Formation and Control of Trajectory during Multijoint Arm Movements in Duchenne's Muscular Dystrophy

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DEDICATIONS

There are many that this work should be dedicated to such as David Lanon who encouraged me to try engineering as a career. Dr. Joseph Heed a teacher, mentor, and friend who has given a helping hand at every step. Also, Casey Albigese a life long devoted family friend who was always there for me since childhood. However, there is one person above all who stands out and that is my wife Jackie. She endured several years of separation without complaint always expressing commitment and support therefore, with a profound gratefulness and love this work is dedicated to her.

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ABSTRACT

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A number of neuropathologies such as Duchenne's muscular dystrophy (DMD), cause disability in the upper extremity due to the loss of muscle strength. This will eventually prevent the individual from being able to move their arm in three-dimensional space so it has been proposed that a robotic orthosis could support and augment movement. This orthosis would need to accommodate the movement capabilities of the user. To accomplish this knowledge of how movements are formed and controlled in the presence of neuromuscular disease need to be determined. For this reason, the formation and control of pointing movements in the horizontal plane made by subjects with DMD are examined.

While the arm was supported in a floatation device, DMD subjects were asked to make pointing movements to various targets from two start positions with trunk movement constrained and unconstrained. The trajectories formed in DMD had essentially straight hand paths that did not necessarily improve with the additional degrees of freedom trunk movement allowed. There is evidence that a hierarchy exists in the kinematic parameters based on the extent of degradation in each feature. The hand paths remain essentially straight at a cost to the other variables, hand velocity profiles improve in modality from constrained to unconstrained configuration, and there is little to no improvement in measures of hand path straightness or the linearity of the joint angular velocity ratio between configurations. The linearity of the joint angular velocity ratio was found to decay at a linear rate related to manual muscle tests.

CHAPTER 1: INTRODUCTION

Several neuropathologies such as Duchenne's muscular dystrophy (DMD), Central cord syndrome, brachial plexus injuries, and poliomyelitis cause disability in the upper extremity due to the loss of muscle strength. As these diseases/injuries advance the individual becomes increasingly incapacitated and therefore reliant on assistance to perform most activities of daily living. If residual muscle power can be augmented by way of a robotic orthosis that supports and actively propels the arm, greater independence for a longer duration can be achieved. Such an orthosis would require knowledge of the desired hand trajectory from initial-to-final position, joint orientations along the trajectory, as well as initiation and termination of movement. Moreover, such orthoses should function as 'naturally' as possible within the physiological limits of the individual such that adaptation for weakness must be considered. Since such a device would be attached directly to the user therefore, control commands from the user must safeguard the user from injury or malfunction. Understanding the effects DMD has on trajectory formation and the consequences of employing compensation mechanisms would be a step towards the goal of realizing a robotic orthosis.

Voluntary integrated muscular activity is necessary to produce a desired point-to-point hand movement if trunk movement is restricted. Because the force-generating capabilities of the affected MD muscle are reduced, these subjects must employ adaptive responses to achieve point-to-point arm movements. Two possible approaches the central nervous system (CNS) could take to achieve this goal are altering muscle activation or system mechanics. Adaptation through muscle activation may take the form of altering muscle contributions via changes in coordination and/or activity level whereas the system

mechanics can be affected by the introduction of another degree of freedom i.e. trunk movement. Motion of the upper body during hand movement could be used to put the joints in an orientation that makes the most effective use of the residual muscle power available. That is, if the CNS exerts motor control over arm motion by specifying trajectory, then the CNS must transform the spatial motor commands into coordinated joint angular patterns by employing muscular forces and/or appropriate compensation mechanisms.

Although the muscle tissues may undergo some morphological changes in neuromuscular diseases, the sensory pathways remain essentially unaffected such that *electromyography* (EMG) could be used as supplemental control input. By studying the underlying EMG activity of point-to-point arm movements the initial muscle activity and agonist selection for a movement can be identified and related to the direction of the desired final position. A consistent partitioning of flexor and extensor initiated movements from rest is observed for each joint with respect to the final position relative to the initial orientation of the forearm (Karst and Hasan, 1991a, b). Therefore, it may be possible to use the EMG as a signal to the orthosis for initiation-termination of movement if this partitioning remains in DMD. To be useful as a possible control input the partitioning of flexor and extensor initiated movements must be present in DMD subjects.

The purpose of this work was to investigate how DMD subjects manage movement execution within the upper extremity musculoskeletal system. Specifically, are compensation mechanisms for the decrease or loss of force production within affected musculature controlled such that the kinematic parameters of the motion are similar to healthy subjects? This work will highlight muscle coordination patterns, strategies, and

biomechanical mechanisms of compensation that are employed by DMD subjects to produce a moment at the elbow and shoulder joints in the transverse plane. Further insight will be gained in the organization of upper extremity motor control and the strategies humans' employ to achieve motion in the presence of local or systemic muscle weakness. Additionally, this work seeks to ascertain whether data supports planning of the movement path in the external or in the joints space. This study determines if established methods based on agonist selection are sufficient for determining the initial direction of the hand movement. To date, clinical assessment of treatment and functional ability in DMD typically are empirically based on such measures as patient responses and manual muscle testing or electromyographic studies. A quantitative measure is needed in order to establish the effectiveness of medical interventions and therapy. Moreover, defining movement parameters in these individuals is a necessary step in the development of control algorithms for an exoskeletal orthosis.

It is therefore hypothesized that in DMD:

A. An empirical method first proposed by Karst and Hasan (1991a, b) is sufficient for determining the direction of the terminal hand position in the transverse plane.
B. A control hierarchy exists where the characteristic of essentially straight hand paths will be conserved but other invariant parameters found in healthy individuals will deteriorate with function in DMD. For example, hand velocity profile and/or the joint angular velocity ratio can be expected to vary yet the hand path will remain essentially straight demonstrating a desire by the CNS to control and execute the planned trajectory.

C. The introduction of another degree of freedom, i.e. trunk translation/rotation will have a positive effect on the kinematic parameters by substituting or standing in for joint rotations not made.

D. Due to pathophysiological changes in muscular structure brought about by the progression of the disease, the ratio of angular velocities in the elbow and shoulder will not always be linear, contrary to results reported for healthy subjects. Because proximal muscles (deltoids) tend to be weaker than distal muscles (brachioradialis) with progression of the disease, it will be easier to propel the forearm about the elbow than the upper arm about the shoulder. Muscles at the shoulder, which are presumed to be weaker, will not only have to manage the inertia of the upper arm but also have to manage force and moment actions transferred due segmental interactions. The result is that the shoulder joint cannot maintain an angular velocity to sustain a linear relationship.

If proven these hypotheses suggest that organization and control of coordinated upper extremity movement in humans has invariable parameters that are maintained so long as movement is physically possible. Such knowledge is required to reduce the number of input requirements to the robotic orthosis. Furthermore, quantitative techniques may result for the assessment of treatment and/or level of function to augment the subjective clinical methods such as manual muscle tests.

CHAPTER 2: BACKGROUND INFORMATION

2.1 Pathophysiology of Duchenne's Muscular Dystrophy

Duchenne's muscular dystrophy (DMD) is an inherited x-linked genetic disease that affects one in approximately every 3,500 male births. The disease primarily affects skeletal and the myocardium muscle causing premature death usually in the early twenties due to respiratory or cardiac failure. Hoffman et al. (1987) and Hutter (1992) characterized the disease as the deficient production of the cytoskeletal structural protein dystrophin associated with the sarcolemma and other transmembrane glycoproteins thereby upsetting intracellular homeostasis resulting in an unstable muscle cell membrane. The result of the missing or non-active protein dystrophin is necrosis of short and/or total segments of fascicular muscle fibers in focal groups, illustrated in Figure 2; the breakdown of myofibrils is triggered by the increased influx of extracellular calcium ions (Horowits et al., 1989).

This imbalance causes the fascicular muscle fibers to go through cycles of cellular necrosis/regeneration with regeneration becoming progressively ineffective. The muscle structure eventually undergoes irreversible degeneration with the subsequent replacement of muscular tissue by fatty as well as connective tissue predominantly consisting of type III collagen in between the muscle fascicles (*cf.* Partridge, 1993; McDonald et. al., 1995; Wang et al., 1999). Given abundance of type III collagen typical of inflammatory fibrosis, Partridge (1993) asserts that lesions in the muscle fascicles are infiltrated by cells that secrete fibrogenic cytokines stimulating collagenous scar tissue. Webster and associates (1988) determined that Type IIb muscle fibers were first to

degenerate/regenerate and attributed this to the lack of dystrophin which is needed to carry out the high frequency contractions demanded by this fiber type.

As the muscle tissue cycles through necrosis-regeneration, the fascicular muscle fibers experience morphological changes i.e. hypertrophy and atrophy. Watkins and Cullen (1982) have shown a mean fiber area increase in DMD muscle with age at a greater rate than in healthy muscle until about age five when it decreases rapidly. Distinctively, Type II muscle fibers tend to be severely reduced and become hypertrophic (Figure 1) in the early stages of DMD while Type I fibers are reduced to 57-82 percent of normal depending on the stage of progression (Buchthal et al., 1971; Partridge, 1993; Wang et al., 1999). The pathological hypertrophy of the Type II fibers in DMD is the result of muscle fiber splitting and not an increase in the myofibrils in addition to the deposition of cellular breakdown products; the increase of substrates secondary to faulty enzymatic pathways; increase in the amount of reactive fat and/or connective tissue in the muscle as

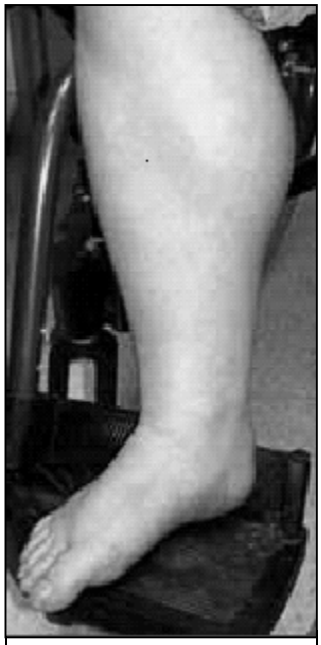


Figure 1: Example of hypertrophy in DMD.

a whole.

Edwards (1980) suggests that as the dystrophic process advances and the muscle fibers available decrease the remaining fibers are subjected to work induced stimuli to muscle fiber hypertrophy and splitting. In a recent study conducted by Wang et al. (1999) the mean diameter of the Type II fibers was reported as 65±21 μm and 48±22 μm for Type I fibers with a variance of 486 and 295 respectively. Other authors have reported different values but of the same order of magnitude (Buchthal et al., 1971; Watkins and Cullen 1982; Coërs and Telerman-Toppet 1977). The authors found that the fiber type disproportion was present by age four and remained unchanged between the ages 4-14 (age period studied by authors). Muscular atrophy later sets in due to denervation, joint contractures preventing muscular contractions and pathological changes within the musculature.

2.1.1 Nerve Fibers in DMD

Notwithstanding these morphological findings, Coërs and Telerman-Toppet (1977) found the density of intramuscular nerves is not reduced. They found that this was the case even in atrophic muscles with a marked reduction in muscle fibers as well as heavy endomysial and perimysial fibrosis. When these researchers followed the individual intramuscular nerves to their end, they found some that formed motor arborizations. Other axons ended in connective tissues with the end being club like or spherical expansion in shape that are small and beaded. Unlike healthy nerve fibers which run a short distance between the intramuscular nerve and the muscle fiber perpendicular to the main direction of the muscle fibers, in DMD they are extended along muscle fibers for a long distance ending several millimeters (up to 10 mm) away from the nerve bundles

producing an abnormal scatter of motor end plates. The resulting aberrant distributions are marked by a longitudinal dispersion of the motor end plates as shown in Figure 2.

The manner in which the disease progresses tends to leave the upper extremity stronger than the lower extremity and with the proximal muscle groups of a limb being weaker than those located more distally. An additional effect is that the *extensor muscle groups* of a limb will tend to be stronger than is their *flexor group counterpart* in the limb. With disease progression and the replacement of muscle fibers with more fatty and connective tissues causing joint contractures thereby limiting the joint's range of motion. Before age nine joint contractures are rare but are present in nearly all DMD individuals by age 13, increasing in both frequency and severity with age. Deep tendon reflex response will also diminish with progression of the disease eventually becoming non-responsive. Degradation within the reflex loop must have an impact on the motor control of the system and the execution of movement.

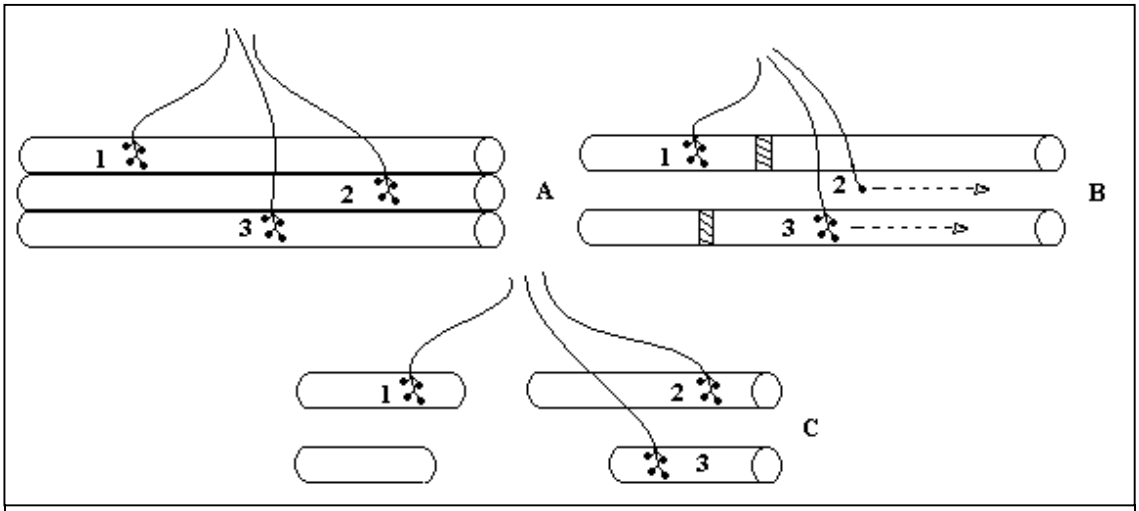


Figure 2: Longitudinal scattering of motor end plates. A represents a normal pattern; B, Necrosis of focal fibers 1 and 3 and total necrosis of fiber 2; C, reinnervation of denervated muscle fiber segment 1 by ectopic end plate of the free axon 2 and a longitudinal displacement of transected fiber 3. *(Adapted from Coërs and Telerman-Toppet, 1977.*

2.1.2 Muscle Force DMD

Horowits et al., (1989) examined single skinned muscle fibers in DMD and discovered no significant change in the fiber's ability to generate active tension in response to calcium, or resting tension in response to stretch. Additionally, these authors found that there were no significant changes in the concentrations of the contractile proteins myosin and actin; elastic protein titin; or the structural proteins nebulin and α-actinin found in fast and slow muscle fibers. The dystrophin protein was found to be absent in the immunocytochemical in DMD however; it was localized at the cell membrane of muscle fibers taken from healthy individuals and from individuals exhibiting severe muscle weakness due to poliomyelitis. These findings suggest that the muscle spindles are still reacting to changes in muscle stretch and still capable of providing efferent feedback in the reflex arc.

It should be noted that these findings contradict those of Wood et al., (1978); these authors reported a decrease in the calcium-activated tension in a population of skinned fibers but this may be due to the fact the fiber types were not separated in this study. Horowits et al. (1989) explains:

"Given the difference in active tension produced by fibers containing fast and slow myosin, the separation of fibers into different types is an important consideration in comparing the disease and control groups. Slow fiber predominance has been reported in DMD (Dubowitz and Brooke, 1973) and may account for the reduction in active fiber tension previously observed by other investigators (Wood et al., 1978)."

In addition, there have been improvements in instrumentation and methodologies since the Wood et al., (1978) study. However, Fick et al., (1990) also reported tension

output from single skinned fibers in DMD is less than in controls. Horowits et al., (1989) go on to explain other qualitative differences between their study and the above other two studies;

"We believe technical differences might account for the discrepancy between the results presented in our report and those obtained by Fink and colleagues. Most importantly, we obtained long, undamaged muscle specimens from all of our patients by open biopsy. In contrast, Fick and Colleagues compared muscle fibers obtained from a control group of unstated age by open biopsy during orthopedic surgery with dystrophic specimens obtained from children by needle biopsy. Because muscle fibers obtained by needle biopsy are relatively short and prone to injury from needle entry, we believe they may be less suitable for quantitative physiological measurements than fibers obtained by open biopsy."

Horowits and colleagues also caution that their results do not have any bearing on the question of whether the activation mechanism is normal in DMD. However, they point out that electromyographic studies of muscle function are consistent with the decrease in the number of muscle fibers without changes to the electrical properties of the cell membranes of surviving fibers (Desmedt et al., 1968). Given these findings, it can be assumed that the absence of dystrophin does not directly interfere with the assembly or force generating function of the contractile apparatus or cell membrane excitability (Horowits et al., 1989). Essentially, the importance here is that the stimulation of the muscle allows for the propagation of the action potential even though the rate is affected as first shown by McComas and Thomas (1968) and described in the following section.

2.1.3 EMG in Duchenne's Muscular Dystrophy

The pathophysiology of DMD must have an effect on the EMG potentials that are produced by affected muscle and its characteristics such as the propagation of the action potentials. EMG has been put to use as an approach for discerning healthy from MD subjects by examining the frequency spectrum and turns of EMG potentials collected with indwelling needle electrodes (Fugsang-Frederiksen et al., 1981, 1985). Panayiotopoulos et al. (1974) used surface EMG electrodes to record an evoked action potential in order to estimate the number of motor units available. Lindeman et al., (1999a) described some changes in the EMG-force characteristics in myotonic MD.

Electrophysiology, muscle activity/synergy, and force during voluntary contractions of dystrophic muscle have been the subject of study among several researchers (McComas and Thomas, 1968; Panayiotopoulos et al., 1974; Hausmanowa-Petrusewcz and Ryniewicz, 1976; Fugsang-Frederiksen, 1981; Fugsang-Frederiksen et al., 1985; Milner-Brown et al., 1986; Martinez and Lõpez-Terradas, 1992; Piotrkiewicz et al., 1993; Rowińska-Marcińska et al., 1997; Kopec, 1997). From this body of research, it is clear that the motor units in DMD are very different from healthy motor units. Some general characteristic differences are smaller EMG potentials having a reduced duration and amplitude with an increase in the number of polyphasic potentials.

Piotrkiewicz and associates (1993) determined there is an increase in the firing rate of the motor unit compared to normal and the increase is more pronounced with the higher force requirements suggesting that the motoneurons are altered in DMD. This contention is later supported by these authors via an examination of the interspike interval of the brachial biceps (Piotrkiewicz et al., (1999). They attribute the alteration of the motor unit

either to the disease itself or to muscular degeneration. This finding is also supported by Martinez and Lopez-Terradas (1992) who suggest that the motor unit remodeling in DMD is mainly myogenic.

McComas and Thomas (1968) found that twitches of dystrophic muscle are relatively slow with approximate contraction time of 92.4 msec and relaxation time of 96.1 ms as compare to normal muscle with times of 63.3 msec and 53.3 msec respectively. Given this, it makes sense that when performing a task that the movement is done at slower rate than in healthy subjects (McDonald et al., 1995). This characteristic could be a result of the severe reduction of fast motor units since it has been shown a muscle's strength correlates to this velocity reduction although weakly (Martinez and Lopez-Terradas, 1992).

Hausmanowa-Petrusewicz and Ryniewicz (1976) demonstrated the disease progression by comparing the EMG potential duration of distal and proximal muscles, i.e. proximal muscles are affected first followed by muscles that are more distal. These authors report mean EMG amplitude of 466-1905 μV for a maximal effort. For evoked potentials using the method of McComas and colleagues (1971), Panayiotopoulos et al. (1974) reported mean amplitudes of 9.32 μV as compared to a mean value for healthy controls of 13.28 μV. The changes in EMG are due to a general loss of active muscle fibers, in other words, the size of each motor unit is reduced, and as a result, the action potentials are smaller. As the disease advances and the muscle fascicles are replaced by fatty and connective tissues so the number of motor units decrease until eventually there may be areas where little if any activity cam be recorded.

2.1.4 Summary

Over time, the disease decreases the patient's functional ability resulting in a need for a wheelchair to maintain mobility, typically by age ten. Eventually the disease also weakens the upper extremity to such an extent that a caregiver's assistance is needed to assist in the performance of most activities of daily living (ADL). In a range of individuals from the age of 3-23 years with a mean age of 13 years the upper extremity strength was reduced to only 4% of a comparable healthy normal group (James and Orr, 1984; McDonald et al., 1995). Before this stage in the progression of the disease is reached, employing muscle coordination strategies and compensation mechanisms can still allow the individual to perform some ADL tasks. However, a DMD affected muscle can deteriorate to a level where it can no longer produce enough force to generate the necessary joint torque for motion to occur. Activation of another muscle or group of muscles within the musculoskeletal system that is capable of compensating for those affected by DMD must be activated. This is especially true if movement is restricted to the arm alone (i.e. no ballistic whipping of the arm via trunk rotations) which leads to the question of how compensation is accomplished, are there strategies that are consistently applied across the population, etc.

2.2 Motor Control and Planning

In healthy individuals, point-to-point arm movements have been described in terms of *spatial hand trajectories* and joint *angular curves* (joint rotations). Morasso, (1981) was first to report that the time course of point-to-point spatial hand trajectories preserve the kinematic characteristic of a roughly straight hand path from initiation to termination of movement. Such movements develop a bell shaped, typically symmetric, unimodal

velocity profile regardless of initial or final position. These facts lead to the hypothesis that the CNS formulates control of these movements in terms of the spatial hand trajectory, termed *spatial motor control* by Morasso, (1981). The spatial motor control thesis implies that other motor control parameters such as joint angles, proprioceptive, exteroceptive, and afferences are subservient to desired hand trajectory in the control hierarchy of arm movement planning and execution.

The predicate for this thesis was Bernstein's (1935) hypothesis that projections of the *Cartesian space* and not projections of *joints and muscles* exist in the higher levels of the central nervous system. The fact that for the same movement there is considerable variance of the hand path in joint space and little variance in hand paths through the external space argues for planning in terms of hand motion. However, contradictory findings have been reported in the literature; for example, Atkeson and Hollerbach (1985) and Soechting and Lacquaniti (1981) found movement trajectories that were curved. In these studies, pointing movements were made in the sagittal plane that were directed outward from the body and terminated near the end of the workspace boundary of the arm without trunk movement. It is suggested by these authors that planned trajectories are constrained by the linearly related (coupled) joint angular velocities towards the end of a spatial pointing movement. Generally, a linear relationship between the joint angular velocities results in the curved hand paths making an argument for planning in terms of joint angles.

2.2.1 Coordinate Transformation

How does the CNS transform a desired trajectory into the desired output? Uno et al., (1989), suggests that it is a three-step process from desire to transformation of visual

coordinates to the desired coordinates of the body followed by generation of the motor commands to motor commands. In other words, coordination and control of multiarticular movement requires the CNS to transform sensory information that resides in its own coordinate frame to the motor output coordinate frame and finally to the coordinates of the external space (*cf.* Saltzman, 1979; Hogan et al., 1987; Soechting and Flanders 1991). These authors also point out that motor and sensory coordinate systems tend to have nonorthogonal axes. The sensory and motor coordinate frames can be defined by their own geometry (i.e. vestibular-ocular) or the musculoskeletal system (i.e. muscular). The direction of stimulus that is most effective in activating peripheral receptors defines the sensory coordinate systems. For example, take the muscle stretch receptors; Pellionisz and Llinás (1980) point out that the direction of force application by the muscles on the skeleton serve as the base vectors of the motor coordinate system. In other words, limb movements require length changes in the musculature; therefore, each muscle length can define axes within a coordinate frame establishing a multidimensional '*muscle space*'.

It is doubtful that the conversion from sensory to motor coordinates takes place in a single step. On the contrary, transformation likely occurs through transitional coordinates that can be embedded in the control circuitry (Hogan et al. 1987). To achieve a desired movement a series of coordinated joint rotations must occur via the actions of muscles on the skeleton. Therefore, a '*joint space*' consisting of all possible joint angles makes up a coordinate system for describing the skeletal kinematics and dynamics. Furthermore, these 'joint space' coordinates must then be transformed into the '*hand space*' coordinates. Considering the kinematic redundancy of the joints and the redundancy of

muscular actions, these transformations are a nontrivial matter. Without scapular motion, the CNS has five degrees of rotation freedom to coordinate during a pointing movement.

Transformations are further complicated by the fact that the relationship between the joint coordinates and the hand coordinates is nonlinear and the mechanical system is indeterminate. An infinite number of postural configurations and muscle force levels are possible to acquire a given target. Greater difficulty arises when the biarticular musculature is considered. Consider the flexion-extension of the elbow where the biceps, brachialis, and brachioradialis muscles act as flexors and the three heads of the triceps act as extensors. Some method for reducing or optimizing the system is required in order for it to be determinate. Moreover, there is the issue of agonist-antagonist coactivation providing no net joint torque. Even though Hogan et al., (1987) provide methods for decreasing this redundancy via impedance and force regulation, clearly there are a number of ways which complicate finding a unique solution.

2.2.2 Kinematic and Kinetic Constraints

Numerous authors have focused their efforts on developing constraints for the mechanical system. Some researchers have examined the kinematic features of single or multijoint arm movement (Morasso, 1981; Abend et al. 1982; Atkeson and Hollerbach 1985; Hollerbach and Atkeson 1987; Kaminski and Gentile 1986; Corradini et al. 1992). Still, others have concentrated their attention on dynamic features of these arm movements (Hollerbach and Flash 1982; Soechting and Flanders 1991; Zajac and Gordon 1989). This body of research has lead to other approaches to CNS motor control modeling which are to optimize a particular movement feature such as smoothness of movement, energy expended, joint torque requirements etc. (Hogan, and Bizzi, 1987;

Hogan and Flash, 1987; Uno et al. 1989; Soechting and Flanders, 1991). To date there has not been one unifying theory on how movement is planned and executed by the CNS, Soechting, and Flanders (1991) postulates it is a misguided pursuit.

Distinguishing between hand and joint based trajectory planning is done at times with some difficulty. Hollerbach and Atkeson (1987) put forth the idea that the velocity profile of the elbow and shoulder joint are unimodal but motion would start in the joint that would develop the greatest excursion. Hogan and Flash (1987) contradict this explanation and point to bimodal velocity profiles created in momentary reversals in joint excursions, which Hollerbach and Atkeson did not see. Soechting and Flanders (1991) suggest the discrepancy could be because Hogan and Flash calculated the joint excursion from measured hand displacement and Hollerbach and Atkeson measure them directly. Kaminski and Gentile (1989) suggest that the "organization of arm movements is hierarchically structured with the important, but different contributions being made on both the hand planning and joint planning levels".

Thus far, the discussion has primarily been on arm movements restricted to the transverse or sagittal plane along straight trajectories. A number of researchers have reported that in general hand trajectories are invariant under translation and rotation as well as amplitude and time scaling (Morasso, 1981; Abend et al., 1982; Flash and Hogan, 1985). This has been shown to be true for multijoint arm movements (restricted to the transverse and sagittal plane) of equal length at different locations, for different orientations of the body, for large and small movements and at different speeds respectively. However, movements are not generally made in a single plane but in three dimensions. Clearly, while writing or drawing the hand is not constrained to follow along

a straight path although, straightness of trajectory is the trend in pointing movements. Abend and colleagues (1982) have shown that the so-called smooth, unimodal velocity profile is not always preserved. They observed bimodal velocity profiles when the subject was instructed to use a curved path to move to a target in the transverse plane. Regardless, because movements are restricted to the transverse plane in this study, it can be safely assumed that the rotations at the joints are single axis rotations.

To produce arm movements the CNS must control the activity of each muscle in the arm to move the hand along the desired path at the preferred speed. It must do so in such a manner that it generates the appropriate forces in each muscle spanning the joint needed to produce the necessary joint torque. For multijoint movements the CNS must alter control of the arm to adapt to the reaction, Coriolis, and centripetal forces generated during multijoint movement (*cf.* Hollerbach and Flash, 1982). Flanders and Colleagues (1997), caution that the focus on kinematic variables and control should not be taken to mean that the kinetics subserve the kinematics, even though they readily admit that reaching is a spatially defined kinematic problem. Their reasoning behind this caution is that evidence suggests there is no preferential control of tonic patterns of muscle activation. In their experiment, they found that curvature seemed to be related to dynamic torque and phasic muscle activity.

Merton (1953) was first to suggest that motor control of posture and movement could be equivalent; in effect, this implies that movement is initiated and controlled based on stretch reflexes. This thesis serves as the fundamental principal behind the equilibrium point hypothesis, which contends that the CNS organizes positional frames of reference for the motor apparatus and shifts the frames in space to produce movement (Feldman,

1986). The shift in the reference frame is brought about by a shift in the threshold properties of the proprioceptive reflex loops and motoneurons. This model and others (i.e. Bizzi, 1979) require an in depth understanding of the reflex arc and its structures which McMahon (1984) and Deutsch and Deutsch (1993) provide (Berkinblit et al., 1986).

2.2.3 Summary

How the CNS locates a position, transforms the position into neural commands, which will drive the limb to that position, has been of interest to many researchers. Various theories and constraints have been proposed for the planning; coordinate transformation, neural command, and execution of limb movement. Current theory points to a course mapping discovered in the premotor areas of the spinal cord (Bizzi et al., 1991; Mussa-Ivaldi, 1999). Stimulation of this structure's circuitry produced convergent force vectors in the musculature. The result is that each muscle generated a force to produce a synergistic effort to move the arm to a new equilibrium. The direction of movement generated coincides with specific sites of stimulation in the premotor areas of the spinal cord (Bizzi et al., 1998, Mussa-Ivaldi, 1999).

This introduction of how the CNS coordinates and prosecutes movement is an overview of what the prevailing thoughts and theory are in motor control. Models have been proposed based on the use of EMG to scale the force output of the muscle (see sect. 2.3). These models are developed based on prevailing motor control theory presented and provide a means of examining actual movements via the neural output of the CNS. In the pursuit of an exoskeletal orthosis, it seems natural to look for invariance in the movements and neural parameters as well; EMG provides such a neural window.

