Bimanual Limb Interaction

by

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Abstract

In this study I investigated the level of neurological interaction between two limbs performing fast, goal directed bimanual movements, and the extent to which the limbs interact and influence each other during movement preparation and production. This experiment focused upon the effect of symmetric and asymmetric bimanual movements of short and long distances performed simultaneously, specifically interaction in response to a movement blocking perturbation. Differences between the EMG patterns of unimanual, equal distance bimanual, and unequal distance bimanual elbow extension movements of 10 and 50 degrees indicated the level of influence seen in movement planning, while differences in kinematic measures indicated the level of interaction during movement production. Results indicated that there was a high level of influence during movement planning and execution, resulting in highly symmetric EMG patterns, but no detectable interaction between the two limbs during movement execution. Blocking the intended movement of one limb had no effect on movement production of the other limb. Once movement was initiated each limb operated independently, displaying characteristic EMG patterns for unblocked movements and modified EMG patterns due to sensory feedback of a blocked movement.

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Introduction

Why is it so difficult to learn to play the piano, playing individual and pairs of notes with your left and right hands? Why is touch-typing just as difficult, even though your fingers move one at a time? Almost all our natural body movements are bimanual, with two limbs moving together, such as when we clap our hands, or two pairs of limbs moving in opposition, such as when we walk, or run. Our bodies perform bimanual movements so naturally that we only become aware of the innate desire for movement symmetry when we try to move our limbs separately, or asymmetrically. When we first try to perform two different left and right hand movements simultaneously, something affects our ability to perform the intended act. Our arms appear to move together, we are unable to easily control one limb without the other acting at the same time. Only through deliberate practice can we learn to produce two different movements simultaneously. The difficulty we feel while trying to perform asymmetric movements represents the resistance that our nervous system exerts to performing different movements simultaneously. Our body's preference to perform bimanual movements symmetrically is known as the "entrainment tendencies" between the two limbs. The degree of entrainment can be dependent on the "coupling strength" which can indicate a level of interlimb interaction in movement production. Of present interest are the circumstances, and the extent to which one limb in a bimanual movement can affect the neurological signals controlling muscles of the other limb.

When it is not necessary, or even beneficial, to perform a bimanual movement symmetrically, we have difficulty producing an asymmetric movement. In a study of bimanual aiming movements over short distances to large targets ('easy') and over long distances to small targets ('difficult'), Kelso, Southard, and Goodman (1979) found that

participants tended to pair their left and right hand movements, with both hands ending the movement (impacting) at the same time. While this type of synchronization would be appropriate for symmetric-bimanual movements, Kelso, et al. (1979) found that when a short movement was paired with a long movement, movements were still synchronized, the short movement took just as long to complete as the long movement. When performed unimanually short movements were completed in 78-82 ms, but when paired with a long movement the short movement times were almost doubled at 133-140 ms (Kelso, et al., 1979, figure 1). In another experiment Kelso, et al., (1979) found that when one hand was forced to take a longer movement path in order to clear a barrier, both hands took the symmetric, longer route, once again with symmetric final target impacts. Participants found it very difficult to produce asymmetric movements in these conditions, the movements they did produce were only different in amplitude, they were still symmetric in time.

Identical (distance) bimanual movements have nearly identical acceleration profiles, with very high cross correlation values and similar amplitudes indicating a high level of symmetry; described as "coupling strength" (Swinnen and Walter, 1988). Bimanual movements of different distances also have similar acceleration profiles, but with differing amplitudes, indicating reduced coupling strength (Swinnen, Walter, Beirinckx, Meugens, 1991). It was therefore assumed that higher coupling strength means stronger interaction between the two limbs. Early evidence of increased coupling strength during symmetrical movements was shown by Kelso, Holt, Rubin, and Kugler (1981). They had participants perform a series of continuous in-phase (homologous muscles contracting simultaneously) cyclical movements of their left and right index fingers. At unexpected intervals a short duration perturbation was applied to one finger. During this 100 ms perturbation the

unperturbed finger modified its trajectory to maintain its phasic relationship with the perturbed finger. Since response time of the unperturbed finger was on the order of 100 ms, Kelso, et al. (1981) believed these results displayed coupling at the spinal level (at the same level as a stretch reflex). Since this level of interaction was found during continuous symmetrical movements, would perturbing a discrete symmetrical movement display the same level of interaction?

One method used to perturb discrete movements is movement blocking, where the limb is mechanically prevented from leaving from a start position (Wadman, et al., 1979). This is a passive method whose influence is not felt until the limb tries to move and encounters a physical barrier, described as an "infinite-mass inertial load". Under movement blocking EMG patterns are unaffected by proprioceptive feedback for approximately 100 ms after onset, which is thought to be due to muscle motor time and stretch reflex response times (approximately 50 ms each). Applied to one limb of a discrete symmetric movement, movement blocking would allow investigation of changes in the perturbed and unperturbed limbs' EMG patterns. Differences in these dependent measures may indicate which parts of movement planning and execution are pre-planned and performed without the use of peripheral feedback, and which parts use feedback to coordinate and control movement.

For fast unimanual movements sensory response times are too slow to provide adequate on-line control. These movements are thought to be prepared centrally and executed in sequence until the movement is completed (Henry & Rogers, 1960; Wadman, Denier van der Gon, Geuze, and Mol, 1979; Shapiro and Walter, 1982). Fast, goal-directed movements are usually characterized by a triphasic burst of EMG (Electromyographic) activity, comprised of agonist muscle activity to initiate movement, an antagonist burst to

brake or slow the movement, and a final agonist burst to "clamp" the limb in position at the end of movement (Berardelli, Rothwell, Day, Kachi, and Marsden, 1984; Enoka, 1988). The amplitude and timing of these EMG bursts determines the distance and speed of limb movement. In order to investigate movement preparation Wadman, Denier van der Gon, Geuze, and Mol (1979) employed a movement blocking (mechanically locking a chain connected to the wrist) paradigm that prevented linear wrist movements of 7.5, 15, 22.5 and 30 cm (approximately 7.5-30 degrees elbow extension). The nature of the prepared response as seen in the pattern of evoked EMG and force was investigated. As expected unblocked movements displayed characteristic EMG patterns for increasing movement distance: later onset of antagonist and second agonist bursts, and reduction in EMG amplitude (see figures 6 and 7, Wadman, et al., 1979). Blocked and unblocked movements showed similar EMG patterns for the shortest movements, but very different patterns for longer distance movements. The shorter 7.5 and 15 cm movements displayed almost identical triphasic EMG patterns for both blocked and unblocked movements, indicating that peripheral feedback did not play a role in controlling the pattern of generated EMG for at least the first 120-130 ms after the onset of the first agonist burst (the reference point for all EMG and kinematic time measures). Differences in EMG were visible after approximately 125 ms, particularly in the abrupt offset of antagonist EMG in blocked movements compared to unblocked movements. Movements of 30 cm also displayed identical first agonist EMG bursts for both blocked and unblocked conditions, but in blocked conditions there was almost no antagonist activity. Since the first agonist burst was not affected by movement blocking, Wadman, et al., (1979) concluded "that for at least the first 100 ms the motor system does not make use of proprioceptive movement information for control" (p. 9). The delay of 100-125

ms between onset of EMG and measurable changes in EMG activity in these blocked trials can be attributed to muscle motor times of approximately 50 ms between EMG onset and movement onset, and a typical stretch reflex delay time of approximately 50 milliseconds after the movement was prevented by blocking. The conclusion of Wadman, et al. (1979) was that "fast-as-possible movements" were first centrally controlled via pre-set muscle commands, then are subject to sensory influence after mechanical and reflex delays amounting to 100-125 ms after agonist EMG onset.

While it was clear that the first 100-125 ms of triphasic EMG activity was centrally planned in blocked unimanual movements, followed by peripheral feedback controlling EMG activity, it was not clear how triphasic EMG patterns were generated in normal, unblocked movements. Was antagonist EMG activity centrally planned, or was it the result of a short delay stretch reflex (see Feldman, 1986)? In order to answer this question, Latash and Gottlieb (1991) had participants perform fast elbow flexion movements of 20 and 36 degrees with 25-50% of trials being mechanically blocked. Kinematic variables of velocity and acceleration started to diverge 40-50 ms after first agonist EMG onset in blocked trials, while EMG patterns diverged after 90-120 ms, supporting the findings of Wadman, et al. (1979). The onset of the antagonist burst was centrally initiated, as it appeared at the same relative time in blocked and unblocked trials, but its amplitude and offset were peripherally controlled, based on two different lines of evidence. Firstly, in a study of fully deafferented patients, Forget and Lamarre (1987), concluded:

> ...the central nervous system can generate a sequence of commands to accelerate and decelerate a limb in the absence of peripheral feedback.

> However, information from the moving limb is required to adjust the magnitude and time of onset of deceleration. (p. 27)

Secondly, Wadman, et al. (1979) found that blocked movements of less than 45-60 degrees displayed correct antagonist burst onsets, but since the limb had not moved, this antagonist activity could not have been triggered by sensory information. Likewise, antagonist EMG offset appeared to be sensory based, as it was fixed at 100-150 ms after agonist onset in movement blocking conditions. Blocked 20 and 36 degree movements displayed some triphasic EMG activity, while fast as possible 45-60 degree movements were characterized by a single continuous sustained agonist EMG burst, with almost no antagonist activity (Wadman, et al., 1979; Latash & Gottlieb, 1991a), indicating complete suppression of antagonist activity in these movements. Additionally, voluntary response times to same limb and opposing limb stimuli in discrete unimanual movements were measured by Latash and Gottlieb (1991b). When participants were instructed to perform an extra extension movement of a blocked limb, this yielded response times of 150-216 ms, while opposing limb response times were 200-260 ms.

While the movement blocking paradigm has proved useful for investigating the nature of movement planning in unimanual movements, it does have its limitations. We (Nagelkerke, Oakey, Mussell, and Franks, 2000) constructed three experiments. The first reproduced the design of Wadman, et al. (1979) using horizontal elbow extension-flexion. The second studied the effect of response complexity on movement initiation time with movement blocking. Finally the third investigated whether or not movement blocking could be used to detect pre-programmed variations in muscle activation patterns.

The first experiment showed that short distance movements of 7.5 and 15 degrees elbow extension displayed nearly identical stereotypical triphasic activity in both blocked and unblocked conditions. These results essentially replicated those of Wadman, et al., (1979) and Latash & Gottlieb, (1991b). In the second study we investigated the EMG patterns of extension movements of 7.5 and 15 degrees, in addition to reversal movements of 7.5 degrees extension - 3.75 degrees flexion and 15 degree extension - 7.5 degrees flexion. Unblocked movements displayed triphasic EMG activity that varied with movement distance and complexity, with short burst durations and short movement time for the 7.5 degree extension movement and significantly longer burst durations and movement times for the 15 degree reversal movement. Muscle EMG patterns of blocked movements were similar to that of unblocked movements for the first 100 ms agonist EMG onset, but the patterns deviated after that. Unblocked movements showed variation in onset, offset, and amplitude of the antagonist burst, as well as second agonist burst, while blocked movement EMG patterns were similar in burst duration times to the unblocked 7.5 degree extension movement. Movement initiation premotor times were significantly longer for reversal movements compared to single extension movements, even in movement blocking conditions. This movement complexity effect (Henry & Rogers, 1960) was attributed to variations in central commands, as it was measured in premotor time (from the stimulus to the onset of first agonist EMG). Feedback processes had a major effect on blocked movement EMG 100 ms after the onset of first antagonist movement, masking any centrally planned movement patterns. The last study was therefore designed to determine if the first 100 ms after agonist EMG onset could be shown to be preprogrammed. The EMG rise rate measure Q30 (Q30 measures the EMG rise rate, or slope, by integrating full-wave rectified agonist EMG over

the first 30 ms) has been shown to vary directly with movement speed (Gottlieb, et al., 1989a, b; Corcos, et al., 1989; Khan, Garry, and Franks, 1999). The third experiment investigated 45 degree extension movements to small 1.5 degree and large 15 degree targets. We reasoned that since Khan, et al., (1999) has shown that Q30 varies with movement speed as a consequence of target size (faster movements to larger targets) and is a preprogrammed pattern executed without the influence of feedback, Q30 would be unaffected by movement blocking. Results of this study showed that Q30 values for movements to the large and small targets were significantly different, and importantly unaffected by movement blocking. EMG patterns of blocked and unblocked movements deviated after 100 ms, indicating that the remaining EMG pattern was the result of feedback-based activity. These experiments showed that movement blocking is restricted in its ability to investigate movement programming, that only the first 100 ms of agonist EMG activity accurately reflects central control as measured by Q30.

The limitations of the movement blocking paradigm may restrict its utility for investigating movement planning of unimanual movements, however, it may still be effectively used to investigate the interaction of bimanual movements during planning and execution. Interactions affecting movement planning would result in changes in EMG , specifically onset times and amplitude, while interactions affecting movement execution would result in changes in movement EMG and kinematics. The response of one limb to a perturbation of the opposing limb (the inter-limb response time), has been measured in continuous cyclical in-phase movements, but it is not known if the results of these experiments are applicable to discrete movements with movement blocking. For example, Kelso, Holt, Rubin, and Kugler (1981) had participants perform cyclical left and right pointer

finger extension and flexion movements, with occasional load perturbations applied to one finger. When movement was constrained by physical barriers to a range of 50 degrees, perturbations applied to one finger resulted in physical correction of the unperturbed finger with a latency of less than 50 ms as measured from angular displacement. Presumably, this response time consisted of a 30-50 ms stretch reflex, indicating that the two fingers may be coupled at the spinal level.

Bimanual aiming movements have displayed remarkable symmetry in both space and time (Kelso, Southard, and Goodman, 1979), and this symmetry was also seen in the execution of unequal distance movements. The pairing of an easy short distance - large target and difficult long distance - small target movements resulted in both movements being performed at the rate required by the longer movement. In these experiments participants would synchronize both finger lift at the beginning of movement and finger press at the end of movement. The short movement was produced much more slowly than if it were performed alone, at less than maximal speed and muscle activation level. The short movement's reduced activation level, and it's synchronization to the long movement, make it more sensitive to possible neural interactions if the longer movement is perturbed.

The present experiment studied limb interaction during discrete bimanual movements to address two questions: What is the nature of interlimb interaction when one limb is forcibly perturbed during a bimanual movement, and does the coupling strength of an intended bimanual movement also predict the strength of limb interaction during movement blocking? The experiment investigated unimanual, bimanual symmetric, and asymmetric movements using EMG and kinematic measures to assess the patterns of single limb versus dual limb movement production under normal and perturbed conditions. Coupling strength,

and presumably the strength of limb interaction to perturbations, was expected to be stronger for the symmetric movements compared to unequal, asymmetric movements. The response of one limb to the blocking perturbation of the opposing limb during a bimanual movement would be an indication of the degree of interlimb interaction - strong interaction would result in a noticeable change in the EMG and kinematics of the unperturbed limb, weak interaction would result in little or no change in the movement of the unperturbed limb. Previous studies have found that unequal bimanual movements are performed symmetrically, with both movement times determined by the longer movement, resulting in a longer duration and slower velocity short movement. Since Q30 measures the rate of EMG rise, and slow movements have reduced EMG rise times, I expect to find reduced Q30 values for 10 degree short movement in the unequal bimanual condition when paired with a long 50 degree movement.

Since this experiment was designed to examine movement preparation and planning for fast, goal directed bimanual movements, and inter-limb responses to perturbations applied to one limb, participants performed fast-as-possible discrete unimanual and bimanual movements of short or long movement distances to targets, and some trials were unexpectedly blocked. It was expected that pre-programmed movement patterns will be evident in both blocked and unblocked EMG patterns, while peripheral feedback would be responsible for generating different EMG patterns between blocked and unblocked movements after 100 ms. Peripheral feedback-based differences in EMG were expected to be seen for both the perturbed and unperturbed limbs, due to the strong inter-limb coupling of symmetric bimanual movements. Comparison of the EMG and kinematic records of normal

and perturbed discrete bimanual movements would indicate the preparation and planning involved in producing these movements and the nature of limb interaction during movement.

The symmetry seen in bimanual movements, with similar movement onset and offset times, is seen as an indication of the "coupling" or interconnection between the two limbs. Rather than being treated as two separate limbs, bimanual aiming movements appear to be controlled as one virtual limb (Al-Senawi & Cook, 1985). Any changes imposed on one limb should in turn affect the characteristics of the un-modified limb, when participants were asked to wear a one pound wrist weight on their non-dominant hand for a period of time they showed movement adaptation in both arms within 6-12 hours. Participants performed extension-flexion stepped tracking trials before, during, and after wearing the weight, and primarily displayed increased movement peak velocity for both hands. Both arms adapted simultaneously to the increased inertial and gravitational load on one arm, and maintained this adaptation when being tested without the weight. The level of simultaneous adaptation to an imposed load on one limb indicates a strong linkage or coupling between the limbs, and the body's innate preference to perform bimanual movements.

The level of limb interconnection, or coupling strength, of a bimanual movement is calculated as a cross correlation coefficient of left and right acceleration patterns (Swinnen, 1992). This gives a measure of movement production symmetry, but does not give a true measure of the level of inter-limb coupling in response to a perturbation such as movement blocking. In order to investigate the level and strength of inter-limb coupling, movement blocking will be applied to one limb of a bimanual movement, and the opposing limb kinematics and EMG times will be measured.

In the present experiment ten participants performed a series of unimanual and bimanual movements with random unexpected movement blocking of one limb. Differences in kinematic and EMG patterns under different testing conditions indicated what movement patterns had changed in each condition. The experiment was designed to investigate the following specific questions about the preparation and production of unimanual, symmetric bimanual, and unequal distance bimanual movements that were performed in either blocked or normal conditions:

1. Kelso, Southard, and Goodman (1979) found that simultaneous short movement and long movements were performed symmetrically in time, the short movement was performed more slowly, with both movements starting and stopping at the same time. Will the timing symmetry evident in multiple joint bimanual movements also be seen in single-joint elbow extension movements?
2. Discrete goal-directed unimanual movements are characterized by triphasic EMG activity, a pattern of agonist, antagonist, agonist activity, that is unaffected by movement blocking for 100-125 ms after first agonist activity onset (Wadman, et al., 1979). Blocking of an intended short movement results in partial suppression of antagonist activity while blocking of a long distance movements results in complete suppression of antagonist activity, due to the later onset of antagonist activity in longer distance movements. Is there a difference in the EMG activity of a blocked movement performed in a unimanual condition as that performed in a bimanual condition?

3. There is some evidence of left and right limb interaction in response to perturbations of one limb in continuous cyclic finger movements. Kelso, Holt, Rubin, and Kugler (1981) had participants perform simultaneous extension-flexion movements of their left and right pointer fingers, with a short term (100 ms) perturbing force applied to one finger. Perturbation resulted in a large 20-25 degree position change of the affected finger and a small 10 degree position change of the unperturbed finger, that maintained their in-phase relationship. What level of interaction would be seen in discrete bimanual elbow extension movements when one arm is blocked?
4. Reaction times tend to increase with increased movement complexity; it generally takes longer to initiate bimanual movements than unimanual movements (Kelso, et al., 1979; Marteniuk, et al., 1984), although this 'bilateral deficit' effect was not seen in all conditions (Swinnen, et al., 1995). Will there be any reaction time difference between unimanual and bimanual movements of the same distance, and will there be any differences between the reaction times of equal distance and unequal distance bimanual movements?

Method

Participants

Ten right handed university students with no upper body abnormalities, and normal or corrected to normal vision were recruited to perform the experiment. After being informed about the general nature of the experiment, participants signed an informed consent form, in accordance with the ethical guidelines of the University of British Columbia.

Apparatus and Task

Participants were seated on a height adjustable chair at a testing station facing two video monitors, a high speed display screen and standard colour computer monitor, with their left and right forearms each placed on a horizontally rotating manipulandum. Participants were positioned with their shoulders abducted approximately 85 degrees (5 degrees down from horizontal), flexed 10 degrees forward, with their forearm resting on a manipulandum, pronated to position their hands palm downwards. The participant's elbows were positioned over the manipulandum's vertical axis of rotation and their hands secured to adjustable hand supports. Participants were secured to the chair at the shoulders and waist by wide safety straps to prevent postural changes over trials. Participants sat facing a high speed video display (Tektronics XY monitor model 620, rotated to give a 10 cm wide by 12 cm high screen) that displayed two sets of two stationary dots representing a starting and target positions, and two moving dots representing each manipulandum position, refreshed at 1000 Hz. The two target dots were positioned at the top of the video display, with the varying start positions indicated below them. Outward elbow extension movements resulted in vertical

movement of the response dots to the target area, with the left and right movement indicator dots horizontally separated by 2 cm on screen.

EMG signals were collected from each arm's triceps and biceps using surface electrodes (Therapeutics Unlimited model 544 pre-amplifier, two 8mm Ag/AgCl disks, 21 mm on center) positioned over each muscle belly, after site preparation with isopropyl alcohol and conductive gel, and a reference ground electrode placed over the participant's right ankle. EMG amplifier gain was adjusted to provide a clean signal with peak amplitude between 1 and 9 Volts, to ensure signal resolution and avoid signal clipping (at 10 volts) before being sampled by the A/D converter.

The manipulandum was instrumented to measure angular position and acceleration, plus horizontal rotational force exerted by the participant's hand. Angular position was measured via optical encoders (Dynapar Model E2025001303), connected to a quadrature interface card (Advantech PCL-833) giving 10,000 counts per revolution, a resolution of 0.036 degrees per count. Angular acceleration of each arm was measured using piezo-electric accelerometers (Kistler model 8638B50, ±50 g) and coupler (Kistler model 5112, frequency response 0.5-5k Hz, 100 mV per gravity) sampled by an analog to digital (A/D) converter (LabMaster PGH, 12 bit bipolar with software selectable gains: ±10.0, ±5.0, ±2.5, ±1.25 Volts). Lateral horizontal force exerted by the hand against the manipulandum was measured by a custom-built load cell (4 arm bridge with 1 strain gauge per arm, machined from 1/4 inch aluminum) coupled to a load amplifier (Northwood Instruments, model IA-102, 500x amplification, range DC-1kHz). The position of the hand plate and integrated force sensor were adjusted for each participant to accommodate different forearm lengths and

the radial distance of the hand plate to the center of rotation for the manipulandum recorded for each participant allowing the calculation of angular torque.

All analog and digital signals were sampled at 1000 Hz by custom computer software (developed with Turbo Pascal 6.0 for DOS) and saved in compressed binary format on the hard disk for later analysis. Visual feedback was provided via a high speed XY-oscilloscope (Tektronix model 620) driven by the 12 bit digital to analog output (DAC) of the LabMaster card at 1000 Hz. The oscilloscope was rotated 90 degrees to give a visual field of 10 cm wide by 12 cm high with two vertically moving dots representing left and right manipulandum position, and four stationary dots in horizontal center of the screen, indicating movement start and target end position. A horizontal black cloth just below chin level blocked participant's view of their arm during movement testing, forcing participants to rely on proprioceptive feedback of arm position and visual feedback on the high speed screen.

Procedure

Participants were asked to perform a series of maximum voluntary contractions (MVCs) of extension and flexion at the beginning of the experiment and again at the end. These consisted of two sequential extension and two flexion contractions at the movement start position against the movement blocking pins. Next participants performed a series of fixed distance extension movements, first to practice the movement, followed by a series of acquisition trials, including unexpected movement blocking trials. Participants practiced forearm extension movements of 10 or 50 degrees starting from a constant position of 22.5 degrees extension (with 180 degrees being full elbow extension). Manipulandum movements of 50 degrees corresponded to a 10 cm displacement of the response dot on the high-speed visual display from the start position dot to the two target indicator dots. At the beginning of

a trial the participant was asked to move the manipulandum to the indicated start position. They lined up their response dot under the left start position and notified the experimenter that they were ready to begin. After a 100 Hz, 100 ms warning tone, participants waited between 1500-2500 ms (random foreperiod) for the 100 ms stimulus tone of 1000 Hz. Data collection commenced 100 ms before the stimulus tone, and continued for three seconds. During normal unblocked trials 2 mm diameter metal pins were lifted into position in front of each manipulandum by electromagnets activated during the 100 ms stimulus tone, then deactivated, allowing the pins to recede. In a blocked movement trial either the left or right pin stayed elevated, preventing the manipulandum tip from moving more than 1-2 mm, less than 0.5 degrees of rotation.

A total of eight movement conditions, in addition to the maximum voluntary contraction (MVC) condition, were performed by each participant, consisting of four unimanual and four bimanual movements. The four unimanual movements consist of both short 10 degree and long 50 degree movements for left and right hand, while the four bimanual movements consisted of either equal distance (short-short or long-long) or "unequal distance" unequal (short-long, long-short) movements. Short and long unimanual movement conditions for each hand were completed before the performing the equivalent bimanual movement, and these conditions were counterbalanced across subjects.

At the beginning of each condition participants performed a series of practice trials of each movement until they were able to reliably stop their movements within five degrees of the target position ten times in row. The movement endpoint was calculated as the first point at which absolute angular velocity dropped below 8 degrees per second. Participants were instructed to move as "quickly and as accurately as possible", receiving verbal feedback from

the experimenter in addition to a display on a computer screen on their target accuracy and movement times. Accuracy feedback consisted of a text message with the number of degrees of target undershoot or overshoot while movement times were displayed in milliseconds.

Research Design

Each participant completed a total of ten testing conditions, consisting of two sets of maximum voluntary contractions (MVC) at the beginning and end of the testing session, and eight unimanual and bimanual, short and long distance movement conditions. Short distance movements consisted of a 10 degree elbow extension movement from the start position while long distance movements consisted of 50 elbow degree extension movements. The four unimanual conditions consisted of short-left hand, short-right hand, long-left hand, and long-right hand movements, while the equal distance or "equal-bimanual" conditions consisted of either a short left and short right movement, or a long left and long right movement performed simultaneously. Unequal distance or "unequal-bimanual" movements consisted of either a short left and long right movement, or long left and short right hand movement.

Dependent Measures

After collection the data were analyzed to convert raw data values into the appropriate data value units. Displacement, acceleration, and force data were multiplied by scaling factors to convert optical encoder count values into degrees for displacement, convert sampled analog voltage values into degrees per second per second for acceleration and Newtons for force. In order to calculate angular velocity, a copy of the displacement data was first smoothed with a fourth-order Butterworth low-pass digital filter with a cut-off frequency of 30 Hz, then differentiated to give velocity. All EMG data were first scaled from

sampled analog voltage values to surface electrode milliVolts using the EMG amplifier gain setting values. Maximum Voluntary Contraction (MVC) trial EMG data were rectified and filtered at 30 Hz and the average muscle activation level calculated over 1.5 to 2.5 seconds of each 3 second MVC trial for extension (triceps) or flexion (biceps). Subsequently, all non-MVC trials were analyzed to scale their EMG values from milliVolts to a percentage of average MVC.

Collected data were analyzed through custom computer programs that used algorithms (described below) to automatically mark specified events on displacement, velocity, acceleration, and force data, while a computer visual editor program was used to manually mark the onset and offset of triceps and biceps EMG activity.

Angular Displacement. The onset of movement was defined as the first point where the displacement position data rose above the value of zero degrees. The end of movement for unblocked movements was defined as the point where angular velocity drops below 8 degrees per second, located just before peak displacement. Peak displacement was found as the point of greatest positive magnitude. Because blocked movements do not achieve velocities of 8 degrees per second, blocked movement offset was defined as the first local displacement maximum.

Angular Velocity. Movement velocity was marked with three event markers: onset, peak velocity, and end of movement. Onset was defined as the first velocity point of 8 degrees per second or greater magnitude, while the end of movement was the last point of velocity greater or equal to 8 degrees per second. Peak velocity was marked as the point of greatest magnitude positive velocity, and end of movement was the last point of velocity greater than or equal to 8 degrees per second. Displacement movement time was calculated

using angular velocity as the interval from velocity onset to velocity offset. Velocity reaction time (Velocity RT) was measured as the interval from stimulus onset to velocity onset, time to peak velocity was the interval from velocity onset to peak velocity, and movement time was calculated as the interval from velocity onset to velocity offset (velocity values greater than 8 degrees per second).

Acceleration. Five acceleration events were marked: acceleration onset, peak positive acceleration, zero acceleration, peak negative acceleration, and second zero acceleration. Zero acceleration was found as the first point at which acceleration crossed from positive to negative, or negative to positive values.

Force. As stated earlier, lateral horizontal force exerted by the hand against the manipulandum was collected for both hands in each condition. Force onset was found as the first point that force first rose and stayed above one Newton for at least 50 ms, while peak force was found as the point of greatest positive force value.

EMG. A visual editor was used to mark the onsets and offset of EMG activity of the triceps and biceps muscle. This editor displayed a 500 ms section of data (150 ms prior to velocity onset and 350 ms after velocity onset). A typical pattern of activity for each trial was a triphasic burst consisting of an accelerating triceps agonist followed by a braking biceps antagonist and a second agonist triceps burst that would clamp the limb into the final position. Exceptions to this triphasic pattern were seen in blocked movements where the braking biceps burst occasionally did not appear (1-5% of blocked short movements, 10-19% of blocked long movements). Editing this data provided the following dependent measures:

- Premotor reaction time (time from stimulus onset to first agonist onset)
- Antagonist burst onset (time from first agonist onset to antagonist burst onset)

- Q30 measure of rectified and integrated triceps EMG for 30 ms from agonist onset, indicating the rise rate and partial measure of the magnitude of the first agonist burst.
- Q100 measure of rectified and integrated triceps EMG over the first 100 ms of agonist activation, essentially the integral (area) of the first triceps EMG burst.

Ensemble Average. Continuous data for displacement, velocity, acceleration, force, and EMG were each combined in "ensemble averages" (see Wadman, et al., 1979 for example) for each testing condition. Ensemble averages were calculated by aligning each trial's data array by a common point, usually the first onset marker, and finding an overall mean of every point for 500 ms, starting 100 ms before the common point.

Data Analysis.

Dependent measures were analyzed separately with a 3 (Condition: unimanual, equal-bimanual, unequal-bimanual) x 2(Distance: 10 degrees, 50 degrees) x 2(Hand: left or right) repeated measures ANOVA. Some dependent measures were also analyzed using a 2 (Condition: equal-bimanual, unequal-bimanual) x 2(Distance) x 2(Hand) x 2 (Blocked/Unblocked) repeated measures ANOVA. The alpha level for the entire experiment was set at 0.05 and the Huynh-Feldt Epsilon factor was used to adjust the degrees of freedom for violation of the sphericity assumption (Huynh & Feldt, 1970). The Tukey HSD (Howell, 1997)method was used for all post-hoc comparisons and statistical significance was assumed if the measured t-value exceeded the critical t-value.

Results

Angular Displacement

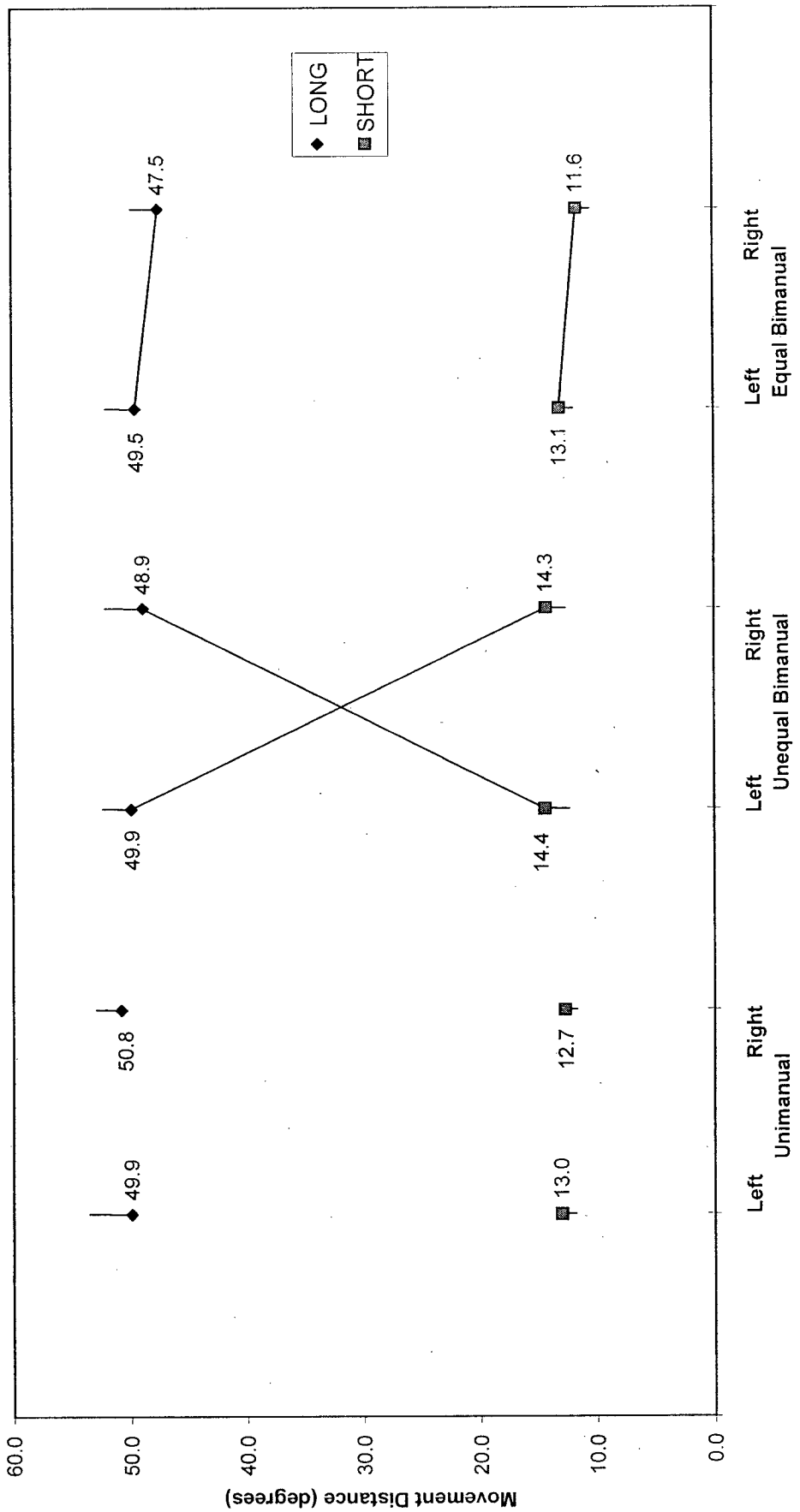
Analysis of movement distance showed main effects for movement condition $F(2, 18) = 16.154$, $p < 0.001$ and movement distance $F(1, 9) = 2793.599$, $p < 0.001$, and a Condition x Distance $F(2, 18) = 15.97$, $p = 0.020$ interaction (see figure 1). There was a significant Condition x Hand interaction, but this is a meaningless result when the results are collapsed over movement distance. In the unequal-bimanual condition the end of movement averaged 14.4 degrees compared to 12.8 degrees for short-unimanual movements and 12.3 degrees for short-short bimanual movements (see figure 2).

Movement blocking was used to investigate the level of limb interaction by looking for movement differences due to perturbation of the opposing arm. Analysis of opposing limb blocking for bimanual movements revealed main effects of movement Condition $F(1, 9) = 32.515$, $p < 0.001$, and Distance $F(1, 9) = 1699.421$, $p < 0.001$, and Blocking $F(1, 9) = 11.158$, $p = 0.009$. There was a significant difference in movement distance for the left hand in the equal-bimanual long movement condition, 49.460 degrees for unblocked movements and 51.320 degrees when the right hand was blocked.

Angular Velocity

Reaction Time. Analysis of the onset of movement showed a main effect for Condition $F(2,18) = 12.580$, $p < 0.001$, and a Condition x Distance interaction $F(2,18) = 11.146$, $p = 0.001$ (see figure 3). Collapsing left and right hand data (see figure 4) there was a significant difference between the unequal-bimanual short movement (RT = 216 ms), the

Figure 1. Movement Distance for Unimanual and Bimanual Conditions



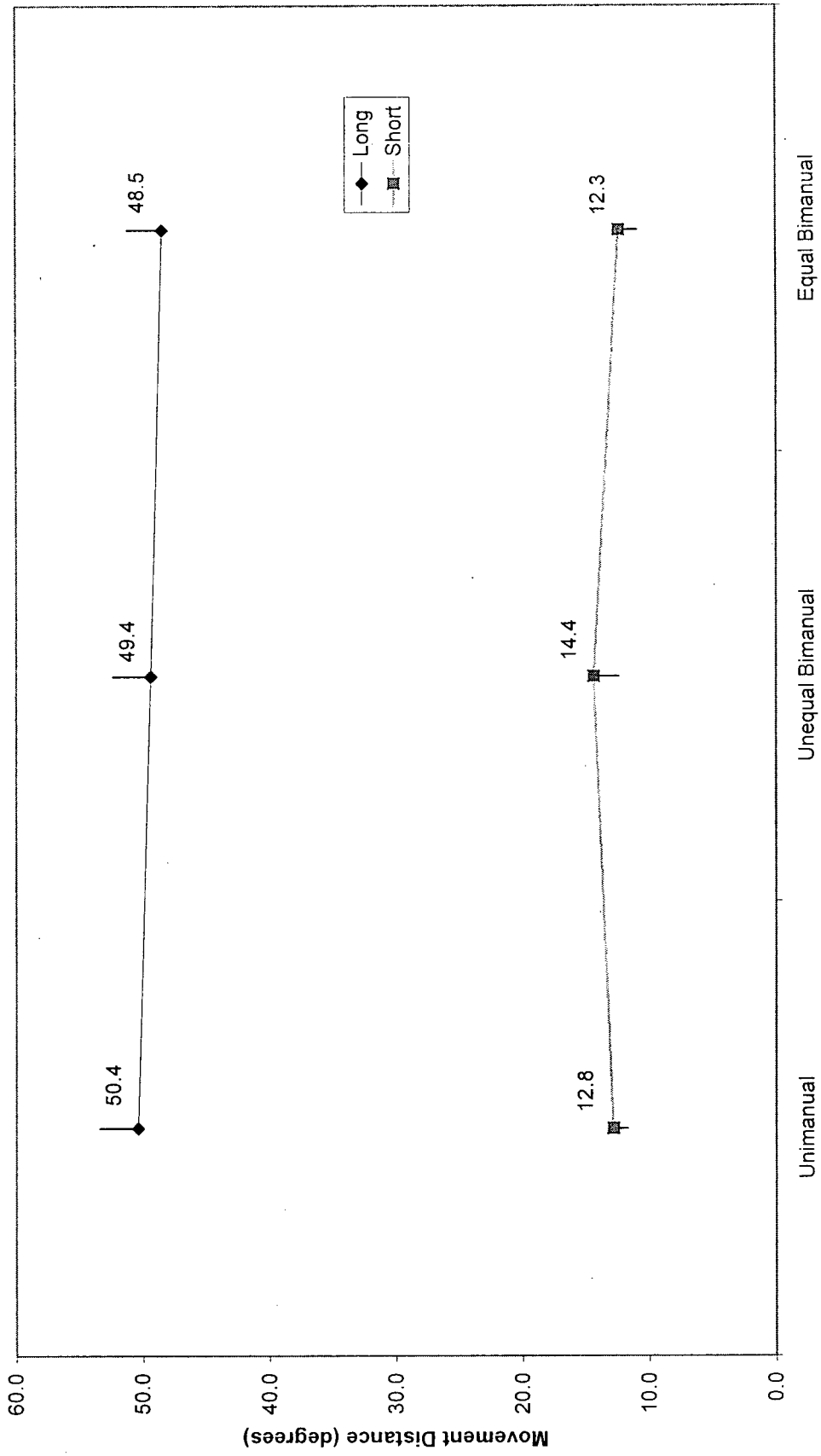


Figure 2. Movement distance (collapsed over hand)

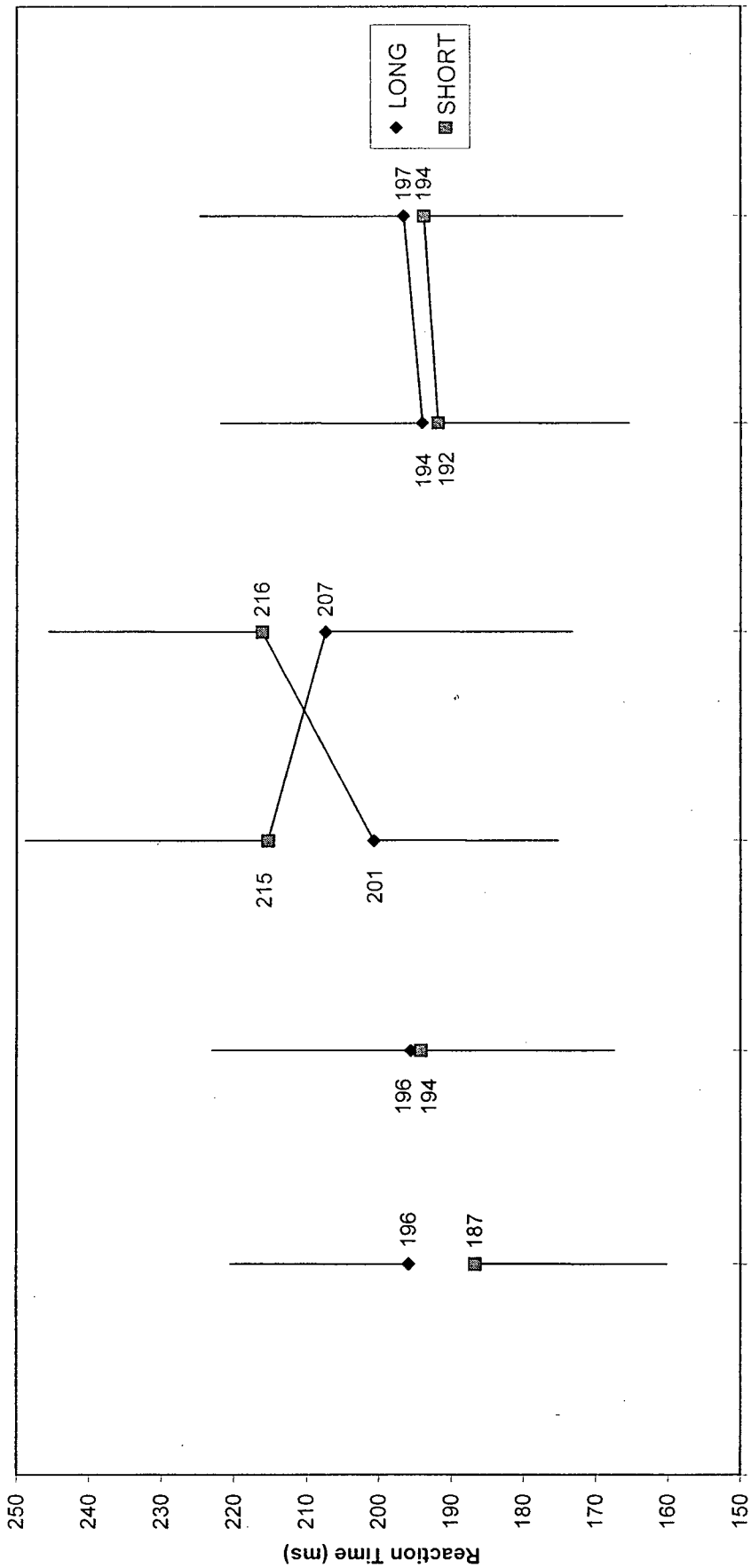


Figure 3. Velocity Onset Reaction Time

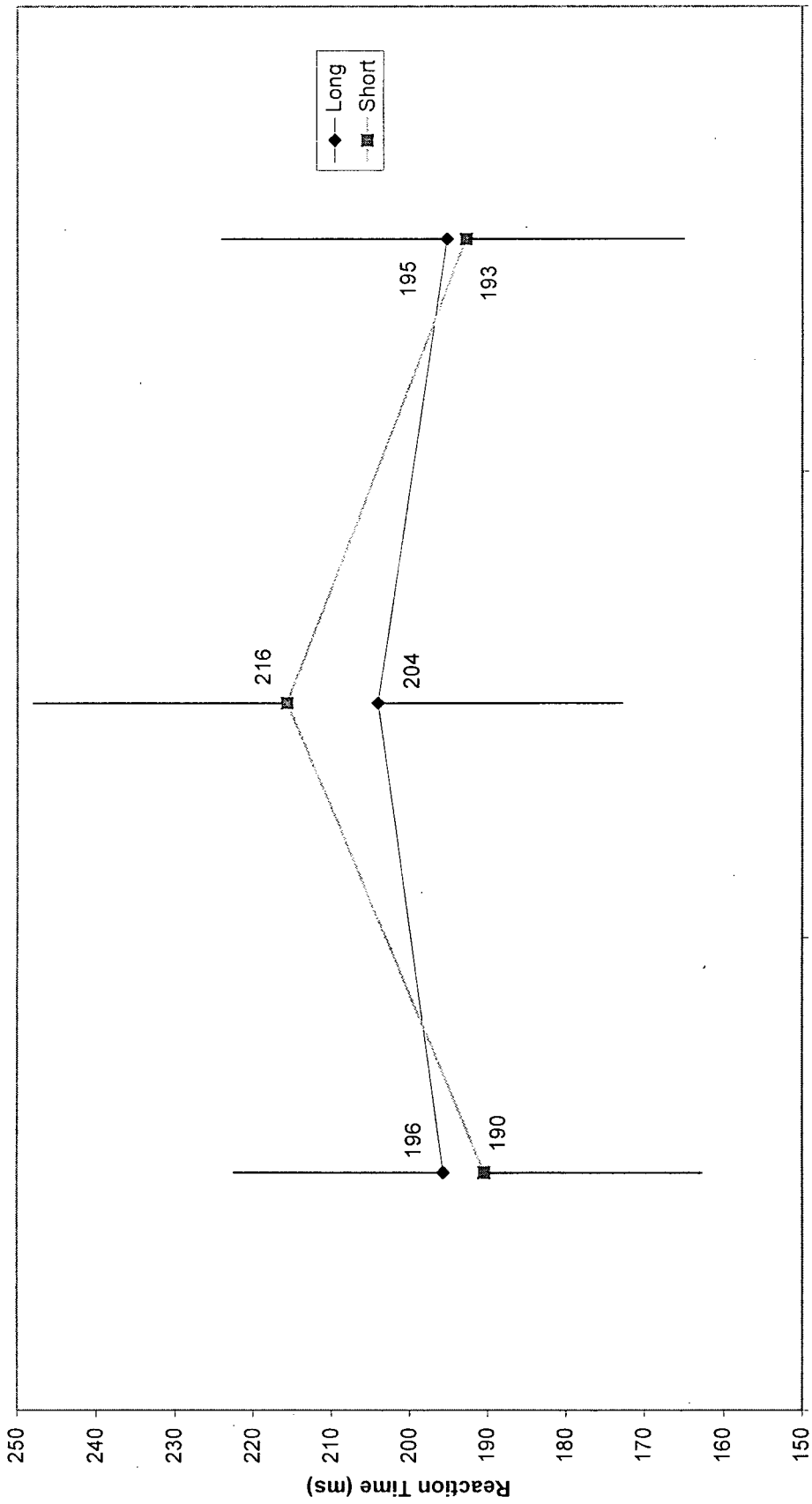


Figure 4. Velocity Onset Reaction Time (collapsed over hand)

unimanual short movement (RT = 190 ms), and equal-bimanual short movement (RT = 193 ms).

