head), bicepsbrachii (short head) and pronator teres showed activity that was affected by motion in two degrees of freedom. The magnitude of the burst was greatest for movements in which the muscle acted as an agonist in both degrees of freedom, less for one degree of freedom movements, and less still for movements in which the muscle was agonist in one degree of freedom and antagonist in the other.

The data illustrating the patterns of muscle activity in biarticular muscles in Experiment 1, when movement time was not explicitly controlled, are shown for two subjects in Figures 3.3 and 3.4. The patterns of muscle activity in movements involving flexion or extension alone, pronation or supination alone, and combinations of the two are shown in Figure 3.3 for Subject D, and in Figure 3.4 for Subject C. The panels to the left (panels A, C, and E in both figures) give the magnitude of individual agonist bursts as a function of the amplitude of elbow movement. Each of the plots is divided into four quadrants in which EMG amplitude is shown for various movement combinations. For example, in Figure 3.3a biceps EMG magnitude for movements involving supination and extension is shown at the lower left; movements involving
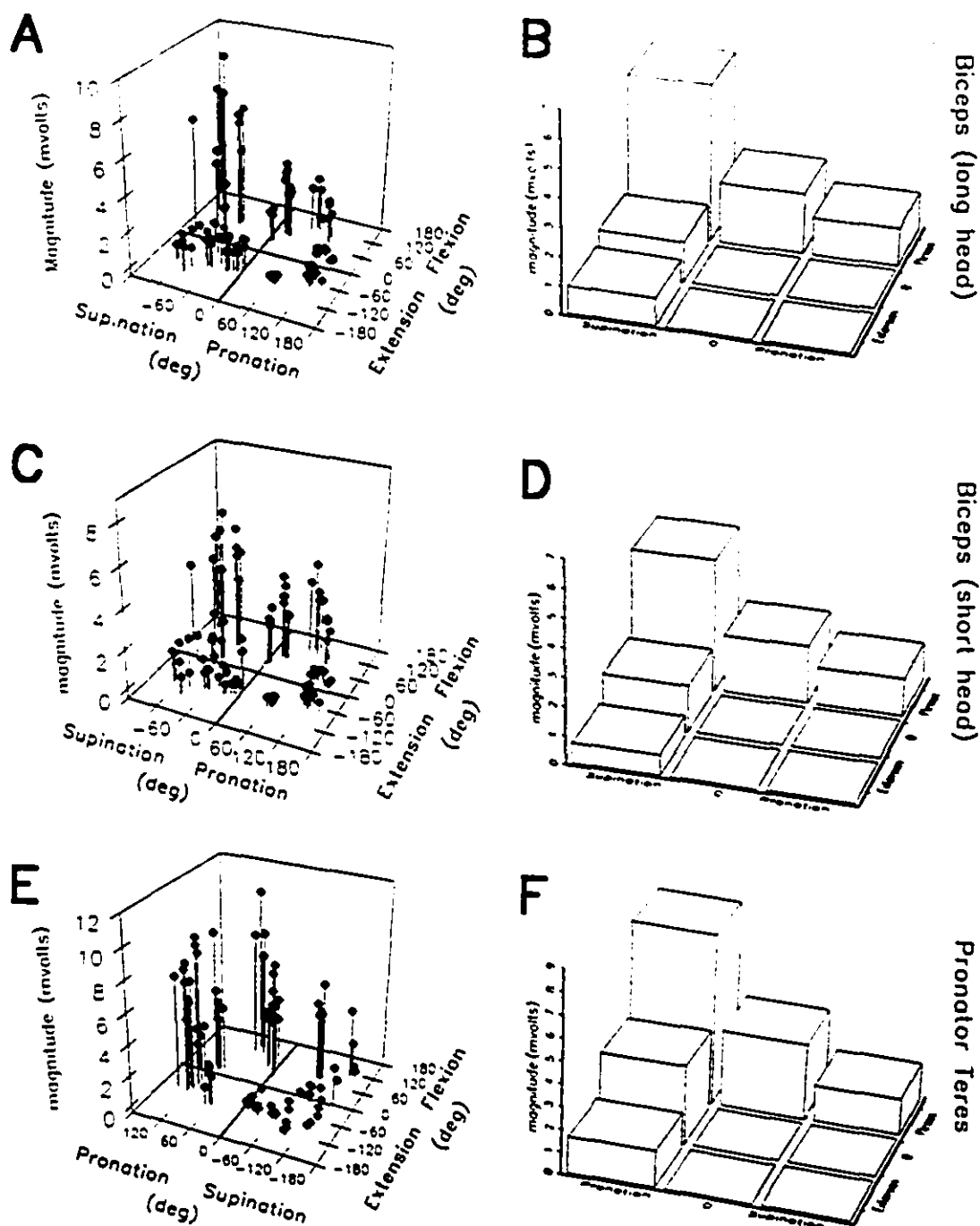
Figure 3.3. Agonist EMG magnitudes for biarticular elbow muscles. A,C,E: EMG agonist burst magnitudes for combinations of elbow flexion / extension, pronation / supination. Each dot represents an individual trial. Pitch and roll axes indicate the amplitude of movement in each degree of freedom. B,D,F: Average EMG agonist burst magnitudes for panels A, C and E respectively. Each block represents the average burst magnitude (across ten trials) for each movement condition. Data shown are from Experiment 1, Subject D.
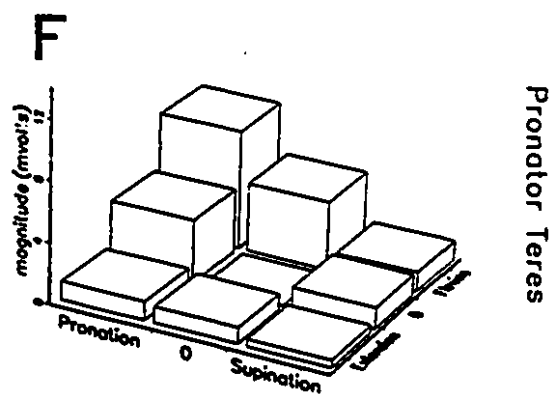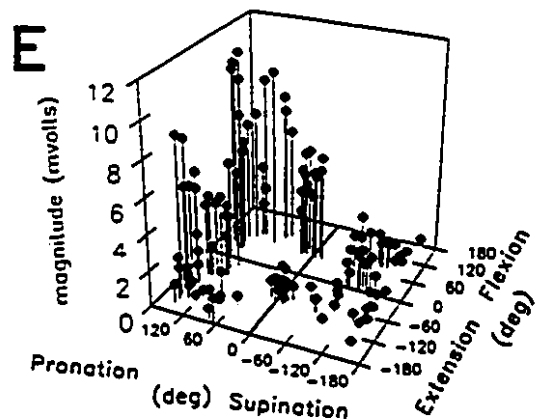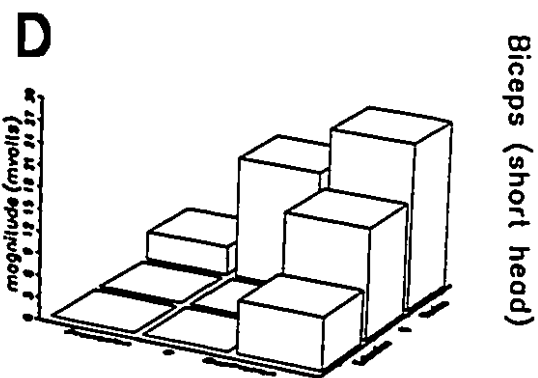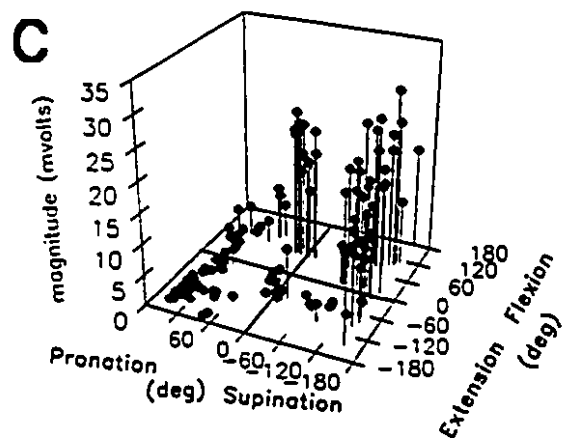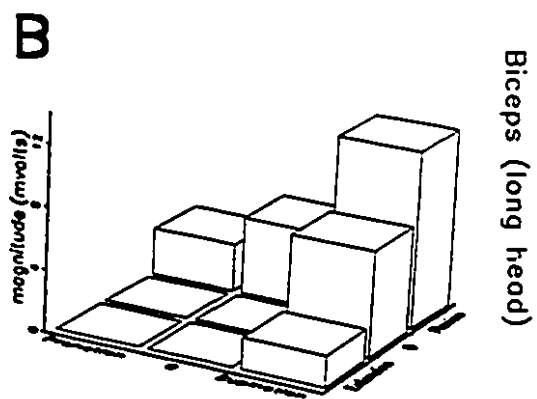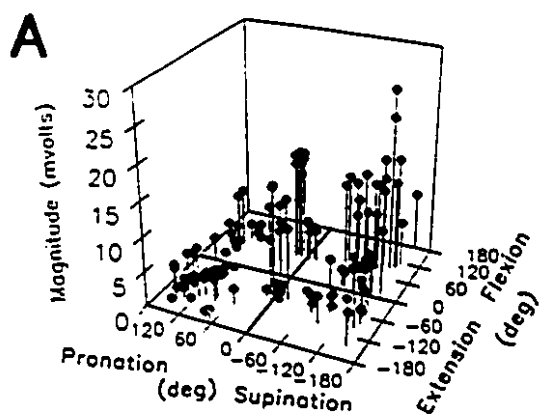
Figure 3.4. Agonist EMG magnitudes for biarticular elbow muscles. Panels on the left display individual trials. Panels on the right display data averaged for each movement condition. Data shown are from Experiment 1, Subject C.

supination plus flexion are shown at the upper left. The EMG amplitudes for flexion alone, extension alone, pronation alone and supination alone are shown along the axes. The panels to the right (panels B, D, and F) present the average EMG magnitude for all movement combinations for the same subject. Average activity is shown for trials involving supination plus extension, supination alone, supination plus flexion, etc. Flat areas indicate movement conditions for which that muscle did not display agonist activity.

An example of the activity pattern seen in biarticular muscles is shown for pronator teres in Figure 3.3f. EMG burst amplitude for pronator teres is greatest during a flexing pronation in which the muscle acts as agonist in both degrees of freedom. The activity level is less during pronation or flexion alone and still less for pronating extensions and supinating flexions, where pronator teres acts as agonist in one degree of freedom and antagonist in the other. Similarly, for biceps (both long and short heads, Figures 3.3 and 3.4 a - d), which is a forearm supinator as well as an elbow flexor, activity is greatest during a supinating flexion. Activity is less during supination or flexion alone, and even less during supinating extensions and pronating flexions in which the muscle serves as agonist in one degree of freedom and antagonist in the other. Comparable patterns were observed in all subjects.

Data representing the activity pattern observed in biarticular muscles in

Experiment 2, when movement time was explicitly controlled, is shown in Figure 3.5 for Subject E, and Figure 3.6 for Subject A. The figures are arranged as in Figures 3.3 and 3.4, with the burst magnitude for individual trials over different movement amplitudes displayed on the left (panels A,C, and E) and the average burst magnitude of a given movement condition displayed as a bar on the right (panels B, D, and F). Note that there are more bars than in Figures 3.3 and 3.4 because of the greater number of movement conditions in Experiment 2 (see Methods). To illustrate, the magnitude of the biceps brachii (long head) first agonist burst for different amplitudes of flexion / extension and pronation / supination is shown in Figures 3.5a,b and 3.6a,b. It can be seen that the magnitude of the burst is greatest for flexing supinations where the muscle acts as an agonist for both flexion and supination. The magnitude is . less for flexions alone or supinations alone, and it is smallest for both flexing pronations and extending supinations. This same basic pattern was observed in all five subjects who participated in Experiment 2.