2.3 EMG-Force Modeling

Although emphasis has been placed primarily on the kinematic or dynamic features of upper extremity movement, muscle activation patterns underlying these features have also been reported using EMG potentials (Lacquaniti et al. 1986; Hasan and Karst, 1989; Flanders and Soechting, 1990; Karst and Hasan 1991, 1991; Buchanan et al. 1993, Theeuwen et al., 1994). The attempt of much of this research is to use EMG signals as a means for determining muscular coordination, selection of agonist during initiation of planar movement, direction of movement and timing. Determining the synergistic actions of upper extremity musculature during dynamic actions is paramount to understanding selection, activation, intensity, and direction of movement of the hand in a plane. To this end several attempts have been made to describe and comprehend the muscle coordination strategies the CNS employs to develop force and motion at the hand based on EMG signals (Karst and Hasan, 1987, 1991a 1991b; Soechting and Lacquaniti, 1981; Buchanan et al., 1986; Flanders and Soechting, 1990; Buchanan et al., 1993; Theeuwen et al., 1994; Soechting and Flanders, 1997).

Triphasic EMG patterns during point-to-point arm movement begin as an initial burst in agonist muscles followed by a pause in agonist activity for fast goal directed movements. While agonist activity is paused, antagonist muscles can be activated or quiet depending on the speed of movement, which is then followed by the resumption of agonist activity (Angel, 1974; Hallett 1986). This pattern was found to hold true for single as well as multiple joint movements (Karst and Hasan, 1991b; Buneo et. al, 1994). Karst and Hasan (1991a, 1991b) used this paradigm to ascertain agonist muscle synergistic actions and characteristics for planar two-joint arm motions. Moreover, this

pattern is known to exist for rapid single joint movements in certain pathologies such as Parkinson's, Huntington's, Upper Motor Neuron Syndrome, Cerebellar Ataxia, and Dystonia. Berardelli et al. (1996) provides an excellent summation of the results of multiple studies concerning these pathologies and their effects on the classical triphasic EMG pattern. Yet, the existence of this pattern in DMD is still to be determined.

Typically, individual muscle contributions in the course of arm movements are based on the maximal force capability of the muscle estimated by its physiological cross sectional area (PCSA). This thesis simply states that the maximum force a muscle can develop is proportional to its PCSA. The contribution of a muscle has been estimated derived from an EMG profile weighted by the PCSA (*cf.* Hof, 1984; Theeuwen et al., 1996; van Bolhuis and Gielen, 1997; Prilutsky and Gregor, 2000). In the case of the DMD subject, the established estimates or estimating methods for PCSA would not hold true due to time dependent pathophysiological changes in the musculature. This does not preclude the use of such methodology as a possible control parameter. PCSA can be estimated based on the percentage of fibers types and their size, which has been well reported on in the literature (Buchthal et al., 1971; Coërs and Telerman-Toppet, 1977; Wang et al., 1999). Yet, the continuous changes in the musculoskeletal system due to the disease make such an approach difficult.

Another method towards determining individual muscle contribution for dynamic actions is generally approached from a mechanical muscle model aspect such as Hill's well-known muscle model. This modeling approach requires knowledge of twitch force; the force-velocity relationship, passive and active tissue characteristics etc. (*cf.* Hof and Van Den Berg, 1981a, b, c). This seems a possible approach because many of these

characteristics have been reported on in the literature for MD subjects (McComas and Thomas, 1968; Belanger and McComas, 1983; Frankeny et al., 1983; Iannaccone et al., 1987; Martinez and Lõpez-Terradas, 1992; Priez et al., 1992; Phoenix et al., 1996; Orzio et al., 1997; Lindeman et al., 1999a, b). Moreover, the EMG potential and PCSA of a muscle can be incorporated into the model to serve as the driving function. However, again the researcher is faced with an ever-changing system that requires the model be adjusted not only to the individual but also to each distinct muscle in the system being modeled.

2.3.1 Mathematical Representations

To realize how the CNS exerts control over upper extremity movement studies of muscle activation timing and amplitude in response to a known external excitation have been done. A significant conclusion from such studies has determined that activation timing and the phasing of shoulder and elbow muscles can vary with direction of movement (Wadman et al., 1980; Karst and Hasan 1991b; Flanders, 1991; Soechting and Flanders, 1997). Flanders (1991) concluded that a muscle could be activated earliest in a movement direction in which the muscle is least active. Assume that EMG potential is linearly related to force magnitude along one direction in space, i.e. the preferred direction. Then the EMG potential of a muscle decreases with the cosine of the angle relative to the force direction in which the maximal EMG potential is developed as in Equation 1 (Georgopoulos et al., 1984; Flanders and Soechting, 1990, Theeuwen et al., 1994).

$$EMG = cF\cos(\phi - \phi_r) \quad \text{Equation 1}$$

Where c is a scaling constant, F is the isometric force magnitude, ϕ is the force direction of maximum EMG, and ϕ_r is the current force direction. Flanders and Soechting (1990) concluded that a multiple cosine function could be applied to the EMG signal to map the directional tuning of a muscle performing an isometric task. Moreover, a muscle's

directional tuning map produced during an isometric effort co-varied with postures at the shoulder and elbow. Buchanan et al. (1986) evaluated elbow torque during isometric contractions and found that EMG potential reached a maximum in the direction of greatest mechanical advantage. Theeuwen et al., (1994) has shown that the preferred direction as predicted by the model coincides with the direction in which the recruitment threshold of motor units is smallest.

The relationship between dynamic motion and EMG can be incorporated in a dynamic activation model based on Hill's muscle model with the force-velocity and series elastic element as in Equation 2 (Wilkie, 1954; Zajac, 1989; Soechting and Flanders, 1997).

$$EMG(t) = \tau_a \dot{\alpha} + \alpha \quad \text{Equation 2}$$

Where $\{0 \leq \text{EMG (t)} \leq 1\}$ is the EMG potential magnitude normalized to the maximal tetanus contraction as is α, τ_a is the muscle twitch time, and $\dot{\alpha}$, α are the force-velocity parameters.

Buchanan et al., (1993) took another approach and related the EMG signal to an applied external moment via a coefficient method where:

$$M_i^e = \sum_{j=0}^{m} EMG_{ij} \rho_j r_j \quad \text{Equation 3}$$

M_i^e is the external measured moment in the i^{th} configuration, EMG_{ij} is the j^{th} muscle's developed potential in the i^{th} configuration, ρ_j is the force-EMG coefficient for the j^{th} muscle, and r_j is the muscle moment arm vector. By measuring the EMG in several directions of force application, it is possible to obtain the number of equations to make

the system determinant. Therefore, the force contribution of an individual muscle is found by Equation 4 where the muscle's line of action is e_j:

$$F_{ij}^{m} = EMG_{ij}\rho_j e_j \quad \text{Equation 4}$$

This model was found adequate in the prediction of muscle forces developed at the wrist with the assumption of muscle EMG-force linearity however, it remains uncertain if it would accurately predict muscle force in a more highly redundant system. Several other EMG-force models have been presented in the literature but most are variations of the models in Equations 1,2, and 3 (Gottlieb and Agarwal, 1971; Wadman et al., 1980; Pollak, 1980; Hof, 1984; Buchanan et al., 1986; Solomonow et al., 1990; McGill, 1992; Theeuwen et al., 1996; Bolhuis et al., 1997; Prilutsky, 2000). A noteworthy fact is that there have been no attempts to date to use these methods in MD.

2.3.2 An Empirical Approach

Karst and Hasan (1987, 1991a, b) and Hasan and Karst (1989) took an empirical approach. First, they define a two-segment model with joints at the elbow, (E) and shoulder, (S). They also define joint angle of each segment as $\theta_{E,}$ and θ_S relative to a fixed coordinate system in the transverse plane are shown in Figure 3. In addition, the target position relative to the start position of the hand was defined as ψ. Movement of the segments was monitored along with the activity of five upper arm muscles for 470 movements with varied start and finish points within the reachable workspace. The EMG was rectified and filtered with conventional methods then quantified by the integral over the first 100 ms of activity. Muscles were considered activated at a threshold value above their baseline and the hand was considered moving at a threshold of .1 m/s. A "±sign" is given to the muscles based on initial activity; muscles activated first were identified as

movement agonists; those activated next as movement antagonists, regardless of their anatomical agonist or antagonist action.

Karst and Hasan (1991b) set out to show it was possible to predict the final position from the start by applying two rules using surface EMG. One, the initial muscle activity at each joint should be such that the distal tip of the limb exhorts a force in the direction of the final tip position. Two, the initial activity at each joint should be such that the initial acceleration of the distal tip is in the direction of the final tip position. Neither of these rules was proven to exist. Instead, they would make use of surface EMG potentials to identify the agonist(s) selected by the CNS and thought to initiate motion in a workspace, quantify its effort by integral of EMG activity, and vise versa for the antagonists.

What these researchers found was a consistent partitioning of flexor an extensor

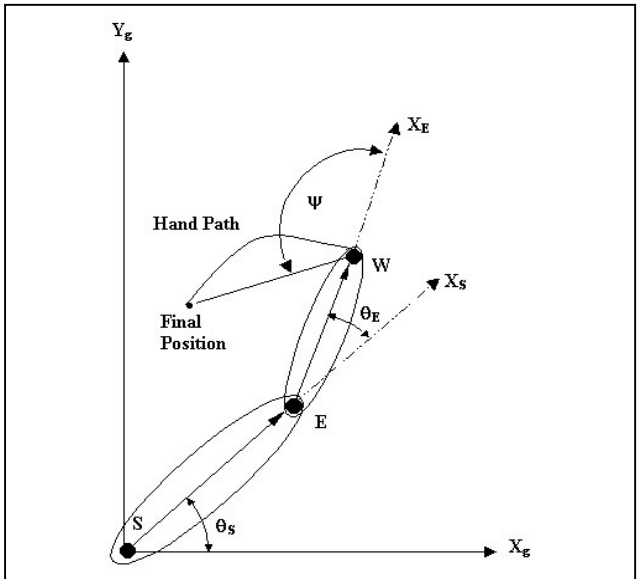


Figure 3: Diagram defining joint angles and the direction of hand movement from initial to final position where the angle ψ defines the target direction relative to the initial orientation of the arm.

initiated movements with respect to ψ and $\boldsymbol{\theta}_E^{\mathbf{int}}$ where *int*=initial. They found that the direction of the final position relative to the start, ψ, is sufficient for determining which movement agonist(s) would be selected, i.e. which muscles would be activated first. In other words, they had identified an empirical rule based on initial muscle activity that could be used to determine the initial direction the hand was to move. This method requires no consideration of the system dynamics or limb trajectory.

2.3.3 Summary

Several EMG models were presented, each with its own benefit; each one of these methods is a possible candidate for an EMG control input. The drawback is they are highly sophisticated, work under rigid constraints, and have demanding signal processing and computational requirements. If partitioning as Karst and Hasan (1991b) have identified appears in DMD, it may be possible to use the initial activity of the muscles to indicate movement direction for the orthosis. Such a method should be considered because once the initial muscle activation pattern is tabularized (i.e. the partitioning of the workspace is known), direction intent can be found based on the monitored muscle activity. Of course, it must be kept in mind that whatever method is considered, the DMD muscle is constantly changing requiring the EMG input to be highly adaptable.

CHAPTER 3: PRELIMINARY STUDY

Myogenic diseases such as muscular dystrophy and others leave an individual with insufficient muscle power to move their arms in 3-D space. The needs of these individuals instigated an initial study into the feasibility of using EMG signals from subjects with the myogenic disorders as a supplementary control input to a robotic orthosis for the upper extremity (Bowen et al., 2001). In the preliminary study, the level of EMG potentials a MD subject could generate and the corresponding muscular synergy were examined in planar point-to-point multijoint upper extremity movements and during isometric activity.

A series of tests were designed to record EMG during maximal isometric force application and to study synergistic muscular activity during dynamic arm motion. A test-bed was constructed where the subjects were seated next to a table and were asked to apply maximum radial force with their elbow flexed at 160^{o} degrees relative to the upper arm. The force transducers (filled dots in Figure 4) were set at an attitude of 30^{o}, 60^{o}, 90^{o},

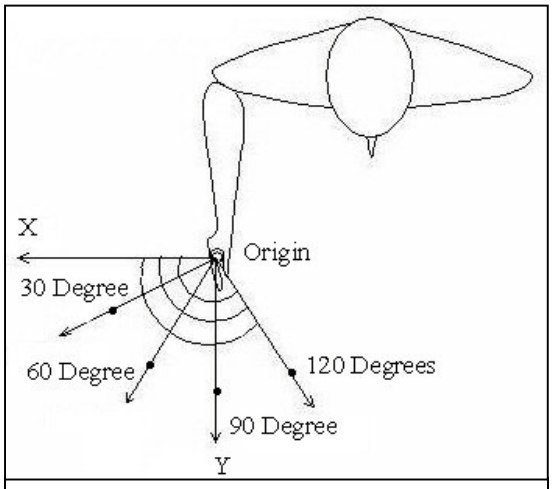


Figure 4: Air Bearing Schematic, air is pumped into the bearing through a fitting seated in the top of the device. An air compressor supplying 120 cubic feet per minute supplies the air at 20 psi.

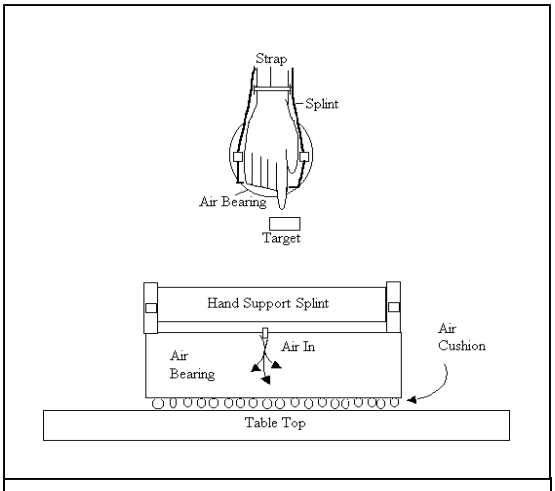


Figure 5: View of table layout with subject seated with their hand positioned at the origin. Isometric and isotonic test positions are located along radial lines at attitudes of 30^{o}, 60^{o}, 90^{o}, and 120^{o} degrees.

and 120^{o} degrees relative to the lateral aspect. Subjects were to push/pull in the anterior-posterior directions. For the dynamic tests, targets were placed along the same attitudes and the subjects were asked to reach for the target with a special arm support provision that ensured frictionless sliding (Figure 5).

In this study, the residual motor activity potentials of MD muscles realized with EMG signals as obtained under isometric and dynamic conditions were at significant levels (see Table 1) and were in the range of reported values (Piotrkiewicz et al., 1993; Edwards, 1980; Lindeman et al., 1999b; Orizio et al., 1997). Potentials emerged at sufficient levels for use in studies determining muscle coordination strategies, direction dependence, estimating muscle contribution, and agonist selection at onset of movement utilizing methods similar to those reported in literature (Lindeman et al., 1992b; Wadman, et al., 1980, Karst and Hasan 1991a, 1991b; Theeuwen et al., 1994). For the MD subjects the EMG potential levels generated are reduced as anticipated. They are also large enough to suggest when, and perhaps how much a muscle is contributing to an effort (Edwards, 1980; Lindeman et al., 1999b; Orizio et al., 1997). This information is essential to understanding how the CNS copes with a neuropathology affecting muscle and function.

Despite being unaware of the force magnitudes being developed, both the MD and

Table 1. Peak action potential levels

Subject	Isometric	Dynamic
Healthy 18 yo	300 μV	100 μV
Healthy 14 yo	250 μV	100 μV
BMD	150 μV	40 μV
DMD	50 μV	---

healthy subjects were capable of maintaining relatively consistent force levels for the test duration. Performance of the isometric tasks required coordinated muscle activity to produce maximal efforts in the desired directions. These facts suggest that in MD while the upper extremity has force-generating capabilities of adequate strength, the isometric force characteristics these individuals produce will mimic the healthy model although at a reduced level.

Breakdown in the ability to maintain straight smooth hand trajectories while executing a movement could be a measure of functional capability. Namely, a greater number of trajectory changes between two points indicate a greater decline in ability or some other variance in a known parameter. In the MD subjects examined, upper body motion was allowed and rotation/translations were large in amplitude at times. However, the hand paths produced were predominantly straight. This suggests that compensation mechanisms were employed in such a manner as to assist the movement and constrain hand motion along roughly straight trajectories. In other words as long as viable compensation mechanisms can be effectively employed to produce a desired point-to-point hand movement, the trajectory will remain relatively straight.

3.1 Summary

In the continuation of this work, the isometric tasks will not be repeated. This is not because there is no interest in this information but for other reasons, the least of which is subject fatigue. This was an issue voiced by the DMD subject tested in this study and to accomplish some statistical significance the target acquisition task must be repeated a number of times throughout the workspace. The test in its current configuration with its current task is too daunting and may be impossible for others with DMD. Another factor

is the duration of the testing, it is difficult to keep young subjects interested and motivated to perform the desired task the longer and more challenging the task is. Consequently, to decrease the time and fatigue factor the tests will be limited to target acquisition.

Limiting the tasks to target acquisitions does not address all changes needed such as the test configuration. It takes muscular effort to transport the arm from an initial posture to its final posture. Therefore, simplifying the kinematics and dynamics of the mechanical system required to carry the arm from one posture to another could be helpful. For example, Miller et al., (1992) have shown that there is two rotation axes contained on a two dimension curved surface describing limb orientation for pointing movements in three dimensions. This can be reduced to one rotational axis if the pointing movements are done in the transverse plane.

Another benefit to having the arm supported in this plane is that the gravitational component of the mechanical system is essentially eliminated. This leaves the muscles free to act as limb movers rather than limb supporters (Hollerbach and Flash, 1982), which will reduce the fatigue. There is no guarantee that fatigue will be eliminated. However, if the muscles are not needed for supporting the arm they will most certainly have more reserves for making a greater number of pointing movements. Realistically the repetition of target acquisition and variation of target location will still be limited due to strength issues.

CHAPTER 4: RESEARCH DESIGN METHODS

These materials and methods were submitted to and approved by the institutional review boards for human experiments at A.I. duPont Hospital for Children, Wilmington, DE and Drexel University, Philadelphia, PA. The experiments were conducted at A.I. duPont Hospital for Children, Wilmington, DE in the Rehabilitation & Pediatric Engineering Research Center. National Institute of Health certification for individual conducting human experimentation was on file at this location and available upon request.

4.1 Participation of Children (Subjects)

Participants were recruited from the patient population attending Muscle Clinic within the Neurology Department of A.I. duPont Hospital for Children. Each participant and guardian (when appropriate) was informed of the risks and benefits of the research and was required to give informed consent. Several factors considered when selecting of subjects were sex, age, available population, ability, intelligence, and subject cooperation.

4.1.1 Subjects

The goals of this study include seeking the effects DMD has on kinematic features and the biomechanical adaptations integrated into the movement for compensation of weakness. Individuals with DMD usually continue to function near or at normal levels until age eight and will not demonstrate sufficient weakness for the study (ref. Sect 3). In other words, the disease has not yet had a gross enough effect on the functional ability to warrant inclusion. Children under 8 years of age would be capable of performing the study tasks required although they present other difficulties in cognitive ability, maturity, and attentiveness.

Due to the functional requirements of performing 90 pointing tasks specified in Section 4.2.3, finding subjects with adequate functional capability was prohibitive. The level of function will be found in the age range of 8-18 years necessary for conducting the movement study. This choice was based on clinical evaluation of functional ability and strength evaluations reported in the literature (McDonald, C. M., et al., 1995; Merlini et al., 1992; Parker et al., 1990; Sunnegardh et al., 1988; McCartney, et al., 1988; Lord et al., 1987; Hosking et al., 1978). However, during recruitment several individuals were identified with sufficient function older than 18 years of age (see Table 2).

The selection criteria for participating in the study were; having some ability to move hand in the transverse plane as in Figure 6 by joint rotations at the elbow and shoulder otherwise the required tasks could not be performed. Participants did not necessarily have to have the strength to move their upper extremity against gravitational forces because the subject's upper extremity was supported in the transverse plane as described in Sect. 3.1. With this support, movement of the arm requires the musculature to overcome the arm segmental inertias only to achieve motion. In other words, participants were able to

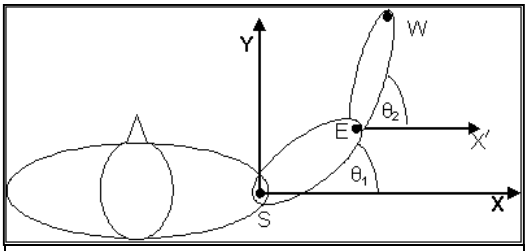


Figure 6: Subject test orientation with the arm supported in the transverse plain. Theta 1 and Theta 2 represent the joint orientation of the upper and forearm respectively. Points S, E and W represent the center of joint rotation for the shoulder, elbow, and wrist respectively.

achieve joints rotations while their arm was supported against gravitational forces.

Another requirement for participation was that the test subject needed or was about to acquire a powered wheelchair for maintaining mobility. This threshold for level of disability ensures that weakness has begun to affect the upper limbs. The criteria for subject selection may have to be adjusted as the study progresses and evidence supports a change, however the preliminary results support this approach (ref. Section 3).

4.2 Data Collection, Equipment, and Specifications

Technical specifications and calibration data of all the equipment specified herein are kept on file at Drexel University, School of Biomedical Engineering, Science, and Health Systems in the Human Performance and Rehabilitation Laboratory and are available upon request. Viewing of medical records was restricted to necessary research staff. All persons collecting data have taken the requisite NIH Human Subjects course as required

Table 2. Demographic and clinical history for subjects tested arranged in the order of strength based on manual muscle tests.

SUBJECT RELEVENT CLINICAL DIAGNOSIS								
				Manual Muscle Test*			Upper Arm Deep Tendon Reflex	Exam Date
Subject	Age	Height (cm)	Weight (kg)	Deltoids	Biceps	Grip		
1	16	152.4	70.3	1	2	3	Absent	5/31/2002
2	17	133	40.1	2+	3	4-	Absent	9/20/2002
3	19	174	87.1	3	3	4	Diminished	6/16/2001
4	11		76.8	3 to 3+	4 to 4+	4+to 5-	Diminished	4/12/2002
5	17	142	63.7	3+ to 4-	4	5	Absent	6/14/2002
6	11	142	71.5	4-	5-	5	Absent	3/22/2002
7	13	132	52.8	4	4+	5	Absent	10/12/2002
* Manual muscle test grade scale is 0-5								
1	Hypertrophy, hypotonia, posterior spinal fusion w/unit rod, sensory exam intact.							
2	No tremor, normal ocular movement, hypotonia, sensory exam intact to light touch.							
3	Elbow Ext. contractures, normal ocular movement, and sensory exam intact.							
4	Normal ocular movement, cushingoid appearance, sensory exam intact to touch.							
5	No tremor, normal ocular movement, hypotonia, sensory exam intact to light touch.							
6	Normal ocular movement, hypotonia, and sensory exam intact to light touch.							
7	No tremor, normal ocular movement, hypotonia, sensory exam intact to light touch.							

4.2.1 Anthropometrics & Clinical Background

The height and weight of each subject was recorded in addition to age and relevant clinical background information relating to strength, reflexes, and other diagnoses (Table 2). Length measurements were taken of the following using a flexible tape: length for each segment. The measurements were from the sternoclavicular joint to acromion process; acromion process to lateral humeral epicondyle; lateral humeral epicondyle to ulnar styloid process for the sternum, upper arm and forearm segments respectively. Circumferences of the arm and trunk segments were taken at wrist below the ulnar styloid process and just below the lateral humeral epicondyle; at just above the lateral humeral epicondyle and most proximal circumference of the upper arm; and lastly the circumference of the trunk at breast and waist level (Table 3).

Although the collection of clinical background information such as manual muscle tests,

Table 3. Subject anthropometric measurements

SUBJECT ANTHROPOMETRICS						
Subject	CICUNFRENCES (cm)					
	Wrist	Elbow 1	Elbow 2	Upper Arm	Waist	Chest
1	18 cm	30 cm	28 cm	32 cm	104 cm	103 cm
2	13 cm	21 cm	19 cm	23.5 cm	76 cm	90 cm
3	6.5 cm	10.5 cm	11 cm	15 cm	110 cm	115 cm
4	16 cm	25 cm	25 cm	21 cm	112 cm	104 cm
5						
6	15.5 cm	26 cm	27.5 cm	33 cm	98 cm	114 cm
7	16 cm	24 cm	23 cm	29	91 cm	101 cm

	LENGTH (cm)		
	Forearm	Upper Arm	Clavicle
1	20.5 cm	30 cm	17.5 cm
2	19 cm	25.5 cm	13 cm
3	27 cm	32 cm	20 cm
4	20 cm	28 cm	17 cm
5			
6	21 cm	26 cm	17 cm
7	19 cm	21 cm	12 cm

state of the disease's progression and level of functional evaluations were not discussed within the proposal, any available information was sought and compiled in Table 2. This information will be useful in making estimations and evaluations based on function and strength within DMD populations reported in existing literature based on other methods (McDonald, C. M., et al., 1995; Merlini et al., 1992; Parker et al., 1990; Sunnegardh et al., 1988; McCartney, et al., 1988; Lord et al., 1987; Hosking et al., 1978). This study is the first step in the genesis of an idea; several other future avenues remain to be explored for which these data will be needed.

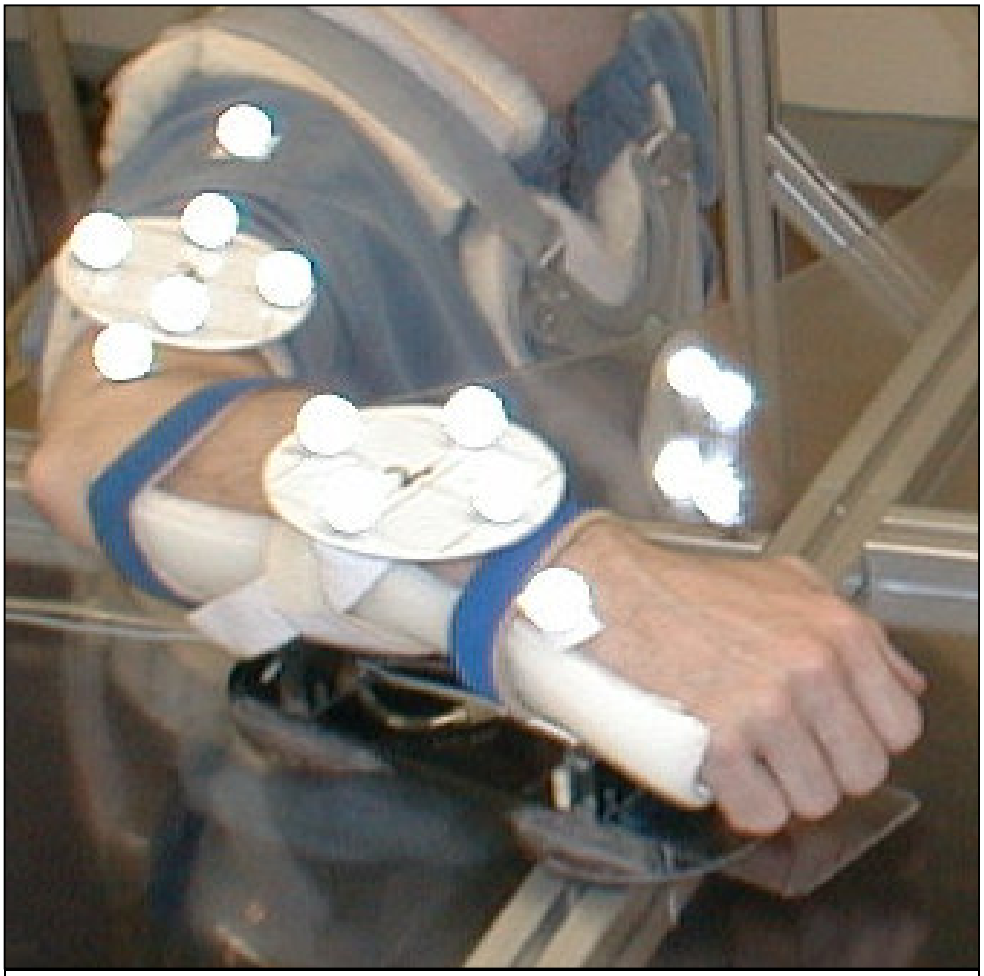


Figure 7: Illustration of marker placement, air bearing carriage, and chest harness. Note that the marker bodies on the upper and forearm were not needed because these segments did not rotate.

4.2.2 EMG

Two BioResearch™ EMG amplifiers were used to monitor muscle activity. Some of the system's operating parameters are fixed including system sensitivity greater than 1.0μV (p-p) with an A to D converter resolution of 0.625μV and a bandwidth of 30-600 Hz, a gain setting of 5000 with a digital noise reduction filter operating at 26 dB. This system is optically isolated to protect the participants from any possibility of shock. Biopolar electrodes 2cm apart with a diameter of of 1cm were employed to collect recordings from eight muscles namely; anterior deltoid, posterior deltoid, latissimus dorsi, pectoralis major, brachioradialis, bicep brachii, lateral head of tricep, and long head of tricep. The site where each EMG electrode was affixed was lightly abraded and cleaned with alcohol and cotton swab before attachment of the electrode. A resting EMG value was recorded at the start and finish of each movement task to establish a baseline value. EMG data were collected on a PC platform with software designed in Labview® for cataloging the data

4.2.3 Kinematics

The kinematics of the upper extremity and trunk were monitored during the movement tasks. The upper extremity model is constructed of two link segments with the hand and forearm forming one segment and the upper arm forming the other as illustrated in Figure 6 and 7. To reconstruct the motion of the arm and the trunk the motion of arm segments and trunk segment will be monitored utilizing MacReflex® system with and absolute accuracy of 0.4 mm. This system was calibrated before the start of testing and has an acquisition rate of 50 Hz. Nine passive markers with a diameter of 1.905 cm will be attached to the subject at the following locations: ulnar styloid process, lateral humeral epicondyle, highest point of the acromion, sternoclavicular joint and 3 markers affixed to

a marker body place mid span of the sternum. Another maker was employed as the ground marker and was fixed to the test table along the 90^{o} degree attitude. This marker was used as a reference for determining the extent of the upper body translations when the trunk is not constrained.

Figure 8: Targets are located at angles of 30˚, 60˚, 90˚, 120˚, and 150˚ degrees relative to the wrist start position. The subject sat in their own wheelchair; the table height was adjusted until the upper arm is abducted 90˚ degrees in the frontal and transverse planes. The forearm was flexed 90˚ degrees and strapped into the air bearing carriage. Subjects were asked to acquire each target five times consecutively from wrist start position. The 30˚, 60˚, 90˚, and 120˚ degree target were acquired fives time from while the subject started while pointing at the 150˚ degree target.