Time to Peak Velocity. The interval from velocity onset to peak velocity showed main effects for Condition $F(2, 18) = 15.237$, $p < 0.001$ and Distance $F(1, 9) = 261.928$, $p < 0.001$, and a Condition x Distance interaction $F(2, 18) = 4.486$, $p = 0.028$ (see figure 5). There was a significant difference between the unequal-bimanual condition (88 ms) and unimanual (75 ms) and equal-bimanual (77 ms) conditions (see figure 6).

Peak Velocity. There was a significant main effect of Condition $F(2, 18) = 4.427$, $p = 0.044$, and Distance $F(1, 9) = 289.291$, $p < 0.001$, and a significant interaction for Condition x Distance $F(2, 18) = 7.894$, $p = 0.003$. The significant difference of the Condition x Distance interaction was between the peak velocity of long movements in the unequal-bimanual condition (333 ms) and long movements in the equal-bimanual condition (376ms) (see figures 7, 8).

Movement Time. Analysis of movement time showed a main effect of Condition $F(2, 18) = 12.954$, $p < 0.001$ and Distance $F(1, 9) = 48.861$, $p < 0.001$, and significant three way interactions $F(2, 18) = 3.756$, $p = 0.043$. Short right hand movements in the unequal-bimanual condition took significantly longer than short right hand unimanual and equal-bimanual movements (see figures 9, 10). Similarly, long distance left hand movements took significantly longer in the unequal-bimanual movement than long left hand movements in the unimanual and equal-bimanual movements, while unequal-bimanual right hand movement times were only significantly different from right hand equal-bimanual movements. For bimanual movements there were no differences in movement time of an unperturbed limb when the opposing limb was blocked.

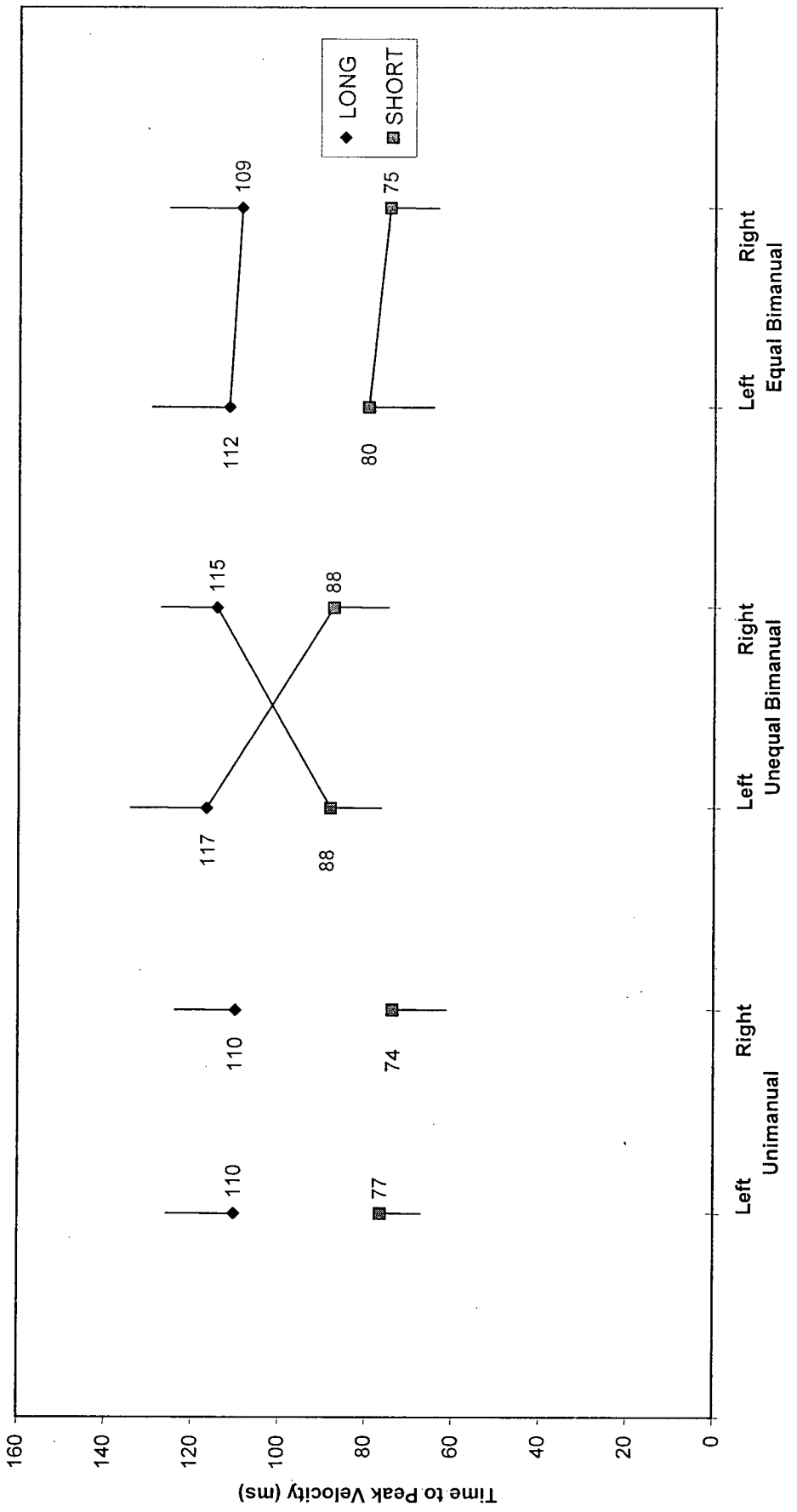
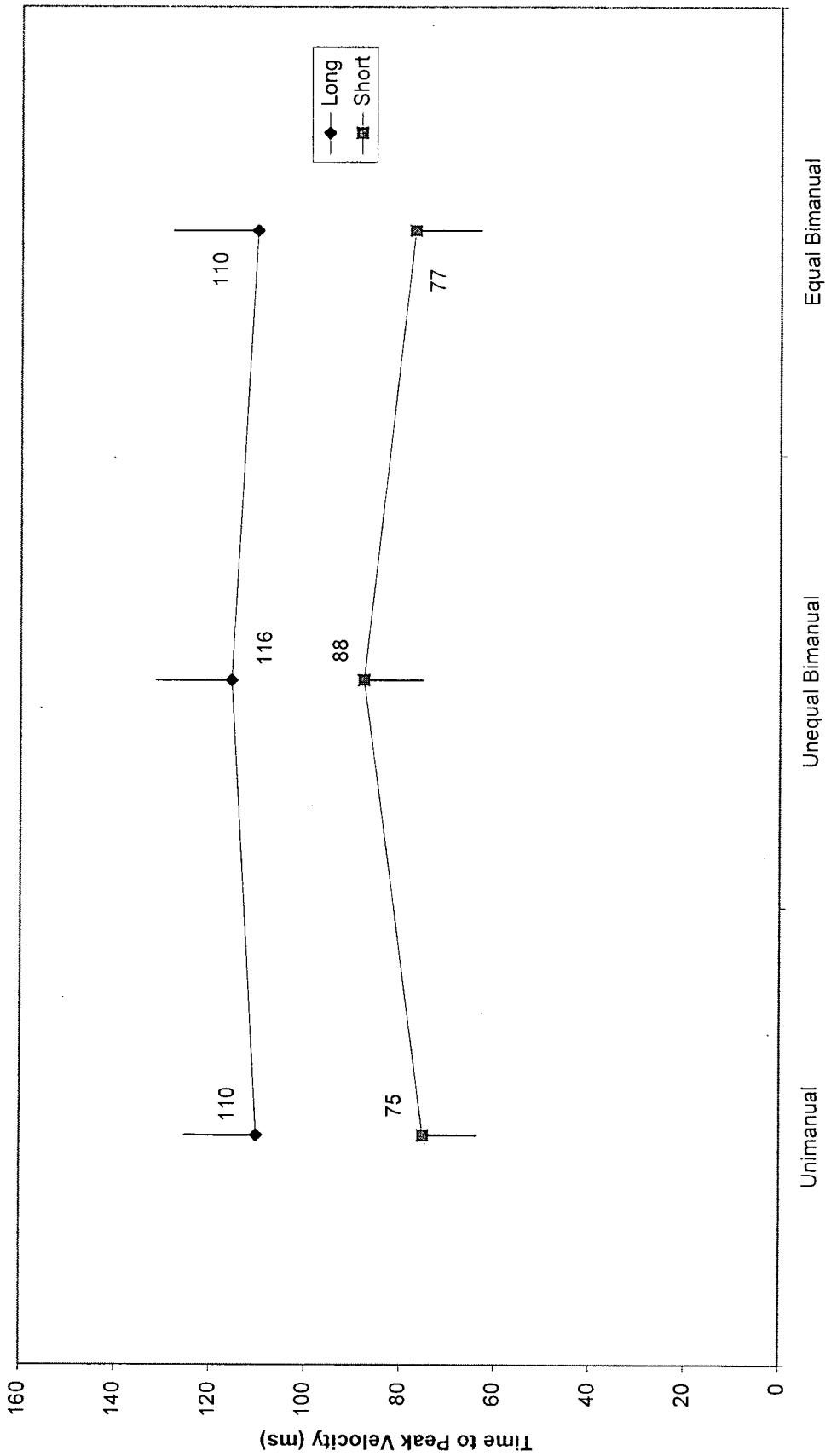


Figure 5. Time to Peak Velocity

Figure 6. Time to Peak Velocity (collapsed over hand)



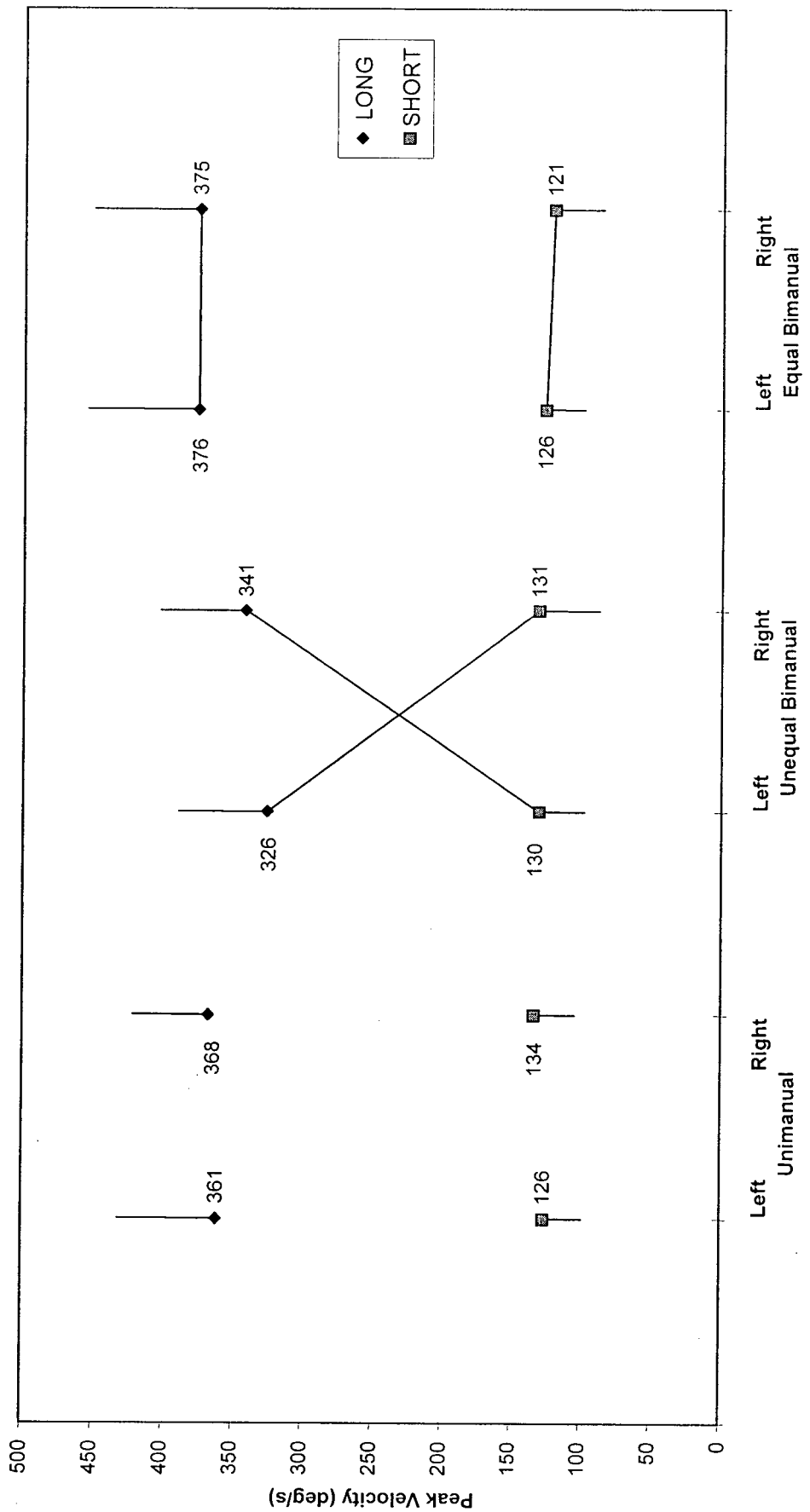
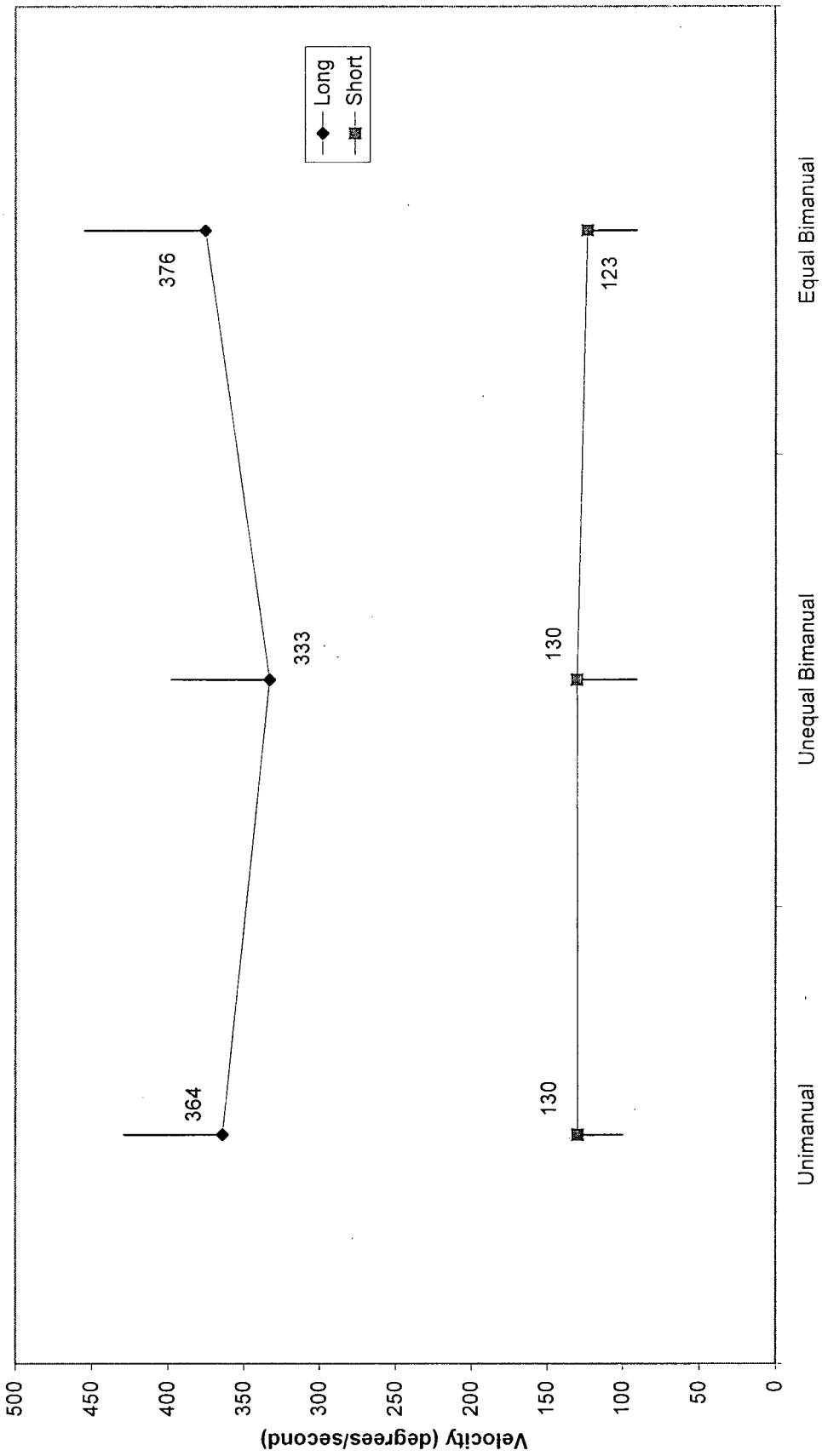


Figure 7. Movement Peak Velocity

Figure 8. Movement Peak Velocity (collapsed over hand)



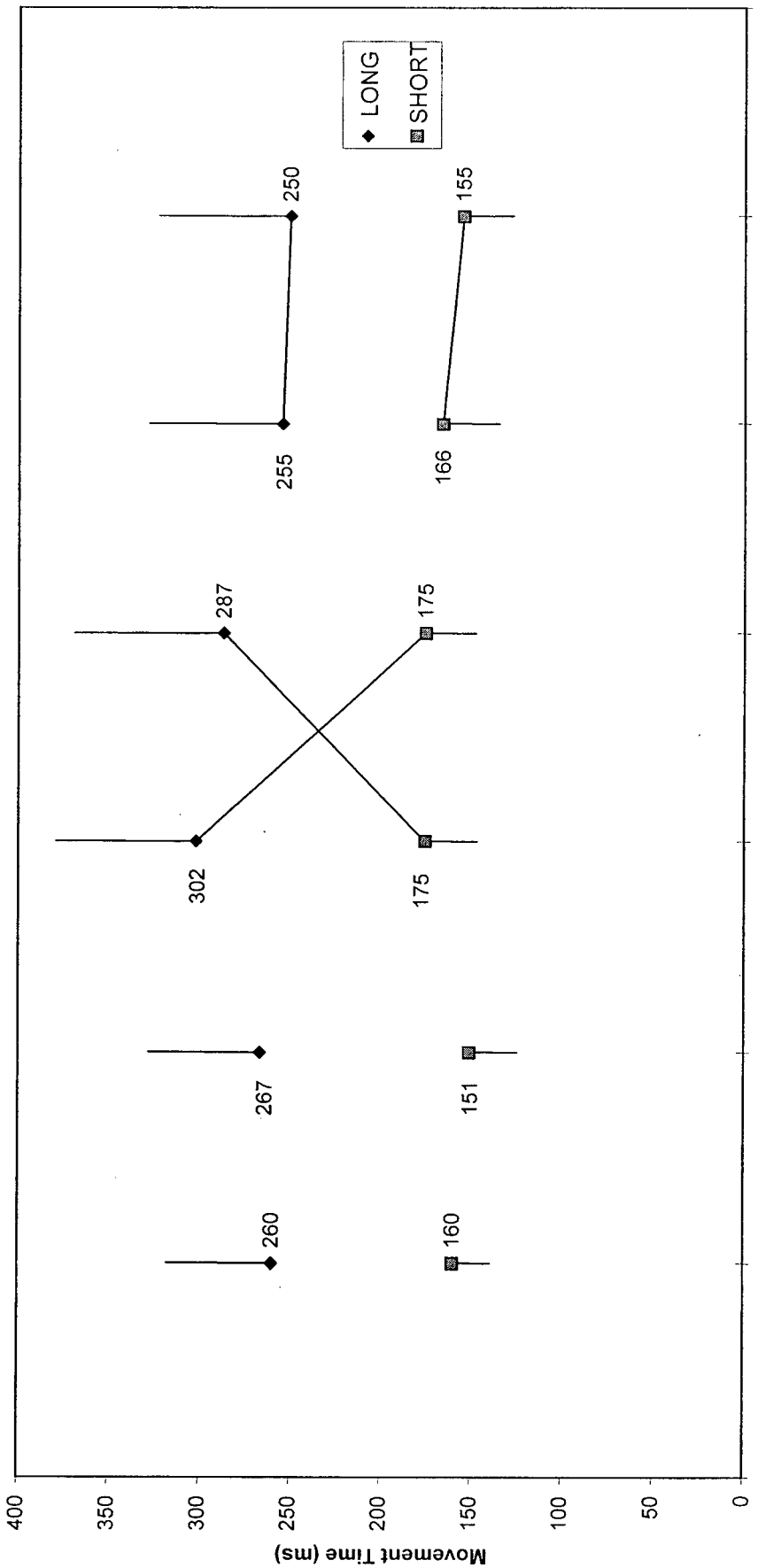


Figure 9. Movement Time

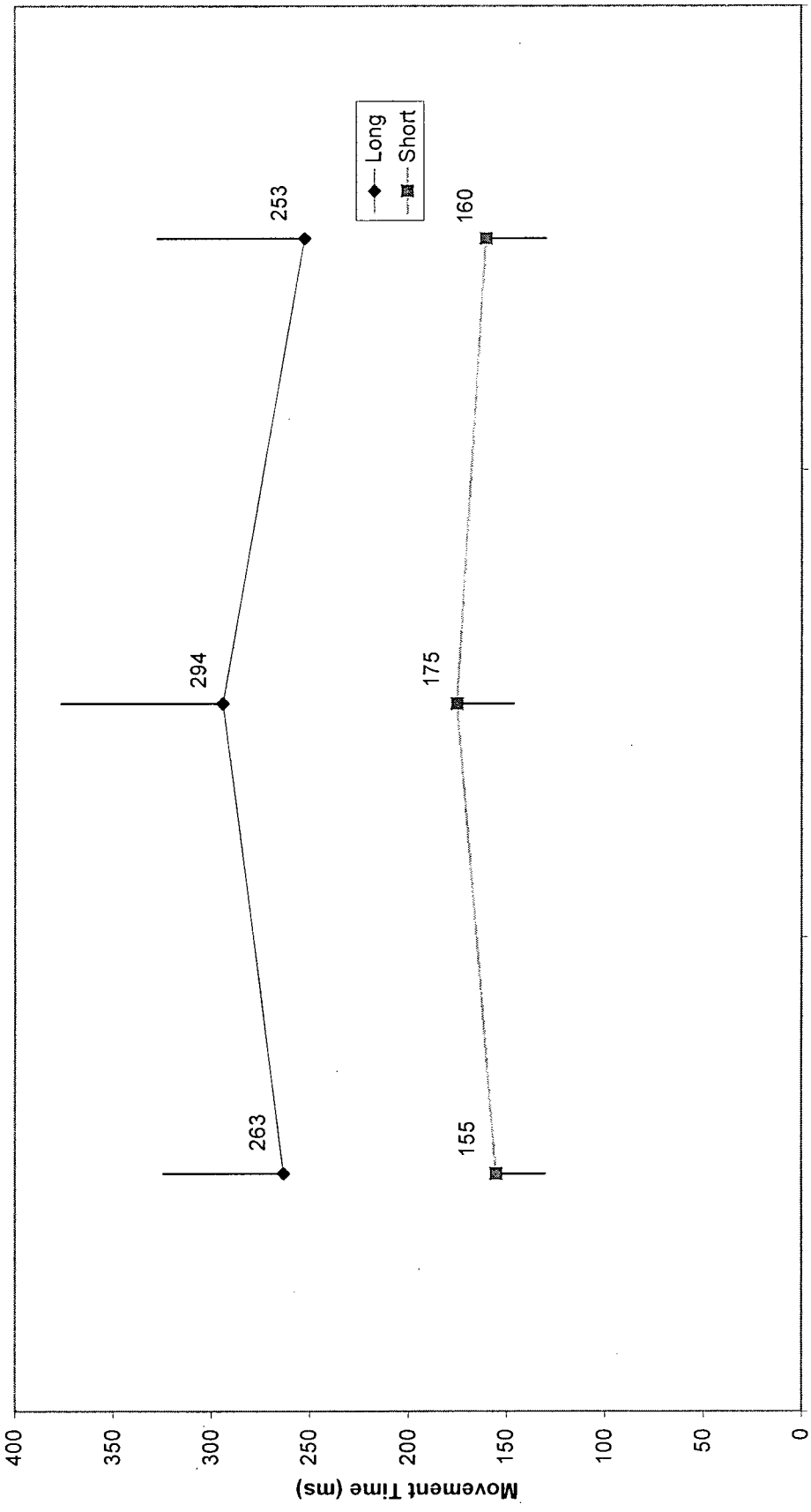


Figure 10. Movement Time (collapsed over hand)

Angular Acceleration

Acceleration Onset. The acceleration reaction time showed a main effect for Condition $F(2, 18) = 10.460$, $p = 0.001$ and a Condition x Distance interaction $F(2, 18) = 12.324$, $p = 0.001$. In the unequal-bimanual condition the shorter distance acceleration onset time (197 ms) was significantly greater than it was in the unimanual (172 ms) or equal-bimanual (174 ms) conditions (see figures 11, 12).

Time to Peak Acceleration. A main effect for Condition $F(2, 18) = 4.684$, $p = 0.023$ was found for time to peak acceleration, with an increase in the unequal-bimanual condition (average 50.5 ms) compared to unimanual (46.5 ms) or equal-bimanual (46.8 ms) movements (see figures 13, 14).

Peak Acceleration. Peak acceleration values displayed main effects for Condition $F(2, 18) = 6.321$, $p = 0.011$ and Distance $F(1, 9) = 68.884$, $p < 0.001$, with no other main effects or interactions Both short and long distance peak acceleration values were reduced in the unequal-bimanual condition (see figure 15)

Force

Peak Positive Force. Peak Positive force showed main effects for Distance and Hand $F(1, 9) = 5.958$, $p = 0.037$. There is a significant Condition x Hand interaction but this is meaningless without a three way interaction involving movement distance. For all long distance movements, across all conditions, the right hand produced greater peak force than the left hand. For short distance movements the right hand produced greater force in the unimanual condition, but produced nearly identical forces in the unequal-bimanual and equal-bimanual movements (see figure 16).

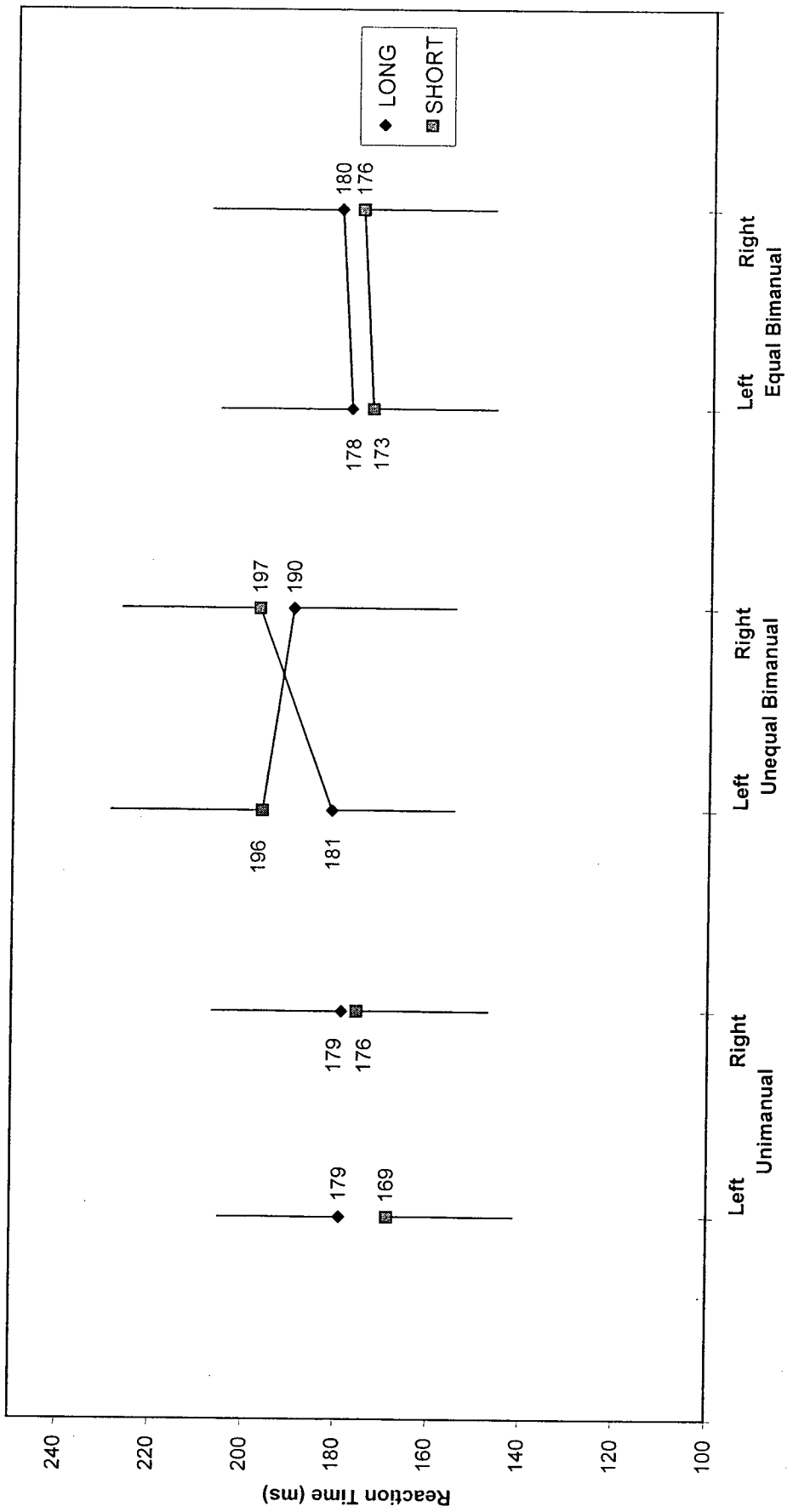
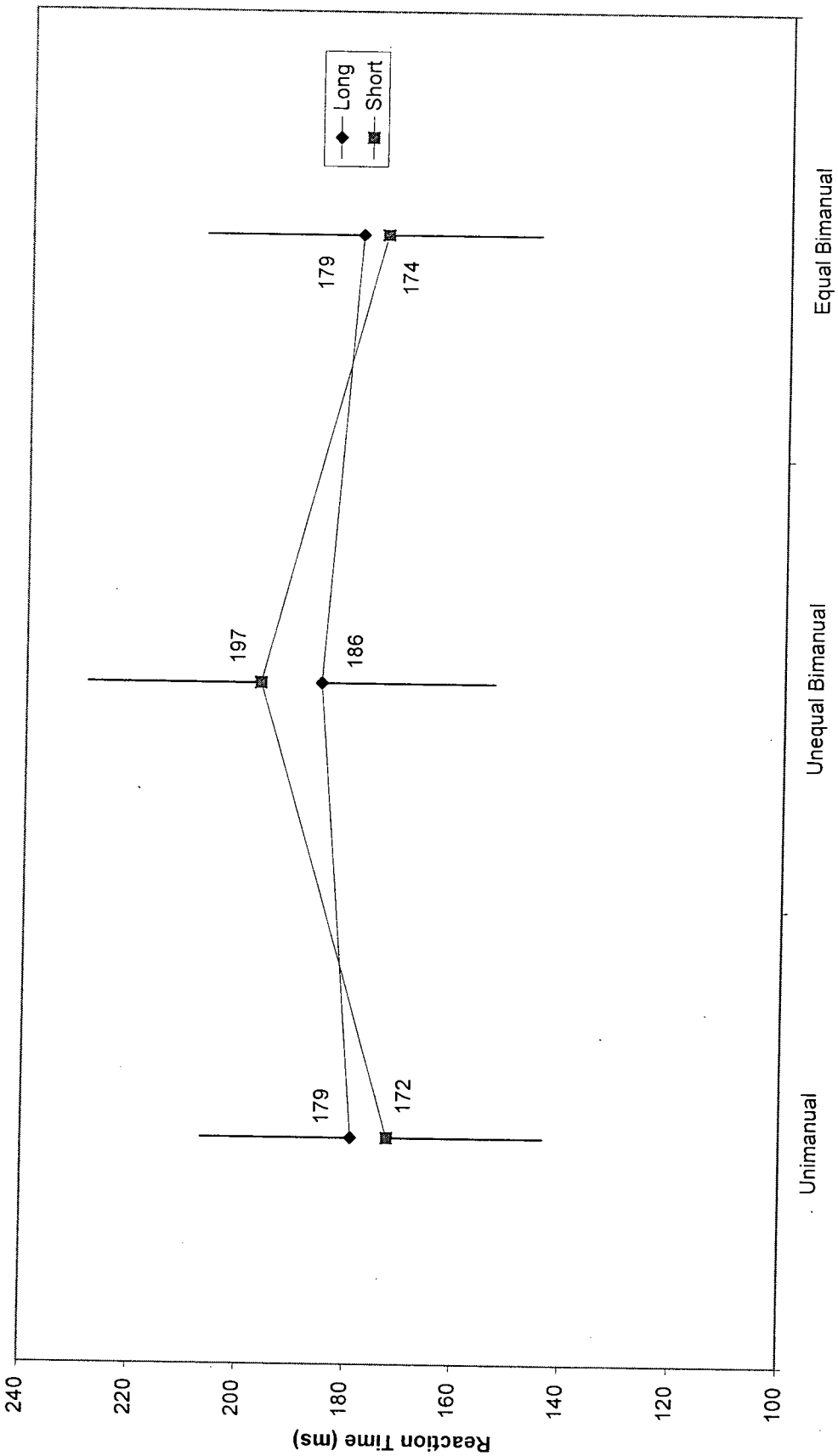


Figure 11. Acceleration Onset Reaction Time

Figure 12. Acceleration Onset Reaction Time (collapsed over hand)