A statistical comparison of first agonist burst magnitudes was performed for Experiments 1 and 2 for each subject using a one-way ANOVA. When biarticular muscles acted as agonists in two degrees of freedom, the magnitude of the first agonist burst was greater than in all other conditions ($p < 0.01$). In addition, in movement conditions in which the muscle acted as agonist in one degree of freedom only, the agonist burst magnitudes were greater than
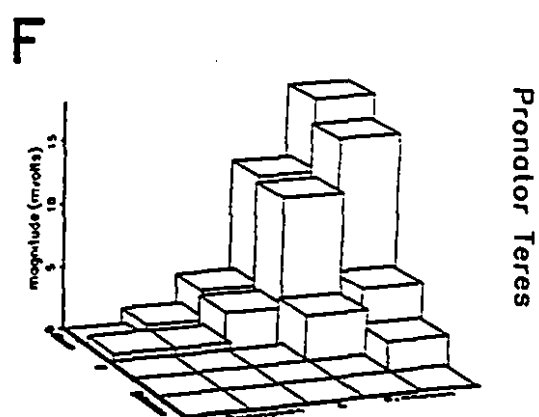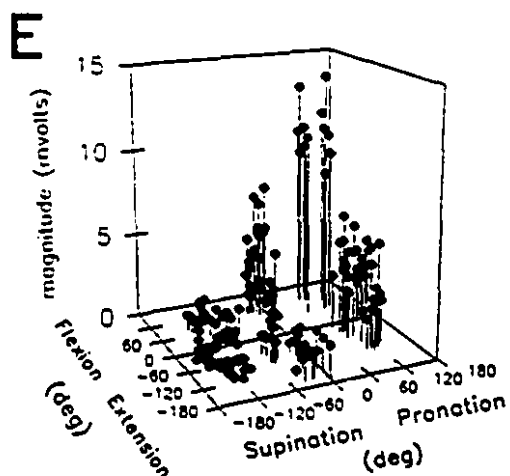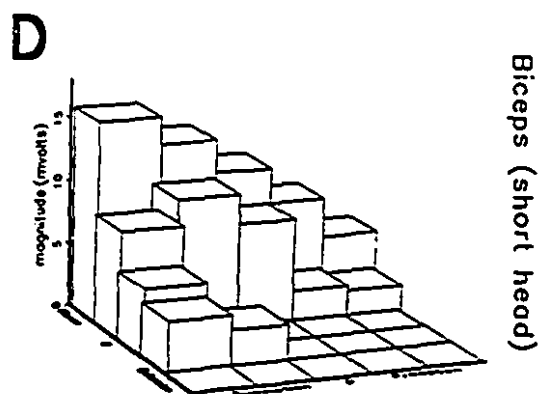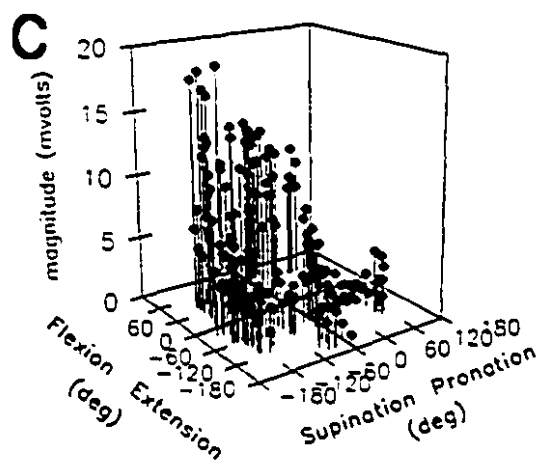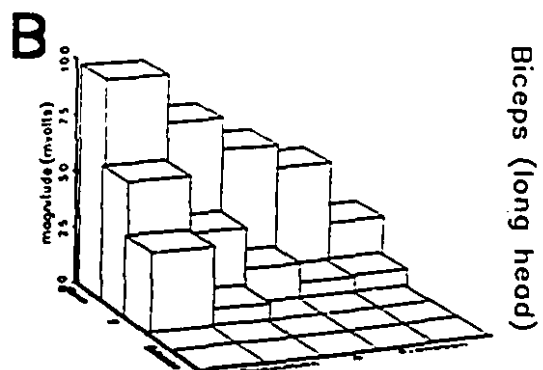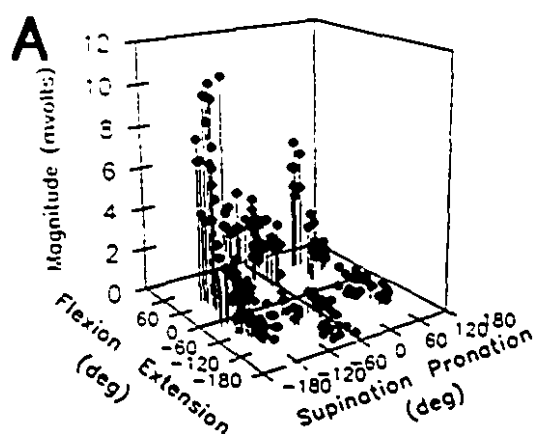
Figure 3.5. Agonist EMG magnitudes for biarticular elbow muscles. Panels on the left display individual trials. Panels on the right display data averaged for each movement condition. Data shown are from Experiment 2, Subject E.
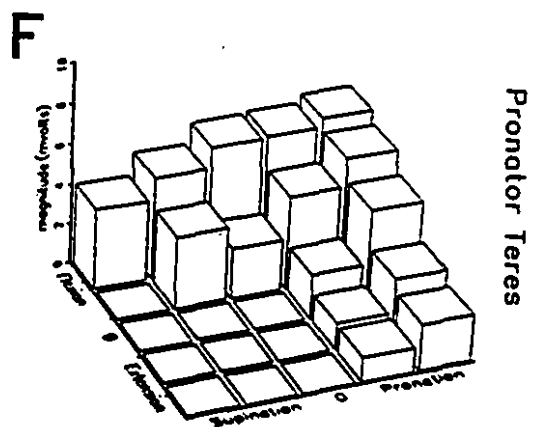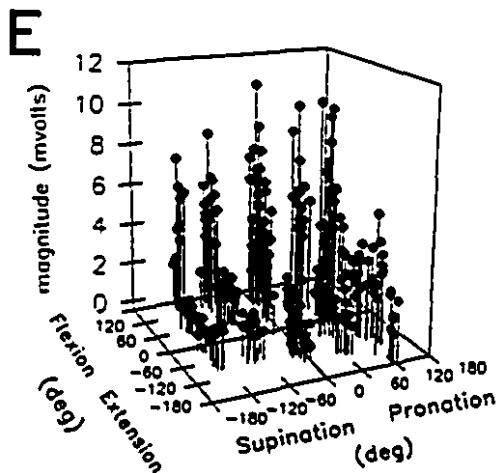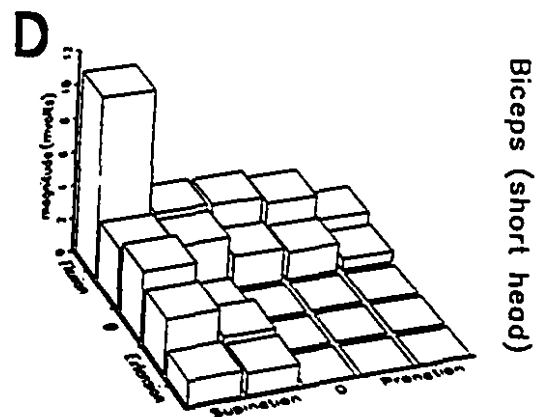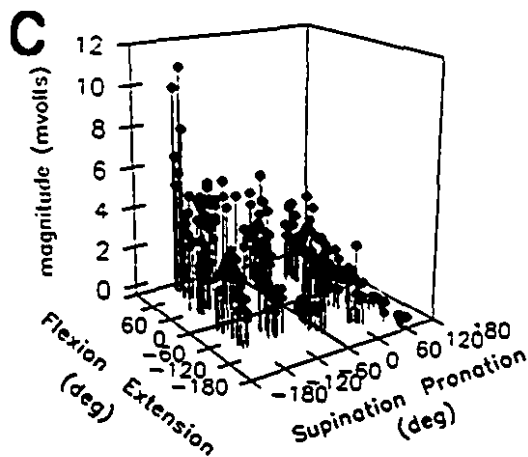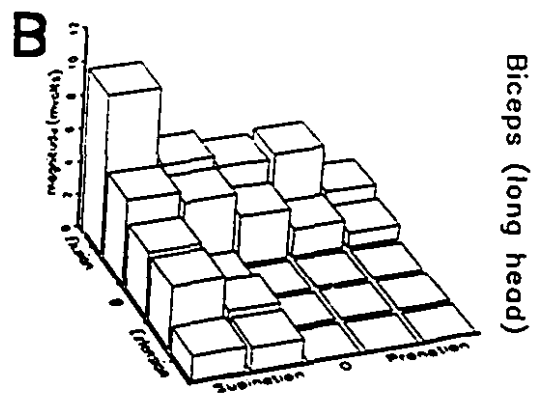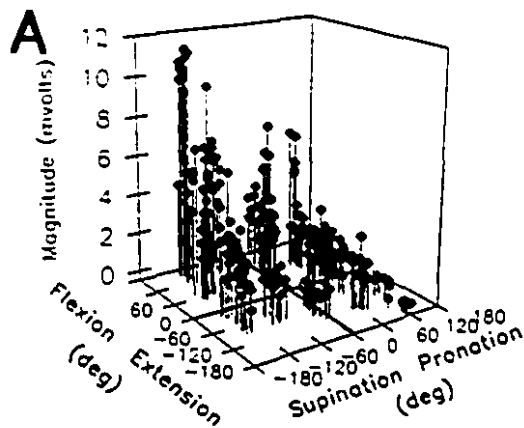
Figure 3.6. Agonist EMG magnitudes for biarticular elbow muscles. Panels on the left display individual trials. Panels on the right display data averaged for each movement condition. Data shown are from Experiment 2, Subject A.

when the muscle acted as agonist in one degree of freedom and antagonist in the second ($p < 0.01$). Similar patterns were observed for all subjects and for all biarticular muscles that were examined. (Note, these results are for movements performed with the upper arm in a vertical position. Movements in which the upper arm was held horizontally displayed similar EMG patterns in both Experiments 1 and 3 and will be discussed in a later section.)

In movements in which a muscle acted as agonist in two degrees of freedom, the magnitude of the agonist burst was the sum of the agonist burst magnitudes of the component one degree of freedom movements. This idea was tested statistically using post-hoc contrasts. Note that these tests provide evidence only for the departure from additivity, rather than additivity itself. Thus, tests for departure from additivity were done both for subjects who performed the experiment under explicit timing conditions (Experiment 2) and those for whom timing was not controlled (Experiment 1). When movement time was controlled, four out of five subjects (Subjects A - D) showed additivity of component EMG magnitudes for both heads of biceps brachii and pronator teres. Subject E only showed this additivity for the short head of the biceps. When movement time was not controlled, in Subjects B, C, and D the magnitude of the agonist burst in two degree of freedom movements did not differ from the sum of the component EMG magnitudes for both heads of biceps brachii ($p > 0.01$). Only one subject (Subject C) showed this additivity

for pronator teres. In all cases in both Experiments 1 and 2, in which the sum of the component EMG magnitudes was not equal to the EMG magnitude in the corresponding two degree of freedom condition, the sum of the two components was always less than the magnitude in the two degree of freedom condition.

A second category of activity was displayed by a number of monoarticular muscles in both Experiments 1 and 2. Some monoarticular muscles, including triceps brachii (long, lateral, and medial heads), anconeus, and pronator quadratus had first agonist bursts whose magnitude did not vary with motion in a second degree of freedom. In other monoarticular muscles, motion in a second degree of freedom did affect the EMG magnitude (to be discussed below).

An example of the pattern displayed by the first group of monoarticular muscles in Experiment 1 is shown for pronator quadratus in Figures 3.7c,d (Subject D) and 3.8a,b (Subject C). It can be seen that the level of pronation-related EMG activity was similar in magnitude for all movements involving pronation, regardless of whether these occurred in isolation or were combined with elbow flexion or extension. Muscles such as pronator quadratus were thus active as agonists for movements in one degree of freedom only. A simultaneous movement in another degree of freedom (e.g., flexion or

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extension) did not greatly affect the amplitude of the burst. Muscles which displayed this patterns of activity included medial head of triceps, lateral head of triceps, long head of triceps (Figure 3.7a, b), anconeus (Figure 3.8a, b) and pronator quadratus (figure 3.7c, d). Some subjects also displayed this pattern for brachioradialis (Figure 3.7e, f) and brachialis (Figure 3.8e, f) (but will be discussed further below). All subjects tested showed similar patterns for these muscles.

Figures 3.9 and 3.10 illustrate activity pattern of muscles in Experiment 2 whose agonist burst magnitude was unaffected by motion in a second degree of freedom. As an example, triceps brachii (long head) displayed a first agonist burst whose magnitude increased when the extension amplitude increased. However, the agonist burst magnitude for this muscle was essentially constant over extension movements of a given amplitude regardless of the amount of accompanying motion in the pronation or supination direction (Figure 3.9a,b).