The kinematic data acquisition system is Macintosh based while the EMG system is PC based and these systems did not have the capability of communicating. Therefore, some other method to coordinate the timing between the EMG and kinematic data acquisition systems had to be employed. This was achieved by utilizing another passive (coordinating) marker that would actively popup when the EMG system started data collection. That is, a spring-loaded popup marker that was hidden in a box until the EMG system was started. The kinematic system was started first, followed by the EMG system and when the EMG system was started, a step motor would release the marker allowing it to rise up out of the box. This event was recorded by the kinematic system establishing a coordinating time index for the two systems (ref. Figure 8). Movement was not started until after the appearance of the popup marker and the time delay was negligible.

The schematics shown in Figures 5, 7 and 10 illustrate the function of the air bearing constructed of polymer disk shaped skid pads mounted to a frame. Air was forced through the bearing at the top such that it floats on a cushion of air escaping from the bottom. An air compressor developing 20 cfm at 10-12 psi was used to float the air bearing. The air bearing is fixed to the test subject by a molded trough mounted (carriage) into which the hand and forearm was strapped on an air bearing frame made from ½ inch Plexiglas.

The carriage also prevents any articulation at the wrist so that the forearm and upper arm segments can be treated as single segments without axial rotation. The air bearing supports the arm on a cushion of air such that the movement from target to target is quasi-frictionless requiring that the subject overcome the inertias of the arm segments and the air bearing. This approach requires an inverse solution because the shoulder does not

remain fixed. This is especially true when the trunk is free to move. To assist the air bearing silicon was sprayed on the table surface. This was done to assist not only in sliding but also for decreasing the coefficient of friction when the air bearing failed at the edge of the disks and caught an edge. A chest harness taken from a HALO fixation device (shown in Figure 7) designed to hold the head in a specific position was use to constrain the trunk motion during the limited trunk movement test.

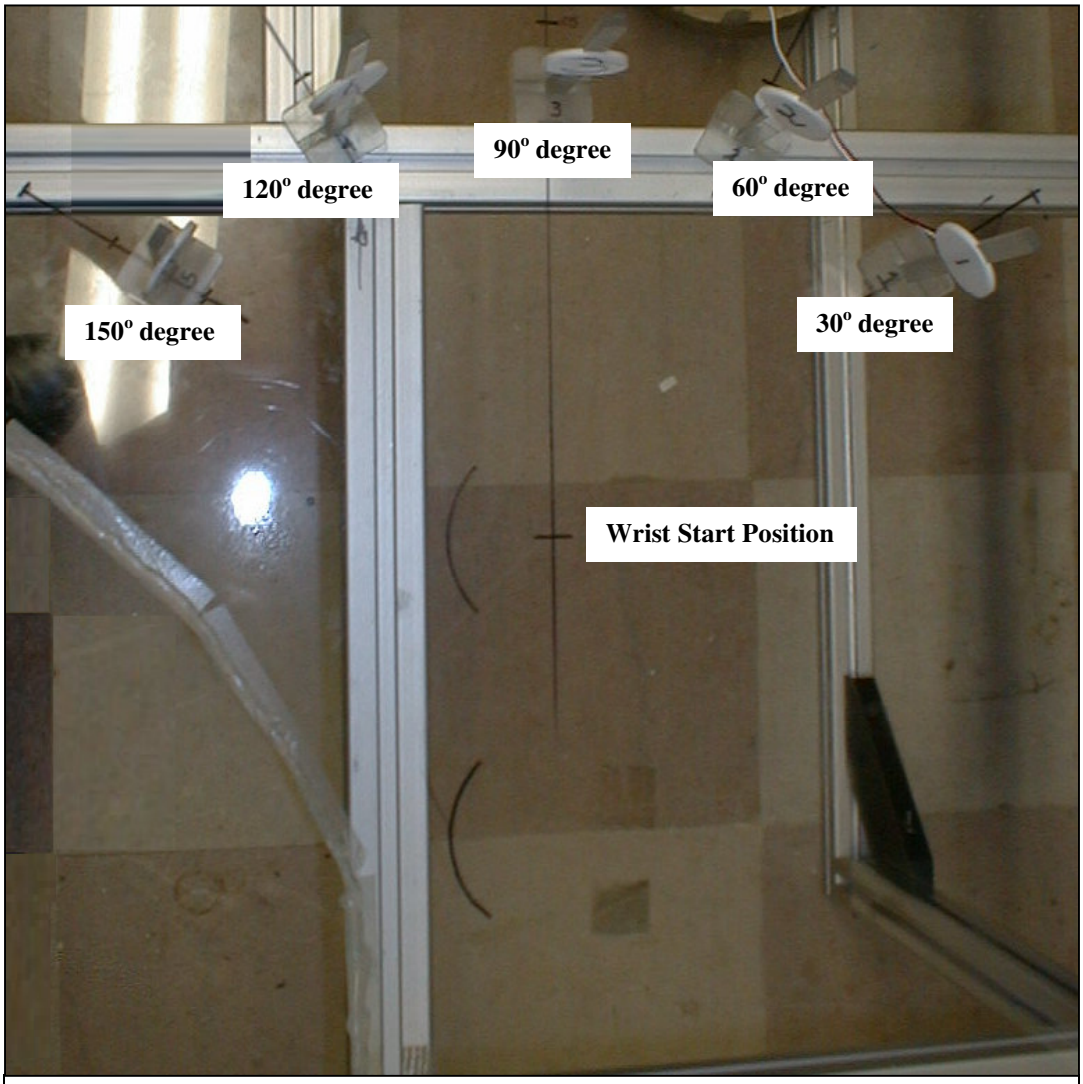


Figure 9: Popup maker was hidden behind the tissue, when the EMG data acquisition system started the maker popped up from under the tissue. Upon appearance of the popup marker the subject was to count, "one thousand one" then execute the pointing movement towards the appropriate target.

4.2.4 Experimental Setup

The air bearing is placed in the fixed start position such that the air disks are aligned with the set start position marked on the table shown in Figure 9 and illustrated in Figure 8. Subjects were seated so that their upper arm was abducted 90^{o} degrees and such that all movements were in the horizontal plane as seen in Figure 7. Each subject was positioned at the table such that when the arm was supported in the air bearing the upper arm was in alignment with the shoulders and the forearm was flexed 90^{o} degrees. Table height was adjusted for each subject to accommodate this arm configuration. This was the start position for reaching forward in to the workspace to acquire a specific target.

Reaching from one position to another in the workspace was examined as well from a single start position. The test subjects were asked to start by reaching into the workspace and pointing at target five. From the target five-start position each remaining target was acquired by moving to and pointing to each target.

The targets were set at a fixed height of 6 cm from the base of the target stand and

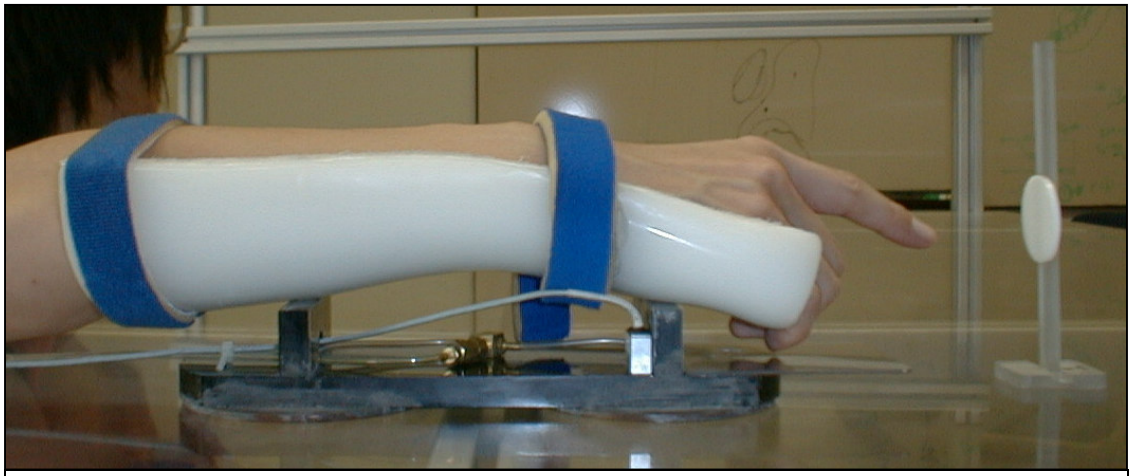


Figure 10: The air bearing rests on two skid pads (front and rear) that air is forced through creating a cushion of air to float the carriage; the cuff is made of heat formable polymer.

numbered to help the test subjects follow instruction. The distance of the targets from a common origin was located at a distance attainable to the individual subject by taking into consideration the affects of the disease such as joint contractures. This meant that the distance to the targets was not fixed but subject specific. The molded trough was lined with additional padding to for those subjects with smaller arms to maintain a restriction on forearm pronation and supination when needed or for additional comfort. Video was taken during the experimentation to help assess and qualify the movement patterns collected. During the constrained trunk motion the chest harness was secured to the wheelchair the subject was seated in, if a chest strap was available on the chair this was also used EMG and Kinematic markers were placed on the test subject in accordance with the descriptions in previous sections.

4.3 Procedure

A hypothesis formed is that given upper extremity musculature weakness, adjustments in posture are made to compensate before and during target acquisition. To prove this and satisfy other goals of the study, i.e. identifying changes in kinematic variables, the following tests configurations were used:

A) Upper Extremity movements performed with the trunk constrained.

B) Upper Extremity movements performed with the trunk unconstrained

The reasoning for the movement tasks being conducted with the trunk in the constrained and unconstrained configuration will be discussed in Sect. 4.2.4.

To perform target acquisition subjects will be asked to move their hand from an initial position to a final position by acquiring a target at the final position with their forefinger. That is, given a start position at the origin the subject would be asked to acquire targets

fixed along the attitudes 30^{o}, 60^{0}, 90^{o}, 120^{o}, and 150^{o} degrees. The subject would move from the start position to each target individually, i.e. move from origin to acquire target fixed along 30^{o} degree attitude, then from the origin to the target fixed along the 60^{o} degree attitude, and so on until each target has been acquired a total of five times. When staring from the target five start position the subjects were instructed to acquire the targets at 30, 60, 90 and 120 degrees from this position at total of five times. This accounts for 80 trials per subject after completion of the constrained and unconstrained configuration.

Subjects were instructed that the task is not a reaction time task as long as they can acquire the target in a 4 sec time window. At the start of each target acquisition, the subjects were instructed to relax until they see the popup marker appear. Upon appearance of the popup marker the subjects were to count, *one-thousand-one* to themselves then execute the move to acquire the target. After the target was acquired, subjects were to maintain the final position until they were told to relax. Upon the command of 'relax', they were to return to the start position.

Target acquisition was conducted with the trunk in a constrained and unconstrained configuration. In the constrained configuration trunk, motion was limited by strapping the upper-body, shoulder, and waist to the seat. While performing target acquisition in the constrained configuration, the musculature of the upper extremity will be required to generate the joint torques necessary to overcome the inertial properties of the arm segments and air bearings in order to produce motion. Additionally, the subjects will have to construct the desired motion without employing mechanisms of compensation but through physical application of muscular efforts. Conversely, when in the unconstrained

configuration the trunk will be free to move allowing the subject the ability to employ mechanisms and strategies of compensation if desired.

To understand how DMD subjects compensate for local weakness a movement task is considered where the subject is asked to acquire targets in a confined workspace. To ensure successful performance of the target acquisition task by the subjects the arm will remain supported by the air bearing throughout the effort. As expected the disabling affect of DMD prohibited some subjects from maintaining their upper extremity in an abducted position in the plane of interest. In other words, the subject did not have the necessary strength to support their arm against gravitational forces. Sustaining the reason given in Sections 3.1 and above, i.e. utilizing an air-bearing to support the arm on air allows most of any muscular effort expended by the subject to overcome system inertia and the friction component between the table and arm during movement to be minimized.

Last, but certainly not least, it is desirable to have experimental results that can be compared with results of similar studies. This makes it necessary to have methods if not the same, then very similar to the works with which the results will be compared. This methodology is constructed with a protocol similar to a body of published works on upper extremity movement concerned with kinematics, dynamics, EMG, and motor control with sufficient deviation to achieve the study goals.

CHAPTER 5: DATA HANDLING

In Section 4 the equipment, their required data acquisition rates, and experiment method were described. This section provides a description of how the data were handled and used once they were collected. The purpose of the data analysis is to elicit some of the electromyographic and kinematic characteristic of multijoint point-to-point hand movements in Duchenne's muscular dystrophy. Therefore, the kinematic model developed in Section 5.1.1 will require the processed MacReflex® position data in order to evaluate the independent variables and their relationship to the dependent variable of hand coordinates $(x_w,\ y_w)$. Specifically, the independent variable is tangential velocity of individual movement tasks. The kinematic model can be constructed from the positional data alone in order to find v_t. In addition, the processed EMG from the eight muscles will provide the activation onset, duration, and amplitude information for the different movement tasks. To get to these variables the data were analyzed using software models developed in Matlab™, an analytical software package developed by Mathworks Inc. Natick MA.

It is important to this study and future work to know the stage of disease progression each of the subjects is at when recruited. This is particularly true if the data or results are to translate to larger populations. McDonald et al. (1995) conducted a ten-year study of 162 patients with DMD to develop a profile of impairment and disability based on manual muscle testing (MMT). Cohen et al., (1982) also published a statistical analysis of muscle strengths attained via MMT by the same rater of 12 subjects over minimum of 41 to a maximum of 84 months. These studies provide a reasonable method for comparing populations at large with the individuals examined.

5.1 Kinematic Marker Data and Arm Model

The MacReflex® data acquisition system comes with software that was developed by the manufacturer for the data collection. It also has a pre-processing capability of direct linear transformation of the coordinate data for each camera. This software was used to sort and export the positional data of the passive markers placed on the upper extremity for further analysis using Matlab. Before the position data were used to construct the kinematic model pictured in Figure 6, it was smoothed utilizing a cubic spline to remove the noise in the positional data. An example of the result is shown in Figure11.

5.1.1 Kinematic Variables

Atkeson (1989) states that the reason it is possible to attain a target in space even with the eyes closed (after viewing the targets position) is because there is an internal representation or model of the forward kinematic transformation from desired hand position to joint angles and muscle lengths. Motor control of the upper extremity can be viewed as a multi-tiered process requiring several transformations from a specific behavioral objective to a desired mechanical output of the motor apparatus and finally to a pattern of muscle activation. In other words the CNS system is faced with the problem of, given a desired hand position, determining what are the necessary joint rotations and corresponding muscle lengths needed to achieve that position. If this is the case then the DMD subjects should organize movement by utilizing the same principles as in the intact musculoskeletal system, at least initially. Invariance in the kinematic features of multijoint arm movements in DMD as compared to the healthy model would support this.

It is speculated herein that the nervous system formulates control of these movements in terms of the spatial hand trajectories. Therefore, this study treats the upper extremity

model as a mechanical device where the CNS centrally controls the movement by converting muscle lengths and joint angles into hand positions. The process the CNS uses to control the movement is treated as the forward kinematic transformation described above. However, the CNS control algorithm for human movement remains ambiguous so that it was not known if the forward kinematic solution cannot be applied directly. All the same, the mathematical formulation for the forward kinematic solution for the hand coordinates can be used to examine possible CNS control algorithms.

Take the arm model, which was depicted in Figure 6 as an idealized two-joint manipulator; therefore, a solution is needed for the hand coordinates $(x_w, \ y_w)$ in Cartesian coordinates. Assuming that movement plans exits, the forward kinematic solution is from

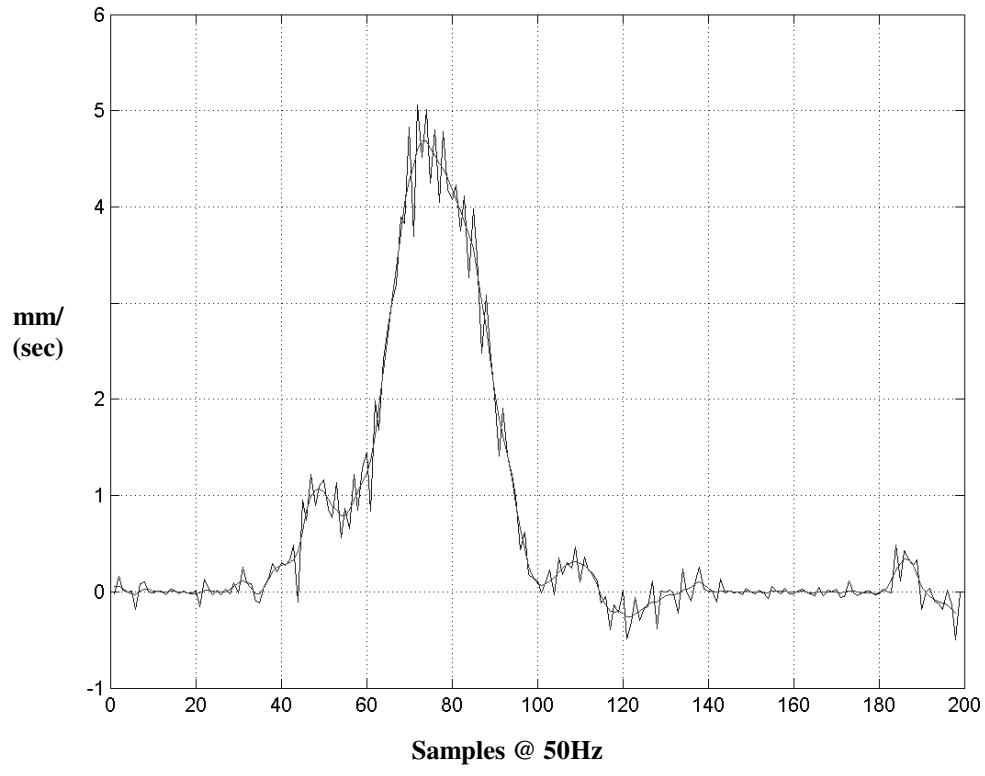


Figure 11: Hand velocity constructed with raw kinematic data over cubic splined data.

the joint angles to the hand coordinates formulated by Equation 5 and depicted as the hand path in Figure 13. All calculations are referenced to an orthogonal Cartesian coordinate system fixed in the shoulder as shown in Figure 6.

$$(x_w,\ \ y_w) = (L_1 * \cos(\theta_s) + L_2 * \cos(\theta_s + \theta_e),\ \ L_1 * \sin(\theta_s) + L_2 * \sin(\theta_s + \theta_e)) \qquad \textbf{Equation 5}$$

Since the position of the wrist, elbow, and shoulder are known (monitored during experiments) the *forward kinematic* transformation can be adapted as an *inverse kinematic* transformation to determine the joint angles (*cf.* Atkeson, 1989; Hogan et al., 1987). The coordination of movement and rates of change between both joints is important to the shape of the hand trajectory. When the joint angles are known the position of the hand can be found directly; however, the same cannot be said for the inverse. This means there are an infinite number of joint orientation combinations that the elbow and shoulder can exhibit for any given position. In other words, there is a mapping from the joint space to the workspace that can be defined as a Jacobian matrix. The indeterminacy of the system results in a Jacobian kernel that is not zero and therefore, has no inverse (*cf.* Gielen et al., 1997, Anton, 1994). The result, i.e. the hand position, can correspond to an infinite set of joint angles to attain any desired hand position. What's more, there is no instruction as to which set of these joint angles the CNS selects in order to attain a given position in the workspace. Fortunately, because the positions of the joints were monitored, their exact orientations (within an acceptable error) due to movement are known. The change in joint angles θ_s and θ_e from the start position of the arm segments were found by using the known hand coordinates $(x_w,\ \ y_w)$ in Equation 6, the inverse kinematic solution. The inverse kinematic transformation model

is necessary to all calculations. In the following sections it will be used as a means to examine the hand trajectory.

$$(\theta_s,\ \theta_e) = \left(\tan^{-1}\left(\frac{y_w}{x_w}\right) - \tan^{-1}\left(\frac{L_2 * \sin(\theta_e)}{L_1 + L_2 * \cos(\theta_e)}\right),\ \cos^{-1}\left(\frac{x_w^2 + y_w^2 - L_1^2 - L_2^2}{2 * L_1 * L_2}\right)\right) \quad \text{Equation 6}$$

A requirement of the spatial motor control thesis is that the hand trajectories be roughly straight while executing point-to-point hand movements. When speaking of trajectory here it refers to the path that the hand follows from point-to-point and the velocity at which the hand moves along that path. Morasso (1981) was first to examine the control of two-joint movements in healthy normal adults, investigating the trajectory of the hand during movements about the elbow and shoulder joints. For such movements the curve plot representing the hand velocity is typically bell shaped and unimodal with hand acceleration and deceleration for half the movement time. From the definition of hand trajectory above and using the hand position, $(x_w,\ y_w)$ from Equation 5, then it follows that Equation 7 is the instantaneous tangential velocity of the hand.

$$V_t = \sqrt{\dot{x}_w^2 + \dot{y}_w^2} \quad \text{Equation 7}$$

It was hypothesized that the variance from straightness will be substantially smaller when the subjects perform target acquisition in the unconstrained trunk configuration as opposed to the constrained. If this were true, it would satisfy part D of the hypotheses developed in the introduction. Assuming there is some repeatability in the hand trajectory this ratio should be smaller for more coordinated or well-controlled movements.

$$R = \frac{\left(\dot{X}_w^{\,2} + \dot{Y}_w^{\,2}\right)^{\frac{3}{2}}}{\left|\dot{X}_w * \ddot{Y}_w - \ddot{X}_w * \dot{Y}_w\right|} \quad \text{Equation 8}$$

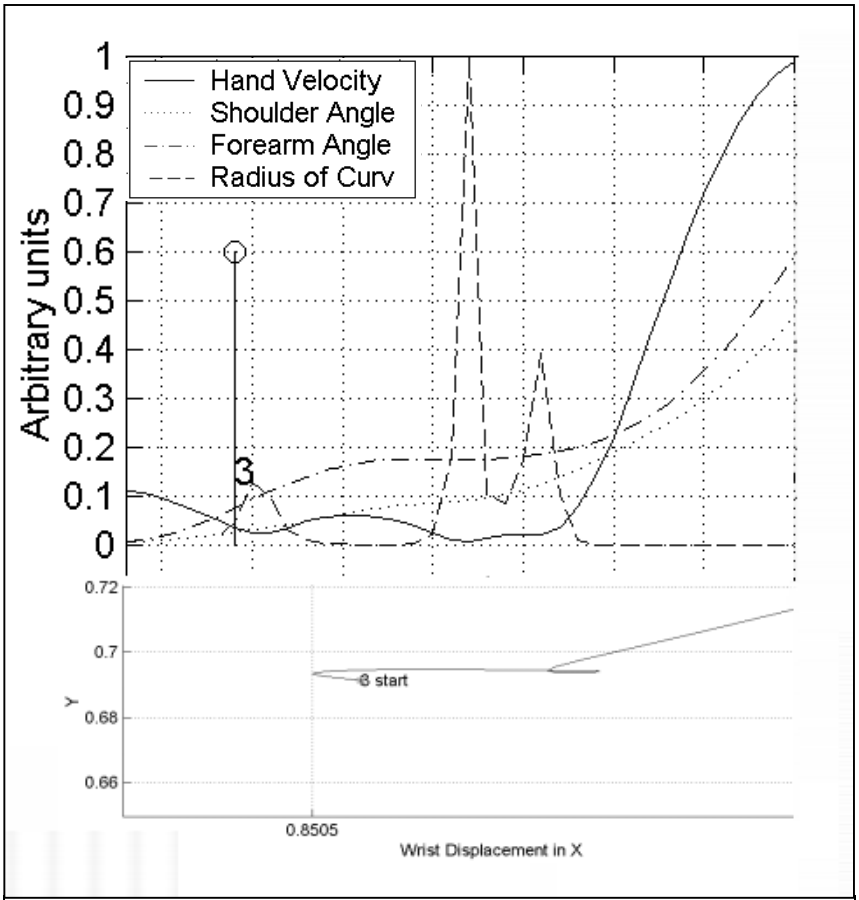


Figure 12: Pictured is a choice of hand movement onset (pole marked 3) after which the start of the movement has large curvature due to infinitesimal changes in the hand path shown. However, the changes in the direction of the trajectory are in the 0.01 mm range, below the resolution of the system kinematic data acquisition.

During the quasi-static holding in the start and stop positions, small hand movements and shifts in posture occur (or vibration of the marker etc.) causing instantaneous changes in the trajectory to appear as highlighted in Figure 12. This means the hand velocity may never really appear to be at zero while trying to maintain the 'start and finish' positions. When examining curvature of the hand path these instances when the hand is not at rest will appear as points of high curvature. This makes a gently curving path appear straight in comparison, this is seen in Figure 13 where the point of highest curvature does not really represent the curvature of the portion of hand trajectory of interest.

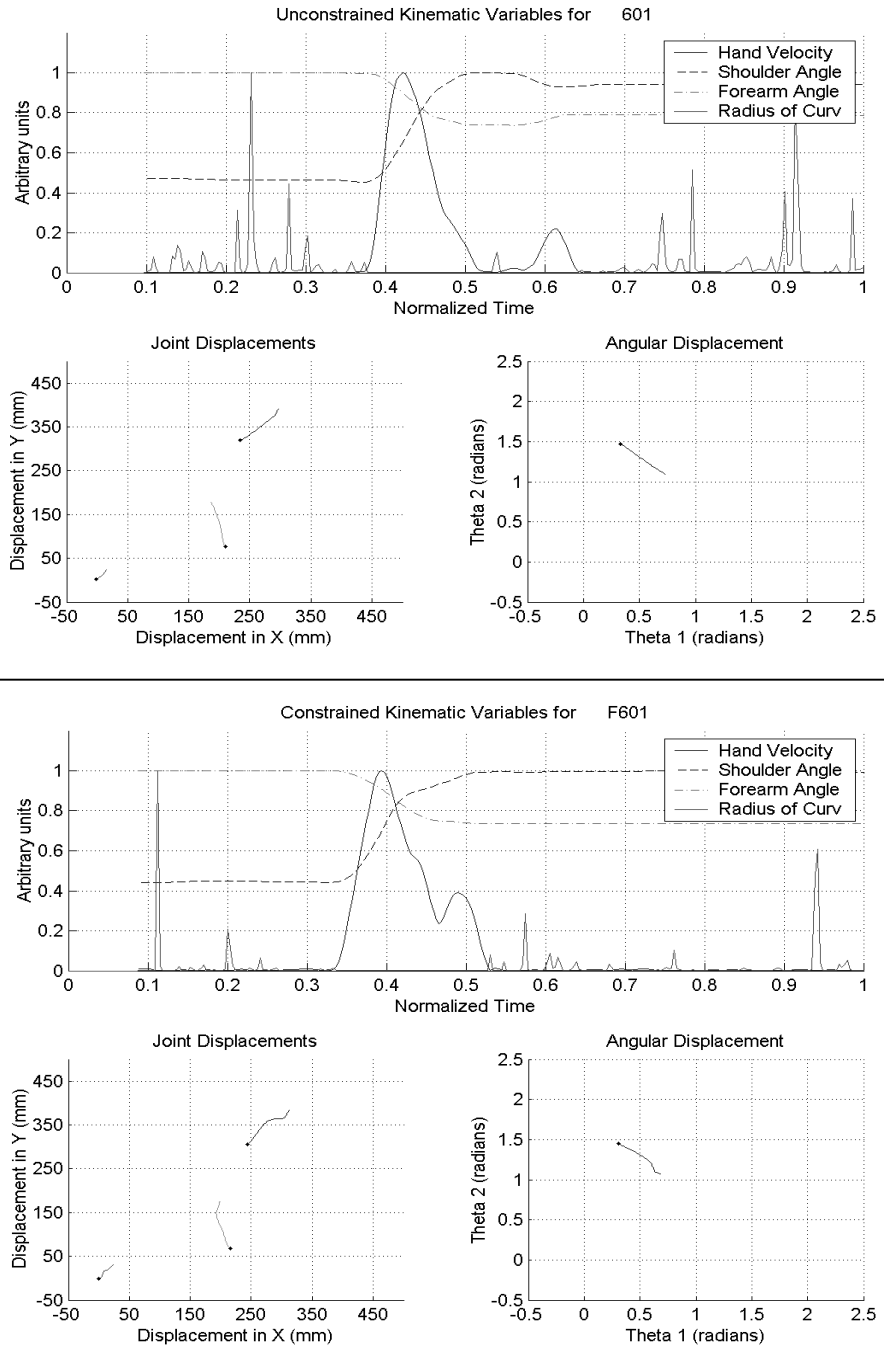
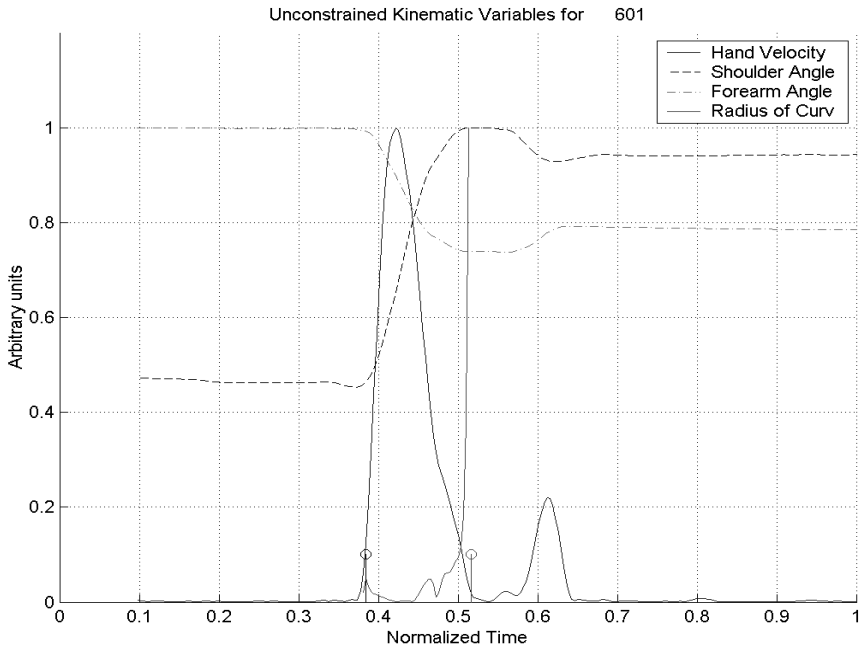


Figure 13: Kinematic parameters normalized to maximum value and time for subject-4 of the first trial while reaching to the target at the 60˚-degree attitude with the trunk unconstrained and constrained. A dot marks the start of the linear and angular displacements producing the kinematic variables. The radius of curvature being produced before and after hand motion is due to infinitesimal directional changes as in Figure 12.