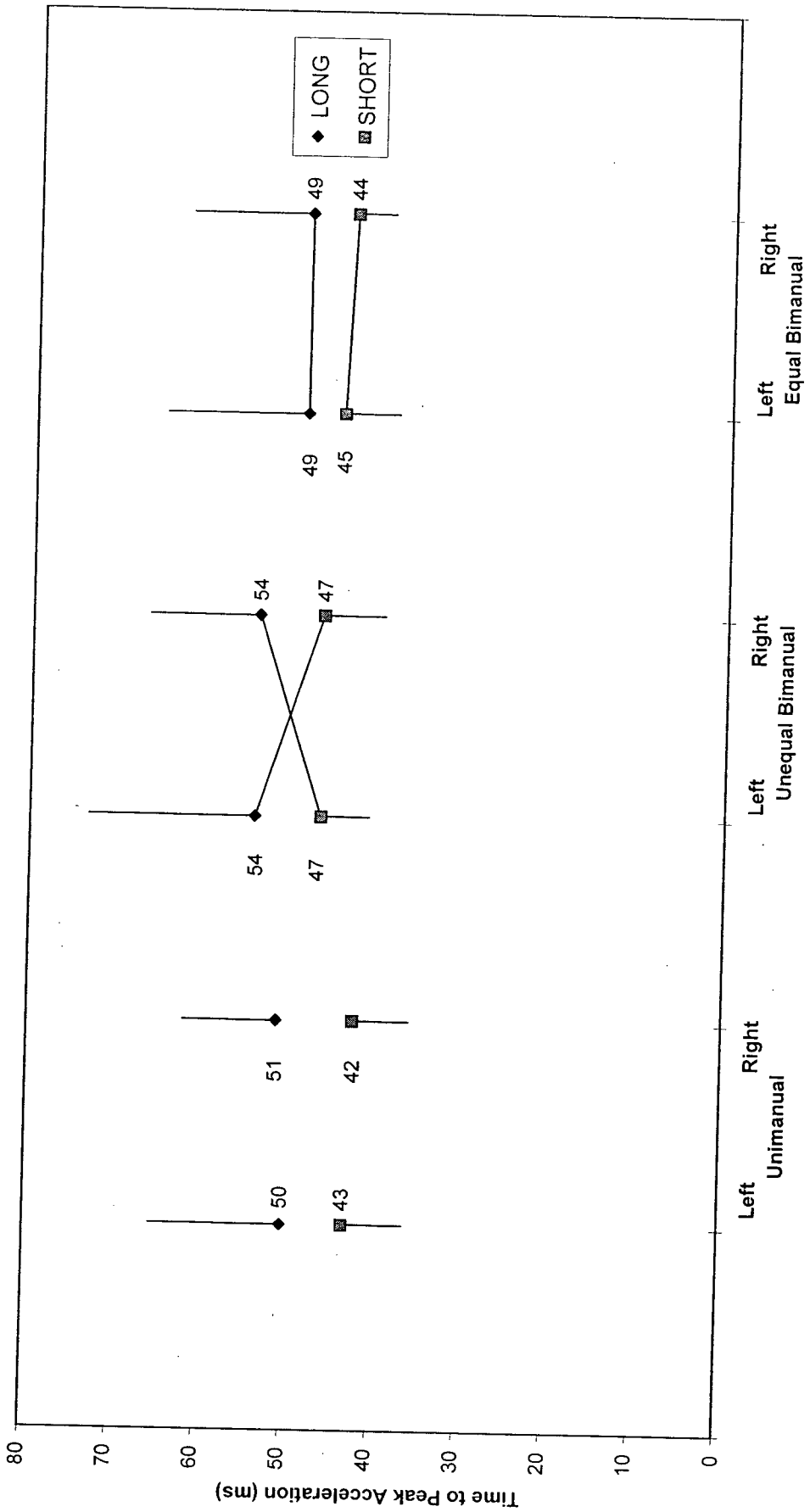


Figure 13. Time to Peak Acceleration

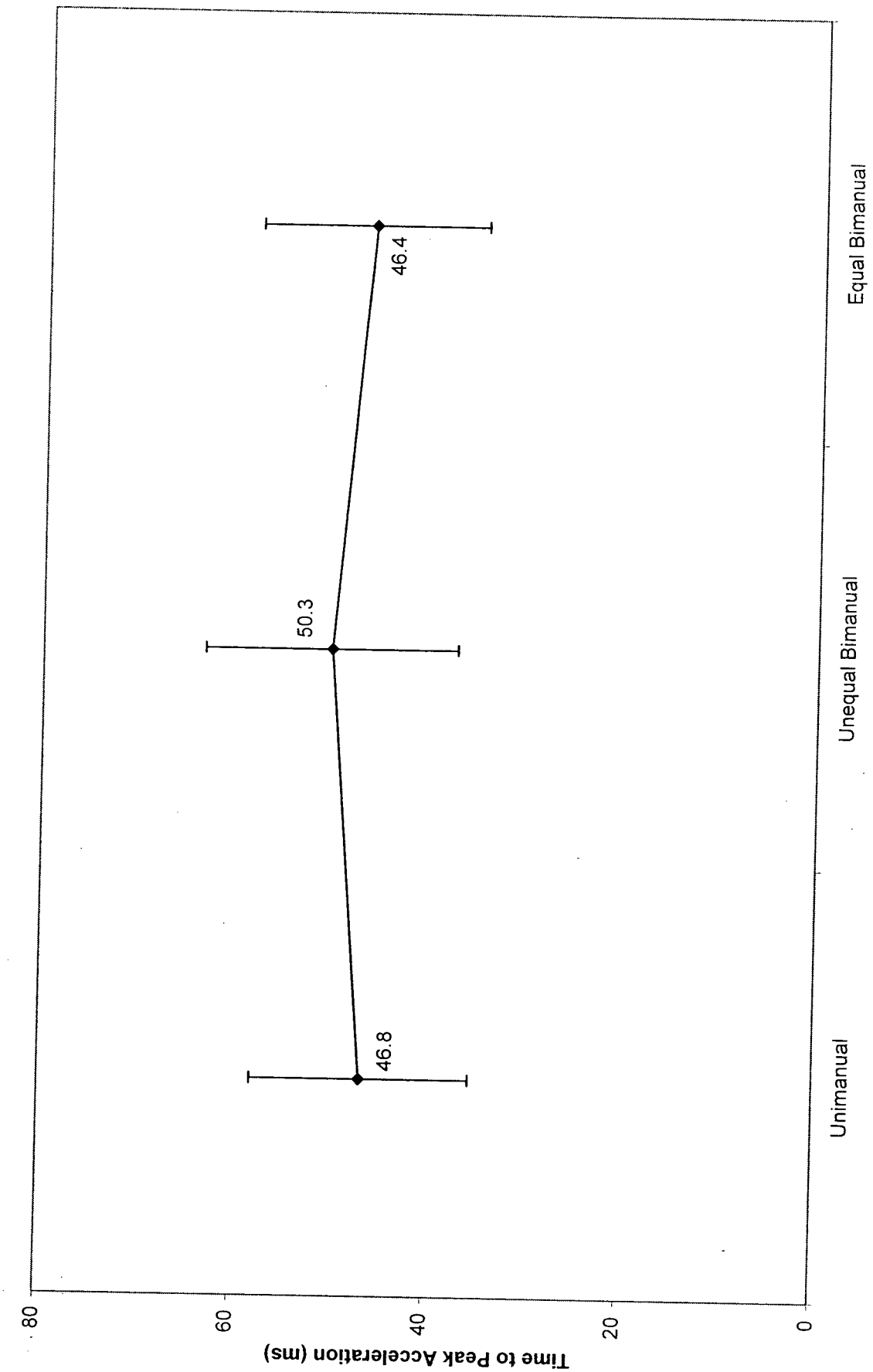


Figure 14. Time to Peak Acceleration (collapsed across hand)

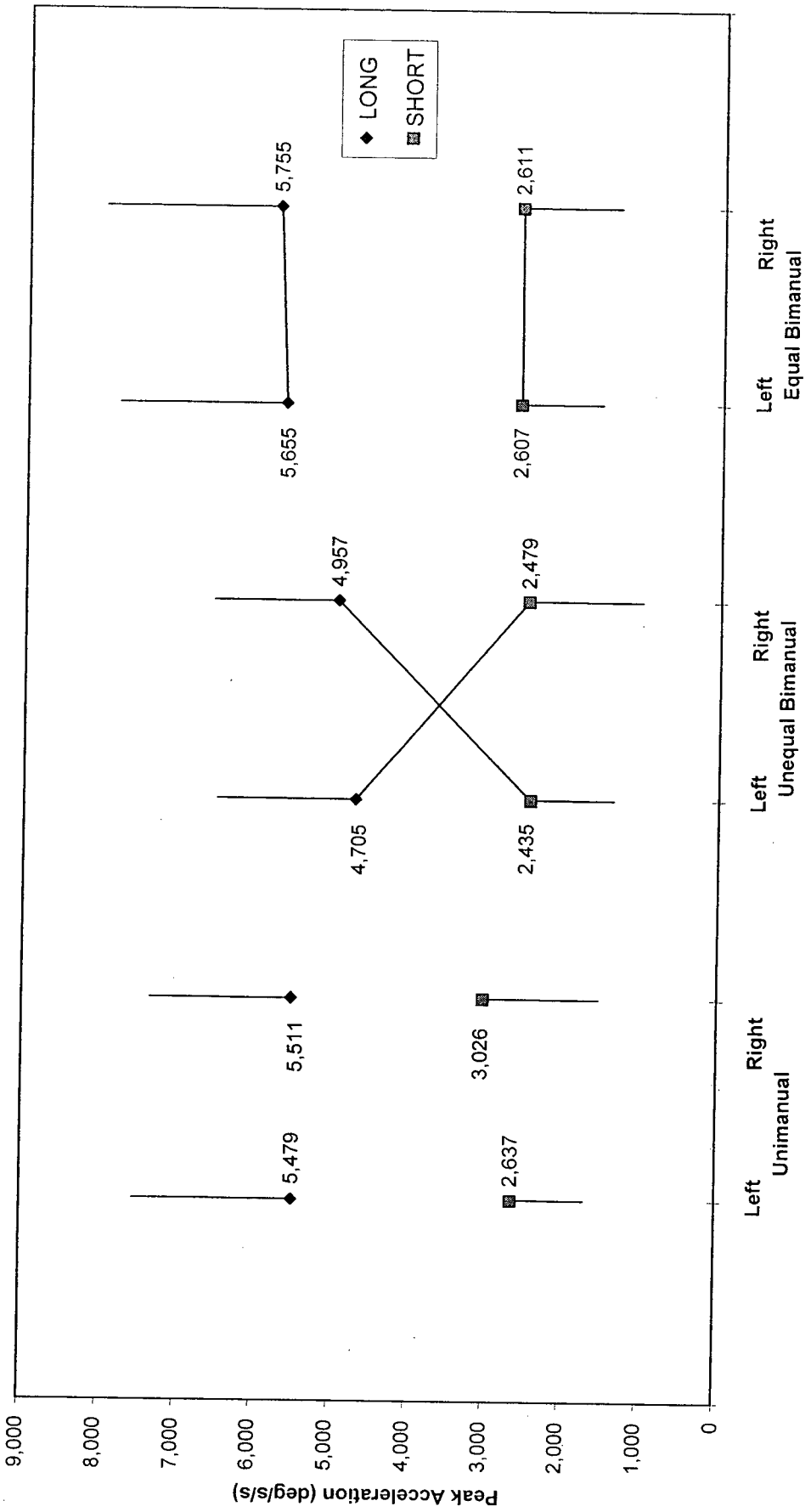
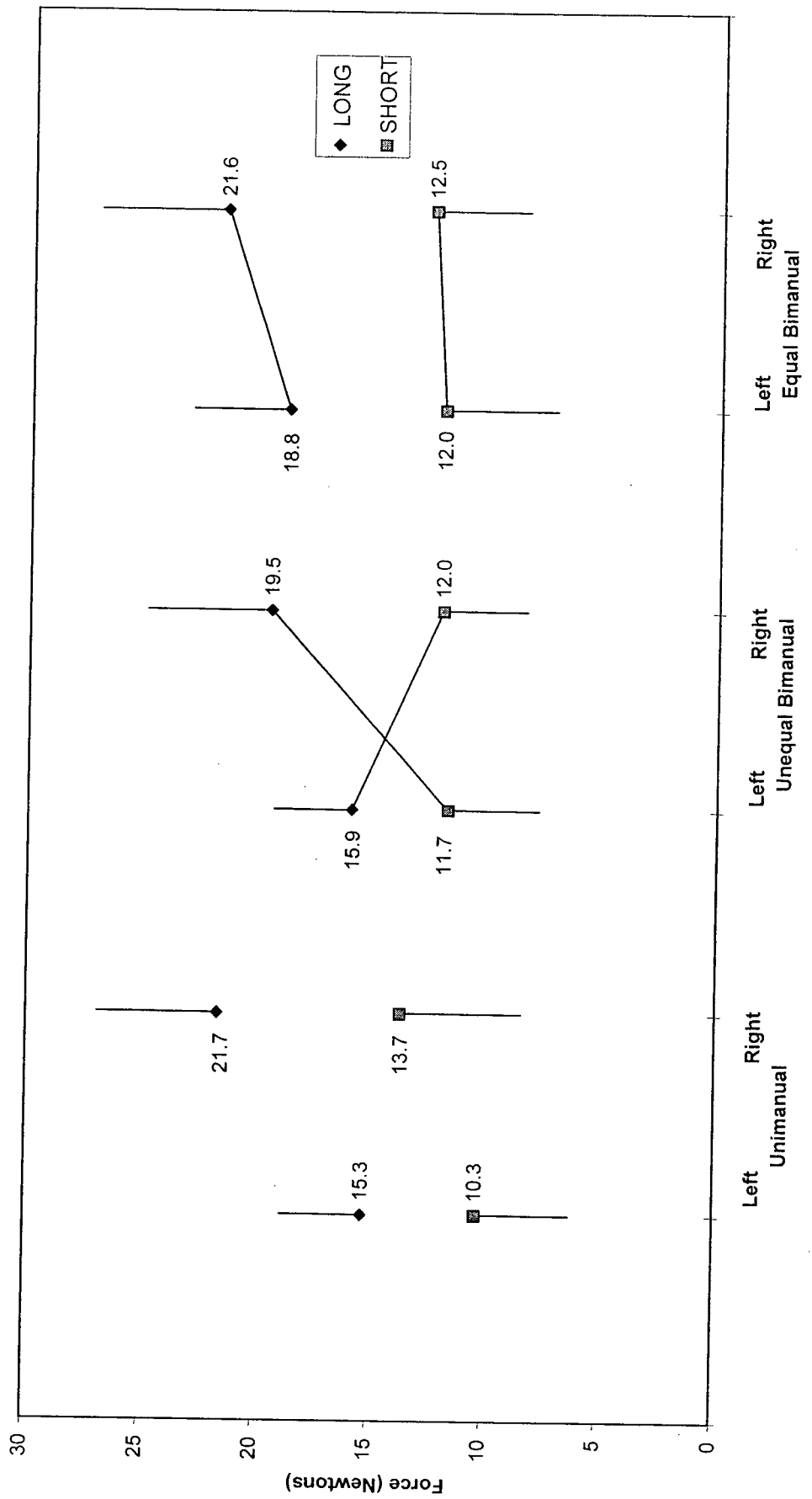


Figure 15. Positive Peak Acceleration

Figure 16. Peak Positive Force



EMG

Triceps Onset. Triceps onset time, or EMG reaction time, had a main effect for movement condition $F(2, 18) = 16.031$, $p < 0.001$, and a Condition x Distance interaction $F(2, 18) = 4.537$, $p = 0.029$ (see figure 17). Collapsing left and right hand data in figure 18 there is a significant increase in short movement triceps onset time in the unequal-bimanual condition (160 ms) compared to unimanual (138 ms) and equal-bimanual (140 ms) conditions.

Q30. The Q30 measure of EMG onset slope and magnitude displayed a main Distance effect $F(1,9) = 24.360$, $p = 0.001$, with long distance movements averaging 2372 (%MVC • ms) and short distance movements averaging 1874 (%MVC • ms). There was a main Hand effect $F(1,9) = 7.982$, $p = 0.02$, with smaller right hand Q30 values than left hand values over all conditions and distances (see figure 19).

Q100. The Q100 measure of EMG area displayed main effects for movement Condition $F(2, 18) = 4.988$, $p = 0.021$ and Distance $F(1, 9) = 93.571$, $p < 0.001$ with larger values for longer movement distances, and smaller values for the unequal-bimanual movement compared to unimanual or symmetric-bimanual movements.

Triceps Onset – Biceps Onset Difference. The difference between agonist triceps onset and antagonist biceps onset displayed a main effect for Distance, and a Condition x Distance interaction $F(2, 18) = 8.548$, $p = 0.002$. For all conditions biceps started later for longer movements (see figure 20). Unimanual movements had left hand biceps EMG starting sooner than right hand biceps onset for both short (69 ms left – 77ms right) and long movements (113 ms left – 125 ms right), while both bimanual conditions had very little

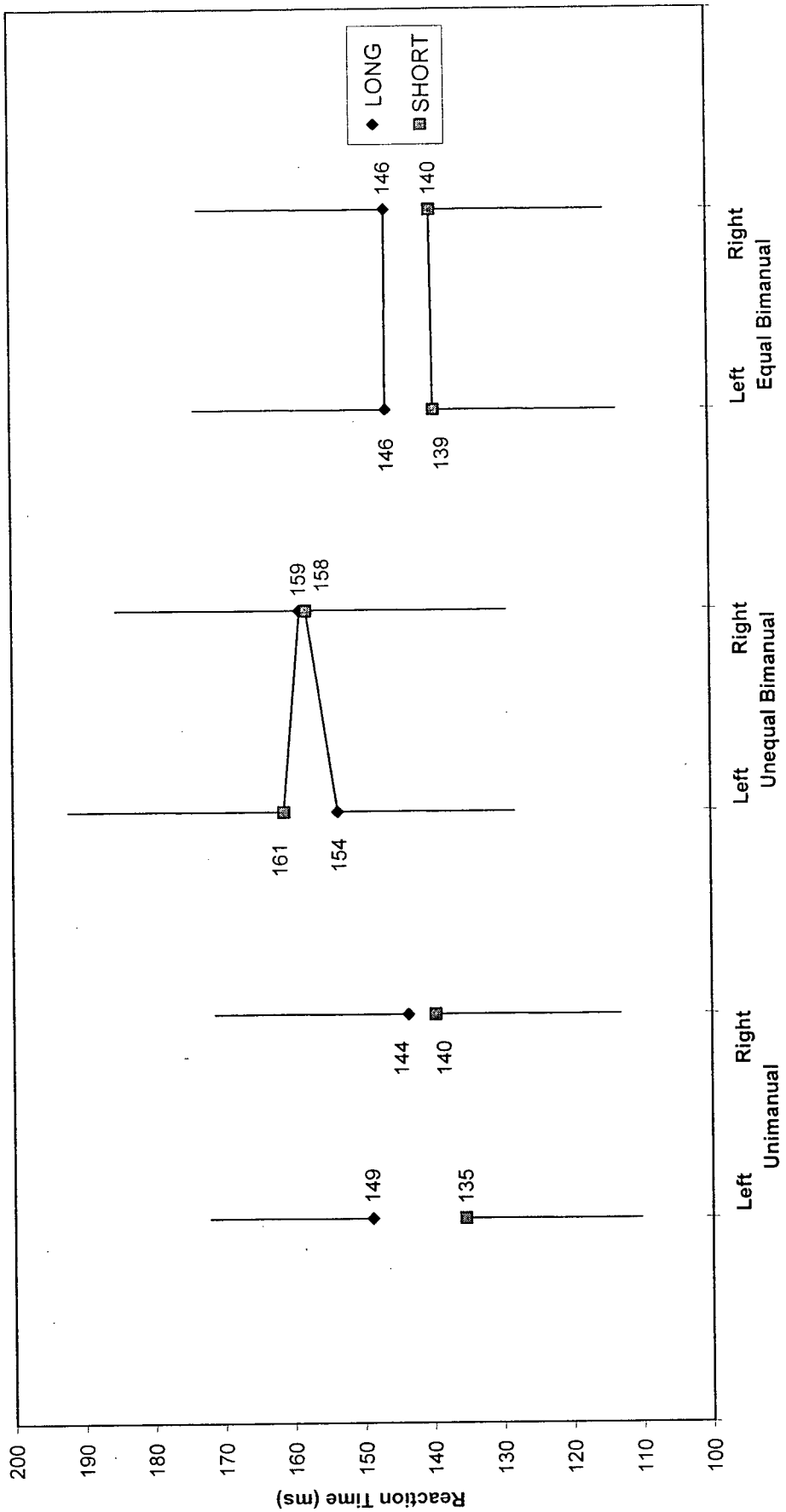
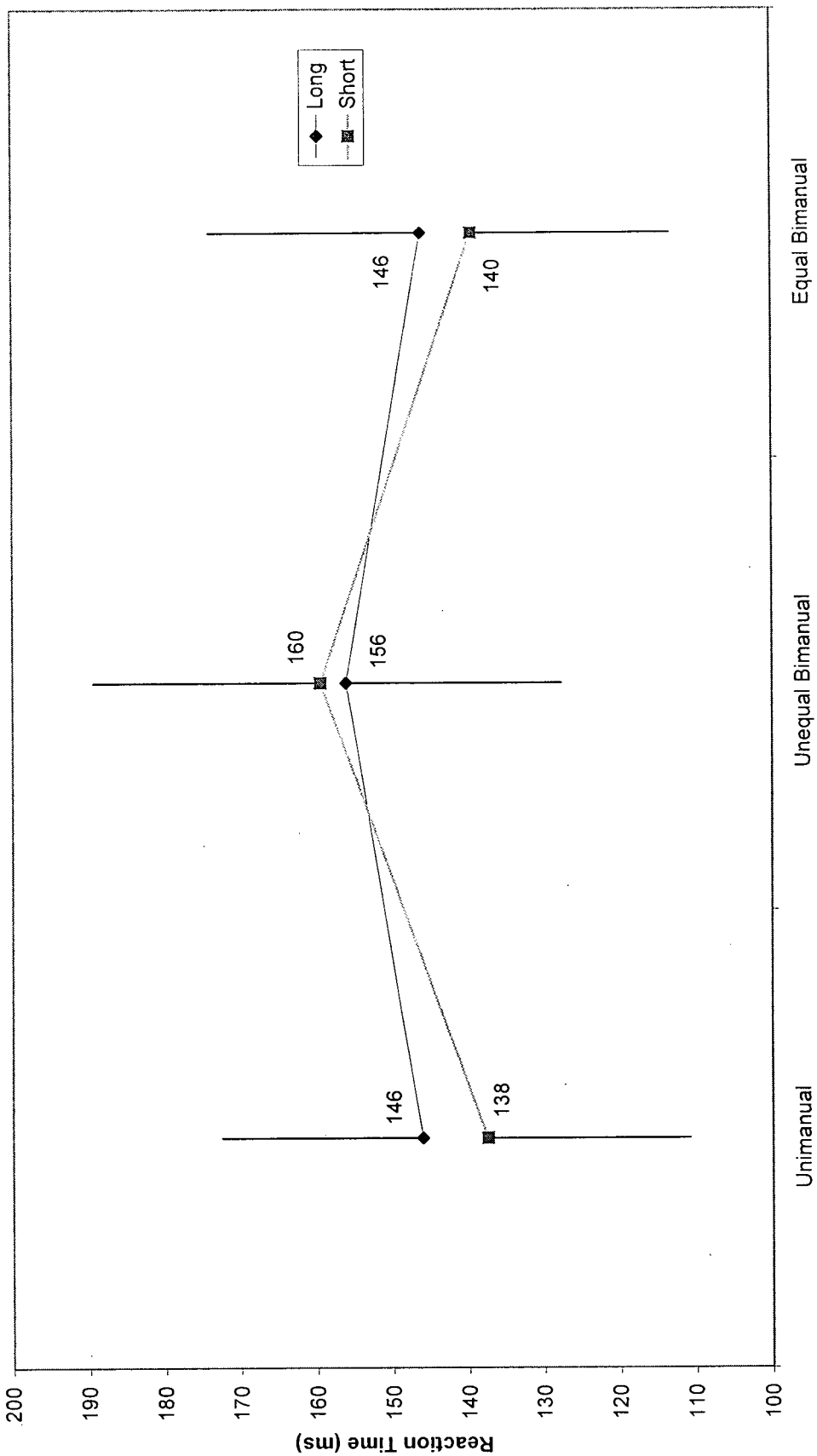


Figure 17. Triceps Onset Reaction Time

Figure 18. Triceps Onset Reaction Time (collapsed over hand)



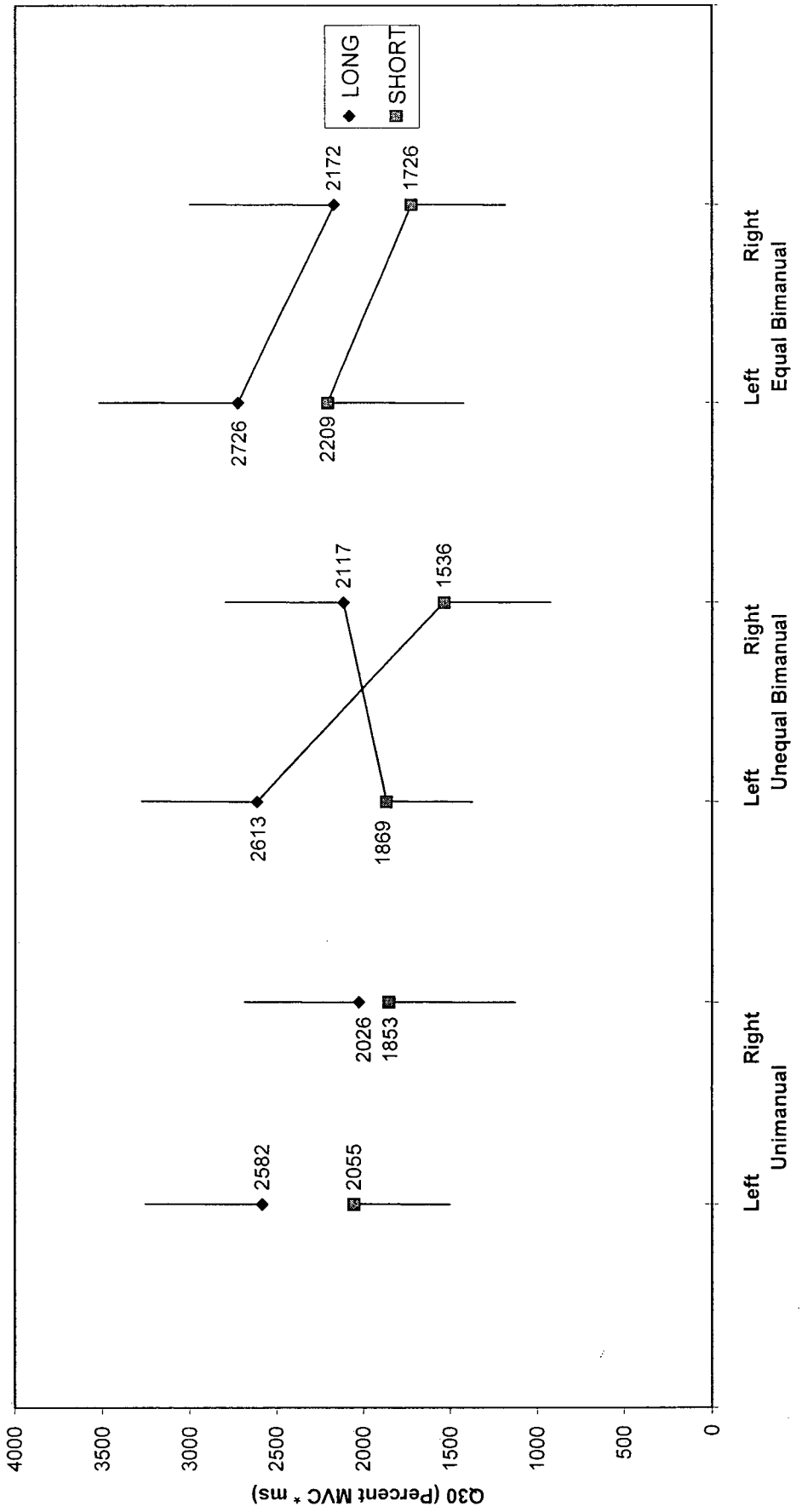
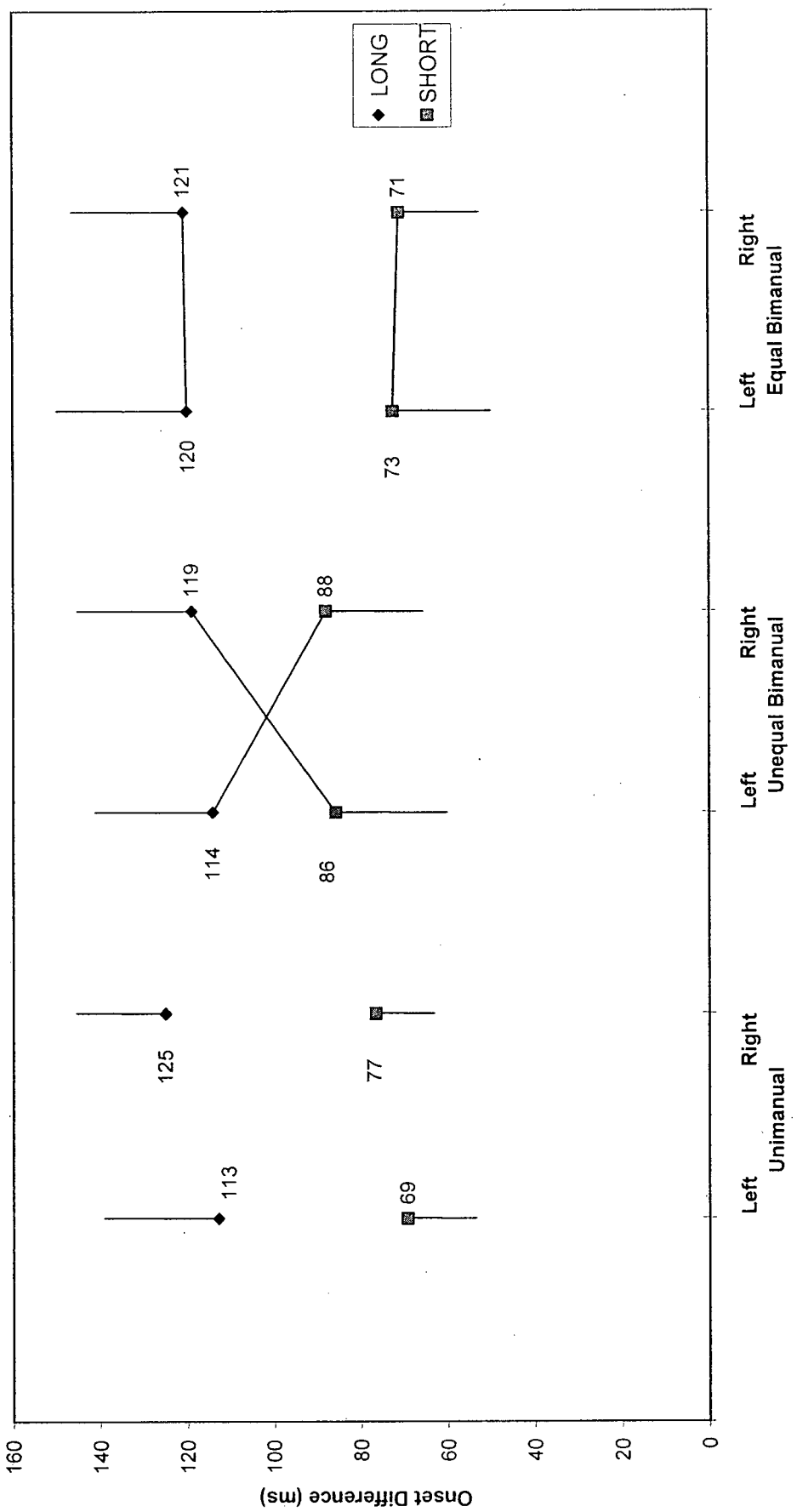


Figure 19. Q30 EMG Onset Slope Measure

Figure 20. Triceps Onset - Biceps Onset Difference



variation between short (73 ms – 71 ms) and long (120 ms – 121 ms) movements, with the shorter movement of unequal-bimanual movements having longer onset times (86 ms - 88 ms) than the short-short bimanual movement (73 ms - 71 ms).

Ensemble Averages

EMG.

- Handedness. No differences were found for the EMG patterns for left and right hands for short or long movement distances (see figures 21, 22). Thus EMG patterns for left and right hands were combined for short and long movements within each condition to produce a single trace of agonist-antagonist EMG burst patterns.
- Burst Pattern. All unblocked movements displayed triphasic EMG patterns with similarities and differences between short and long movements: 1) similar slope of EMG rise for first agonist onset, 2) increased peak EMG amplitude for longer movements, and 3) later onset of antagonist activity in long distance movements compared to short distance movements (see figures 23, 24).
- Unimanual/Bimanual. Unimanual and similar-distance bimanual movements displayed similar EMG burst patterns for short and long distance movements, while unequal-bimanual movements displayed different EMG burst patterns for short distance movements (see figures 25, 26). Both unimanual and equal-bimanual short distance movements displayed similar first agonist EMG amplitudes and agonist onset times, while the unequal-bimanual movement has reduced first agonist amplitude and delayed antagonist onset (see figure 25).
- Same Hand Blocking. Movement blocking affected the produced pattern of EMG activity for the blocked hand, for short movements there was very little difference in

Figure 21. Short Unimanual Movement EMG

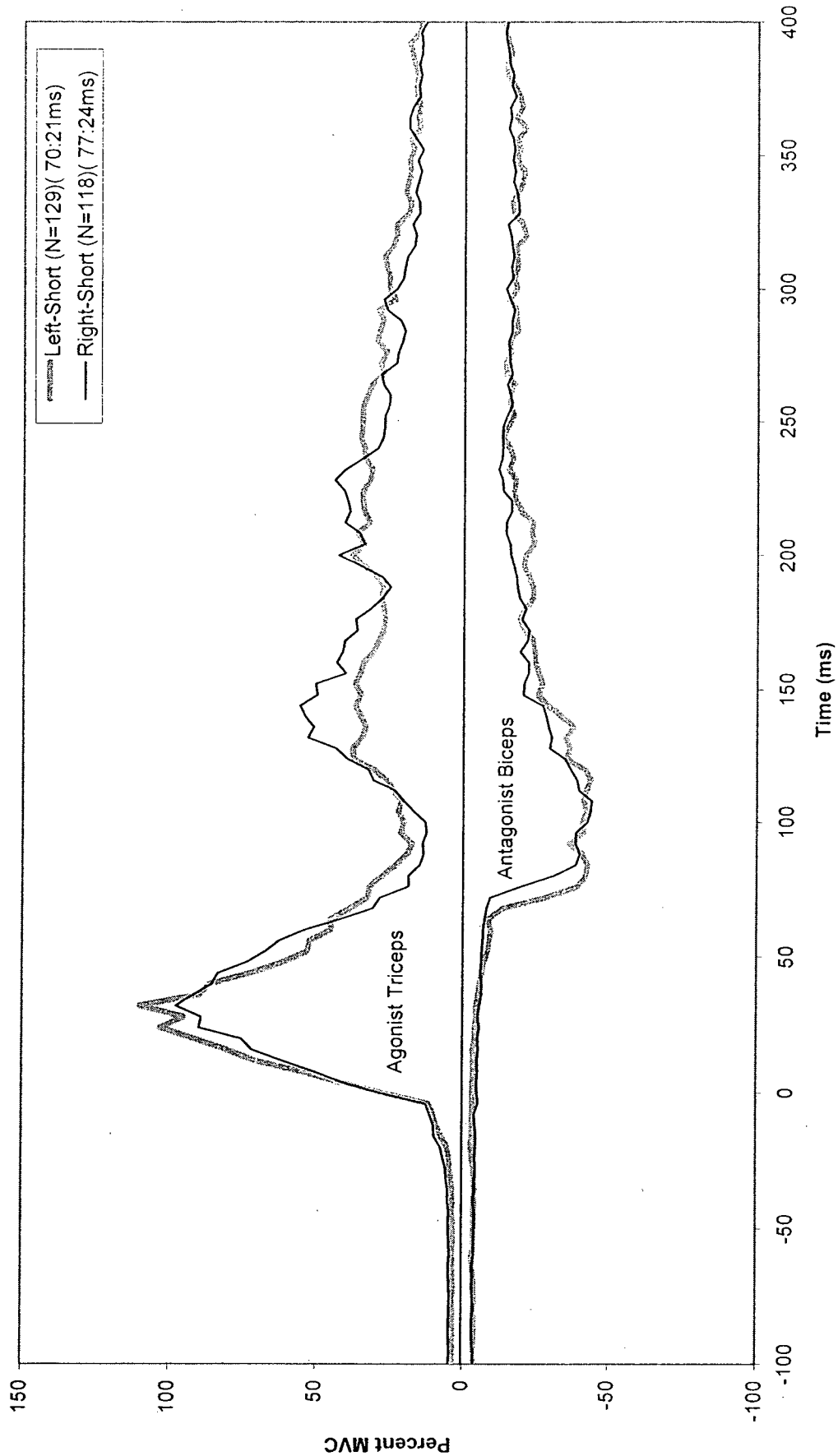
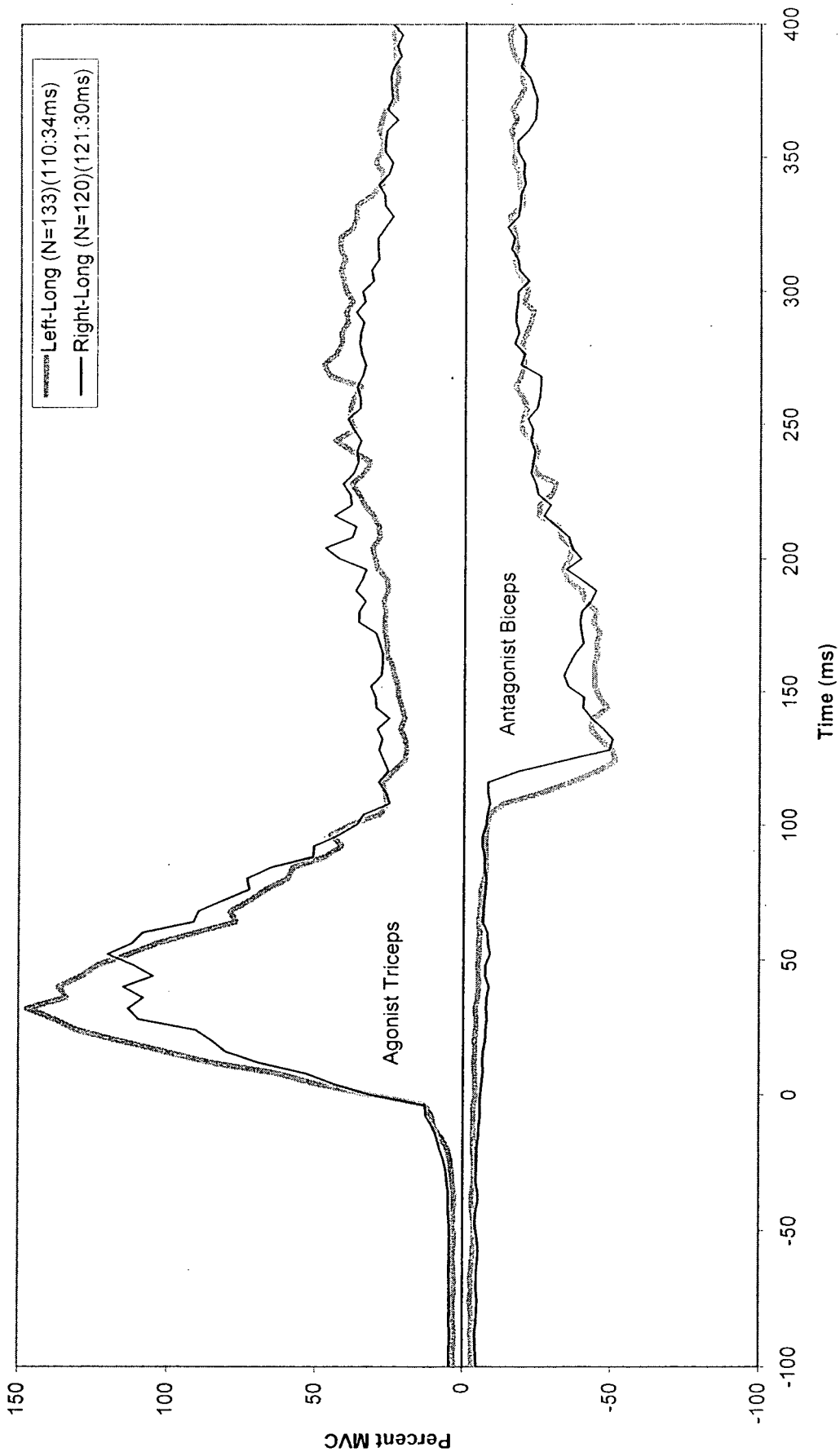


Figure 22. Long Unimanual Movement EMG



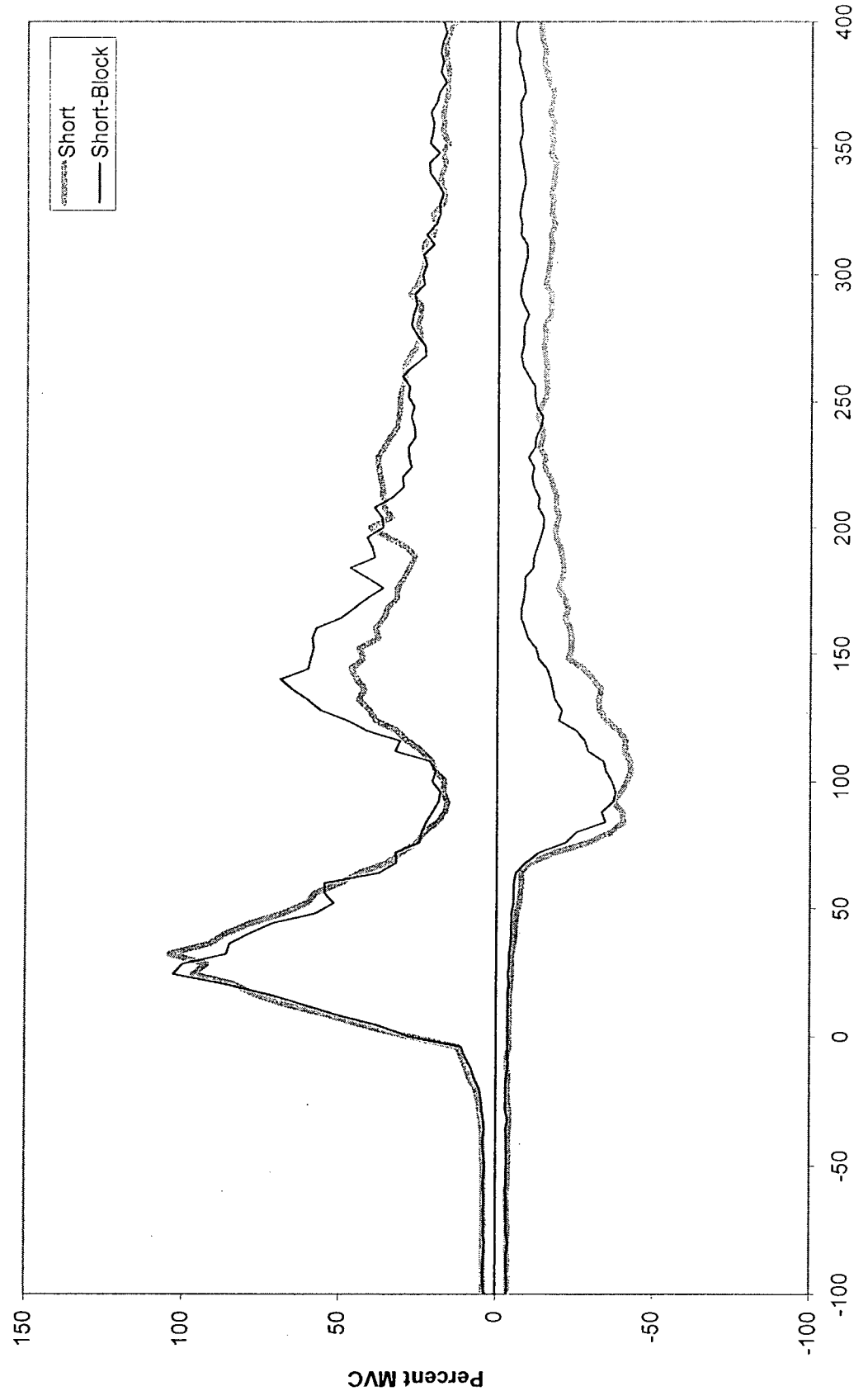


Figure 23. Short Unimanual Movement EMG (collapsed across hand)