For the monoarticular muscles triceps brachii (medial, lateral, and long heads), anconeus, and pronator quadratus, statistical comparisons revealed significant differences in agonist burst magnitude between large and small amplitude movements ($p < 0.01$). Motion in the second degree of freedom produced no significant differences in burst magnitude ($p > 0.01$). All subjects showed this pattern for these muscles in Experiments 1 and 2.
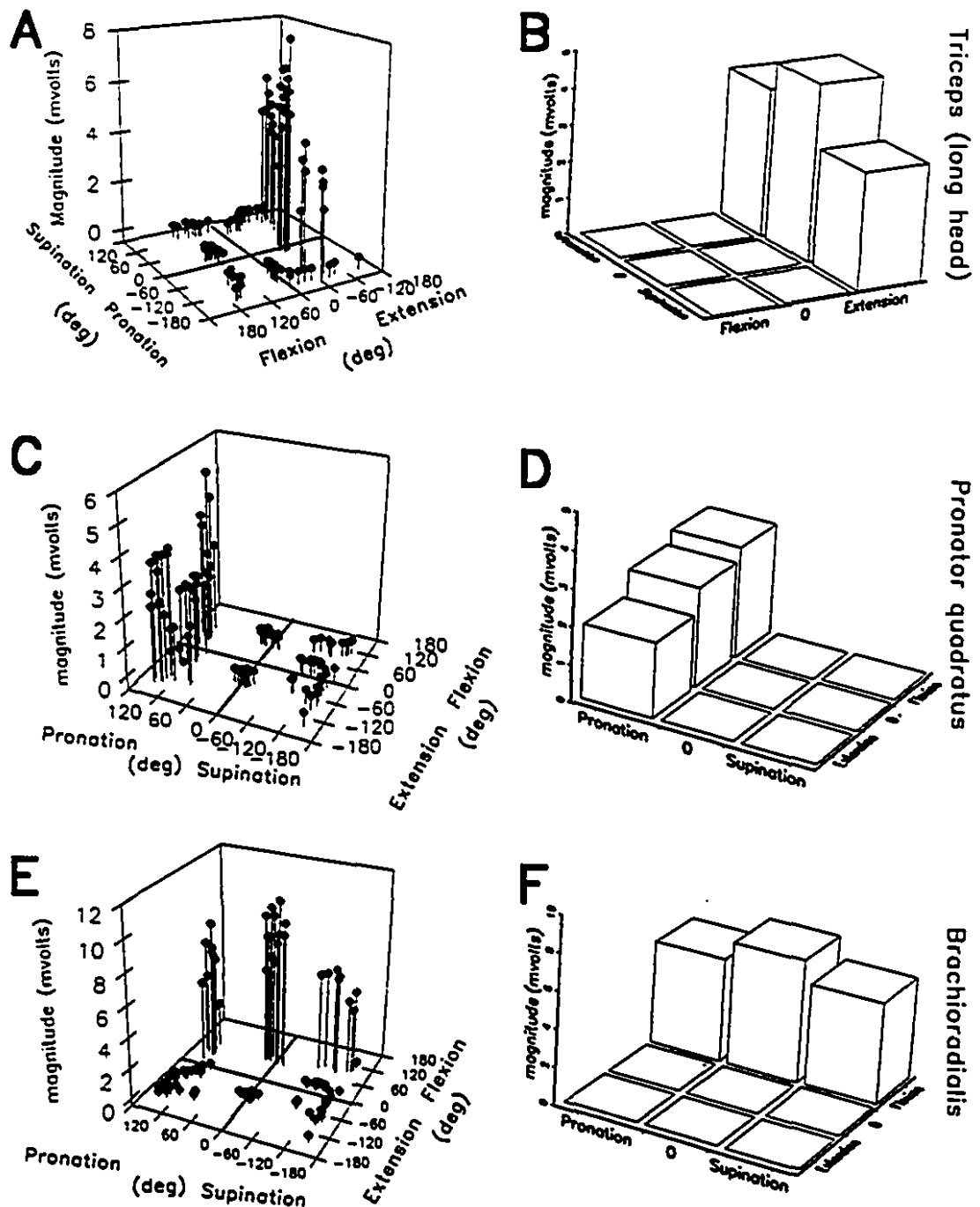
Figure 3.7. Agonist EMG magnitudes for monoarticular elbow muscles. Panels on the left display individual trials. Panels on the right display data averaged for each movement condition. Data shown are from Experiment 1, Subject D.
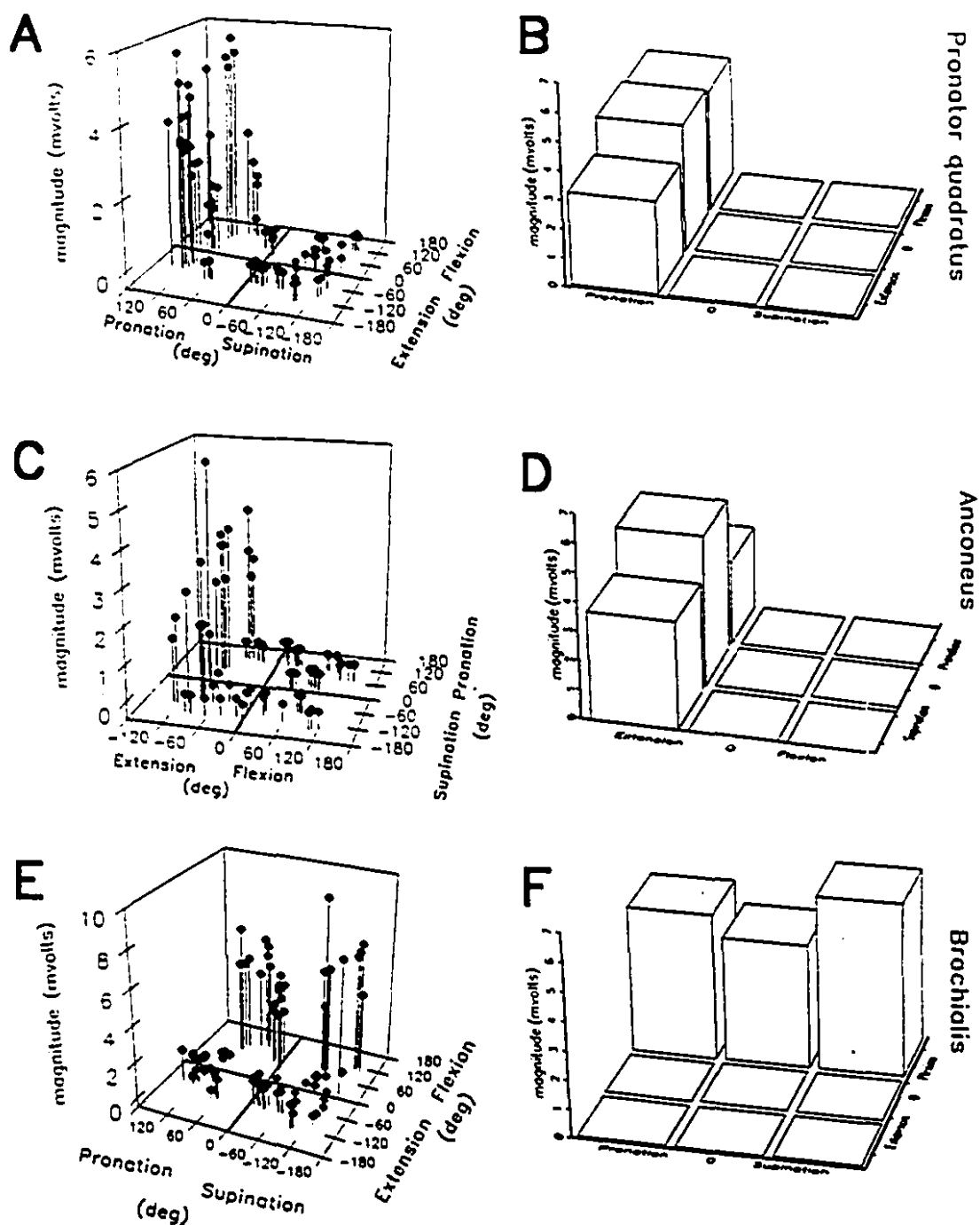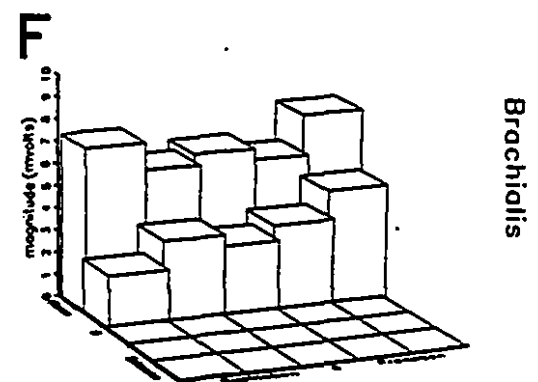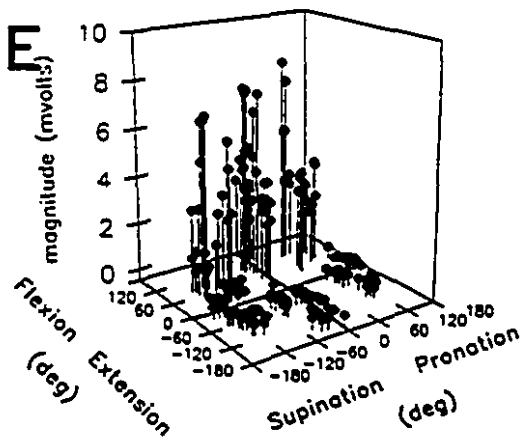
Figure 3.8. Agonist EMG magnitudes for monoarticular elbow muscles. Panels on the left display individual trials. Panels on the right display data averaged for each movement condition. Data shown are from Experiment 1, Subject C.
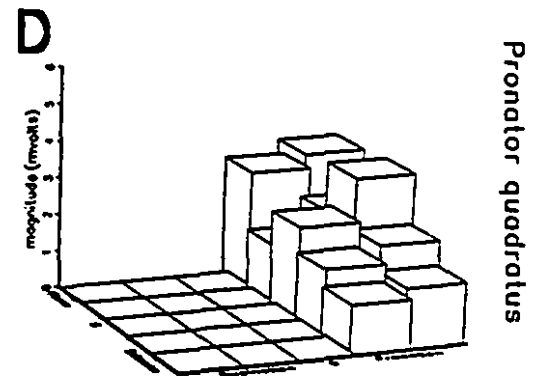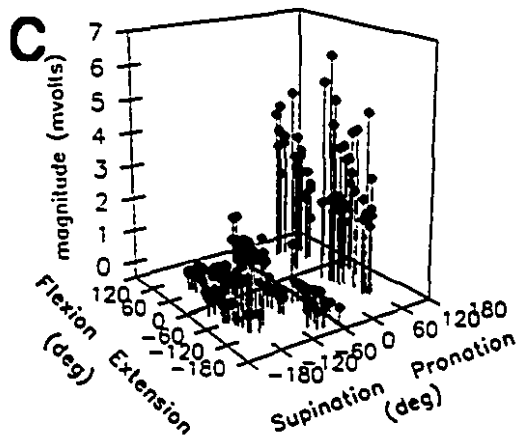
Figure 3.9. Agonist EMG magnitudes for monoarticular elbow muscles. Panels on the left display individual trials. Panels on the right display data averaged for each movement condition. Data shown are from Experiment 2, Subject E.
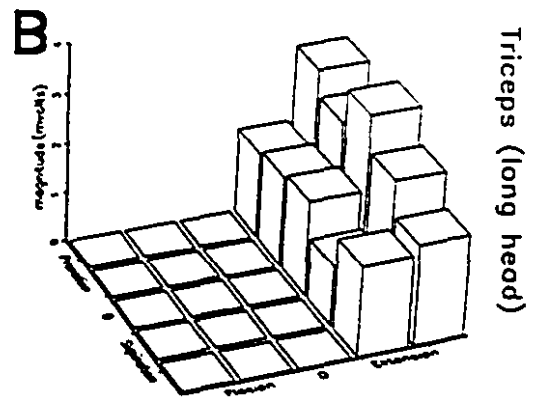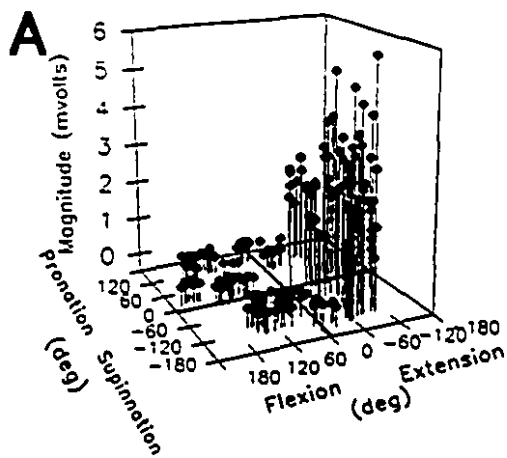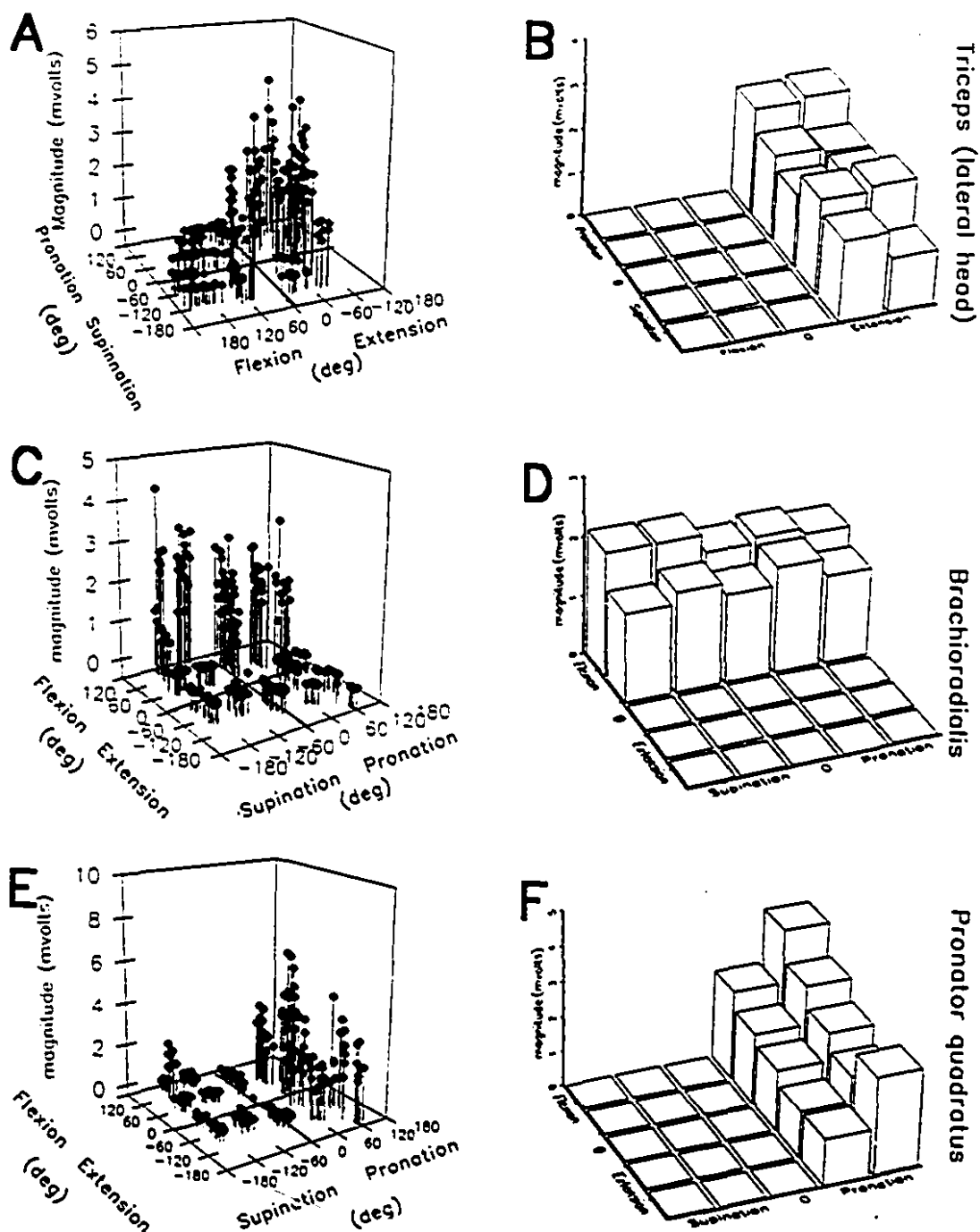
Figure 3.10. Agonist EMG magnitudes for monoarticular elbow muscles. Panels on the left display individual trials. Panels on the right display data averaged for each movement condition. Data shown are from Experiment 2, Subject A.

Activity in other monoarticular muscles was affected by motion in a second degree of freedom. As seen in Figure 3.2, the burst magnitude of brachioradialis (an elbow flexor) is greater for a flexing pronation than for a flexing supination. This is also observed in brachialis. Biomechanically, brachialis and brachioradialis produce torque only in the flexion direction. Given the reduction in biceps brachii activity during flexing pronations (Figures 3.1, 3.2), the increase in monoarticular flexor activity may occur as a compensatory response.

Statistical tests were performed on the monoarticular elbow flexors brachialis and brachioradialis, whose magnitude appeared to be affected by pronation or supination motion. In three of the four subjects tested in Experiment 1 the burst magnitude during flexing pronations increased relative to its magnitude during flexion alone or flexing supinations ($p < 0.01$). In four out of five subjects tested in Experiment 2, the burst magnitude was greater in flexing pronations than in flexions alone or flexing supinations ($p < 0.01$). However, a given subject showed this pattern for only one muscle or the other. For example, in Experiment 2, two subjects showed a brachialis burst magnitude that was greater for flexing pronations than for flexing supinations, while for two others the brachioradialis burst magnitude was greater.

In the three subjects who performed Experiment 3, where the

movements were made with the upper arm held horizontally, the behaviour displayed by each of the mono- and biarticular muscles remained the same as in the upper-arm-vertical condition with the following exception: the absolute magnitude of the agonist burst changed for biceps brachii (long head) and triceps brachii (long head), both of which act across the shoulder. In all three subjects, these muscles displayed a decrease in the magnitude of the agonist burst to a level lower than that of their single joint counterparts (i.e. biceps brachii (short head) and triceps brachii (lateral head) ($p < 0.01$). As a result, in two of three subjects the biceps brachii burst magnitude was *not* greater when it was acting as agonist in two degrees of freedom than in one degree of freedom ($p > 0.01$). Figure 3.11 illustrates the reduction in biceps brachii (long head) agonist burst magnitude during combined flexion / supination for these two subjects. Only large amplitude movement conditions (e.g. 140° flexion with 140° supination, 140° flexion alone, etc.) are shown for purposes of clarity.

BEHAVIOUR OF BIARTICULAR MUSCLES DURING TWO DEGREE OF FREEDOM MOVEMENTS

In movements involving two degrees of freedom the situation arises where a biarticular muscle may act as agonist to the motion in one degree of freedom and antagonist in the other. In Experiment 4, the behaviour of biarticular elbow muscles was examined in depth during movements of this type.

Biceps Brachii (long head)
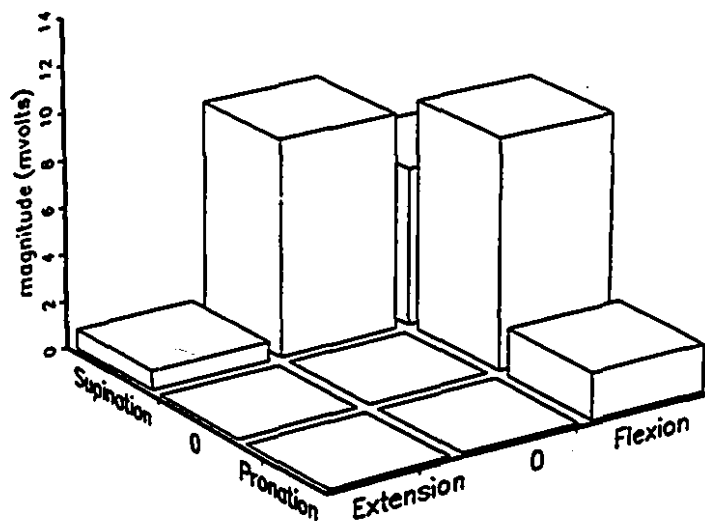
Subject F
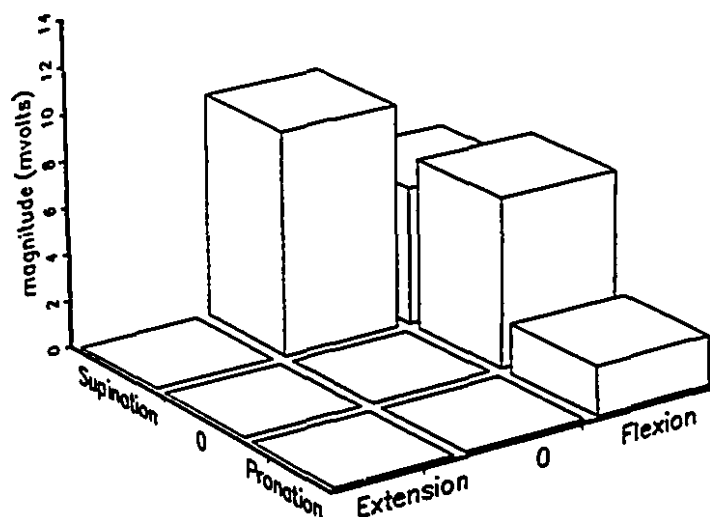
Subject A

Figure 3.11 Average biceps brachii agonist burst magnitudes for two subjects in Experiment 3 (upper arm held horizontal). Note that the magnitude of agonist burst in the flexion / supination condition is not greater than in the flexion and supination conditions alone.