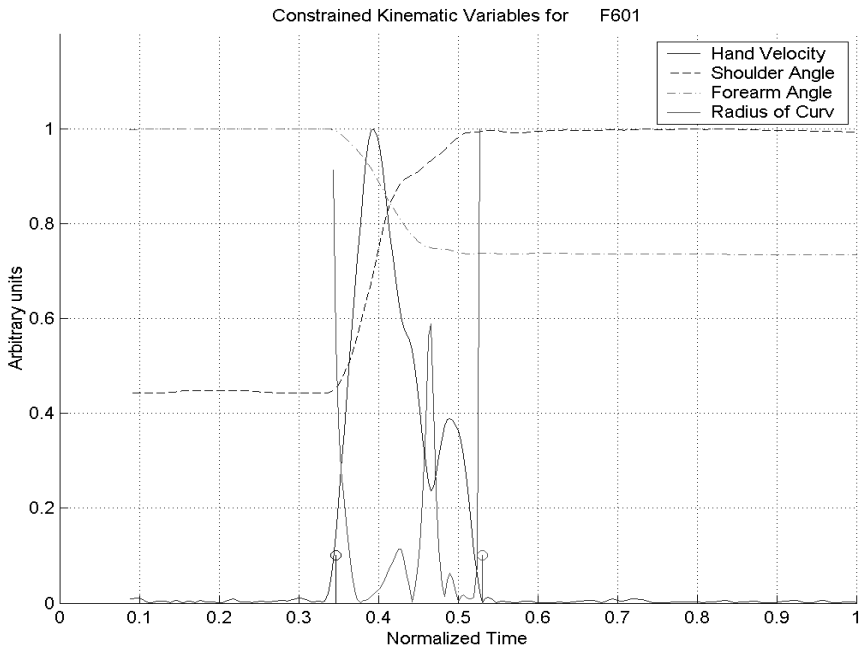


Figure 14: Kinematic results of subject-4 for the first trial while reaching towards the target at the 60˚-degree. The radius of curvature plotted in this figure starts and terminates when the hand velocity first exceeds and slows to 0.4 mm/s marked by vertical lines topped with open circles. In Figure 13, there are more infinitesimal rotations in the unconstrained condition as opposed to the constrained. In addition, it is visually clear that the unconstrained trajectory is straighter than the constrained in Figure 13 confirmed by radius of curvature values.

Onset/end of hand movement was determined as the instant when the tangential hand velocity exceeds 0.4 mm/s (resolution possible with MacReflex is .4 mm). In these figures, the data are normalized to the variable's maximum value and in time. The threshold velocity chosen for determining onset of movement was contingent on several factors. First, because the air bearing supports the arm the test subject must maintain the start position without changes in posture. This is not always possible such that small changes in velocity and direction occur. At the start of movement, 0.4 mm/s worked well but termination of movement was not as clear, again because of the inability to maintain a fixed position once the target is reached. For this reason, the value of the termination velocity was typically 0.4 mm/s but at times ranged up to .6 mm/s. The radius of curvature that is of interest lies along the portion of the trajectory that is between these points as seen in Figures 14.

Angular rotations of interest at the elbow and shoulder joints also occur between the onset/end points. However, changes in joint orientation do occur before the start of movement and after the end of movements in some subjects because of postural adjustment. In addition, acquisition of a target did not always occur in a single movement but in segments with multiple velocity profiles as in the case illustrated in Figure 15. This required that the selection of hand velocities be visually inspected after the placement of the onset/end markers to ensure that the kinematic parameters from the portion of trajectory that is of interest were included.

Special attention was paid to the selection of onset/end points because their choice determines the period over which the statistical analysis on the kinematic parameters is done. Poor choice of these onset/end points will generate skewed statistical results; an

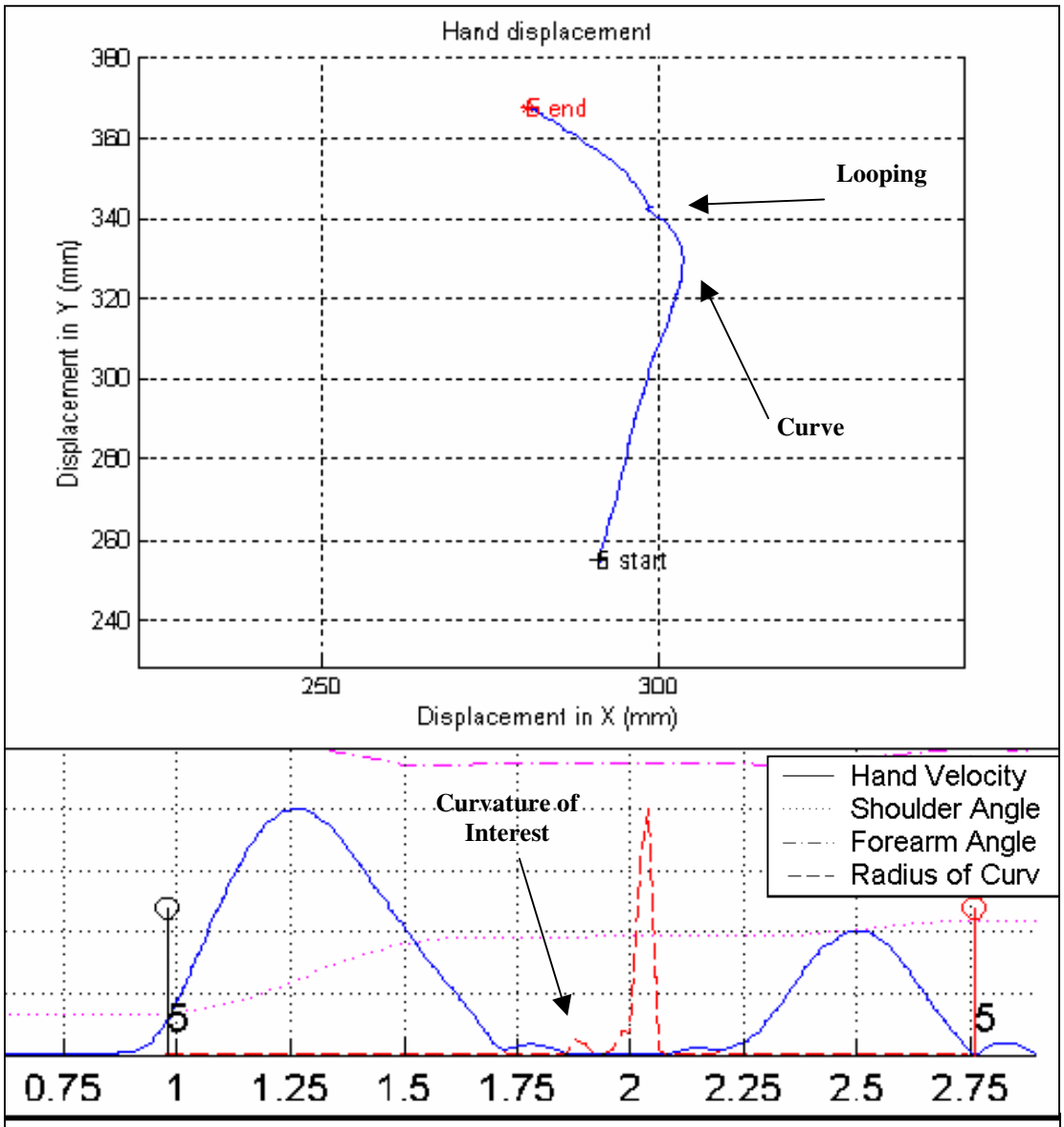


Figure 15: The graph depicts a target acquisition trajectory marked at the start and end of movement, the corresponding values for the hand velocity, changes in theta 1 and theta 2, and the radius of curvature. Note that the movement is executed with segmented movements producing two distinct 'straight' paths. The largest radius of curvature occurs in the velocity valley as hand makes small changes in trajectory by looping or shifting rearward and forward along the trajectory's path. The *curvature of interest* for the *curve* in the path is marked above; its radius appears smaller than it should due to the magnitude of the 'infinitesimal looping' radius. This 'looping' is not done to change the path of the hand but within its trajectory as above. All the subjects produced this looping on occasion but its appearance was irregular.

examination of Figure 15 will illustrate the point. In the effort, the movement was made in two segments; approximately two-thirds through the movement there is a momentary stoppage and then resumption of movement. Visual inspection was done to ensure that the entire movement was considered.

It has been suggested herein that a possible method for quantifying the quality of movement is to investigate the radius of curvature of the hand trajectory as in equation 8. However, an initial examination of the results has placed doubt on this concept such that another method must be sought. The method proved adequate when the hand path was essentially straight and made in a single movement. However, at times, the DMD subjects made halts, reversed direction, or loops before continuing along the *desired* trajectory as in Figure 15. No pattern was identified in the appearance of this feature between subjects or conditions. The velocity valleys usually occur over shorter periods than the one in Figure 15 but they always occurred during deceleration near the end of the movement. This 'looping' when present obscures the actual value for radius of curvature of interest. It could be possible to filter such perturbations out of the trajectory but this feature may point to a breakdown in the bio-controller.

A coupling of the curvature and the speed of movement has been shown to exist while tracing patterns in a plane (Lacquaniti et al., 1983; Pollick and Sapiro, 1996; Viviani and Flash 1995), where the hand velocities are slower for more curved paths. Furthermore, it has been suggested by others that joint angular velocities are the rule by which the CNS accomplishes movement planning (Viviani and Cenzato, 1985; Viviani and Flash 1995). Lacquaniti et al., (1983) point to the linear relationship of the angular velocities as

evidence that there is some consideration given to the coupling of the elbow and shoulder joints.

The model's kinematic linkage representing the arm develop arm movements that involve elbow and shoulder joint rotations confined to the horizontal plane, as illustrated in Figure 6. Because the arm motion is restricted to the horizontal plane, the only changes in joint rotations are single axis rotations that occur about the Z-axis of each joint.

This means the angular velocity and acceleration vectors vary in size but the direction is constant, dependent only on the joint angle change of, θ_s and θ_e calculated in the inverse kinematic model. The angular velocity and acceleration determined by taking the time derivative of θ_s, and θ_e, are $\Omega = \left(\frac{d\theta_s}{dt}, \frac{d\theta_e}{dt} \right) = (\dot{\theta}_s, \dot{\theta}_e)$ and $\dot{\Omega} = \left(\frac{d^2\theta_s}{dt^2}, \frac{d^2\theta_e}{dt^2} \right) = (\ddot{\theta}_s, \ddot{\theta}_e)$ respectively. Target acquisitions generate shoulder and elbow joint rotations that produce a constant ratio of angular velocity during movement in a planer space in healthy subjects illustrated in Figure 16 (Lacquaniti et al., 1986; Gielen et al., 1997). Therefore, it may be that a quantitative measure of effective control is the correlation coefficient from the linear regression of the ratio of angular velocity.

Examining the data in Figure 16 qualitatively the ratio of angular velocity appears to follow the published trend in both the constrained and unconstrained trunk test condition. If there is an argument that movement is planned and executed in the joint-space it could be made by showing little variability exists in the linearity of the angular velocity ratio. This could also be the case in the instances of improved linearity between the constrained vs. the unconstrained trunk configuration.

A comparison of correlation coefficients of least squares fit lines highlights the usefulness of this quantity as a measure of movement quality and control as demonstrated

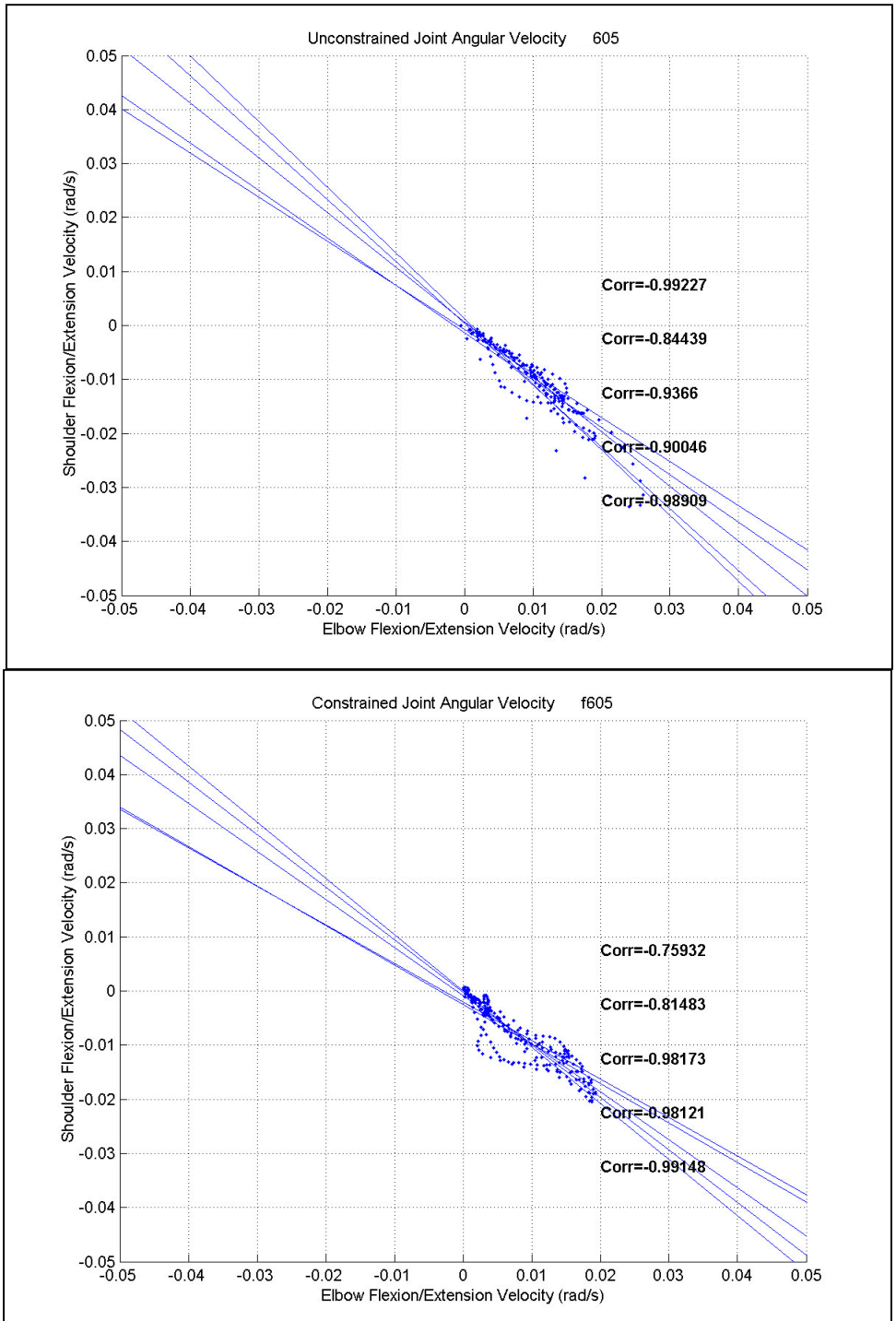


Figure 16: Angular velocity of the elbow vs. shoulder for the constrained (top) and unconstrained (bottom) trunk test condition. Plotted are five trials of reaching for the target at the 60-degree attitude by subject-4 for each condition. (Data corresponds to data presented in Figures 12 and 13.

in Figures 16. Moreover, improvement in the correlation coefficient in the unconstrained trunk condition over the constrained reveals the desire to maintain a constant ratio of angular velocity. Since effective control of movement is in part, issues of strength mechanical compensations are employed in order to adapt to weakness in the upper extremity muscle groups.

5.2 EMG

The BioResearch™ EMG amplifiers systems have a fixed collection rate of 3000Hz with a fixed gain of 5000 which far exceeds the limits required. EMG data were rectified, low pass filtered with a Butterworth 4^{th} order filter with a cutoff frequency of 12Hz, see example in Figures 17 and 18. Not all the EMG recordings are going to be useful as a determinant of movement direction due to problems in the recording of the signal as revealed in Figures 17 and 18. This is regardless of whether the failure is due to motion artifacts, loss of skin contact, thickness of adipose tissue, ambient 60Hz noise or lack/level of action potential propagation, when occurred the erroneous EMG data were discarded from further use.

The popup marked used to time coordinate the EMG and the kinematic variables. By eliminating the kinematic data recorded before the appearance of the popup marker, the kinematic and EMG data are coordinated to the same time line.

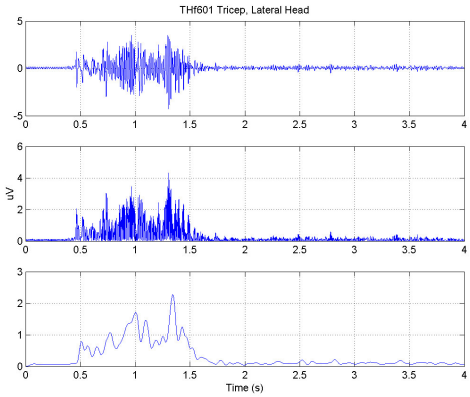


Figure 17: Result of the EMG processing is an enveloped EMG corresponding to the kinematic results (see Figure 12). Activity of the lateral head of the triceps while moving towards the 60-degree target with the trunk constrained.

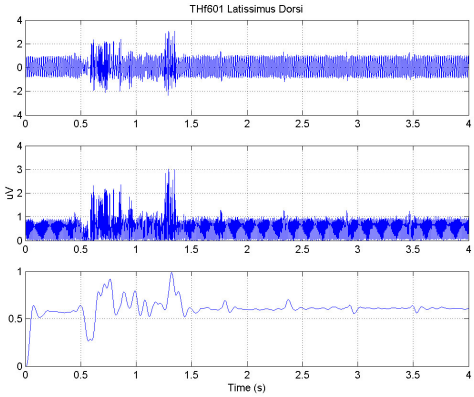


Figure 18: Result of the EMG processing is an enveloped EMG corresponding to the kinematic results (see Figure 12). The activity of the latissimus dorsi was recorded in conjunction with the lateral head of the triceps in Figure 17, note the 60 Hz noise visable in the signal.

CHAPTER 6: RESULTS AND DISCUSSION

In this study, seven subjects with DMD were asked to make pointing movements from two start positions with the arm abducted 90˚ degrees and the forearm flexed 90˚ degrees and while pointing at the target at the 150˚ attitude. Movements were made towards targets located at attitudes of 30˚, 60˚, 90˚, 120˚, and 150˚ degrees in the sagittal plane from two test configurations, a constrained and unconstrained trunk. Each target was to be acquired five times from each start position with no speed or accuracy requirements.

Given the pathophysiology of DMD, it is not surprising that some of the subjects were not able to complete all the study tasks. Subject-1 and three were the weakest of the subjects tested according to the manual muscle tests results presented in Table 2. These subjects were physically exhausted by the challenges of the tasks additionally; these subjects had the most severe of joint contractures (diagnosed by the medical staff of A.I. duPont Muscle Clinic), which prevented them from being able to reach all the targets. Consequently, they were not capable of making reaching movements from the 150˚ degree start position to the other targets, all the same they made 30 pointing movements. However, these individuals would benefit most from the proposed robotic orthosis and their effort provided information on the movement capabilities and characteristics in DMD in the later stages of the disease.

Subject-4 although capable of reaching the target located at 150˚ from the origin the subject asked to stop because of the exhausting effort; therefore, this subject completed all the trials with the exception of the 150˚ target acquisition from the origin. Subjects-5, and subject-7 were able to complete all the tasks without any trouble. In addition, even though subject-2 made an effort to acquire the 60˚ and 120˚ in the constrained trunk

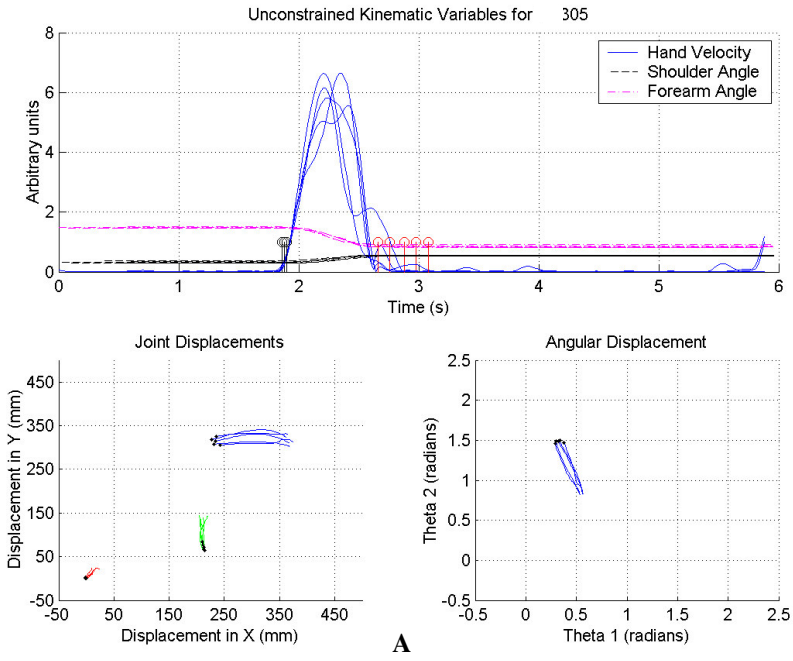
position from both start positions he did not make movements of adequate distance to be included in the evaluation.

According to the manual muscle tests presented in Table 2, subject-6 should have been capable of completing the entire exam without difficulty; however, this was not the case. While executing the pointing movements from the fixed trunk position the subject made an error in the pointing pattern. Rather than reaching for the 120˚, 90˚, and 60˚ from the 150˚ degree target position he pointed to the 30˚ degree target from each of the remaining targets. He was not asked to repeat any of the tasks due to fatigue issues asked to stop after he had completed the 30˚ degree acquisition from the 150˚ degree position. He was asked if he could acquire the 30˚ degree target from the 120˚, 90˚, and 60˚ degree targets at least once and he complied. It is likely that determination and boredom were more an issue than strength in completing the tasks based on verbalizations of the subject.

Tables containing the individual statistics for the five trial of each target position are presented in Appendix I.

6.1 Kinematic Features and Movement Strategies

For stereotypical pointing movements in healthy individuals, essentially straight hand paths are generated while producing bell shaped velocity profiles. The hand paths created by the DMD subjects tested were also essentially straight with bell shaped velocity profiles and most appear similar to the results seen in Figures 19A-B and 20A-B. This evidence supports the line of reasoning that the CNS is planning the movement in the hand-space. In other words, in the hierarchy of movement planning, the hand is of primary importance (Kaminski and Gentile, 1989).



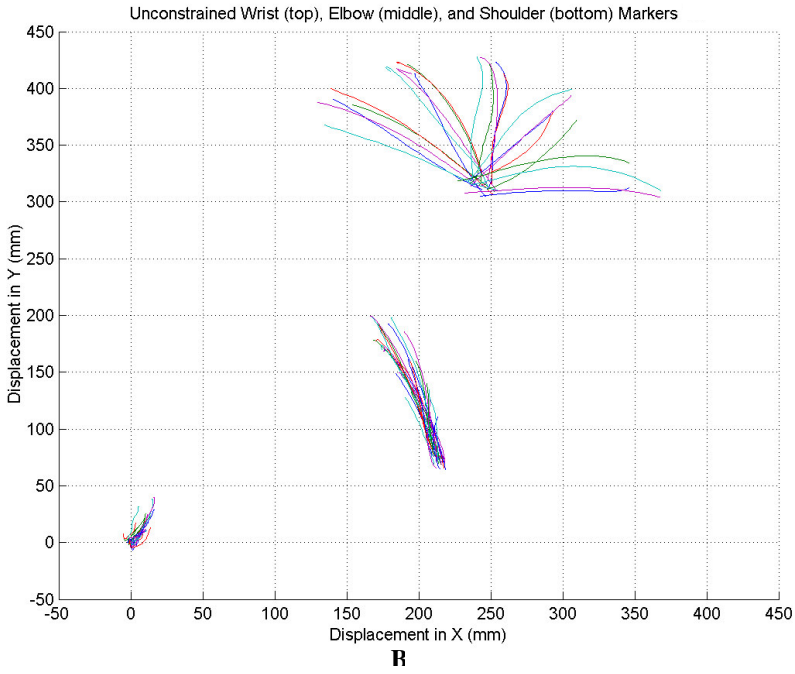
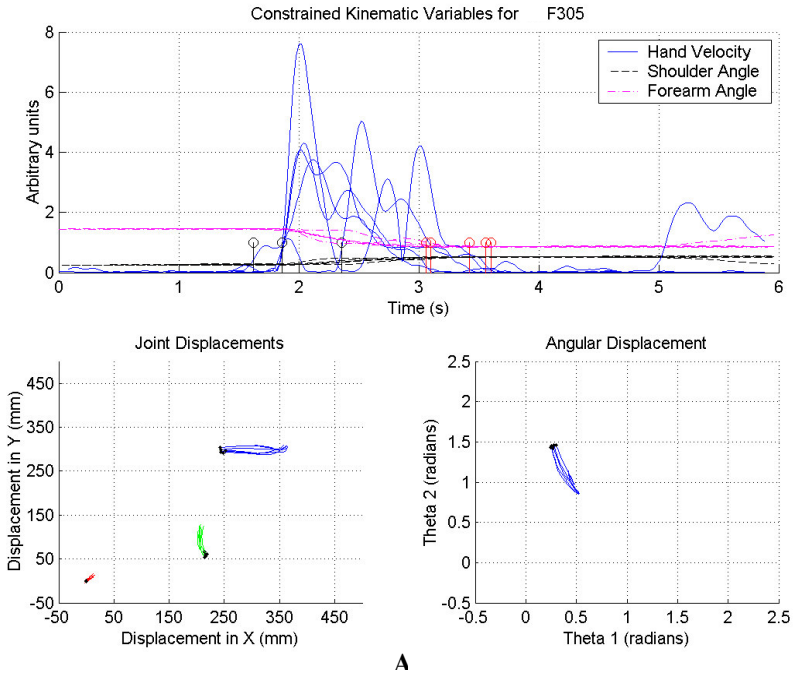


Figure 19: Results of pointing movements made by subject-4 in the unconstrained trunk condition. Velocity profiles are marked at 0.4 mm/s at the start and end of the five pointing movements made from the origin to the 30˚ degree target are presented in A. The corresponding change in θ_1 and θ_2 (dot marks start) are presented along with the displacement at the joints. In B, the hand, elbow, and shoulder paths created while making pointing movements from the origin to each of the targets.



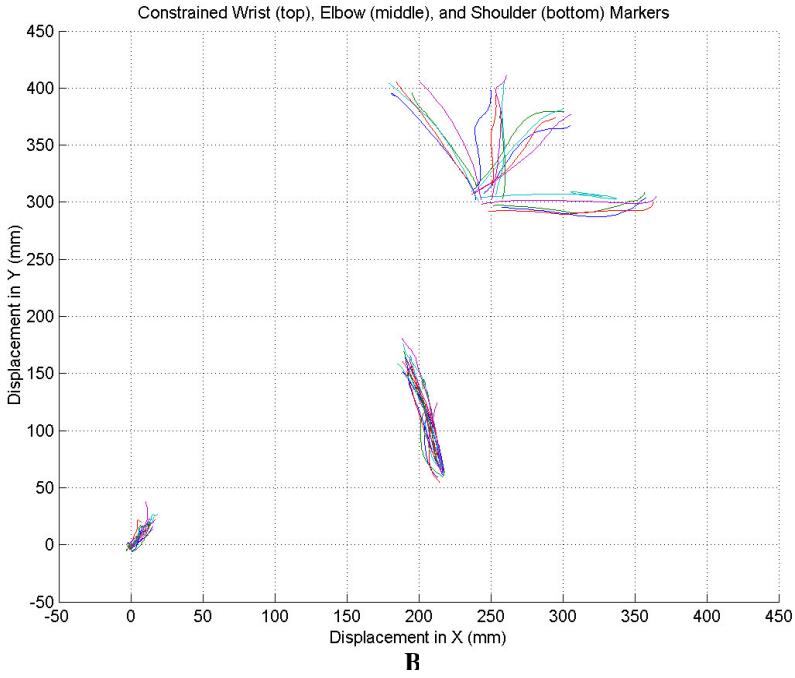
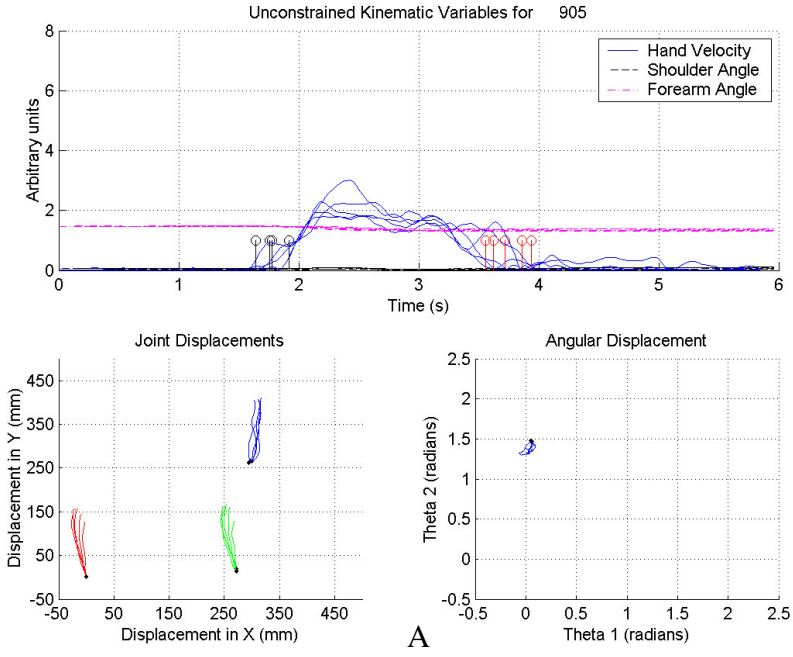


Figure 20: Results of pointing movements made by subject-4 in the constrained trunk condition. Velocity profiles are marked at 0.4 mm/s at the start and end of the five pointing movements made from the origin to the 30˚ degree target are presented in A. The corresponding change in θ_1 and θ_2 (dot marks start) are presented along with the displacement at the joints. In B, the hand, elbow, and shoulder paths created while making pointing movements from the origin to the targets located at 150˚, 120˚, 90˚, 60, and 30˚ degrees.

Notice that the hand path and velocity profile features is ‘controlled’ better in the unconstrained (Figure 19A-B) then in the constrained trunk condition (Figure 20A-B). The claim that CNS planning occurs in the hand-space is supported by the fact that with an increase in degrees of freedom (i.e. trunk rotation-translation), there is improvement in the quality of the kinematic features. Moreover, this suggests that the CNS makes adaptations for weakness to maintain movement along the desired or planned trajectory. Kaminski and Gentile (1989) put forth the notion that there is a subordinate joint planning strategy in the management of movement. The results presented in Figures 19 and 20 imply that adaptation and/or compensation for weakness is being made by the DMD subjects in the joint space providing further evidence of this subordinate relationship.