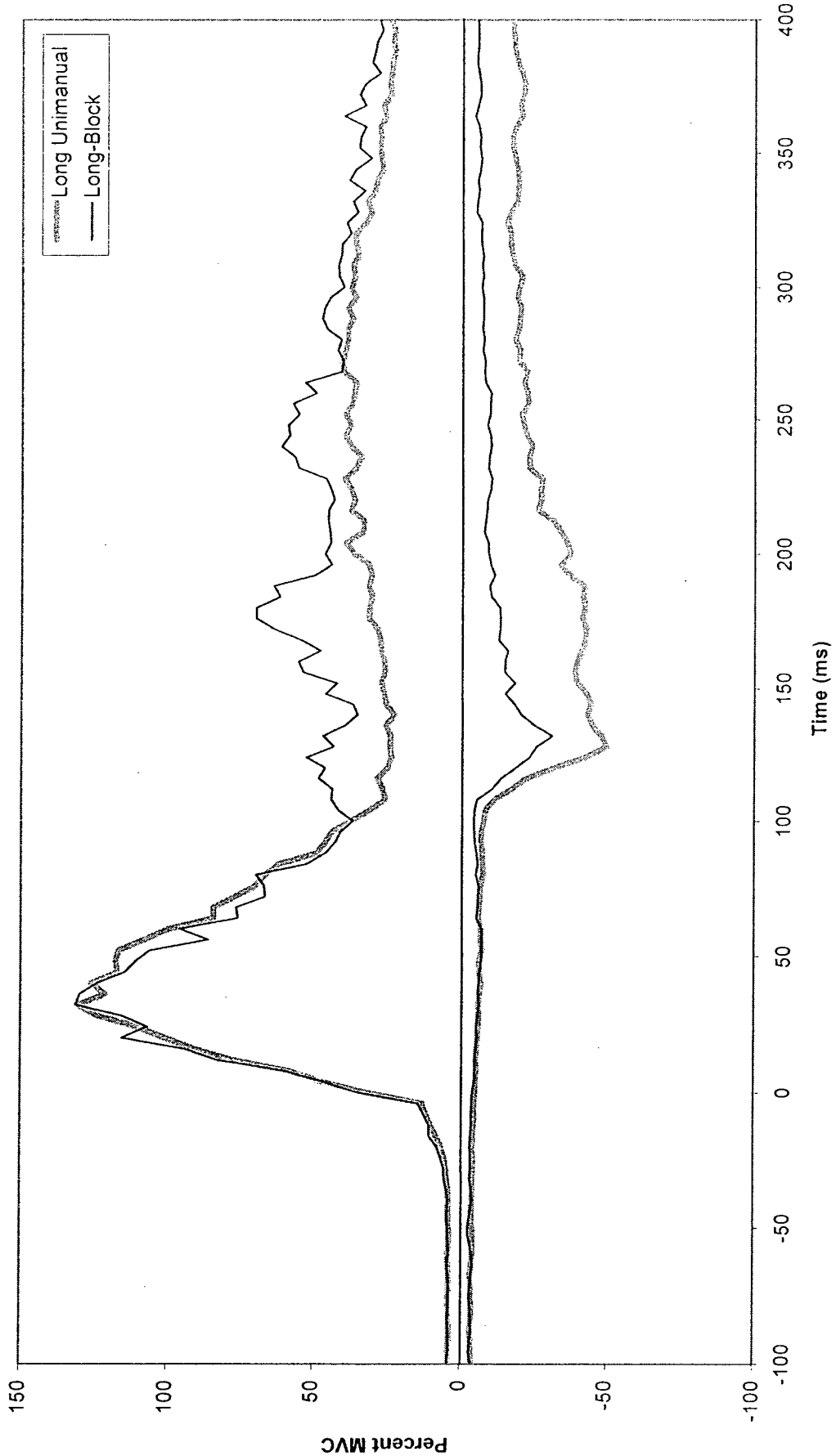


Figure 24. Long Unimanual Movement EMG: Effect of Blocking

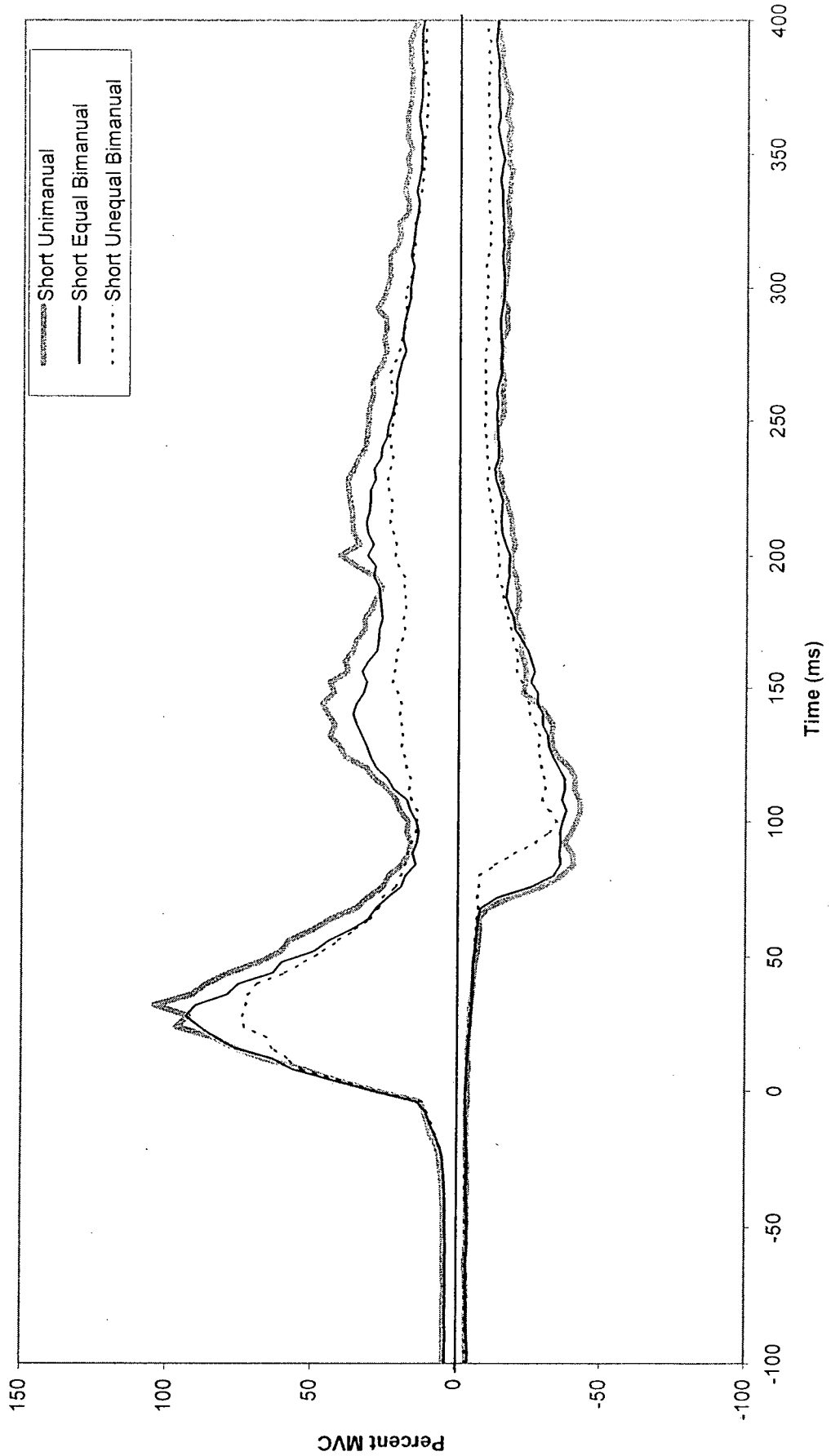


Figure 25. Short Movement EMG Patterns (Unimanual and Bimanual)

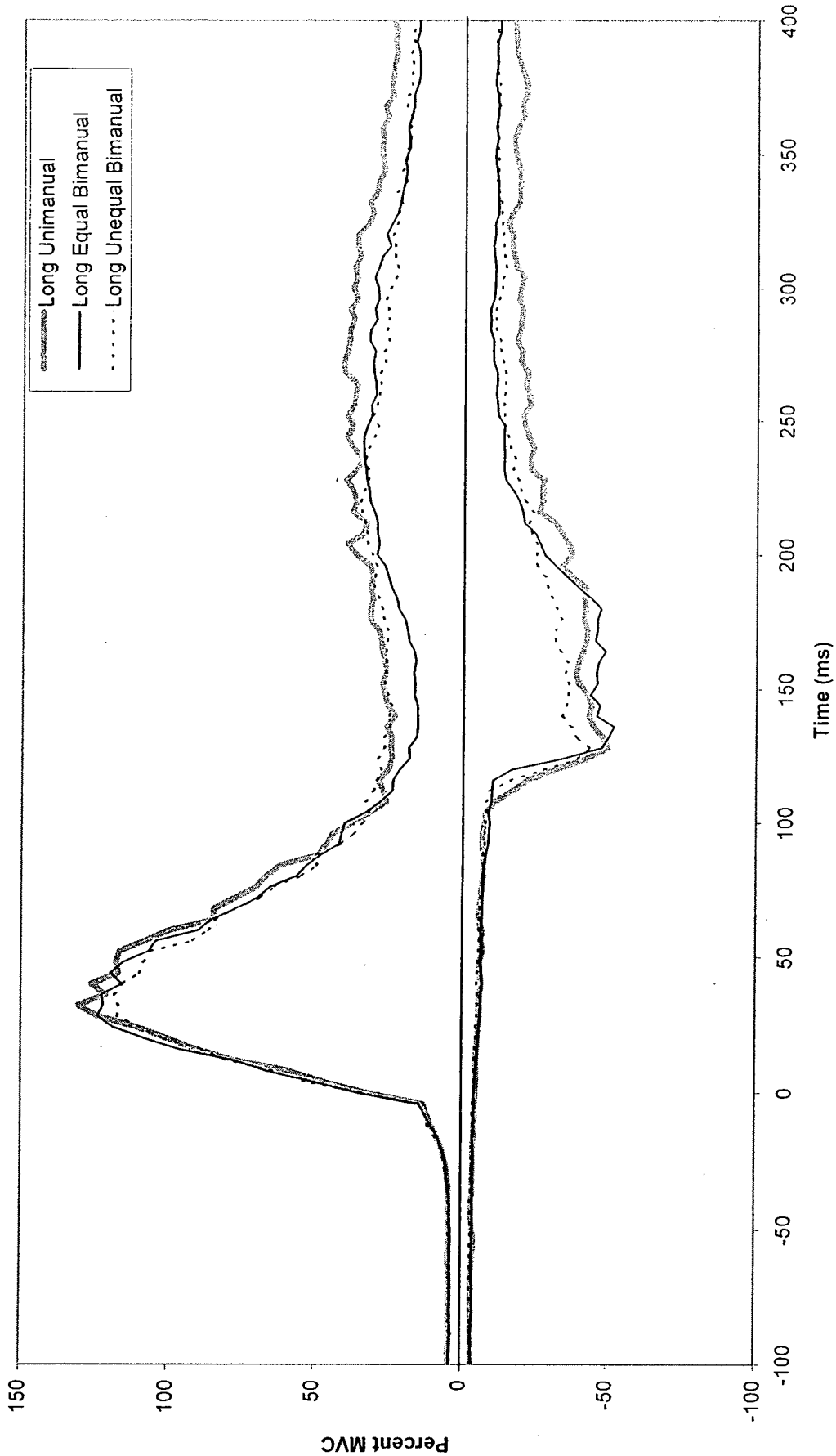


Figure 26. Long Movement EMG Patterns (Unimanual and Bimanual)

the EMG burst patterns for approximately 100 ms after agonist onset, with reduced antagonist EMG amplitude and increased agonist amplitude after 100 ms (see figs. 23, 27, 29). The EMG pattern of long movements is affected more drastically by movement blocking: since antagonist onset is typically after 100 ms: antagonist EMG amplitude is greatly reduced, and agonist activity is elevated (see figures 24, 28, 30).

- Opposing Hand Blocking. In order to investigate the effect of blocking on the opposite, unblocked hand, various EMG and kinematic measures were analyzed for differences. Blocked limb EMG patterns were attenuated approximately 100-125 ms after agonist onset, with reduction in the antagonist biceps EMG, and an increase in the second agonist triceps EMG (see figures 27, 28). The unblocked limb's EMG patterns were analyzed for differences in antagonist burst onset and offset times. In addition, unblocked limb movement time and movement distances were analyzed for the effects due to opposing limb blocking. Analysis of the antagonist onset time revealed no significant main effect for movement blocking $F(1, 9) = 1.608$, $p = 0.237$, as did antagonist offset time $F(1, 9) = 1.721$, $p = 0.222$. There was no measurable interaction between the two limbs during movement blocking, perturbing one hand has no perceivable effect on movement production of the opposing hand (see figures 23, 24, 27, 28, 29, 30).

Kinematics. As expected, short distance movements had reduced movement times compared to the long distance movements, with similar movement times for both unimanual and equal-bimanual movements (see figure 9). While unequal-bimanual movements usually resulted in slower production of the short distance movement as seen in movement time (see figures 31, 32), this experiment showed only minor increases in both short and long movement times

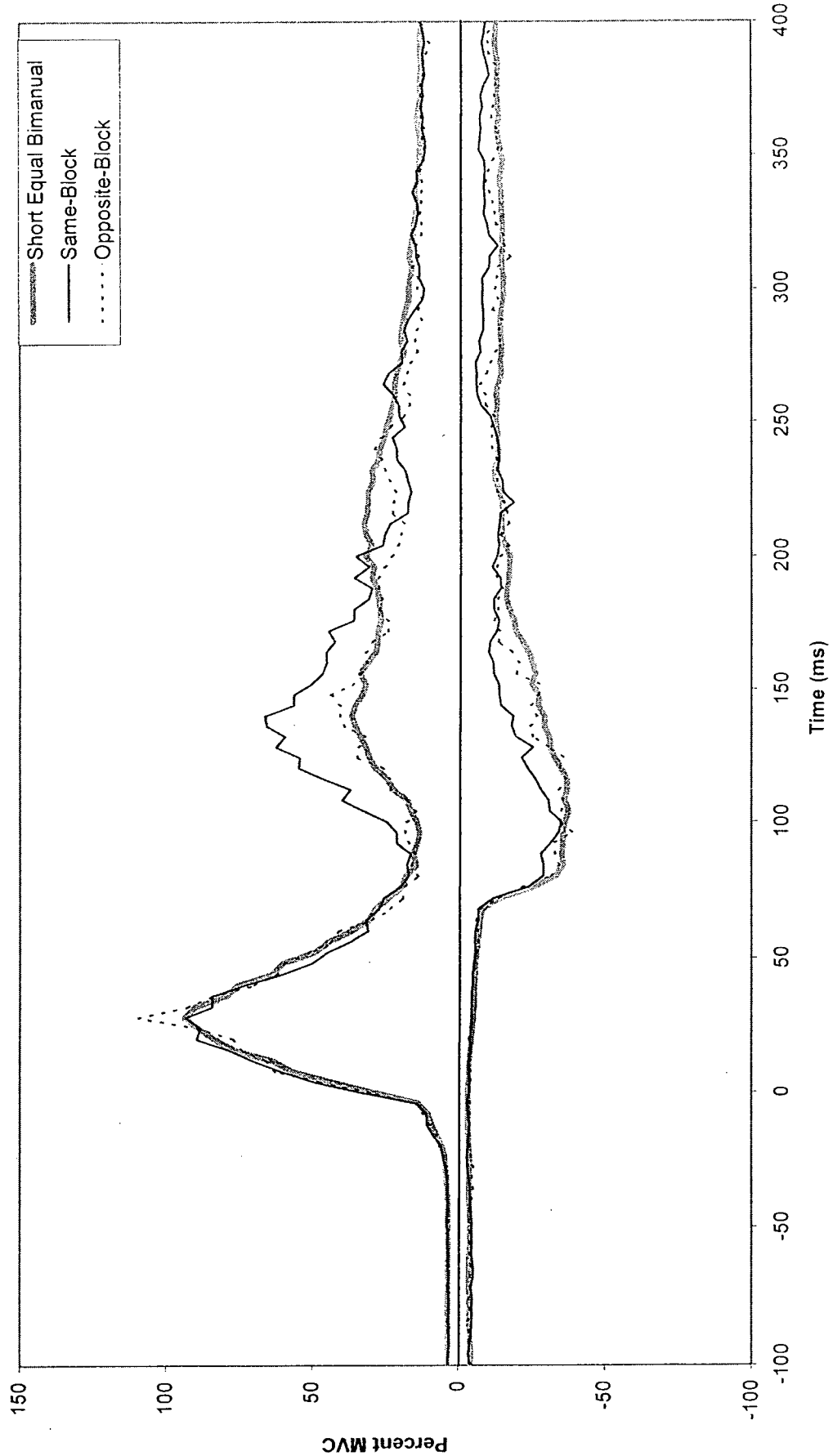


Figure 27. Short Equal Bimanual EMG: Effect of Blocking Hand

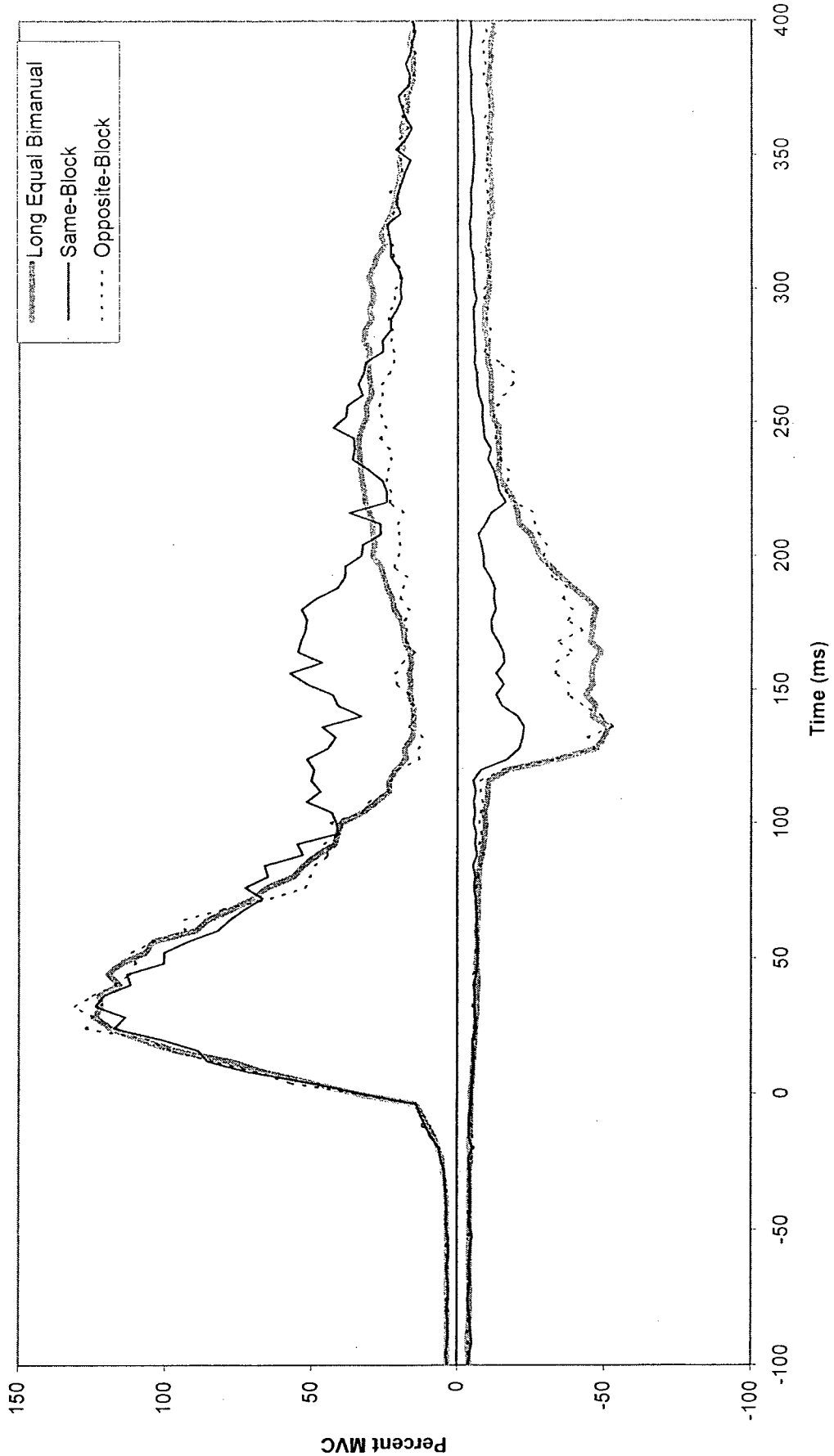


Figure 28. Long Equal Bimanual EMG: Effect of Blocking Hand

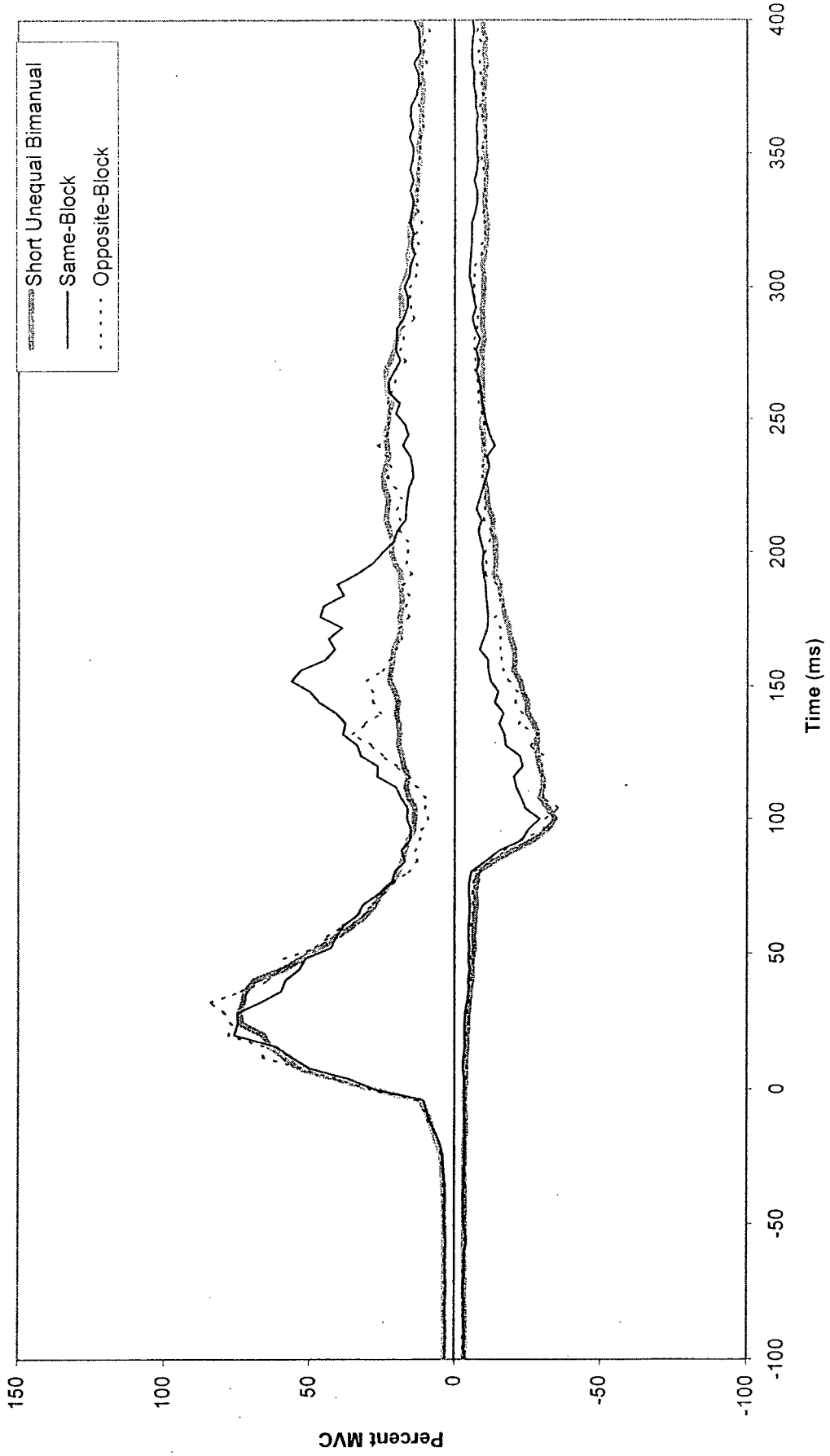


Figure 29. Short Unequal Bimanual EMG: Effect of Blocking Hand

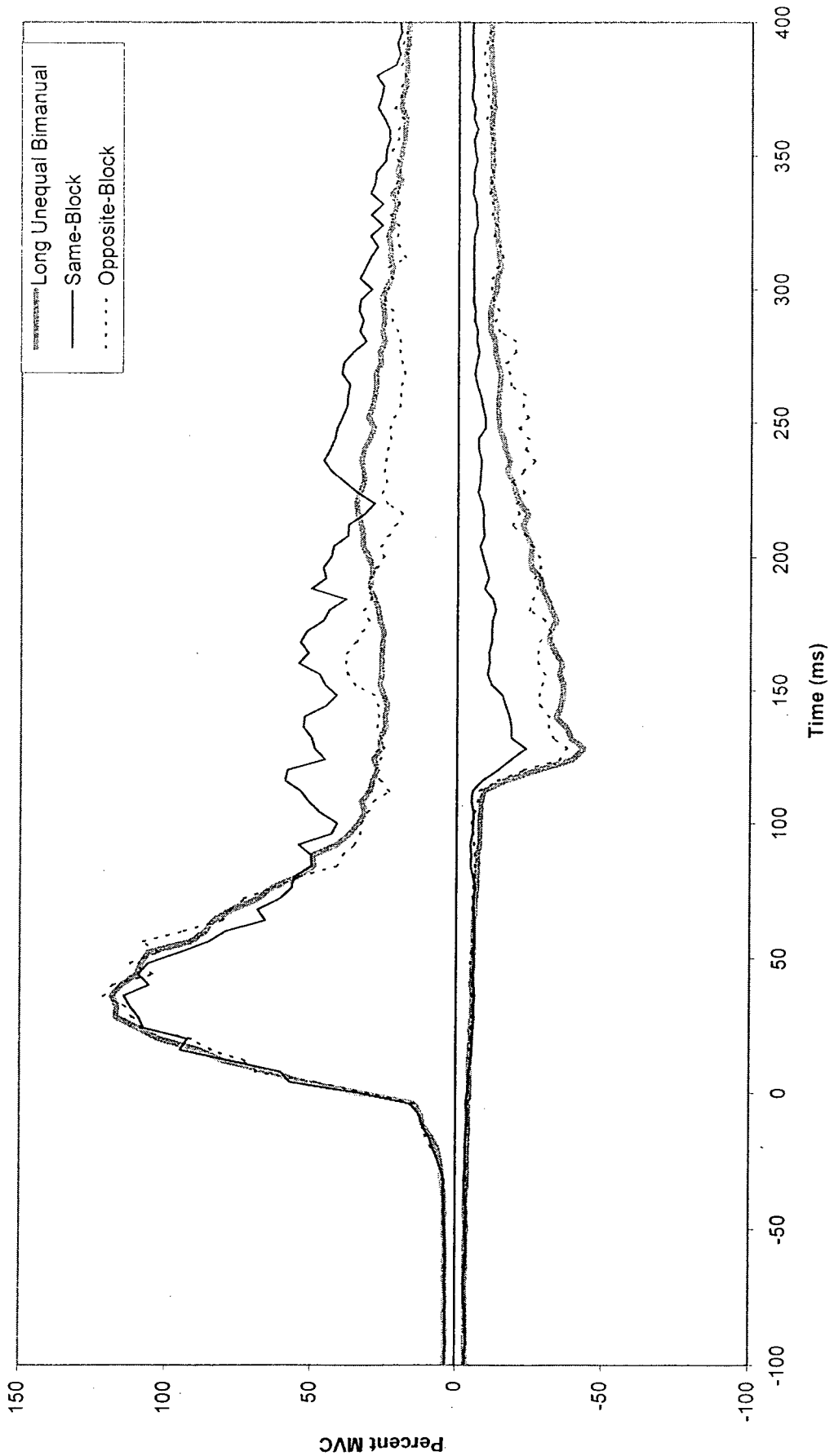


Figure 30. Long Unequal Bimanual EMG: Effect of Blocking Hand

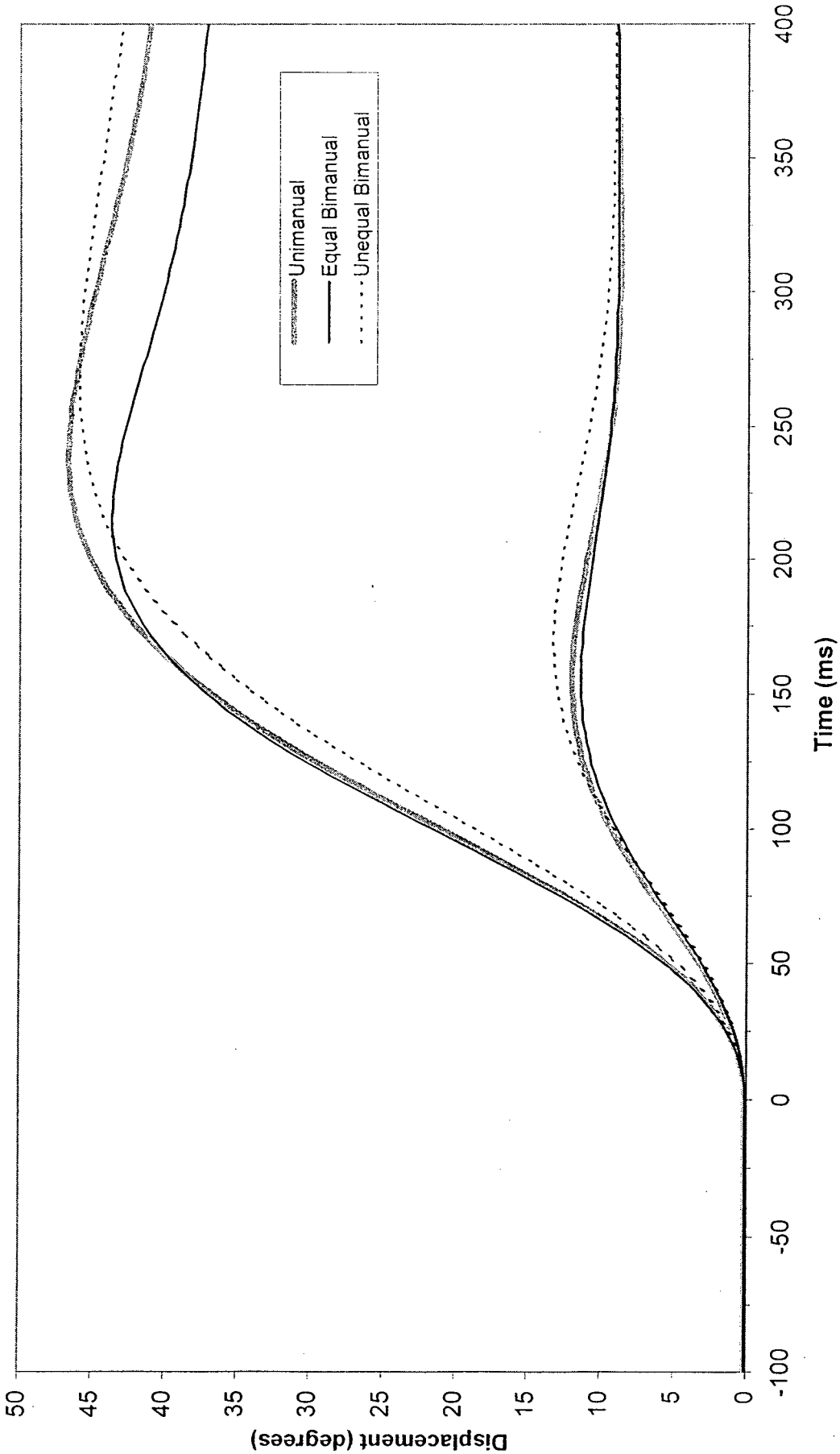


Figure 31. Displacement Profile

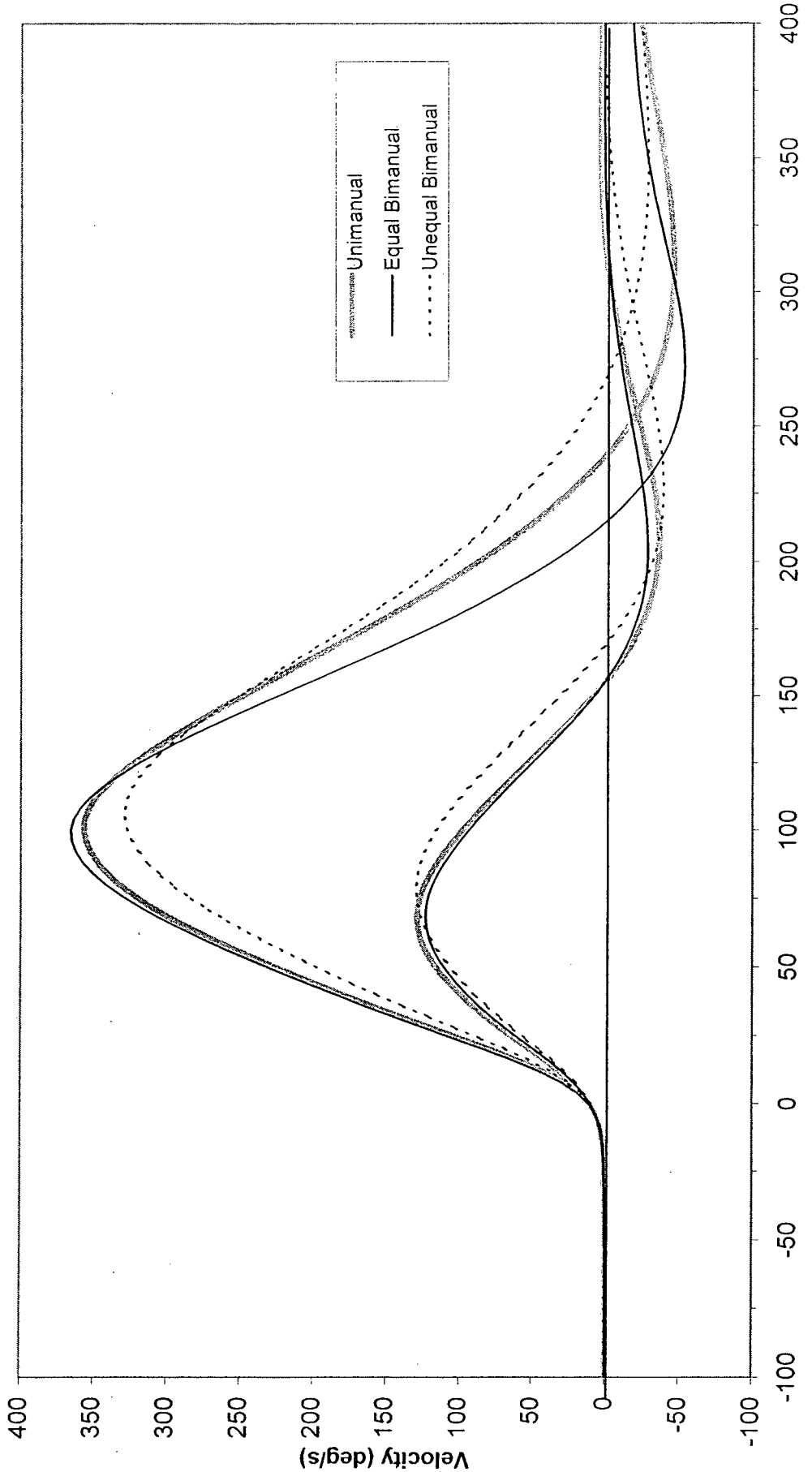


Figure 32. Velocity Profile

compared to unimanual or equal-bimanual movements (see figure 9). While part of the increase in the short movement time may be due to increased short movement distance, there is no corresponding increase in the long movement distance (see figure 1), and conversely no decrease in short distance peak velocity (see figure 7).

The pairing of long and short distance movements resulted in longer movement times for both movements, but for different reasons. The long distance movement had a reduced peak velocity value, while maintaining movement distance, indicating a decreased amplitude but longer duration movement impulse, while the short distance movement displayed a similar amplitude impulse of longer duration (see figure 31). These kinematic differences indicate differences in the muscle activation patterns producing movement, as seen in recorded muscle EMG.

Discussion

This thesis was designed to investigate the *influences*, *affects*, and *interactions* of limbs during bimanual movements. Decisions about the nature of an intended movement influenced the generation of EMG patterns used to create that movement, differences in EMG patterns were clearly visible before any movement began. Movement blocking affected the blocked limb's EMG patterns through reflexive responses to the physical prevention of movement, causing stereotyped changes in agonist and antagonist EMG. Blocking one limb of a bimanual movement had no measurable affect on the unblocked limb, indicating that there was no measurable interaction between the two limbs once movement began. Four unimanual and four bimanual movement conditions were used to compare and contrast the effects of performing short and long movements singly and paired together.

Unimanual Movements. Discrete, goal-directed single joint movements are characterized by a triphasic pattern of EMG activity consisting of a first agonist burst that accelerates the limb, a decelerating antagonist burst, and a second agonist burst that clamps the limb at the end of movement (Berardelli, Rothwell, Day, Kachi, and Marsden, 1984; Enoka, 1988). This EMG pattern generates muscle force that accelerates the limb, in a sinusoidal pattern of positive acceleration followed by negative acceleration. Typically, each EMG burst is symmetrical with equal onset and offset slopes, creating positive and negative acceleration profiles of nearly equal amplitude. Previous studies have shown that it is possible to alter acceleration and deceleration EMG durations, creating acceleration symmetry ratios ranging from 2:5 to 1:2 for acceleration time vs. deceleration time (Brown & Cooke, 1990).

This experiment consisted of short distance unimanual movements of 10 degrees and long distance unimanual movements of 50 degrees, with participants instructed to move "as quickly and as accurately as possible". Participants responded to these instructions by performing elbow extension movements with maximum agonist muscle activation levels, extending the duration of the agonist and antagonist EMG bursts in order to achieve longer movement distances. This technique of fixing the amplitude of agonist activation (at maximum) and modulating activation time is known as the "speed-insensitive" strategy, whereas fixing duration and modulating EMG amplitude is known as the "speed sensitive" strategy (Latash & Gottlieb, 1991a). Long distance movements were characterized by longer duration EMG bursts resulting in longer duration and larger amplitude positive acceleration profiles. Short distance movements had first agonist bursts of approximately 75 ms (as

measured from agonist onset) and peak amplitude of approximately 100% MVC (see figure 22), while long distance movements had agonist burst durations of approximately 100-110 ms and peak amplitudes of approximately 125% MVC (see figure 23). Antagonist onset time for the short unimanual movement was approximately 73 ms measured from agonist onset, while long movement antagonist onset time was approximately 118 ms (see figure 20). Overall, unimanual movements were powered by a fixed magnitude EMG burst with modulation of burst duration to achieve movement distance: short duration triphasic burst patterns produced short movements while longer duration triphasic patterns produced long distance movements.

Short distance unimanual movements had very similar EMG and kinematic patterns for left and right hands even though these movements were performed by different limbs at different times. Long distance EMG and kinematic patterns were also similar for left and right hands, yet were distinctively different from short distance movement patterns. Kinematic and EMG data for left and right hands were combined for short movements, and separately for long distance movements (see figures 22, 23). Similarly, left and right hand data were combined for similar distance movements in both unequal and equal distance bimanual movements, giving EMG and kinematic data for short and long distances over all three conditions: unimanual (see figures 22-23), equal distance bimanual movements (see figures 27-28), and unequal distance bimanual movements (see figures 29-30).