A

Extension

Supination

Biceps Brochii

Triceps Brochii

Pronotor Teres

time (msec)

Subject C

B

Flexion

Pronotion

Biceps Brochii

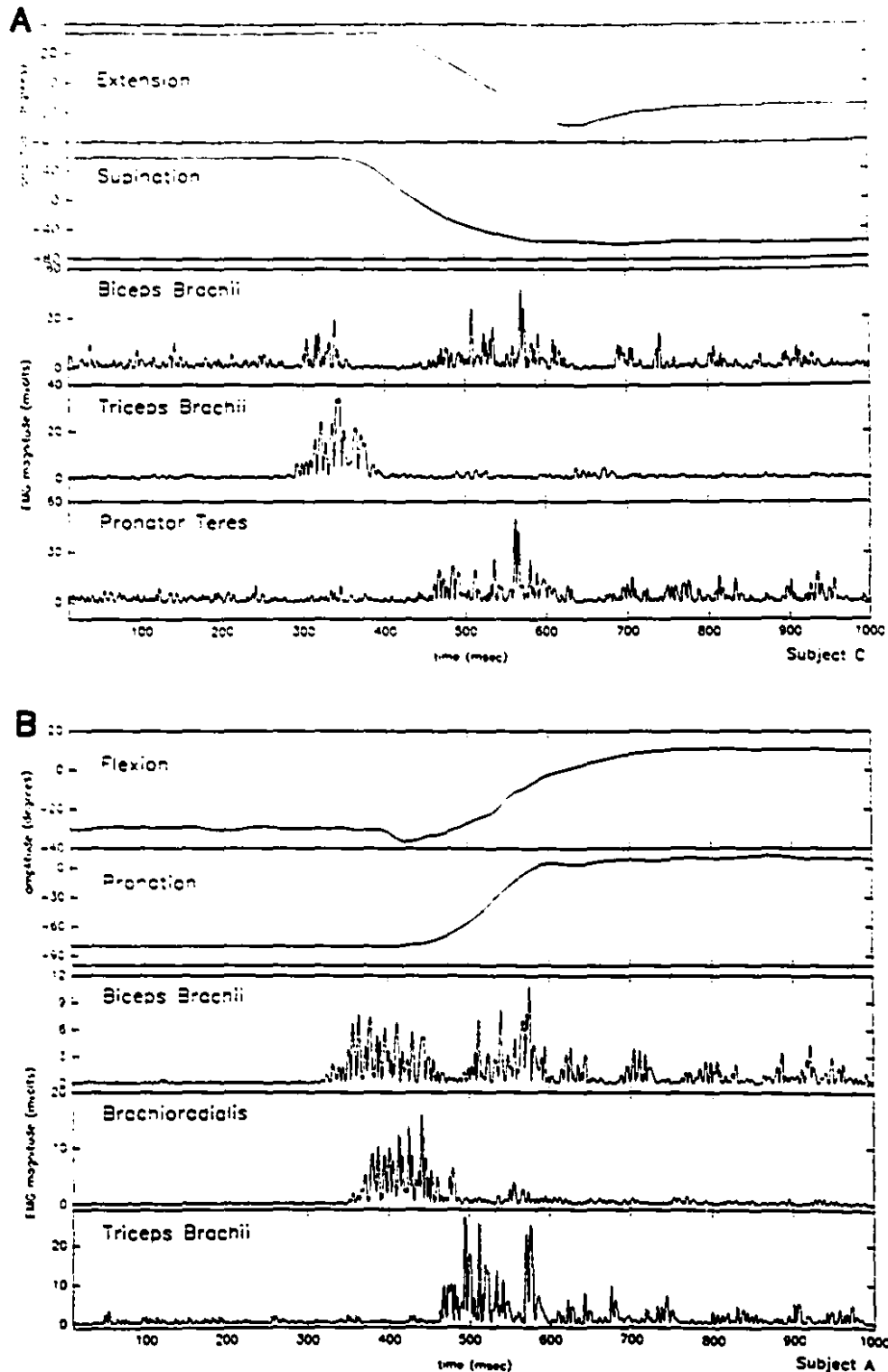Brochioradialis

Triceps Brochii

time (msec)

Subject A

Figure 3.12: Kinematic patterns and EMG activity for two different two degree of freedom movements in which biceps brachii acts as agonist to motion in one degree of freedom and antagonist to motion in the second. A: During an extending supination, biceps brachii displays burst activity along with triceps brachii (a pure agonist) and pronator teres (a pure antagonist). B: During a flexing pronation, biceps brachii again shows agonist and antagonist components. Data are from two different subjects.

It was found that pronator teres and biceps brachii displayed an activity pattern which had both agonist and antagonist components within the same movement. That is, the timing of the bursts was such that there was activity in these muscles that was concurrent with activity in both pure agonists and pure antagonists. Figure 3.12 shows the activity patterns of mono- and biarticular muscles during two different two degree of freedom movements. The pattern is quite general, so examples from different subjects are shown. Note that in these figures the background level of EMG activity is often greater than in previous records. Recall that, as shown in Figures 3.3 - 3.6, the movement conditions depicted in these figures are ones in which the biarticular muscles display bursts of low magnitude. Figure 3.12a depicts an extending supination (Subject C) and Figure 3.12b depicts an flexing pronation (Subject A). In these records, biceps brachii displays both agonist and antagonist activity. During an extending supination (Figure 3.12a) biceps brachii (long head) displays an initial burst of activity concurrent with triceps brachii, a monoarticular elbow extensor which acts as agonist to the extension movement. The biceps muscle then displays a second burst of activity concurrent with pronator teres, a monoarticular forearm pronator which acts as antagonist to the supination movement. Similarly, Figure 3.12b depicts a flexion combined with a pronation, where biceps brachii (short head) displays an initial burst of activity with brachioradialis (an elbow flexor) and a later burst of activity with triceps brachii (an elbow extensor). Comparable findings are

presented in Figure 3.13 for Subject D during a flexion / pronation (panel A), and for Subject A during an extension / supination (panel B).

Figure 3.14 illustrates two instances where pronator teres is both agonist and antagonist. Panel A depicts a combined flexion / supination performed by Subject D. It can be seen that pronator teres exhibits a burst of activity at the same time as biceps brachii (an agonist to the flexion and supination movements) and a later, reduced burst of activity at the same time as pronator quadratus (an antagonist to the supination movement). Panel B depicts an extending pronation (Subject E) where pronator teres displays a small burst concurrent with triceps brachii and a second larger burst with biceps brachii. Figure 3.15 presents two further instances of pronator teres displaying both agonist and antagonist muscle activity during flexing supinations for Subject F (Panel A) and Subject I (Panel B).

This pattern was seen in all subjects for pronator teres; however, it was not present in all trials. For biceps brachii this pattern was seen in all but two of nine subjects, but again was not present in all trials. A detailed examination of the frequency of this behaviour was undertaken for two subjects. For subject H, it was found that in 69% of the trials involving flexing supination or extending pronation, pronator teres displayed both agonist and antagonist components. For this same subject, biceps brachii showed both agonist and
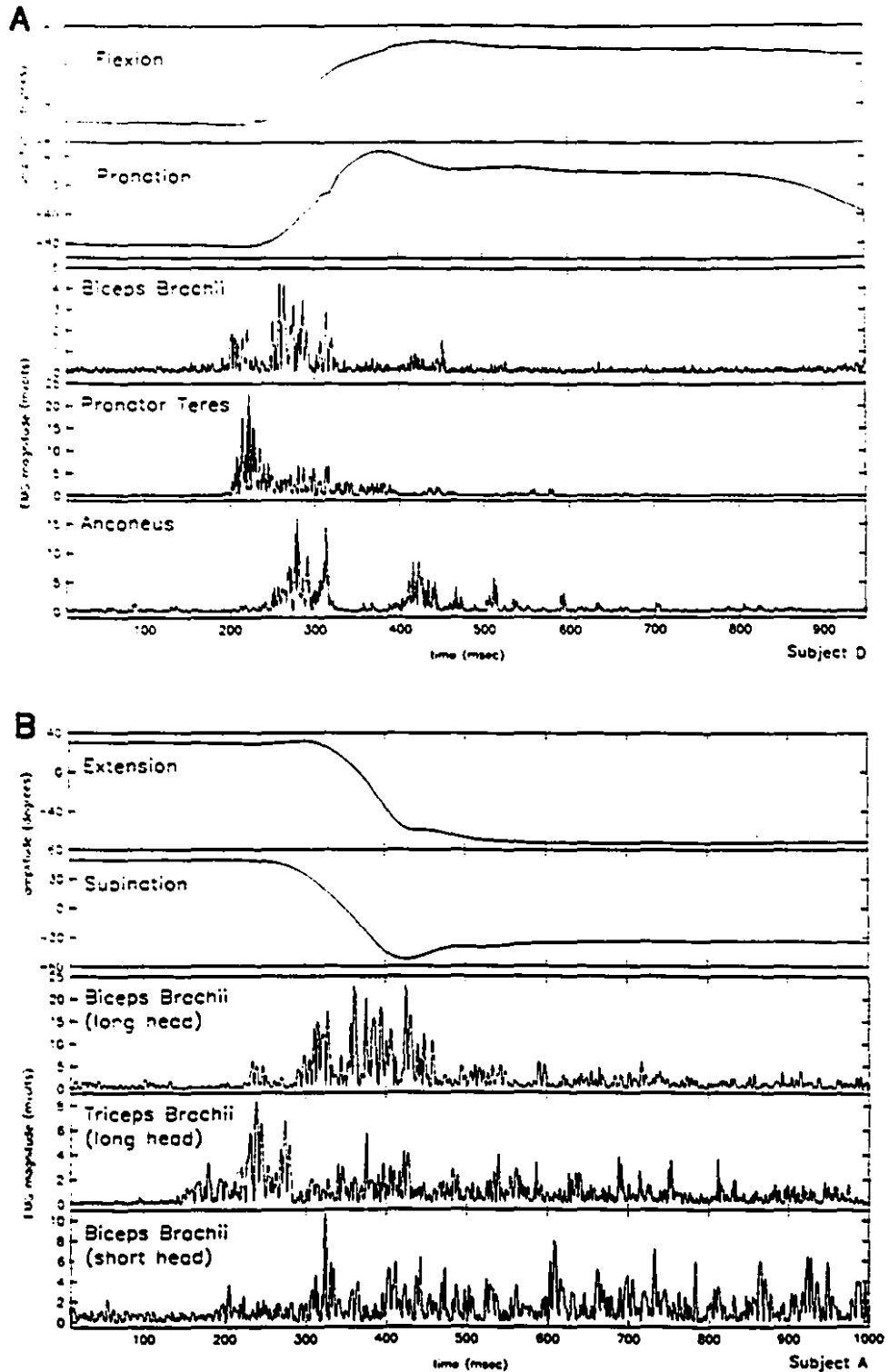
Figure 3.13: Kinematic patterns and EMG activity for two different two degree of freedom movements in which biceps brachii acts as agonist to motion in one degree of freedom and antagonist to motion in the second degree of freedom. In both Panel A (Subject D) and Panel B (Subject A) biceps brachii displays both agonist and antagonist EMG activity components.

Figure 3.14: Kinematic patterns and EMG activity for two different two degree of freedom movements in which pronator teres acts as agonist to motion in one degree of freedom and antagonist to motion in the second. A. Pronator teres displays a large agonist burst concurrently with biceps and a smaller antagonist burst with pronator quadratus during a flexing supination. D. The opposite pattern is seen in a trial involving an extending pronation. Pronator teres displays a small agonist burst and a larger antagonist burst. Data from two different subjects are shown.
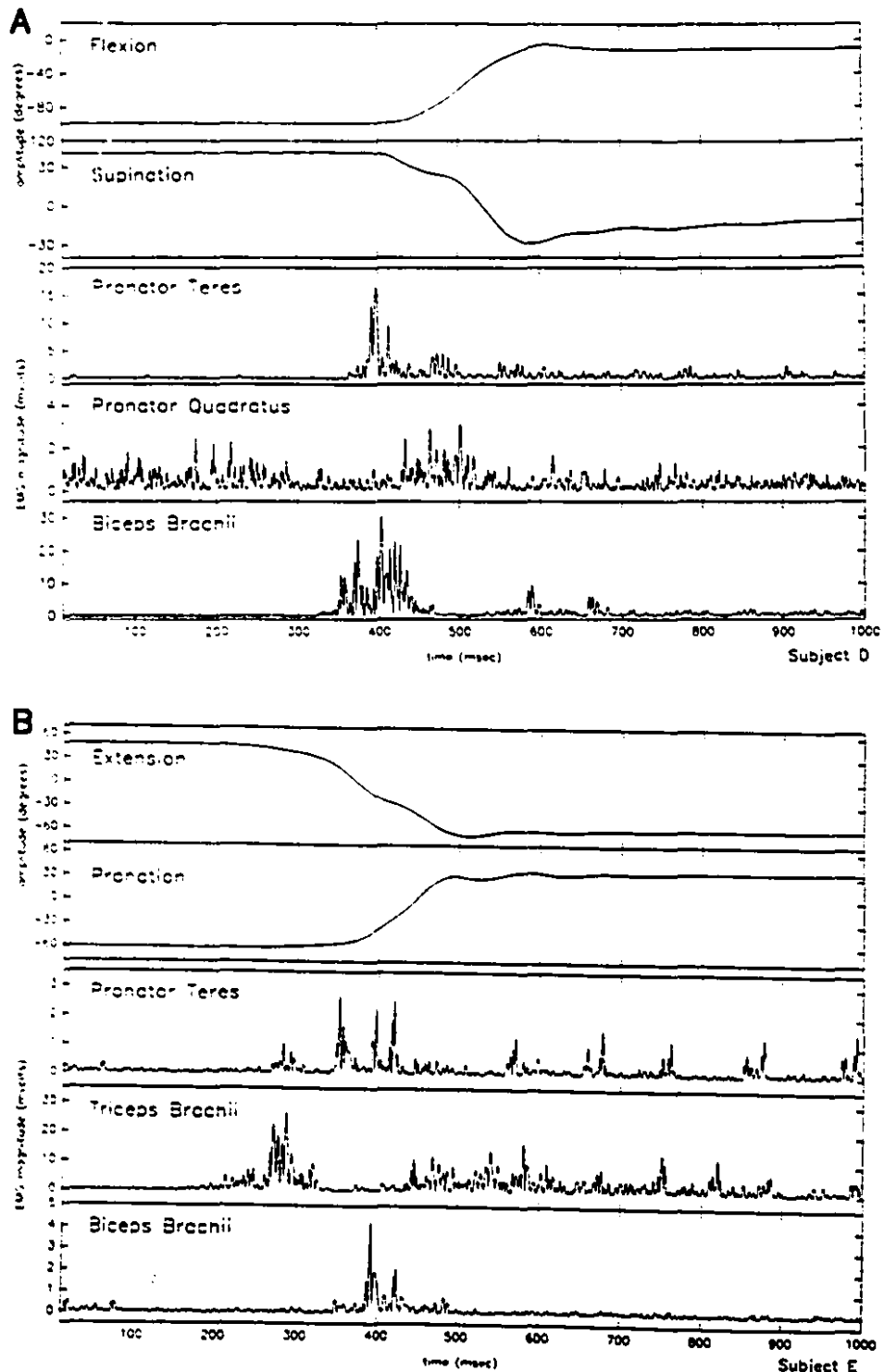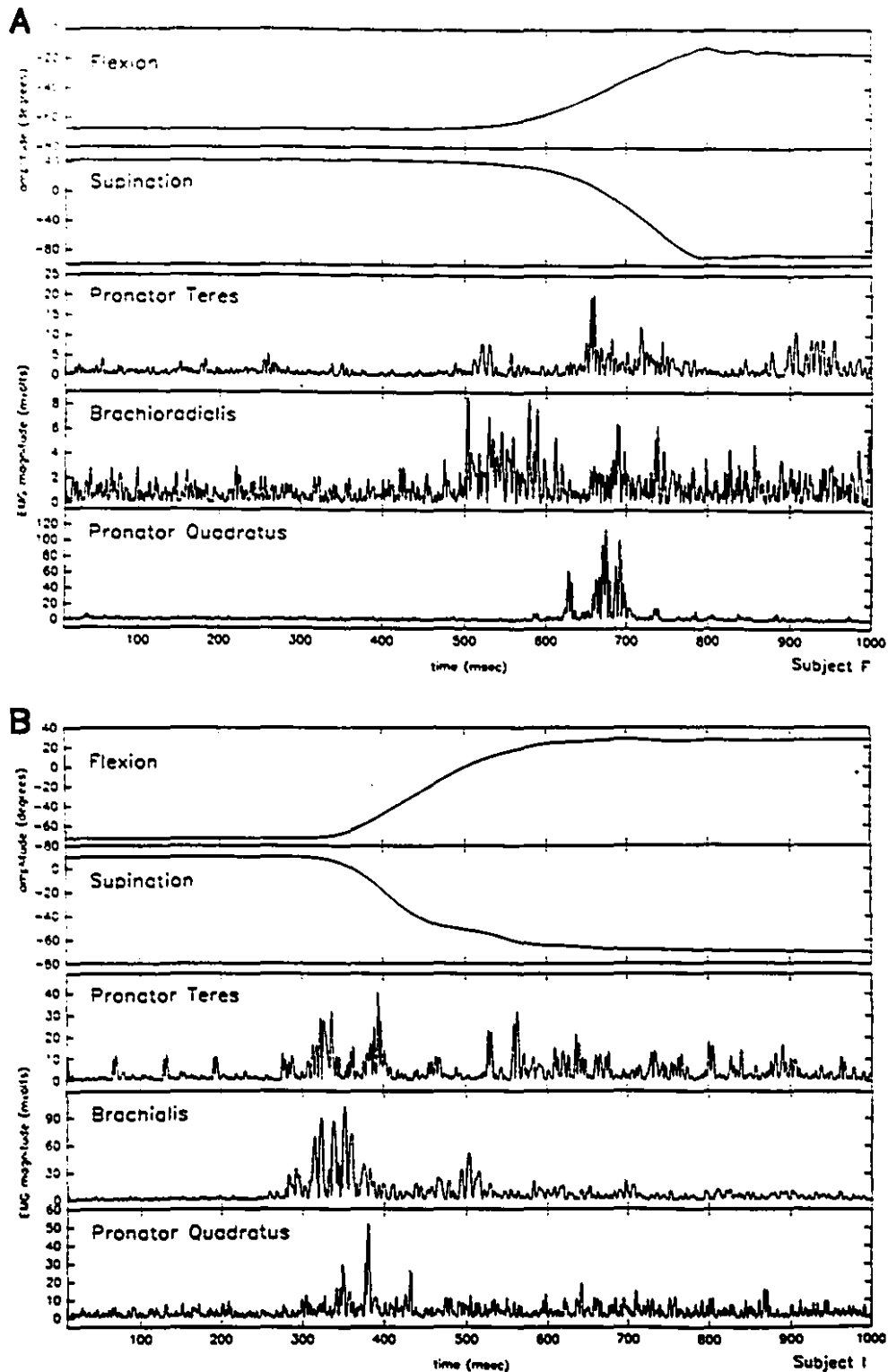
Figure 3.15: Kinematic patterns and EMG activity for two different two degree of freedom movements in which pronator teres acts as agonist to motion in one degree of freedom and antagonist to motion in the second degree of freedom. In both Panel A (Subject F) and Panel B (Subject I) biceps brachii displays both agonist and antagonist EMG activity components.

antagonist activity in 39% of trials involving flexing pronation or extending supination. For subject F, pronator teres displayed both agonist and antagonist components within a single movement in 55% of the trials where it was agonist to motion in one degree of freedom and antagonist to motion in the second. Biceps brachii exhibited agonist and antagonist activity 40% of the time.