Subject-3 had the most severe restrictions of movement attributed to the diagnosed joint contractures. Arm stiffness is further compounded by weakness, which must have an effect on performance. Alterations in the mechanical properties of the muscles (i.e. viscous and elastic) as well as impairment of the reflexes must be dealt with by the CNS in order to produce a desired movement. Additionally, the muscles about a joint must be coordinated in their agonist and antagonist role to maintain the necessary joint torque needed to develop motion in the desired direction.

The stiffness within the musculature and joints of subject-3 was severe enough that it prevented him from attaining the targets at the 60˚ and 30˚ attitudes in both the constrained and unconstrained test configurations. He could attain all the targets but his reach was restricted given the constraints placed on his movements by the test conditions. When reaching towards the 90˚ degree target subject-3 developed a velocity profile with



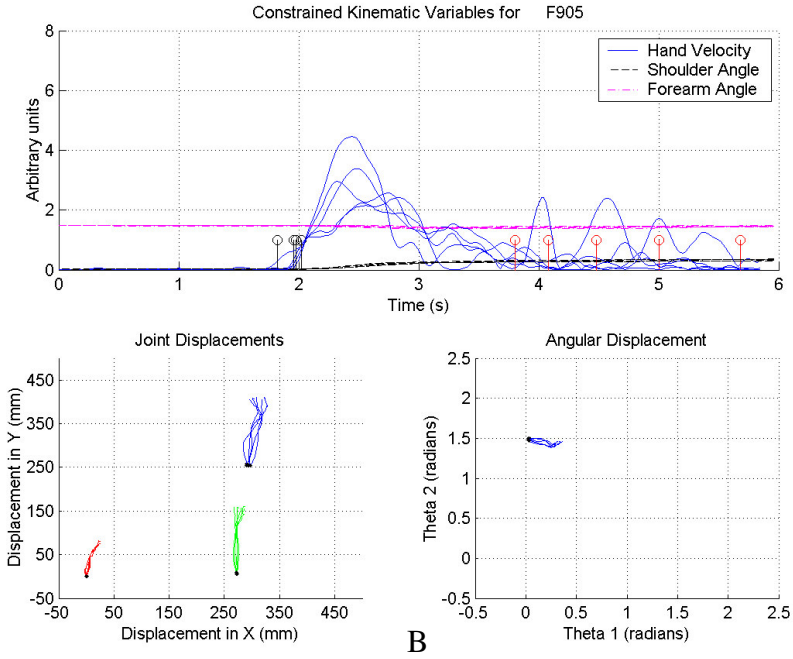
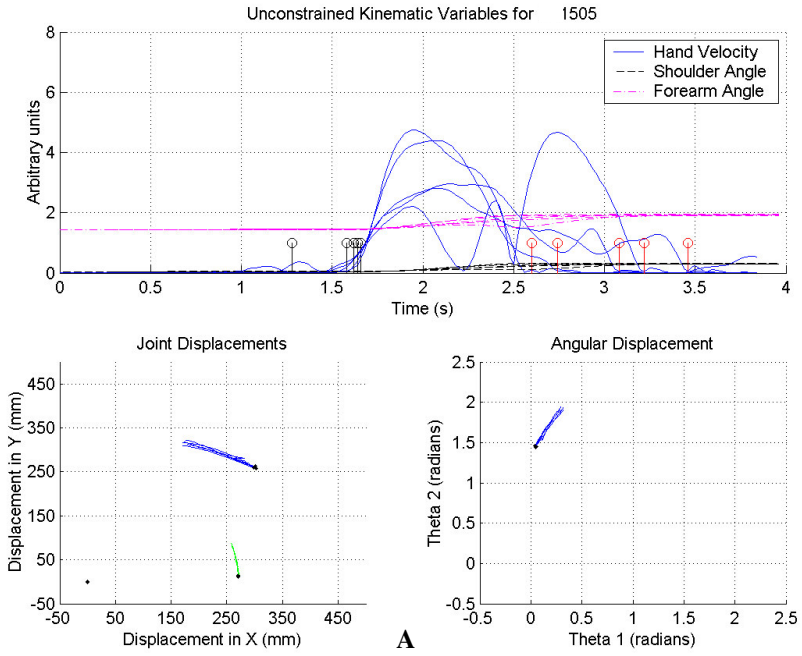


Figure 21: Kinematic features of subject-3during acquisition of the 90˚ degree target in the constrained an unconstrained trunk condition. In unconstrained configuration (A), there is very little contribution to the movement via the joint rotation. However, in the constrained trunk configuration (B) the joints are rotated to achieve the target. The shoulder angle θ_1 changes the greatest in (B) due to joint contracture about the elbow joint that restrict its rotation.

a peak that is essentially flat throughout the movement. Examining the plot of θ_1 vs. θ_2 in Figure 21A-B it can be seen that during the restricted trunk condition the joint rotations were greater than in the unrestricted. Joint rotations in the arm were eliminated because the introduction of an additional of trunk rotation-translation allowed him to adapt to the limits in strength and joint mobility. This adds further support to the view that there is a hierarchy at work in the coordination of movement. But it also suggests that the adaptation need not take place in the arm musculature or joints.

In Figure 21A, the introduction of the additional degree of freedom allowed for forward movement of the arm by maintaining the joint position and leaning forward at the waist. However, while reaching across the body to the 120˚ and 150˚ degree targets there is little difference in the quality of the kinematic features as seen in Figure 22A-B. Moreover, the hand paths developed during acquisition of the 150˚ degree target shown in Figure 23 when examined for straightness remain essentially straight in both test configurations.

The compensation strategy used by this subject was also dependent on the location of the target in the workspace. The movement employed a compensation mechanism of increasing joint rotation at both the elbow and shoulder joints but this did not necessarily improve straightness of the hand paths as the results in Figure 23A-B illustrate. There is essentially no dramatic improvement in the quality of the velocity profiles in the 120˚ and 150˚ degree acquisitions as there was in the acquiring the 90˚ degree target position. In fact, a joint reversal occurred in the unconstrained configuration coincident with a direction change in the hand path during the 150˚ degree acquisition. This is not s



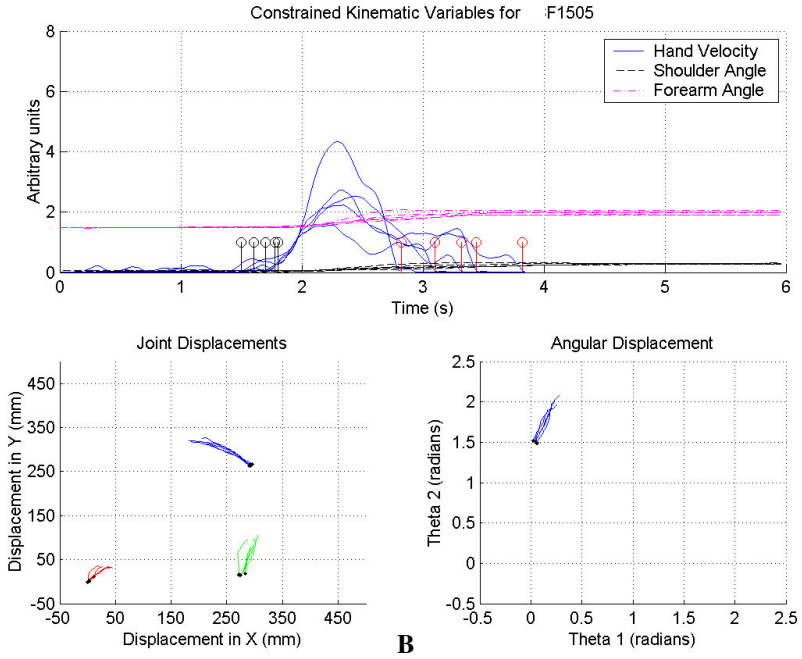
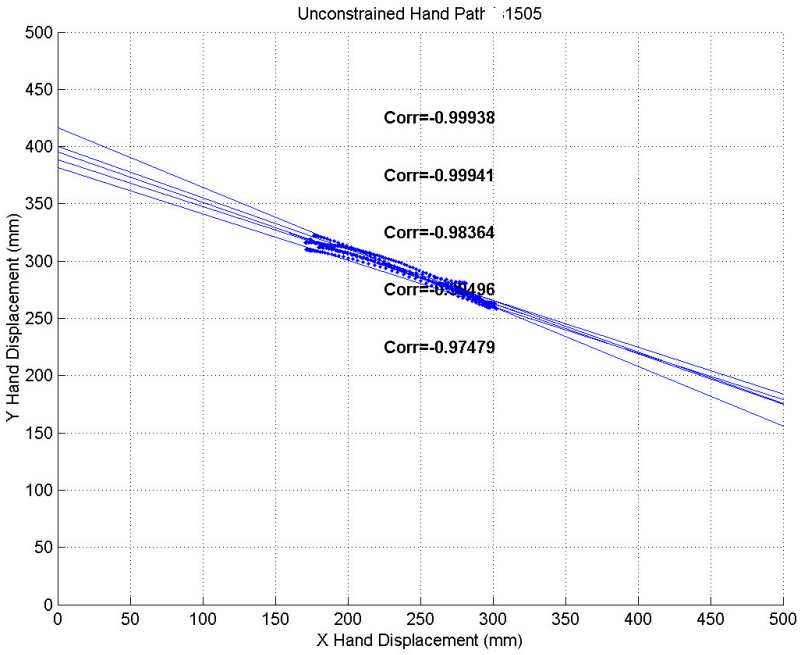


Figure 22: Kinematic features of subject-3 during acquisition of the 150˚ degree target in the constrained an unconstrained trunk condition. In unconstrained configuration (A), there is joint reversal coincident with a velocity valley. Joint excursions are of the same order of magnitude unlike in Figure 21A and B. Additionally, Hand paths are essentially unchanged, and the velocity profiles unimproved as was the case in Figure 23.

stereotypical of movements whether straight or curved. The importance of this observation will be discussed further later on in this section (Abend et al., 1982).

Thus far, the kinematic features of hand movement from near the body out into the workspace have been examined. However, the subjects also made pointing movement requiring them to reach across the workspace at the boundary of the target placement. A set of typical kinematic features of these movements are demonstrated in Figures 24A-B. Simple visual comparisons of the movements made out in and across the workspace were not always straight as were the pointing movements presented for movements made outward from the body.

During large pointing movements i.e. from 150˚ to 30˚ degrees, the path of the hand illustrated in Figures 25A-B and 26A-B remained essentially straight. This was the stereotypical result for all the subjects who made these movements. However, for shorter pointing movements from 150˚ to 120˚ degrees, such as those in Figures 27A-B and 28A-B, the paths trended towards a more curved path. During acquisition of the 120˚ degree target from the 150˚ degree target the timing of joint movement onset was not coupled. Movement onset of the forearm was first and continued until peak velocity was reached. Onset of upper arm movement followed the forearm starting midway between start and peak hand velocity continuing until the target was acquired (see Figure 27A-B) resulting in a curved plot of $\theta 1$ vs. θ_2. The joint that moved furthest (shoulder) did not necessarily move first as Kaminski and Gentile (1986) reported for healthy subjects. This difference may have more to do with method differences; they constrained the arm in a manipulandum constraining the shoulder joint than the identifiable implementation of compensation strategy by the DMD subjects.



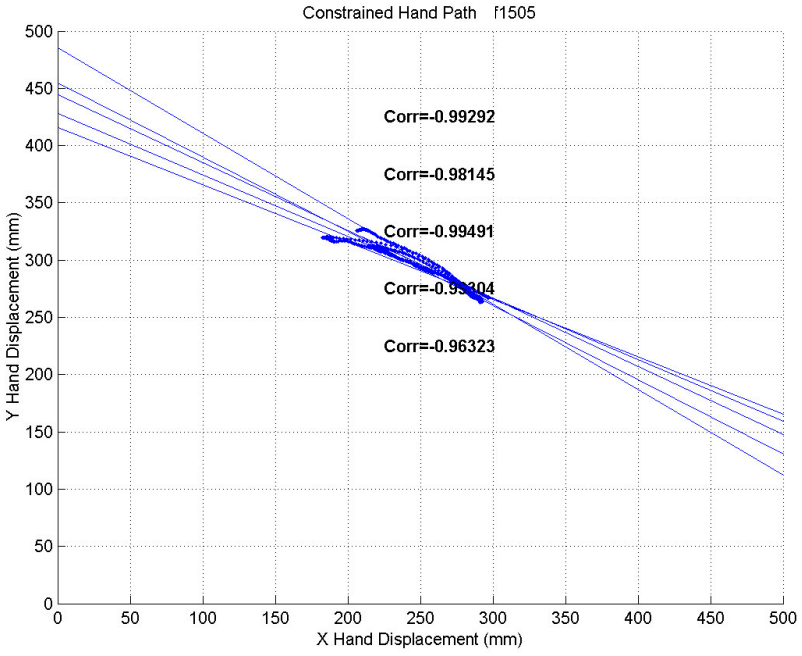
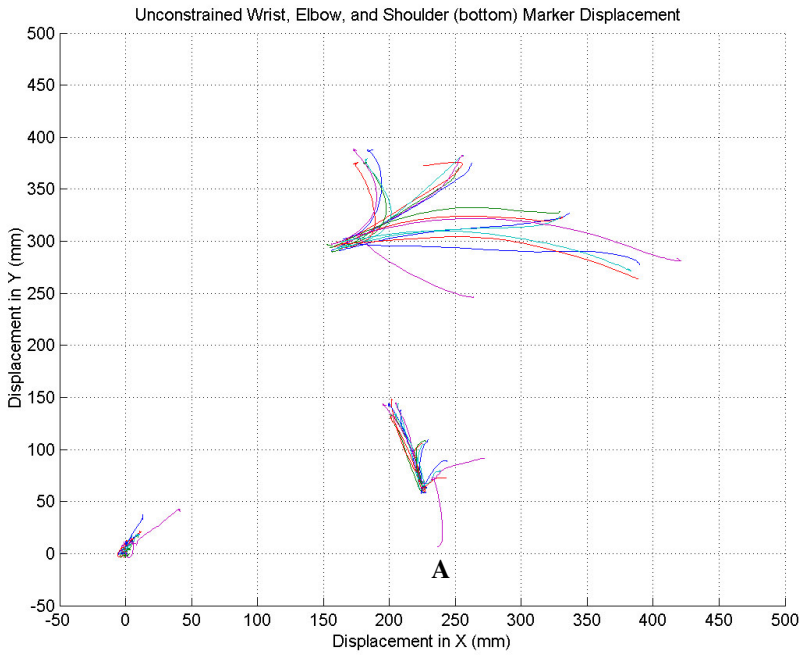


Figure 23: Correlation to straightness to a least squares fit line of each hand path for the trials presented in Figure 24A and B.



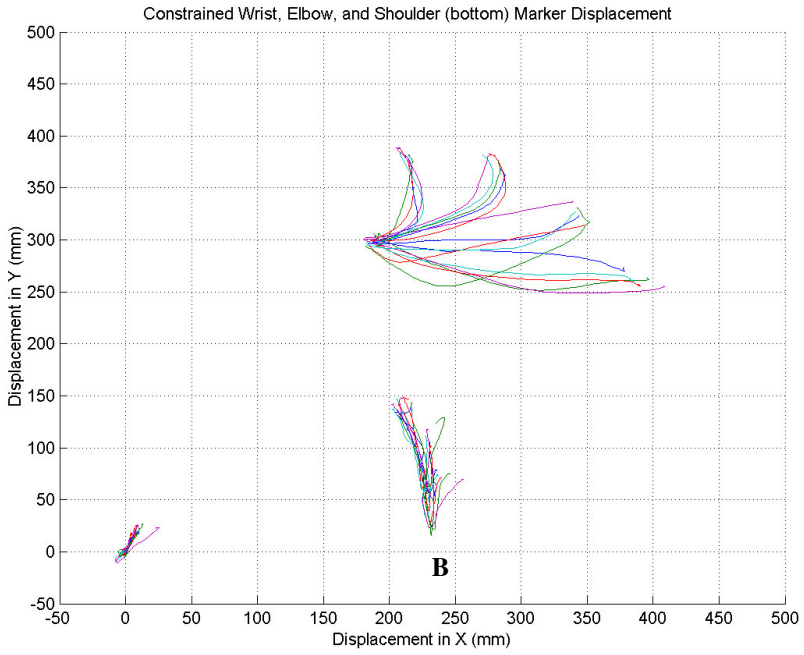
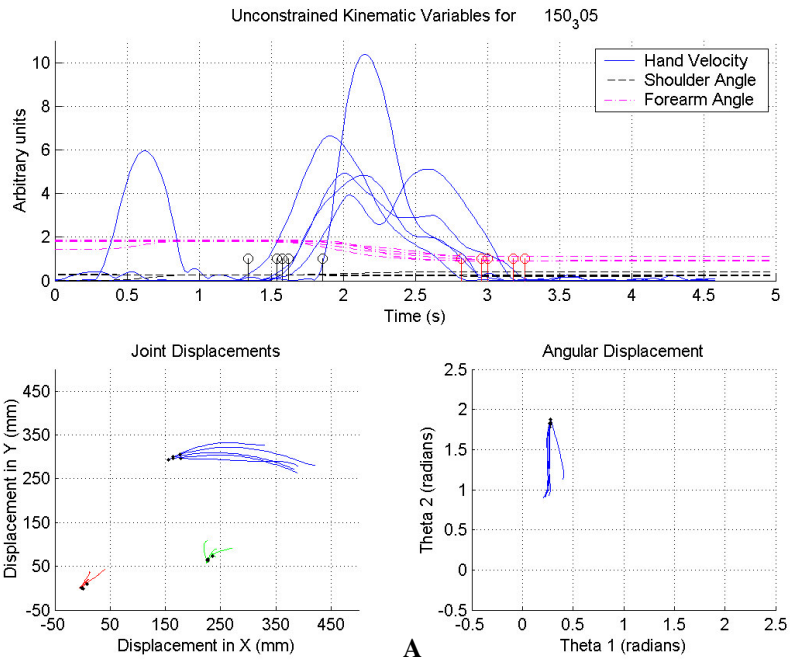


Figure 24: Hand paths develop by subject-5 while making pointing movements to each of the targets from the 150˚ target position. In both the constrained and unconstrained trunk configuration, the paths to the 30˚, 60˚, and 90˚ (correlation coefficient to a least squares fit line>.90) targets are essentially straight. Yet, both test configurations produced a curved path for the 120˚ target acquisition (see Figure 28).



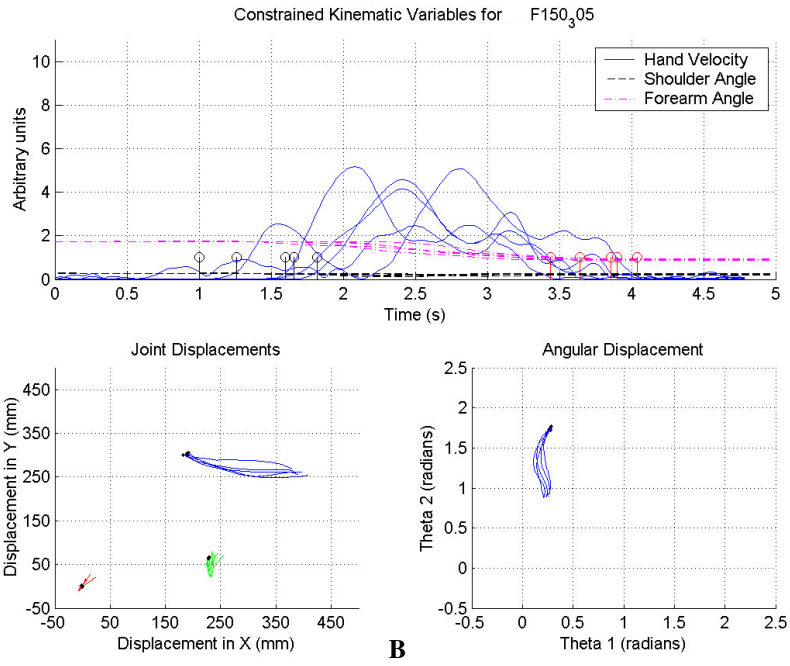
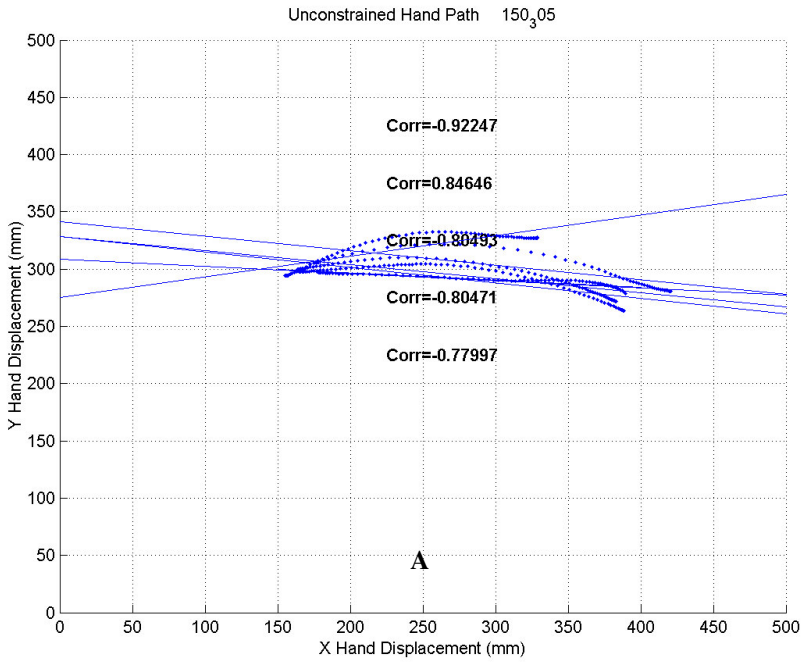


Figure 25: Pointing movements made by subject 5 starting from the 150˚ degree target moving towards the 30˚ degree target. Hand paths remain essentially straight (see Figure 26) and joint excursions are of the same order of magnitude. The correlation coefficient to a least squares fit line is .88 for the constrained and .83 for the unconstrained test configurations.



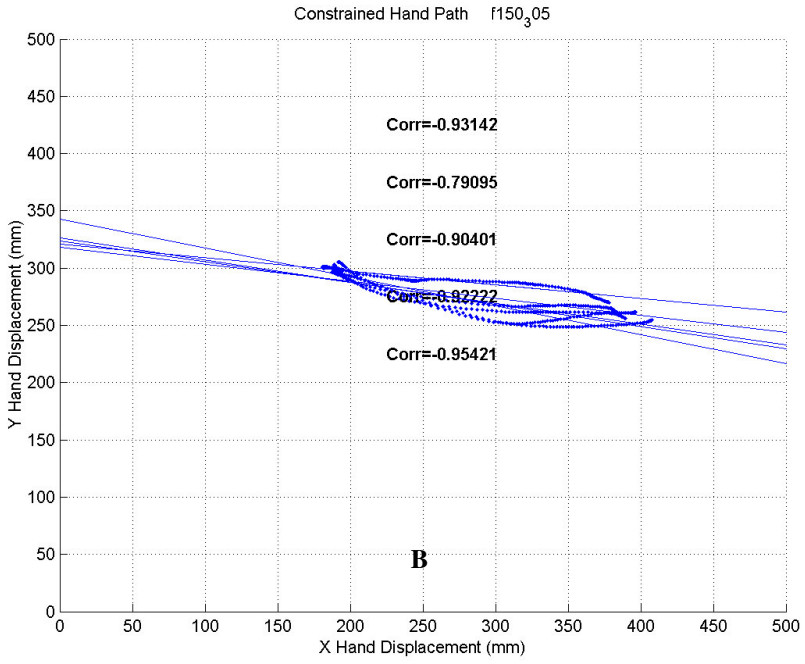
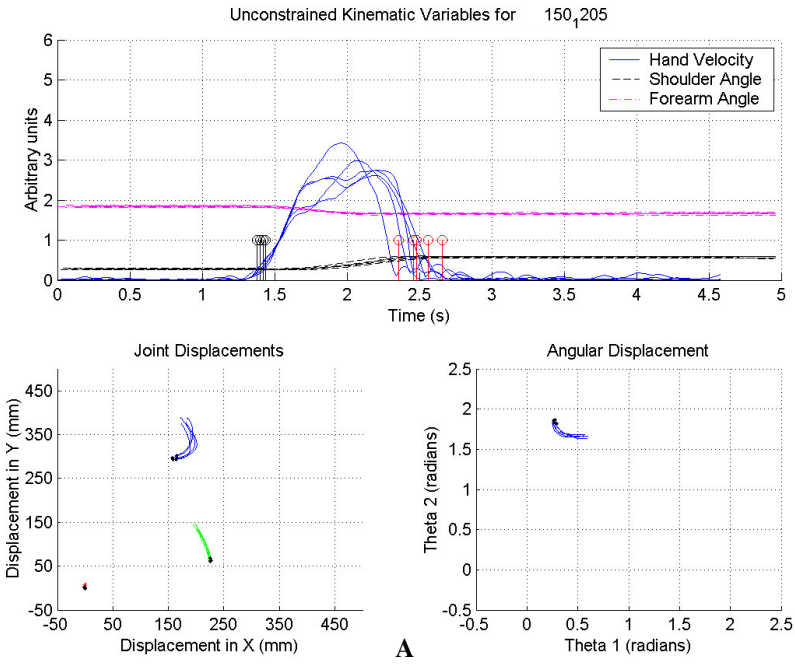


Figure 26: Target acquisitions from the 150˚ target to the 30˚ target made by Subject 5. All the DMD subjects that made these movements produce hand paths that were similar in nature to those presented above.



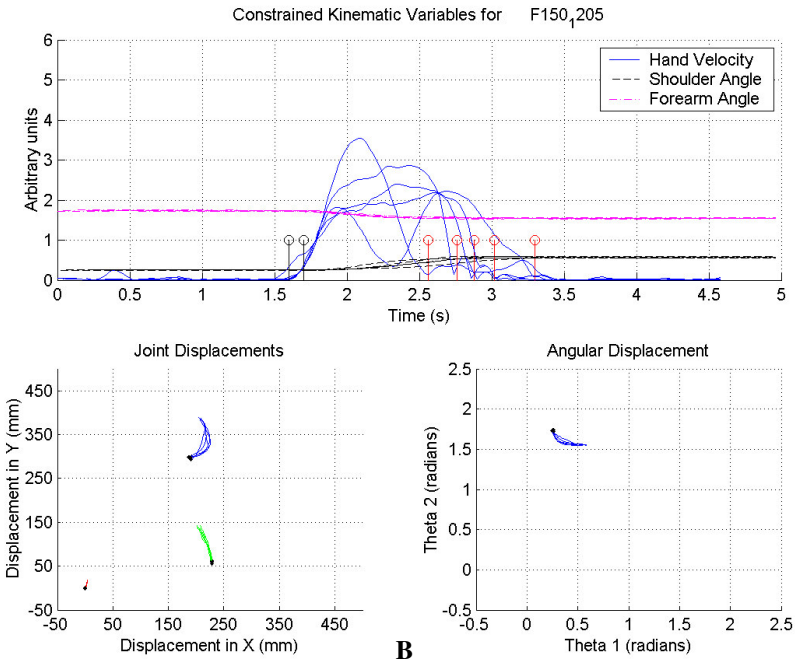
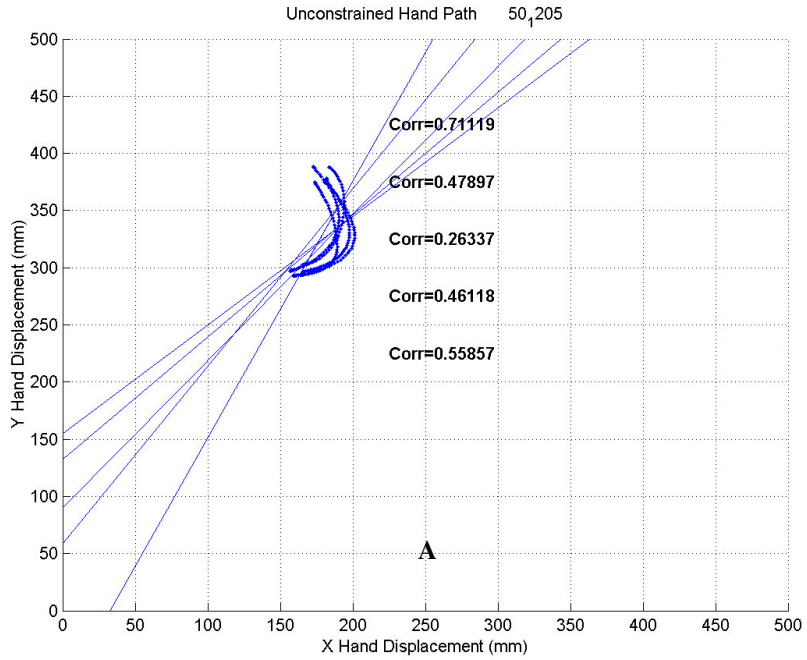


Figure 27: Curved paths are created when subject-5 (typical of all subjects) made pointing movements from the 150˚ target to the 120˚ target. Movement onset began in the forearm followed by the upper arm producing a curved hand path in the workspace and in the joint space. The excursion of the shoulder joint was greater than that of the elbow even though onset started in the elbow first contrary to the findings of Kaminski and Gentile (1986).



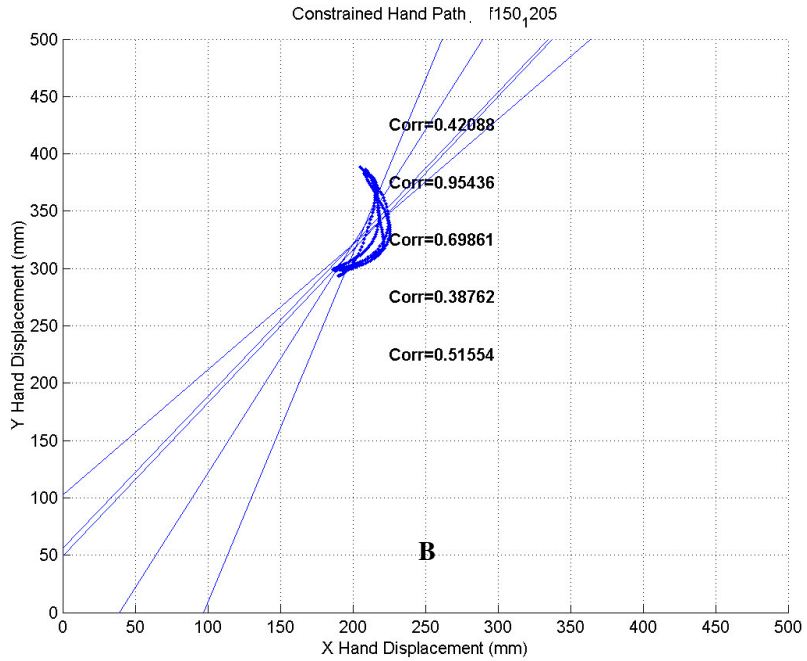


Figure 28: Target acquisitions made by subject-5 from the 150˚ target moving to the 120˚ degree target. Paths are not considered straight due to their low correlation too least square fit line.