When a unimanual movement was blocked, where movement was mechanically prevented, the pattern of EMG was attenuated by peripheral feedback approximately 100-125 ms after agonist onset. This time delay was composed of muscle motor time of approximately 50 ms, and feedback response time of approximately 50 ms (Wadman, et al.,

1979; Latash & Gottlieb, 1991b; Brown & Cooke, 1981). There was very little difference between the EMG patterns of blocked and unblocked short distance unimanual movements for the first 100-125 ms from first agonist onset (see figure 22). However, EMG differences due to blocking after this critical time (100ms after agonist onset) were characterized by reduction of antagonist burst intensity and an increase in the second agonist burst magnitude (see figure 22). Long distance unimanual movements were characterized by a first agonist burst of approximately 100-120 ms duration, compared to 75-90 ms for short movements, and antagonist burst onset times of 110-120 ms compared to 60-70 ms for short movements (see figures 22-23). Movement blocking had a different effect on short distance EMG patterns compared to long distance EMG, where antagonist EMG was affected more for long distance movements. In short distance movements antagonist EMG had already reached peak amplitude by 100 ms, so any reflex attenuation due to blocking only reduced the antagonist amplitude towards the end of the burst, slightly shortening the burst duration (see figure 22). On the other hand, long distance movements had first agonist durations of approximately 100 ms and antagonist onset times of approximately 110 ms. Therefore reflex attenuation due to blocking occurred at or after antagonist onset which resulted in reduced or in some cases no measurable antagonist EMG activity. Consequently this led to reduced peak amplitude and overall burst duration compared to unblocked movements (see figure 23). Under movement blocking the stretch reflex increased and extended agonist EMG activity to correct for limb position error, and through reciprocal inhibition also reduced antagonist EMG. The inhibition effect was more pronounced in the EMG patterns of intended long distance movements as antagonist activity had not yet begun when it was inhibited, whereas

in short movements antagonist EMG was essentially completed when inhibition had begun (see figures 22, 23).

The differences and similarities of EMG patterns seen in short and long blocked movements indicate at least two distinct mechanisms to create and maintain an intended movement. The first mechanism allows the generation of movement without the necessity of peripheral feedback (Lashley, 1917; Bizzi, et al., 1978; Nougier, et al., 1996), while the second mechanism results in movement corrections due to peripheral feedback (Wadman, et al., 1979; Forget & Lamarre, 1987). While various models have been proposed to explain these control processes (see St. Onge, Adamovich, and Feldman, 1997; Gottlieb, 1996), ultimately it is the membrane potentials of extensor and flexor motoneurons that determine if resting muscles will contract to initiate movement (St. Onge, Adamovich, and Feldman, 1997). Membrane potential changes can occur under central control without peripheral feedback and creates reasonably predictable limb movements in deafferented monkeys (Bizzi, 1980), and deafferented human patients (Lashley, 1917; Forget and Lamarre, 1987). However, peripheral sensory information is required to control activation of the antagonist muscle to provide correct timing and amplitude control of antagonist EMG for accurate movements (Forget and Lamarre, 1987; Nougier, et al., 1996. A comparison of blocked and unblocked EMG patterns, aligned at the onset of the first agonist burst, showed a divergence 50-60 ms after blocked movements impacted with the barrier. Movement typically started 50-60 ms after the first agonist EMG onset, so reflex-based EMG differences due to movement blocking were typically seen 100-120 ms after the initial agonist burst onset. This stretch reflex compensates for movement inaccuracies due to load (Forget and Lamarre, 1987; Adamovich, Levin, and Feldman, 1997), and provides coordination for multiple joint

movements (Bizzi, 1980). Rapid movements of the human elbow joint are generated by shifting the equilibrium point of agonist and antagonist muscle activation thresholds at maximum speed, approximately 600 degrees per second, which results in the generation of a triphasic EMG pattern for short movements, and feedback-attenuated triphasic EMG for longer movements (Feldman, Adamovich, Levin, 1995; Gottlieb, 1996).

Equal Distance Bimanual Movements. Equal distance bimanual EMG reaction times were less variable compared to their unimanual counterparts (see figure 16), as were the antagonist biceps onset times (see figure 20). Overall, equal distance bimanual movements were very similar to their unimanual counterparts with first agonist burst durations of around 75 ms and peak intensities of around 100% MVC for short movements (see figures 22, 27) and durations of around 100 ms and intensities of 125% MVC for long movements (see figures 23, 28). The movement patterns were more symmetric left and right for equal distance bimanual movements than for their unimanual counterparts with less variation in agonist premotor times (see figure 16), antagonist biceps onset times (see figure 20), time to peak acceleration (see figure 12), and time to peak velocity (see figure 7). Differences between left and right movements are more pronounced once movement has begun, with differences in the time to peak velocity (see figure 5), and overall movement distance (see figure 1).

Decreased variability between left and right hand EMG and kinematic variables during equal distance bimanual movements, and the lack of reaction time increase compared to equivalent unimanual movements, indicated that left and right EMG patterns were being synchronized within the nervous system. This would suggest evidence for a single neural

path of movement control (Anson and Bird, 1993). When two movements are performed simultaneously, there is the expectation of a decrease in performance in initiation (increased reaction time) and production (decreased peak force) known as the "bimanual deficit", due to each side of the brain inhibiting the other through the corpus callosum, or "transcallosal inhibition" (Ohtsuki, 1994). Most unimanual-bimanual experiments have found reaction time increases with bimanual movements compared to unimanual movements, but these differences vary by experiment. In their experiments Anson and Bird (1993) found a significant 10 ms increase in finger extension reaction times for equal distance bimanual movements compared to unimanual movements, but non-significant 6 ms increases for equal distance elbow flexion movements. For whole arm movements Marteniuk, et al., (1984) found a significant 12 ms increase in reaction time for equal distance bimanual movements while unequal distance bimanual movements had non-significant 6 ms increases.

In this experiment, the pairing of two equal distance movements did not result in increased reaction time or increased movement times compared to unimanual movements of the same distance (see figure 36). Indeed equal distance movements performed simultaneously were more symmetric than their unimanual counterparts, as reflected in dependent variables peak velocity (figure 7), peak acceleration (figure 15), triceps onset reaction time (figure 16), and biceps onset time (see figure 20). The increased synchrony of left and right hand EMG onset in bimanual movements indicated that equal distance bimanual movements were being planned and performed similarly to unimanual movements, as a single movement performed over two limbs (Al-Senawi & Cooke, 1985; Kelso, et al., 1983; Anson & Bird, 1993). This ability to combine movements appears to be limited to proximal muscle sets, such as that of the elbows, and is not available for more distal muscle

sets such fingers, where bilateral movements have increased reaction times compared to unimanual movements (Anson & Bird, 1993). The ability to plan a single movement and express it over two limbs has clear advantages for symmetric equal movements, however this also means that unequal asymmetric movements would be more difficult to perform. While equal distance bimanual movements would have a common temporal structure and equal EMG intensities, unequal movements require different EMG intensities, or 'metrical specifications' in order to generate different distance movements (Swinnen, et al., 1991).

The symmetry of EMG onset seen in equal distance bimanual movements is expressed before actual limb movement occurs, without any action there could not be any limb *interaction* creating the initial EMG symmetry. Interaction between the two limbs can only occur after movement has begun, and the effect of movement blocking is only seen in the affected limb after delays of 100-120 ms from first agonist onset. EMG patterns are therefore *influenced* by the type and nature of the intended movement, since EMG differences occur before any limb interaction is possible. The decision to perform an equal distance bimanual movement resulted in highly synchronized, symmetric EMG patterns for left and right arms.

The implications of a single neural path for bimanual movements during movement blocking was also clear; since a single movement pattern is being simultaneously performed over two limbs, there would not be any mechanism for limb interaction or coordination. Limb interaction is required to coordinate continuous bimanual movements such as gait, but there is no such necessity for discrete bimanual movements. Indeed, limb interaction could be detrimental if the two limbs interacted with positive feedback, an unstable increasing amplitude response to any perturbation (Kelso, et al., 1981). Blocking one limb of an equal

distance bimanual movement had no measurable effect on the unblocked limb for the 150-250 ms of movement. Blocked movements produced changes in same limb muscle EMG 100-120 ms after the onset of agonist activity which was composed of 50-60 ms of motor time, and 50-60 ms stretch reflex time. Voluntary responses to movement blocking have been measured at 200-260 ms in the contralateral arm, and 150-216 ms in the blocked arm (Latash and Gottlieb, 1991b). The blocked limb displayed the characteristic blocking effects seen in unimanual movements: shortened antagonist burst duration for short movements, reduced antagonist EMG burst and increased agonist activation for long distance movements (Wadman, et al., 1979 see appendix A For a more comprehensive discussion on the blocking paradigm).

Unequal Distance Bimanual Movements. Agonist EMG reaction times were longer for unequal bimanual movements compared to similar distance unimanual and equal distance bimanual movements (see figure 16). Both equal and unequal bimanual movements shared a single overall temporal pattern whose overall duration was determined by movement distance. In the case of unequal bimanual movements the overall pattern duration was determined by the increased time taken to generate the longer of the two movements, with the shorter movement's EMG amplitude reduced to produce a shorter movement (Latash and Gottlieb, 1991). Unequal bimanual movement EMG onset times displayed two differences compared to unimanual and equal bimanual movements, an overall reaction time increase of 10-20 ms for both hands, and longer times for the shorter movement EMG onset compared to longer movement EMG onset (see figure 16). The overall reaction time increase may be attributed to the increased complexity in planning and executing an unequal bimanual

movement, controlling not only the overall EMG timing, but also modulating EMG amplitude for each hand (Henry and Rogers, 1960). The 2-4 ms difference in short and long distance EMG onset times, and the fact that fast movement EMG onset occurs before short distance EMG onset, can be attributed to the reduced amplitude and reduced onset slope of the short movement's EMG (as measured by Q30). With reduced onset slope it took longer for short movement EMG bursts to rise to a threshold above the background EMG noise (see figure 16).

While longer distance 50 degree movements of unequal bimanual movements were performed correctly (see figure 1), their overall movement time was longer (see figure 31, 32) with reduced peak velocity (see figure 7), and longer times to peak velocity (see figure 5). The shorter 10 degree movement of unequal bimanual movements was not performed correctly, overshooting with average movement distances of 14.4 degrees compared to 12.8 degrees for unimanual and 12.3 degrees for equal distance bimanual movements (see figures 1, 2). The primary cause of these overshoots was a 'late braking' effect caused by an approximately 15 ms onset delay of the short movement agonist EMG compared to short unimanual or equal distance bimanual movements (see figure 25). This delay in antagonist onset must be centrally controlled, influenced by the type and distance of the intended movement, as modulation of antagonist EMG onset times has been seen in fully deafferented patients performing similar movements that generated triphasic EMG activity (Forget and Lamarre, 1987). However, correct modulation of antagonist EMG amplitude needed for accurate movement targeting requires intact peripheral feedback, as deafferented patients tended to over or undershoot more than normal participants with intact peripheral senses (Forget and Lamarre, 1987).

In order to simultaneously perform both a short and long distance movement participants were forced to find a compromise EMG temporal pattern. Since movements were being performed as fast as possible, at maximum EMG levels were used, only EMG onset and offset timing could be varied to vary movement distance. In the unequal distance condition the shorter movement EMG pattern was lengthened to make it similar to the long distance pattern, resulting in overall synchronization of both the agonist and antagonist EMG onsets (see figure 37). The overall consequence of this compromise strategy was slower movement velocities, longer movement times with good long movement accuracy, but consistent overshoots of the short movement (see figures 1 and 31). This temporal coordination of EMG patterns has been seen in discrete 3D bimanual movements in space (Keslo, Southard, and Goodman, 1979a, b), and in cyclical patterns of movement (Kelso, et al., 1981; Shik and Orlovskii, 1976).

Blocking of one limb of an unequal distance bimanual movement greatly affected the pattern of EMG of the blocked limb, yet had no measurable effect on the unblocked limb. Blocking the short movement of an unequal bimanual movement affected the blocked limb's pattern of EMG after 100-120 ms, resulting in reduced antagonist biceps EMG after onset, and increased agonist triceps EMG (see figure 29, compare 'Short Unequal Bimanual' & 'Same Block' data traces), the same pattern seen in blocked unimanual movements (Wadman, et al., 1979). Blocking of the long distance limb had no visible effect on short movement EMG (see figure 29, compare 'Short Unequal Bimanual' and 'Opposite Block' data). Blocking of the longer movement in an unequal distance bimanual movement resulted in extension of the first agonist triceps EMG burst and reduction in the onset amplitude of the

antagonist biceps EMG, while blocking the opposite limb performing the short movement had no visible effect on EMG production (see figure 30).

General Discussion

This thesis was designed to investigate the level and strength of limb interactions during bimanual movements. Unimanual, equal distance bimanual, and unequal distance bimanual movements of short and long distances were performed, while movement blocking was used to perturb limb movements and elicit responses to the effects of this "infinite mass" inertial load. Specifically several questions were addressed during this investigation.

Was movement symmetry seen in bimanual elbow extension movements?

All bimanual movements displayed symmetry, with the strongest symmetry seen in equal distance movements and weakest in unequal distance bimanual movements. Most of the kinematic event differences for the left and right hands were small, on the order of 2-15 ms over total movement times of 350 - 500 ms for equal and unequal distance movements. The only major differences found were with the longer distance of an unequal movement at the second zero crossing of acceleration (the velocity "end of movement") with differences of 130-140 ms (see figures 39 & 40). This difference was due to reduced negative acceleration for braking of the long distance movement resulting in a longer movement time.

Equal distance bimanual movements performed in this experiment displayed very strong symmetry, with triceps EMG onset time differences of less than one millisecond (see figure 17), movement onset time differences of less than four milliseconds (see figure 3), and total movement time differences of 11 milliseconds for short movements and five milliseconds for long movements (see figure 9). These results were comparable to the results

of three dimensional bimanual aiming movements reported by Kelso, et al. (1979) and Marteniuk, et al. (1984) who found that equal distance bimanual movements had very similar movement onset times (see figures 31, 34), movement times (see figures 32, 35), and total response times (see figures 33, 36). These two studies used tapping movements with physical targets as movement endpoints which participants would have naturally used to stop and stabilize their hands upon impact. These studies also found that when unequal distance movements were performed together, they were produced symmetrically with the short movement executed more slowly, at a reduced velocity, resulting in total movement times similar to that of the long distance movement. Short distance movement times were nearly doubled when a short movement was paired with a long movement (see figure 32).

However, the unequal movement conditions performed in the present experiment did not exhibit this effect for short distance movement times (compare figures 9 and 32). In this experiment short distance movement times were increased by only 9-24 ms compared to their unimanual and equal bimanual counterparts, while the longer movement times were 20-47 ms longer (see figure 9). The present experiment used points in space as movement targets, providing no physical barrier to impact at the end of movement. This protocol required participants to move a low inertia manipulandum using elbow extension movements, and the end of the movement was set by the experimenter as the first point when movement velocity dropped below eight degrees per second (close to peak displacement). In the Keslo, et al. (1979) and Marteniuk, et al. (1984) experiments participants appear to have synchronized the tactile end of movement, while in the present study movements were synchronized up to the point of peak negative acceleration.

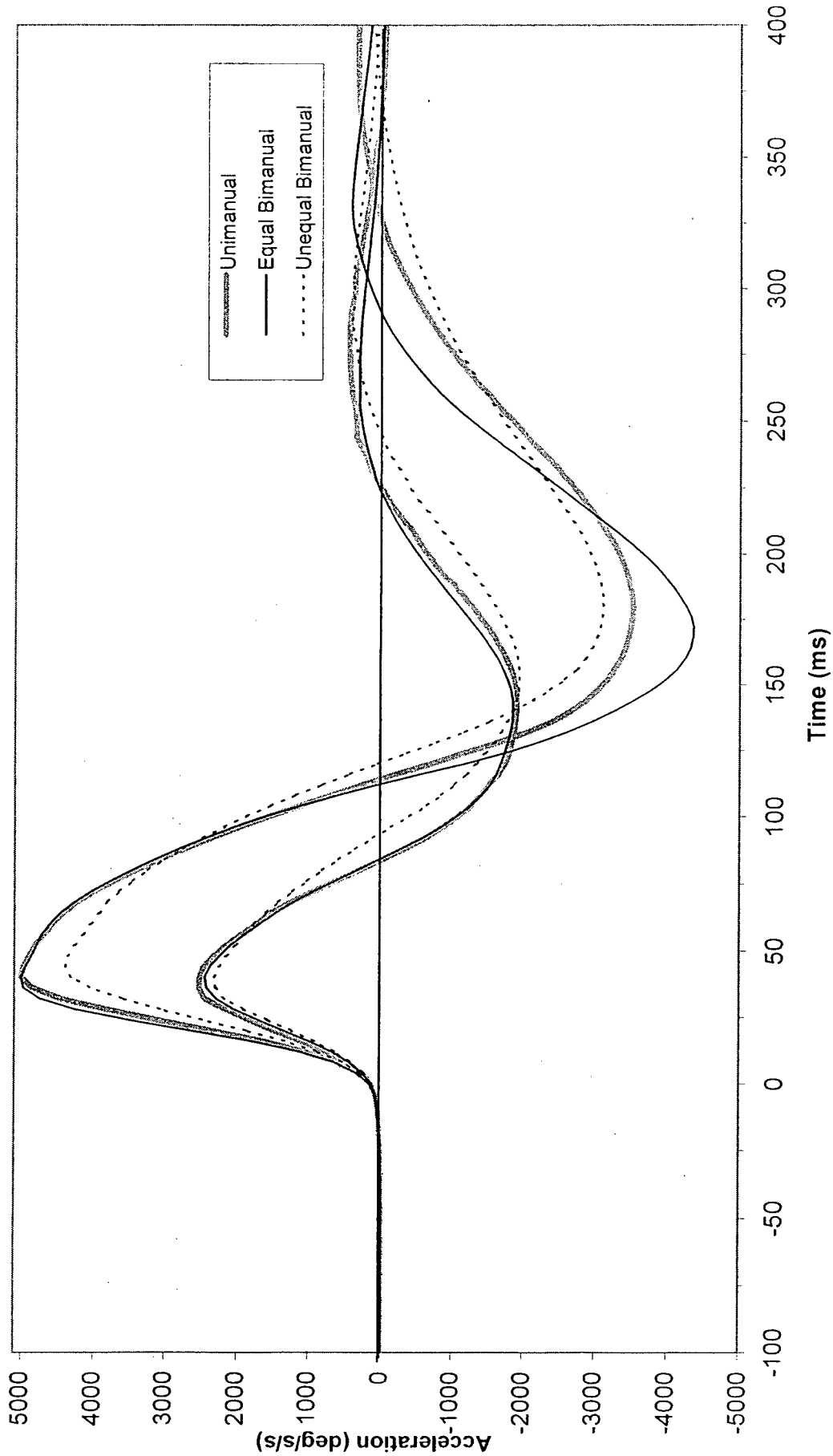


Figure 33. Acceleration Profile

Figure 34. RT and TRT: Kelso, Southard, and Goodman (1979)

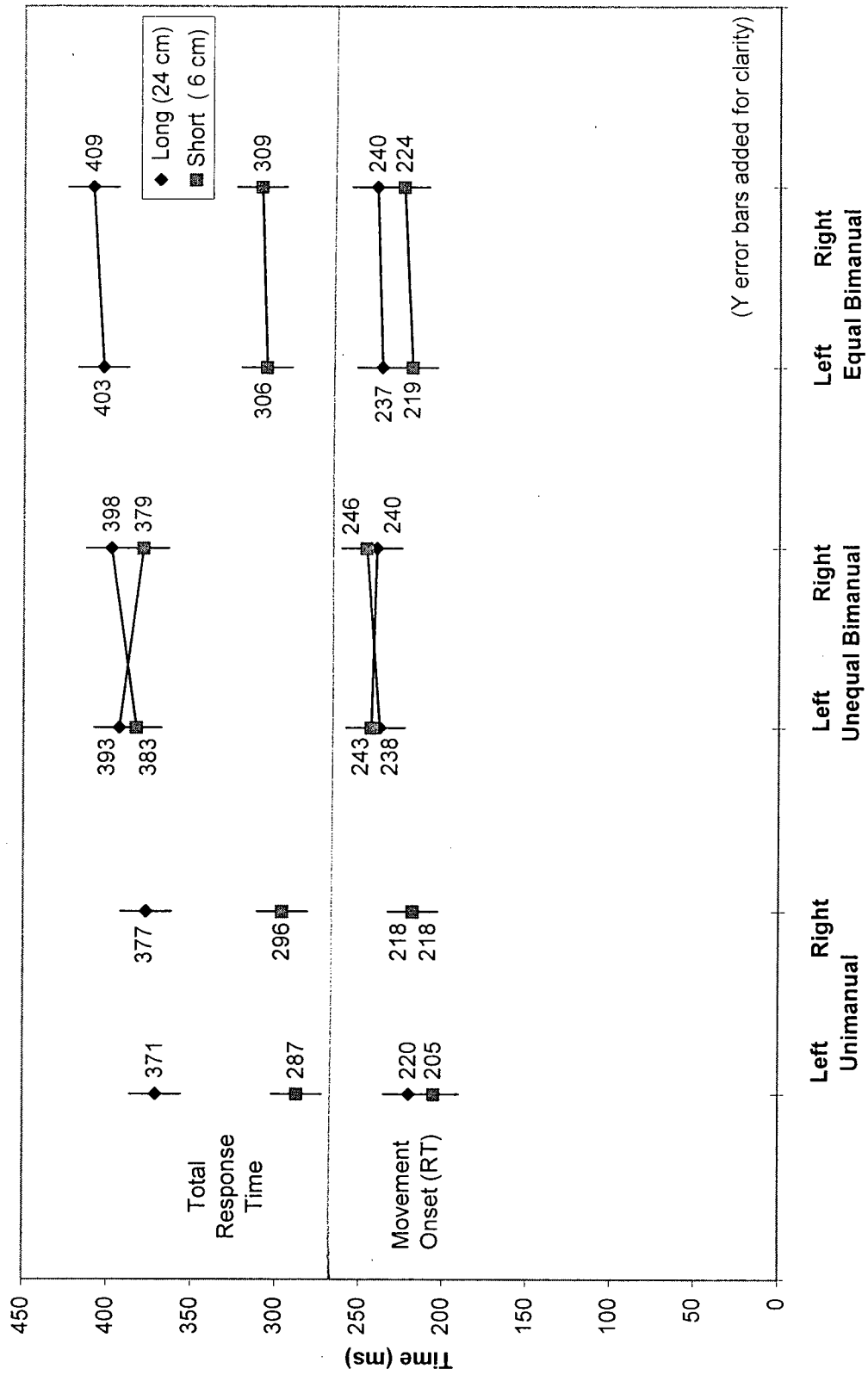


Figure 35. RT and TRT: Marteniuk, MacKenzie, and Baba (1984)

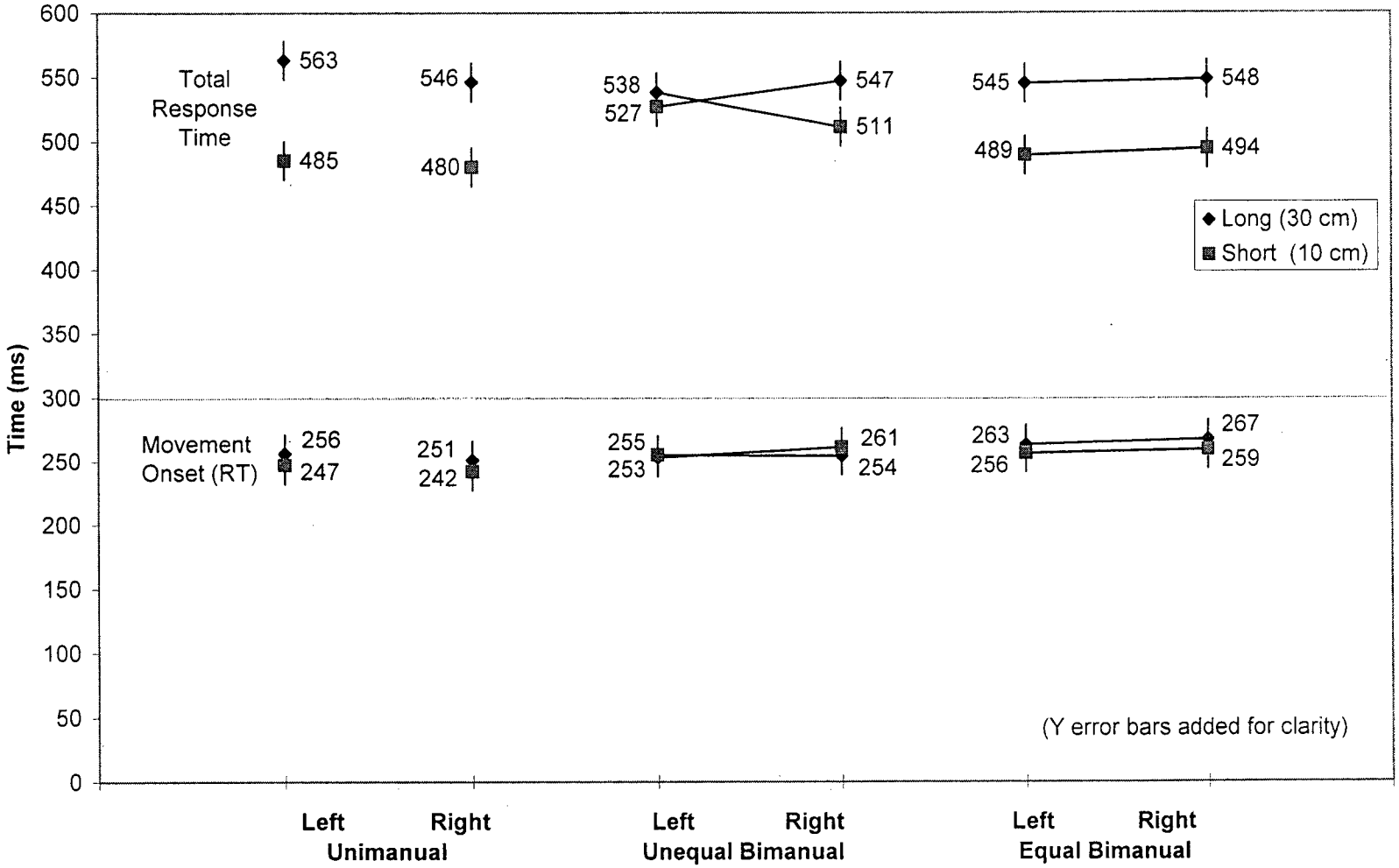
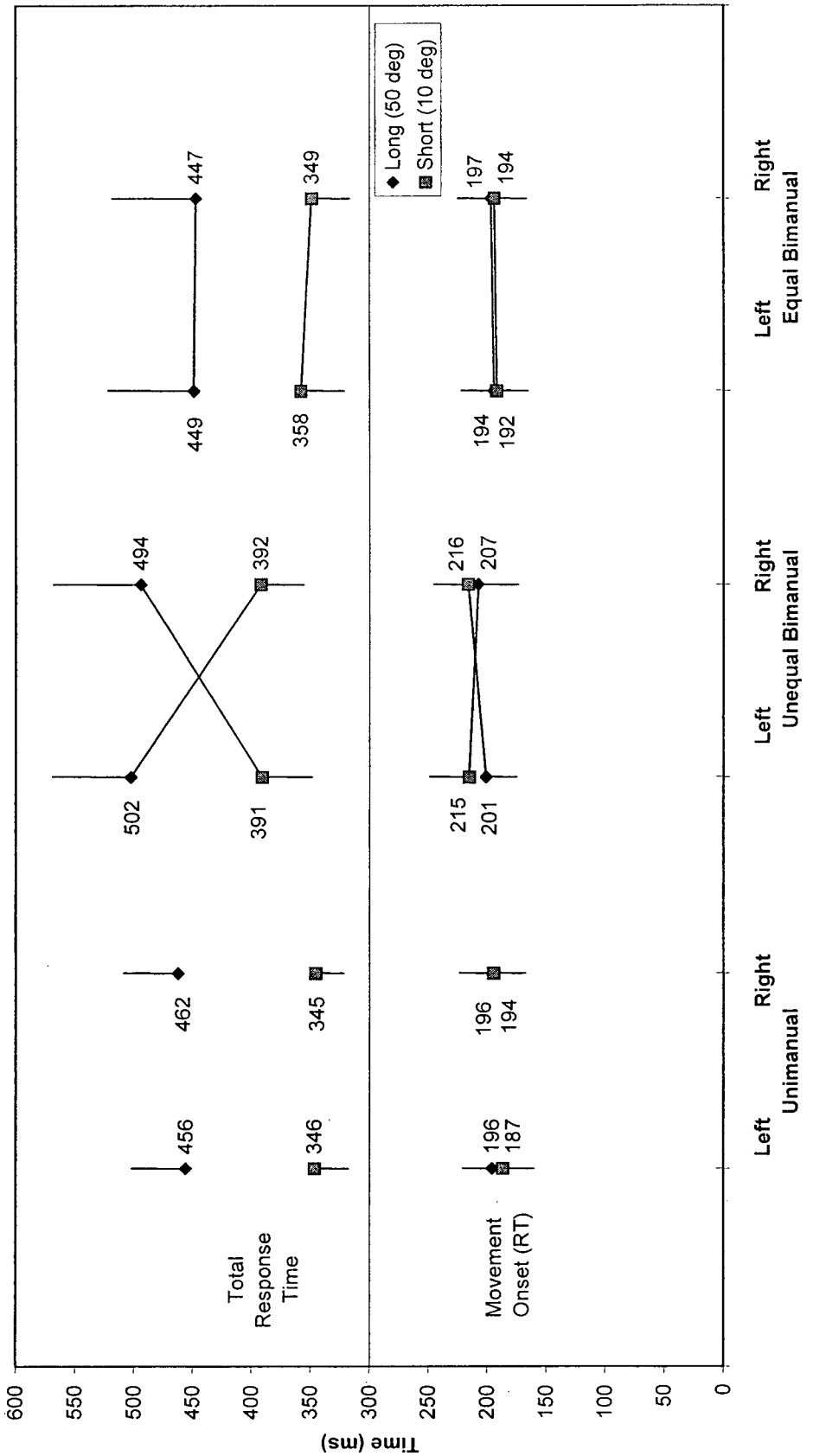


Figure 36: Experiment RT and TRT (Velocity onset to Offset)



Analysis of the unequal distance kinematics indicate that the times of acceleration onset, peak acceleration, zero acceleration, and time of peak negative acceleration were very highly correlated for all bimanual movements, whereas final acceleration offset (second zero crossing) was poorly correlated (see figure 37) with large differences (see figures 38, 39, 40). There were two possible reasons for the breakdown in correlation between peak negative velocity and acceleration offset, the first being later onset of antagonist EMG that resulted in short distance overshoot (see figure 20), and reduced peak negative acceleration in the long distance movement (see figure 33). The average difference between left and right hand event markers for acceleration onset and peak velocity were small, (0 to 15 ms), yet the average difference for acceleration offset was 133 ms for short left hand – long right hand movement and 140 ms for the long left hand – short right hand movement (see figure 38).

The second possible reason for poor correlation values at acceleration offset was that fast discrete point-to-point movements in space necessitate the generation of triphasic EMG activity to accelerate, decelerate, and brake the limb's movement (Berardelli, et al., 1984; Enoka, 1988) while discrete point-to-point movements into a physical barrier can be performed without triphasic EMG activity (specifically without antagonist muscle activity), depending on the participant's movement strategy (Waters & Strick, 1981). If movements were performed without antagonist activity for limb braking, relying instead on physical impact with the target to stop the limb, then the end-of-movement impact would occur at approximately peak velocity, a point of very high correlation in the kinematic patterns of this experiment.

Figure 37. Triceps and Biceps EMG Burst Onset Times

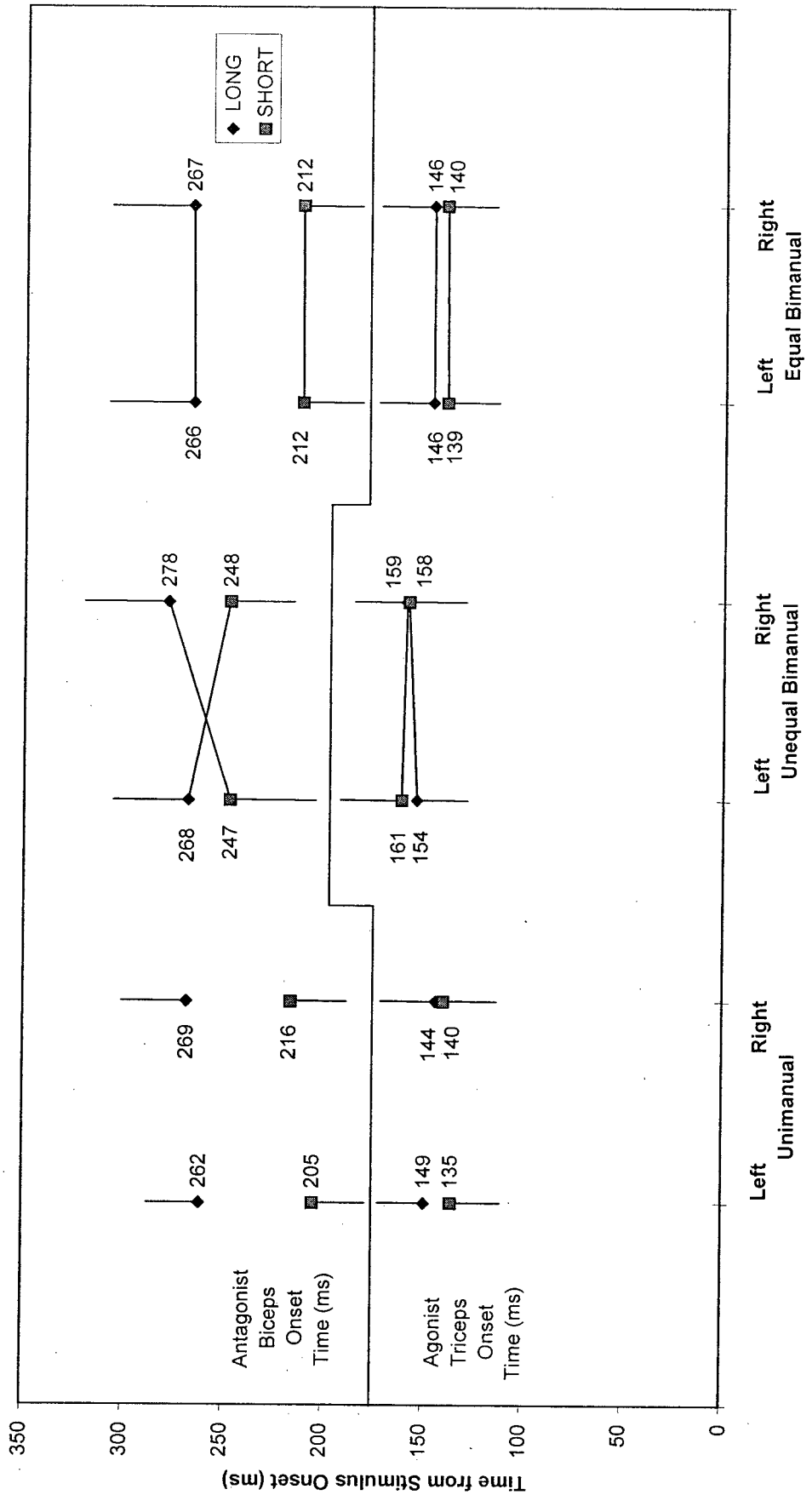
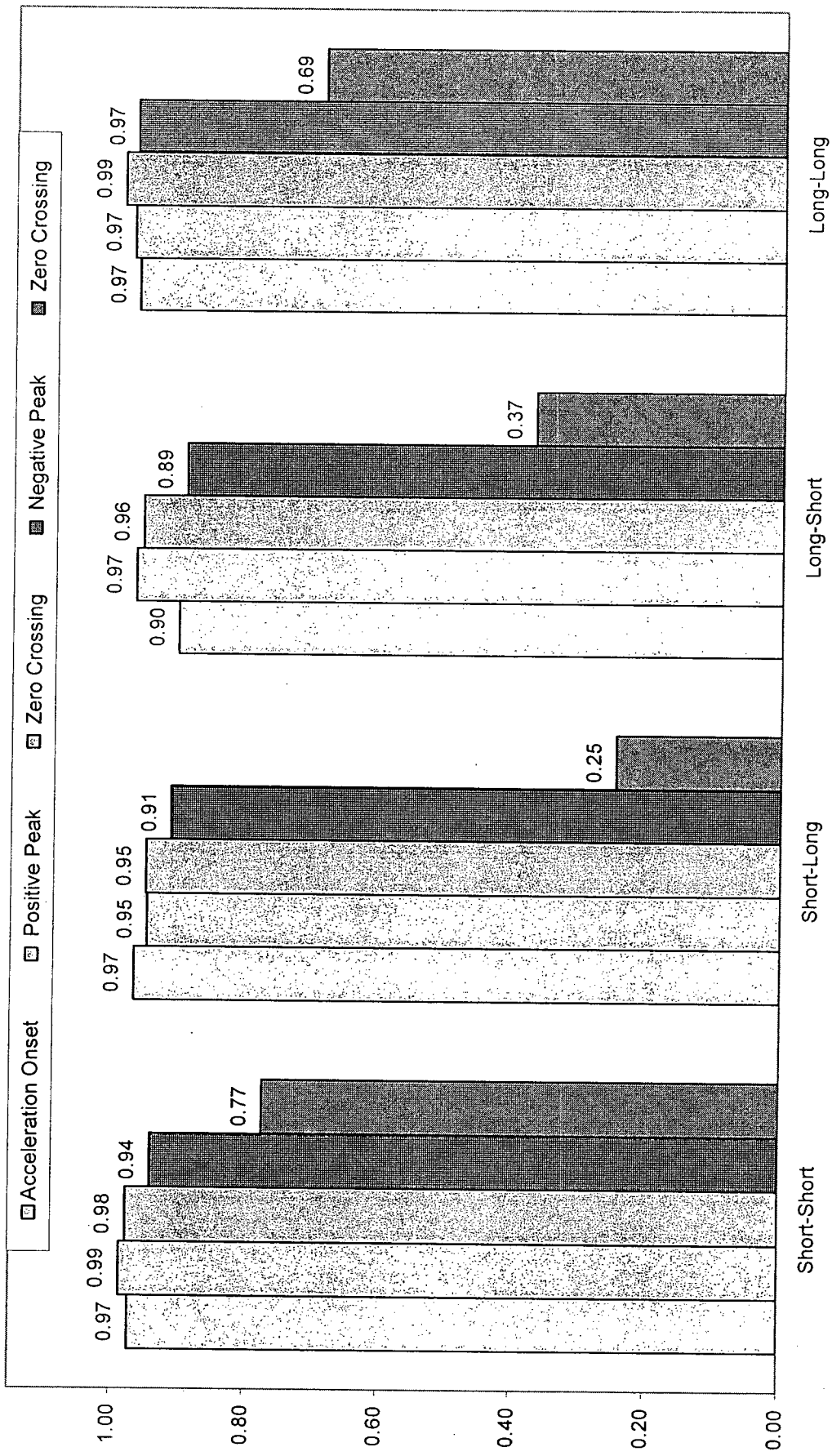


Figure 38. Acceleration Event Correlations



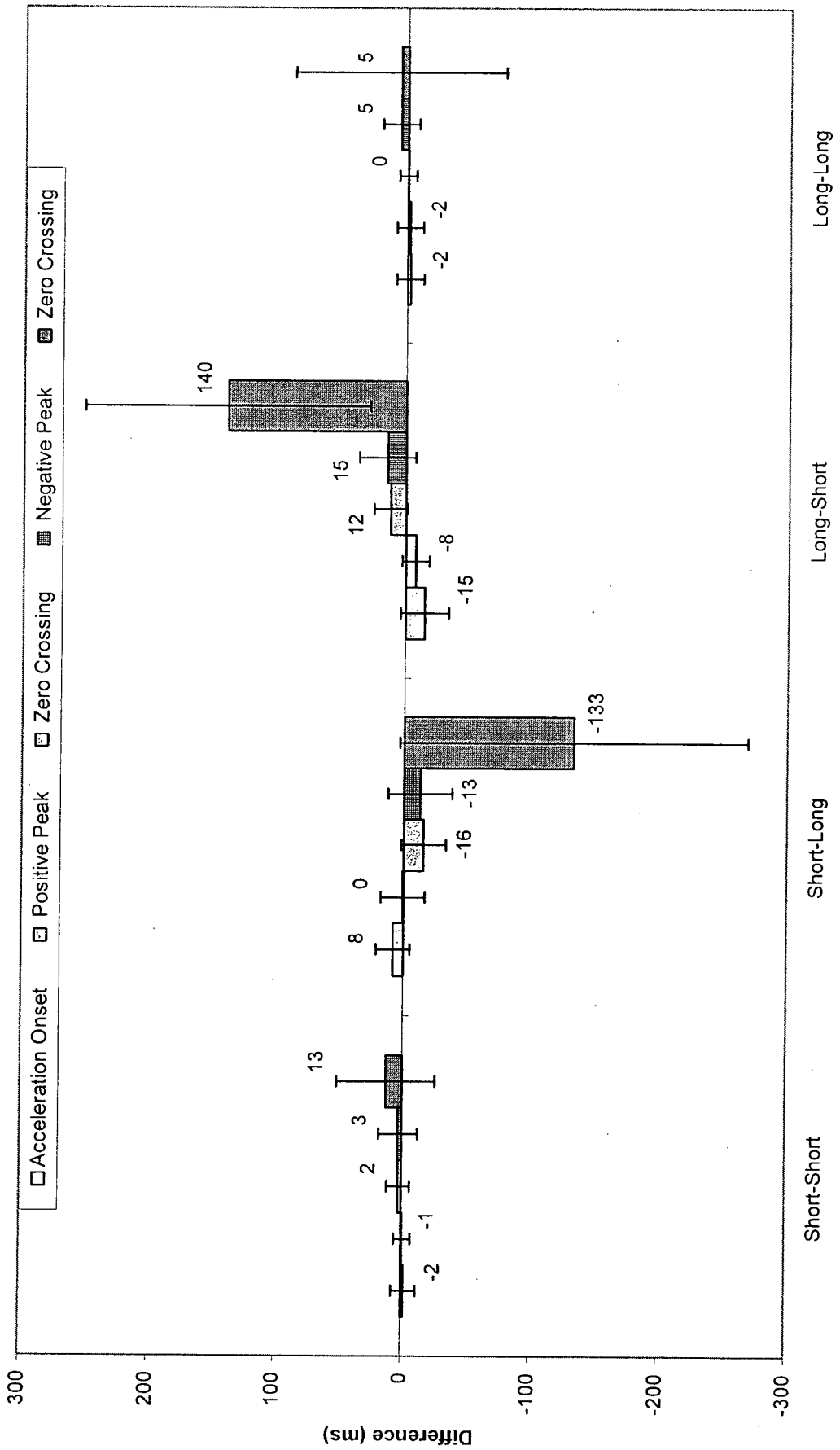
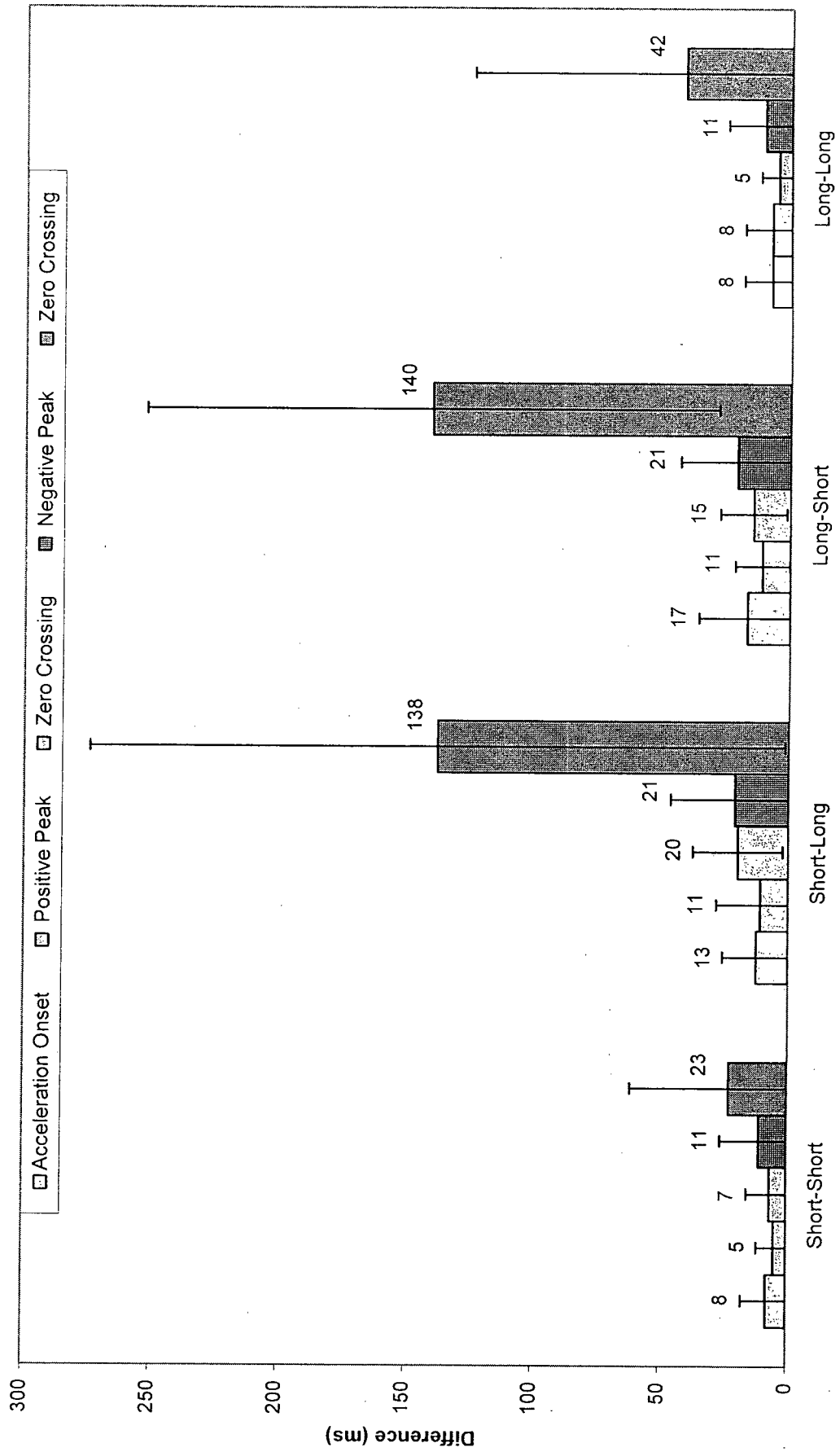


Figure 39. Acceleration Event Time Differences

Figure 40. Acceleration Event Time Absolute Differences



In this experiment unequal distance bimanual movements were performed with a high degree of temporal symmetry, requiring participants to modulate EMG duration in order to correctly perform the two different movements. Movement symmetry was very strong throughout the movement, with average differences of less than 16 ms for acceleration onset, time of peak acceleration, first zero line crossing, time of negative peak acceleration, but differences of 130-140 ms at the second acceleration zero line crossing (see figures 39, 40). The pairing of a short and long distance movement resulted in an overshoot for the short movement due to a longer movement time brought on by later onset of antagonist EMG, while the long distance movement was on target even though it had reduced peak acceleration and peak velocity values (see figures 31, 32, 33).