Five subjects were tested in Experiment 4 in conditions designed to study biarticular muscles acting as agonist in one degree of freedom and antagonist in the second. In this study, motion in the second degree of freedom was systematically varied to assess the effect of movement amplitude on the magnitude of the EMG signal (both agonist and antagonist components) in these movement conditions. The remainder of this portion of the Results section presents data from Experiment 4 only.

While biarticular muscles displayed both agonist and antagonist activity within the same movement, the magnitude of one burst was almost always greater than the other. As an example, in Figure 3.14a, pronator teres has an agonist burst whose magnitude is greater than its antagonist burst. In Figure 3.14b the opposite pattern is exhibited; the antagonist burst is larger than the agonist burst for pronator teres. The one which was larger was not directly dependent on the amplitude of motion in each degree of freedom. For example,

during a series of 125° supinations where the amplitude of flexion movement was increased each trial, pronator teres (a flexor and pronator) displayed an antagonist burst whose magnitude was greater than that of the agonist burst when the amount of flexion was less than 75°. When the amount of flexion exceeded 75° the agonist burst was greater in magnitude than the antagonist burst, even though the amplitude of the supination movement was greater (125°). Thus the crossover point where antagonist activity exceeds agonist activity in magnitude occurred when the amplitude of movement in the pitch degree of freedom was 50° less than the amplitude of movement in the roll degree of freedom. This pattern was seen for pronator teres in four out of five subjects who performed this portion of the experiment. For these subjects, the crossover point occurred at a flexion / extension amplitude that ranged from 15° to 55° less than the supination / pronation amplitude. In the remaining subject (Subject I), the magnitude of the pronator teres antagonist burst was always less than its agonist burst.

Similar behaviour was exhibited by biceps brachii. The EMG pattern associated with motion in the flexion / extension degree of freedom predominated that in the pronation / supination degree of freedom in relation to which burst magnitude was greater. In Figure 3.12a the magnitude of the second biceps brachii burst, which occurs with pronator teres as antagonist to the extension / supination movement, is greater than the initial agonist burst

component. Figure 3.12b shows an instance, for a subject from Experiment 4, where the magnitude of both biceps brachii bursts are approximately equal (although the area under each burst reveals that the magnitude of the agonist component is indeed greater). The pattern was seen in other manipulations as well, in which subjects performed a series of discrete flexing pronations in which the amplitude of flexion motion was held constant at 85° while the amplitude of pronation motion was increased in each trial. It was only when the amplitude of the pronation movement reached 110° that the biceps brachii displayed an antagonist component that was greater in magnitude than the agonist component. That is, the crossover point where biceps brachii displayed a greater magnitude antagonist burst component occurred only after the amplitude of motion in the pronation / supination degree of freedom was 25° greater than in the flexion / extension degree of freedom. This pattern of behaviour in biceps brachii was exhibited by all five subjects tested in Experiment 4.

The point at which muscles switched from exhibiting greater agonist to greater antagonist activity, or vice versa, was assessed by examining the onset time of the larger of the two bursts. Figure 3.16a shows the time of burst onset for biceps brachii (long head), brachialis, and triceps brachii (long head) relative to movement onset for a series of discrete movements where the amplitude of pronation is held constant while the amplitude of flexion is

increased. Data are shown for Subject H. Note again that it is the onset time

of the larger of the two biceps bursts that is shown here. It can be seen that

the point at which the onset of the larger biceps brachii burst switches from

aligning with the agonist to aligning with the antagonist is at a flexion angle

(60°) that is 45° smaller than the pronation angle (105°). A second example is

illustrated in 3.16b for Subject G. In this series of movements, the amplitude

of flexion motion is maintained constant at 85° while the amplitude of pronation

motion is increased in each trial. Once again, the point at which the larger of

the two biceps brachii bursts changes from aligning with brachialis, a pure

agonist, to aligning with triceps brachii, a pure antagonist, is at a flexion angle

that is 25° less that the pronation angle.

In a similar manner, for a series of discrete flexing supinations, pronator

teres activity switches from being predominantly antagonist to predominantly

agonist at a flexion amplitude that is 50° smaller than the supination amplitude

for Subject F (Figure 3.17a), and 35° smaller than the supination amplitude for

Subject A (Figure 3.17b). In general, we observed that the identity of the larger

of the two bursts switched when the amplitude of movement in the flexion /

extension degree of freedom was smaller than the amplitude of movement in

the pronation / supination degree of freedom by an average of 35° for pronator
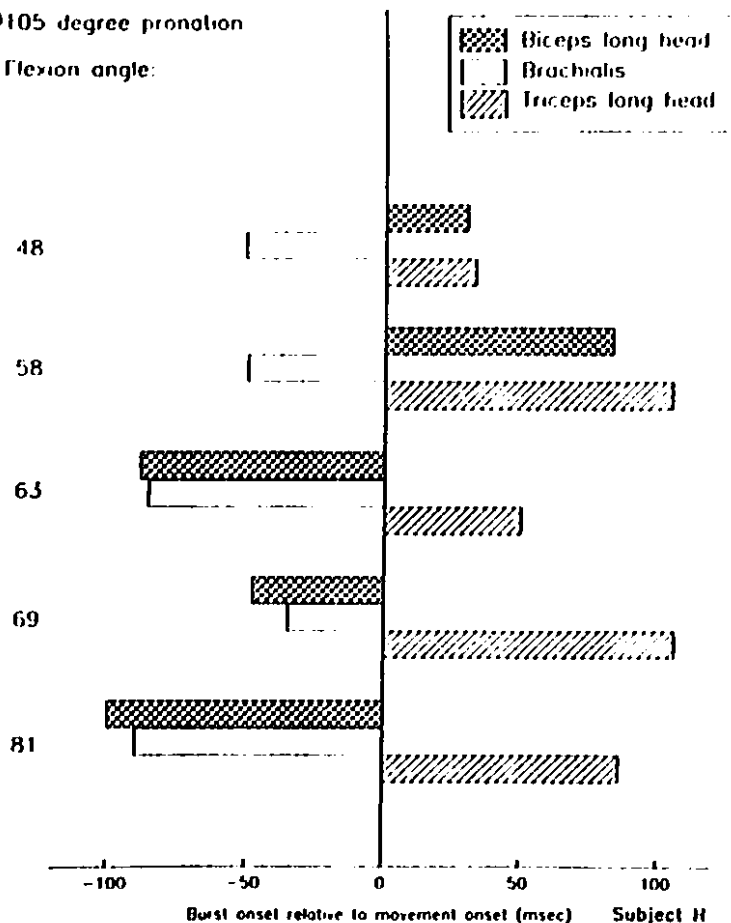
Figure 3.16. Experiment 4: Burst onset time relative to movement onset for a series of discrete two degree of freedom movements. Data are shown for two different subjects. Each set of three bars shows the burst onset during a single trial for three muscles: a biarticular (larger burst shown), a pure agonist, and a pure antagonist. A: Pronation movement amplitude is constant while flexion angle is increased. B: Flexion angle is held relatively constant while pronation angle is increased for each trial.

A 125 degree supination

Flexion angle:

	Pronator teres
	Biceps long head
	Pron. Quadratus

93°

88°

75°

73°

71°

61°

55°

52°

-100   -50   0   50   100   150

Burst onset relative to movement onset (msec)   Subject F

B 135 degree supination

Flexion angle:

	Pronator teres
	Bicep long head
	Triceps long head

142

123

110

98

82

-100   -50   0   50   100

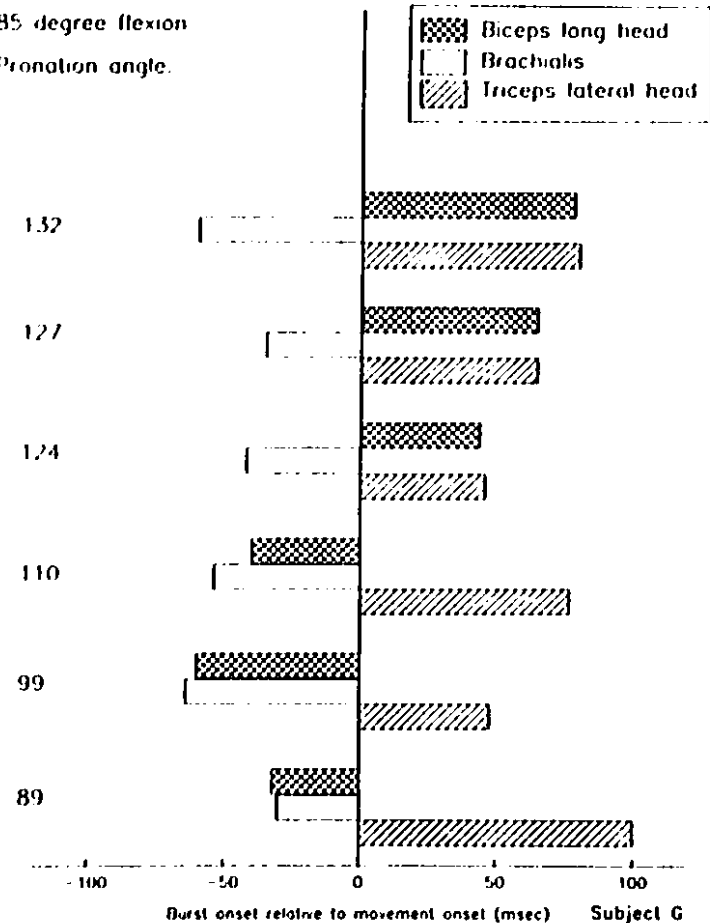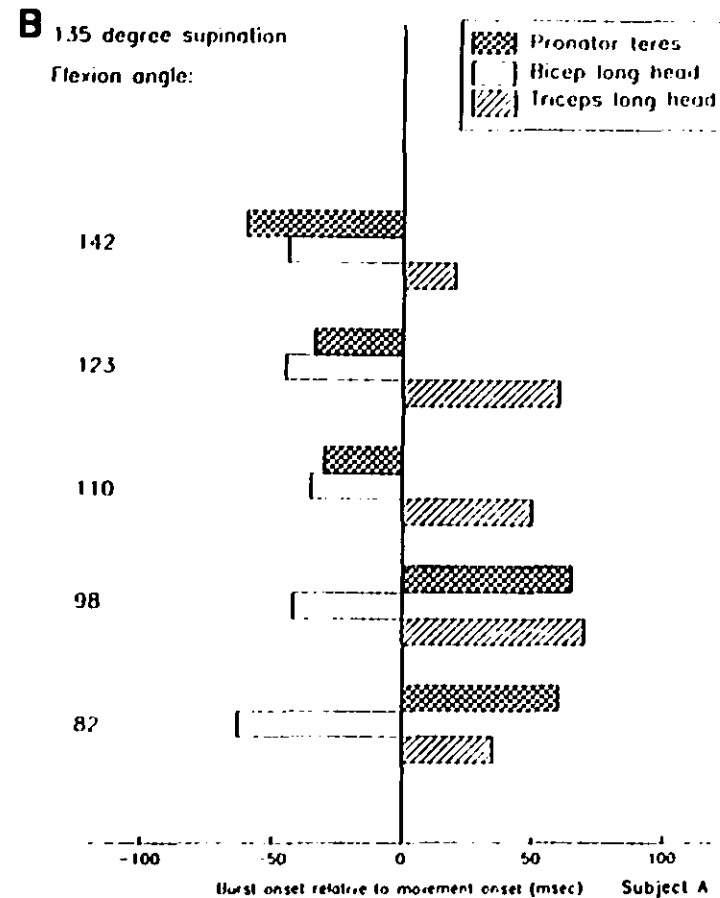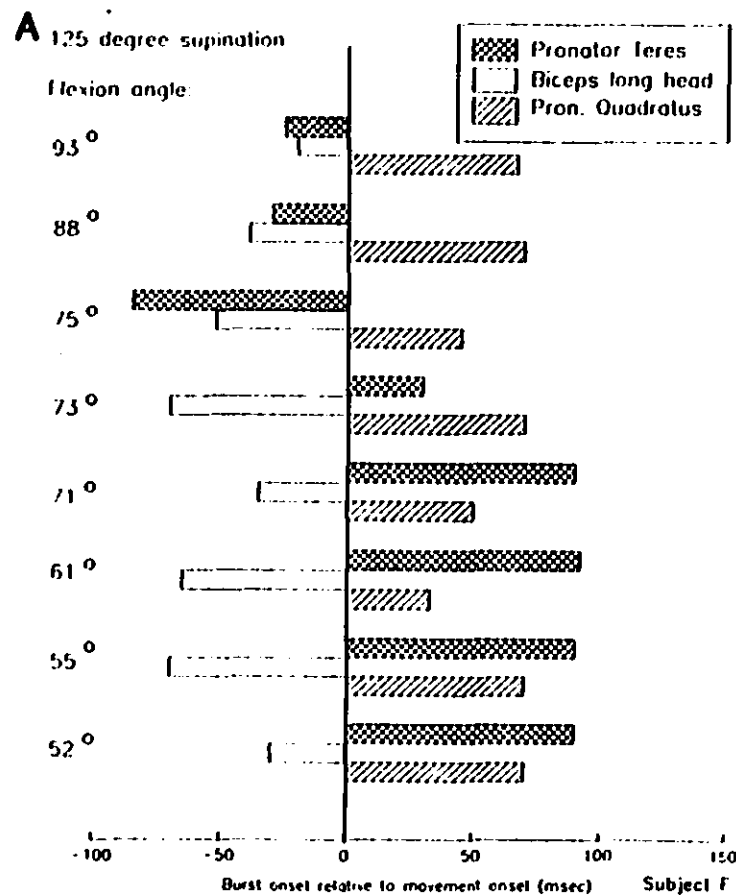Burst onset relative to movement onset (msec)   Subject A

Figure 3.17. Experiment 4: Burst onset time relative to movement onset for a series of discrete two degree of freedom movements. Data are shown for two different subjects. Each set of three bars shows the burst onset during a single trial for three muscles: a biarticular (pronator teres), a pure agonist, and a pure antagonist. For pronator teres, the onset time is shown for the larger of its two bursts. A: Note that, for a series of 125° supinations, the flexion angle at which pronator teres changes from displaying a larger antagonist burst to displaying a larger agonist burst is less than 125°. B: For a series of 135° supinations, pronator teres begins to display agonist activity at a flexion angle that is less than 135°.

teres (four subjects) and 40° for biceps brachii (five subjects).

Two of the five subjects (Subjects H and I) repeated the movements from a more flexed starting position. Whereas the movements shown in Figures 3.12 through 3.17 were performed starting with the elbow at -70° (-90° = fully extended), these movements were performed starting with the elbow at -40°. Both pronator teres and biceps brachii continued to show both agonist and antagonist components in EMG activity. Similarly, both muscles displayed a crossover from a greater antagonist burst magnitude to a greater agonist burst magnitude which was not directly dependent on the movement amplitude in the two degrees of freedom. The magnitude of the agonist burst in both muscles was smaller in the more flexed initial position in one of two subjects (Subject H, $P < 0.01$).