6.2 Quantifying Quality of Movement

It had been speculated in Section *5.1.1* that the radius of curvature might prove to be a measure for quantifying the quality of movement. In both configurations high to very little curvature occurred as was illustrated in Figures 14. After examining the data, it is clear that using the radius of curvature for such purposes would not be a good approach. A difficulty in using radius of curvature is the 'looping' described above can occur in either the constrained or unconstrained trunk configurations whether reaching outward from the body or across the workspace. When a reversal in joint rotation (looping) occurs, it can produce infinite results in the radius of curvature obscuring the radius of curvature of interest; this effect was shown in the previous Figures 12 and 13. Curvature shown in these figures is typical for all subjects seen at the start (at the end as well) or along the trajectory when looping occurs as in Figures 15, 29, and 30.

In the figures above, there is visible curvature in some of the paths outside of the 'looping' as in Figure 28 where the trajectory is curved and segmented. Instances of high curvature in the trajectory appeared in the early stages of acceleration or in the late stage of deceleration as the hand approaches the target. Timing of high curvature coincides with changes in the hand velocity, inspection of the velocity profiles reveals multiple bell shaped profiles or multiple peaks within a profile, features present in Figures 19-22, 25, and 27. This argues for the notion that these high curvatures are due to aiming adjustments being made in the beginning and near the end of the movement. Subsequently, these instances of high radius of curvature can obscure the curvature of the overall trajectory. Curvature of the hand trajectory has possibilities as a method for

quantifying movement quality but the linear relationship of the angular velocities presents a less complicated approach.

6.2.1 Ratio of Angular Velocities

Even though the radius of curvature does not present itself as an easy to use quantifier of the movement quality, another variable does; specifically, the angular velocity. Lacquaniti et al., (1986) have shown that a linear relationship exists between the angular velocities of the arm joints. In other words, a tight coupling exists between the angular

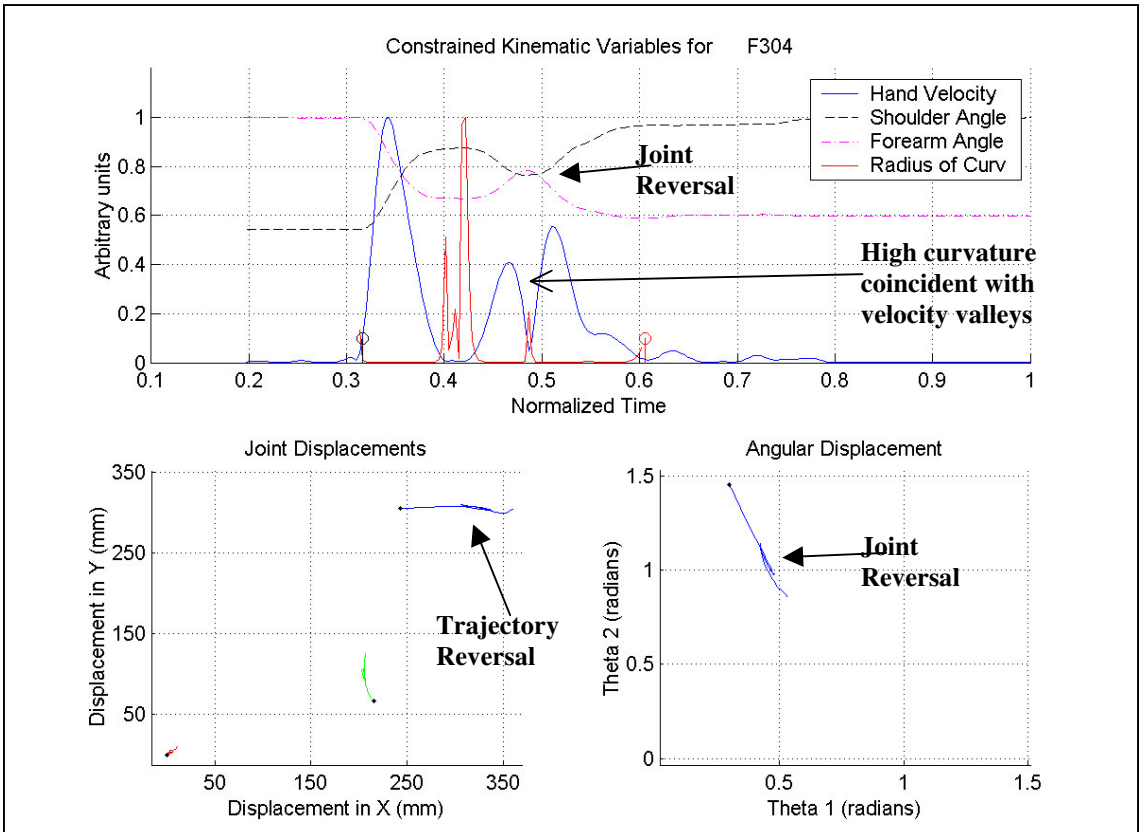


Figure 29: Kinematic results of subject-7 reaching from the 150˚ target across the workspace to the 30˚ target. In this figure 'looping' occurred during the acceleration phase of the movement producing instances of high radius of curvature. A joint reversal was coincident the change in trajectory and therefore the result of the bio-controller and not postural changes. The bio-controller relies on muscle properties and the reflex arch both of which are interrupted by DMD. The kinematics results above suggest that a consequence of the changes to muscle architecture is the timing of the agonist and antagonist muscle activity.

velocity of the elbow and shoulder joint, which is taken to facilitate the mapping between the internal and external coordinate systems.

Abend and colleagues (1982) first pointed out that points of high radius of curvature occurred in the valleys of the velocity profiles which suggests that curvature was responsible for slowing of movements. These researchers further reveal that joint reversals are required for producing some straight as well as curved trajectories. The joint reversals themselves did not impart a valley in the velocity profile in their study.

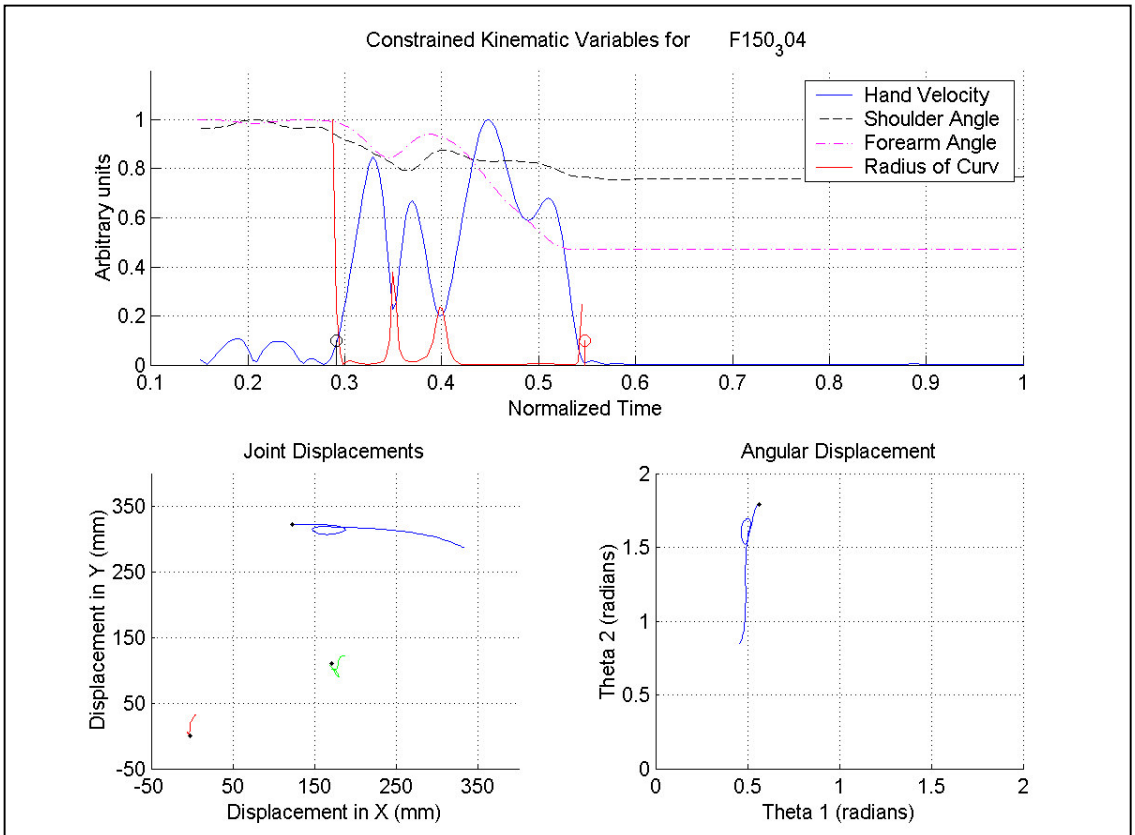


Figure 30: High radius of curvature produced during trajectory formation for subject-4 in the fixed trunk condition while reaching for the 30-degree target. Instances of greatest curvature occur in the valleys of the three distinct velocity profiles scaled to their maximum. The first profile is developed during the greatest portion of the hand trajectory towards the target. A halt in the trajectory occurs followed by two subsequent direction reversals thereby producing two additional bell shaped velocity profiles. Coincident with the second velocity valley is a joint reversal in both the shoulder and elbow.

Therefore, they suggested that the valleys are a result of the multijoint bio-controller as opposed to the events involving the individual joints. Previously these features were shown in the Figure 15 to be part or the result of pointing movements made by individuals with DMD. However, in Figure 29 it can be seen that a joint reversal can develop a coincident valley, a feature not seen by Abend and colleagues. A possible explanation is that the disease is interfering with the bio-controller, whereas the joint reversal is a loss of effective control of the movement.

These joint reversals are coupled such that the hand either loops or shifts rearward and forward along the intended path (Figure 29 and 30). This feature was even produced by the subject-7 considered strongest according to the manual muscle tests ratings. The difference between the cases in Figure 29 is the joint reversal occurred in the deceleration phase while reaching outward from the body into the workspace. In Figure 30 the 'looping' occurs during acceleration while reaching from the 150˚ degree target to the 30˚ target. It is suggested that this momentary loss of control is due to the effects of the disease on the musculature and subsequently the bio-controller. In section 2.1 the changes in muscle architecture and function in DMD were discussed here, it is suggested that there are consequences for bio-controller (Sect 6.2.2).

It was hypothesized that the linear relationship of the shoulder and elbow joint angular velocities would be degraded in some subjects related to strength and function. If the hand trajectory remains essentially straight and the linear relationship of the joint angular velocities degrades, it suggests that the path of the hand is of primary importance. In Figures 16 and 32, a least square line is fit to the ratio of angular velocities and the correlation to 'straightness' is presented. A coupling exists between the joint angular

changes such that the ratio of angular velocity is linear in some subjects, which is in agreement with previous reports (Lacquaniti et al., 1986; Gielen et al., 1999). However, as predicted this feature deteriorated with an increased functional requirement or impairment due to progression of the disease (see Table 4).

It is suggested here that a controlled movement correlates to the linearity of the ratio of shoulder to elbow angular velocity. That is, an effectively controlled movement maintains a high correlation to linearity in this ratio and therefore a linear coupling of the joint angular velocity. Given this notion of effective control, it follows that decay of linearity (coupling) suggests decay in the effectiveness of the bio-controller, actuators (muscles), or both. The consequence of decoupling the elbow and shoulder joint are the 'looping' and joint reversals that are coincident with valleys in the hand velocity profile

Table 4. Descriptive statistics for the correlation coefficient for all target acquisitions for specific movement configuration and start position. The correlation to linearity decreases with an increase in physical effort such as reaching out into and across the workspace. Additionally, there is a decrease in the linearity with increasing deviations in the ratio of angular velocity as DMD progresses.

Overall Correlation Coefficient for linearity of Ratio of Joint Angular Velocity					
Subject	Configuration	Origin Start		150˚ Start	
		Mean	Stdev.	Mean	Stdev.
1	Unconstrained	0.65	0.30		
	Constrained	0.67	0.22		
2	Unconstrained	0.63	0.26	0.52	0.15
	Constrained	0.64	0.46	0.44	0.19
3	Unconstrained	0.65	0.24		
	Constrained	0.57	0.21		
4	Unconstrained	0.81	0.25	0.64	0.19
	Constrained	0.82	0.19	0.39	0.12
5	Unconstrained	0.85	0.16	0.39	0.19
	Constrained	0.74	0.15	0.60	0.12
6	Unconstrained	0.79	0.15	0.26	0.17
	Constrained	0.83	0.13	0.41	0.18
7	Unconstrained	0.85	0.11	0.50	0.28
	Constrained	0.89	0.13	0.50	0.29

in Figures 29 and 30 discussed previously.

Ideally, the values of the ratio would be tightly grouped as in the case marked out in Figure 32 but as effective control fails the coupling of the elbow and shoulder joint is degraded. In Table 4 the mean correlation to linearity for all pointing movements in a given test configuration is presented. Subjects are presented in ascending strength based on decay in linearity of the ratio of shoulder to elbow angular velocity and the extent of deviation. This is a strong liner relationship between the decay in the ratio of angular velocity and strength illustrated in Figure 31. The result is a grading of strength

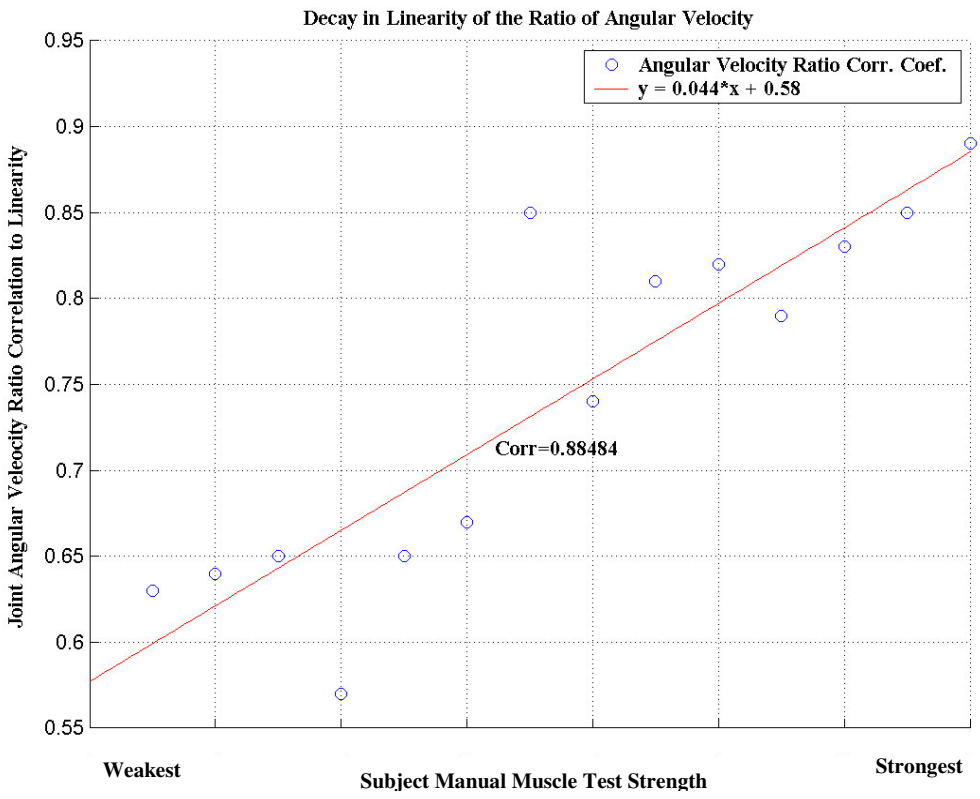


Figure 31: Results for reaching made outward into the workspace in the constrained and unconstrained trunk configuration for all subjects. Plotted are the correlation coefficients for the ratio of angular velocity to a least squares fit line. The results are ordered from weakest to strongest according to the manual muscle test results in Table 2 and its shown that decay in the linearity of the joint angular velocity ratio is strongly related to strength reduction. Correlation coefficients for each subject a presented in the constrained followed by the unconstrained order; note that linearity increases slightly in the unconstrained configuration in all but two cases.

predicated on the linearity of the ratio of joint angular velocity created while reaching outward from the body similar to manual muscle testing presented in Table 2. Because this method is less subjective than the opinion of the rater performing manual muscle test it is submitted as a means for quantifying functional ability.

Correlation to linearity decreased in the angular velocity ratio for all the subjects making the reaching movements from the 150˚ position to the other targets. This adds support to the notion that the linearity of the angular velocity ratio corresponds to the functional ability to perform a movement task. When linear coupling of the joint angular velocity breaks down a compensating mechanism must be employed to maintain the characteristic of a straight hand path. In the case presented in Figure 32, postural adjustments were made to project the hand towards the target compensating for the drift in the aim.

Trunk rotation/translation was substituted for joint angular changes due to the failure of the bio-controller to coordinate the efforts of the musculoskeletal system. Specifically, the mechanical properties of the DMD muscle have changed due to alterations in the muscle structure. Function of the muscle relies on mechanical behavior of its structure i.e. muscle spindles and Golgi tendon organ for control perturbation, dampening and inhibition. In order to maintain an essentially straight trajectory in the presence of the pathophysiology of DMD the CNS employed the additional joint with three addition degrees of freedom. This strongly suggests that a hierarchy exists in the bio-controller where maintenance of straight trajectories takes precedence over other characteristics such as the ratio of joint angular velocities.

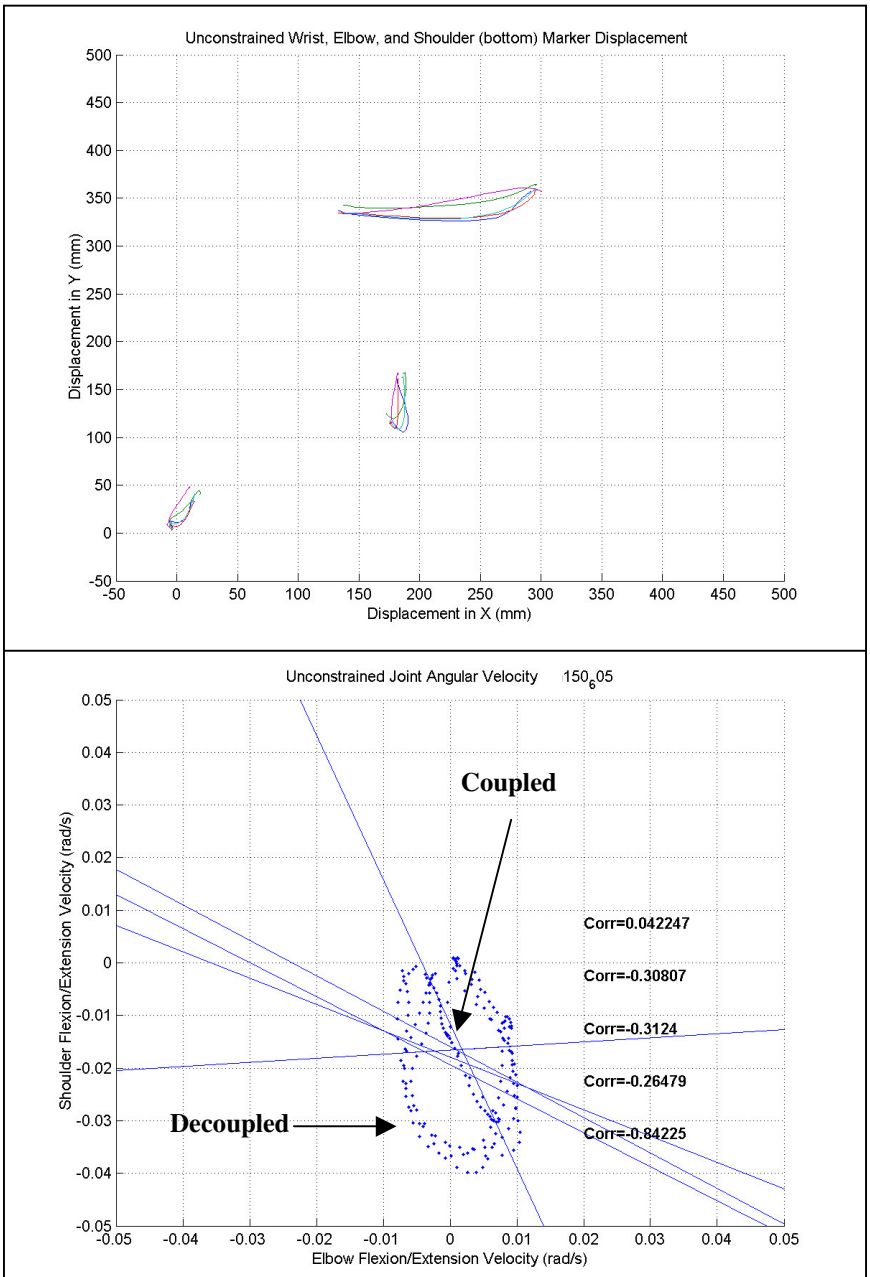


Figure 32: Top plot contains the shoulder, elbow, and wrist joint displacements for subject-7 making point movements across the workspace from the 150˚ to the 60˚ target. The bottom plot contains the corresponding of the shoulder vs. the elbow joint angular velocity. Essentially straight hand paths were created in all the trials yet in only one of the five trials did the angular velocity remains linearly coupled. The trunk is compensating for the failure of the joint angular velocity ratio to remain linear by projecting the arm forward. The importance of the straight trajectory is reflected in the effort to maintain the shortest path.

6.3 Proprioceptors and Muscle Control

Many have put forward the notion that muscle behaves as a spring whose stiffness is a function of its activation and length, i.e. the equilibrium hypothesis (Feldman, 1966; Cook, 1979, Bizzi et al., 1981a, b, 1982a, b, 1984). Polit and Bizzi (1979) have shown that the initial position of the arm is dependent on proprioceptive information. If the CNS is to maintain a desired position, the simultaneous activation of agonist and antagonist muscle groups must develop the appropriate forces. Lestienne et al., (1981) suggest that the CNS accomplishes this through a scalar coding of the agonist and antagonist forces. It is a well-established fact that the force developed by a muscle is a function of its length and as such a function of the stretch experienced by the muscle.

The bio-controller is dependent on feedback and feed forward information to the muscle via the motoneuron, Golgi tendon organ, and muscle spindles. In the control loop, motoneurons instruct the muscle to activate via efferent action potentials. Action potentials are sent to the muscle fibers by way of ventral nerve roots, which terminate at motor end plates. The Golgi tendon organ, a type Ib afferent senses the stretch experienced by the muscle (i.e. tendon stress) and inhibits its motoneuron at some threshold level. The muscle spindles (stretch receptor) termed type Ia afferent are imbedded at each end and within the extrafusal muscle fibers experience the same length change as the overall muscle. Both afferents produce action potentials that are sent back to the spinal cord via its dorsal roots to interneuron to inhibit the motoneuron contralaterally. The spindle itself is imbedded with an intrafusal efferent that signals an adjustment in the length of the spindle, which is coactivated with the motoneuron (Deutsch and Deutsch, 1993).

It is likely that several factors are playing roles in the apparent loss of control and actions of the musculature during voluntary movements made by DMD subjects. First in DMD, muscle function and strength deteriorate as the disease cycles the muscle through breakdown and repair. Since muscular structure is directly connected to its function, it is suggested here that the behavior of the Golgi tendon organ and the muscle spindles is altered. This view is supported by the fact that connective tissue embeds the muscle fibers in addition to fibrils that have been replaced with adipose tissue (Partridge et al., 1993).

Connective tissues cause a stiffening of the structure dampening the stretch of some fibers and influencing others subsequently altering the behavior of the muscle spindles and Golgi tendon organ (Cornu et al., 1998). The outward effects are seen in the absent or diminished deep tendon reflex seen in DMD subjects. The adipose tissue deposited during the course of the disease affects the muscle fiber conduction velocity delaying and inhibiting the propagation of action potentials (Buchthal et al., 1971). Other known muscle responses include increased twitch time for the DMD fibers (McComas and Thomas, 1968; Horowits et al., 1989), altered behavior of the motor units (Piotrkiewicz et al., 1999; Martinez and López-Terradas, 1992), and marked decrease in type II (fast) muscle fibers (Wang et al., 1999). The consequence is an interruption or delay in the reflex arc, which in turn affects the *fine-tuning* of the agonist and antagonist muscular effort absent in the EMG recordings (see section 6.5).

The ‘looping’ or reversing of trajectory occurs near the start of the acceleration phase or near the end of deceleration phase. During the acceleration phase, the musculature must overcome the inertia of the two-link system and coordinate the actions in both links

such that the arm is projected in the desired direction. Interference in the reflex arc causes the untimely activation of movement antagonists producing a loop in the trajectory. Once the arm started along planned path there were no further adjustments made until deceleration had begun. During deceleration, perturbations in the velocity profile reflect adjustments made by CNS to adjust the final approach trajectory. Again, since the reflex arc is impaired looping occurs because there is a disruption of the stimulation, inhibition, and propagation of proprioceptive signals. This is a plausible explanation due to the impaired stretch receptors responsible for the diminished or absence of the arm's deep tendon reflex (see table 2) for the described behavior.

6.4 Hand Velocities

Unimodal, symmetrical velocity profiles are typical for movements requiring low accuracy however; bimodal hand velocity profiles are also possible in the development of curved trajectories (Morasso, 1981, Abend et al., 1982). From the hand velocity profiles and paths presented in the figures above, it is clear that the disease is having a detrimental effect upon the smoothness of the velocity profile. The explanation forwarded for these results is a disruption of the bio-controller that is occurring due to the morphological changes occurring in the DMD muscle.

The ratio of peak hand velocity to the average (V_{max}/V_{mean}) has been used to describe movements with reported values ranging from 1.5 to 6 (Hogan, 1985; Soechting, 1984; Cooke et al., 1989). Ostry et al., (1987) have shown that the ratio of V_{max}/V_{mean} is not a constant but decreases as movement duration increases. Also, it has been observed that hand velocity profiles created during reaching movements are scaled by the ratio of V_{max}/V_{mean} such that as movement amplitude increased so does peak velocity and by

necessity the mean velocity (Cooke et al., 1989). The results for the ratio V_{max}/V_{mean} produced by DMD subjects tested ranged from a mean of 1.71 to 2.57 and are presented in Table 5. The movement amplitude of some subjects varied, as did the self-selected speed of movement. Subsequently, the ratio of V_{max}/V_{mean} is subjected to time and amplitude scaling accounting for differences. Despite that, the mean value of V_{max}/V_{mean} is 2.1 over the spread of V_{max}/V_{mean} (illustrated in Figure 33) where values of 1.88 to 1.90 have been reported (Hogan, 1984; Ostry et al., 1987).

A higher ratio of V_{max}/V_{mean} has been identified in DMD subjects but intuition points towards a slower rate due to the decrease in fiber contraction velocity. The extent of variance in the ratio of V_{max}/V_{mean} suggests as Cook et al., (1989) contend that the inability to produce a constant ratio reflects the inability to scale a basic or underlying

Table 5. V_{max}/V_{mean} for pointing movements made towards targets at attitudes of 30˚, 60˚, 90˚, 120˚, and 150˚ degrees set at equal distance from a common origin as in Figures 8 and 9. The start position had the origin set at the wrist with upper arm abducted and the forearm flexed 90˚ degrees respectively. Pointing movements were made outward from the body towards the specified targets for a total of five acquisitions apiece.

Subject	Configuration	V_{max}/V_{mean}	
		Mean	Stdev.
1	Unconstrained	2.11	0.46
	Constrained	1.98	0.31
2	Unconstrained	1.88	0.10
	Constrained	2.03	0.26
3	Unconstrained	1.71	0.17
	Constrained	2.03	0.35
4	Unconstrained	1.84	0.24
	Constrained	2.31	0.26
5	Unconstrained	1.91	0.12
	Constrained	1.87	0.16
6	Unconstrained	2.08	0.47
	Constrained	2.19	0.24
7	Unconstrained	1.89	0.07
	Constrained	2.31	0.29

movement, and thus this is a reflection of control of movement trajectory. Moreover, symmetric hand velocity profiles are produced during reaching movements (Hollerbach and Flash, 1982, Atkeson and Hollerbach, 1985) and under varied loads (Ruitenbeek, 1985; Lestienne, 1979). However, asymmetrical profiles have been shown to exist where the acceleration period is shorter than the deceleration when high accuracy is required (Soechting, 1984; Taylor and Birmingham, 1948). In all efforts made by the DMD subjects the acceleration phase was shorter than the deceleration with the except for subject-four and seven in the unconstrained, see Table 6. The velocity profiles generated

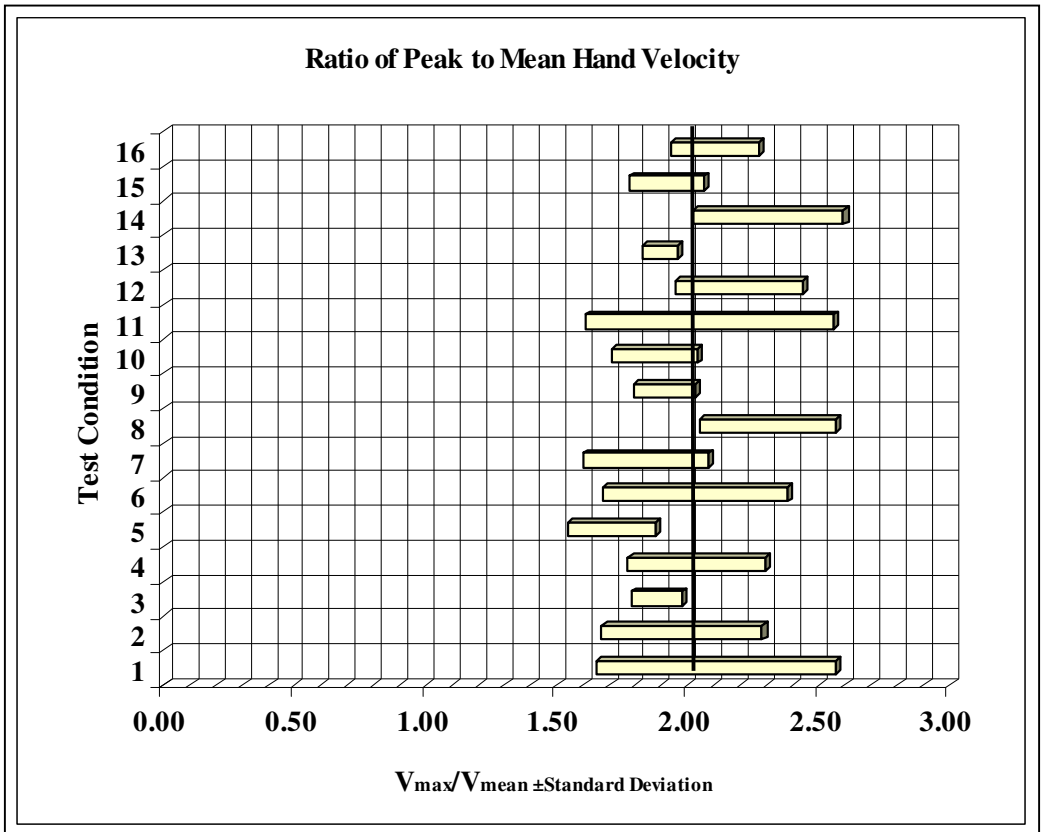


Figure 33: Ratio of V_{max}/V_{mean} for each subject performing in the constrained followed by the unconstrained trunk configuration corresponding to the data in Table 5. Values shown are the mean over the five trials ± the standard deviation. Subjects-one (1, 2), three (5, 6), and six (13, 14) made movements of the same amplitude.

by DMD subjects while executing pointing movements was similar in character to those found in the elderly (Cooke et. al., 1989).