Comparisons of unequal distance bimanual movements showed high correlation of only qualitative (temporal-structural) characteristics, while comparison of equal distance bimanual movements showed high degrees of both qualitative (temporal) and quantitative (amplitude-magnitude) characteristics (see also Swinnen, Beirinckx, Meugens, and Walter, 1991). The qualitative or structural characteristics of the movements of the equal and unequal movements were very similar (time to peak acceleration, time peak velocity), since both movements were a single extension movement generated by a single sinusoidal cycle of acceleration created from a burst of triphasic EMG. The quantitative (metrical) characteristics of the equal distance bimanual movements were very similar, with nearly identical peak velocity and peak acceleration values (see figures 7, 14). The unequal distance bimanual movements had different quantitative values, as indicated by the significantly different peak acceleration and peak velocity values for short and long movements.

The high level of temporal symmetry seen in bimanual movements, and the high level of quantitative symmetry seen in equal distance bimanual movements but not in unequal distance bimanual movements indicated that both left and right elbow movements shared a common temporal pattern that governed the generation of EMG for both limbs. This temporal pattern resulted in very closely timed EMG onsets and offsets for bimanual movements, but caused short movement overshoots in the unequal distance bimanual condition.

Overall, bimanual movement symmetry was very strong, with EMG onset and offset differences of less than 13 ms, and left and right hand acceleration event differences of at most 13 ms over movement times of 150-250 ms for equal distance bimanual movements, but end of movement differences of around 140 ms for unequal distance bimanual movements. Both equal and unequal distance bimanual elbow extension movements displayed very strong overall symmetry, with major differences appearing only at the end of movement, the point where movement velocity drops below eight degrees per second.

Was there a difference in EMG activity of a blocked movement performed in a unimanual condition compared to the bimanual conditions?

Blocking one limb of a bimanual movement resulted in characteristic changes in EMG activity of the blocked limb similar to that seen in blocked unimanual movements. Movement blocking triggers the agonist muscle's stretch reflex in response to the increased muscle force produced when the muscle is prevented from shortening. The effect of the stretch reflex is typically seen 100-120 ms after the onset of EMG activity, due to an approximately 50-60 ms muscle motor time, and 50-60 ms reflex response time (Wadman, et

al., 1979). In short distance movements the triphasic pattern of EMG was almost completed by 100 ms, so movement blocking resulted in reduced antagonist EMG and an increased antagonist EMG (see figure 29). The effect of movement blocking appears constant, starting approximately 100-120 ms after the onset of agonist EMG, where antagonist EMG decreases and agonist EMG increases for another 100-200 ms (see figures 29, 30). The stretch reflex is an autogenic, monosynaptic reflex, innervating the same muscle group it via the muscle spindle and Golgi tendon organs through 1a afferent and alpha & gamma motoneurons.

The extent to which antagonist EMG was affected by movement blocking depended on the timing of the antagonist burst; short movement bursts were mostly completed around 100 ms, so they were not severely affected, while the long distance movement antagonist EMG burst started at approximately 100 ms, so it was more affected with reduced magnitude and duration (see figure 28). The patterns of agonist and antagonist EMG for each hand were influenced by movement distance and number of limbs performing the movement. Equal distance bimanual movement EMG patterns were nearly identical to their unimanual counterparts, so blocked movement EMG appeared the same for both conditions. Unequal distance movement EMG patterns were different from their unimanual counterparts, as the short distance's antagonist onset times were considerably longer than in the short unimanual condition, with reduced peak velocity and acceleration values. However these EMG patterns were influenced by the nature of the intended movement, while the effect of movement blocking was consistent for all intended movements after 100-120 ms. While there were differences in the EMG patterns of blocked unequal distance bimanual movements compared to their unimanual counterparts, these differences were due to the nature of movement

preprogramming, and the effect of movement blocking on these different EMG patterns, and not due to a changes in the nature of movement blocking.

What level of interaction was seen in bimanual movements when one arm was blocked?

Blocking or perturbing one limb during a bimanual movement has been shown to cause an interaction in continuous, cyclical extension flexion movements of left and right hand pointer fingers (Kelso, et al., 1981). The effect of this interaction was a small 10 degree sympathetic response of one finger to a 25 degree perturbation of the opposite finger during a 50 degree continuous extension-flexion movement. While no measure of response time was reported by Kelso, et al. (1981), the fastest reasonable response time would be a minimum of 50 ms for an active perturbation (stretch reflex plus some spinal conduction time), and minimum 100-120 ms for a passive perturbation such as movement blocking (muscle motor time plus stretch reflex latencies). Voluntary responses to movement blocking of an opposing limb have been measured at 200-260 ms for elbow flexion movements (Latash & Gottlieb, 1991b).

The use of passive movement blocking resulted in changes in the EMG patterns for the blocked limb 100-120 ms after onset of agonist EMG, due to the combination of muscle motor time and stretch reflex latencies (see figures 22, 23). Movement blocking resulted in early offset of the antagonist EMG burst for short movements and reduced EMG activity for long movements (see figures 22, 23). Analysis of the unblocked hand's antagonist biceps onset and offset times revealed no differences due to blocking of the opposing limb (see Appendix B). Additional analysis of movement time and movement distance of unblocked movements revealed no differences due to blocking of the opposing limb.

While the nature of the intended movement clearly influences the production of EMG patterns early in movement preparation and execution, as seen in agonist triceps EMG onset time differences for unequal bimanual movements (see figure 16), or antagonist biceps onset times in unequal short movements (see figure 25), there was no measurable interaction between the two limbs during movement blocking. With no clear interaction between the two limbs during a bimanual movement, what would be the shortest expected latency to a perturbation such as movement blocking? When instructed to, participants have responded to movement blocking by flexing the opposing limb, with reaction times of 200-260 ms, but these times could be decreased somewhat with a higher probability of movement perturbation trials (Latash & Gottlieb, 1991b).

While limb interaction has clearly been shown in continuous cyclical gait movements of spinalized cats (Shik & Orlovskii, 1976), and demonstrated in some continuous cyclical finger movements (Kelso, et al., 1981), the coordination and synchronization of EMG seen within this experiment's discrete, point-to-point, goal directed movements can not be due to an *interaction* if before any action has taken place. There must be a separate mechanism within the nervous system that allows the pre-planning of movements, where the nature of the intended movement (number of limbs, distances, speed, etc) *influences* the organization and expression of EMG patterns. This experiment has demonstrated that while there is a high level of symmetry in these discrete movement EMG patterns, this is due to the influence of the desired movement, and that there was no measurable interaction between the two limbs. Two limbs performing a discrete bimanual movement have synchronized EMG patterns generating movement force, but the limbs operate completely independently, perturbations of one limb had no affect on the other.

Was there a reaction time increase for bimanual movements compared to unimanual movements?

Generally, movement reaction times increase for one of two reasons; increased movement complexity, or increased movement accuracy constraints. A single movement response, an elbow extension movement for example, will have a shorter reaction time than a two part sequential ("serial") response of elbow extension-flexion, due to the increased time taken to preprogram the second movement (Henry and Rogers, 1960). However, reaction time will not increase if the first movement takes enough time that the second movement can be prepared "online" (during the execution of the movement). In addition two responses performed simultaneously ("parallel") have been shown to have increased reaction times only for more distal bimanual finger responses compared to more proximal bimanual elbow responses (Anson and Bird, 1953).

This experiment consisted of three movement conditions: unimanual, equal distance bimanual, and unequal distance bimanual movements over short and long distances for both the left and right hands, a total of four unimanual and four bimanual conditions. There were no reaction time differences between unimanual and equal distance bimanual movements, yet reaction times increased for unequal bimanual movements, although only significantly for the limb performing the shorter movement. The lack of difference between unimanual and equal distance bimanual movements compares favorably with Anson and Bird (1953), who concluded that a single neural path was available for both elbow unimanual and bimanual movements, but not available for the more distal bimanual finger movements.

Both the long and short movements of the unequal bimanual condition had increased reaction times compared to their unimanual and equal bimanual counterparts, but only the

short movement's 20 ms RT difference was significant (see figure 16). Only two factors can account for this RT increase, the bimanual movement itself, or the differences in movement distance. The fact that some bimanual movements can be initiated as quickly as unimanual movements (above) eliminates the bimanual nature of the movement as a factor. Since reaction times increase with increased target accuracy demands (Fitts, 1953), the RT difference may be due to perception of target size. Participants were given two types of visual feedback that expressed target error in absolute terms rather than relative terms. Visual feedback on the XY oscilloscope was in absolute units, with 10 cm indicating a 50 degree movement, 1 cm of screen position error represented an error of 50% for a 10 degree movement, but only a 10% error for a 50 degree movement.

Conclusions

This study investigated the interaction of two limbs performing elbow extension movements of 10 and 50 degrees, as unimanual, equal distance bimanual, and unequal distance bimanual movements. The expected increase in reaction time for bimanual movements was only found in unequal distance bimanual movements, there were no reaction time differences between equal distance bimanual and unimanual movements. Bimanual movements displayed less EMG and kinematic variability than unimanual movements, indicating the adoption of a single overall temporal pattern for EMG generation, resulting in high correlation for all acceleration profile events except for the end of movement indicator in unequal bimanual movements. Bimanual movements used a single common temporal pattern, unequal distance movements adopted the EMG temporal pattern of the longer movement, requiring modulation of short distance EMG amplitude to produce the short

movement, but the later antagonist onset resulted in target overshooting for short distance movements.

In all conditions movement blocking resulted in increased agonist and decreased antagonist activity for the blocked limb 100-125 ms after agonist onset through the action of the stretch reflex. Blocking of long distance movements resulted in reduction or complete suppression of antagonist EMG, while blocking of short distance movements reduced antagonist EMG after the burst had already peaked, except for the unequal movement condition which resulted in some reduction of the peak antagonist amplitude. Blocking of one limb had no measurable effect on the EMG or kinematics of the unblocked limb in any of the bimanual movement conditions in the interval before a possible voluntary response, indicating there was no limb interaction for discrete bimanual elbow movements.

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Appendix A: Literature Review

Movement EMG Patterns

Fast, voluntary point-to-point single joint movements are characterized by a stereotypical pattern of three (agonist, antagonist, and agonist) distinct muscle contractions. The electromyographic (EMG) patterns associated with these contractions consist of three overlapping bursts of activity (Angel 1977, Hallett 1979) followed by low level activation for an extended period of time while holding on the target. The first EMG burst shortens the agonist muscle, accelerating the limb towards the target, while the EMG burst in the lengthening antagonist muscle creates a negative acceleration force on the limb. A second agonist burst, coactive with the end of the antagonist burst, stabilizes the limb at the end of movement (Wadman et al, 1979). The intensity, duration, and onset time of each of the three bursts can yield various desired movement outcomes. Triphasic EMG patterns, or reciprocal innervation,

is associated with well-learned movements, it is energy efficient but requires accurate load anticipation to minimize movement error. When a movement is first learned, or the joint load is unknown, movement error can be minimized by co-contracting both the agonist and antagonist muscles, increasing joint stiffness and reducing the effect of load anticipation errors. During the development of motor skill we are able to more accurately anticipate movement loads, switching from a co-contraction strategy to reciprocal innervation (Ghez, 1991).

EMG can be generated by external perturbing forces, such as in the classic knee-jerk reflex, clinically used to test correct neurological functioning of lower limbs. Striking the

knee cap briefly stretches the knee extensor muscle, generating a signal that excites neural connections in the spinal cord, causing excitation of the main motor nerve controlling the knee, generating movement (Ghez, 1991). Neurological damage occasionally occurs which destroys this reflexive pathway, but still allows voluntary control of movement. In the study of one such patient, Lashley (1917), documented a patient with partial paralysis of his lower legs, no knee jerk reflex in the left knee, but still voluntary control of movement. After instruction he was able to accurately produce requested leg extension and flexion movements with no vision of his leg, with movement accuracy dependent only on the rate of movement, with greater accuracy for faster movements. Thus, accurate voluntary movements can be produced in the absence of reflex feedback influence on EMG generation. Indeed the role of feedback in skilled motor performance appears to diminish as a movement is learned, appearing to become an automatic "motor program" run off uninfluenced by peripheral feedback (Keele, 1968). However, this is not always the case, as movements learned under the influence of visual feedback are adversely affected by the removal of vision (Khan, et al., 2002), and peripheral feedback continues to be used at the neurological and reflex level to regulate movement and posture (Adamovich, et al., 1997).

The ability to perform accurate movements in the absence of neurological feedback, and conversely have dependence on feedback, indicate two neurological pathways for control of movements, one for fast-accurate movements, and one for the control of slower movements using stretch reflexes for muscle length regulation (Merton, 1953). Smooth movements were thought to be controlled by a stretch-reflex servo mechanism using gamma-muscle spindle activation to control main muscle length. This servo system operates as a negative feedback loop, increasing muscle activation in response to muscle stretch, and

decreased activation with muscle shortening. Stretching the muscle increases the spindle firing rate, these neural impulses are carried to the spinal cord via the Ia afferent, act directly on the alpha motor neuron, increasing muscle activation. Descending commands for normal smooth movements, acting on the small intrafusal (gamma) motor neuron, would control the tension within the muscle spindle, input sensor for the servo mechanism. Thus descending commands could control main muscle activation through the stretch-reflex mechanism. Descending commands for fast urgent movements were thought to directly influence the alpha motor neuron, bypassing propagation delays in the hypothesized servo mechanism. In order to study the response of the stretch reflex mechanism to large and small changes in muscle tension, Merton (1953) had participants perform a 1 kg isometric thumb flexion while receiving electrical stimulation along their ulnar nerve. Powerful stimulation caused muscle activation, shortening the muscle spindle, reducing spindle activation and hence reducing muscle activation. Stimulation just above motor nerve threshold still elicited a powerful corrective response to a relatively small increase in tension (3-4% of 1 kg background tension), indicating that small changes in muscle spindle length triggered large corrective muscle forces, a length-sensitive servo system. Gamma motor neurons innervate muscle spindles increasing or decreasing spindle tension, providing a control mechanism to adjust muscle spindle, and hence, main muscle length. Thus the two pathway system provides direct muscle activation via the alpha motor neuron for fast movements, and a feedback-based length-sensitive servo mechanism for slower movements via gamma motor neuron activation. In each case descending commands act either directly on the muscle resulting activation, or indirectly via tuning of length-sensitive servo mechanism based on the gamma reflex loop.

Movement without Neurological Feedback

Deafferentation Studies

Mammalian deafferentation, studying the characteristics of central control mechanisms in the absence of peripheral feedback, were performed by Bizzi et al (1979) Bizzi (1980) studying neck rotation of Macaque monkeys. These primates performed 30 to 40 degree head aiming movements in no vision conditions before and after deafferentation of Ia afferent fibers, severing reflex control. An opposing constant torque load was applied in five to ten percent of trials starting at the onset of measurable EMG, for a duration of 400-800 milliseconds. Head movements completed during the torque load would undershoot the target, while removal of opposing torque resulted in proper targeting. In intact animals, combined muscle and reflexive torques were not able to fully correct for the perturbation torque, measurements before and after deafferentation show that reflexes contributed only 10-30% of required corrective torque to perturbations. Added inertial loads slowed overall movement patterns, reducing movement velocity, extending overall movement time, but not perturbing movement endpoint. These experiments show that without additional feedback, a constant torque load perturbed intended movements, removal of the torque load allowed accurate target acquisition, with or without intact reflexes, while added inertial loads slow overall movement production without affecting movement endpoint. Agonist and antagonist muscles acting under central control, in the absence of reflexive feedback, acted as length-adjustable elastic components, working in concert to accurately rotate the monkey's head in response to a stimulus. Aiming movements were only slowed by added inertial loads, eventually resulting in accurate target acquisition, while offsetting torques prevented accurate target acquisition until the torques were removed. From the effects of inertial loads it can be

concluded that muscles are able to allow movement completion after an end-point has been set, and from the offset torque perturbation that muscles act as length-adjustable elastic components.

Further experiments were conducted by Polit and Bizzi (1979) to investigate the characteristics of forearm aiming movements in mammalian primates. Macaque monkeys performed no vision forearm aiming movements before and after dorsal rhizotomy preparation, which cuts the Ia afferents, eliminating stretch reflexes (along with all other sensory and reflex loops). Movements were performed with assisting and opposing torque loads, and increased inertial loads before and after preparation. Assisting torque loads forced aiming movements to overshoot the target, opposing torques forced target undershoot, while removal of reflex loops resulted in increased error. Added inertial loads slowed overall movement, reducing peak velocity and extending movement time, but not affecting overall target accuracy. An additional condition had the elbow shifted forward slightly, affecting the relationship between elbow angle and overall forearm-body angle. Intact monkeys were able to accurately compensate for this shift, but after preparation these same monkeys were able to accurately aim the elbow joint but not compensate for the positional shift. The effect of inertial and torque loads confirm that the muscles controlling the monkey's forearm function similarly to neck muscles, these muscles act as length-adjustable elastic components, and that sensory input for reflexes plays an integral role in movement error correction, and overall coordination of movement over many joints.

The ability of muscles to accurately control movement in the absence of neurological feedback gave rise to the Alpha model of movement control (Bizzi, 1980). As we have seen, muscles can act as adjustable elastic actuators, with their active lengths controlled by the

level of alpha (α) motoneuron activation, and coordinated interaction of agonist and antagonist muscles gives accurate limb control. In the absence of spinal reflex control through deafferentation, higher motor control centers are still able to generate muscle activation patterns that allow relatively accurate control of fast movements. Deafferented patients usually rely on vision to coordinate movement, without it limb coordination is severely handicapped, but with vision of the limb deafferented patients are able to perform accurate fine motor skill activities (Nougier, et al., 1996). The Alpha control model has some serious theoretical inconsistencies, contradictory predictions and discrepancies with observed behavior. Since muscle activation length is set by alpha motoneuron activation levels, muscles shorter than their activation length cannot be activated and muscles longer than their active length would always be active. Furthermore, according to the alpha model, for any given muscle activation level, muscles will contract more against a heavier load than a lighter load, meaning that a small perturbation would result in violent uncontrollable opposing movement, while light loads would be very difficult to move, and isotonic, no load movements, would be impossible (Latash, 1993, p. 25).

Feedback-Based Movement

The Role of Peripheral Feedback

The role of peripheral feedback in the generation or control of very fast or "ballistic" movements is unclear; if movements can be accurately performed without peripheral control, what role does feedback play? The influence of feedback is clearly seen in differences in the EMG of perturbed and unperturbed trials of human thumb flexion movements (Hallett and Marsden,1979). Integrated EMG patterns measured from thumb agonist and antagonist muscles were very highly correlated to distance traveled in unperturbed conditions. Agonist

EMG increased with opposing loads, indicating increased opposing force to the load, remained at an elevated level for constant opposing loads, and decreased when the opposing force load tapered off, indicating a servo-like response to increasing and decreasing loads. First agonist EMG onset and offset times were not affected by the perturbation, indicating that EMG amplitude increases and decreases were primarily due to muscle responses to the load, and not due to reflex action, but changes in load after the first agonist burst caused augmentation of the second agonist burst.

The antagonist EMG burst could be totally eliminated by a sufficiently large opposing torque, indicating peripheral feedback control (Feldman, Adamovich, & Levin, 1995), yet the burst was seen in patients with pan-sensory neuropathy, with poor timing, and amplitude coordination, indicating central control (Nougier, et al., 1996). From this we can conclude that the antagonist burst is a product of both peripheral feedback and central programming, with feedback playing the larger role. Also, overall temporal patterning of EMG was fixed by the central nervous system (CNS) while feedback could only partly adjust EMG amplitude in the first agonist burst, but could modify or even completely eliminate the antagonist EMG burst.

Aiming movements using the wrist, elbow or shoulder appear to use different underlying control strategies depending on the magnitude and speed of the intended movement. In studies of various sized movements of the human wrist and elbow Berardelli et al. (1984) found that EMG patterns fall into two distinct patterns depending on movement distance. Fast small-amplitude movements of 15-30 degrees had relatively constant first agonist burst durations of around 75 ms with variable EMG amplitude controlling movement range, while larger fast movements of 60-105 degrees had fixed (near maximal) amplitudes

and extended durations, and slow movements are composed of longer duration, low amplitude EMG bursts. Triphasic EMG activity therefore is not a fixed or stereotypical pattern, but variable to suit task demands, with a minimal first agonist duration of approximately 75 ms for even the smallest movements.

One method of classifying movement EMG patterns was indirectly proposed by Latash & Gottlieb (1991a), based on the factors underlying movement (equilibrium point shift speed), but now generally used to describe measured EMG patterns. Slow movements generally have a fixed duration, less than maximal amplitude EMG pattern, this pattern was dubbed the "Speed Sensitive Strategy", while fast movements generally have variable duration, near maximal amplitude EMG bursts, known as the "Speed Insensitive Strategy". These terms were originally defined in terms of the speed of change of agonist and antagonist muscle activation levels, the limb's equilibrium point (EP), with speed sensitive movements being driven by less than maximal speed shifts in the EP, and speed insensitive movements being driven by maximal changes in equilibrium point shifts. This original definition starts to break down with very short movement distances, where movements driven by maximal speed equilibrium point shifts (speed *insensitive*) are forced exhibit less than maximal EMG (speed *sensitive*) due to minimal EMG burst duration (Berardelli, 1984). Since the speed of equilibrium point shift can only be inferred indirectly, this classification system is generally only used to describe the pattern of recorded EMG, with speed sensitive EMG patterns characterized by less than maximal EMG patterns of fixed duration, and speed insensitive EMG patterns characterized by near maximal EMG activation patterns of variable duration. The selection of EMG patterns for movement is variable to cope with muscle fatigue, or unexpected loads that can extend a first agonist burst by up to 25 ms (Berardelli, et al., 1984).

Larger limb movement is usually studied in an "isotonic", or a zero external force (especially gravity) condition, horizontal joint rotation movements of the wrist (Lee et al., 1986), elbow (Brown and Cooke, 1981), or shoulder (Angel, 1977), or shoulder and elbow combined (Wadman et al., 1979, Smeets et al, 1990, Gomi and Kawato, 1996, Ghafouri & Feldman, 2001). Horizontal elbow extension-flexion movements are much less affected by the natural dampening properties of muscle and connective tissue than a much lower mass appendage such as the thumb, forcing participants to actively use their muscles to accurately start and stop each movement. The pattern of agonist and antagonist EMG for elbow movements shows characteristic triphasic activity, even when movement was mechanically blocked, preventing movement (Wadman, et al., 1979). Linear wrist movements of 7.5, 15, 22.5 and 30 cm resulted from elbow and shoulder movements with upper arm triceps and biceps muscles acting as the elbow agonists and antagonists. EMG durations increased with longer distance movements while overall amplitude remained constant, the relative pattern of agonist-antagonist-agonist activity remained constant for all unblocked movements. Increased inertial loads resulted in longer overall movement times and longer EMG patterns, while decreased loads resulted in reduced duration EMG patterns and faster movements. When movement was completely prevented in the movement blocking condition, overall EMG patterns were unchanged for the first 100 ms from agonist onset. Very short 7.5 cm blocked movement EMG appear identical to unblocked EMG over a period of over 300 ms (see figure 9, page 10, Wadman, et al., 1979). In blocked movements the basic triphasic pattern appears, but with the antagonist burst peak slightly reduced at 100 ms after agonist onset, and the second agonist burst appearing approximately 25 ms early. These results lead Wadman et al to suggest that muscle activation patterns were pre-set by the central nervous

system and "run off" without the need for feedback control. Longer amplitude movements of 15 and 22.5 cm were more attenuated after movements were blocked, due to a reduction in antagonist burst amplitude and duration, and also a reduced second agonist burst amplitude. Blocked movements of 30 cm had markedly different EMG patterns, with continuous agonist activation and no antagonist activation. Antagonist activity, which starts at around 100 ms in unblocked 30 cm movements, is completely suppressed in blocked movements while agonist activity is sustained. From the invariance of first agonist onset, Wadman et al. concluded that movement planning follows two steps, first selection of muscle activation level, and secondly burst durations that together would generate the correct limb movement. Peripheral feedback did not appear to influence production of the first agonist burst, and only partly involved in control of the antagonist burst, totally eliminating it in longer blocked movements.

In order to investigate the role of peripheral feedback in agonist and antagonist EMG burst production, movement perturbations were introduced around the onset of voluntary movement in a series of stepped tracking trials (Brown and Cooke, 1981). Participants performed 48 degree horizontal isotonic elbow extension-flexion movements with an assisting or opposing torque of 3-5 Nm applied after the presentation of the new target on some trials. The 50 ms duration torque load was timed after stimulus presentation to appear 30-120 ms before voluntary movement onset. As in the Wadman et al 1979 experiments, the first agonist EMG burst was not initially affected by the perturbations; 100 ms after onset EMG increased in opposed load conditions, and decreased in assisting load conditions. Opposing load perturbations applied 35-75 ms before agonist onset resulted in increased EMG activity only 100 ms after onset, while assisting loads applied up to 110 ms before

agonist onset resulted in decreased EMG activity only 100 ms after onset, a total latency of 220 ms. Perturbations introduced elsewhere within the movement profile elicited stretch reflex responses with normal 50-60 ms latency times, indicating that peripheral feedback was not being used around the time of the first agonist EMG burst, and the perturbation response was being delayed by another mechanism. This delay could be "due to a gating of peripheral input during a preparatory period for movement." (p. 354), produced by the participant's task instructions to "ignore the perturbation as much as possible and concentrate only on performing the movement." (p. 355). Thus while production of the first agonist EMG burst appears to be unaffected by peripheral feedback, the effect of short duration perturbations completed before EMG initiation are still seen, but only 100 ms after EMG onset.

The long 200 ms response delays found by Brown and Cooke (1981) are normally attributed to long-loop reflexes, while shorter delays of 30-60 ms are attributed to spinal stretch reflexes. Movement blocking elicited differences in EMG activity 100 ms after agonist onset, or approximately 50 ms after physical contact with the barrier (using an average 50 ms motor time) indicating that a stretch reflex was responsible for EMG modification (Wadman et al., 1979). However, the statement that "...muscle activation patterns are preset over this [100 ms] period and are not immediately modified by proprioceptive information." (Wadman et al, 1979), caused confusion, appearing to indicate that the first agonist burst, of less than 100 ms duration, was completely generated by central commands, in the absence of peripheral commands. In order to see if early perturbations could influence first agonist EMG Lee, et al., (1996) performed perturbation experiments using fast, 100 ms - 40 degree wrist flexion movements against a standard opposing load. Unexpected increases in opposing loads resulted in decreased movement velocity, target

undershoot, decreased antagonist EMG, and an additional agonist burst after the first agonist burst. Decreased opposing loads resulted in increased movement velocity, target overshoot, increased antagonist EMG, and suppression of the additional agonist burst. Compensatory increases in agonist EMG activity occurred 30 ms after onset of movement, indicating spinal level mechanisms for correction, rather than long-loop reflexes. The long delays of the Brown and Cook (1981) studies indicate that participants were able to suppress peripheral feedback from immediately affecting movement production via stretch reflexes prior to first agonist activity, and use long loop reflexes to respond to perturbations.

Modifications in triphasic EMG due to movement perturbations are seen only 100 ms after the onset of the first agonist burst, with the antagonist and second agonist bursts greatly affected by peripheral feedback. As seen in Lashley (1917), deafferented patients were able to consistently produce accurate movements due in part to the agonist EMG burst initiating movement. The role of peripheral feedback in the production and control of fast goal directed elbow movements was investigated by Forget & Lamarre (1987), where three deafferented patients and ten normal control participants performed 40 and 90 degree flexion movements to a 10 degree wide target (centered at 115 degrees of elbow flexion), with instructions to "rapidly and accurately move from resting position to target zone". All participants and patients produced triphasic EMG activity, with 90% target accuracy for normals, and 50% accuracy for patients, due to small, poorly coordinated antagonist bursts. Decelerating bursts for deafferented patients were not properly scaled for movement magnitude, and incorrectly timed for limb position. Normal participants properly scaled the magnitude of the antagonist burst and timed antagonist EMG onset at a fixed interval with respect to limb peak velocity. Antagonist EMG burst timing is also not affected by the

influence of transcranial magnetic stimulation of the motor cortex, which affects first and second agonist EMG amplitudes and onsets, indicating a different, possibly subcortical, control mechanism (MacKinnon & Rothwell, 2000). Thus it appears that afferent feedback plays an important role in the amplitude and timing of the antagonist EMG burst.

Position Reference in Movement

While it is clear that feedback plays an important role in movement EMG timing, it is not clear what type of feedback is used to correct movements. In order to discover to nature of corrective feedback, Smeets et al (1990) studied the effect of changing mass on a linear pulling motion and associated delays in EMG correction. A torque motor simulated 0.7, 5.0 (normal), and 20 kg loads being pulled horizontally 8 or 16 cm towards participants, with 20% probability of an increasing or decreasing load. Modified inertial loads did not affect EMG production until 90-110 ms after agonist onset (65-85 ms after the start of movement), increasing loads resulted in longer agonist burst duration (delayed offset), and delayed antagonist onset, while decreased loads resulted in shorter agonist burst duration (earlier offset) and earlier antagonist onset. Modifications in EMG were triggered when either force, displacement or velocity of the limb did not match the profile of the desired movement. Position differences of 0.6 cm can be seen approximately 25 ms before EMG modification, but thresholds of this magnitude are difficult to perceive, and the 25 ms transport time is not adequate time for typical feedback loops. Joint velocity differences of 0.6 radians/second (34 degrees/second) can be seen approximately 37 ms before EMG modification, a readily perceived threshold and sufficiently long feedback loop time. Force profiles showed no clear separation between normal and perturbed trials before EMG modification, indicating that force differences could not have triggered the changes in EMG. Changes in movement

velocity can be readily detected either in the shortening agonist muscle, or lengthening antagonist muscle. In the shortening agonist muscle an α-γ co-activation mechanism, with γ-activation corresponding to movement velocity and α-activation corresponding to movement force, would adequately control both force and velocity of movement. Perturbation induced differences between the desired γ-activation movement velocity and actual limb velocity would result in EMG modification through stretch reflexes. One intrinsic property of velocity control of movement is the limb's displacement over time, the duration of movement from a given start position determines the limb's final position.

The Feldman Two Component EP Model

The combination of velocity and position control of movement gives rise to an internal reference of desired limb position over time, encapsulated in the Lambda (λ), or equilibrium point model of muscle control (Feldman, 1986; Feldman & Levin, 1995). The Lambda model states that descending motor commands define a threshold value for the tonic stretch reflex, thus defining fixed force-length characteristics for the muscle. Complementary pairs of muscles acting across a joint would be separately affected by two descending commands, a reciprocal (R) command that increases activation in one muscle while decreasing activation in the other, and the complementary (C) command that increases or decreases activation in both muscles simultaneously. Limb movements would be performed by selecting appropriate C and R commands that cause a force-torque imbalance across the active joint, causing limb movement to attain a new position of equilibrium where movement ends. The R command controls equilibrium point position, and consequently limb position, while the C command controlling muscle activation levels leads to fast or slow movements. The C command, controlling complementary muscle activation and static

tension, also controls muscle stiffness and subsequently joint stiffness, with increased stiffness at higher complementary muscle activation. Changes in the equilibrium point (R command) are monotonic; single speed, continuously increasing in value, reflecting the desired position of the limb. Slow changes in the EP reflect slow changes in limb position, while fast limb movements are driven by fast changes in EP, to a maximum of 600 degrees per second (Feldman, Adamovich, and Levin, 1995), thus EP shifts are completed at about peak movement velocity.

One consequence of the equilibrium point model is easy adaptation to different assisting or opposing loads by corrective shifts in the joint's equilibrium point. A series of trials were run with differing assisting or opposing loads for a random number of trials to study one trial learning for force generation (Weeks, et al., 1996). Opposing loads resulted in target undershoot, assisting loads resulted in overshoots, in 94% of first trials with new force, with only 37% of following trials having targeting errors. Participants were able to correct their response upon presentation of a new perturbing force, accurately presenting the new corrective force in succeeding trials, confirming one trial learning of corrective forces. Participants adopted different muscle force-length equilibrium points for each perturbing force, generating the appropriate corrective force in successive trials. When presented with a new perturbing force, participants would initially generate the previous force, resulting in target error, then adjust their response with a new corrective force. Participants adopted different equilibrium points along similar force-length curves in response to different perturbing forces, rather than adopt different force-length curves by also changing overall limb stiffness, for example. This is interpreted within the Lambda model as selecting different R values, and leaving C constant, for each new perturbing force presented.

The Feldman Three Component EP Model

The two component lambda model, using R and C commands, is adequate for modeling the start of fast movements, but runs into problems modeling the end of movement. The level of muscle co-contraction required to stop very fast movements results in very high muscle tension, which can lead to limb oscillations at the end of movement. An additional component is required to adjust the dampening qualities of the muscles involved in movement in order to address the problem of limb oscillation. St-Onge, Adamovich, and Feldman (1997) performed a series of experiments, including movement blocking, to compare empirical and model data for a three component lambda model, using, R- the reciprocal command, C-coactivation command, and μ-time-dimensional variable influencing the dependency of the threshold of the stretch reflex on movement velocity. The μ-time constant variable specifies a time constant characterizing the activation of dynamic γ- and β-motoneurons, providing end of movement limb oscillation dampening. Experimental results matched model predictions of a monotonic ramp-shaped pattern changes in the R command, where shift rates are dependent on movement speed, and movement distance is encoded by the duration of equilibrium shift. Strong perturbations may result in substantial differences in kinematic and EMG patterns despite similar control patterns for an intended movement, control patterns are not influenced by peripheral feedback, but feedback will be involved in the creation of a new central command.

One method to determine the magnitude of both the equilibrium point and complementary command during a limb movement is to measure overall limb stiffness in both directions, then calculate individual joint stiffness values. Slight force perturbations

were applied at various points through a horizontal right hand aiming movement by a parallel link arm powered by torque motors to measure limb stiffness (Gomi & Kawato, 1996). After resolving overall limb stiffness into individual joint equilibrium points, it was found that the equilibrium point for movement varied continuously over the time of movement. At the beginning of movement the equilibrium point moved past the intended target in order to increase initial acceleration, then started back towards the starting point to decrease movement speed, then returned to intended end point overshooting and undershooting in order to dampen out the end of the movement. The equilibrium point was calculated to be continuously changing well past the end of physical movement, acting as a classic "inverted pendulum" under-dampened dynamic system, with the necessity of feedback to create equilibrium point shifts to dampen the smallest movements. This contradicts the underlying concepts of the hypothesis that the equilibrium point is set without the necessity of feedback, and that physical movement lag behind any changes in the equilibrium point. One possible cause of the contradictory equilibrium point results was an incorrect assumption of the non-linear translation of perturbation magnitude to limb stiffness (as discussed by Ghafouri & Feldman, 2001).

The Latash & Gottlieb EP Model

The underlying concept of the equilibrium point (EP) hypothesis, that of a single control parameter for equilibrium position, and hence intended limb position, explains all but a few points about limb joint control. In the case of small amplitude, fast goal-directed limb movements characterized by triphasic EMG activity, the appearance of the antagonist EMG burst poses a problem, as it appears while the limb is still accelerating towards the intended target position. The appearance of triphasic EMG in movement blocking conditions, where

no physical movement occurs, appears to contradict the notion of the Equilibrium Point model, that EMG patterns are sensory based. If no movement is permitted, then sensory input would not trigger the antagonist EMG burst. From this problem Latash & Gottlieb (1991b) hypothesized that a single central relative position command must yield two different muscle threshold signals controlling both the agonist and antagonist muscles. This version of the Equilibrium Point Model (Latash & Gottlieb) was used to examine isometric conditions, treated as "heavily loaded, short distance isotonic movements" (p. 179). A single central command controls both agonist and antagonist lambdas, with two different time profiles: the agonist lambda monotonically increases creating the agonist EMG burst while the antagonist lambda first monotonically increases, then decreases, triggering the antagonist EMG burst, then increases again to the final antagonist endpoint (an "N" shaped profile). Thus antagonist EMG is triggered in both unblocked and blocked conditions, with or without the influence of feedback. The original Feldman (1986) Lambda model relies on movement induced changes in the antagonist muscle length to trigger the antagonist EMG burst, which would not occur in isometric movements. Even deafferented patients had an obvious, yet reduced amplitude, antagonist burst, a possible sign of "absent reflex actions while the patients still used the same central programs" (Forget & Lamarre, p. 188). The dual strategy hypothesis answers some questions about movement control, but still leaves some questions about the nature of the antagonist Lambda control mechanism, and the role of feedback in both agonist and antagonist EMG generation.