It has been observed in movements in which biarticular muscles act as agonist in one degree of freedom and antagonist in the other that the muscle is active regardless of the amplitude of motion in each degree of freedom. Figure 3.18 shows the kinematics and corresponding EMG activity for a series of ten flexing pronations performed rhythmically. The top two traces show the amplitude of movement in the flexion / extension and supination / pronation degrees of freedom. The third and fifth traces show the rectified EMG activity for biceps brachii and pronator teres, and the fourth and sixth traces show the

Pitch

60
0
-60

Roll

40
0
-40
-80

<-Sup.Pro-> <-Ext Flex-> amplitude (deg.)

Biceps Brachii (long)

raw data

Biceps Brachii (long)

20
15
10
5
0

Pronator Teres

raw data

Pronator Teres

9
6
3
0

EMG magnitude

iEMG(mvolts)

iEMG(mvolts)

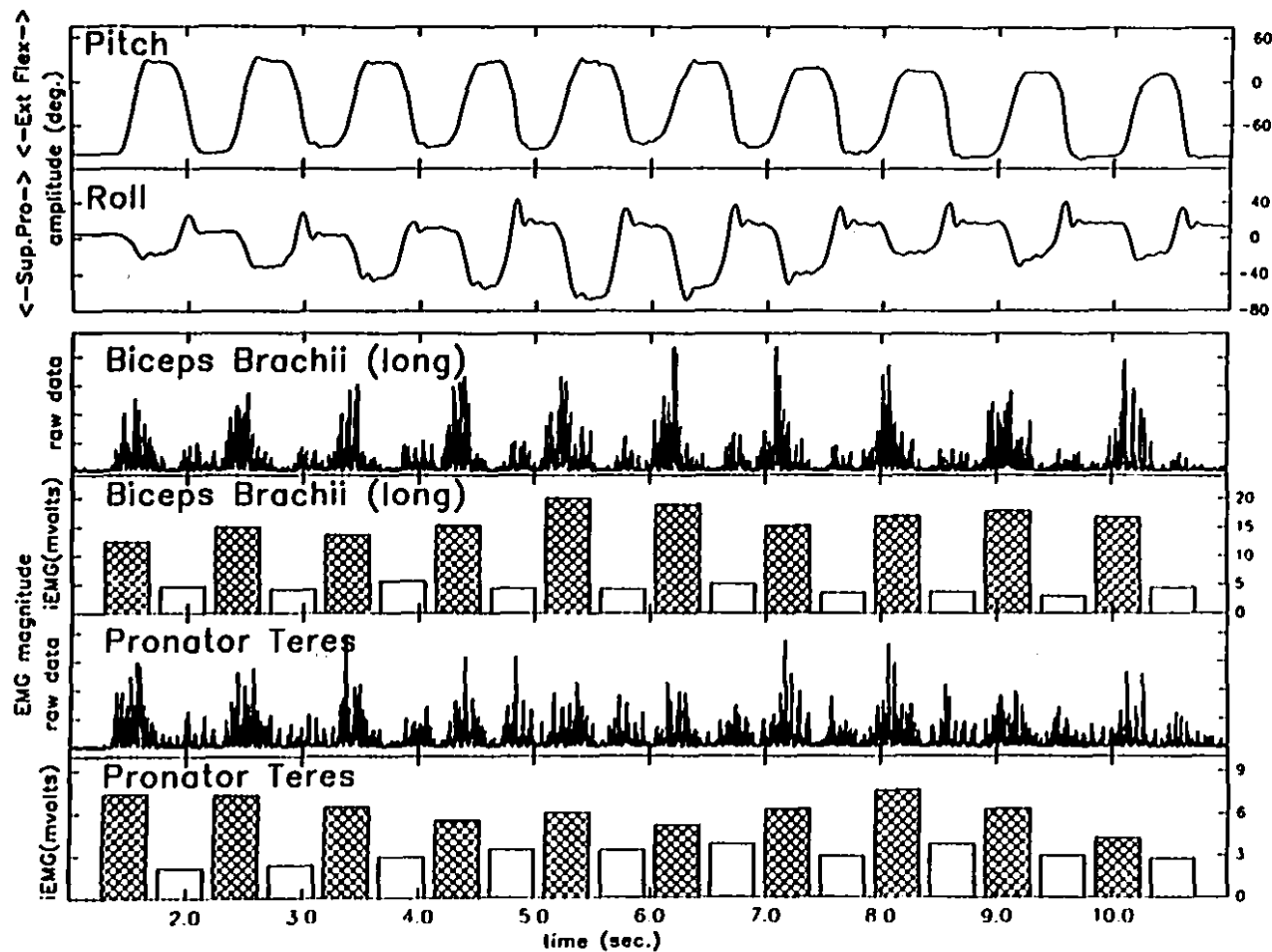2.0   3.0   4.0   5.0   6.0   7.0   8.0   9.0   10.0

time (sec.)

Figure 3.18. Kinematic patterns and EMG activity for a series of 10 rhythmic flexions and extensions performed simultaneously with pronations and supinations. The top two traces show the amplitude of flexion / extension and pronation / supination movements, respectively. The magnitude of each muscle burst is represented by the height of the bars directly underneath each EMG trace. Cross-hatched bars correspond to bursts occurring during the flexing pronations, open bars correspond to bursts occurring during the extending supinations. Data shown are for one trial (Subject A).

corresponding magnitudes of each burst for the two muscles. The amplitude of flexion / extension was held constant (75°) while the amplitude of supination / pronation was gradually varied (40° - 130°). In this situation, pronator teres acts as agonist in one degree of freedom and antagonist in the other, be it a flexing supination or and extending pronation (biceps brachii acts as agonist or antagonist in both degrees of freedom). The figure shows that pronator teres exhibits activity during both the flexion / supination phases and the extension / pronation phases. At no point is the phasic activity eliminated, even when the amplitude of the supination or extension is greater than the amplitude of the flexion or pronation. All five subjects show a similar pattern for pronator teres. Likewise, all subjects display this behaviour for biceps brachii during rhythmic flexing pronations coupled with extending supinations.

TIMING OF BURST ACTIVITY ONSET IN ONE AND TWO DEGREE OF FREEDOM ELBOW MOVEMENTS RELATIVE TO MOVEMENT ONSET

The timing of muscle burst activity relative to arm movement onset was examined in two ways: the onset of activity burst for a given muscle across all movement conditions, and the onset of activity burst for a given movement condition across all muscles. As described in section 3.1, initially muscles were classified qualitatively as displaying agonist burst activity, antagonist burst activity, tonic activity, or no activity in each movement condition. Biarticular

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muscles displaying both agonist and antagonist activity were classified according to which burst was larger in magnitude for purposes of this analysis. Muscles displaying agonist burst activity were subsequently analyzed separately from muscles displaying antagonist burst activity.

Burst onset relative to movement onset for a given movement condition across muscles

Both when movement timing was explicitly controlled through use of an audio metronome and when it was not explicitly controlled, the pattern of EMG timing was similar. For a given discrete movement, no differences in muscle burst onset time were observed either for muscles acting as agonists or for muscles acting as antagonists (p > 0.01, for both agonists and antagonists). All five subjects tested in the two timing conditions (Experiments 1 and 2) as well as the three subjects tested with the upper arm held horizontally (Experiment 3) displayed this pattern. Figure 3.19 displays muscle burst onset times relative to movement onset for all eight muscles recorded from Subject D in Experiment 1. Three different movements are shown. As an example, during a flexing supination (top panel), biceps brachii (both long and short heads) and brachialis all increased their activity approximately 40 ms prior to movement onset, while triceps brachii (both medial and lateral heads), anconeus, pronator quadratus, and pronator teres increased their activity

approximately 60 ms after movement onset. It should be noted, however, that individual subjects showed occasional onset differences in particular muscles ($p < 0.01$). These differences would typically only occur within one movement condition and there were no instances where a particular muscle displayed an onset time that was significantly different from the other co-agonists or co-antagonists across all subjects. To illustrate, in the middle panel of Figure 3.19, it can be seen that for a flexing pronation, the onset of biceps brachii (short head) was significantly earlier than the onset of pronator teres ($p < 0.01$). However, no other subjects displayed this pattern during a flexion pronation. Figure 3.20 depicts the onset time of the first agonist and antagonist bursts relative to the onset time of arm motion for three representative movements performed by Subject A in Experiment 2. The time of burst onset for all eight muscles recorded from this subject are shown. It can again be seen that when movement time was explicitly controlled, muscles which act as agonists (bars to the left of 0) all increase their activity at approximately the same time, while all muscles which act as antagonists (bars to the right of 0) increase their activity at the same time in the three movements.

Burst onset relative to movement onset for a given muscle across movements

Individual muscles displayed burst onset times which did not vary across movement conditions in which the muscle acted as agonist ($p > 0.01$).
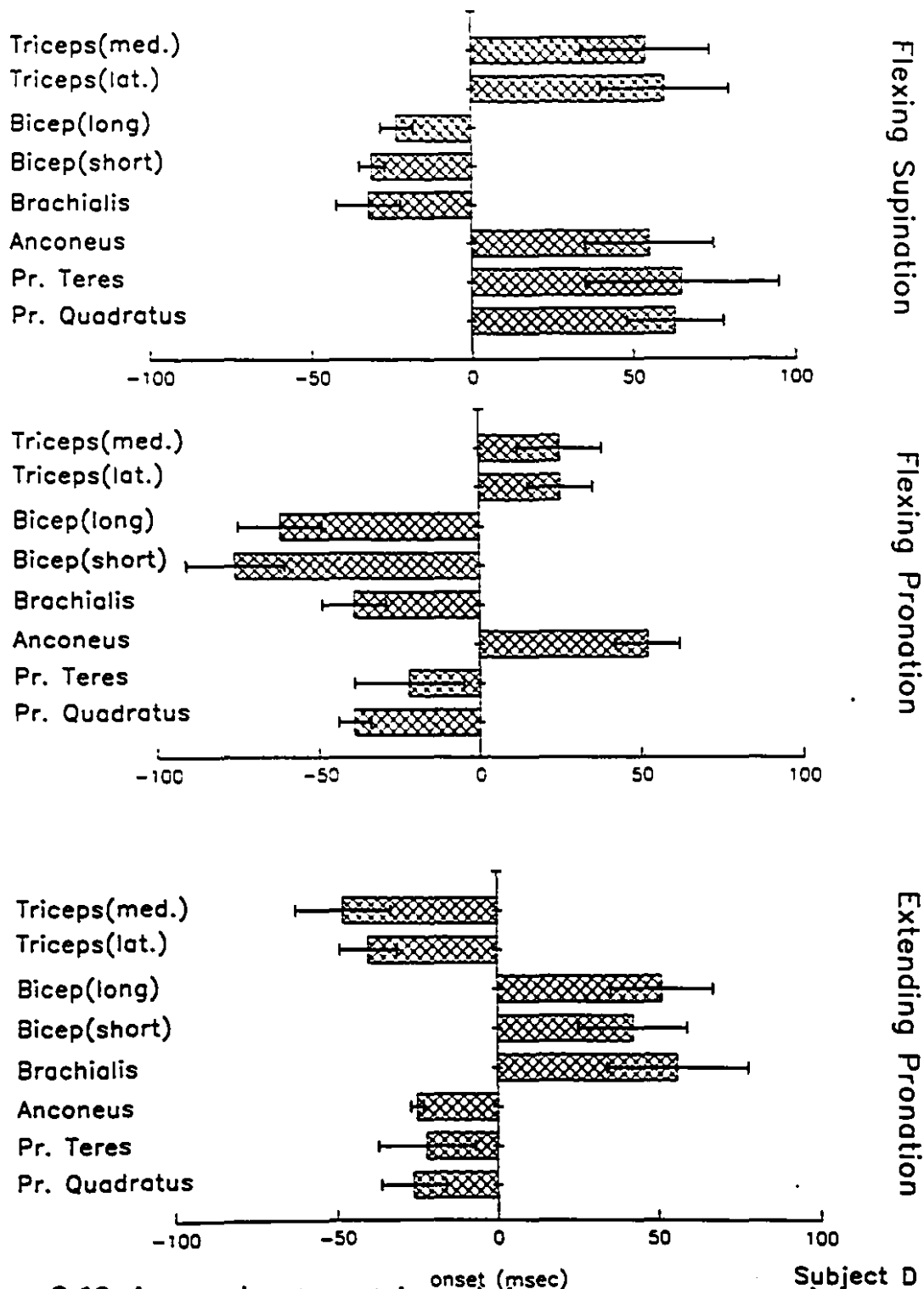
Figure 3.19. Average burst onset time relative to movement onset time across eight muscles for three different movements. Bars to the left of 0 are agonists, bars to the right of 0 are antagonists. All muscles acting as agonists increase activity at approximately the same time. Muscles acting as antagonists show the same pattern. Data shown are from Experiment 1, Subject D.
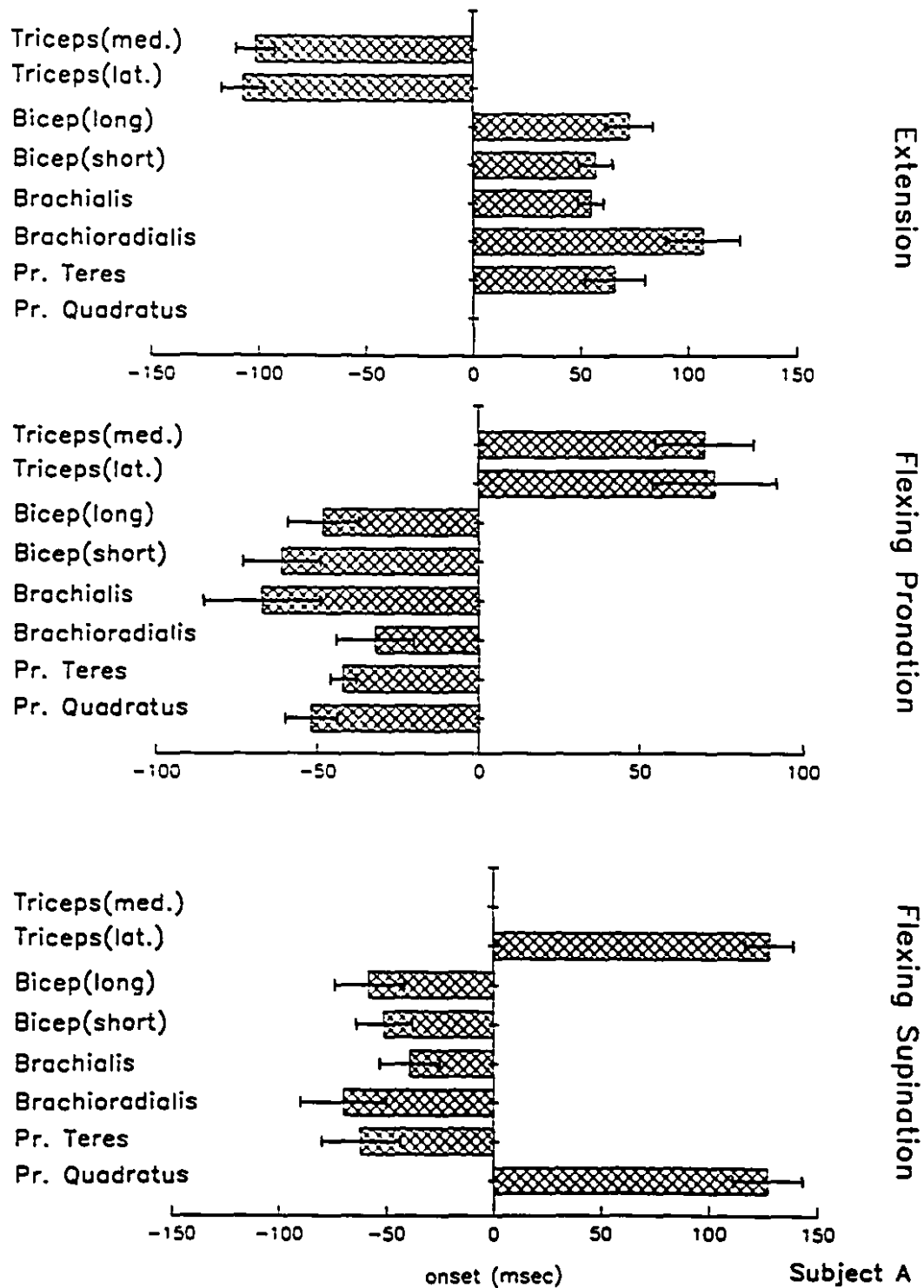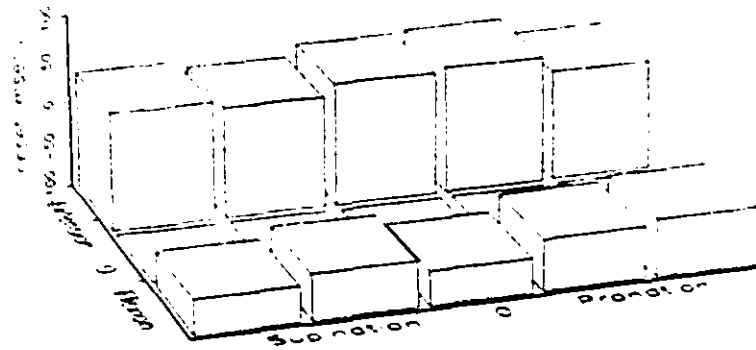
Figure 3.20. Average burst onset time relative to movement onset time across eight muscles for three different movements. Bars to the left of 0 are agonists, bars to the right of 0 are antagonists. All muscles acting as agonists increase activity at approximately the same time. Muscles acting as antagonists show the same pattern. Data shown are from Experiment 2, Subject A.

