VISCOELASTIC PROPERTIES OF THE HUMAN WRIST DURING THE STABILIZATION PHASE OF A TARGETED MOVEMENT

by

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B.Sc. University of British Columbia 1990

THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF SCIENCE
in the School
of
Kinesiology

SIMON FRASER UNIVERSITY

December 1997

APPROVAL

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DEGREE: Master of Science

TITLE OF THESIS:

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ABSTRACT

Joint stability is a critical factor in the control of human movement. An understanding of the methods by which the neuromuscular system maintains mechanical stability in the face of a wide range of changing mechanical environments is important in the accurate modeling of motor control dynamics.

Under normal operating conditions, the neuromuscular system is able to rapidly modulate the mechanical impedance of a joint in order to maintain stability during postural or movement tasks. This is particularly evident following a targeted movement; the mechanics of the limb is controlled so that motion is stopped as quickly as possible with minimal oscillation about the final position. In fact, the human motor control system is very good at this type of task considering that rapid voluntary movements are most often stopped without any noticeable oscillation.

The mechanical impedance of a joint consists of inertial, viscous, and elastic components. Its mechanical properties are determined primarily by the activation of the muscles that act about the joint. Stability is therefore effected by intrinsic muscle properties and proprioceptive feedback. It has been suggested that reflex feedback from muscle proprioceptors is responsible for damping oscillations. There is however, a body of evidence that suggests the central nervous system may more readily employ viscoelastic properties intrinsic to the muscle as a means of controlling joint stability. The intrinsic elastic property of muscle has been investigated in detail, especially with respect to its role in postural control. Viscous properties have not yet been studied in great depth.

The objective of this study was to investigate the intrinsic and reflexive viscoelastic properties of the human wrist during the stabilization phase of a targeted voluntary movement. The oscillation of the hand about its final position was exaggerated by reducing the viscosity of the wrist joint with the aid of a robot manipulandum. The velocity trace was modeled to estimate the viscoelastic properties of the joint. Wrist stiffness increased when the target width was decreased. Both stiffness and viscosity increased for faster movements and decreased when the distance to the target was increased.

DEDICATION

To my parents – without your support this work could not have been accomplished

ACKNOWLEDGMENTS

I owe a great deal of thanks to my supervisor, Dr. Ted Milner, for his dedication, support, and insistence in high standards of research; for allowing me the freedom to make mistakes and learn from them. I am grateful to Dr. Parveen Bawa for her academic support and, more importantly, for her friendship. I owe thanks to my good friends Rod Rempel and David Franklin – one helped get me into the lab, the other urged me to get out. Thanks are extended to Charles Pierce for his technical help and insightful comments. Thanks are extended to all of my labmates with whom I worked, without whose friendship this thesis would not have been nearly as enjoyable. I am indebted to Shona McLean, Laurie Klak, and Sherry Lloyd – I would never have been able to weave my way through the administrative "challenges" without your help. I have very much appreciated the support and friendship of Dr. Silia Chadan, Craig Asmundson, and Dr. Richard Ward throughout my stay at SFU. Finally, I must thank my subjects who unselfishly volunteered their time for this study.

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INTRODUCTION

The neuromuscular system's ability to modulate joint mechanical properties is of fundamental concern in the study of human motor control. The mechanical properties of a joint determine how it will respond to perturbing forces during postural control and how it will act during voluntary movement.

In general, humans are able to maintain stable mechanical behaviour in the face of a wide range of mechanical operating environments. Goal directed movements are usually very smooth and there is a strong tendency to return to the original trajectory or target when the limb is perturbed. The stability of a joint is determined by its mechanical impedance or ability to resist disturbance forces that would move it away from its intended posture or trajectory. This is determined to a great extent by the viscoelastic properties of the muscles controlling a joint. The neuromuscular system can, therefore, change a joint' s mechanical impedance by modulating voluntary muscle activation and reflex feedback gains.

Considerable progress has been made in the understanding of postural control of single joints. Numerous single joint studies have been conducted to determine the mechanics of the joint during both imposed and voluntary movements. One main result of such studies is the finding that muscles are essentially spring-like and that the central nervous system may employ this characteristic in the control of posture and movement.

Recently, techniques have been developed to estimate the time-varying viscoelasticity of a single joint during movement (Bennett *et al.*, 1992; Bennett, 1993; Milner and Cloutier, 1993; Milner and Cloutier, 1997). These studies have indicated that the viscoelastic parameters depend on muscle activation and the speed of movement. These experiments have all been conducted with a fixed target size.

For purposes of analysis, a goal directed movement may be divided into a number of phases: pre-movement, acceleration to peak velocity, deceleration to the target, stabilization at the target, and post-movement. For many tasks, the stabilization phase is a critical component of the movement. Stability may be important in order to prepare for

another event (such as catching a ball) or to interact with the environment (such as moving a switch).

The purpose of this thesis was to investigate how the neuromuscular systems adapts the mechanics of a joint in response to different targets. In particular, the intent was to determine the viscoelastic characteristics of the wrist joint during the stabilization phase of a movement when the distance to the target was changed, the accuracy requirements of the target were changed, or the velocity of movement toward the target was changed.

For normal targeted human movements the oscillations about the target are small in amplitude and they damp out very quickly. For this study, the natural damping of the wrist was decreased by employing the novel technique of applying negative viscosity to the wrist with a torque motor (Milner and Cloutier, 1993). Using this technique, the number and amplitude of these oscillations were increased but the frequency of the oscillations remained at the natural frequency of the system. These oscillations were then modeled with a second order model to determine the viscoelasticity of the joint.

WRIST ANATOMY

The wrist is a complex joint allowing flexion, extension, abduction, adduction, and circumduction. The skeletal framework of the wrist consists of eight carpal bones that articulate with the distal aspect of the radius, the metacarpals, and with each other. The carpal bones are arranged in two transverse rows of four bones each, located in the proximal part of the hand. Together, the carpal bones form the carpus. The trapezium, trapezoid, capitate, and hamate bones form the distal row of the carpus. The proximal row is formed by the scaphoid, lunate, triquetrum, and pisiform. Within each row of carpal bones, the palmar, dorsal, and interosseous ligaments restrict movement. The proximal and distal rows of the carpus form the midcarpal joint. The ligaments allow more freedom of movement between the rows than between the individual carpal bones such that flexion and extension are possible. The distal end of the radius forms a concave surface that together with the articular disc of the ulna articulate over a convex surface formed by the proximal row of the carpus. The midcarpal joint and the wrist joint work in concert to increase the range of flexion and extension available to the hand.

Many degrees of freedom exist for the wrist joint due to the complex interactions available within and between the carporadial and midcarpal joints. However, the wrist as a whole essentially undergoes biaxial rotation about the flexion-extension and radial deviation-ulnar deviation axes. Although there is a great deal of variation between individuals, the normal ranges of motion of the wrist complex are generally considered to be 85-90 degrees of flexion, 75-80 degrees of extension, 15-20 degrees of radial deviation, and 35-37 degrees of ulnar deviation (Crouch, 1983).

The musculature that generates movement of the hand about the wrist is also complex. Most of the muscles that act across the wrist joint are biarticular because they have at least part of their origin on the distal end of the humerus. Nevertheless, it should be noted that these muscles have very slight actions on the elbow. The primary actions of the wrist muscles are in the movement of the hand and fingers. All of these muscles have their bellies in the proximal part of the forearm and have long tendons that cross the wrist. The tendons are held down at the wrist by flexor and extensor retinacula. These

transverse bands of fascia prevent the tendons from protruding when the hand is flexed or extended. As indicated in Table 1, the muscles may be classified based on their location and function. The muscles of the anterior group serve as flexors of the wrist and have their origin on the medial epicondyle of the humerus. These muscles insert on the carpals, metacarpals, or phalanges. Wrist flexion is effected predominantly by three muscles: flexor carpi ulnaris (FCU), flexor carpi radialis (FCR), and palmaris longus (PL). The finger flexors may also act in assisting wrist flexion. The posterior group of muscles acts as wrist extensors. These muscles have their origin on the lateral epicondyle or the lateral supracondylar ridge of the humerus and they insert on the metacarpals or phalanges. There are four primary wrist extensor muscles: the extensor carpi radialis longus (ECRL), extensor carpi radialis brevis (ECRB), extensor carpi ulnaris (ECU), and the extensor digitorum comunis. Although the wrist extensor muscles are antagonistic to the wrist flexors, the ECRL acts synergistically with the FCR to radially deviate the hand. In a similar manner, the ECU acts synergistically with the FCU to produce ulnar deviation of the hand. The above mentioned primary wrist flexors and extensors comprise the superficial group of muscles of the forearm. In addition, there are layers of deeper muscles I the forearm. These muscles are summarized in Table 1 together with their actions, origins, and insertions (Crouch, 1983).

Muscle	Action	Origin	Insertion
Anterior Group - Superficial Muscles			
Flexor carpi radialis	wrist flexion radial deviation	medial epicondyle of the humerus	base of the second and third metacarpals
Palmaris longus	wrist flexion	medial epicondyle of the humerus	Palmar aponeurosis
Flexor carpi ulnaris	wrist flexion ulnar deviation	medial epicondyle of the humerus, olecranon process, and the proximal two-thirds of the posterior surface of the ulna	pisiform, hamate, and base of the fifth metacarpal
Flexor digitorum superficialis	finger flexion wrist flexion	medial epicondyle of the humerus, coronoid process of the ulna, and the anterior surface of the radius	anterior surface of the second phalanx of each finger
Anterior Group - Deep Muscles			
Flexor digitorum profundus	finger flexion wrist flexion	medial epicondyle of the coronoid process of the humerus, the interosseous membrane, and the anterior surface of the ulna	anterior surface of the base of the distal phalanges of each finger
Posterior Group - Superficial Muscles			
Extensor carpi radialis longus	wrist extension radial deviation	lateral supracondylar ridge of the humerus	posterior surface of the base of the second metacarpal
Extensor carpi radialis brevis	wrist extension radial deviation	lateral supracondylar ridge of the humerus	posterior surface of the base of the third metacarpal
Extensor digitorum communis	finger extension wrist extension	lateral epicondyle of the humerus	posterior surface of the second and third phalanges of each finger
Extensor carpi ulnaris	wrist extension radial deviation	lateral epicondyle of the humerus	base of the fifth metacarpal
Posterior Group - Deep Muscles			
Abductor pollicis longus	thumb abduction wrist extension	posterior surface of the middle of the radius and ulna, and the interosseous membrane	base of the first metacarpal
Extensor pollicis brevis	thumb extension wrist extension	posterior surface of the middle of the radius, and the interosseous membrane	base of the first phalanx of the thumb
Extensor pollicis longus	thumb extension wrist extension	posterior surface of the middle of the ulna, and the interosseous membrane	base of the distal phalanx of the thumb

Table 1. Muscles that act about the wrist.