Muscle compliance properties, as well as length-sensitive stretch reflexes, contribute to muscle EMG changes when movements are perturbed, but the relative contribution of each is not clear. Application of an unexpected perturbing force results in immediate changes in

muscle force due to muscle length-tension and force-length properties, while reflexive changes in EMG are usually seen within 100 ms (Latash and Gottlieb, 1991b). The exception to this is perturbations applied around the first agonist burst, where reflexive changes in EMG are only seen approximately 100 ms after the first agonist onset (Wadman, 1979). With expected opposing elastic loads, EMG differences occur approximately 125 ms before measurable differences in muscle torque, but with unexpected loads EMG differences appear later, approximately 30 ms before muscle torque differences appear (Gottlieb, 1994). Differences in muscle torque appear approximately 100 ms after movement onset in both expected and unexpected load conditions. For inertial loads the difference is more dramatic, muscle torque differences appear just after movement onset, expected inertial load EMG differences appear approximately 50 ms before movement onset, while EMG differences appear 250 ms *after* movement onset for unexpected inertial loads. Elastic loads produced a reciprocal effect in muscles, increasing agonist activity and decreasing antagonist activity, expectation of the load only reduced and delayed this effect. With prior knowledge of increased inertial loads participants increased both extensor and flexor EMG, with unexpected loads participants produced the opposite, creating greater EMG with decreased inertial loads. Unexpected changes in either elastic or inertial load produce greater trajectory effects due to the reduced amplitude and later onset of corrective EMG patterns. Muscle-joint compliant properties appear to account for most of the minimizing effects load changes, and "that there is no evidence for a large contribution by length-sensitive stretch reflexes to this process." (p. 545). While stretch reflexes may not appear to contribute much to overall muscle force generation, they do play a role in adaptation to movement load.

Tests of Feldman and Latash & Gottlieb Models

Different patterns of antagonist EMG within a triphasic EMG pattern are predicted for the Feldman and Latash & Gottlieb versions of the Equilibrium Point model under conditions of movement with decreased inertial loads. The Feldman EP model predicts early antagonist onset with decreased inertial load, due to the antagonist muscle reaching its threshold activation length due to increased movement stretching the muscle, while the Latash & Gottlieb model predicts no change in antagonist onset (Latash, 1994). A torque motor was used to simulate the reduction of a manipulandum's moment of inertia by approximately 40%, known as an "unloaded" trial, in order to address these predictions (Latash, 1994). The first agonist duration was significantly shorter for expectedly unloaded trials compared to normal or unexpectedly unloaded trials, further reinforcing the concept that the first agonist burst is centrally planned. Antagonist onset time was also reduced for expectedly unloaded trials, and also unchanged for normal and unexpectedly unloaded trials, confirming that antagonist onset is centrally planned. But agonist activity is also subject to feedback modification, large amplitude movements of more than 45-60 degrees display no antagonist activity when they are mechanically blocked (Wadman, et al. 1979). The integral of the first agonist EMG burst decreased non-significantly, while the antagonist EMG integral decreased significantly, in unexpectedly unloaded trials compared to normal trials. EMG patterns for known unloading trials were different than normal or unexpectedly unloaded trials, indicating central control of EMG onsets and offsets, and no influence of peripheral feedback on EMG timing. Differences in EMG integrals for normal and unexpectedly unloaded trials indicate that peripheral feedback does play a role in influencing EMG magnitude during perturbed trials. These results also confirm the predictions of the

Latash & Gottlieb Equilibrium Point Model, which predicted that there is a separate control lambda for antagonist muscles, that there would be no change in agonist onset. The Feldman model, which predicted early onset of antagonist EMG in reduced inertial load conditions, failed in its prediction.

Similarly, the two models have contradictory predictions for antagonist onset in conditions of high opposing loads, where the Latash & Gottlieb Equilibrium Point Model states that the antagonist EMG burst is centrally planned and would be expressed due to the "N" shaped antagonist lambda, regardless of high opposing load or "infinite load" encountered with movement blocking (Latash, 1994). The Feldman model states that the reciprocal (R) and co-contraction (C) commands together create muscle activation thresholds for both the extensor and flexor muscles that cause triphasic antagonist bursts in isometric conditions for short movements, but not for movements larger than 40-55 degrees because the unstretched antagonist muscle would be less than its threshold activation length (Feldman et al, 1995, p. 447). While both models agree that antagonist EMG bursts will be seen in isotonic and isometric conditions, they disagree if an antagonist burst will be seen if a large opposing load stops a large amplitude intended movement. The Latash & Gottlieb EP model predicts that halting a fast movement would still elicit an antagonist burst, although smaller in amplitude while the Feldman model predicts that the antagonist burst will be suppressed by a high opposing load, and expressed upon load release. In order to test these predictions, seven participants performed elbow flexion movements of 60 degrees, with random elastic opposing loads of up to 60 Nm applied after movement onset, halting movements within 5 degrees of the start position, releasing movement after 50-100 ms, permitting participants to accurately achieve their intended target position (Feldman et al, 1995). Antagonist EMG

bursts were not seen during the halting of movement, but were clearly seen after release of the opposing load, as predicted by the Feldman Equilibrium Point model. Given the short interval of less than 120 ms. from the initiation of movement to load release, participants could not have made any voluntary movement corrections. Suppression of the antagonist burst until approximately 50 ms after the release of opposing load, the "unloading reflex" (Forget an Lamarre, 1987), indicate that peripheral feedback is critically involved in antagonist EMG production. The speed of equilibrium point shift, calculated in isometric conditions from EMG onset to achievement of steady state, is confirmed by the halting load condition to be approximately 600 degrees/second, offering an explanation of why the first agonist EMG burst are not affected by perturbations. Perturbations effects in EMG of fast, goal-directed movements would only be seen approximately 100 ms after EMG onset, due to approximately 50 ms muscle motor time to movement onset, very short (zero) sensory time, and 50 ms stretch reflex response times. An intended movement of 60 degrees, for example, would have an equilibrium point shift of approximately 100 ms, ending at the same time as the first perturbation induced changes in EMG would become expressed 100 ms after EMG onset. Response to perturbations depends on the speed of movement, and the type of perturbation.

The role of peripheral feedback in the first agonist EMG burst preparation is unclear, and the statement "for at least the first 100 ms the motor system does not make use of proprioceptive movement information for control" (Wadman, et al., 1979, p. 9), implied that agonist activation was totally prepared and performed centrally, in the absence of peripheral feedback. In reality the first agonist EMG burst is directly affected through stretch reflex feedback loops, and by manipulation of muscle length activation thresholds, with early

agonist onset times seen for opposing loads compared to assisting loads (Adamovich, Levin, and Feldman, 1997). Opposing loads increase an agonist muscle's length forcing earlier activation to a decreasing stretch-reflex activation length, while assisting loads decrease muscle length resulting in later activation. This effect was only seen with larger perturbations of 8-15 Nm, and was not seen in previous studies with perturbations of 3-5 Nm (Brown and Cooke, 1986) due to the large recruitment of stretch reflex pathways for movement production.

Integrated Movement Control: The Gottlieb Model

Fast voluntary movements are affected differently by various types of external perturbing loads, whether it be elastic, viscous or inertial. Tests of these three types of loads in both expected and unexpected testing conditions were conducted to study the effects of these loads and develop a better model of movement control (Gottlieb, 1996). In order for participants not to alter their central commands in response to the perturbations, changes in loads must be applied smoothly, based on movement kinematics rather than delivered abruptly, and especially not before movement has begun. Unexpected elastic loads produce very little change in phasic EMG, but cause final aiming errors requiring correction, unexpected inertial loads produce some phasic changes in EMG but do not lead to any final position errors, while unexpected changes in viscous loads sometimes alter phasic EMG, and sometimes produce final position errors. These results imply the need for a new control model based on three components, an α-λ-γ (Apha-Lambda-Gamma) control model for single joint movement. The alpha (α) component is an excitation pattern based on estimates of the required dynamics of the intended movement used to activate muscle directly as seen in deafferentation studies. This feedforward component would be based on extensive training,

and internal model of the intended movement. The lambda (λ) component would be analogous to a virtual trajectory of movement, a kinematic reference for error correction for slower movements. Fast movements would rely almost exclusively on the alpha component at first, almost the entire duration of a short movement, while longer movements would shift to lambda control for movement corrections and completion. Slower movements would be almost entirely lambda based, since the delays due to reflex loop timing would not seriously affect movement performance. The gamma (γ) component sets the gains and thresholds of various component reflexes, allowing for continuous reflex-response adjustment during movements. This component accounts for differing reflex gains seen with known and unexpected loadings, and also explains reflex-reversal effects found in the analysis of gait (Gottlieb, 1996, p. 3226). The three component alpha-lambda-gamma model includes an additional element, an intelligent controller coordinating the various parts of movement, especially complex patterns such as gait, for example, and is very knowledgeable at decomposing intended movements into their appropriate components.

Modeled and used extensively within robotic control systems, these interlinked, hierarchical, yet independent control processes both integrate and decompose descending commands and peripheral feedback at the same time (Raibert, 1986; Brooks, 1986, 1989). An intelligent controller functionally midway between the brain and limb is able to integrate limb position with descending commands, for example, to transform actions into limb or body coordinates. This functional integration is seen artificially in the actions of the spinalized frog where an acid stimulus triggers the so called "wiping reflex", the coordinated movement of the rear limb to contact the forward limb and remove an irritant (Fukson, et al, 1980). A normal healthy frog wanting to scratch it's elbow would not have to plan the entire

movement, it would only plan to touch its rear leg to a certain location on his front leg, and the movement would be coordinated automatically. Such movements are planned according to a body-based coordinate system, with a global center of reference in the head in the head centered around the eyes and Vestibular system. A dynamic 'map' of limb location and physical space is maintained within the brain, creating the body's kinesthetic sense or virtual body image, neural damage to sensory pathways causes reference problems as limbs appear to drift into 'uncomfortable' positions, giving rise to phantom limb pain (Melzack, 1992). At a lower level within the spinal cord, the dynamic interaction of these controllers under central control may be responsible for limb coordination in movement (Kelso, Southlard, and Goodman, 1979; Tuller, Turvey, and Fitch, 1982)

One consequence of such a 'smart' central control system would be the ability to appropriately compensate for damage or loss of certain control channels, such as with neurological damage resulting in proprioceptive deafferentation (Lashley, 1917, Nougier, et al., 1996). With the loss of peripheral feedback the smart internal controller would have to fall back on using only alpha (α) motoneuron control characterized by impulse timing of EMG, where the EMG burst amplitude and duration are preset by central commands, and executed without the use of feedback control loops. Two deafferented patients participated in a series of experiments investigating the limits of movement control of forearm supination-pronation compared to normal test participants (Nougier, et al., 1996). In continuous supination-pronation movements of 60 degrees deafferented patients produced the highest spatial error (35 degrees no vision, 2 degrees with vision), but were consistently good with movement amplitude production (approximately 10 degrees error, vision and no vision). With an 8 second delay between the presentation of new movement targets, control

participants performed movements with greater amplitude error (13 degrees) than the deafferented patient (approximately 8 degrees). A second experiment consisting of alternating supination and pronation of 20 degrees amplitude would occasionally have magnetic brake activation preventing movement to the first target, and releasing at the presentation of the second target. Results of this experiment showed that upon brake release of the prevented supination movement, patients performed a small supination before performing a full amplitude pronation movement while control participants did not move at all. Both of these results indicate that in the absence of peripheral feedback deafferent patients used impulse-timing of EMG for forearm pronation-supination movements rather than force-length control of their limbs as seen in elbow extension-flexion movements (Bizzi, 1980).

The timing of the individual EMG bursts in triphasic activity is both central an peripherally based, the first agonist burst being centrally initiated with peripheral feedback playing a minor role (Wadman, et al., 1987), while the antagonist burst amplitude and timing are dependent on feedback for accuracy (Forget & Lamarre, 1987). Stimulation of the cranial motor cortex with a Transcranial Magnetic Stimulation (TMS) system during the reaction period between presentation of stimulus and the first expression of agonist EMG allows the measurement of increased evoked EMG (MEP) activity compared to integrated EMG activity (IEMP) due to increased cortical excitability (MacKinnon & Rothwell, 2000). With TMS stimulation, the onset time of first agonist MEP was decreased by approximately 10 ms, while the ratio of MEP:IEMP values increased by approximately seven times, indicating that TMS stimulation released motor commands early, and with increased amplitude. Five of nine participants showed increased MEP:IEMP activity preceding the

second agonist burst, also indicating central control of the second agonist burst. Antagonist EMG was not preceded by any change in the ratio of evoked motor potential and integrated EMG activity (MEP:IEMP), indicating antagonist activity is not initiated by the same cortical mechanisms as the agonist bursts. This research shows that changes in motor cortical excitability controlling the initiation of movement occurs less than 23 ms before the onset of first agonist EMG, rather than the larger 83-100 ms reported in previous studies.

Under isometric movement blocking conditions, where movement is prevented by a load of "infinite mass", muscles act as static force generators where force levels reflect the descending command activation levels (Ghafouri & Feldman, 2001). Horizontal arm extension movements to three target locations were randomly blocked with resulting isometric force production reaching steady state after 150 ms, compared to more than 500 ms to reach steady state in normal isotonic conditions. This indicates that the descending command controlling agonist muscular activation also reaches steady state within 150 ms, at approximately the same time as peak velocity is reached in unblocked normal movements. In order to study the patterning of sequential movements, participants were also asked to perform a secondary force production in response to blocked movements, initiating secondary force production after initial muscle force production reached steady state, well before the end of first movement in unblocked conditions. These results show that the equilibrium point shifts driving movement are fast, monotonically increasing functions of approximately 600 degrees per second, reaching a steady state at approximately peak velocity of unblocked movements.

Experiments in isometric force production of elbow flexion with unexpected mechanical extension or flexion perturbations were conducted to study the production of

compensatory EMG production (Flanders & Cordo, 1987). Forced flexion movements of isometric flexion force production resulted in reduced agonist activity and increased antagonist activity, an anticipated reciprocal activation pattern accountable for by known stretch and unloading reflexes. Forced extension movements resulted in increased activation of both agonist and antagonist compared to the isometric flexion condition, this co-activation pattern was not expected, and not consistent with know reflex mechanisms. Unloading reflexes were seen in shortening agonist muscles (forced flexion), but not in shortening antagonist muscles (forced extension), a phenomenon know a "reflex reversal" seen in spinalized cats (Forssberg et al. 1977).

Conclusions

These various models of movement control all attempt to describe the underlying mechanisms governing voluntary movement, and movement in general, each model has its strengths, weaknesses, and special cases where it fails in its predictions. The alpha model of exclusively central, muscle length-threshold control applies only in the special case of sensory deafferentation, or for the first part of very fast movements within the three component alpha-lambda-gamma model (Gottlieb, 1996). The original two component lambda model describes movements as driven by shifts in muscle length-thresholds for both agonist and antagonist muscles, using sensory feedback to maintain movement and posture for slow and fast movements (Feldman, 1986). For very fast movements an additional component is required to help dampen out limb oscillations at the end of movement, giving rise to the three element Feldman lambda model (St-Onge, Adamovich, and Feldman, 1997). However, this model makes an incorrect prediction that all EMG is triggered by peripheral feedback, and that the antagonist EMG would be triggered early by an unloading trial, while

the Latash and Gottlieb model (Latash, 1994) correctly predicts no change in antagonist EMG onset latency. But the Latash and Gottlieb model also makes an incorrect prediction that antagonist EMG would not be suppressed by high opposing loads, while the Feldman model correctly predicts suppression of the antagonist EMG burst until after the release of the opposing loads. Each of the models can not be both correct and incorrect depending on external situations, necessitating the development of another model that allows movement without reflexive feedback, movement with feedback, movement with or without perturbations from the external environment. This final model integrates direct alpha motoneuron control of muscles, lambda movement regulation, and gamma reflex control, and decentralized autonomous spinal control structures for movement coordination (Gottlieb, 1996). The three component Gottlieb alpha-lambda-gamma control model is able to make limited predictions about fast movement control, but the complex interactions of spinal control structures limits the scope of these predictions to those covered originally by the mechanistic alpha and lambda models. If very fast movements are primarily under alpha control for the first part of movement, then switch to lambda control later, there will be a way to influence the switch from one control structure to another, but this is not obvious from the model, it makes no clear predictions on the influence of strategy.

Appendix B: Statistical Results

ANOVA Condition Code Legend

USL –Unimanual Short Left	MSL –Unequal bimanual Short Left	BSL –Equal bimanual Short Left
USR –Unimanual Short Right	MSR –Unequal bimanual Short Right	BSR –Equal bimanual Short Right
ULL –Unimanual Long Left	MLL –Unequal bimanual Long Left	BLL –Equal bimanual Long Left
ULR –Unimanual Long Right	MLR –Unequal bimanual Long Right	BLR –Equal bimanual Long Right

Movement Distance

Movement Distance (degrees)

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Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
12.990	12.670	49.930	50.820	14.440

MSR	MLL	MLR	BSL	BSR
14.270	49.880	48.880	13.080	11.610

BLL	BLR
49.460	47.460

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	48.753	2	24.376	16.154	0.000	0.000	0.000
Error	27.162	18	1.509				

Greenhouse-Geisser Epsilon: 0.9725
Huynh-Feldt Epsilon : 1.0000

Distance	39374.764	1	39374.764	2793.599	0.000	.	.
Error	126.852	9	14.095				

Greenhouse-Geisser Epsilon:

Hand	13.804	1	13.804	2.694	0.135	.	.
Error	46.112	9	5.124				

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .

Condition							
*Distance	31.945	2	15.972	5.085	0.018	0.027	0.020
Error	56.537	18	3.141				

Greenhouse-Geisser Epsilon: 0.7966
Huynh-Feldt Epsilon : 0.9404

Condition							
*Hand	20.533	2	10.266	4.635	0.024	0.037	0.029
Error	39.869	18	2.215				

Greenhouse-Geisser Epsilon: 0.7614
Huynh-Feldt Epsilon : 0.8845

Distance							
*Hand	0.019	1	0.019	0.005	0.944	.	.
Error	32.014	9	3.557				

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .

Condition							
*Distance							
*Hand	6.066	2	3.033	1.336	0.288	0.286	0.287
Error	40.869	18	2.271				

Greenhouse-Geisser Epsilon: 0.7180
Huynh-Feldt Epsilon : 0.8170

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.245	2	8	12.320	0.004
Pillai Trace =	0.755	2	8	12.320	0.004
H-L Trace =	3.080	2	8	12.320	0.004

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.457	2	8	4.760	0.043
Pillai Trace =	0.543	2	8	4.760	0.043
H-L Trace =	1.190	2	8	4.760	0.043

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.374	2	8	6.700	0.020
Pillai Trace =	0.626	2	8	6.700	0.020
H-L Trace =	1.675	2	8	6.700	0.020

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.802	2	8	0.985	0.415
Pillai Trace =	0.198	2	8	0.985	0.415
H-L Trace =	0.246	2	8	0.985	0.415

Velocity Reaction Time

Velocity Reaction Time (velocity > 8 deg/s)

MON 6/24/02 2:21:15 PM

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Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
186.680	194.190	195.950	195.540	215.310

MSR	MLL	MLR	BSL	BSR
216.120	200.720	207.450	191.750	193.840

BLL	BLR
193.980	196.590

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	7133.576	2	3566.788	12.580	0.000	0.001	0.000
Error	5103.711	18	283.539				
Greenhouse-Geisser Epsilon:			0.9316				
Huynh-Feldt Epsilon :			1.0000				
Distance	48.896	1	48.896	0.284	0.607	.	.
Error	1548.859	9	172.095				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	311.696	1	311.696	2.268	0.166	.	.
Error	1236.982	9	137.442				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	1647.635	2	823.817	11.146	0.001	0.002	0.001
Error	1330.445	18	73.914				

Greenhouse-Geisser Epsilon: 0.7833
Huynh-Feldt Epsilon : 0.9193

Condition							
*Hand	11.683	2	5.841	0.222	0.803	0.698	0.715
Error	473.564	18	26.309				

Greenhouse-Geisser Epsilon: 0.6185
Huynh-Feldt Epsilon : 0.6679

Distance							
*Hand	1.825	1	1.825	0.011	0.918	.	.
Error	1462.873	9	162.541				

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .

Condition							
*Distance							
*Hand	243.283	2	121.641	1.123	0.347	0.345	0.347
Error	1950.124	18	108.340				

Greenhouse-Geisser Epsilon: 0.9479
Huynh-Feldt Epsilon : 1.0000

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.259	2	8	11.437	0.005
Pillai Trace =	0.741	2	8	11.437	0.005
H-L Trace =	2.859	2	8	11.437	0.005

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.381	2	8	6.494	0.021
Pillai Trace =	0.619	2	8	6.494	0.021
H-L Trace =	1.623	2	8	6.494	0.021

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.838	2	8	0.772	0.494
Pillai Trace =	0.162	2	8	0.772	0.494
H-L Trace =	0.193	2	8	0.772	0.494

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.775	2	8	1.159	0.361
Pillai Trace =	0.225	2	8	1.159	0.361
H-L Trace =	0.290	2	8	1.159	0.361

Time to Peak Velocity

Time to Peak Velocity (ms)

THU 6/27/02 3:33:45 PM

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Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
76.500	73.790	110.340	110.140	88.210

MSR	MLL	MLR	BSL	BSR
87.630	116.780	114.500	79.580	74.740

BLL	BLR
111.820	108.850

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	1976.223	2	988.112	15.237	0.000	0.000	0.000
Error	1167.267	18	64.848				
Greenhouse-Geisser Epsilon:			0.9334				
Huynh-Feldt Epsilon :			1.0000				
Distance	30713.600	1	30713.600	261.928	0.000	.	.
Error	1055.336	9	117.260				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	153.680	1	153.680	1.938	0.197	.	.
Error	713.550	9	79.283				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	292.780	2	146.390	4.486	0.026	0.037	0.028
Error	587.403	18	32.634				

Greenhouse-Geisser Epsilon:		0.8050				
Huynh-Feldt Epsilon :		0.9539				
Condition						
*Hand	40.429	2	20.215	0.752	0.486	0.429	0.436
Error	483.651	18	26.869				

Greenhouse-Geisser Epsilon:		0.5995				
Huynh-Feldt Epsilon :		0.6403				
Distance						
*Hand	5.985	1	5.985	0.123	0.733	.	.
Error	436.405	9	48.489				

Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Distance						
*Hand	25.732	2	12.866	0.533	0.596	0.546	0.569
Error	434.128	18	24.118				

Greenhouse-Geisser Epsilon:		0.7394				
Huynh-Feldt Epsilon :		0.8502				

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.197	2	8	16.274	0.002
Pillai Trace =	0.803	2	8	16.274	0.002
H-L Trace =	4.068	2	8	16.274	0.002

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.371	2	8	6.782	0.019
Pillai Trace =	0.629	2	8	6.782	0.019
H-L Trace =	1.696	2	8	6.782	0.019

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.559	2	8	3.158	0.098
Pillai Trace =	0.441	2	8	3.158	0.098
H-L Trace =	0.789	2	8	3.158	0.098

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.907	2	8	0.409	0.677
Pillai Trace =	0.093	2	8	0.409	0.677
H-L Trace =	0.102	2	8	0.409	0.677

Peak Velocity

Peak Velocity (deg/s)

THU 6/27/02 2:17:16 PM

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IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
126.080	133.500	361.000	367.500	130.170

MSR	MLL	MLR	BSL	BSR
130.730	325.460	341.410	126.370	120.590

BLL	BLR
375.950	375.270

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	7247.395	2	3623.698	4.427	0.027	0.050	0.044
Error	14734.786	18	818.599				
Greenhouse-Geisser Epsilon:			0.6541				
Huynh-Feldt Epsilon :			0.7204				
Distance	1585045.602	1	1585045.602	289.291	0.000	.	.
Error	49311.589	9	5479.065				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	478.801	1	478.801	0.777	0.401	.	.
Error	5545.677	9	616.186				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	12393.785	2	6196.893	7.894	0.003	0.006	0.003
Error	14130.997	18	785.055				

Greenhouse-Geisser Epsilon: 0.8492
Huynh-Feldt Epsilon : 1.0000

Condition							
*Hand	791.394	2	395.697	0.946	0.407	0.376	0.383
Error	7526.441	18	418.136				

Greenhouse-Geisser Epsilon: 0.6484
Huynh-Feldt Epsilon : 0.7120

Distance							
*Hand	319.154	1	319.154	0.526	0.487	.	.
Error	5459.493	9	606.610				

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .

Condition *Distance							
*Hand	340.117	2	170.059	0.471	0.632	0.619	0.632
Error	6503.538	18	361.308				

Greenhouse-Geisser Epsilon: 0.9284
Huynh-Feldt Epsilon : 1.0000

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.237	2	8	12.883	0.003
Pillai Trace =	0.763	2	8	12.883	0.003
H-L Trace =	3.221	2	8	12.883	0.003

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.331	2	8	8.085	0.012
Pillai Trace =	0.669	2	8	8.085	0.012
H-L Trace =	2.021	2	8	8.085	0.012

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.619	2	8	2.460	0.147
Pillai Trace =	0.381	2	8	2.460	0.147
H-L Trace =	0.615	2	8	2.460	0.147

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.919	2	8	0.354	0.712
Pillai Trace =	0.081	2	8	0.354	0.712
H-L Trace =	0.089	2	8	0.354	0.712

Movement Time (Velocity greater than 8 degrees per second)

Movement Time (Velocity > 8 deg/s)(ms)

IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
159.760	150.940	259.840	266.560	175.400

MSR	MLL	MLR	BSL	BSR
175.330	301.690	286.580	165.990	154.700

BLL	BLR
254.650	250.420

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	19446.253	2	9723.126	12.954	0.000	0.001	0.000
Error	13510.444	18	750.580				
Greenhouse-Geisser Epsilon:			0.8473				
Huynh-Feldt Epsilon :			1.0000				
Distance	338799.387	1	338799.387	48.861	0.000	.	.
Error	62405.866	9	6933.985				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	896.533	1	896.533	1.232	0.296	.	.
Error	6550.337	9	727.815				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	3569.928	2	1784.964	2.574	0.104	0.137	0.135
Error	12482.859	18	693.492				
Greenhouse-Geisser Epsilon:			0.5683				
Huynh-Feldt Epsilon :			0.5955				
Condition							
*Hand	292.749	2	146.374	0.728	0.496	0.474	0.494
Error	3616.781	18	200.932				

Greenhouse-Geisser Epsilon:		0.8222					
Huynh-Feldt Epsilon :		0.9819					
Distance							
*Hand	47.628	1	47.628	0.120	0.737	.	.
Error	3559.575	9	395.508				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Condition							
*Distance							
*Hand	1246.214	2	623.107	3.756	0.043	0.046	0.043
Error	2986.013	18	165.890				
Greenhouse-Geisser Epsilon:		0.9531					
Huynh-Feldt Epsilon :		1.0000					

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.175	2	8	18.830	0.001
Pillai Trace =	0.825	2	8	18.830	0.001
H-L Trace =	4.707	2	8	18.830	0.001

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.738	2	8	1.418	0.297
Pillai Trace =	0.262	2	8	1.418	0.297
H-L Trace =	0.354	2	8	1.418	0.297

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.900	2	8	0.442	0.658
Pillai Trace =	0.100	2	8	0.442	0.658
H-L Trace =	0.111	2	8	0.442	0.658

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.594	2	8	2.734	0.125
Pillai Trace =	0.406	2	8	2.734	0.125
H-L Trace =	0.683	2	8	2.734	0.125

Acceleration Onset (RT)

Acceleration Onset Time (ms)

FRI 6/28/02 7:52:11 AM

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IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
168.790	175.740	179.120	178.900	196.420

MSR	MLL	MLR	BSL	BSR
197.140	181.270	189.790	173.170	175.480

BLL	BLR
177.710	180.130

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	6039.421	2	3019.711	10.460	0.001	0.001	0.001
Error	5196.402	18	288.689				
Greenhouse-Geisser Epsilon:			0.9518				
Huynh-Feldt Epsilon :			1.0000				
Distance	0.027	1	0.027	0.000	0.990	.	.
Error	1592.683	9	176.965				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	357.075	1	357.075	1.837	0.208	.	.
Error	1749.718	9	194.413				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	1931.688	2	965.844	12.324	0.000	0.002	0.001
Error	1410.631	18	78.368				

```
Greenhouse-Geisser Epsilon:       0.7597
Huynh-Feldt Epsilon       :       0.8819
Condition
*Hand              25.533      2       12.767     0.383   0.687   0.607   0.627
Error             599.963     18       33.331


Greenhouse-Geisser Epsilon:       0.6646
Huynh-Feldt Epsilon       :       0.7361
Distance
*Hand               0.456      1        0.456     0.002   0.961     .       .
Error            1662.784      9      184.754


Greenhouse-Geisser Epsilon:        .
Huynh-Feldt Epsilon       :        .
Condition
*Distance
*Hand             280.196      2      140.098     1.259   0.308   0.307   0.308
Error            2003.644     18      111.314


Greenhouse-Geisser Epsilon:       0.9582
Huynh-Feldt Epsilon       :       1.0000
------------------------------------------------------------------------------


Multivariate Repeated Measures Analysis

Test of: Condition          Hypoth. df  Error df       F           P
  Wilks' Lambda=      0.287       2          8        9.924       0.007
  Pillai Trace =      0.713       2          8        9.924       0.007
  H-L Trace    =      2.481       2          8        9.924       0.007

Test of: Condition          Hypoth. df  Error df       F           P
         *Distance
  Wilks' Lambda=      0.354       2          8        7.295       0.016
  Pillai Trace =      0.646       2          8        7.295       0.016
  H-L Trace    =      1.824       2          8        7.295       0.016

Test of: Condition          Hypoth. df  Error df       F           P
         *Hand
  Wilks' Lambda=      0.773       2          8        1.173       0.357
  Pillai Trace =      0.227       2          8        1.173       0.357
  H-L Trace    =      0.293       2          8        1.173       0.357

Test of: Condition          Hypoth. df  Error df       F           P
         *Distance
         *Hand
  Wilks' Lambda=      0.749       2          8        1.343       0.314
  Pillai Trace =      0.251       2          8        1.343       0.314
  H-L Trace    =      0.336       2          8        1.343       0.314
```

Time to Peak Acceleration

Time to Peak Acceleration (ms)

FRI 6/28/02 8:20:19 AM

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IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
43.160	42.450	50.310	51.220	46.520

MSR	MLL	MLR	BSL	BSR
46.510	54.140	53.880	44.590	43.490

BLL	BLR
48.830	48.710

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	361.569	2	180.785	4.694	0.023	0.031	0.023
Error	693.325	18	38.518				
Greenhouse-Geisser Epsilon:			0.8452				
Huynh-Feldt Epsilon :			1.0000				
Distance	1358.114	1	1358.114	4.458	0.064	.	.
Error	2742.033	9	304.670				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	1.387	1	1.387	0.016	0.903	.	.
Error	799.077	9	88.786				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	60.981	2	30.491	1.673	0.216	0.225	0.222
Error	328.024	18	18.224				

Greenhouse-Geisser Epsilon:			0.7102				
Huynh-Feldt Epsilon :			0.8051				
Condition							
*Hand	2.617	2	1.308	0.071	0.932	0.881	0.906
Error	332.162	18	18.453				

Greenhouse-Geisser Epsilon:			0.7365				
Huynh-Feldt Epsilon :			0.8456				
Distance							
*Hand	4.602	1	4.602	0.124	0.733	.	.
Error	333.782	9	37.087				

Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance							
*Hand	4.516	2	2.258	0.104	0.902	0.838	0.865
Error	392.252	18	21.792				

Greenhouse-Geisser Epsilon:			0.7176				
Huynh-Feldt Epsilon :			0.8164				

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.366	2	8	6.922	0.018
Pillai Trace =	0.634	2	8	6.922	0.018
H-L Trace =	1.730	2	8	6.922	0.018

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.606	2	8	2.605	0.134
Pillai Trace =	0.394	2	8	2.605	0.134
H-L Trace =	0.651	2	8	2.605	0.134

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.963	2	8	0.155	0.859
Pillai Trace =	0.037	2	8	0.155	0.859
H-L Trace =	0.039	2	8	0.155	0.859

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.967	2	8	0.135	0.876
Pillai Trace =	0.033	2	8	0.135	0.876
H-L Trace =	0.034	2	8	0.135	0.876

Peak Acceleration

Peak Acceleration (degrees/second/second)

FRI 6/28/02 8:15:38 AM

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Welcome to SYSTAT!
IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
2637.180	3026.150	5479.120	5510.780	2435.410

MSR	MLL	MLR	BSL	BSR
2478.940	4704.620	4956.490	2607.410	2611.280

BLL	BLR
5654.580	5754.650

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	7108634.017	2	3554317.009	6.321	0.008	0.015	0.011
Error	1.01218E+07	18	562320.696				
Greenhouse-Geisser Epsilon:			0.7751				
Huynh-Feldt Epsilon :			0.9062				
Distance	2.20428E+08	1	2.20428E+08	68.884	0.000	.	.
Error	2.87999E+07	9	3199984.736				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	560292.334	1	560292.334	0.441	0.523	.	.
Error	1.14371E+07	9	1270787.360				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	2639270.404	2	1319635.202	2.828	0.086	0.113	0.107
Error	8399477.150	18	466637.619				

Greenhouse-Geisser Epsilon: 0.6531
Huynh-Feldt Epsilon : 0.7188

Condition							
*Hand	127193.367	2	63596.684	0.254	0.778	0.648	0.656
Error	4503601.591	18	250200.088				

Greenhouse-Geisser Epsilon: 0.5515
Huynh-Feldt Epsilon : 0.5717

Distance							
*Hand	2320.561	1	2320.561	0.003	0.959	.	.
Error	7356413.007	9	817379.223				

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .

Condition *Distance							
*Hand	448505.519	2	224252.759	0.781	0.473	0.470	0.473
Error	5168165.957	18	287120.331				

Greenhouse-Geisser Epsilon: 0.9700
Huynh-Feldt Epsilon : 1.0000

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.255	2	8	11.667	0.004
Pillai Trace =	0.745	2	8	11.667	0.004
H-L Trace =	2.917	2	8	11.667	0.004

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.691	2	8	1.791	0.228
Pillai Trace =	0.309	2	8	1.791	0.228
H-L Trace =	0.448	2	8	1.791	0.228

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.808	2	8	0.948	0.427
Pillai Trace =	0.192	2	8	0.948	0.427
H-L Trace =	0.237	2	8	0.948	0.427

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.848	2	8	0.717	0.517
Pillai Trace =	0.152	2	8	0.717	0.517
H-L Trace =	0.179	2	8	0.717	0.517

Peak Positive Force

Peak Positive Force (Newtons)
FRI 6/28/02 9:36:00 AM

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IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
10.330	13.710	15.330	21.700	11.730

MSR	MLL	MLR	BSL	BSR
12.030	15.860	19.500	12.020	12.540

BLL	BLR
18.800	21.630

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	44.688	2	22.344	1.696	0.211	0.224	0.223
Error	237.115	18	13.173				
Greenhouse-Geisser Epsilon:			0.5990				
Huynh-Feldt Epsilon :			0.6396				
Distance	1364.176	1	1364.176	101.208	0.000	.	.
Error	121.310	9	13.479				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	241.968	1	241.968	5.958	0.037	.	.
Error	365.485	9	40.609				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	23.716	2	11.858	1.799	0.194	0.200	0.194
Error	118.627	18	6.590				

```
Greenhouse-Geisser Epsilon:       0.8599
Huynh-Feldt Epsilon       :       1.0000
Condition
*Hand              62.554      2       31.277     4.475   0.026   0.049   0.043
Error             125.803     18        6.989


Greenhouse-Geisser Epsilon:       0.6528
Huynh-Feldt Epsilon       :       0.7184
Distance
*Hand              62.208      1       62.208     4.614   0.060     .       .
Error             121.339      9       13.482


Greenhouse-Geisser Epsilon:       .
Huynh-Feldt Epsilon       :       .
Condition
*Distance
*Hand               1.372      2        0.686     0.260   0.774   0.739   0.774
Error              47.452     18        2.636


Greenhouse-Geisser Epsilon:       0.8483
Huynh-Feldt Epsilon       :       1.0000
-------------------------------------------------------------------------------


Multivariate Repeated Measures Analysis

Test of: Condition          Hypoth. df  Error df      F           P
  Wilks' Lambda=      0.421       2          8        5.503       0.031
  Pillai Trace =      0.579       2          8        5.503       0.031
  H-L Trace    =      1.376       2          8        5.503       0.031

Test of: Condition          Hypoth. df  Error df      F           P
           *Distance
  Wilks' Lambda=      0.696       2          8        1.751       0.234
  Pillai Trace =      0.304       2          8        1.751       0.234
  H-L Trace    =      0.438       2          8        1.751       0.234

Test of: Condition          Hypoth. df  Error df      F           P
           *Hand
  Wilks' Lambda=      0.627       2          8        2.382       0.154
  Pillai Trace =      0.373       2          8        2.382       0.154
  H-L Trace    =      0.595       2          8        2.382       0.154

Test of: Condition          Hypoth. df  Error df      F           P
           *Distance
           *Hand
  Wilks' Lambda=      0.921       2          8        0.345       0.718
  Pillai Trace =      0.079       2          8        0.345       0.718
  H-L Trace    =      0.086       2          8        0.345       0.718
```

Triceps – Biceps Onset Difference

Triceps - Biceps Onset Difference (ms)

FRI 6/28/02 11:42:35 AM

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IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
69.320	76.600	112.760	125.040	85.800

MSR	MLL	MLR	BSL	BSR
88.130	114.100	118.980	72.470	71.080

BLL	BLR
120.060	120.830

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	876.956	2	438.478	2.654	0.098	0.121	0.114
Error	2973.597	18	165.200				
Greenhouse-Geisser Epsilon:			0.6957				
Huynh-Feldt Epsilon :			0.7830				
Distance	51406.381	1	51406.381	163.744	0.000	.	.
Error	2825.498	9	313.944				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	569.852	1	569.852	1.211	0.300	.	.
Error	4235.404	9	470.600				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	2132.951	2	1066.475	8.548	0.002	0.003	0.002
Error	2245.853	18	124.770				

```
Greenhouse-Geisser Epsilon:       0.9923
Huynh-Feldt Epsilon       :       1.0000
Condition
*Hand              517.553      2      258.777     3.314   0.060   0.083   0.076
Error             1405.624     18       78.090


Greenhouse-Geisser Epsilon:       0.6878
Huynh-Feldt Epsilon       :       0.7710
Distance
*Hand               78.570      1       78.570     0.593   0.461       .       .
Error             1191.612      9      132.401


Greenhouse-Geisser Epsilon:       .
Huynh-Feldt Epsilon       :       .
Condition
*Distance
*Hand               11.850      2        5.925     0.082   0.922   0.915   0.922
Error             1300.910     18       72.273


Greenhouse-Geisser Epsilon:       0.9584
Huynh-Feldt Epsilon       :       1.0000
-------------------------------------------------------------------------------


Multivariate Repeated Measures Analysis

Test of: Condition          Hypoth. df  Error df      F          P
  Wilks' Lambda=      0.459      2          8        4.716      0.044
  Pillai Trace =      0.541      2          8        4.716      0.044
  H-L Trace    =      1.179      2          8        4.716      0.044

Test of: Condition          Hypoth. df  Error df      F          P
           *Distance
  Wilks' Lambda=      0.358      2          8        7.181      0.016
  Pillai Trace =      0.642      2          8        7.181      0.016
  H-L Trace    =      1.795      2          8        7.181      0.016

Test of: Condition          Hypoth. df  Error df      F          P
           *Hand
  Wilks' Lambda=      0.488      2          8        4.203      0.057
  Pillai Trace =      0.512      2          8        4.203      0.057
  H-L Trace    =      1.051      2          8        4.203      0.057

Test of: Condition          Hypoth. df  Error df      F          P
           *Distance
           *Hand
  Wilks' Lambda=      0.984      2          8        0.064      0.938
  Pillai Trace =      0.016      2          8        0.064      0.938
  H-L Trace    =      0.016      2          8        0.064      0.938
```

Q 30 Agonist Onset Slope Measure

Q30 Measure (Percent MVC * ms)

FRI 6/28/02 12:17:05 PM

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Welcome to SYSTAT!
IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
2055.020	1853.240	2582.240	2025.590	1868.470

MSR	MLL	MLR	BSL	BSR
1536.330	2613.110	2116.590	2208.880	1726.390

BLL	BLR
2725.700	2171.860

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	611333.287	2	305666.644	1.365	0.281	0.281	0.281
Error	4031407.606	18	223967.089				
Greenhouse-Geisser Epsilon:			0.8818				
Huynh-Feldt Epsilon :			1.0000				
Distance	7433946.081	1	7433946.081	24.360	0.001	.	.
Error	2746570.864	9	305174.540				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	5735277.080	1	5735277.080	7.982	0.020	.	.
Error	6466811.758	9	718534.640				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	492954.516	2	246477.258	2.211	0.138	0.141	0.138
Error	2006636.934	18	111479.830				

Greenhouse-Geisser Epsilon:		0.9675					
Huynh-Feldt Epsilon :		1.0000					
Condition							
*Hand	104406.243	2	52203.122	0.836	0.450	0.439	0.450
Error	1124272.704	18	62459.595				
Greenhouse-Geisser Epsilon:		0.8927					
Huynh-Feldt Epsilon :		1.0000					
Distance							
*Hand	290673.633	1	290673.633	0.728	0.416	.	.
Error	3591572.455	9	399063.606				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Condition							
*Distance							
*Hand	104437.176	2	52218.588	0.604	0.557	0.506	0.523
Error	1555978.661	18	86443.259				
Greenhouse-Geisser Epsilon:		0.7023					
Huynh-Feldt Epsilon :		0.7930					

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.677	2	8	1.909	0.210
Pillai Trace =	0.323	2	8	1.909	0.210
H-L Trace =	0.477	2	8	1.909	0.210

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.625	2	8	2.404	0.152
Pillai Trace =	0.375	2	8	2.404	0.152
H-L Trace =	0.601	2	8	2.404	0.152

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.870	2	8	0.598	0.573
Pillai Trace =	0.130	2	8	0.598	0.573
H-L Trace =	0.150	2	8	0.598	0.573

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.918	2	8	0.356	0.711
Pillai Trace =	0.082	2	8	0.356	0.711
H-L Trace =	0.089	2	8	0.356	0.711

Q100 Agonist burst Integral Measure

Q100 First Agonist burst Integral (%MVC * ms)

IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

USL	USR	ULL	ULR	MSL
5393.690	5238.680	8914.690	8317.700	4731.730

MSR	MLL	MLR	BSL	BSR
3987.200	8058.000	7633.520	5185.980	4508.880

BLL	BLR
8587.710	7759.920

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Distance	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Hand	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	2.000	3.000	3.000	3.000	3.000		
Distance	2.000	1.000	1.000	2.000	2.000		
Hand	2.000	1.000	2.000	1.000	2.000		

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	1.49304E+07	2	7465200.038	4.899	0.020	0.029	0.021
Error	2.74265E+07	18	1523695.738				
Greenhouse-Geisser Epsilon:			0.8122				
Huynh-Feldt Epsilon :			0.9656				
Distance	3.40888E+08	1	3.40888E+08	93.571	0.000	.	.
Error	3.27879E+07	9	3643104.091				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	9780659.008	1	9780659.008	0.830	0.386	.	.
Error	1.05994E+08	9	1.17771E+07				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Condition							
*Distance	203229.833	2	101614.917	0.169	0.846	0.844	0.846
Error	1.08263E+07	18	601459.775				
Greenhouse-Geisser Epsilon:			0.9919				
Huynh-Feldt Epsilon :			1.0000				
Condition							
*Hand	711296.722	2	355648.361	0.397	0.678	0.623	0.651
Error	1.61056E+07	18	894755.541				

Greenhouse-Geisser Epsilon: 0.7532
Huynh-Feldt Epsilon : 0.8717

Distance						
*Hand	61934.720	1	61934.720	0.056	0.818	. .
Error	9942794.716	9	1104754.968			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .

Condition							
*Distance							
*Hand	739279.777	2	369639.889	1.033	0.376	0.372	0.376
Error	6439143.911	18	357730.217				

Greenhouse-Geisser Epsilon: 0.9195
Huynh-Feldt Epsilon : 1.0000

Multivariate Repeated Measures Analysis

Test of: Condition		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.460	2	8	4.699	0.045
Pillai Trace =	0.540	2	8	4.699	0.045
H-L Trace =	1.175	2	8	4.699	0.045

Test of: Condition *Distance		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.965	2	8	0.143	0.869
Pillai Trace =	0.035	2	8	0.143	0.869
H-L Trace =	0.036	2	8	0.143	0.869

Test of: Condition *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.907	2	8	0.411	0.676
Pillai Trace =	0.093	2	8	0.411	0.676
H-L Trace =	0.103	2	8	0.411	0.676

Test of: Condition *Distance *Hand		Hypoth. df	Error df	F	P
Wilks' Lambda=	0.801	2	8	0.996	0.411
Pillai Trace =	0.199	2	8	0.996	0.411
H-L Trace =	0.249	2	8	0.996	0.411

Blocking Effects: Antagonist Biceps Onset

SAT 7/20/02 1:15:37 AM

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Welcome to SYSTAT!
IMPORT successfully completed.
Number of cases processed: 10
Dependent variable means

NSLU	NSLB	NSRU	NSRB	NLLU
85.800	69.550	88.130	98.860	114.100

NLLB	NLRU	NLRB	ESLU	ESLB
92.720	118.980	119.670	72.470	69.370

ESRU	ESRB	ELLU	ELLB	ELRU
71.080	73.250	120.060	121.270	120.830

ELRB
119.710

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	1.000	1.000	1.000
Distance	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Hand	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Blocking	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	1.000	2.000	2.000	2.000	2.000	2.000	2.000
Distance	2.000	1.000	1.000	1.000	1.000	2.000	2.000
Hand	2.000	1.000	1.000	2.000	2.000	1.000	1.000
Blocking	2.000	1.000	2.000	1.000	2.000	1.000	2.000
Within factor							
Condition	2.000	2.000					
Distance	2.000	2.000					
Hand	2.000	2.000					
Blocking	1.000	2.000					

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	244.283	1	244.283	0.912	0.365	.	.
Error	2410.409	9	267.823				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Distance	55812.106	1	55812.106	88.793	0.000	.	.
Error	5657.064	9	628.563				
Greenhouse-Geisser Epsilon:			.				
Huynh-Feldt Epsilon :			.				
Hand	2654.456	1	2654.456	5.990	0.037	.	.
Error	3988.324	9	443.147				

Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Blocking	457.314	1	457.314	1.608	0.237	.	.
Error	2560.115	9	284.457				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Condition							
*Distance	5355.753	1	5355.753	9.658	0.013	.	.
Error	4990.636	9	554.515				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Condition							
*Hand	2384.708	1	2384.708	9.266	0.014	.	.
Error	2316.331	9	257.370				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Condition							
*Blocking	402.273	1	402.273	1.785	0.214	.	.
Error	2028.366	9	225.374				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Distance							
*Hand	5.968	1	5.968	0.013	0.910	.	.
Error	4016.624	9	446.292				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Distance							
*Blocking	125.139	1	125.139	0.472	0.510	.	.
Error	2387.543	9	265.283				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Hand							
*Blocking	1689.350	1	1689.350	5.202	0.049	.	.
Error	2922.982	9	324.776				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Condition							
*Distance							
*Hand	7.526	1	7.526	0.046	0.836	.	.
Error	1482.891	9	164.766				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Condition							
*Distance							
*Blocking	163.823	1	163.823	0.524	0.487	.	.
Error	2811.714	9	312.413				
Greenhouse-Geisser Epsilon:						.	
Huynh-Feldt Epsilon :						.	
Condition							
*Hand							
*Blocking	1328.833	1	1328.833	9.254	0.014	.	.

Error	1292.324	9	143.592			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Distance						
*Hand						
*Blocking	97.813	1	97.813	0.796	0.396	. .
Error	1105.932	9	122.881			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Distance						
*Hand						
*Blocking	4.523	1	4.523	0.025	0.878	. .
Error	1630.682	9	181.187			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				

Blocking Effects: Biceps Offset

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Dependent variable means

NSLU	NSLB	NSRU	NSRB	NLLU
152.010	141.910	156.930	161.830	190.810

NLLB	NLRU	NLRB	ESLU	ESLB
166.410	182.140	191.180	142.680	132.020

ESRU	ESRB	ELLU	ELLB	ELRU
135.260	134.720	193.750	199.840	191.680

ELRB
182.760

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	1.000	1.000	1.000
Distance	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Hand	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Blocking	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	1.000	2.000	2.000	2.000	2.000	2.000	2.000
Distance	2.000	1.000	1.000	1.000	1.000	2.000	2.000
Hand	2.000	1.000	1.000	2.000	2.000	1.000	1.000
Blocking	2.000	1.000	2.000	1.000	2.000	1.000	2.000
Within factor							
Condition	2.000	2.000					
Distance	2.000	2.000					
Hand	2.000	2.000					
Blocking	1.000	2.000					

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	581.788	1	581.788	1.340	0.277	.	.
Error	3906.177	9	434.020				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Distance	72765.165	1	72765.165	253.251	0.000	.	.
Error	2585.922	9	287.325				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Hand	182.116	1	182.116	0.300	0.597	.	.
Error	5465.161	9	607.240				

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .

Blocking	747.793	1	747.793	1.721	0.222	. .
Error	3911.032	9	434.559			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Condition

*Distance	6955.088	1	6955.088	10.009	0.011	. .
Error	6254.032	9	694.892			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Condition

*Hand	2625.210	1	2625.210	4.966	0.053	. .
Error	4757.699	9	528.633			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Condition

*Blocking	26.651	1	26.651	0.090	0.771	. .
Error	2660.891	9	295.655			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Distance

*Hand	335.531	1	335.531	0.579	0.466	. .
Error	5211.656	9	579.073			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Distance

*Blocking	2.003	1	2.003	0.002	0.963	. .
Error	7730.217	9	858.913			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Hand

*Blocking	1185.377	1	1185.377	1.556	0.244	. .
Error	6855.328	9	761.703			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Condition
*Distance

*Hand	20.235	1	20.235	0.061	0.811	. .
Error	3004.159	9	333.795			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Condition
*Distance

*Blocking	214.601	1	214.601	0.327	0.581	. .
Error	5905.101	9	656.122			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Condition
*Hand

*Blocking	1777.556	1	1777.556	6.622	0.030	. .
Error	2415.871	9	268.430			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Distance
*Hand

*Blocking	27.973	1	27.973	0.092	0.769	. .
Error	2742.537	9	304.726			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .
Condition
*Distance
*Hand

*Blocking	1186.466	1	1186.466	2.575	0.143	. .
Error	4147.161	9	460.796			

Greenhouse-Geisser Epsilon: .
Huynh-Feldt Epsilon : .

Blocking Effects: Movement Distance

Comparison of the distance (and movement times) of normal limb movements and movements of the unblocked limb of a blocked partner.

ANOVA Condition Code Legend

M***	B***	- Unequal (Mixed) distance movement	- Bimanual (equal) distance movement
*S**	*L**	- Short distance movement	- Long distance movement
**L*	**R*	- Left hand data	- Right hand data
***U	***B	- Unblocked Movement Pair	- Unblocked Movement Pair

```
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Dependent variable means
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MSLU	MSLB	MSRU	MSRB	MLLU
14.440	15.410	14.270	15.600	49.880

MLLB	MLRU	MLRB	BSLU	BSLB
50.490	48.880	49.670	13.080	13.420

BSRU	BSRB	BLLU	BLLB	BLRU
11.610	11.970	49.460	51.320	47.460

BLRB
49.110

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	1.000	1.000	1.000
Distance	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Hand	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Blocking	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	1.000	2.000	2.000	2.000	2.000	2.000	2.000
Distance	2.000	1.000	1.000	1.000	1.000	2.000	2.000
Hand	2.000	1.000	1.000	2.000	2.000	1.000	1.000
Blocking	2.000	1.000	2.000	1.000	2.000	1.000	2.000
Within factor							
Condition	2.000	2.000					
Distance	2.000	2.000					
Hand	2.000	2.000					
Blocking	1.000	2.000					

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	78.540	1	78.540	32.515	0.000	.	.
Error	21.739	9	2.415				

Condition	78.540	1	78.540	32.515	0.000	. .
Error	21.739	9	2.415			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Distance	51290.663	1	51290.663	1699.421	0.000	. .
Error	271.631	9	30.181			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Hand	49.841	1	49.841	4.031	0.076	. .
Error	111.291	9	12.366			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Blocking	39.105	1	39.105	11.158	0.009	. .
Error	31.542	9	3.505			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Distance	40.703	1	40.703	4.834	0.055	. .
Error	75.776	9	8.420			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Hand	17.756	1	17.756	3.897	0.080	. .
Error	41.001	9	4.556			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Blocking	0.163	1	0.163	0.117	0.740	. .
Error	12.529	9	1.392			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Distance						
*Hand	6.123	1	6.123	1.539	0.246	. .
Error	35.804	9	3.978			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Distance						
*Blocking	2.280	1	2.280	0.466	0.512	. .
Error	44.012	9	4.890			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Hand						
*Blocking	0.077	1	0.077	0.010	0.921	. .
Error	66.713	9	7.413			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						

*Distance							
*Hand	0.189	1	0.189	0.046	0.835	.	.
Error	36.993	9	4.110				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Condition							
*Distance							
*Blocking	8.603	1	8.603	1.107	0.320	.	.
Error	69.954	9	7.773				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Condition							
*Hand							
*Blocking	0.333	1	0.333	0.087	0.774	.	.
Error	34.351	9	3.817				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Distance							
*Hand							
*Blocking	0.105	1	0.105	0.017	0.898	.	.
Error	54.084	9	6.009				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Condition							
*Distance							
*Hand							
*Blocking	0.002	1	0.002	0.000	0.988	.	.
Error	57.393	9	6.377				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					

Blocking Effects: Movement Time

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Number of cases processed: 10
Dependent variable means

MSLU	MSLB	MSRU	MSRB	MLLU
175.400	178.990	175.330	182.450	301.690

MLLB	MLRU	MLRB	BSLU	BSLB
289.250	286.580	279.230	165.990	172.280

BSRU	BSRB	BLLU	BLLB	BLRU
154.700	156.970	254.650	245.310	250.420

BLRB
251.080

Repeated measures factors and levels

	Dependent Variables						
Within factor							
Condition	1.000	1.000	1.000	1.000	1.000	1.000	1.000
Distance	1.000	1.000	1.000	1.000	2.000	2.000	2.000
Hand	1.000	1.000	2.000	2.000	1.000	1.000	2.000
Blocking	1.000	2.000	1.000	2.000	1.000	2.000	1.000
Within factor							
Condition	1.000	2.000	2.000	2.000	2.000	2.000	2.000
Distance	2.000	1.000	1.000	1.000	1.000	2.000	2.000
Hand	2.000	1.000	1.000	2.000	2.000	1.000	1.000
Blocking	2.000	1.000	2.000	1.000	2.000	1.000	2.000
Within factor							
Condition	2.000	2.000					
Distance	2.000	2.000					
Hand	2.000	2.000					
Blocking	1.000	2.000					

Univariate and Multivariate Repeated Measures Analysis

Within Subjects

Source	SS	df	MS	F	P	G-G	H-F
Condition	29571.844	1	29571.844	30.574	0.000	.	.
Error	8705.017	9	967.224				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Distance	396109.506	1	396109.506	68.456	0.000	.	.
Error	52076.730	9	5786.303				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Hand	1368.900	1	1368.900	1.292	0.285	.	.
Error	9538.369	9	1059.819				

Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Blocking	52.900	1	52.900	0.086	0.776	. .
Error	5526.761	9	614.085			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Distance	5412.602	1	5412.602	3.496	0.094	. .
Error	13934.309	9	1548.257			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Hand	6.889	1	6.889	0.036	0.854	. .
Error	1729.965	9	192.218			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Blocking	50.176	1	50.176	0.347	0.571	. .
Error	1302.865	9	144.763			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Distance						
*Hand	0.090	1	0.090	0.000	0.990	. .
Error	4595.653	9	510.628			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Distance						
*Blocking	1424.442	1	1424.442	1.173	0.307	. .
Error	10926.619	9	1214.069			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Hand						
*Blocking	133.225	1	133.225	2.048	0.186	. .
Error	585.494	9	65.055			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Distance						
*Hand	2006.472	1	2006.472	5.442	0.045	. .
Error	3318.247	9	368.694			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				
Condition						
*Distance						
*Blocking	109.892	1	109.892	0.343	0.572	. .
Error	2882.019	9	320.224			
Greenhouse-Geisser Epsilon:		.				
Huynh-Feldt Epsilon :		.				

Condition							
*Hand							
*Blocking	4.356	1	4.356	0.040	0.845	.	.
Error	969.433	9	107.715				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Distance							
*Hand							
*Blocking	151.710	1	151.710	1.222	0.298	.	.
Error	1117.223	9	124.136				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					
Condition							
*Distance							
*Hand							
*Blocking	97.032	1	97.032	0.792	0.397	.	.
Error	1102.082	9	122.454				
Greenhouse-Geisser Epsilon:		.					
Huynh-Feldt Epsilon :		.					

Appendix C: Table of Perturbation Studies

A summary of motor control and perturbation studies in eight columns:

- Reference: citation reference in APA reference style.
- Subjects: details about test subjects, species, sex, age where possible.
- Movement: description of test conditions, limb or joint being investigated.
- Perturbation: nature of applied perturbation or load, with stated values.
- Probability: stated probability or ratio of trials with applied perturbation or load.
- Onset: timing and nature of perturbation application.
- Duration: duration of perturbation, details of perturbation release.
- Conclusions: summary of study results, stated or implied predictions, references to other studies with similar or contrasting results.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Henry, M. H. (1953). Dynamic kinesthetic perception and adjustment. Research Quarterly, 24, 176-187.	12 humans.	Hand pressing against moving plate. 1) Constant position 2) Constant pressure	Irregular changes in elastic force-position.	Constant	(continuous)	15 seconds of irregular cam rotation.	1) Constant position: average pressure error was 0.065 lbs. 2) Constant pressure: average response error of 0.71 lbs, with perception threshold of 1.25 lbs. 3) Sub-threshold corrections must be attributed to reflexive responses.
Merton, P. A. (1953). Speculations on the servo-control of movements. In J.L. Malcolm, J.A.B. Gray & G.E.W. Wolstenholm (Eds.), The Spinal Cord (pp. 247-260). Boston: Little, Brown.	1 human (minimum).	~1 kg isometric force production of thumb, adductor pollicis muscle.	Electrical stimulation of ulnar nerve: 1. Threshold 2. Super-threshold.	(unknown)	During isometric steady force production (~1 kg).	(unknown)	1) Muscle spindle, detecting muscle length changes, acts as sensor for a negative feedback servo loop controlling muscle length: Stretch Reflex. 2) Steady voluntary or posture effort set intrafusal fiber length (through small motoneuron), which adjusts main muscle length through stretch reflex loop. 3) Faster direct feedforward control is available by direct impulses to muscle through large motor nerve (bypassing spindle-reflex loop).
Angel, R. W. (1977). Agonist muscle activity during rapid arm movements: central versus proprioceptive influences. Journal of Neurology, Neurosurgery, and Psychiatry, 40, 683-686.	7 humans 6 male, 1 female, 21-59 yrs.	Shoulder flexion of suspended 750 mm aluminum rod at arm's length through 15 cm (11.3 degrees).	Isometric movement blocking with electromagnet.	50% of trials.	Trial onset.	Full Trial.	1) First agonist EMG was nearly identical for unblocked and blocked movements. 2) Antagonist EMG was significantly reduced (or silent) in blocked movements, compared to unblocked movements. 3) EMG patterns result from interaction of central program with perceptual feedback: this accounts for trials with partial antagonist EMG patterns.
Bizzi, E., Dev, P., Morasso, P., Polit, A. (1978). Effect of load disturbances during centrally initiated movements. Journal of Neurophysiology, 41, 542-556.	3 Macaque monkeys, vestibulect omized, and Cervical Rhizotomy (C1-T3).	30-40 degrees head rotation left and right.	0.035-0.12 Nm opposing torques on head/neck.	5-10% of trials.	At measurable EMG onset.	400-800 ms.	1) Corrective torque contribution of reflexes was only 10-30% of required torque. 2) Combined mechanical and reflexive torques never fully compensated for perturbations.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Hallett, M., Marsden, C. D. (1979). Ballistic flexion movements of the human thumb. Journal of Physiology, 294, 33-50.	2 humans (minimum).	Flexion of distal thumb joint 5, 10, 20 degrees from flexion angles of 10, 20, 30 degrees.	Constant, increasing, decreasing opposing torques, 0.04, 0.08, 0.16 Nm.	(unknown)	At acoustic 'click' stimulus or 25, 40, 55, 65 ms after agonist EMG onset.	(unknown)	1) Rectified and integrated agonist EMG correlated with the distance traveled. 2) Opposing stretch perturbations increased agonist EMG amplitude, while assisting release decreased EMG amplitude. 3) Stretch or halt occurring between agonist bursts augmented the second agonist burst (onset time, amplitude, duration). 4) Stretch occurring between agonist bursts diminished antagonist EMG activity, while stop eliminated it. Release increased antagonist EMG. 5) Timing of EMG bursts is fixed by CNS, feedback modifies EMG response to perturbations.
Polit, A., and Bizzi, E. (1979). Characteristics of motor programs underlying arm movements in monkeys. Journal of Neurophysiology, 42, 183-194.	3 Macaque monkeys, training before and after Dorsal Rhizotomy (C2-T3).	Elbow aiming movements up to 70 degrees (from resting position) to a 12 degree wide target	Constant assisting or opposing torques (unknown magnitude)	20% of testing trials.	150-200 ms before movement initiation (after target presentation).	100-180 ms.	1) After bilateral dorsal rhizotomy trained monkeys were able to aim accurately at target even if arm briefly perturbed (under no vision conditions). 2) Constant opposing or assisting loads offset movement endpoint, on load removal target was attained in prepared animals. 3) Shifting elbow position disrupted aiming (elbow angle was maintained), intact monkeys were able to compensate.
Wadman, W. J., van der Gon, J. J., Geuze, R. H., Mol, C. R. (1979). Control of fast goal-directed arm movements. Journal of Human Movement Studies, 5, 3-17.	7 human males.	7.5, 15, 22.5, 30 cm linear movement.	1) Increased/ decreased inertial load. 2) Blocking - isometric.	(unknown)	(unknown)	Full trial.	1) Main pattern of EMG: Triphasic (reciprocal inhibition). 2) First agonist duration increases with increasing distance, generally intensity does not change (Speed Insensitive Strategy). 3) Movement blocking resulted in no differences in EMG activity for first 100 ms: either stereotypical blocked triphasic EMG, or only single extended agonist burst. 4) Muscle motor time (MT) not reported, use of passive perturbation caused confusion: 50 ms MT + 50 ms Stretch Reflex = 100 ms delay. 5) For real delayed reflex response see Brown & Cooke (1981).

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Bizzi, E. (1980). Central and peripheral mechanisms in motor control. In G. Stelmach & J. Requin (Eds.), Tutorials in Motor Behavior (pp. 131-143). New York: North-Holland.	Macaque monkeys, with (neck) Cervical Rhizotomy (C1-T3) and (arm) Dorsal Rhizotomy (C2-T3).	1) Head /neck aiming movements 2) Arm aiming movements.	1) 315 and 726 g-cm torque loads and unknown inertial load. 2) unknown torque loads.	(unknown)	At measurable EMG onset.	Torque load: 100-180 ms. Inertial load: Full Trial.	Additional results to Bizzi et al (1978) and Polit and Bizzi (1979): 1) Increased inertial loads slow overall movement pattern. 2) Increased inertial load slowed overall head aiming movement, reduced peak velocity, extending movement time, but did not affect movement endpoint.
Brown, S. H., Cooke, J. D. (1981). Responses to force perturbations preceding voluntary human arm movements. Brain Research, 220, 350-355.	6 humans.	1 trial = 40 elbow Ext-Flex movements: 48 degree Step Tracking.	3-5 Nm (est.)	40 Ext-Flex movements: % Normal % Assisting % Opposing. (unknown).	30-120 ms before EMG onset (after target presentation).	50 ms.	1) First Agonist burst altered ~100 ms after onset. a. Increased amplitude for opposed movements. b. Decreased amplitude for assisted movements. 2) No reflexive responses elicited for perturbations 30-120 ms before EMG onset, no change seen until after 100 ms of onset: 130-220ms delays (no typical reflex latency of 50-60 ms). 3) Typical reflex latencies seen during hold condition and during movement
Kelso, J. A., Holt, K. G., Rubin, P., Kugler, P. N. (1981). Patterns of human interlimb coordination emerge from the properties of non-linear, limit cycle oscillatory processes: theory and data. Journal of Motor Behavior, 13, 226-261.	12 humans, 6 single hand, 6 bimanual.	50 degree index finger extension-flexion over 9 seconds, ~1.5 Hz.	Exp 1: 40.8 oz-in Exp 1: 20.4 oz-in in flexion direction.	(unknown)	During extension movement.	Experiment 1: 100 ms 2: End of trial.	1) Homeokinetic theory states biological systems as ensembles of non-linear, limit cycle oscillatory processes coupled and mutually entrained at all levels of organization. 2) Perturbation of one hand during continuous movement results in both hands stopping until release of load, short delay in one cycle (100 ms). 3) Smaller constant load disrupted one cycle of perturbed hand, other hand maintained pattern, proper resumption of pattern with no timing delay, some attenuation in perturbed finger, only slightly reduced amplitude in other finger.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Day, B. L. & Marsden, C. D. (1982). Accurate repositioning of the human thumb against unpredictable dynamic loads is dependent upon peripheral feed-back. Journal of Physiology, 327, 393-407.	9 humans 21-29 yrs.	3 degree thumb extension and 10 degree thumb flexion against torque motor.	0.0042 normal, 0.0084, 0.0056, 0.0028 Nsm/rad viscous load.	32 trials: 8 normal, 8 of each other viscous loads.	Trial onset.	Full Trial.	4) No final position error was found with increased viscous friction, but small overshoots found with reduced viscous friction. 5) EMG activity increased with increased viscous friction, EMG decreased with decreased friction. 6) Thumb sensory anaesthetization resulted in undershoot to increased viscous friction, overshoot for reduced viscous friction. 7) First 100 ms of agonist EMG was unaffected by voluntary intervention.
Berardelli, A., Rothwell, J. C., Day, B. L., Kachi, T., Marsden, C. D. (1984). Duration of the first agonist EMG burst in ballistic arm movements. Brain Research, 304, 183-187.	5 of 7 humans	1) 15, 30, 60, 90, 105 degree wrist and elbow flexion.	(none)	(none)	(none)	(none)	1) Short movements (15-30°) have fixed duration, amplitude modulated agonist I bursts (Speed Sensitive strategy: Latash & Gottlieb, 1991a).
		2) 15, 60 degree wrist flexion.	2.2 Nm opposing torque.	Load: 100 % Control: 0% (full knowledge)	(unknown)	Full Trial.	
Al-Senawi, D., Cooke, J. D. (1985) Matching of movements made independently by the two arms in normal humans. Journal of Motor Behavior, 17, 321-334.	6 right handed humans.	Stepped tracking (see Brown & Cooke 1981).	1 lb (0.45 kg) mass worn on left wrist for minimum one week.	(none)	(none)	(none)	1) Both weighted and unweighted arms had increased peak-velocity/amplitude ratios during testing (no weight worn during testing). 2) Provides support for single movement command hypothesis: single movement expressed over two limbs. 3) Interaction of command with limbs with second order mechanical properties yields similar movements even when made independently.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Lee, R. G., Lucier, G. E., Mustard, B. E., White, D. G. (1986). Modification of motor output to compensate for unanticipated load conditions during voluntary movements. Canadian Journal of Neuro Science, 13, 97-102.	20 humans 20-45 yrs.	Wrist flexion of 40 degrees completed within 100 ms.	0.25 Nm up to 1.5 Nm 1.6 Nm down to 0.25 Nm.	< 20% change in opposing load.	Start of trial.	Full Trial	1) Unanticipated increase in opposing torque load resulted in increased agonist activity and decreased antagonist activity. 2) Unanticipated decrease in opposing load resulted in decreased agonist activity, increased antagonist activity. 3) EMG differences seen at 30 ms after movement onset, indicating spinal level mechanisms, rather than long-loop responses.
Flanders, M., Cordo, P. J. (1987) Quantification of peripherally induced reciprocal activation during voluntary muscle contraction. Electroencephalography and Clinical Neurophysiology, 67, 389-394.	4 humans. performing flexion isometric force production.	On visual stimulus increase isometric torque from 3 Nm to 20 Nm.	14 degree, 140 deg/sec forced extension or flexion.	50% Isometric 25% Flexion 25% Extension	At anticipated onset of isometric torque response.	Full Trial	1) Forced flexion movements resulted in reduced agonist activity and increased antagonist activity compared to isometric flexion condition (reciprocal activation: expected). 2) Forced extension increased EMG activity in both agonist and antagonist muscles compared to isometric flexion condition (coactive activation: unexpected).
Forget, R. & Lamarre, Y. (1987). Rapid elbow flexion in the absence of proprioceptive and cutaneous feedback. Human Neurobiology, 6, 27-37.	3 functionally deafferented humans, 3 women, 34, 40, 42 yrs.	Rapid elbow flexion movements of 40 and 90 degrees.	(none)	(none)	(none)	(none)	1) Both deafferented and intact patients produce triphasic EMG patterns. 2) Information from the moving limb is required to adjust the magnitude and onset time of antagonist activity. Deafferented patients tended to overshoot or undershoot due to inadequate antagonist amplitude and timing.
Hayashi, R., Becker, W. J., Lee, R. G. (1990). Effects of unexpected perturbations on trajectories and EMG patterns of rapid wrist flexion movements in humans. Neuroscience Research, 8, 100-113.	5 humans 22-47 yrs.	Wrist flexion of 20 degrees	1.8 Nm opposing or assisting torque.	90% Normal, 10% Perturbed.	60 ms before to 60 ms after EMG onset (selected random trials).	30 ms.	1) Opposing perturbations resulted in greater maximum velocity and target overshoot, while assisting perturbations reduced peak velocity and target undershoot.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Smeets, J. B., Erkelens, C. J., Denier van der Gon, J. J. (1990). Adjustments of fast goal-directed movements in response to an unexpected inertial load. Experimental Brain Research, 81, 303-312.	6 humans.	8/16 cm linear hand pulling motion, 10/20 degree elbow flexion.	0.7, 5, 20 kg inertial masses (through torque motor)	Normal: 80% Changed: 20%	Stepped Tracking: at appearance of stimulus target.	Full Trial.	1) Agonist EMG activity unchanged for 90-110 ms after onset (65-85 ms from movement onset) due to modified inertial load. 2) With added inertial load agonist EMG offset was delayed, antagonist onset was also delayed. With reduced inertial load agonist offset occurred earlier, as did antagonist onset. 3) Latency between load induced velocity differences (>0.6 rad/s) to measurable EMG change was 37 ms (stretch reflex?).
Latash, M. L., Gottlieb, G. L. (1991b) An equilibrium-point model for fast single-joint movement: II. Similarity of single-joint isometric and isotonic descending commands. Journal of Motor Behavior, 23, 179-191.	8 male humans.	20 or 36 degrees right elbow flexion (no accuracy constraints). Manipulandum moment: 0.86 Nms^2/rad.	Movement Blocking-isometric.	25-50%	Start of trial.	Full Trial.	1) Blocked acceleration and velocity profiles diverge from unblocked profiles 40-50 ms after agonist onset (barrier impact: motor time). 2) Blocked EMG patterns diverge after 90-120 ms: agonist EMG amplitude increased, antagonist decreased. 3) In blocked trials one subject showed a 10 Hz, another 20 Hz EMG oscillation not seen in isotonic movements. 4) Left elbow contralateral biceps onset reaction times were 200-260 ms (presumably from agonist onset). 5) Reversal on movement blocking: onset reaction time was 216 ms for 7 subjects, 150 ms for one subject. Marked decrease in extensor antagonist amplitude before reversal burst (beginning of single agonist burst?).
				Three experimental series: 1) No extra response. 2) Flex left elbow on right arm blocking (Contralateral Response) 3) Extend right elbow on flexion movement blocking.			
Gottlieb, G. L. (1994) The generation of the efferent command and the importance of joint compliance in fast elbow movements, Experimental Brain Research, 97, 545-550.	1 human.	36 degree elbow flexion to a 3 degree target. Manipulandum and limb inertia: 0.16 $Nm.s^2/rad$ (estimated)	Elastic loads: 0.1, 0.2, 0.4 Nm/deg	11 normal trials. 10 large, 10 small, 50 medium trials randomized.	At onset of movement	Full trial.	1) EMG patterns change in well defined manner when load is known in advance. 2) During an unexpected load change, EMG pattern changes are smaller and later, while trajectory perturbations are larger. 3) No large effects seen, joint compliant properties minimized effects of external load on trajectory. 4) "No evidence for a *large* contribution by length-sensitive stretch reflexes to this process".
			Inertial loads: -0.057, 0, +0.171 $Nm.s^2/rad$	Same, using inertial loads.	At onset of measurable velocity.	Full trial.	

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Latash, M. L. (1994). Control of fast elbow movement: a study of electromyographic patterns during movements against unexpected decreased inertial load. Experimental Brain Research, 98, 145-152.	5 humans, 29-51 yrs.	40 degree elbow flexion (130 to 90 degrees) Manipulandum inertia: 0.086 Nm s^2 rad.	40% decrease in manipulandum inertia: Unload (via torque motor) or Unperturbed.	Series 1: 6 unloaded, 6 unperturbed Series 2: all unloaded.	Beginning of movement.	Full Trial.	1) No differences in antagonist burst latency in *unexpectedly* unloaded and perturbed trials. 2) Decrease in antagonist latency during *expected* unloadings. 3) Significant increase in antagonist EMG integral in unexpectedly unloaded trials compared to unperturbed trials. 4) Support for equilibrium point hypothesis assuming central control of antagonist onset, and reflex-induced EMG amplitudes.
Feldman, A. G., Adamovich, S. V., Levin, M. F. (1995). The relationship between control, kinematic and electromyographic variables in fast single-joint movements in humans. Experimental Brain Research, 103, 440-450.	7 humans, 1 woman, 6 men. 22-55 yrs.	55-70 degree elbow flexion to 6 degree target. Manipulandum inertia: 0.03 kg-m^2 (est.)	Elastic loads: 12-20 Nm/$deg^{1/2}$ (per subject). Dampening value 0.01-0.03 Nms/deg.	30-40% of testing trials had elastic load.	After leaving 1 degree start window.	Full trial, Equifinality test: released 100 ms after velocity < 30 deg/s.	1) Fast EP shifts (600 deg/s) are completed at around time of peak velocity for unperturbed movements. Isometric blocked muscles act as force generators: end of EP shift reflected in force stabilization. 2) In high opposing load condition antagonist burst suppressed. 3) Kinematic and EMG patterns represent a long lasting response of system to the short-duration monotonic control pattern, external forces and proprioceptive feedback. • *Prediction*: For long distance movements, braking can be prepared online, resulting in minimal-simple RT. Short movements need to be totally preplanned meaning longer RT. In a short/long choice RT experiment both long and short movement will have extended RT's, while long/long RT would have minimal-simple RT.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Gomi, H., & Kawato, M. (1996). Equilibrium-point control hypothesis examined by measured arm stiffness during multi-joint movement. Science, 272, 117-120.	3 humans, 1 female, 2 male. 26-34 yrs.	Left to right movements in front of body. Manipulandum Mass: 0.65 kg. Inertia: 4.4 N/(m/s).	Small force perturbations measuring limb stiffness.	(unknown)	Various positions throughout movement.	0.2 seconds.	1) High shoulder and elbow stiffness (67.9 and 78.0 Nm/rad) indicate equilibrium point shift is monotonic. 2) Low shoulder and elbow stiffness (19.5 and 15 Nm/rad) indicate equilibrium point shift is very complex to initiate and end movement. 3) Measured elbow stiffness: 5-21 Nm/rad, indicating an 'N' shaped EP profile: first leading to accelerate, then lagging to decelerate limb. -Stiffness values challenged by Ghafouri & Feldman (2001)
Gottlieb, G. L. (1996). On the voluntary movement of compliant (inertial-viscoelastic) loads by parcellated control mechanisms. Journal of Neurophysiology, 76, 32073229.	19 humans (total) 14 Male 5 Female 17-52 yrs.	36 or 54 degree flexion (from 120 degree extension) to 3 degree target. Manipulandum Inertia: 0.086 kg m^2 Limb Inertia: 0.095 kg m^2, Total: M = 0.16 Nms^2/rad.	Three load types: **Elastic:** K = 0, 12, 24 Nm/rad. **Viscous:** B = 0, 1.3, 1.4, 2.61, 2.87, 3.14 Nms/rad. **Inertial:** M = 0.13, 0.30, 0.57, 0.90 Nms^2/rad.	1) 80% heavier, 20% lighter. 2) 80% lighter, 20% heavier.	100 ms after stimulus tone. (Change would not be noticed until torque applied).	Full Trial.	1) With larger loads, subjects increase torque duration (maintaining amplitude). Subjects modulate torque most for inertial loads, less for viscous loads, least for elastic loads. Predictable conditions result in greater modulation. 2) Predictable Conditions: slower movements for large inertial and viscous loads than small loads, no difference for large or small elastic loads. Unpredictable conditions: largest changes in movement kinematics. 3) Predictable loads that slow movement delay antagonist onset; only unpredictable inertial loads affect antagonist onset. 4) Muscle viscous properties cause initial changes in muscle forces due to unexpected changes in load, reflex responses are seen 50-70 ms later. 5) Three element control: feedforward α specifies muscle activation patterns, reference λ controls reflex feedback loop, γ modulates degree and manner of multiple reflex mechanisms contribute to system stability.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Nougier, V., Bard, C., Fleury, M., Teasdale, N., Cole, J., Forget, R., Paillard, J., Lamarre, Y. (1996). Control of single-joint movements in deafferented patients: evidence for amplitude coding rather than position control. Experimental Brain Research, 109, 473-482.	Two deafferented patients, and 10 normal control subjects.	Forearm supination - pronation:	Isometric braking.	(unknown)	Isometric: brake applied on presentation of first target	Isometric: brake released on presentation of second target.	1) Supination-pronation movement away and back to origin: deafferented patients systematically produced second (unnecessary) pronation movement when first supination prevented
		1) 60 degrees isotonic supination-pronation. 2) 15-80 degrees Isotonic supination-pronation 3) 20 degree supination, 20 degree pronation. Isometric movement blocking of first supination movement.					
Weeks, D. L., Aubert, M. P., Feldman, A. G., Levin, M. F. (1996). One-trial adaptation of movement to changes in load. Journal of Neurophysiology, 75, 60-74.	6 humans: 4-Female 2-Male 20-40 yrs.	60 degree elbow flexion	Spring like: 4.2-120 Nm at target.	Load switched every 5-10 trials: 1) Opposing/ Normal 2) Assisting/ Normal 3) Random alternating Opposing/ Normal/Assisting.	After leaving 1 degree start window.	2 seconds, then gradual reduction.	1) In first perturbed trial 94% of primary movements were in error, followed by a corrective secondary movement. Opposing loads produced undershoots while assisting loads produced overshoots. 2) Secondary trials with same perturbations were 63% successful: one trial adaptation was occurring. 3) Removal of perturbing force resulted in opposite error production.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
Adamovich, S. V., Levin, M. F., Feldman, A. G. (1997). Central modifications of reflex parameters may underlie the fastest arm movements. Journal of Neurophysiology, 77, 1460-1469.	6 humans: 25-55 yrs.	50-70 degrees elbow flexion.	10-15 Nm	33% Normal, 33% Assisting, 33% Opposing.	50 ms after stimulus.	50 ms	1) Greater peak velocity in opposing torque condition, similar amplitude but later in assisting torque condition compared to normal. 2) Decreased biceps EMG onset for opposing torque (111 ms), assisting torque (148 ms), than normal condition (201 ms). 3) Earlier agonist EMG onset seen with opposing load compared to EMG onset with assisting load (predicted). 4) Normal agonist EMG onset was later than both assisting and opposing torque conditions (not predicted: triggered response?). 5) Early antagonist burst seen at 36 ms after perturbation onset (86 ms after stimulus) in both assist and normal trials. Subjects actively held start position in space, no starting barrier.
St-Onge, N., Adamovich, S. V., Feldman, A. G. (1997). Control processes underlying elbow flexion movements may be independent of kinematic and electromyographic patterns: experimental study and modeling. Neuroscience, 79, 295-316.	9 humans, 2 female, 7 male, 22-55 yrs. Manipulandum moment: 0.05 kg m^2	1) 20,40,60,80 elbow flexion. 2) Self pace moderate speed. 3) Additional inertial mass. 4) 60 degree flexion.	Expt. 4): Spring like load ($L=kx^{1/2}$) (Linear loads induced oscillation)	Expt. 4) 30-40% of trials. Instruction: Do not make corrections if the movement ended outside the target window	Movement onset.	During movement, 100-150 ms hold period, 50 ms unloading time.	Five tests of theory and model: 1) Shifts in R (EP) command are fast and monotonic. 2) Monotonic EP shift speeds are different for different movement speeds, but independent of movement distance. (EP profile not long duration multi-phasic, or N-shaped). 3) Distances are encoded by duration of shift in equilibrium state. 4) Subjects produce same control patterns despite EMG and kinematic activity elicited by strong load perturbations. 5) Antagonist burst suppression seen in 60 degree blocked movement, burst appears when unloaded.

Reference	Subjects	Movement	Perturbation	-Probability	-Onset	-Duration	Conclusions
MacKinnon, C. D., & Rothwell, J. C. (2000). Time-varying changes in corticospinal excitability accompanying the triphasic EMG pattern in humans. Journal of Physiology, 528, 633-645.	9 humans: 7 Male, 2 Female, 27-45 years.	20 degree wrist extension or flexion.	Biphasic Transcranial Magnetic Stimulation (TMS) at 80% and 110% of resting threshold	80% Normal trials 20% TMS trials.	80-320 ms after audio stimulus cue.	(TMS pulse duration)	1) TMS pulses evoked EMG patterns approximately 10 ms earlier in agonist muscles but not in antagonist muscles as seen in MEP:IEMG time-integration ratio (also seen in evoked H reflexes). 2) Five of nine subjects showed similar TMS shifts in agonist 2 activity. 3) Changes in motor cortical excitability mediating movement initiation occur about at 23 ms before EMG onset rather than previously reported 80-100ms.
Ghafouri, M., & Feldman, A.G. (2001). The timing of control signals underlying fast point-to-point arm movements. Experimental Brain Research, 137, 411-423.	4 humans.	~30 cm reaching, 3 targets. (~60 degrees elbow) plus secondary movement. Elbow and shoulder rotation.	Isometric	33% Blocking.	At stimulus.	1.5 seconds	1) In isometric condition EP shift is seen in muscle activation level. 2) EP shifts are fast monotonic shifts (600d/s), completed around time of peak velocity in isotonic movements. 3) Onset of secondary movement only occurs after steady state achieved at ~275 ms (earlier in isometric condition). 4) Secondary movement onset latency may be partly due to decreased probability of blocked trials.