Similarly, no differences among burst onset times were found when the muscle was classified as antagonist (p > 0.01). This pattern held for all subjects tested in the two timing conditions as well the three subjects who performed the experiment with the upper arm held horizontally.

Figure 3.21 shows burst activity onset times over different amplitudes of flexion / extension and pronation / supination for triceps (long head), biceps (long head), and pronator quadratus. Data shown are from Experiment 1, Subject A. It can be seen that for triceps brachii (top panel), the taller bars which represent those movement conditions in which the muscle acted as antagonist are all essentially the same height (corresponding to a burst onset time of approximately 45 msec after movement onset). The shorter bars, which represent those movement conditions for which triceps brachii acted as agonist, are all of similar height as well (corresponding to a burst onset time of approximately 50 msec prior to movement onset). Areas on the figure which are flat represent those movement conditions for which the muscle displayed neither agonist or antagonist burst activity. Comparable behaviour can be seen for the other two muscles in Figure 3.21, and was observed for all other muscles tested.

Figure 3.22 displays muscle burst onset times relative to movement onset times across all movement conditions for three muscles recorded during

Triceps (long head)

Biceps (long head)
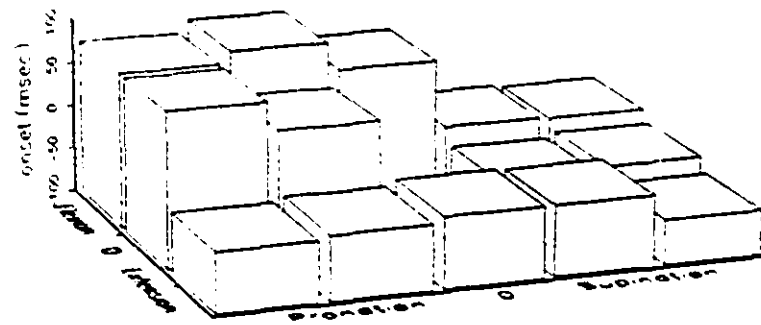
Pronator Quadratus

Figure 3.21. Burst onset time relative to movement onset time for three muscles across all movement conditions. Each bar represents the average of all trials within one movement condition. Taller bars correspond to antagonist movement conditions, shorter bars correspond to agonist movement conditions. Flat areas indicate lack of phasic activity. Data are from Experiment 1, Subject A.
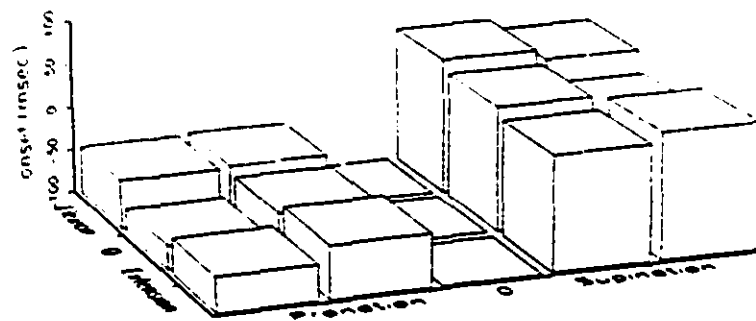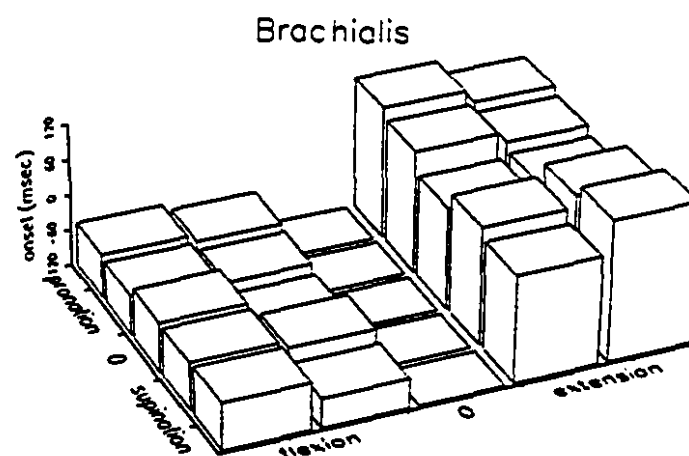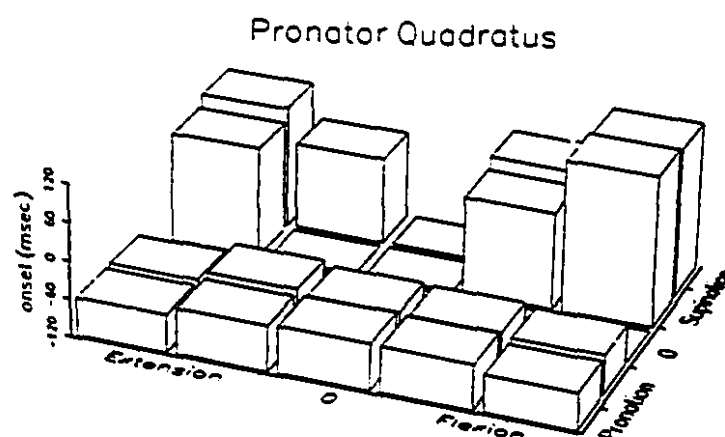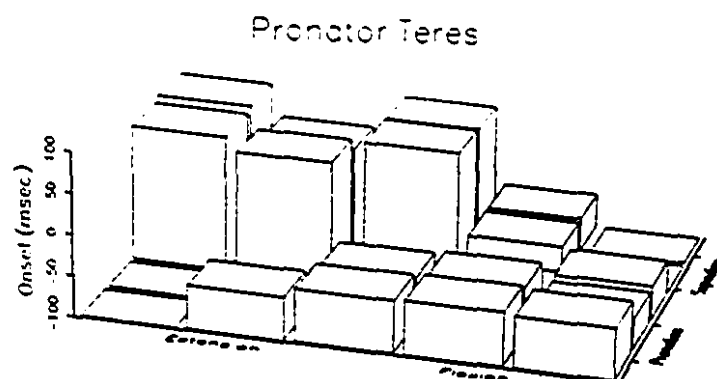
Figure 3.22. Burst onset time relative to movement onset time for three muscles across all movement conditions. Each bar represents the average of all trials within a single movement condition. Taller bars correspond to those movement conditions in which the muscle acts as antagonist, shorter bars correspond to the conditions in which the muscle acts as agonist. Flat areas indicate lack of phasic activity for that muscle. Data shown are from Experiment 2, Subject E.

Experiment 2. Data are shown for Subject E. Note that there a greater number

of bars in each panel reflecting the greater number of movement conditions

performed in the second experiment. It can be seen, for example, that in all

movement conditions for which pronator teres (top panel) acts as agonist the

bars are approximately the same height. The same is true for all movement

conditions for which the pronator teres acts as agonist. Again, similar findings

are observed for pronator quadratus (Figure 3.22, middle panel), brachialis

(Figure 3.22, lower panel) and all other muscles recorded, across all subjects.

When movement time was not explicitly controlled, although there were

no significant differences in an individual muscles' burst onset times, the overall

variability of onset times was greater than when movement time was

controlled. For example, the agonist burst onset mean and standard deviation

for biceps brachii long head (Subject B) was -45 ± 20 msec when timing was

explicitly controlled, and -47 ± 42 msec when it was not controlled. Other

subjects showed comparable patterns.

DURATION OF MUSCLE BURSTS

To investigate the possibility that differences in EMG burst magnitudes

were due, in part, to differences in burst duration, both agonist and antagonist

muscle burst durations were examined in different movement conditions for

Experiments 1, 2 and 3. Agonist burst durations were analyzed separately from antagonist burst durations.

Within a condition, the duration of the individual muscle bursts varied between 90 and 160 milliseconds. However, the average burst duration did not vary across movement conditions ($p > 0.01$). Movement durations across movement conditions are depicted in Figure 3.23 for three muscles from Experiment 1 (Subject C), and in Figure 3.24 from Experiment 2 (Subject E). In Figure 3.23, bars corresponding to average burst durations for each of the movement conditions are all seen to be nearly the same height. Note that, although there is no statistical difference in burst duration, there is trend towards greater duration with motion in either direction in a second degree of freedom (observe the "V" shape among many rows of bars in the two figures). In contrast to Figure 3.23, where bars corresponding to burst duration were similar in height, figure 3.24 depicts both biceps (short head) and brachialis burst durations which fall into two groups. The taller bars (and, hence, longer burst durations) correspond to those movement conditions for which these muscles act as agonist, while the shorter bars represent those movement conditions in which these muscles act as antagonist. In general, it was observed that for a number of muscles, the duration of the agonist burst was longer than the duration of the antagonist burst by approximately 80 msec. Within all movement conditions for which a muscle acted as agonist, however,

the movement durations were not significantly different ($p > 0.01$). This behaviour was observed in all subjects both when movement timing was explicitly controlled and when it was not. This was also the case for the three subjects who repeated the experiment with the upper arm held in a horizontal position. Thus the variation in agonist burst magnitude noted above was not due to an increase in burst duration but presumably to an increase in the amplitude of the burst.

Figure 3.23. Average burst duration for three muscles across all movement conditions. Each bar represents the average duration of all trials within a single movement condition. Flat areas indicate lack of phasic activity for that muscle. Data are from Experiment 1, Subject C.
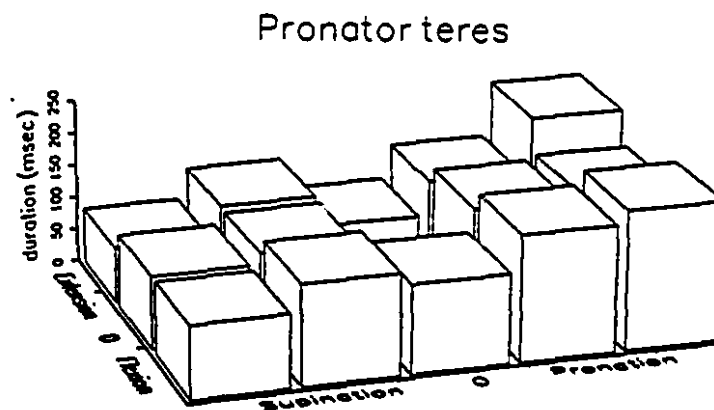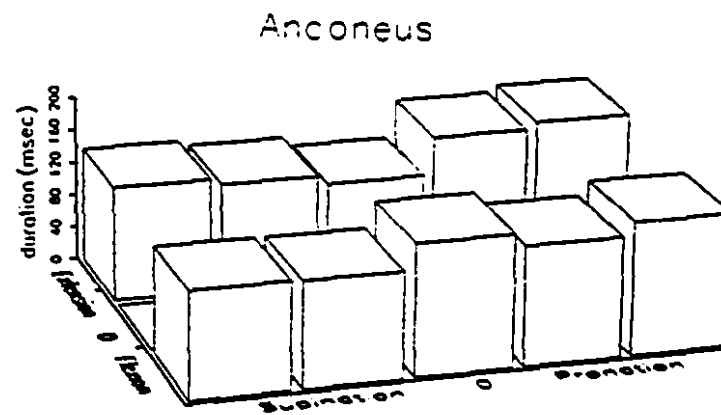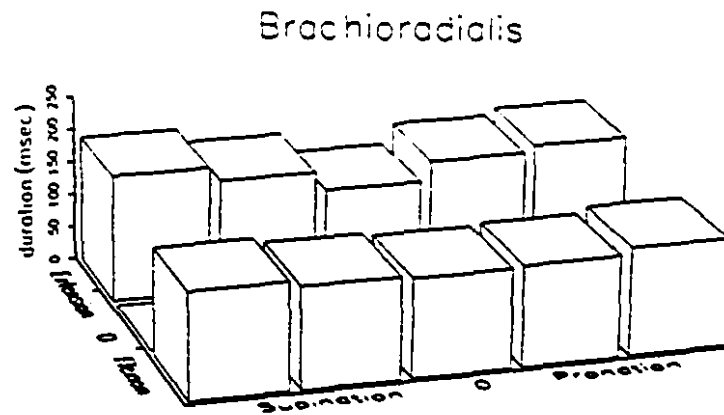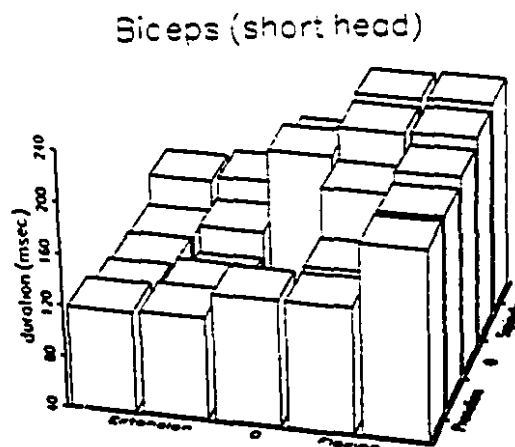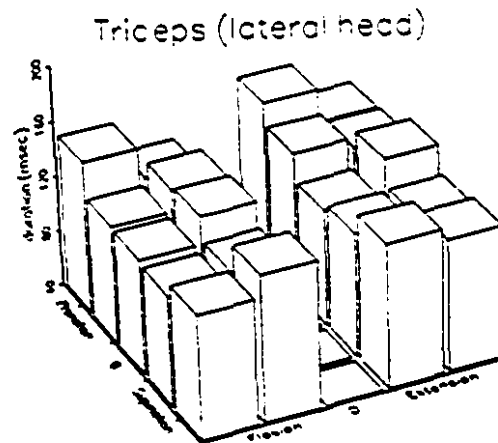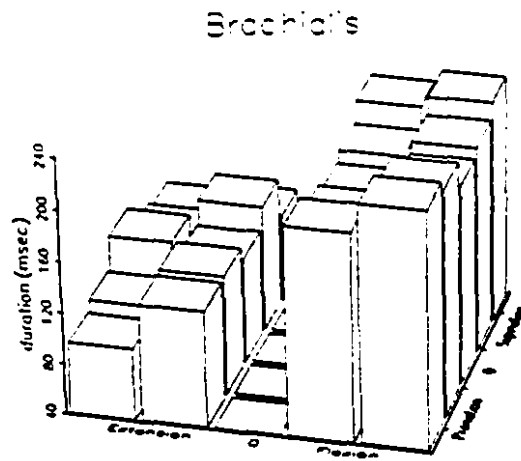
Figure 3.24. Average burst duration for three muscles across all movement conditions. Each bar represents the average duration of all trials within a single movement condition. For brachialis and biceps brachii, taller bars correspond to the condition in which the muscle acts a agonist. Flat areas indicate lack of phasic activity for that muscle. Data are from Experiment 2, Subject E.

4. DISCUSSION

Electromyographic correlates of motion in two degrees of freedom about the elbow were examined in this thesis. It was found that muscles fall into two categories with respect to agonist burst magnitude: those whose burst magnitude varies with motion in a second degree of freedom in a graded fashion and those whose burst magnitude does not. It was also observed that during movements in which biarticular muscles act as agonist to motion in one degree of freedom and antagonist in the other, the muscle activity has both agonist and antagonistic components. In this situation, the identity of the burst displaying a greater magnitude is not determined solely by the amplitude of motion in each degree of freedom. Lastly, it was seen that the onset and duration of muscle activity did not vary with motion in a second degree of freedom. This was observed both when timing was explicitly controlled and when it was not.