MECHANICS

Models of Muscle Mechanics

The A.V. Hill Model

The earliest model of muscle mechanics that is still widely used today is the macroscopic model proposed by A.V. Hill (Hill, 1938). Hill adopted a phenomenological systems physiology approach with his model to provide an understanding of the mechanics of muscle as a whole. The origins of this model may be traced back to the experiments of Gasser and Hill (1924) who conducted force and length change measurements of maximally stimulated isolated frog muscles. They showed that the contractile properties of muscle resembled a passive viscoelastic system. The Hill model was based primarily on thermal measurements of maximally stimulated muscle exposed to an isotonic quick release (Hill, 1938). Hill suggested that the mechanical properties of muscle could be modeled by a passive damped elastic element connected in series with a nonlinear contractile element.

In current versions of the Hill model, the contractile element accounts for the dynamics of muscle excitation and mechanical contraction. The energy conversion process from neural excitation to mechanical contraction is represented by equations that incorporate muscle length-tension and force-velocity properties as well as a rate limiting term representing calcium activation and deactivation. The maximum tension as a function of muscle length is multiplied by the activation level to determine the length-tension relationship. For a muscle that is shortening, the force-velocity relationship is defined by a hyperbolic curve in which the contractile force decreases as the shortening velocity increases. This curve is defined such that the x-intercept is located at the maximum velocity of unloaded shortening and the y-intercept is defined at the maximum isometric force. For a muscle that is lengthening, the force-velocity relationship has a very steep slope at low lengthening velocity and levels off to a constant force for higher velocities.

The series element represents the stiffness of passive connective tissue; it includes the passive elasticity associated with active and inactive muscle fibers, tendon, and aponeurosis (Zajac, 1989). The series element is modeled as a nonlinear spring whose length and stiffness is determined by the instantaneous muscle force.

Although Hill did not include it in his original model, the results of an experiment performed by Katz (1939) suggested that a second elastic element be added to the model. He suggested that this element should be in parallel with the contractile element and that it would be due to passive force generation from connective issues within and surrounding the muscle. In spite of the fact that the addition of the parallel elastic element improves the model, it is not usually included in muscle model simulations because it generates very little force except at the extremes of the range of motion, well beyond the normal physiological range.

The Sliding Filament Model

In contrast to the previous model, the Sliding Filament Model proposed by A.F. Huxley (1957) is a biophysical model of the dynamics of the actin-myosin interaction. This type of model allows insight into the actual mechanisms of muscle contraction at the molecular level.

Huxley assumed that a muscle's active mechanical properties are due to the interaction between actin and myosin. The model assumes that the head projecting from the myosin filament forms an elastic linkage or cross-bridge with the actin filament that can be attached or detached. When the muscle is activated, a cross-bridge will attach to the actin filament, undergo a conformational change creating a force that shortens the actin-myosin complex, and later detach from the actin filament. This cycle is repeated as long as the muscle is activated. The combined effect of cycling cross-bridges along the myosin filament produces a sliding of the actin filament with respect to the myosin filament. It is assumed that the cross-bridges act independently of each other, and attach and detach with rate constants that vary independently of the activation level.

The mechanical properties exhibited by whole muscle are a result of the sum of the actions of the myofilaments that comprise it. Force and stiffness depend on the availability of a binding site on the actin filament to which the myosin head may attach; therefore, these properties are dependent on the activation level. The force and stiffness of a muscle fiber are also dependent on the movement of the actin-myosin complex because cross-bridges can be forcibly detached due to over stretching, resulting in decreased force and stiffness in the fiber as a whole.

Huxley's model provides a relatively simple and concise mathematical description of the way muscles are believed to work on a molecular level. Although he showed that his model was consistent with many of the macroscopic features of muscle behavior, subsequent modifications have been made to the basic theory to account for new observations. For example, two attachment states were suggested to account for transient tension changes following step shortening and lengthening (Huxley and Simmons, 1971). Other researchers have suggested multiple states of attachment and detachment to account for the numerous complex details of muscle mechanical, biochemical, and thermodynamic behavior (Huxley and Simmons, 1971; Eisenberg and Hill, 1978; Eisenberg *et al.*, 1980)

Intrinsic Stiffness and Viscosity

Muscle Fiber Stiffness

When muscle is lengthened, a restoring force develops that resists the movement and tends to bring the muscle back to its original position. Early work in muscle mechanics suggested that muscles behave like nonlinear springs (Gasser and Hill, 1924; Fenn and Marsh, 1935; Hill, 1938). When a muscle is lengthened or shortened such that the cross-bridges are deformed, the cross-bridges generate a restoring force that opposes the displacement. Muscle, therefore, exhibits elastic behavior and consequently has the property of stiffness. Stiffness can be defined as either static or dynamic. When a muscle fiber is displaced and held at a new steady state length it generates a new steady state force. The static stiffness of a muscle fiber is defined from these two different steady states as the incremental change in force, ΔF, produced by the muscle in response to an

imposed change in length, ΔL, divided by that change in length. This relationship is summarized by the equation

$$K_m = \frac{\Delta F}{\Delta L}, \qquad [1]$$

where the variable K_m is the elastic coefficient of the muscle. Whereas static stiffness is easily measured, time varying stiffness is more difficult to measure. It must be estimated by modeling the mechanical behavior and estimating the elastic, viscous, and inertial contributions to the force. Some aspects of muscle behavior are best discussed in terms of compliance. Mathematically, compliance is the inverse of stiffness and is defined by the equation

$$C_m = \frac{\Delta L}{\Delta F}. \qquad [2]$$

In practice, a muscle's viscoelastic behavior is history dependent and therefore its compliance may not simply be the reciprocal of its stiffness for a given set of operating conditions.

The stiffness of a muscle fiber has both passive and active components. However, the length-tension curve shows that passive components only become significant when the fiber length approaches the maximum of its physiological range (Edman, 1966; Gordon *et al.*, 1966). If the mechanics are described in terms of the Sliding Filament Theory, the muscle fiber stiffness can be thought of as a relative measure of the number of attached cross-bridges. This is because stiffness is proportional to the number of attached cross-bridges. (Huxley and Simmons, 1971; Ford *et al.*, 1981).

Several studies have shown that the number of attached cross-bridges, and therefore, the stiffness varies with the velocity of a displacement (Edman, 1978; Ford *et al.*, 1985; Sugi and Tsuchiya, 1988; Lombardi and Piazzesi, 1990). As the stretch velocity is increased both force and stiffness increase with force rising more rapidly than stiffness (Lombardi and Piazzesi, 1990). Similarly, as the shortening velocity is increased force and stiffness decrease with stiffness falling more gradually than force. (Ford *et al.*, 1985; Edman, 1993). Griffiths *et al.* (1993) isotonically shortened muscle fibers at constant velocity and confirmed these results by estimating the number of attached cross-bridges

using x-ray diffraction techniques. They suggested that negatively strained cross-bridges could account for the difference between force and stiffness because they would reduce the tension and still contribute to the muscle fiber stiffness.

Muscle Fiber Viscosity

Materials that generate resistive forces related to the velocity of a disturbance are said to have the property of viscosity. Muscles exhibit this property and like stiffness, the relationship between force and velocity may be summarized by a simple equation

$$B_m = \frac{\Delta F}{\Delta V}, \qquad [3]$$

where B_m is the coefficient of viscosity of the muscle and ΔV is the change in muscle velocity. Like stiffness, instantaneous viscosity cannot be measured directly because any change in the length of a muscle fiber will result in the generation of elastic forces from the cross-bridges. Therefore, instantaneous viscosity must be estimated by modeling the dynamic behavior of stiffness and viscosity simultaneously. A steady state viscosity may be estimated from the slope of the force-velocity curve identified by A.V. Hill (1938). Based on this relation, the viscosity of a shortening muscle fiber should be maximum near zero velocity and approach zero as the shortening velocity is increased. Similarly, in lengthening fibers viscosity should be maximum near zero velocity and get smaller as the velocity is increased.

If viscosity is determined by the force-velocity curve, an explanation of the underlying physiology of viscosity depends on the physiological processes that account for the force-velocity relationship. There are different views on how the force-velocity curve is explained physiologically. Huxley originally proposed that the number of attached cross-bridges is related to the relative velocity of the sliding myofilaments. For muscle fibers lengthening at low velocity, he suggested that the average strain in the attached cross-bridges must increase. In addition, further attachments are made while these cross-bridges are stretched contributing to the enhancement of force. As the lengthening velocity is increased, the progressive increase in average strain is balanced by the reduced time that the cross-bridges may remain attached. Consequently, the force

enhancement decreases with lengthening velocity such that force becomes constant. When a muscle fiber shortens attached cross-bridges will be compressed and therefore generate negative tension which detracts from the overall force generated by the fiber. According to Huxley's theory, as the rate of shortening increases, a progressively larger population of the cross-bridges will not detach in time to prevent the generation of negative tension. The velocity of shortening therefore reaches a limit when the negative tension equals the positive tension.

Huxley has suggested that the force-velocity relationship of a muscle fiber may be explained in terms of to a change in the average cross-bridge strain as a function of velocity (Huxley, 1957; Ford *et al.*, 1985). Conversely Iwamoto *et al.*,(1990) suggest that the relationship is due to changes in the populations of attached and detached cross-bridge states. It should be noted that muscle fiber viscosity has not been attributed to a dashpot-like system where the movement of myofilaments is resisted by a viscous myoplasm.

The force-velocity relationship for the lengthening muscle may be determined by measuring the force change in response to constant velocity stretches of various lengths. When the velocity of stretch is low and the sarcomere lengths are long, the force is observed to rise rapidly followed by a very slow rise to a higher level until it reaches a plateau. This phenomenon is known as creep (Flitney and Hirst, 1978). As the stretch velocity is increased, the force increment gets progressively smaller. Lombardi and Piazzesi (1990) characterized the force-velocity relationship at high velocities as having two distinct phases. The first phase is transitory - the force rises to a velocity dependent maximum value and then decreases. Next, there is a steady state phase where the increase in force with velocity is low and it decreases progressively to a limiting value as velocity is increased further (Edman *et al.*, 1978; Lombardi and Piazzesi, 1990). The variability in the force response has been suggested to result from inhomogeneity in the distribution of length changes within the sarcomeres of a fiber (Colomo *et al.*, 1988). The length change for weak sarcomeres is much greater than that for strong sarcomeres. At high stretch velocities, the weak segments are responsible for most of the increase in velocity keeping the force increase small. In addition, Lombardi and Piazzesi suggest that at low stretch

velocity, the average strain in the attached cross-bridges increases and more cross-bridges become attached contributing to the force enhancement. As the velocity is increased, the time for a cross-bridge to attach is reduced and, therefore, fewer cross-bridges actually bond with the actin filament. The force generation at high velocity is, therefore, dependent on the cycling rate of the cross-bridges (Lombardi and Piazzesi, 1990; Lombardi *et al.*, 1992).