Cooke and Colleagues found normal appearing velocity profiles during the acceleration phase of the movement in elderly subjects. Additionally, there were small oscillations in the velocity profile during an extended deceleration phase. They provide evidence that the source for the alteration in the velocity profiles was likely a cerebellar dysfunction. However, they did not know if the dysfunction was due to a failure in the proprioceptive, central motor program or visual feedback mechanisms. In the DMD subjects tested, the acceleration phase of the velocity profile appears relatively normal but the deceleration phase was often extended and had small oscillations as the subject approached the target. The velocity profile results of the DMD subjects were similar to the velocity profiles by the elderly as described by Cooke et al., (1989) but in DMD, the pathophysiology of the disease in known. Therefore, it was suggested herein that the

Table 6. Mean acceleration and deceleration time in seconds for each subject reaching for all targets in a defined test condition.

		Time to peak Velocity				Time from Peak to Stop			
		Origin Start		150˚ Start		Origin Start		150˚ Start	
Subject	Configuration	Mean	Stdev.	Mean	Stdev.	Mean	Stdev.	Mean	Stdev.
1	Unconstrained	0.51	0.14			0.74	0.28		
	Constrained	0.47	0.09			0.89	0.50		
2	Unconstrained	0.60	0.09	0.57	0.06	0.75	0.10	0.60	0.15
	Constrained	0.49	0.01	0.08	0.06	0.66	0.17	0.60	0.07
3	Unconstrained	0.67	0.07			1.07	0.24		
	Constrained	0.61	0.08			1.29	0.67		
4	Unconstrained	0.44	0.07	0.43	0.52	0.40	0.17	2.22	1.04
	Constrained	0.37	0.11	0.09	0.09	0.83	0.32	2.22	0.22
5	Unconstrained	0.58	0.04	0.74	0.07	0.85	0.31	0.68	0.26
	Constrained	0.54	0.09	0.30	0.23	0.73	0.13	0.68	0.14
6	Unconstrained	0.40	0.07	0.53	0.09	0.55	0.16	0.62	0.15
	Constrained	0.50	0.13	0.22	0.16	0.66	0.12	0.62	0.04
7	Unconstrained	0.36	0.03	0.37	0.04	0.33	0.07	0.39	0.11
	Constrained	0.35	0.10	0.10	0.07	0.53	0.16	0.39	0.14

abnormalities seen in the velocity profile are a result of a failing peripheral proprioceptive feedback or reflex arch in the aiming of the movement.

6.5 EMG

Hallett (1986) described three distinctive EMG patterns that can be recognized tonic, ballistic, and reflex. Tonic EMG activity is characterized as continuous activity lasting the duration of the movement. Reflex EMG patterns are seen with stretch reflexes lasting 10-30 msec (Hallett 1994). Ballistic triphasic EMG patterns with delaminated muscle activation of agonist-antagonist-agonist introduced in Section 2.3 have durations of 50-100 msec. The triphasic pattern was expected in this study because it is synonymous with the kinematics of rapid multijoint arm movements, even in some with motor impairments (Barardelli et al., 1986; Barardelli et al., 1996). However, the results differed from the expectation in that often the DMD subjects generated EMG patterns that exhibited tonic or ballistic behavior as well as remaining quiet (Figure 33). Nonetheless, when a subject produced the ballistic EMG pattern, the synergistic effort of the muscles was clear.

The EMG of trial two for movements made from the 150˚ target across the workspace to the 30˚ target is presented in Figure 34 and the kinematic results are illustrated in Figure 34. Pictured in these two figures are the results of movements made by a stronger subject and many of the kinematic and EMG characteristics discussed are seen. Shown in Figure 34 are the ballistic triphasic EMG patterns of agonist-antagonist-agonist burst activity. The corresponding kinematic features of an essentially straight hand path and unimodal velocity profile are shown in Figure 34. Tonic EMG patterns are also present in the bicep and deltoids in this trial and the brachioradialis remained relatively quiet.

However, neither the EMG nor the velocity characteristics remained consistent between test configurations or even trials.

To illustrate this point recall the kinematics results presented in Figure 30, these results are of trial 4 for the same test condition as in Figures 34 and 35. However, in Figure 30, the hand velocity profile is certainly not smooth and bell shaped. Moreover, a disruption of the velocity profile and path is reflected in the EMG patterns produced. That is, not only is the kinematic characteristics affected but patterns of muscle activity revealed through EMG are also altered as shown in Figure 36. In Figure 36 there is no appearance of the triphasic muscle activation pattern, muscles are activated and stay

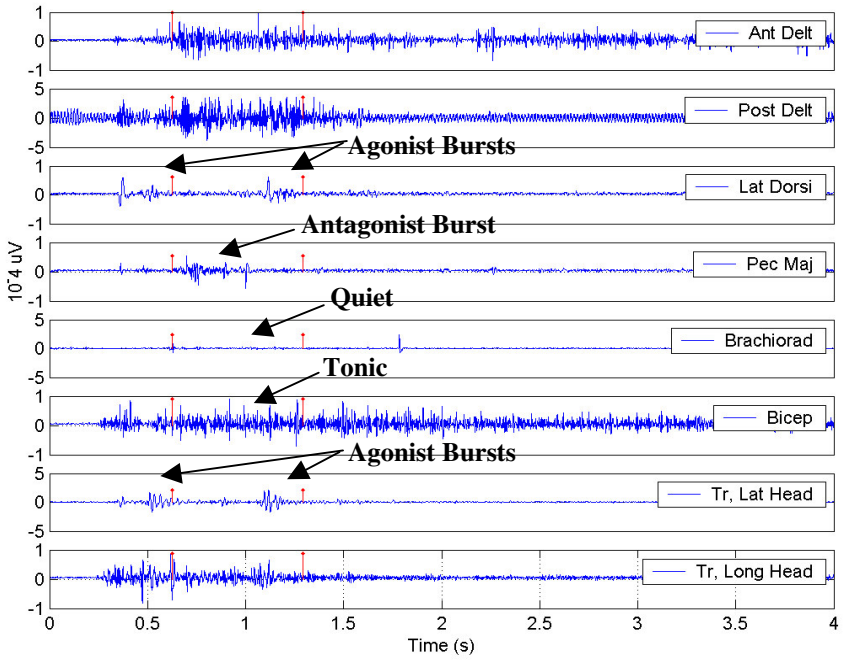


Figure 34: Figure displays the EMG recordings from the muscles of subject-7 while reaching from the target set at the 150˚attitude to the target at the 30˚ attitude. This was the second trial with the trunk in the fixed configuration. Standing stems mark the onset and termination of the movement. There is both tonic and ballistic behavior observed in the EMG recordings in addition to no recorded activity in the brachioradialis. Tonic activity was present in the deltoids and bicep yet triphasic EMG activity is present in the latissimus dorsi, pectoralis major, and triceps.

active throughout the movement with the exception of the brachioradialis and lateral head of the triceps.

It has been shown that subject-7 is a stronger test subject and still there were no consistent patterns in the timing or magnitude of muscle activation. At times muscle activation patterns present a clear agonist-antagonist relationship or synergy, such as those in Figure 33. Such results are also similar to those reported for healthy, disabled (Berardelli, et al., 1996) and elderly subjects Cooke, et al., (1989) even so, this was not typical. During many efforts muscles activate essentially in unison shortly before movement commences and remain active throughout the movement as shown in Figure 36. Demonstrated are but two of the variety of activation patterns produced between trials

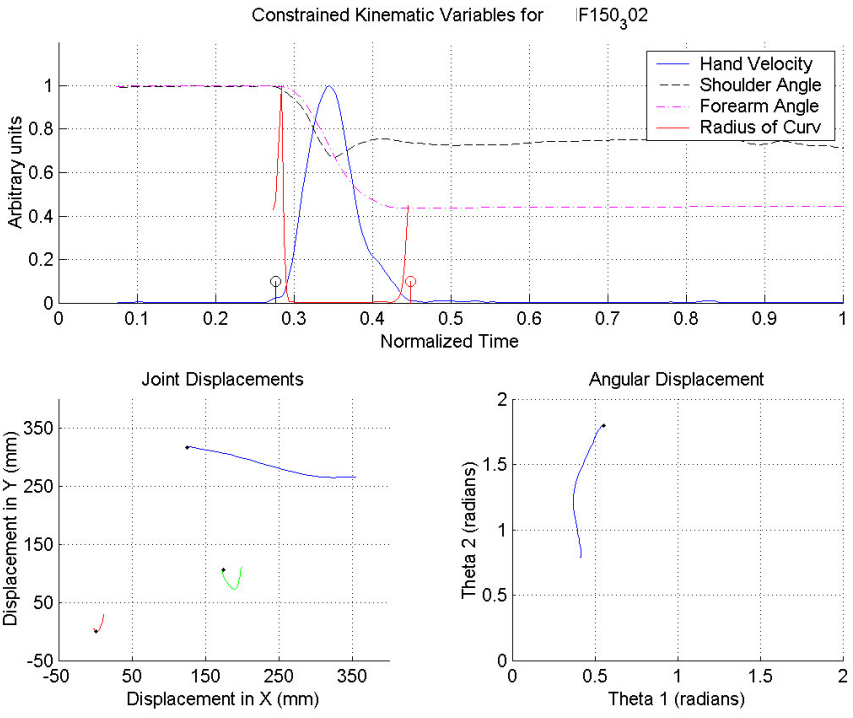


Figure 35: Muscle activity for the fourth reaching movement made by subject-7 from the 150˚ target to the 30˚ target while in the fix trunk configuration. The movement onset and termination are marked with standing stems. The muscle activity in this figure corresponds to the kinematic results shown previously in Figure 30.

within the same test condition and between the two test configurations.

There was no discernable difference in the muscle activation patterns in the constrained and unconstrained condition. Although there were negligible differences in muscle activation patterns in the unconstrained and constrained test configurations, the quality in modality of the velocity profile does improve when the trunk adds additional degrees of freedom. This suggests that when the trunk is free to move it substitutes for the lack of muscular control and/or coordination by making adjustments that help sustain not only a straight hand path but a smooth hand velocity profile as well. Although there is some improvement in the 'straightness' of the hand path when the trunk in unconstrained,

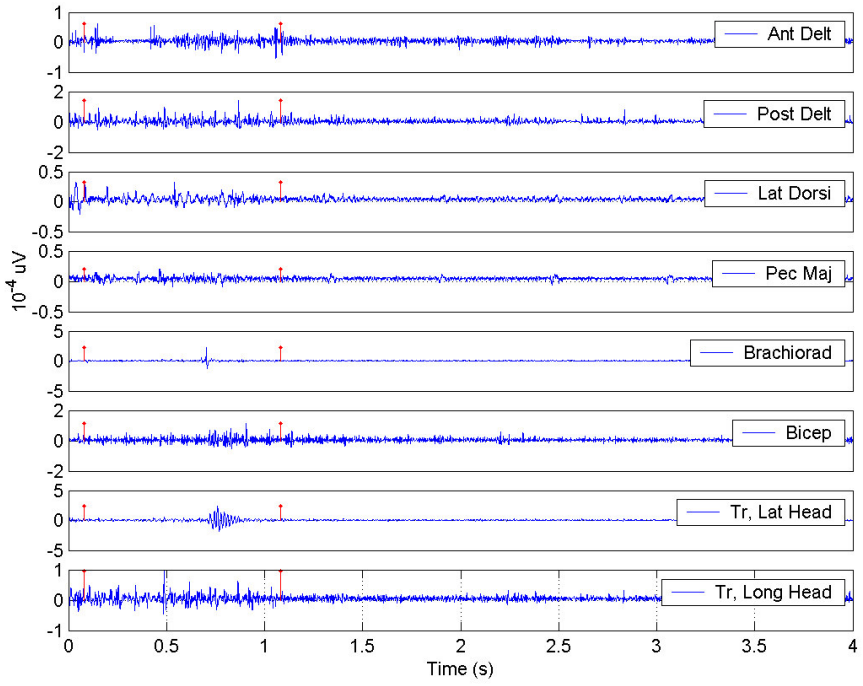


Figure 36: Hand velocity profiles for the five trials of subject-7 pointing from the 150˚ target to the 30˚ target in the unconstrained (left) and constrained (right) trunk configuration. An improvement in the quality of the hand velocity profile is clearly visible when the trunk is free to add its translational/rotational degrees of freedom to the movement.

it may be due to the marked improvement in the smoothness of the velocity profiles as seen in Figure 37. This was a performance characteristic consistent with all the subjects, meaning they all had improvements in the smoothness of the hand velocity profile. Regardless of start and terminal points or configuration, the only characteristic that has remained consistent throughout is an essentially straight hand path.

It had been proposed that the empirical method of Karst and Hasan (1987, 1991a, b) and Hasan and Karst (1989) would be an adequate method for identifying the direction the bio-controller intended the hand to travel. In this method, five muscles were monitor pectoralis major, posterior deltoid, biceps, brachioradialis, and tricep. Muscles are considered activated at a threshold value of 10 times the standard deviation of the EMG baseline for more than 7.5 ms and their effort is quantified by the integral of the enveloped EMG over 100 ms of activity. A sign ± is assigned to each muscle for either flexor or extensor activity at a joint, this was done from numerous start and terminal points. Based on the onset and magnitude (by sign ±) of initial flexor or extensor muscle

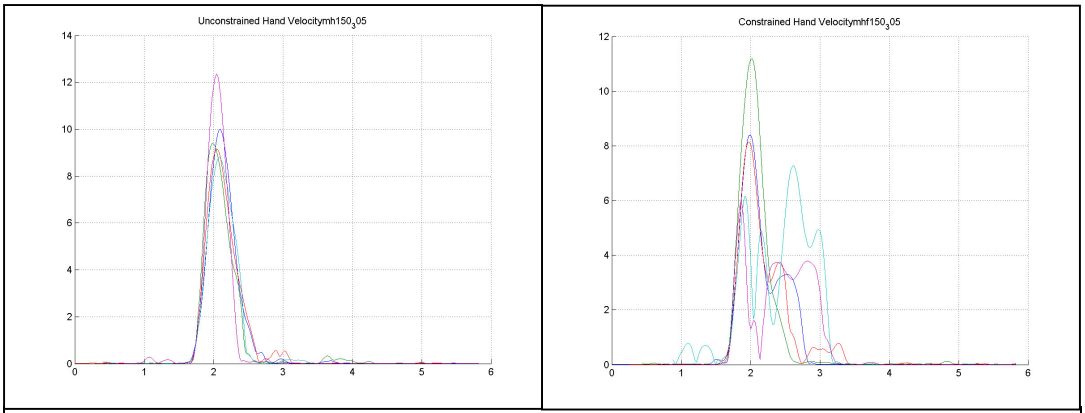
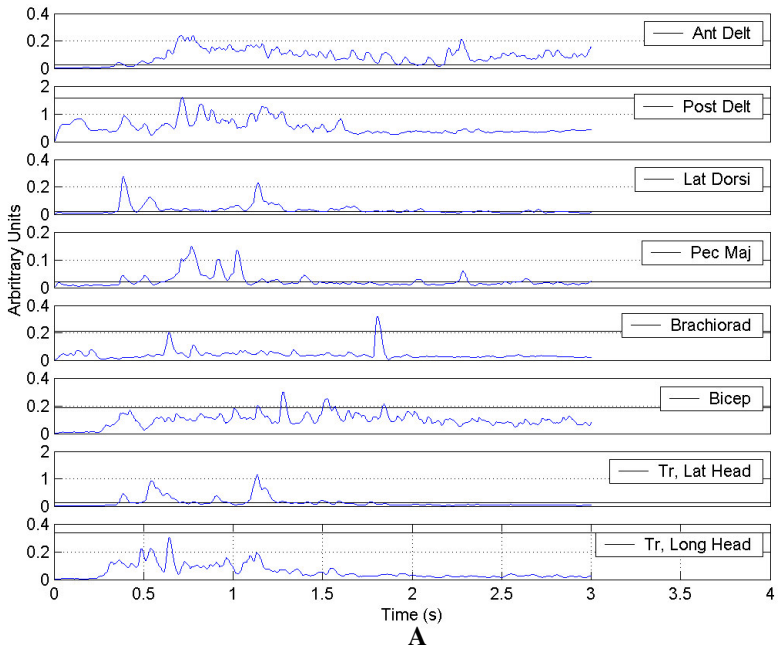


Figure 37: Kinematic results for second trial of subject-7 making a pointing movement from the 150˚ target to the 30˚ with the trunk in the constrained configuration. The subject produced an essentially straight hand path and unimodal velocity profile; data corresponds to EMG shown in Figure 33.

activity and the orientation of the elbow (θ_2) the terminal direction of the hand was identified.

Returning to the EMG data in Figures 34 and 36 and rectifying the EMG signal followed by filtering with a second order low-pass Butterworth filter with a cutoff frequency of 12 Hz, results are illustrated in Figure 38A, B. With the appropriate threshold to indicate muscular activity the onset and magnitude of initial muscle activity in Figure 38A and B can be defined. However, patterns in muscle initial activity did not appear between trials as shown in Figure 38, which forms the basis of partitioning the workspace based on initial extensor and flexor activity. To apply the Karst and Hasan method successfully ballistic type patterns are a requisite. This may be in fact what the CNS is attempting given the results in Figure 38A however; the CNS must cope with the pathophysiology of DMD disrupting the agonist-antagonist-agonist relationship. It demonstrates that although patterns of initial muscle activity are preserved under some conditions in DMD movement control demands can alter muscular synergy.



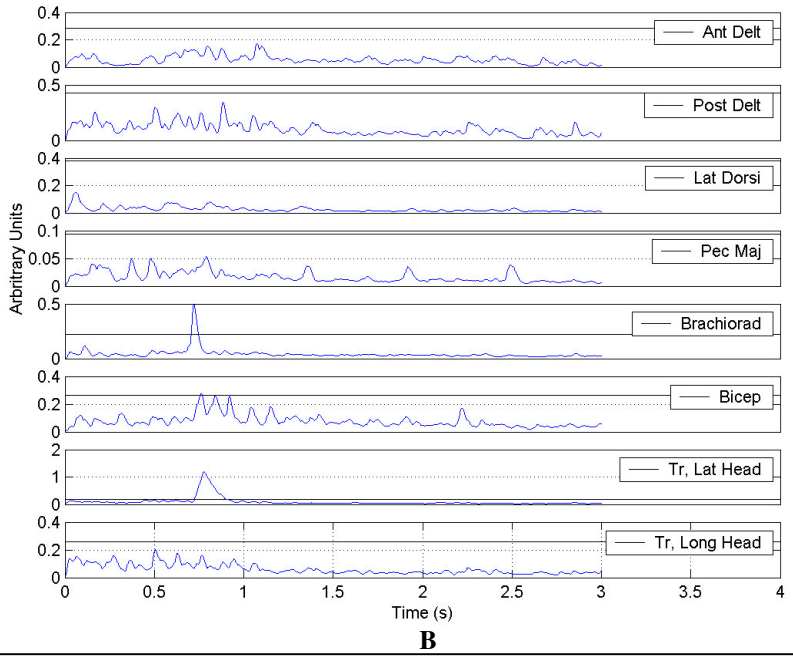


Figure 38: Enveloped EMG patterns for the second (A) and fourth (B) trials of movements made from the 150˚ target to the target at 30˚ by subject-7. A pattern of initial flexor and extensor activity from the EMG results was sought using methods similar to Karst and Hasan (1987, 1991a, b). Although an identifiable flexor/extensor activity produces patterns in the EMG with agonist-antagonist relationships appears in A, there was no consistent relationship in onset of muscular activity between for a group of trials, see B.

CHAPTER 7: CONCLUSIONS

It has been proposed that individuals with some neuropathies could benefit from a robotic exoskeleton. The device would augment residual strength by supporting and actively propelling the arm in space. For such a device, a control system is required. Two critical parameters of control are the direction of the final hand position from a given arm posture and the path to the target. Once a target direction and path are defined, a set of joint rotations for the shoulder and elbow and their rate of change are needed. Regardless of how they are chosen, they must be properly controlled to produce *coordinated* movement of the hand along a desired trajectory. The orthosis control should also accommodate the 'natural' adaptations and movement strategies employed by the user. This level of complexity is required if the orthosis is to help *actively* produce movements of a 'natural' form.

Towards this end several questions and hypotheses were developed and examined in this study. Specifically; 1. Can terminal hand direction be determined using a methodology first proposed by Karst and Hasan; 2. Do additional degrees of freedom introduced with trunk (rotation/tilt) in DMD improve arm movement kinematics in the transverse plane; 3. Does the ratio of joint angular velocity maintain a linear relationship in the presence of a motor impairment; 4. Is there a control hierarchy in the development and maintenance of arm kinematics in DMD?

7.1 Karst and Hasan EMG Methodology

Since EMG provides a window into the motor activity of the musculature, it was proposed that it could also play a role in the control of the orthosis. Therefore, it was asked if EMG activity of arm musculature were at adequate levels to be used as a

possible input and if so how might it be used? EMG records were found at levels that reflected some muscular synergy (Bowen et al., 2001) consequently, it was hypothesized that the empirical EMG method proposed by Karst and Hasan (1987, 1991a, b) and Hasan and Karst (1989) for determining terminal hand position from an initial posture could be applied in DMD. The method requires a partitioning of the workspace based on the initial posture of the elbow (θ_2 of model presented in Figure 3) and initial muscle activity. Subsequently, this study examined the kinematic and muscular activity of pointing movements made by individuals with DMD. Subjects were asked to make pointing movements in the transverse plane with their arm supported in a floatation device from two initial positions to several targets with the trunk constrained and unconstrained.

This methodology was attractive for two reasons first; it would provide a possible means for using EMG as control input for the orthosis by identifying the terminal direction utilizing initial forearm posture θ_1 and flexor/extensor muscle activity. Although there are other methods for using EMG to estimate muscular contributions that could have been used to determine terminal direction most have complicated signal processing and calculation needs. As a result it is likely that longer response times for the control system would ensue. This brings to light the second attractive reason for this approach, the minimal processing and calculation requirements once partitioning of the workspace had been established. Of course, even if successful it would be limited to the partitioning of extensor or flexor activated movements established in the plane from the initial postures tested.

The Karst and Hasan methodology is predicated on the triphasic motor activity of a burst of agonist muscle activity followed shortly by a burst of antagonist muscle activity and ending with another burst of agonist muscle activity burst. This type of muscle activation pattern is synonymous with ballistic movements that produce essentially straight trajectories and unimodal velocity profiles (Abend et al., 1982; Hogan et al., 1987; Berardelli et al., 1996). This ballistic pattern of muscular activity was identifiable in most of the subjects in at least one trial however; it was not consistently repeated within any set of target acquisitions of any subject tested.

Typically, the subjects tested produced EMG activation patterns where onsets of muscular activity were in quasi-unison or varied from ballistic to tonic type activity. While some subjects did not produce a single triphasic EMG pattern others did but not repeatedly. Moreover, variability in EMG activity was present in individual trials while starting from the same initial posture moving to the same final position. The absence of repeated muscle activation patterns prevented the use of the Karst and Hasan method as a means for determining the terminal hand position from a given initial posture. Even though the method failed under the conditions tested, this should not be taken as evidence towards the validity of the method forwarded by Karst and Hasan.

Several faults could have contributed to the lack of success of this methodology in DMD such as the subject not being training for the task. If training time were made available to the subject it is possible that the results could have been more consistent. Additionally, the quality of electromyographic electrodes, leads, and system could certainly have a detrimental effect. This conclusion is based on the 60 Hz noise embedded in the EMG signals such as in Figure 18. However, none of the results

preclude the use of EMG as an auxiliary control input for the proposed exoskeletal robotic orthosis.

7.2 Effect of Additional Degrees of Freedom

A hypothesis of the study was trunk movement contributed to an improvement in arm movement kinematics i.e. hand trajectories, velocity profile, ratio of angular velocity etc. To investigate this hypothesis the subjects were asked to perform a number of pointing movements with the trunk constrained and unconstrained from two initial postures to select targets. Common kinematic features thought to be synonymous with the formation and control of movement such as the straightness of hand trajectory, modality of the hand velocity profile and linearity of joint angular velocity are examined.

The hand paths created by the DMD subjects tested were essentially straight with a high correlation coefficient to a least squares fit line in both the constrained and unconstrained trunk configuration. This was the case for both the constrained and unconstrained trunk configurations for most subjects. Some curving in the hand paths was present but usually near the end of movement. This suggests a strategy of ballistically projecting the arm towards the target then making aiming adjustments in the final approach. Although the straightness of hand path improved in the unconstrained over the constrained the curvature of the hand trajectory remained small in the most DMD subjects. The nature of the hand paths generated by the DMD subjects in this study supports the contention that hand paths are planned in the hand space and '*straightness*' of trajectory is of primary importance.

In the kinematic construction of the arm model shown in Figure 3 the hand coordinates (x, y) and joint orientations (θ_1, θ_2) are dependent variables. Given that the

hand paths remain essentially straight in the constrained and unconstrained test configuration some other kinematic variable must be altered to accommodate the straight hand path. What was found was a significant change in the hand velocity profile modality when performing the movement tasks in the constrained vs. the unconstrained test configuration. In this study while the subjects were in the constrained trunk test configuration, they all produced velocity profiles that were less 'smooth' than profiles produced in the unconstrained truck configuration as exampled in Figures 19, 20, 21, 22, 25, and 27.

These results show an alteration in the hand velocity profile modality between the unconstrained vs. constrained trunk configuration. Specifically, there is less acceleration/deceleration occurring along the hand path during the movement in the unconstrained than in the constrained configuration. This improvement occurred while making reaching movements from both start positions i.e. whether reaching out into or across the workspace. This demonstrates that the additional degree(s) of joint freedom are incorporated into the arm movements such that unimodality of the hand velocity profile is restored.

Hogan (1984) proposed that symmetry of the hand velocity profile is due to the CNS minimizing the rate of change of acceleration, in other words the amount of jerk. The theory is based on the goal to produce a smooth movement in the most energy efficient manner. This return to the more recognized classic unimodal velocity profiles supports the opinion if the theory of minimum jerk holds that movements are organized in such a manner as to reduce energy cost.

7.3 Ratio of Joint Angular Velocities

A coupling of the angular velocity has been shown to exist in healthy subjects such that the ratio is linear (Lacquaniti et al., 1986; Gielen et al., 1999). Subsequently it was hypothesized that the linearity in the joint angular velocity ratio of the shoulder ($\dot{\boldsymbol{\theta}}_1$) versus the elbow ($\dot{\boldsymbol{\theta}}_2$) would not be maintained. The motivation for this hypothesis is the impact of DMD pathophysiology has on the musculature and its likely affect on the muscle performance. That is, progression of DMD is marked by a fast twitch muscle fiber reduction (Buchthal et al., 1971), an increase in muscle stiffness (Cornu et al., 1998), distal/proximal muscular strength differentials (Partridge et al., 1993), a decline in motor unit remodeling (Piotrkiewicz et al., 1999), a reduction in action potential propagation velocity and an increase fiber contraction times (Horowits et al. 1989) to name a few.

The results show that the linearity of the ratio of joint angular velocity remained essentially unchanged between the constrained and unconstrained trunk configurations for each subject. Although this was not always the case in fact, there were some instances when the correlation to linearity improved in the unconstrained trunk configuration vs. the constrained. However, the trend was no notable improvement or further decay in the linearity of the ratio of angular velocity with changes in constrained or unconstrained trunk configurations.

A decay in the linearity of the ratio of joint angular velocity with progression of the disease exists and is illustrated in Figure 31. In this study, the stronger the subject the higher the mean correlation to linearity of the ratio angular velocity for all the trials was. This was the result whether reaching into or across the workspace. This result illustrates how the disease affects muscular control and the importance of some variables with

respect to others. Improvement in a hand space parameter of hand tangential velocity and the lack of improvement in the ratio of angular velocity linearity suggests that the central motor program is not adapting the additional degrees of freedom the trunk provides to accommodate arm joint space.

7.4 Hierarchical Control

The notion that arm movements are planned in extracorporeal hand space as straight hand paths forwarded by Morasso (1981) suggests that hand paths are of primary importance in the organizational hierarchy of arm movement. The spatial motor control thesis implies that other motor control parameters such as joint angles, proprioceptive, exteroceptive, and afferences are subservient to desired hand trajectory in the control hierarchy of arm movement planning and execution. Therefore, it was hypothesized that a control hierarchy would manifest itself in the kinematic parameters.

First, it can be noted that the results show hand trajectories follows essentially straight paths in the constrained and unconstrained trunk configuration although, with some exceptions. Generally this is the case whether reaching into or across the workspace examined within this study. Second, there is a reduction in the hand velocity profile modality and an increase in overall quality when the trunk is free to move as opposed to the fixed. Again, this was the result whether reaching into or across the workspace. Third, the linearity of the angular velocity ratio remained essentially unaffected by the introduction of additional degrees of freedom allowed in the unconstrained trunk configuration.

A slight improvement in the ‘straightness’ of the hand path occurred in a few instances moving from the constrained to the unconstrained configurations However, it

was as likely to have no improvement or even a slight deterioration in the so called 'straightness' of hand path. This lack of improvement in the hand path 'straightness' suggests that this characteristic is produced at the best possible level of control in both configurations. However this is done at a cost to the other kinematic parameters monitored. This assertion is forwarded because an improvement in the smoothness of the hand velocity profile was not synonymous with an improvement in the overall 'straightness' of trajectory. Additionally, the linearity of the joint angular velocity ratio decays with disease progression and the characteristic of a straight hand path remained relatively consistent regardless of trunk configuration and/or disease progression.