SUPERPOSITION OF COMMANDS FOR MOTION IN DIFFERENT DEGREES OF FREEDOM

Movements in the two degrees of freedom about the elbow generally subserve different functions and appear to be controlled separately. Flexion and extension about the elbow act to bring the arm to a specific location, while rotating the forearm acts to orient the hand. The production of motion in these

two degrees of freedom is interdependent due to a number of biarticular muscles. Nevertheless, many studies have suggested that reaching movements (involving shoulder and elbow flexion / extension) and rotation of the forearm may be planned separately (Jeannerod and Biguer 1982; Lacquaniti and Soechting 1982; Perenin and Vighetto 1988; Soechting and Flanders 1993).

A number of lines of evidence have been presented here which likewise suggest that control of elbow movements is organized in terms of commands for motion in individual degrees of freedom. These control signals may act alone or be superimposed. (As noted previously, however, EMG activity is a dependent measure and inferences about control on the basis of EMG must be made with caution.)

One demonstration consistent with these ideas is that, in a number of muscles, agonist burst magnitudes observed in two degree of freedom arm movements were approximately the sum of those magnitudes seen in the individual movements which comprised them. The additivity seen in agonist burst magnitudes suggests that the central commands for motion in the component degrees of freedom may themselves be additive.

A second demonstration indicating superposition of central commands is that biarticular muscles may display activity during both agonist and

antagonist phases of the same movement. For example, during a flexing supination, pronator teres may fire together with muscles acting as pure agonist as well as with those acting as pure antagonists. This occurs regardless of which component motion - the flexion or the supination - is greater in amplitude. One might have expected the muscle to display purely agonist or antagonist activity depending on which degree of freedom was of greater amplitude, or required a greater amount of torque. The data show, however, that the muscles can synchronously act as agonist and antagonist. This may indicate that, even when a muscle must perform antagonistic functions in a single movement, the control signals subserving the individual degrees of freedom may be superimposed.

RELATING SINGLE DEGREE-OF-FREEDOM CONTROL SIGNALS TO INDIVIDUAL MUSCLE COMMANDS

If, as suggested, control is organized at the level of degrees of freedom, there must exist a mapping between control signals at that level and control signals at the level of commands to individual muscles. The present findings in conjunction with previous work on isometric two degree of freedom elbow movements may provide insight into the nature of this mapping. Under isometric conditions, van Zuylen et al. (1988) identified specific motor unit sub-populations whose recruitment thresholds were dependent on torques in

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individual degrees of freedom, or on combined torques in two degrees of freedom. Thus, control at the level of individual degrees of freedom may be mapped onto control at the level of individual muscles using these sub-populations of motor units as a neuro-anatomical substrate.

It is not possible to directly compare between previous isometric studies and the present anisometric study due to the effects of the force-velocity relationship and the change in lines of muscle action. The similarity in results may provide insight as to how sub-populations of motor units in muscles may serve as the neural substrate associated with EMG activity magnitude. For example, a muscle may contain sub-populations of motor units, some with recruitment thresholds that vary with motion in a second degree of freedom and some that do not. Activation of only single degree-of-freedom motor units will not affect the direction of arm movement differently from activation of two degree-of-freedom motor units: a muscle contraction will pull the muscle origin towards the muscle insertion regardless of which motor units are active. However, activation of a subset of motor units *will* affect the magnitude of activity in that muscle. Thus, a muscle architecture arranged on the basis of task-dependent subunits may provide a means by which the nervous system can regulate muscle activation at the level of kinematic degrees of freedom.

Previous studies examining two degree of freedom shoulder / elbow movements in the horizontal plane (Karst and Hasan 1991b) and in the sagittal plane (Flanders and Herrmann 1992; Flanders et al. 1994) have found that the timing of muscle activity varied for movements in different directions. In contrast, we have found that across all muscles tested the agonists start firing together and the antagonists likewise come on together. In studies such as those reported by Karst, Flanders, and colleagues, subjects typically produced point-to-point arm movements in which relatively straight line hand paths were observed. Asynchronies in the timing of joint motion and hence muscle activation are necessary to achieve such straight line hand paths. In the present study, orientation of the forearm did not alter the trajectory of the hand and thus subjects were not constrained to couple movements as in observed in target-directed pointing. Nor were subjects specifically constrained to produce simultaneous two degree of freedom movement at the elbow. One interesting finding of the present study is that, for movements in which timing was not explicitly controlled, synchronous agonist and antagonist burst onsets were nevertheless observed.

Previously, it has been noted that forearm rotation is uncoupled from elbow motion (Lacquaniti and Soechting 1982). That is, the torques produced

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by rotation of the forearm do not affect the torques produced during flexion and extension of the arm. In a system such as that involving shoulder and elbow, variable muscle burst onsets may help produce smooth movement in the face of inertial, coriolis, and other interaction torques. In coordinating multiple muscle activity patterns in the present type of movements, it may be unnecessary to stagger muscle onsets. Synchronous timing of muscle bursts seen in the present study may simply reflect this uncoupling.

Muscle burst durations were found to not vary over the different movement conditions. This indicates that phenomena such as the gradation of agonist burst magnitude with motion in a second degree of freedom primarily reflect changes in burst amplitude rather than burst duration. The relatively constant burst duration observed here is consistent with previous work on single joint elbow movements within this range of amplitudes (Brown and Cooke, 1984) and double joint movements where movement time was held constant over different movement amplitudes (Buneo et al., 1994).

FRAME OF REFERENCE FOR MOTION PLANNING

A number of studies have focused on the issue of motion planning coordinates in arm movements. Some have provided kinematic evidence that motion planning occurs at the endpoint, or hand, level (Abend, Bizzi, and

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Morasso 1982; Flash and Hogan 1985). Specifically, straight line hand paths and bell-shaped hand velocity profiles were observed. Other studies assert that motion may be planned at the joint level on the basis of similar joint angular velocity profiles, or straight motion paths in joint coordinates (Hollerbach and Atkeson 1988, Ostry and Munhall 1994, Soechting and Lacquaniti 1981).

Electrophysiological evidence is consistent with the idea that goal-directed movements are planned at the hand level. Recordings from cortical cells in the monkey reveal that cell activity is broadly tuned to the direction of hand movement in multi-joint arm motion. The activity of cell populations is greatest for hand movements in a particular direction and decreases progressively as direction changes. Similar patterns have been reported in motor cortex (Caminiti et al. 1990; Georgopoulos et al. 1982, 1986; Kalaska et al. 1989; Schwartz et al. 1988), cerebellar cortex (Fortier et al. 1989), and parietal cortex (Kalaska et al. 1983, Kalaska 1988). Mussa-Ivaldi (1988) has argued that these observations may not relate to the direction of movement, but to the ensemble of muscles by which the direction is implemented. However, other studies have shown that cortical cell activity can be related to the direction of movement irrespective of the particular muscles being used (Crutcher and Alexander 1990; Thach 1978).

Relationships between parameters of the EMG signal and motion in each

degree of freedom have been demonstrated in the present studies. This suggests that control signals for motion may be planned at the level of the systems' degrees of freedom. While in multi-joint tasks this would correspond to motion at the joint level, these data cannot support one possibility over the other. This is due to the nature of the task used in these experiments. Position of the hand does not change with rotation of the forearm. Moreover, there is a monotonic relationship between hand position and elbow angle when all other joints are immobile. Hence specification of the hand location cannot be dissociated from specification of the elbow joint angle over different amounts of pronation or supination. While the present data does not provide direct support for motion planning at a joint level coordinate frame over an endpoint level one, it does suggest that control signals may, at some level, be organized with respect to the systems kinematic degrees of freedom.

At the joint level, control must be coordinated with other functions such as position of the hand in space. Thus, constraints at the joint level may be embedded in some higher order plan for multiple degree of freedom movements. Evidence for planning at both a hand and joint level has been reported previously in studies of multi-joint arm and jaw movements (Flanagan et al. 1990, 1993; Ostry and Munhall 1994).

COORDINATION OF MULTIPLE MUSCLES: IMPLICATIONS OF THE PRESENT FINDINGS ON THE

In a number of studies examining EMG activity during multiple degree of freedom movements, it has been found that relative muscle activity varies with motion in a second degree of freedom. This is particularly interesting in light of the fact that certain monoarticular muscles display this alteration in activity. For example, in the present set of experiments the magnitude of brachioradialis activity is less than that of biceps brachii during a combined flexion / supination. However, during a combined flexion / pronation the relative magnitudes of brachioradialis and biceps activity is reversed. This pattern may be contrasted with that exhibited during one degree of freedom flexion movements. In this situation the relative activation of these two muscles remains fairly constant for flexion movements of different amplitudes (i.e., they scale in magnitude similarly). Thus, synergistic relationships between elbow muscles are not fixed. In light of the variation in relative muscle activation, one may consider the concept of muscle synergy.

The term muscle synergy may be used in a number of different ways. Muscles exerting torque about a joint in the same direction may be thought of as anatomical synergists. For example, pronator quadratus and pronator teres both rotate the forearm in the pronation direction. Similarly, supinator and biceps brachii, which both act to supinate the forearm, can be considered as

anatomical antagonists to the pronators. During motion in more than one degree of freedom, however, muscles having different actions may be activated simultaneously. As an example, during a combined 140°-pronation / 70°-flexion, pronator quadratus acts as agonist while triceps brachii and biceps brachii may be activated together as antagonists. In this situation, pronator teres and biceps brachii can be thought of as physiological, or functional, synergists (Hasan et al. 1985).

The term "physiological synergy" may itself refer to different things. In one sense, a synergy may represent a fixed pattern of relative activation among a number of muscles (Buchanan et al. 1986). A more broad definition would consider all muscles active during a task as synergists (Jamison and Caldwell 1993). A great deal of research into the nature of muscle synergy has examined muscle activation patterns for a given task. Despite all the patterns possible, only a limited set of patterns are typically observed (Hasan 1990). This has been taken as evidence that such synergies provide a solution to the problem of muscular redundancy.

The present data suggest that the choice of muscle synergy may result from a combination of physiological and biomechanical factors. Functional synergies may not completely arise as a result of explicit central planning. Rather, the amount of activation displayed by a muscle may be partly

determined by its musculo-skeletal properties. That is, each muscle has a unique origin and insertion (even the different heads within a muscle) and thus presumably a line of action different from other muscles. Studies have shown that, for forces exerted in different directions, a muscles' activity may be represented by a tuning curve having a maximum for movements along its line of action (Flanders and Soechting 1990; Miller et al. 1992). It has been argued that for different movements, muscles that become more effective from a mechanical point of view will receive more activation than other muscles involved (van Zuylen et al. 1988). Thus, the activation patterns observed may be partly determined by a mechanism that accounts for the mechanical action of all muscles involved in the movement (van Zuylen et al. 1988).

The demonstration that monoarticular muscles may increase their activity with motion in a second degree of freedom indicates that functional synergies are also coordinated at a neural level. The increase of a monoarticular muscle's activity may compensate for a reduction in biarticular muscle activity. This compensation is not predicted purely on the basis of biomechanics. Hence, coordination of multiple muscles in multi-degree of freedom movements must be based on a combination of both neural control and biomechanical factors.

FUTURE STUDIES

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Based on the findings of the present investigation, several directions of future research suggest themselves. This future research would extend the present set of results as well as address some of the issues raised by them. The scope of these experiments ranges from an analysis of measures already obtained to a modelling of two degree of freedom elbow motion.

First, the magnitude of the antagonist and second agonist burst should be analyzed. This would provide a more complete picture of the effects of motion in more than one degree of freedom on the different components of the triphasic burst pattern and allow further comparison with results obtained from single joint studies. Presently, only the magnitude of the first agonist was analyzed quantitatively.

Second, the present study consisted of movements having fairly constant movement times while amplitude and, therefore, peak velocity were varied. In light of previous studies relating a movement's temporal characteristics to EMG activation patterns (Brown and Cooke 1990; Cooke and Brown 1990; Gottlieb et al. 1989b; Hoffman and Strick 1990), an investigation of multi-degree of freedom movements under more controlled temporal conditions is warranted. That is, it would be interesting to see the variation in burst magnitude over conditions of, for example, constant acceleration duration in each degree of freedom. While this might prove a difficult task for subjects to perform, results

would permit a direct comparison between motion in the single and multi-degree of freedom situations.

Third, an examination of muscle activation during static torque production is suggested. These isometric contractions would be performed in the various arm configurations (ie, start and end positions) used in these studies. Static tuning curves during force production in different directions at the wrist provided insight into the role of muscle mechanics in shoulder / elbow motion (Flanders and Soechting 1990). That is, the direction in which a muscle displayed the greatest amount of activation in counteracting a force was along that muscle's mechanical line of action. A similar analysis in the present context would consist of subjects counteracting torques in both the pronation / supination and flexion / extension directions, applied both alone and in combination. This force production would be repeated for a number of static postures corresponding to the start and end positions used in the anisometric task. By observing the effects of posture on EMG activity magnitude, one could determine the extent to which the present EMG magnitude findings are related to biomechanical constraints versus neural constraints.

Fourth, an extension of the present studies through the addition of motion in a third degree of freedom should be investigated. As an example, performance of the present paradigm with the addition of a grasping motion

would provide insight into the control of reaching, orientation, and grasping at the EMG level. In the studies reported here, the magnitude of the agonist burst varied while timing of muscle activity did not. Comparable findings under conditions of motion in three degrees of freedom, which presumable subserve three different functions, would provide further evidence that these may be planned in parallel. Similarly, an electromyographic analysis of combined shoulder / elbow / forearm motion would be informative. Specifically, a change in the timing of EMG activity with the addition of motion at the shoulder (unlike that seen with elbow / forearm motion alone) would support the notion that these are parallel, interdependent processes. Evidence for this has been seen at the level of sensori-motor transformations (Flanders and Soechting 1993) and kinematics (Lacquaniti and Soechting 1982).

Fifth, the finding that a muscle may simultaneously act as agonist and antagonist is unique. A further exploration of this phenomenon seems appropriate. Examination of biarticular muscles about different joints during multiple degree of freedom movements would assess the generalizability of this finding. Furthermore, it was observed that when a muscle displays both agonist and antagonist bursts, the identity of the burst having the greater magnitude is not related to the amplitude of motion in each degree of freedom. It was suggested that the observed predominance of motion in the flexion / extension direction may be related to the relative torque requirements of the

task. An inverse dynamics procedure, which would provide a measure of torque exerted in each degree of freedom, would address this possibility.

Finally, a model of motion in two degrees of freedom about the elbow is necessary to address some of the issues raised in these studies. To be informative, such a model would have to incorporate physiological and biomechanical properties of the arm under dynamic conditions. One could then explore the form of the central commands and their coordination in controlling such a system. Comparable simulated and empirically derived data would support the form of neural organization proposed by the model in the absence of direct evidence. To date, a model exploring the organization of commands to different degrees of freedom and their associated electromyographic activity has not been reported.

SUMMARY

The determinants of arm motion in multiple degrees of freedom were investigated by examining electromyographic activity associated with two degree of freedom elbow movements. Movements of varying amplitude were performed in each degree of freedom both separately and together.

Muscles fell into two categories with respect to agonist burst magnitude

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- those whose burst magnitude varied with motion in a second degree of freedom in a graded fashion and those whose burst magnitude did not. In biarticular muscles, the magnitude of the agonist burst in movements in which a muscle acted as agonist in two degrees of freedom was approximately the sum of the magnitudes of the bursts in the component one degree of freedom movements. Burst magnitudes for one degree of freedom movements were, in turn, greater than for movements in which the muscle was agonist in one degree of freedom and antagonist in the other.

Certain monoarticular muscles, including triceps brachii (long head), triceps brachii (lateral head), and pronator quadratus had first agonist bursts whose magnitude did not vary with motion in a second degree of freedom. Other monoarticular elbow flexors brachialis and brachioradialis had agonist burst magnitude which were affected by pronation or supination movement.

During movements in which biarticular muscles act as agonist to motion in one degree of freedom and antagonist in the other, the muscle activity may display both agonist and antagonist components in the same movement. It was found that, for pronator teres and biceps brachii, the timing of the bursts was such that there was activity in these muscles concurrent with activity in both pure agonists and pure antagonists. This behaviour was present over a wide range of movement amplitudes in the two degrees of freedom.

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The onset of burst activity relative to movement onset did not vary for movements of different amplitudes in each degree of freedom. This was true both for those muscles acting as agonists, and those acting as antagonists. In addition, the duration of burst activity did not vary with movements of different amplitude in each degree of freedom.

Together these findings suggest that central commands for motion in individual degrees of freedom may be planned in parallel and simply superimposed in producing two degree of freedom movements.

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