In contrast, Iwamoto *et al.* (1990) investigated the force-velocity relationship by varying the load on the fiber and measuring the resulting change in velocity. Their results showed that when a ramp decrease in load was applied to the fiber the force-velocity curve was always convex except for a hump in the high force region that increased as the velocity increased. They suggested that the increase in the shortening velocity was due to an increase in the proportion of cross-bridges that remain attached until the force is reduced to zero. They attempted to simulate this behavior with a model. However, they were not able to predict the experimentally observed hump at high forces and suggested that the series elastic component may be responsible for this effect. A significant finding from their work was that the slope of the velocity-force curve approaches infinity as the force approaches zero. This suggests that viscosity cannot simply be determined from the slope of the force-velocity curve in this region.

Whole Muscle Viscoelasticity

Stiffness

The elastic behavior of a whole muscle is complex and may be affected by several factors, some of which give rise to nonlinear behavior. In terms of the Hill model, stiffness is created passively by the stretching or shortening of the series and parallel elastic components. Although the model assumes that passive stiffness should be a fixed function for a given muscle, the length-tension dynamics are in fact nonlinear and, therefore, stiffness may change. The muscle is connected in series to its aponeurosis and tendon. The effect of this series connection is that the compliance of the entire musculotendon unit is obtained from the sum of the compliance contributions from the muscle, tendon and aponeurosis. Rack and Westbury (1969) showed that for small loads,

the stiffness of the tendon and aponeurosis is much greater than that of muscle; the length change in the musculotendon unit is therefore taken up by the muscle. As the imposed load increases, the tendon-muscle stiffness ratio decreases and more of the stretch is taken up by the tendon. In the cat, this ratio was constant for in isometric conditions for the force range of 20-80% of the maximal. (Baratta and Solomonow, 1991). The dynamic effect of the tendon is somewhat more complicated. In contrast to the static results of Baratta and Solomonow, Roleveld (1994) reported that the tendon's viscoelastic properties do, in fact, have a moderate effect on the dynamic response of a load–moving musculotendon unit. Furthermore, Griffiths (1991) found that the length change for slow to moderate stretches of maximally contracted isometric cat medial gastrocnemius muscle were taken up entirely in the tendons. For higher velocity stretches, the length change was shown to take place mostly in the muscle. Passive properties also result in a nonlinear dependence of stiffness on muscle length (Rack and Westbury, 1969). Single joint studies have shown that stiffness greatly increases towards the extremes of the range of motion of the joint as the passive elements are stretched but remains mains nearly constant in the joint's mid–range (Kearney and Hunter, 1990).

Active stiffness is generated intrinsically from the elastic deformation of the cross-bridges formed by actin-myosin bonds. The relation between force and intrinsic stiffness is complex and nonlinear because it depends on the amplitude and velocity of the length change, as well as the level of activation of the muscle prior to the displacement. In addition, time varying stiffness can be relatively high compared with static stiffness. Joyce *et al.* (1969) measured the force response of constant velocity movements generated with various stimulation rates of the cat soleus muscle. They suggested that the amplitude and activation dependence exhibited by the force could be summarized by the slopes of a family of length-tension curves. Rack (1966) applied sinusoidal oscillations to cat soleus muscle and showed that the stiffness increased approximately linearly with frequency of the oscillations and decreased nonlinearly with stretch amplitude. For small amplitude changes of up to 2% of the physiological range of the muscle length the change in force is proportional to the change in length (Rack and Westbury, 1974). Rack referred to the high time varying stiffness in this region as the

short-range stiffness. Experiments with human subjects have shown that the short-range stiffness increases linearly with the muscle's level of activation (Ma and Zahalak, 1985). The extent of this region also increases when the muscle is alternately stretched and shortened compared to when the muscle is held isometric (Rack and Westbury, 1974). Rack and Westbury suggest that this result occurs because the cross-bridges that attach when the muscle is shortened can be extended further before they are forcibly detached. For stretch amplitudes beyond this range a drop in force occurs which results in a short period of negative time varying stiffness called yielding (Joyce *et al.*, 1969). The yielding phenomenon occurs because the cross-bridges become stretched beyond the capacity of the actin-myosin bonds to resist the imposed strain. Consequently, they break and reattach at a new position on the muscle's length-tension curve. As the amplitude of the displacement is increased the stiffness decreases, because on average the cross-bridges will have to cycle more and will, therefore, spend more time in a detached state. The amount of yielding is inversely proportional to both velocity of stretch and level of muscle activation (Joyce *et al.*, 1969; Rack and Westbury, 1974; Nichols and Houk, 1976; Houk and Rymer, 1981).

When muscle is shortened, the yielding phenomenon is not observed and the muscle force drops continuously until the imposed length change is stopped. Joyce *et al.* (1969) showed that in contrast to lengthening, stiffness is independent of the shortening velocity. In addition, Nichols (1984) has shown that as the velocity of stretch increases, larger amplitude stretches are required to produce yielding.

Viscosity

Viscous mechanical properties are more difficult to measure than are static elastic properties because they must be measured under dynamic conditions and are generally of small magnitude. As a result, there have been far fewer studies examining muscle viscous properties than elastic properties. However, some work has been done to determine the mechanical effects of viscosity with respect to muscle activation, amplitude and velocity of displacement, and frequency of oscillation.

Like stiffness, muscle viscosity has been found to increase with activation level (Rack, 1966; Joyce and Rack, 1969). This result is in agreement with the muscle fiber theories and experiments that suggest that viscous properties result from cross-bridge mechanics. Because activation level also affects stiffness, the viscous and elastic properties had to be measured simultaneously in Rack's experiments (Rack, 1966; Joyce and Rack, 1969). Rack sinusoidally oscillated tetanized and passive cat soleus muscles with small amplitudes and low frequencies. He assumed that for small amplitude oscillations with constant activation, stiffness and viscosity could be considered constant. The experiments showed that viscosity increased with frequency when the muscle was oscillated at low frequencies (up to 5 Hz). This increase, however, was small compared with the corresponding increase observed for stiffness. As the frequency of oscillation was increased further, viscosity became constant and then began to decrease. Kirsch *et al.* (1994) applied stochastic perturbation sequences to the passive cat soleus and medial gastrocnemius muscles. They oscillated the muscle at much higher frequencies than those used in Rack's experiment and found that viscosity decreased nonlinearly with oscillation frequency in agreement with his results. Furthermore, these results indicated that viscosity decreased nonlinearly with increased velocity in agreement with the force-velocity curve.

Kirsch *et al.* (1994) also investigated the effect of oscillation amplitude on muscle viscoelasticity. Whereas stiffness was observed to decrease for small increases in oscillation amplitude, viscosity did not appear to change. In the same study, however, these investigators reported that both stiffness and viscosity decreased when the amplitude of step muscle stretches was increased. The relative decrease in stiffness was much greater than that of viscosity for these perturbations so that, as the stretch amplitude was increased, muscle properties became more viscous in nature.

Kirsch *et al.* (1994) compared predictions of three viscoelastic models to the stiffness frequency responses for stochastic perturbations of cat medial gastrocnemius muscle. The first model consisted of a stiffness and elastic element in parallel. The second model was similar to the first with the addition of a series elastic element to account for tendon compliance. The third model consisted of a stiffness and elastic

element in series coupled with a parallel elastic element. All models accurately predicted the stiffness magnitude. However, only the first model accurately predicted the observed phase shift towards 90 degrees as the frequency was increased. This model is essentially a second order linear model of muscle viscoelasticty.

The force-velocity curve has been shown to be asymmetric near zero velocity for both active and passive muscle (Harry *et al.*, 1990; Krause *et al.*, 1995). Harry *et al.* (1990) stretched and shortened active frog sartorius muscles at various constant velocities and measured the force response. Their results indicated that the slope of the force-velocity relation during shortening was approximately one quarter of the magnitude of the slope during lengthening. Krause *et al.* (1995) conducted a similar experiment using passive frog sartorius muscle. Like the previous experiment, their results showed that the force-velocity curve is asymmetric about zero velocity; furthermore, they found that the viscosity is discontinuous at zero velocity. These results provide further support for the idea that viscosity at low velocity should not be determined from the slope of the force-velocity curve and that alternative methods should be used for this measure.

Lin and Rymer (1993) investigated the mechanical properties of decerebrate cat soleus muscle in response to a stretches beyond the short-range stiffness region. They applied constant velocity stretches to the muscle while holding the initial length and activation constant. Their results were similar to those of the muscle fiber experiments performed by Lombardi and Piazzesi (1990) indicating that following yield the force response was essentially viscous. Lin and Rymer suggested that the force response of the yield and steady state phases of the stretch reflect a transition between the transient elastic behavior of the short-range stiffness to a more stable steady state viscous behavior. Kirsch's (1994) results also support these findings.

Single Joint Mechanics

Joint Viscoelasticity

The stiffness and viscosity observed at a joint in response to an angular perturbation is more complex than that of a single muscle because there are more

structural components contributing to the overall viscoelasticity. Generally, a torque about a particular axis of rotation cannot be achieved through the action of a single muscle. Synergist muscles must be activated such that the required torque vector is achieved through the vector summation of the torque produced by the individual muscles. The activation of synergist muscles, all of which have different moment arms, creates off axis torques which are balanced by stresses in bone, connective tissue, and contraction of other muscles. Another factor contributing to a more complex joint viscoelasticity is that when a joint is displaced, the agonist muscles are shortened and the antagonist muscles are stretched. The viscoelastic properties are, therefore, affected by the force of the shortening cross-bridges in the agonists and the resistive forces of the stretched cross-bridges in the antagonists.

Since muscle viscoelastic properties are nonlinear, joint viscoelastic properties are also nonlinear. Nonlinearities arise from the contribution to viscoelastic properties from multiple muscles, as well as the nonlinearities inherent in the properties of muscles themselves. In addition, the geometrical transformation between joint angle and muscle length involves nonlinear scaling by the moment arm. Frictional forces within the joint will also affect the joint dynamics.

Whereas the torque generated about a joint is a vector quantity whose magnitude is determined from the difference between the torques created by agonist and antagonist muscles, the joint viscoelastic properties are scalar. The elastic and viscous contributions from each muscle acting about the joint, therefore, sum to produce the viscoelastic properties of the joint. Consequently, the mechanical impedance of a joint my be increased by agonist/antagonist muscle cocontraction without generating torque. Joint stiffness and viscosity are defined in a similar manner as muscle stiffness and viscosity except that the joint properties are expressed as rotational quantities. Joint stiffness is defined as the change in torque, $\Delta\tau$, produced in response to a small angular displacement, $\Delta\theta$, divided by that change in angle

$$K_J = \frac{\Delta\tau}{\Delta\theta}. \qquad [4]$$

Similarly, joint viscosity is determined from the torque response to a change in angular velocity $\Delta\theta$

$$B_J = \frac{\Delta\tau}{\Delta\dot{\theta}}. \qquad [5]$$

A useful property when describing joint viscoelasticity is that of damping. Damping is a measure that describes how effective the viscous forces of an oscillating system are in decreasing the amplitude of the oscillations. An overdamped system that is displaced from its equilibrium position will return to that position without overshoot and slow down as it nears the equilibrium position and require a relatively long time to come to rest. An underdamped system will undergo a series of decaying sinusoidal oscillations about the final position before it reaches equilibrium. Damping depends on the inertia (I), viscosity, and stiffness of a joint. The damping ratio is defined as

$$\varsigma = \frac{\mathrm{B}}{2\sqrt{\mathrm{KI}}}. \qquad [6]$$

The damping coefficient is defined as:

$$\beta = \frac{\mathrm{B}}{2\,\mathrm{I}}. \qquad [7]$$

Since the inertia about a single joint is often almost constant, changes in the damping ratio are mainly the result of changes in joint stiffness and/or viscosity.