The velocity profile results also support the contention that in the hierarchy of control, maintenance of a unimodal velocity profile is not of a higher order than that of producing essentially straight hand paths. When degrees of freedom are added to the system i.e. unconstrained trunk configuration, their employment by the CNS causes an improvement in the *smoothness* of the velocity profile. This demonstrates the flexibility of the central motor program to adapt degrees of freedom such as trunk translation/rotation to the preservation of straight hand trajectories with unimodal velocity profiles. This suggests that the angular velocity ratio is subservient in the control hierarchy to the desire to maintain the straight trajectory and the desire to maintain symmetrical unimodal hand velocity profile.

7.5 Summary

Hand path were essentially straight in both the unconstrained and constrained trunk configuration and were affected the least by breakdown in movement control. There was some curvature in the hand paths usually near the end of movement as opposed to an

arched path suggesting impairment in aiming control in the final approach due to slowed muscle and/or reflex responses. All subjects reflected an improvement in the smoothness of the hand velocity profile when pointing movements were made in the unconstrained as opposed to the constrained trunk configuration. Given additional degrees of freedom, the central motor program adapted it to improve the smoothness of the hand velocity profile and not the hand path or joint angular velocity ratio. The hypothesis that the hand path is of primary importance in the control hierarchy is supported by the fact that decay is seen in both the joint angular velocity ratio and in the hand velocity profile and not the hand paths. Moreover, it reflects how the CNS employs as compensation additional degrees of freedom outside the limb making a pointing movement. It also adds credence to the theory that minimizing jerk (Hogan 1984) where the central bio-controller is managing acceleration changes.

Additionally, it was proposed that the ratio was sufficient as a means for quantifying the quality of movement control. A closer look at the geometry of the joint angular velocity profile is warranted. This is because several of the subjects had produced loops in the profile indication mutual rotation at varied angular velocity in the elbow and shoulder. It may be that a more robust quantitative analysis other than examination of linearity could provide quantitative measure of finer resolution such as the surface area contained within the hysteresis of the joint angular velocity profile.

In this study there were seven subjects seen, this was due to the difficulty in recruiting subjects capable of performing the task from a limited available population. To further this work a greater number of subjects from beginning to end stages of the disease should be seen to ensure that the results apply to populations at large. As it stands the results are

interesting but difficult to draw conclusions for whole populations based on the limited data set.

Training of subjects should also take place before the task is performed to diminish alterations in muscle activation patterns seen as a new movement is learned. Although this requirement would significantly extend the testing time to avoid fatigue, it may produce more effective EMG results for use in the Karst and Hasan methodology. It would also be beneficial if more sophisticated EMG equipment were employed in any future study which could also make the application of this methodology more effective.

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APPENDIX A: TABLES CONTAINING DESCRIPTIVE STATISTICS

Descriptive statistics for all Subjects, an F indicates that the trunk was in the fixed configuration. The 150_30 etc. indicates that the start position was at the 150-degree target and just 30 etc. indicates a start position form the origin. Empty rows are due to failure to complete the task or corrupted data. The first set of tables contain the acceleration/deceleration times, the second the V_{max}/V_{maen} and the last peak velocity.

	Acceleration Time (sec)					Deceleration Times (sec)					
Subject/Task	Max	Min	Mean	Std	Var	Max	Min	Mean	Std	Var	Trials
DM30	0.44	0.26	0.32	0.10	0.01	0.74	0.40	0.58	0.17	0.03	3
DMF30	0.36	0.22	0.28	0.06	0.00	1.10	0.70	0.86	0.16	0.02	5
DM60	0.44	0.30	0.36	0.07	0.01	0.88	0.72	0.80	0.08	0.01	3
DMF60	0.94	0.22	0.60	0.33	0.11	0.86	0.38	0.58	0.19	0.04	5
DM90	0.48	0.36	0.42	0.08	0.01	0.58	0.40	0.49	0.13	0.02	2
DMF90	1.02	0.30	0.56	0.29	0.08	1.00	0.38	0.66	0.27	0.07	5
DM120	0.54	0.42	0.49	0.06	0.00	0.70	0.38	0.53	0.16	0.03	3
DMF120	0.94	0.36	0.52	0.24	0.06	0.68	0.42	0.54	0.10	0.01	5
DM150	0.46	0.36	0.41	0.05	0.00	0.46	0.30	0.36	0.09	0.01	3
DMF150	0.76	0.38	0.53	0.15	0.02	1.22	0.36	0.66	0.34	0.12	5
DM60_30	0.24	0.24	0.24	0.00	0.00	0.44	0.44	0.44	0.00	0.00	1
DMF60_30	0.62	0.28	0.39	0.13	0.02	0.44	0.22	0.28	0.09	0.01	5
DM90_30	0.42	0.42	0.42	0.00	0.00	0.70	0.70	0.70	0.00	0.00	1
DMF90_30	0.96	0.24	0.44	0.30	0.09	0.60	0.26	0.39	0.13	0.02	5
DM120_30	0.44	0.44	0.44	0.00	0.00	0.56	0.56	0.56	0.00	0.00	1
DMF120_30	1.04	0.38	0.75	0.28	0.08	0.78	0.30	0.58	0.19	0.04	5
DM150_30	0.44	0.32	0.40	0.05	0.00	0.94	0.56	0.78	0.14	0.02	5
DMF150_30	0.80	0.38	0.52	0.17	0.03	0.80	0.44	0.58	0.14	0.02	5

	Acceleration Time (sec)					Deceleration Times (sec)					
Subject/Task	Max	Min	Mean	Std	Var	Max	Min	Mean	Std	Var	Trials
LS90	1.20	0.28	0.73	0.38	0.15	1.66	0.84	1.23	0.33	0.11	5
LSF90	0.92	0.46	0.61	0.18	0.03	3.12	1.30	2.06	0.67	0.45	5
LS120	0.80	0.42	0.60	0.15	0.02	1.86	0.48	1.18	0.52	0.27	5
LSF120	0.68	0.42	0.53	0.11	0.01	1.54	0.48	0.86	0.45	0.20	5
LS150	1.16	0.34	0.68	0.34	0.11	1.36	0.46	0.80	0.35	0.13	5
LSF150	0.98	0.54	0.69	0.17	0.03	1.46	0.50	0.95	0.37	0.14	5

	Acceleration Time (sec)					Deceleration Times (sec)					
Subject/Task	Max	Min	Mean	Std	Var	Max	Min	Mean	Std	Var	Trials
JM30	1.36	0.40	0.64	0.40	0.16	1.42	0.60	1.05	0.32	0.10	5
JMF30	0.50	0.32	0.44	0.07	0.01	0.84	0.54	0.69	0.11	0.01	5
JM60	0.66	0.46	0.54	0.09	0.01	1.52	0.72	1.13	0.28	0.08	5
JMF60	0.92	0.36	0.55	0.22	0.05	1.28	0.58	0.78	0.28	0.08	5
JM90	0.66	0.48	0.54	0.07	0.01	1.42	0.80	1.02	0.24	0.06	5
JMF90	1.10	0.26	0.69	0.34	0.12	1.08	0.40	0.85	0.26	0.07	5
JM120	0.64	0.54	0.58	0.05	0.00	0.80	0.44	0.62	0.13	0.02	5
JMF120	0.58	0.42	0.50	0.06	0.00	0.96	0.58	0.79	0.14	0.02	5
JM150	0.82	0.46	0.61	0.13	0.02	0.52	0.34	0.41	0.07	0.00	5
JMF150	0.60	0.44	0.53	0.08	0.01	0.74	0.40	0.51	0.14	0.02	5
JM150_30	1.02	0.34	0.62	0.25	0.06	1.22	0.56	0.86	0.24	0.06	5
JMf150_30	1.84	0.34	0.82	0.59	0.35	2.32	1.22	1.50	0.46	0.22	5
JM150_60	0.68	0.52	0.60	0.07	0.00	1.04	0.84	0.92	0.10	0.01	4
JMF150_60	1.28	0.68	0.94	0.27	0.07	1.24	0.62	0.92	0.29	0.08	5
JM150_90	1.00	0.48	0.63	0.21	0.05	0.74	0.12	0.58	0.26	0.07	5
JMF150_90	0.58	0.36	0.42	0.09	0.01	1.48	0.76	0.99	0.29	0.08	5
JM150_120	0.86	0.58	0.76	0.13	0.02	0.44	0.24	0.36	0.07	0.01	5
JMF150_120	1.06	0.42	0.80	0.26	0.07	0.60	0.30	0.45	0.11	0.01	5

	Acceleration Time (sec)					Deceleration Times (sec)					
Subject/Task	Max	Min	Mean	Std	Var	Max	Min	Mean	Std	Var	
RM60	0.58	0.30	0.39	0.12	0.01	1.20	0.72	0.97	0.19	0.04	5
RMF60	0.62	0.26	0.37	0.14	0.02	1.78	0.70	1.42	0.45	0.21	5
RM90	0.70	0.32	0.49	0.18	0.03	0.68	0.32	0.43	0.14	0.02	5
RMF90	0.78	0.30	0.49	0.19	0.04	1.22	0.52	0.82	0.26	0.07	5
RM120	0.94	0.50	0.66	0.18	0.03	1.08	0.62	0.81	0.17	0.03	5
RMF120	0.70	0.44	0.55	0.10	0.01	0.60	0.24	0.43	0.14	0.02	5

	Acceleration Time (sec)					Deceleration Times (sec)					
Subject/Task	Max	Min	Mean	Std	Var	Max	Min	Mean	Std	Var	Trials
JH30	0.66	0.34	0.51	0.15	0.02	0.86	0.68	0.79	0.07	0.00	5
JHF30	0.80	0.30	0.49	0.19	0.04	1.06	0.62	0.78	0.20	0.04	5
JH60	0.88	0.58	0.74	0.11	0.01	0.96	0.54	0.73	0.16	0.02	5
JHF60											
JH90	0.78	0.58	0.65	0.08	0.01	0.74	0.44	0.59	0.12	0.01	5
JHF90	0.58	0.40	0.50	0.08	0.01	0.66	0.38	0.54	0.12	0.01	5
JH120	0.66	0.44	0.55	0.11	0.01	1.12	0.54	0.88	0.23	0.05	5
JHF120											
JH150	0.68	0.38	0.55	0.13	0.02	0.88	0.58	0.76	0.12	0.01	5
JHF150											5
JH150_30	0.78	0.62	0.68	0.06	0.00	1.04	0.66	0.81	0.17	0.03	5
JHf150_30	0.52	0.46	0.50	0.03	0.00	1.24	0.74	0.99	0.23	0.05	5
JH150_60	0.76	0.62	0.67	0.05	0.00	0.72	0.46	0.58	0.12	0.01	5
JHF150_60											
JH150_90	0.80	0.54	0.66	0.10	0.01	0.80	0.32	0.55	0.21	0.04	5
JHF150_90	0.76	0.46	0.62	0.12	0.01	1.26	0.70	0.92	0.23	0.05	5
JH150_120	0.66	0.38	0.55	0.10	0.01	0.64	0.30	0.47	0.13	0.02	5
JHF150_120	0.74	0.46	0.58	0.10	0.01	0.62	0.32	0.46	0.11	0.01	

	Acceleration Time (sec)					Deceleration Times (sec)					
Subject/Task	Max	Min	Mean	Std	Var	Max	Min	Mean	Std	Var	Trials
TH30	0.58	0.36	0.45	0.09	0.01	0.72	0.22	0.56	0.20	0.04	5
THF30	0.54	0.20	0.27	0.15	0.02	1.52	1.02	1.18	0.22	0.05	5
TH60	0.40	0.26	0.33	0.05	0.00	0.54	0.26	0.41	0.12	0.02	5
THF60	0.32	0.26	0.29	0.03	0.00	0.78	0.56	0.70	0.10	0.01	5
TH90	0.52	0.44	0.49	0.04	0.00	0.66	0.50	0.56	0.07	0.00	5
TMF90	0.48	0.34	0.41	0.07	0.00	1.64	0.26	0.98	0.50	0.25	5
TH120	0.58	0.38	0.46	0.07	0.01	0.34	0.18	0.27	0.06	0.00	5
THF120	0.88	0.36	0.51	0.21	0.04	0.66	0.36	0.46	0.13	0.02	5
TH150	0.56	0.42	0.49	0.06	0.00	0.30	0.14	0.20	0.06	0.00	5
THF150											
TH150_30	0.96	3.38	1.79	1.03	1.06	4.40	2.16	2.93	0.96	0.91	5
THF150_30	0.76	0.34	0.55	0.17	0.03	1.86	0.48	0.96	0.57	0.32	5
TH150_60	0.84	0.38	0.58	0.19	0.04	1.14	0.44	0.73	0.29	0.08	5
THF150_60	0.56	0.32	0.42	0.09	0.01	1.06	0.48	0.67	0.23	0.05	5
TH150_90	0.92	1.46	1.26	0.21	0.05	2.38	2.18	2.28	0.08	0.01	5
THF150_90	0.40	0.34	0.37	0.02	0.00	0.84	0.58	0.72	0.10	0.01	5
TH150_120	0.54	2.06	1.54	0.60	0.35	3.38	2.74	2.95	0.26	0.07	5
THF150_120	0.46	0.24	0.36	0.08	0.01	0.64	0.42	0.50	0.09	0.01	5

	Acceleration Time (sec)					Deceleration Times (sec)					
Subject/Task	Max	Min	Mean	Std	Var	Max	Min	Mean	Std	Var	Trials
MH30	0.42	0.26	0.33	0.08	0.01	0.66	0.24	0.38	0.16	0.03	5
MHF30	1.00	0.24	0.51	0.34	0.11	1.42	0.52	0.76	0.37	0.14	5
MH60	0.76	0.24	0.38	0.22	0.05	0.36	0.20	0.28	0.07	0.01	5
MHF60	0.24	0.22	0.23	0.01	0.00	0.72	0.46	0.62	0.10	0.01	5
MH90	0.34	0.30	0.32	0.02	0.00	0.28	0.20	0.24	0.03	0.00	5
MHF90	0.38	0.26	0.32	0.05	0.00	0.68	0.28	0.51	0.18	0.03	5
MH120	0.54	0.32	0.39	0.09	0.01	0.38	0.20	0.32	0.07	0.01	5
MHF120	0.36	0.28	0.32	0.04	0.00	0.46	0.30	0.38	0.06	0.00	5
MH150	0.50	0.30	0.38	0.09	0.01	0.56	0.30	0.40	0.12	0.01	5
MHF150	0.44	0.28	0.35	0.06	0.00	0.56	0.26	0.40	0.12	0.01	5
MH150_30	0.44	0.28	0.36	0.06	0.00	0.66	0.36	0.54	0.12	0.01	5
MHF150_30	0.96	0.20	0.46	0.29	0.08	1.36	0.56	0.81	0.33	0.11	5
MH150_60	0.66	0.32	0.41	0.14	0.02	0.54	0.30	0.40	0.10	0.01	5
MHF150_60	0.44	0.30	0.39	0.06	0.00	0.60	0.36	0.51	0.09	0.01	5
MH150_90	0.40	0.28	0.32	0.05	0.00	0.50	0.28	0.36	0.10	0.01	5
MHF150_90	0.36	0.32	0.34	0.02	0.00	0.36	0.22	0.30	0.06	0.00	5
MH150_120	0.50	0.22	0.33	0.11	0.01	0.32	0.18	0.26	0.05	0.00	5
MHF150_120	0.36	0.24	0.29	0.05	0.00	0.26	0.22	0.24	0.02	0.00	5

	Peak/Mean Velocity					
Subject/Task	Max	Min	Mean	Std	Var	Trials
DM30	2.30	1.58	1.92	0.36	0.13	3
DMF30	3.00	1.85	2.54	0.47	0.23	5
DM60	2.35	2.03	2.21	0.16	0.03	3
DMF60	2.33	1.62	1.87	0.29	0.08	5
DM90	3.92	1.77	2.85	1.52	2.31	2
DMF90	2.75	1.91	2.22	0.34	0.12	5
DM120	1.79	1.70	1.76	0.05	0.00	3
DMF120	2.26	1.81	2.11	0.18	0.03	5
DM150	1.87	1.55	1.69	0.16	0.03	3
DMF150	2.94	1.67	2.24	0.52	0.27	5
DM60_30	2.36					1
DMF60_30	2.22	1.55	1.80	0.26	0.07	5
DM90_30	2.35					1
DMF90_30	1.86	1.66	1.76	0.09	0.01	5
DM120_30	1.98					1
DMF120_30	3.16	1.96	2.29	0.50	0.25	5
DM150_30	2.99	2.08	2.57	0.38	0.14	5
DMF150_30	2.57	2.36	2.44	0.08	0.01	5

	Peak/Mean Velocity					
Subject	Max	Min	Mean	Std	Var	Trials
LS90	1.97	1.28	1.57	0.26	0.07	5
LSF90	3.21	2.04	2.43	0.47	0.22	5
LS120	1.88	1.54	1.67	0.17	0.03	5
LSF120	2.24	1.58	1.78	0.27	0.07	5
LS150	2.28	1.61	1.89	0.25	0.06	5
LSF150	2.10	1.64	1.87	0.18	0.03	5

	Peak/Mean Velocity					
Subject	Max	Min	Mean	Std	Var	Trials
JM30	1.97	1.54	1.82	0.17	0.03	5
JMF30	2.06	1.66	1.88	0.16	0.02	5
JM60	2.14	1.67	1.93	0.22	0.05	5
JMF60	2.24	1.89	2.03	0.14	0.02	5
JM90	2.23	1.89	2.01	0.13	0.02	5
JMF90	1.91	1.44	1.65	0.19	0.04	5
JM120	1.88	1.58	1.77	0.14	0.02	5
JMF120	1.98	1.55	1.78	0.16	0.03	5
JM150	2.18	1.72	2.03	0.19	0.04	5
JMF150	2.33	1.72	2.02	0.25	0.06	5
JM150_30	2.29	1.75	2.02	0.22	0.05	5
JMf150_30	3.06	1.73	2.24	0.50	0.25	5
JM150_60	2.14	0.00	1.53	0.87	0.75	4
JMF150_60	2.15	1.50	1.93	0.26	0.07	5
JM150_90	2.33	1.82	2.05	0.21	0.05	5
JMF150_90	2.64	1.79	2.08	0.33	0.11	5
JM150_120	1.64	1.37	1.54	0.10	0.01	5
JMF150_120	1.81	1.36	1.56	0.22	0.05	5

	Peak/Mean Velocity					
Subject/Task	Max	Min	Mean	Std	Var	Trials
JH30	2.16	1.59	1.86	0.23	0.05	5
JHF30	2.61	1.94	2.33	0.25	0.06	5
JH60	1.92	1.64	1.78	0.10	0.01	5
JHF60						
JH90	2.00	1.65	1.83	0.12	0.02	5
JHF90	2.15	1.73	1.92	0.18	0.03	5
JH120	2.26	1.68	2.02	0.27	0.07	5
JHF120						
JH150	2.13	1.69	1.94	0.16	0.03	5
JHF150	2.00	1.73	1.85	0.11	0.01	5
JH150_30	2.73	1.94	2.21	0.30	0.09	5
JHf150_30	2.71	1.76	2.21	0.39	0.15	5
JH150_60	2.34	1.82	2.07	0.25	0.06	5
JHF150_60						
JH150_90	2.69	1.89	2.16	0.33	0.11	5
JHF150_90	3.31	2.20	2.74	0.52	0.27	5
JH150_120	2.19	1.84	1.96	0.14	0.02	5
JHF150_120						

	Peak/Mean Velocity					
Subject	Max	Min	Mean	Std	Var	Trials
RM60	3.80	2.42	3.16	0.60	0.36	5
RMF60	3.98	2.33	3.00	0.67	0.45	5
RM90	2.26	1.59	1.89	0.29	0.08	5
RMF90	2.98	1.81	2.33	0.51	0.26	5
RM120	3.56	2.33	2.86	0.45	0.20	5
RMF120	2.14	1.79	1.95	0.14	0.02	5

	Peak Velocities (mm/s) Statistics Across Trials					
Subject	Max	Min	Mean	Std	Variance	Trials
TH30	6.64	5.55	6.16	0.48	0.24	5
THF30	7.61	3.76	4.96	1.55	2.42	5
TH60	6.26	3.48	4.83	1.13	1.28	5
THF60	5.96	4.07	4.86	0.78	0.61	5
TH90	4.50	3.04	3.93	0.54	0.29	5
RMF90	4.54	2.11	3.46	0.93	0.87	5
TH120	6.04	4.91	5.56	0.46	0.21	5
THF120	5.53	4.74	5.13	0.37	0.13	5
TH150	8.99	6.84	7.43	0.89	0.79	5
THF150						
TH150_30	11.50	6.80	8.70	1.74	3.02	5
THF150_30	9.38	3.69	7.12	2.10	4.41	5
TH150_60	7.62	3.40	5.94	1.58	2.49	5
THF150_60	8.50	5.06	7.20	1.35	1.81	5
TH150_90	5.99	3.19	4.63	1.00	0.99	5
THF150_90	6.58	3.50	4.80	1.20	1.45	5
TH150_120	3.70	1.95	2.81	0.75	0.57	5
THF150_120	3.80	2.43	3.11	0.50	0.25	5

	Peak/Mean Velocity					
Subject	Max	Min	Mean	Std	Var	Trials
MH30	2.19	1.68	1.94	0.23	0.05	5
MHF30	2.88	2.19	2.37	0.30	0.09	5
MH60	2.66	1.72	1.98	0.38	0.14	5
MHF60	3.06	2.37	2.72	0.33	0.11	5
MH90	1.95	1.86	1.90	0.04	0.00	5
MHF90	2.79	1.86	2.37	0.40	0.16	5
MH120	2.04	1.68	1.82	0.15	0.02	5
MHF120	2.23	1.78	2.07	0.19	0.04	5
MH150	1.87	1.81	1.83	0.02	0.00	5
MHF150	2.39	1.76	2.01	0.23	0.05	5
MH150_30	2.45	1.90	2.07	0.22	0.05	5
MHF150_30	2.40	1.81	2.16	0.22	0.05	5
MH150_60	2.27	1.86	2.03	0.15	0.02	5
MHF150_60	2.26	1.80	1.97	0.19	0.04	5
MH150_90	2.21	1.79	2.00	0.18	0.03	5
MHF150_90	1.97	1.64	1.79	0.16	0.02	5
MH150_120	2.20	1.54	1.81	0.31	0.09	5
MHF150_120	2.53	1.63	2.08	0.33	0.11	5

Peak Velocities Statistics Across Trials						
Subject/Task	Max	Min	Meam	Std	Var	Trials
DM30	9.51	4.83	7.35	2.36	5.56	3
DMF30	9.54	6.53	7.89	1.28	1.64	5
DM60	7.38	6.29	6.74	0.57	0.32	3
DMF60	8.76	3.44	5.87	2.29	5.24	5
DM90	7.31	5.90	6.61	1.00	0.99	2
DMF90	8.49	5.19	7.00	1.48	2.19	5
DM120	8.11	5.44	6.35	1.52	2.32	3
DMF120	9.17	5.36	7.85	1.59	2.54	5
DM150	7.80	6.84	7.47	0.54	0.29	3
DMF150	8.03	5.72	6.88	0.95	0.90	5
DM60_30	5.81					1
DMF60_30	6.68	4.68	5.77	0.93	0.86	5
DM90_30	8.55	8.55	8.55	0.00	0.00	1
DMF90_30	9.93	4.62	7.59	2.26	5.13	5
DM120_30	8.42					1
DMF120_30	11.52	7.30	8.62	1.90	3.61	5
DM150_30	15.86	8.73	13.25	2.86	8.20	5
DMF150_30	17.53	11.49	13.94	2.63	6.94	5

	Peak Velocities (mm/s) Statistics Across Trials					
Subject	Max	Min	Meam	Std	Var	Trials
LS90	3.01	1.84	2.27	0.46	0.21	5
LSF90	4.46	2.24	3.09	0.89	0.79	5
LS120	4.52	1.73	2.66	1.13	1.29	5
LSF120	4.24	3.02	3.55	0.52	0.27	5
LS150	4.74	2.82	3.92	0.95	0.90	5
LSF150	4.33	1.57	2.68	1.02	1.05	5

Peak Velocities Statistics Across Trials						
Subject/Task	Max	Min	Meam	Std	Var	Trials
JM30	4.69	2.66	3.60	0.85	0.72	5
JMF30	5.29	3.86	4.36	0.55	0.30	5
JM60	4.18	2.79	3.54	0.59	0.35	5
JMF60	4.28	2.54	3.34	0.74	0.55	5
JM90	4.47	3.59	4.06	0.33	0.11	5
JMF90	4.47	2.01	3.06	0.94	0.88	5
JM120	5.43	4.70	5.09	0.34	0.11	5
JMF120	4.85	3.92	4.43	0.37	0.14	5
JM150	5.47	3.54	4.39	0.80	0.64	5
JMF150	5.47	3.54	4.28	0.76	0.59	5
JM150_30	10.38	4.83	6.38	2.36	5.55	5
JMf150_30	5.17	2.54	4.30	1.07	1.14	5
JM150_60	4.88	0.00	3.59	2.04	4.16	4
JMF150_60	4.54	2.72	3.69	0.73	0.53	5
JM150_90	5.45	3.93	4.48	0.60	0.36	5
JMF150_90	5.36	3.00	4.09	1.00	0.99	5
JM150_120	3.42	2.62	2.90	0.32	0.10	5
JMF150_120	3.54	2.18	2.64	0.57	0.33	5

Peak Velocities (mm/s) Statistics Across Trials						
Subject	Max	Min	Meam	Std	Var	Trials
RM60	7.93	6.50	7.18	0.61	0.37	5
RMF60	6.55	4.25	5.55	0.92	0.84	5
RM90	6.26	3.29	4.71	1.24	1.55	5
RMF90	7.92	3.69	5.39	1.59	2.52	5
RM120	8.02	5.31	6.40	1.10	1.21	5
RMF120	9.97	7.49	8.45	1.00	1.00	5

Peak Velocities Statistics Across Trials						
Subject	Max	Min	Meam	Std	Var	# Trials
JH30	5.45	4.07	4.96	0.59	0.35	5
JHF30	6.72	5.63	6.38	0.44	0.19	5
JH60	4.98	3.40	4.17	0.63	0.40	5
JHF60						
JH90	5.62	4.37	5.05	0.47	0.22	5
JHF90	7.36	5.71	6.27	0.65	0.42	5
JH120	5.87	4.53	5.36	0.52	0.27	5
JHF120						
JH150	5.71	4.99	5.49	0.30	0.09	5
JHF150	8.05	4.82	6.30	1.29	1.67	5
JH150_30	10.86	8.06	9.82	1.30	1.69	5
JHf150_30	10.41	0.00	7.19	4.27	18.25	5
JH150_60	9.20	7.43	8.58	0.68	0.47	5
JHF150_60						
JH150_90	7.32	5.39	6.52	0.72	0.52	5
JHF150_90	7.59	5.27	6.88	0.94	0.88	5
JH150_120	5.56	3.07	3.92	0.97	0.95	5
JHF150_120						

	Peak Velocities (mm/s) Statistics Across Trials					
Subject	Max	Min	Meam	Std	Var	# of Trials
TH30	6.64	5.55	6.16	0.48	0.24	5
THF30	7.61	3.76	4.96	1.55	2.42	5
TH60	6.26	3.48	4.83	1.13	1.28	5
THF60	5.96	4.07	4.86	0.78	0.61	5
TH90	4.50	3.04	3.93	0.54	0.29	5
THF90	4.54	2.11	3.46	0.93	0.87	5
TH120	6.04	4.91	5.56	0.46	0.21	5
THF120	5.53	4.74	5.13	0.37	0.13	5
TH150	8.99	6.84	7.43	0.89	0.79	5
THF150						
TH150_30	11.50	6.80	8.70	1.74	3.02	5
THF150_30	9.38	3.69	7.12	2.10	4.41	5
TH150_60	7.62	3.40	5.94	1.58	2.49	5
THF150_60	8.50	5.06	7.20	1.35	1.81	5
TH150_90	5.99	3.19	4.63	1.00	0.99	5
THF150_90	6.58	3.50	4.80	1.20	1.45	5
TH150_120	3.70	1.95	2.81	0.75	0.57	5
THF150_120	3.80	2.43	3.11	0.50	0.25	5

	Peak Velocities (mm/s) Statistics Across Trials					
Subject	Max	Min	Meam	Std	Var	Trials
MH30	10.12	4.33	7.81	2.19	4.80	5
MHF30	7.04	5.13	5.85	0.77	0.59	5
MH60	10.03	6.74	8.30	1.44	2.06	5
MHF60	7.54	5.63	6.50	0.79	0.63	5
MH90	9.65	7.23	8.91	0.98	0.97	5
MHF90	6.40	5.04	5.60	0.57	0.33	5
MH120	9.31	6.77	7.55	1.01	1.02	5
MHF120	7.97	5.72	6.53	0.88	0.77	5
MH150	9.19	6.25	7.30	1.19	1.42	5
MHF150	7.28	5.97	6.62	0.49	0.24	5
MH150_30	12.35	8.80	9.94	1.42	2.01	5
MHF150_30	11.20	5.96	8.20	1.93	3.72	5
MH150_60	9.79	7.19	8.61	0.95	0.91	5
MHF150_60	8.97	6.52	7.58	1.08	1.17	5
MH150_90	8.71	5.23	7.20	1.25	1.56	5
MHF150_90	7.55	4.83	6.41	1.07	1.14	5
MH150_120	5.42	2.08	3.44	1.37	1.89	5
MHF150_120	4.69	2.64	4.07	0.83	0.69	5

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Principles of Systems Analysis Applied to Biomedicine, *BMES-511-512* Drexel University sequence of two graduate level courses designed to introduce systems analysis applied to biological systems for those with life science backgrounds.

Biomedical Mechanics, *BMES 641-643* Drexel University sequence of three graduate level courses to introduce advanced analytical tools applied to living systems and structures.

Publications

Bowen R., C., Seliktar, R., Rahman T, Alexander, M., *"Feasibility of Electromyographic Potentials of Persons with Muscular Dystrophy as Control Inputs for Power Orthosis.*" Proceedings of Progress in Motor Control III: From basic science to application." University of Montreal, Montreal Canada August 15-19, 2001.

Bowen R., C., Seliktar, R., Rahman T, Alexander, M., *"Surface EMG and Motor Control of the Upper Extremity in Muscular Dystrophy: A Pilot Study."* Proceedings of the 23rd Annual International Conference of the IEEE-EMBS October 25-28, 2001, Istanbul, Turkey.

Bowen R., C., Seliktar, R., Rahman T, Alexander, M., *"Arm Motion and EMG in Children with Duchenne's Type Muscular Dystrophy; A Pilot Study.*" Proceedings of the XIVth Congress of the International Society of Electrophysiology and Kinesiology; "Standardization for a better exchange of ideas", Vienna, Austria, June 22-25, 2002.