The torque about a joint produced by a particular muscle may be determined by multiplying its moment arm by the force that it generates. For small joint angle displacements, the change in joint angle may be determined from the muscle length change divided by the muscle's moment arm. Using these two relations, for a particular muscle the joint viscoelasticity can be related to muscle viscoelasticity as follows:

$$k_j = \frac{\Delta\tau}{\Delta\theta} = \frac{r\Delta F}{\Delta\ell/r} = r^2 k_m \qquad [8]$$

and

$$b_j = \frac{\Delta\tau}{\Delta\dot{\theta}} = \frac{r\Delta F}{\Delta\dot{\ell}/r} = r^2 b_m. \qquad [9]$$

Several system identification techniques have been used to measure joint viscoelastic properties. Some of these techniques include step torque perturbations (Gielen *et al.*, 1984; Gottlieb and Agarwal, 1988; Bennett *et al.*, 1992; Milner, 1993), step position displacements (MacKay *et al.*, 1986b; Bennett, 1993a; Bennett, 1993b), ramp and hold position displacements (Gielen and Houk, 1984; Gielen *et al.*, 1984; Ma and Zahalak, 1985; MacKay *et al.*, 1986b), sinusoidal length displacements (Agarwal and Gottlieb, 1977; Cannon and Zahalak, 1982; MacKay *et al.*, 1986b; Bennett, 1994), and stochastic perturbations (Hunter and Kearney, 1982; Kearney and Hunter, 1982; Weiss *et al.*, 1986a; Weiss *et al.*, 1986b; Weiss *et al.*, 1988).

Experiments employing oscillations of the human ankle oscillations have shown that joint stiffness is linearly dependent on joint torque (Agarwal and Gottlieb, 1977; Hunter and Kearney, 1982; Weiss *et al.*, 1986a; Weiss *et al.*, 1986b; Weiss *et al.*, 1988). With respect to the direction of joint movement, the torque-stiffness relationship has been shown to be asymmetric in the both the human ankle and the human wrist. For example, stiffness was greater with ankle extension than with ankle flexion in the ankle (Hunter and Kearney, 1982), but for the forearm it was greater with flexion than with extension in the wrist (Milner and Cloutier, 1993; Milner and Cloutier, 1997).

Like stiffness, joint viscosity has been shown to increase with activation (Hunter and Kearney, 1982; Becker and Mote, 1990). In contrast to stiffness, however, viscosity was shown to be greater for extension than for flexion in the wrist (Milner and Cloutier, 1997). Hunter and Kearney (1982) have suggested that such asymmetry may be due to differences in intrinsic or reflex properties between the active muscles.

As indicated previously, joint viscoelastic properties result from the additive elastic and viscous properties of the active muscles about the joint. The viscoelasticity of a joint may be modulated without changing its torque by cocontraction of the agonist and antagonist muscles. Several studies have reported that joint stiffness and viscosity increase with cocontraction (Lacquaniti *et al.*, 1982; Akazawa *et al.*, 1983; De Serres and

Milner, 1991; Milner and Cloutier, 1993). These properties provide the central nervous system with a very useful tool that may be employed to maintain stability.

In a series of experiments using stochastic perturbations, Weiss *et al.* (1986a, 1986b) investigated the passive and active mechanics of the human ankle joint. Their results showed that the stiffness and viscosity of passive joints exhibit position dependence (Weiss *et al.*, 1986a). They observed that the passive stiffness of the human ankle is small and nearly constant in the midrange of a joint's range of motion and that it increases dramatically at either extreme. The passive viscosity changed throughout the range of motion such that a uniform damping ratio was maintained. In contrast, the active mechanical characteristics for the human ankle did not show any position dependence (Weiss *et al.*, 1986b).

As with muscle viscoelasticity, small amplitude changes in joint angle result in mechanical characteristics that are largely dominated by the elastic properties of the cross-bridges. Stiffness has been shown to decrease linearly with increasing movement amplitude in experiments with the ankle (Agarwal and Gottlieb, 1977; Kearney and Hunter, 1982; Gottlieb and Agarwal, 1988), the wrist (Gielen and Houk, 1984; De Serres and Milner, 1991), and the elbow (Joyce *et al.*, 1974; Cannon and Zahalak, 1982; Ma and Zahalak, 1985; MacKay *et al.*, 1986a; Gottlieb and Agarwal, 1988). Fewer studies have reported the effects on viscosity. Kearney and Hunter applied position perturbations to the ankle and modeled the force response with a second order transfer function. Their results indicated that viscosity decreased with amplitude (Kearney and Hunter, 1982). This finding has been supported by the results of MacKay *et al.* (1986a) who applied step torque displacements to the elbow. Both of these experiments showed that the decreased viscous response was not as great as the decrease in the stiffness response. Gielen and Houk's (1984) experiment with the wrist indicated that the force dependence on velocity was, in fact, nonlinear. They showed that the force response varied as a power function of velocity, which had an exponent value of 0.17.

Recently, several investigators have looked at the viscoelastic characteristics of joints during voluntary targeted movements (Bennett *et al.*, 1992; Bennett, 1993b;

Milner, 1993; Milner and Cloutier, 1993; Milner and Cloutier, 1997). Bennett *et al.* (1992) investigated the phasic mechanical characteristics of the human elbow during sinusoidal movement by measuring the response to small pseudorandom torque perturbations. They compared the stiffness during movement to that when holding a steady posture. Their results showed that the stiffness dropped during the movement to such an extent that the maximum stiffness recorded for the movement was less than the minimum stiffness observed when subjects were required to hold a steady posture. Milner (1993) suggested that the larger stiffness seen during maintenance of posture in Bennett's experiments was due to a cocontraction strategy that is not employed when the limb is moved voluntarily. He found that during relaxed posture the elbow was more compliant than during movement. As the target was approached, the stiffness gradually increased. In contrast, the damping ratio was observed to change erratically during the movement and drop as it neared the target.

As with stiffness, viscosity has also been reported to increase in the human elbow as a target is approached so as to stabilize the joint (Bennett, 1994). Using a paradigm very similar to the proposed work, Milner and Cloutier (1993, 1997) investigated the damping characteristics of the wrist during rapid targeted flexion movements. Throughout the movement a load with negative viscosity was used to reduce the natural damping of the wrist. This had the effect of enhancing the oscillations about the target so that the time course of the viscoelastic properties could be modeled. The first study showed that cocontraction of the flexor and extensor muscles was used to stabilize the joint about the target. Furthermore, the stiffness was reported to increase with decreasing oscillation amplitude. The most recent study confirmed this result and revealed more detail about the effects of viscosity. Viscosity was reported to increase as the joint became more stable which is again consistent with the observation that viscosity increases with decreased oscillation amplitude or velocity.

Mechanical Stability

The mechanical impedance of a limb may be broken down into three types of resistance to motion - inertial, viscous, and elastic. The inertial component of impedance

depends on the mass m of the system, and produces a resistance to motion proportional to the acceleration of motion. Viscous friction resists the movement in proportion to the velocity of movement and the viscosity b. The elastic resistance to motion is proportional to the displacement of the limb and the stiffness k.

Rack (1981) used the transfer function of a second-order linear system to model the mechanical behavior of the human neuromuscular system. In order to describe the system he considered the movement of a load combining elastic, viscous, and inertial elements. A load that is driven by sinusoidal oscillations will have a displacement $r\sin(wt)$ where r is the oscillation amplitude, w is the angular frequency, and t is time. The inertial resistive force due to the mass of the load is $-mrw^2 \cdot \sin(wt)$. A viscous friction η, will provide a force sinusoid $\eta rw \cdot \cos(wt)$ resisting the movement. This force leads the movement by 90° and has increasing amplitude in proportion with the frequency of movement. The elastic impedance, k, for this movement is in phase with the displacement. The overall mechanical impedance for the load is determined from the magnitude of the vector sum of these three types of resistance to motion. The phase difference between the movement and the force response due to the system's mechanical impedance is, therefore, dependent on the relative magnitudes of the mass, viscosity, and stiffness of the limb.

Rack used a vector representation of the frequency response to describe the relationship between the amplitude and phase of the input/output properties of the human neuromuscular system. The human system was represented by a load combining elastic, viscous, and inertial elements. Rack showed that at low frequencies the total mechanical impedance is dominated by the stiffness of the elastic component resulting in a force component $kr\sin(wt)$ in phase with the position sinusoid. As the frequency of the driving oscillations increases, the viscous component adds a progressively larger force $\eta rw \cdot \cos(wt)$ and the inertial component adds a force $-mrw^2 \cdot \sin(wt)$, which increases even more steeply. Rack's analysis shows that for a limb held in a steady posture, the elastic component contributes the greatest to the overall mechanical impedance. As the limb is moved and the frequency of movement is increased, the viscous and inertial elements become more important in the determination of its impedance.

It can be assumed that the mass of a human limb does not change appreciably during a movement. For motion of about a single joint, the moment of inertia does not change if the configuration of the limb does not change with respect to the center of the rotation of the joint. Therefore, it can be assumed that, in this case, the inertia is constant and that the inertial resistive force in response to an imposed acceleration is both constant and immediate. The force response due to the intrinsic viscoelastic resistance from cross-bridge mechanics and connective tissues is also immediate because the restoring force is generated as soon as these structures are displaced. The force response from reflexes, however, is delayed due to the time required for the conduction of neural impulses through the neural pathways (including signal processing for transcortical reflex pathways), and excitation--contraction coupling.

Research in control theory has shown that the greatest benefit gained from feedback loops is that the control becomes less dependent on the dynamics of the actuator and more dependent on the control signal as the gain of the feedback is increased. It follows that if the overall mechanical impedance of the system is influenced more by reflex feedback than by the intrinsic muscle mechanics, then the mechanical behavior of the system becomes less sensitive to variations arising from nonlinearities in the behavior of the muscle. A servo-control system is most effective when it has a high feedback loop gain and a short delay because small errors in the feedback signal result in a large correction signal that is applied quickly. Control theory has also shown, however, that the effect of feedback control is somewhat limited as the frequency at which the system is controlled is increased. It is well known that the combination of high feedback gain and long feedback delays may lead to oscillatory instability.

Because the reflex latencies in the human neuromuscular system are significant, the effect of the reflex response cannot simply be determined from the magnitude of its force response. The delayed response will result in the reflex force being phase shifted relative to the other components of mechanical impedance. Reflex feedback contributes to muscle force whether the muscle is shortening or lengthening. Consequently, the phase of the movement in which the reflex force response occurs will influence the mechanical stability of the limb.

The effect of reflexes on mechanical stability has been investigated in a number of studies (Rack, 1981; Brown *et al.*, 1982; Jacks *et al.*, 1988; Prochazka and Trend, 1988; De Serres and Milner, 1991; Shadmehr and Arbib, 1992; Milner and Cloutier, 1993; Bennett *et al.*, 1994; Stein *et al.*, 1995). The movement frequency and reflex response latency determines the phase of the reflex force. If reflex force leads displacement by 90° such that it is in phase with the velocity of lengthening muscle, the effect of the reflex will be to oppose the movement with a viscous force. As the frequency of the imposed movement is increased, the phase lag of the reflex response increases (Rack, 1981). One result of Rack's analysis is that the combination of a long delay and high feedback gain has the potential to cause the system to become mechanically unstable. This effect has been shown in experiments with human subjects using driven sinusoidal movements of the thumb (Brown *et al.*, 1982), and elbow (Rack, 1981). If the reflex response lags the movement by 90° it will contribute a force proportional to velocity as the muscle is shortening. In effect, this adds negative viscosity to the system resulting in an assistive rather than a resistive force to the imposed movement. Long feedback delays can result in responses that are applied too late to have their intended corrective effect. If the feedback gain is sufficiently high, the corrective response will instead lead to oscillatory instability.

Theoretical analyses of joint stability confirm that long reflex feedback delays can indeed result in unstable mechanical behavior (Hogan, 1985; Shadmehr and Arbib, 1992). Hogan has argued that feedback can only be effective below a frequency that is determined by the length of the feedback delay. As this delay increases, the frequency at which feedback can significantly modulate impedance is decreased. He notes that instability rarely occurs in normal human motor control because the reflex feedback delays are within the temporal limits at which most tasks can be performed with stability.

Inherent properties of muscle also aid in the maintenance of stability. For example, in the generation of force muscles behave like low pass filters. When a muscle is stimulated by a slow train of α--motoneuron action potentials, it generates a similar train of force impulses. However, a fast train of action potentials will lead to a tetanic force output. The intermittent nature of the stimulation is filtered out and does not result

in an equivalent intermittent force response (Rack, 1981). The result of this low pass filtering behavior in the normal neuromuscular system is that the reflex force response from high frequency movements becomes attenuated. The reflex response to high frequency movements, therefore, has less of a destabilizing effect than does the response to low frequency movements (Bawa *et al.*, 1976; Bawa and Stein, 1976; Zahalak and Heyman, 1979). Perhaps a more important reason why instability is prevented in the normal system is that the CNS exploits the immediate resistance provided by the intrinsic viscoelasticity of muscle. Instability due to reflex feedback delay is, therefore, avoided entirely. If agonist and antagonist muscles are coactivated, the CNS can modulate the net joint stiffness and control the behavior of the system with greater stability (Hogan, 1985). This cocontraction strategy has been observed in human subjects with studies involving the finger (Akazawa *et al.*, 1983) and wrist (De Serres and Milner, 1991; Milner *et al.*, 1995). The disadvantage of this control strategy is that it involves a higher metabolic energy cost than is required for reflex mediated feedback. Presumably, the CNS will attempt to modulate the amount of cocontraction required for stable control such that the energy cost is minimized. Another property that aids in stability is the nonlinear nature of resistance to movements of different amplitudes. Joyce *et al.* (1974) showed that stiffness decreased nonlinearly with oscillation amplitude. This effectively reduces the natural frequency of the joint and, therefore, increases stability by limiting the growth of oscillations.

Despite the fact that these mechanisms are available to the neuromuscular system, instability resulting from reflex feedback has been observed by several investigators in both normal and pathological states. Excessive oscillations have been observed in subjects with neuropathological conditions such as Parkinson's disease, cerebellar dysfunction, spinal cord injury and hemiplegia (Stein and Oguztoreli, 1976; Stein and Lee, 1981). They have also been induced artificially (Jacks *et al.*, 1988; Prochazka and Trend, 1988; Milner and Cloutier, 1993; Bennett *et al.*, 1994). Prochazka and Trend (1988) applied frequency-modulated vibrations to the elbow flexor and extensor tendons of normal human subjects to produce reflex-generated movements. The application of the vibrations had the effect of artificially increasing the gain of the reflex pathway. At low levels of cocontraction, they observed activation dependent tremor in the forearm with a

frequency of 3-5 Hz. The oscillation frequency increased to 5-8 Hz for 100 percent cocontraction. Similar results were obtained in another experiment from their laboratory in which instability was induced in the forearm with amplitude-modulated electrical stimulation of the biceps and triceps muscles of human subjects (Jacks *et al.*, 1988).

Milner and Cloutier (1993) investigated the roles of intrinsic muscle mechanics and reflex feedback in stabilizing targeted movements. Their subjects were required to perform wrist flexion movements while exposed to a load that had the property of negative viscosity, thus removing the natural damping of the wrist. It was shown that the oscillation amplitude increased when the natural frequency was close to a critical frequency of 6-7.5 Hz. When the wrist oscillated near this critical frequency, the reflex torque lagged the angular velocity of the wrist by 180°. Although the oscillations were observed to increase, mechanical instability was never observed. The authors suggested that stability was maintained because the torque contributed by the intrinsic muscle impedance dominated the reflex torque. This result lends further support to an earlier study conducted in their laboratory in which the torque produced in response to a stretch of the wrist originated mostly from intrinsic mechanics rather than reflex pathways (De Serres and Milner, 1991). In addition, this result is supported by an experiment conducted by Bennett (1994). He used sinusoidal position perturbations of the human elbow to show that the mechanical impedance of the arm decreased for frequencies between 6-7 Hz, due to reflex delay.

Numerous single joint studies with human subjects have shown that joints are underdamped during maintenance of posture (Agarwal and Gottlieb, 1977; Gottlieb and Agarwal, 1978; Hunter and Kearney, 1982; Becker and Mote, 1990) and during voluntary movements (Bennett *et al.*, 1992; Bennett, 1993b; Milner and Cloutier, 1993; Milner and Cloutier, 1997). Whereas Bennett (1992, 1993b) reported that the damping ratio of the elbow joint was erratic during movement and decreased as the limb neared a target window, a subsequent experiment indicated that the joint viscosity scaled with the joint stiffness so as to maintain constant damping (Bennett, 1994). Becker and Mote (1990) reported that the damping ratio increased with muscle activation and decreased as muscle fatigue increased.

For many tasks, the transition between limb deceleration and steady posture is perhaps the most important part of the movement. During this part of the movement, the limb must be stabilized in order for the task to be successfully performed. The present study was intended to explore in detail the viscoelastic joint properties of the human wrist during the stabilization phase of a voluntary targeted movement. In particular, it was designed to investigate how its viscoelastic properties change when the characteristics of the target change. There were two main components to this study. First, the effects of an accuracy constraint on the viscoelastic properties exhibited by the wrist were investigated by requiring subjects to stabilize the hand in target windows of different widths. Previous studies investigating viscoelastic properties during voluntary targeted movements have all used a single target window with a single accuracy constraint (Bennett *et al.*, 1992; Bennett, 1993b; Milner and Cloutier, 1993; Milner and Cloutier, 1997). The second component of this experiment was to investigate the effect of velocity on the viscoelastic joint properties as the joint is stabilized. Bennett (1993b) looked at the joint stiffness during movement as a function of velocity although he did not focus on a particular phase of movement as was done for this study. With few exceptions, previous experiments where viscosity has been investigated have used a single velocity. Furthermore, researchers who have employed different ramp velocities have examined only imposed movements as opposed to voluntary movements (Gielen and Houk, 1984; Gielen *et al.*, 1984).

We know very little about how the neuromuscular system controls joint mechanical properties during stabilization. The results of this experiment allow us to determine the variation of viscoelastic parameters with different target characteristics. This will lead to greater insight into the control schemes used by the neuromuscular system. We can then better predict how the system will behave when exposed to various environmental conditions.

METHODS

General Overview

To examine the effects of target constraints and the velocity of movement on joint viscoelasticity, subjects were required to make repeated wrist flexion movements from a stationary position and stabilize the hand within the boundaries of a target window. The natural damping of the wrist was removed during the stabilization phase to exaggerate the natural oscillation. Subjects made movements of two different amplitudes with various combinations of three accuracy constraints, three movement velocities, and three levels of negative viscosity. The experiment was conducted on two consecutive days. On the first day of testing, subjects performed a series of wrist flexion movements for training. They were exposed to each of the experimental conditions so that modulation of wrist mechanics due to practice effects would be diminished. In addition, the moment of inertia of the wrist and manipulandum was measured and the relative levels of negative viscosity required to remove natural damping were determined. Following a brief retraining period on the second day, subjects again performed the flexion movements under the same experimental conditions. This time, however, the natural damping of the wrist was unexpectedly removed by applying a load with negative viscosity on random trials.

Subjects

Ten male right-handed volunteers without known history of musculoskeletal disorders participated in this study. The subjects ranged in age from 22 to 43 years with a mean age of 30.6 years. The experiment was conducted with the right hand only.

Apparatus

Subjects were seated comfortably with the right forearm resting on a padded support. The hand was secured in a wrist manipulandum by clamping the palm such that the hand was positioned midway between pronation and supination (Figure 1). The manipulandum was coupled to a torque motor (PMI U16M4) and the subject was positioned with his flexion/extension axis centered over the shaft of the motor. Because

the wrist rotates about two axes formed by the two rows of carpal bones, there is no anatomically defined center of rotation in its flexion/extension plane. In order to align the joint and motor axes each subject's range of motion when secured in the manipulandum was compared with their normal range of motion. The axes where assumed to be colinear when the subject had the largest possible range of motion with no lateral movement of the radial and ulnar proximal heads. The manipulandum stabilized the wrist, restricting movement to the horizontal plane. Movement of the subject's fingers was restricted by splinting the hand with Thermoplast® splinting material. Movement of the wrist was, therefore, restricted to that produced by the wrist flexor and extensor muscles alone.

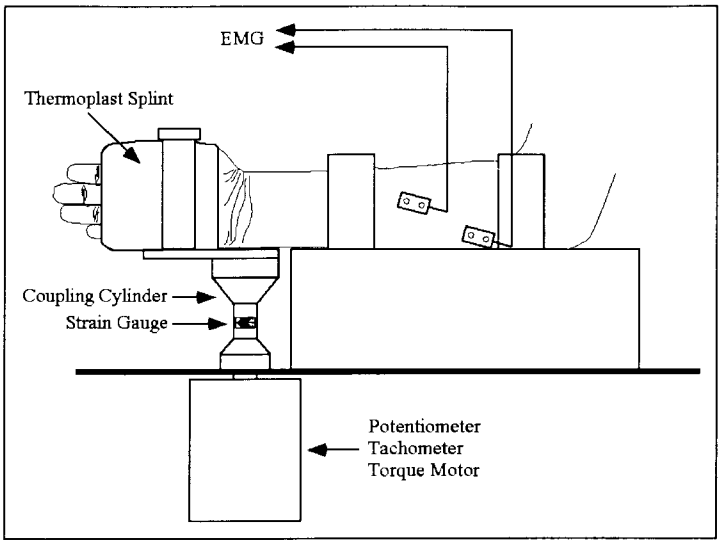

Figure 1. The apparatus is constructed so that the right arm is secured to a supporting block maintaining the hand in a neutral position half way between pronation and suppination. The hand is immobilized in the position of function with a Thermoplast® splint and secured to the manipulandum restricting movement to the flexion - extension plane. The EMG electrodes for the flexor muscles are diagrammed.

The manipulandum was servo controlled with a Pentium 100 MHz computer. The motor could generate a maximum torque of 5 Nm. Kinematic measurements were made with a potentiometer and tachometer coupled to the shaft of the motor. Position data from the potentiometer were accurate to ±0.75 degrees, accounting for noise. Angular velocity measurements with the tachometer were accurate to ±1.5 degrees/second. Pilot

experiments showed that more error resulted from intertrial variability than from the measurement accuracy of the apparatus. To provide information about muscle activation patterns, electromyographic activity was recorded from four superficial muscles of the forearm: ECU, ECRL, FCU, and FCR. EMG was recorded using active, bipolar, stainless steel surface electrodes (Liberty Mutual MYO 111) bandpass filtered with cut-off frequencies of 45 and 550 Hz to remove electrical noise. The placement of the electrodes over each muscle was determined by observing the EMG activity with an oscilloscope while the subject performed flexion and extension movements. The electrodes were placed where each signal was maximal. The position, velocity, torque, and EMG measurements were amplified and digitized at 2 kHz.

Moment of Inertia Measurement

The moment of inertia, *I*, is defined as torque divided by angular acceleration

$$I = \frac{\tau}{\ddot{\theta}}. \qquad [10]$$

Torque was measured with a linear strain gauge attached to a cylinder coupling the shaft of the motor and the manipulandum. Angular acceleration was obtained by numerical differentiation of the angular velocity record measured with the tachometer. These data were low pass filtered at 200 Hz with a first order Butterworth filter. Small amplitude high frequency oscillations were applied with the torque motor and the resulting torque and angular acceleration traces were recorded. The moment of inertia of the wrist and manipulandum could then be determined by dividing the torque record by the angular acceleration record during periods of high acceleration. Because reflex forces are delayed with respect to their time of initiation, it can be assumed that forces due to reflexes would not be in phase with the driving torque applied by the motor. Because the torque and acceleration records were not phase shifted with respect to one another, it can be assumed that reflex forces generated in response to the driving oscillation were either non existent or too small to have a measurable effect on the inertia estimate.

Viscoelastic forces from the motor shaft or coupling cylinder due to torsion could

be neglected because the wrist-manipulandum system was oscillated at a frequency well below the natural frequency of the motor shaft and coupling cylinder (19Hz). The periods of high acceleration occurred when velocity was lowest. During these periods of high acceleration, viscous forces generated from the wrist muscles would be relatively small and can be considered negligible. However, elastic forces due the wrist musculature need not be small. Consequently, the wrist-manipulandum system was sinusoidally oscillated at two different frequencies: 8 Hz and 10 Hz so that both stiffness and inertia could be estimated. The driving torque for these oscillations was adjusted so that the peak to peak amplitudes were the same for both frequencies. If θ is the peak amplitude and θ_0 is an offset (the zero point), the equation of motion is

$$\tau - \left(K(\theta - \theta_0) + I\ddot{\theta}\right) = 0 . \qquad [11]$$

The stiffness, K, and moment of inertia, I, can be estimated using a least squares approach to minimize

$$\sum \left[\tau_i - \left(K(\theta_i - \theta_0) + I\ddot{\theta}_i\right)\right]^2 , \qquad [12]$$

where the subscript i refers to the data sampling matrix. The minimization is done by setting the derivatives of Equation 12 equal to zero. This leads to the following set of equations

$$K\sum(\theta_i - \theta_0)^2 - I\sum\left((\theta_i - \theta_0)\ddot{\theta}_i^2\right) = \sum\left((\theta_i - \theta_0)\tau_i\right), \qquad [13]$$

and

$$K\sum\left((\theta_i - \theta_0)\ddot{\theta}_i\right) - I\sum\ddot{\theta}_i^2 = \sum\ddot{\theta}_i\tau_i . \qquad [14]$$

This system of equations has solutions for inertia and stiffness as follows:

$$I = \frac{\sum\left((\theta_i - \theta_0)\ddot{\theta}_i\right)\sum\left((\theta_i - \theta_0)\tau_i\right) - \sum(\theta_i - \theta_0)^2\sum\ddot{\theta}_i\tau_i}{\left(\sum\left((\theta_i - \theta_0)\ddot{\theta}_i^2\right)\right)^2 - \sum(\theta_i - \theta_0)^2\sum\ddot{\theta}_i^2}, \qquad [15]$$

and

$$K = \frac{\sum\ddot{\theta}_i^2\sum\left((\theta_i - \theta_0)\tau_i\right) - \sum\left((\theta_i - \theta_0)\ddot{\theta}_i\right)\sum\ddot{\theta}_i\tau_i}{\sum\ddot{\theta}_i^2\sum(\theta_i - \theta_0)^2 - \left(\sum\left((\theta_i - \theta_0)\ddot{\theta}_i\right)\right)^2} . \qquad [16]$$

The subject's hand was oscillated for 8 seconds at either 8 or 10 Hz with the subject instructed to relax. The position trace was then analyzed for compliance with this instruction by looking for variations in the maxima and minima. This method was very sensitive, often showing the effects of involuntary muscle contractions that were not actually perceived by the subject. The position, acceleration, and torque values were recorded from oscillations during the last few seconds to avoid any artifact induced by the motor when it was first turned on. Following these calculations, the system was oscillated at the other frequency. These position, acceleration, and torque values were added to the data matrix. The same number of peaks was added to the matrix for both frequencies. Inertia and stiffness values were then calculated using Equations 15 and 16. Ten estimates for stiffness and inertia were obtained and these results were averaged for a final value for each subject.

It was also necessary to account for the moment of inertia of the motor shaft. This value was determined by rapidly accelerating the manipulandum toward a physical stop with the motor turned off. The peak torque observed before impact was then divided by the peak acceleration to obtain the moment of inertia. The moment of inertia of the motor shaft was determined to be 0.00056 Nm. This value was added to each moment of inertia measurement of the wrist-manipulandum system.

The method of estimating moment of inertia was validated using metal plates of known moment of inertia. The moment of inertia of a square metal plate of mass, M, and length, x, is given by

$$I = \frac{Mx^2}{6}. \qquad [17]$$

The plates were oscillated and the moment of inertia estimated in the same manner as the hand. The measured moment of inertia should yield the same values as those calculated using Equation 17.

Experimental Procedure

When the subject was ready to begin he was presented with a video display as shown in Figure 2. The display indicated the desired target distance, target width, and peak velocity range. With the assistance of visual feedback from the video display the subject would start by placing a hairline cursor in a one degree wide start window located twenty degrees into extension. When a steady position had been maintained within this window for 500 ms the cursor changed colour signaling the subject that a movement could be initiated. At this time, the controller was programmed to compensate for friction so that the subject was presented with a frictionless axis about which to move. The subject then moved the cursor to the indicated target window and attempted to stabilize the movement within the target boundaries. Following the movement, knowledge of the results of the peak velocity and level of accuracy was provided via the video display. Peak velocity was represented as a bar graph, above the velocity target display - the bar turned green if the correct velocity range was achieved. The subject was provided with feedback of accuracy by showing his actual range of movement with respect to the left and right boundaries of the target during the stabilization period. Figure 2 shows the display as a result of unsuccessful trials due to (a) incorrect velocity (b) movement outside the target boundaries and (c) a successful trial in which the movement stayed within the required velocity range and target boundary.

On random trials in which the correct maximum velocity was achieved, one of three levels of positive velocity feedback was used to control the motor. The positive velocity feedback was applied immediately after the wrist velocity reached zero in the target window. This created a load with the characteristics of negative viscosity that effectively reduced the damping of the wrist so that the hand behaved like an underdamped oscillator.

Subjects were instructed not to react to the increased oscillations produced as a result of the decreased damping. This instruction was similar do the "do not resist" instruction which has been shown to be effective by Crago *et al.* (1976) and Lacquaniti *et al.* (1982). Crago *et al.* (1976) investigated the ability of human subjects to voluntarily modify their responses to mechanical disturbances of the elbow. They reported that

subjects could suppress their long latency reflex responses when given the instruction to "not intervene voluntarily." Lacquaniti *et al.* (1982) applied large perturbations to the elbow with viscoelastic loads. They reported that when subjects were given the instruction not to resist the perturbations and to remain as relaxed as possible, the EMG activity was silent except for one very small burst of triceps activity concomitant with the first (and largest) flexor perturbation.

Three levels of negative viscosity were chosen based on observations during the training phase of the experiment. The amount of negative viscosity ranged from almost imperceptible, yet large enough to enhance oscillations, to a level that induced large oscillations without causing the oscillations to grow. The three levels of negative viscosity where used so that different oscillation amplitudes and velocities would be produced for each of the trial conditions. The effect of amplitude and velocity dependence of stiffness and viscosity could then be investigated.

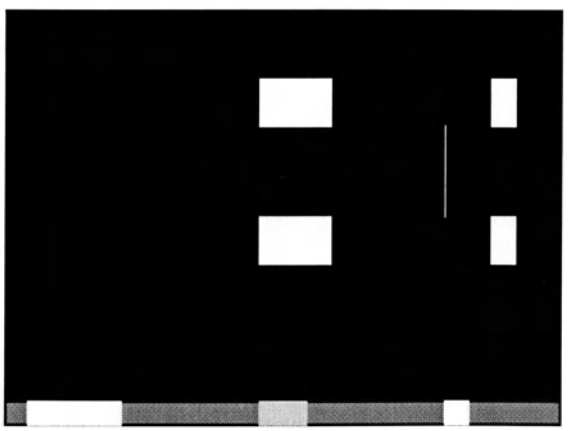

Figure 2. A video display is presented to the subject when he is ready to begin a movment. The hair line cursor travels from right to left on the display for a flexion movement. In this diagram, the display shows a start window to the right of the cursor and the target window to the left. The desired peak velocity range is indicated in the bar at the bottom of the display. In this case the middle range has been selected as indicated by the darker colour. (220 deg/s).

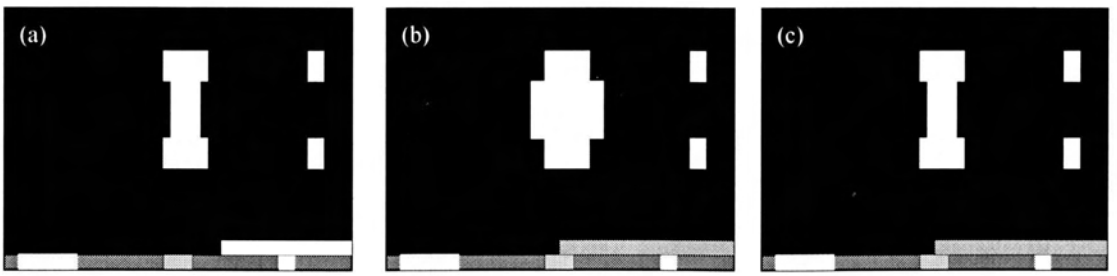

Figure 3. Visual feedback following a trial. In panel (a) the movement was unsuccessful because the peak velocity was lower than the desired peak velocity range. In panel (b) the movement was unsuccessful because the subject failed to stabilize his hand within the desired target boundary. The feedback for a successful movement is shown panel (c).

The EMG, torque, velocity, and position traces were displayed following trials where the subject met the velocity and accuracy constraints whether or not the negative viscosity was applied. These traces were examined for evidence of compliance with the "do not react" instruction. This was indicated by deviations in the velocity record from smoothly decaying oscillations or from bursts in the EMG records that did not appear in previous records.

To test the effects of target amplitude, subjects moved to one of two target windows requiring flexion movements of 20 or 40 degrees. These movement were chosen because they are well within the operating range of normal human movements. The target window had a width of 3, 5, or 7 degrees to test the effects of accuracy. Pilot experiments revealed that the 3 degree target was the smallest width for which subjects could repeatedly stabilize oscillations. These experiments also showed that the 7 degree target width was narrow enough that subjects did not reach a ceiling effect. Movements were made with each of the six combinations of these conditions for a target peak velocity of 220 degrees/second.

To test the effects of movement velocity two more target peak velocities were used. Movements of 20 degrees were made at a slower target velocity (120 degrees/second) with the 3 and 5 degree target windows. This target velocity was chosen to be fast enough to ensure that the movement was made as a single smooth trajectory without sub-movements (Milner and Ijaz, 1990). The 7 degree target window was not used at this velocity because pilot data had shown that the 5 and 7 degree target windows

had the same effect. A high target velocity (420 degrees/second) was used for movements of 40 degrees with the 5 and 7 degree target windows. This velocity was chosen to be slow enough that the task could be successfully completed with reasonable frequency. Pilot experiments showed that at this velocity subjects could not consistently stop a movement within the 3 degree target window. Therefore, this combination of peak velocity and target boundary was omitted. In total, there were ten movement conditions as summarized in Table 2.

Amplitude (degrees)	Accuracy (degrees)	Velocity (degrees/second)
20	3	120
20	5	120
20	2	220
20	5	220
20	7	220
40	3	220
40	5	220
40	7	220
40	5	420
40	7	420

Table 2. Summary of the experimental conditions.

For each movement condition, three levels of negative viscosity were used for a total of thirty experimental conditions. Three successful trials for each movement condition were obtained. On average, the negative viscosity loads were presented once every five trials.

In order to control for any training effects, subjects were required to perform two practice sessions. On the first day, subjects were presented with each of the movement conditions, without the addition of negative viscosity. They were required to perform movements under each condition until fourteen successful trials were made. Further trials were then performed with various levels of negative viscosity to ascertain appropriate limits as discussed above. Prior to the data collection on the next day, each subject performed the flexion movements until three successful trials were achieved for each of the movement conditions without exposure to the negative viscosity loads. To ensure that

the practice effects had indeed stabilized, the peak velocity values for blocks of data with similar trial conditions were compared for trials near the beginning of the experiments and at the end of the experiment.

ANALYSIS

The analysis of the EMG data and the parameter estimation of wrist stiffness and viscosity was carried out off-line. Trials were rejected if the stabilization phase of the movement showed evidence of reaction to the effects of negative viscosity (non-uniform velocity or position traces) or if fewer than three half-cycles of oscillation within the target window were apparent. Other criteria for rejection were excessive cocontraction at any time during the movement or a non-zero mean velocity at the end of the movement indicating a position drift away from the target. Excessive cocontraction was determined by visual inspection and comparison of the EMG activity to previous trials.

EMG Analysis

The EMG activity from the individual trials were quantified by determining the root mean squared values during four time intervals as defined by Milner and Cloutier (1993). These time intervals correspond to different phases of the task: (1) pre-movement, (2) movement to the target, (3) stabilization at the target, and (4) post-movement. Using this method, the interval boundaries were determined such that the EMG for a particular interval would be expected to contribute to the wrist torque during the corresponding phase of the movement.

Mechanical Impedance Parameter Estimation

The viscoelastic parameters were estimated using the velocity trace. The combined system of the wrist, Thermoplast® cast, manipulandum, and torque motor may be modeled as an underdamped harmonic oscillator as described by the following equation:

$$I\ddot{\theta} + B\dot{\theta} + K\theta = -B_m\dot{\theta} \qquad [18]$$

where I is the combined inertia of the wrist, plastic mold, manipulandum, and motor, B is the wrist viscosity, K is the wrist stiffness, and B_m is the viscosity of the motor. The inertia was measured as previously described. The oscillation amplitude and angular

velocity were measured directly with the potentiometer and tachometer. The solution to Equation 17 for θ(t) can be differentiated to give

$$\dot{\theta}(t) = -\frac{K\theta_0}{I\omega} e^{-\beta t} \sin \omega t , \qquad [19]$$

where

$$\omega = \sqrt{\frac{K}{I} - \left(\frac{B + B_m}{2I}\right)^2} \qquad [20]$$

and

$$\beta = \frac{B + B_m}{2I} . \qquad [21]$$

By convention, θ_0 represents the maximum oscillation amplitude, ω is angular velocity, and β is the damping parameter. The inertia, I, was independently measured as described above while θ_0 was measured from the position records. Stiffness and viscosity were then estimated by fitting Equation 19 to the velocity trace and solving for K and B.

Parameter estimation was conducted by fitting Equation 19 to the velocity data with a nonlinear least squares routine. A modified Marquardt--Levenberg procedure was employed which combines two nonlinear least squares methods. A "steepest descent" algorithm is used far from the local minimum, then as the minimum is approached the procedure continuously transforms into an inverse Hessian method to get a good local approximation to the solution (Press *et al.*, 1989). This method has been used successfully by Milner and Cloutier (1993; 1997) who reported that the variance accounted for by the model was almost always greater than 99%.

This second order model is not valid over the whole of the stabilization phase of movement because stiffness varies with amplitude and viscosity varies with velocity. Since the viscoelastic parameters are time varying, the model is only locally valid over a small range. Consequently, the viscoelastic parameters were estimated separately over each half-cycle of the oscillation and considered to be constant for that particular interval. The oscillations occurred over a very narrow bandwidth that was close to the natural frequency of the joint. As a result, the amplitude and velocity dependence of the

viscoelastic parameters could be considered to be more important than their frequency dependence.

The analysis was carried out as follows. First, the velocity trace was displayed with vertical bars separating each half-cycle as defined by velocity zero crossing points. The number of half-cycles to be analyzed was then chosen and the stiffness and viscosity were estimated by fitting Equation 19 to the velocity for each half-cycle. For each set of estimated parameters, the variance accounted for by the model was calculated to determine the goodness of the fit. The viscosity parameter estimated by the model is that of the wrist/motor system. Therefore, the negative viscosity that was applied by the motor was subtracted from this value to determine the viscosity of the wrist alone.

Statistical Analysis

This experiment has a three factor within subjects design, with factors: accuracy, target amplitude, and target peak velocity. Not all of the experimental conditions were used with every other condition. For example, the 3 degree target width was not used with the 420 deg/s peak target velocity and the 7 degree target width was not used with the 120 deg/s peak target velocity. Consequently the experiment was an incomplete design and the data was grouped into blocks of trials with similar conditions. The statistical analysis could have been carried out using t-tests. However, to reduce the possibility of type I error inherent to multiple t-tests, one-way analysis of variance tests were conducted on each of the blocks to test the effect of a particular factor. A summary of the eleven one-way analysis of variance tests is presented in Table 3. A significance level of 0.05 was used for all statistical tests.

Effect of Target Width			
Comparison	Target Amplitude (deg)	Target Peak Velocity (deg/s)	Target Width (deg)
A	20	120	3 5
B	20	220	3 5 7
C	40	220	3 5 7
D	40	420	5 7

Effect of Target Peak Velocity			
Comparison	Target Amplitude (deg)	Target Width (deg)	Target Peak Velocity (deg/s)
A	20	3	120 220
B	20	5	120 220
C	40	5	220 420
D	40	7	220 420

Effect of Target Amplitude			
Comparison	Target Peak Velocity (deg/s)	Target Width (deg)	Target Amplitude (deg)
A	220	3	20 40
B	220	5	20 40
C	220	7	20 40

Table 3. Trial grouping for statistical comparisons.

RESULTS

Moment of Inertia

The measured moment of inertia of the combined hand, Thermoplast® cast, and manipulandum system ranged from 0.00322 kg-m^2 to 0.00541 kg-m^2 with an average of 0.00469 kg-m^2. Subtracting the moment of inertia of the manipulandum and Thermoplast® cast, 0.00127 kg-m^2, the moment of inertia of the hand ranged from 0.00195 kg-m^2 to 0.00414 kg-m^2 with an average value of 0.00342 kg-m^2. The moment of inertia of the motor shaft was 0.00056 kg-m^2.

For one subject the moment of inertia of the hand was estimated with an anthropometric method which uses measurements of the wrist to first interphalangeal joint of the middle finger length, the wrist to middle finger tip length, and the subject's body mass (Winter, 1990). The moment of inertia of the hand using this method was 0.0037 kg-m^2 compared with 0.00342 kg-m^2 for our method.

The results of the moment of inertia validation procedure are diagramed in Figure 4. Error bars are not shown on the figure because, in all cases, they were smaller than the data point symbol. The regression equation for the actual and estimated inertia data has slope = 0.96 $\pm$ 0.02 and intercept = 0.00003 kg-m^2 $\pm$ 0.00015 kg-m^2. The estimated inertia values are slightly higher than the actual values. Ideally, the slope should be equal to one and the intercept should be zero. The slope value indicates that the combined error of the torque and velocity conversion factors is approximately 4%. This could be the result of too large a torque conversion factor and/or too small a velocity conversion factor. The intercept was not statistically significant from zero. The inertia estimates were corrected for this 4% error for the viscoelastic parameter estimation procedure. The values reported above are the corrected estimates of inertia.

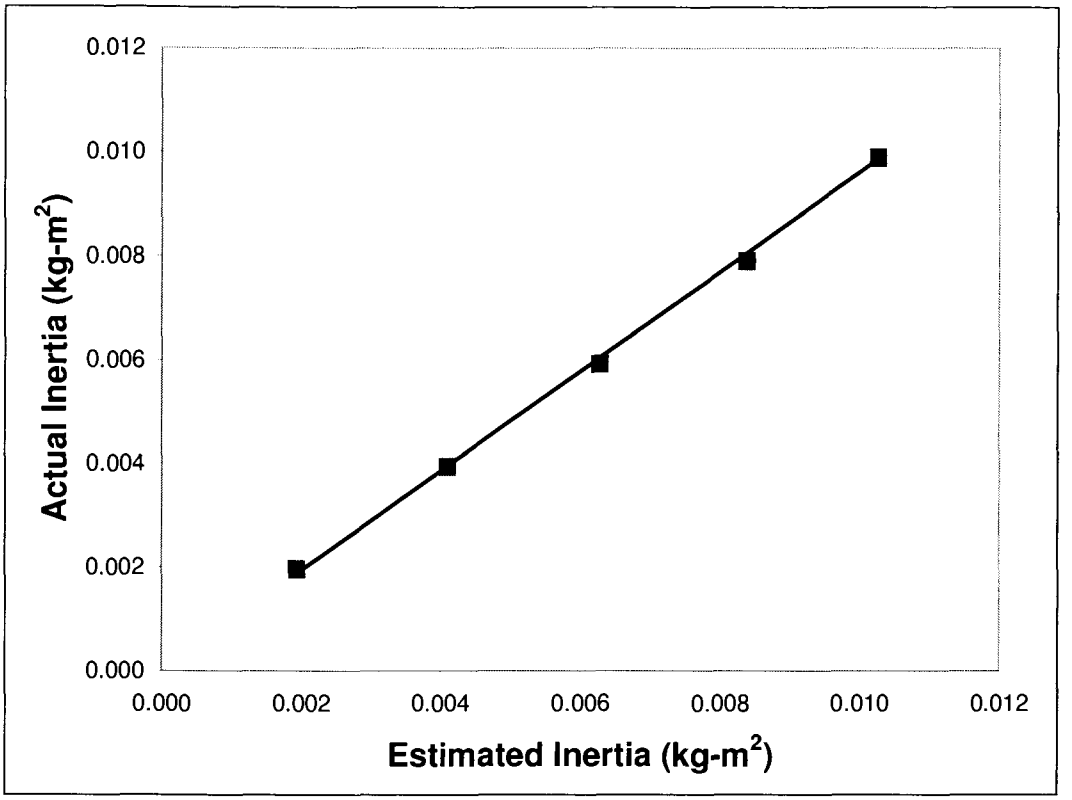

Figure 4. Calibration chart for the measurement of moment of inertia. The hand measurements listed in the legend are the moment of inertia of the hand-Thermoplast® cast-manipulandum system.

Kinematics

Effect of Negative Viscosity

The application of negative viscosity at the start of the stabilization phase of the movement had the intended effect of decreasing the natural damping of the wrist. This resulted in more oscillations with larger amplitudes for all velocities (Figure 5). Generally, the oscillations were more symmetrical and sinusoidal when the target peak velocity was 220 deg/s or 420 deg/s as compared with 120 deg/s. In addition, more irregularity in the oscillation amplitude was observed in blocks of movements performed under the same experimental conditions when the target peak velocity was 120 deg/s.

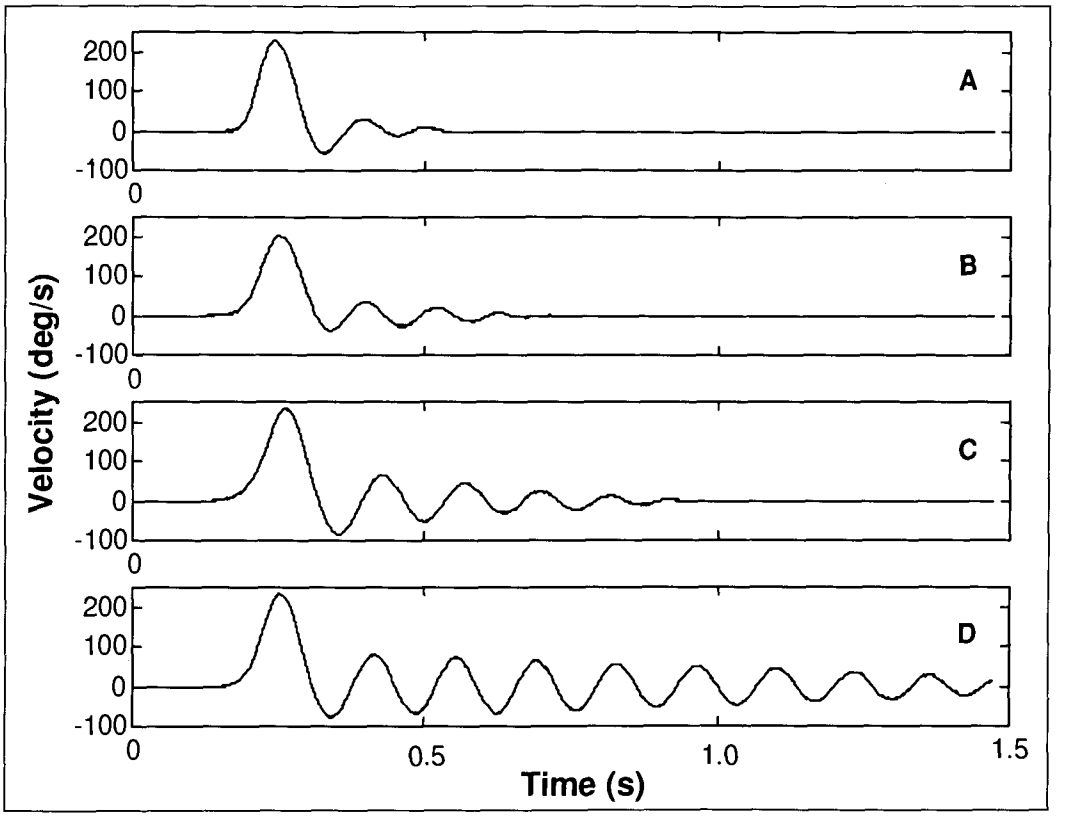

Figure 5. The effect of negative viscosity. Velocity traces are shown for movements to a 20 degree target with a width of 5 degree and a peak target velocity of 220 deg/s with four levels of negative viscosity. Negative viscosity levels were **A:** zero; **B:** 0.0012 Nms/rad; **C:** 0.0014 Nms/rad; and **D:** 0.0018 Nms/rad.

The amount of positive velocity feedback required by the motor to generate oscillations for each condition is presented in Table 4. In general, this command had to be

much higher for the 120 deg/s peak target velocity than for the faster velocities. In contrast, there was no difference in the amount of positive velocity feedback required to generate oscillations between the 220 deg/s and 420 deg/s movement velocities, the target accuracy constraints, or the target amplitudes.

Target Amplitude (deg)	Target Width (deg)	Negative Viscosity Level	Target Peak Velocity (deg/s)					
			120		220		420	
			Minimum	Maximum	Minimum	Maximum	Minimum	Maximum
20	3	1	0.0016	0.0027	0.0008	0.0015		
		2	0.0017	0.0027	0.0010	0.0017		
		3	0.0020	0.0028	0.0012	0.0019		
	5	1	0.0017	0.0024	0.0009	0.0016		
		2	0.0021	0.0025	0.0012	0.0017		
		3	0.0020	0.0026	0.0014	0.0020		
	7	1			0.0007	0.0015		
		2			0.0011	0.0018		
		3			0.0000	0.0021		
40	3	1			0.0011	0.0018		
		2			0.0013	0.0019		
		3			0.0000	0.0021		
	5	1			0.0010	0.0020	0.0008	0.0015
		2			0.0013	0.0021	0.0011	0.0017
		3			0.0015	0.0022	0.0013	0.0020
	7	1			0.0012	0.0020	0.0008	0.0014
		2			0.0010	0.0021	0.0011	0.0016
		3			0.0014	0.0021	0.0012	0.0019

Table 4. Positive velocity feedback required for each level of negative viscosity at each of the experimental conditions.

Additionally, fewer of the 120 deg/s trials met the acceptance criteria because the positive velocity feedback required by the motor to effect oscillations for these particular trials was generally quite high. The high velocity feedback very often led to considerably asymmetric and irregular oscillations that could not be adequately modeled. Consequently, a relatively large proportion of the trials with the 120 deg/s target velocity were excluded from the analysis.

Two series of statistical tests were conducted using the peak velocity and EMG to determine if there was a difference between the control and perturbed (added negative viscosity) trials prior to the application of the negative viscosity load. Trials were grouped according to the ten experimental conditions so that like trials could be matched.

The mean peak velocities across each of these conditions are shown in Figure 6. The control and perturbed trials were compared across all subjects and the three target peak velocities targets using a paired t-test. The peak velocity reached in the control trials was not significantly different than that of the perturbed trials (P=0.060).

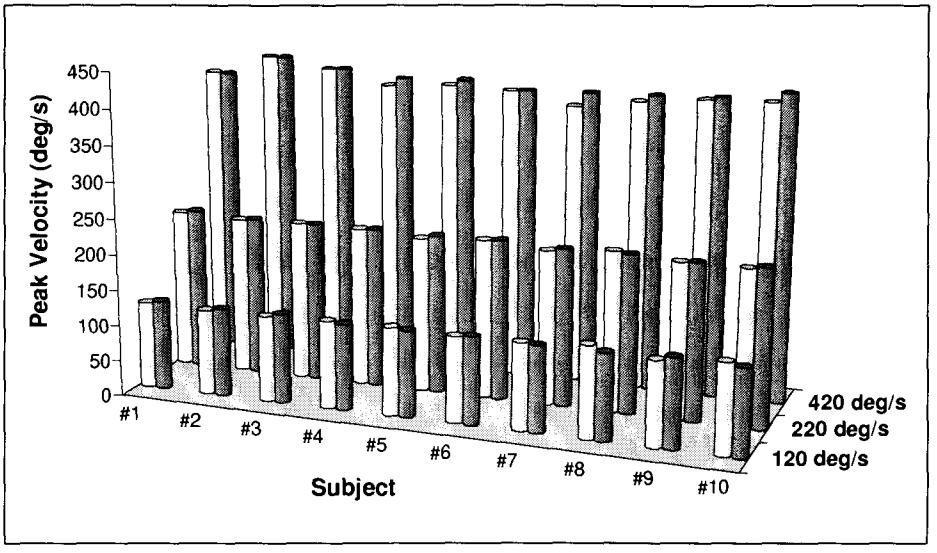

Figure 6. A comparison of the peak velocities reached for controlled (light) and perturbed (dark) trials for each subject and peak velocity target.

The EMG activity during the pre-movement and movement phases was compared for each subject and muscle at each of the three velocity targets using a paired t-test in a similar manner to that of the peak velocity t-test. The EMG activity was not significantly different between the control and perturbed trials during these movement phases (P=0.967).

Oscillation Amplitude

Trials are grouped and presented according to the accuracy constraint (Figure 7), peak velocity target (Figure 8), and target amplitude (Figure 9). The oscillation amplitudes over the five half-cycles of the oscillation are shown. Half-cycles 1, 3, and 5 are wrist extensions whereas oscillations 2 and 4 are flexion movements. In all three groups the amplitude of the second half-cycle was larger than that of the first half-cycle ($P<0.001$). The amplitudes then decreased as expected from the second to the fifth half-cycles. The oscillation amplitudes tended to increase as the target width increased although the difference was not significant ($P=0.075$). The oscillation amplitude increased with velocity ($P<0.001$) and decreased with target amplitude ($P<0.01$).

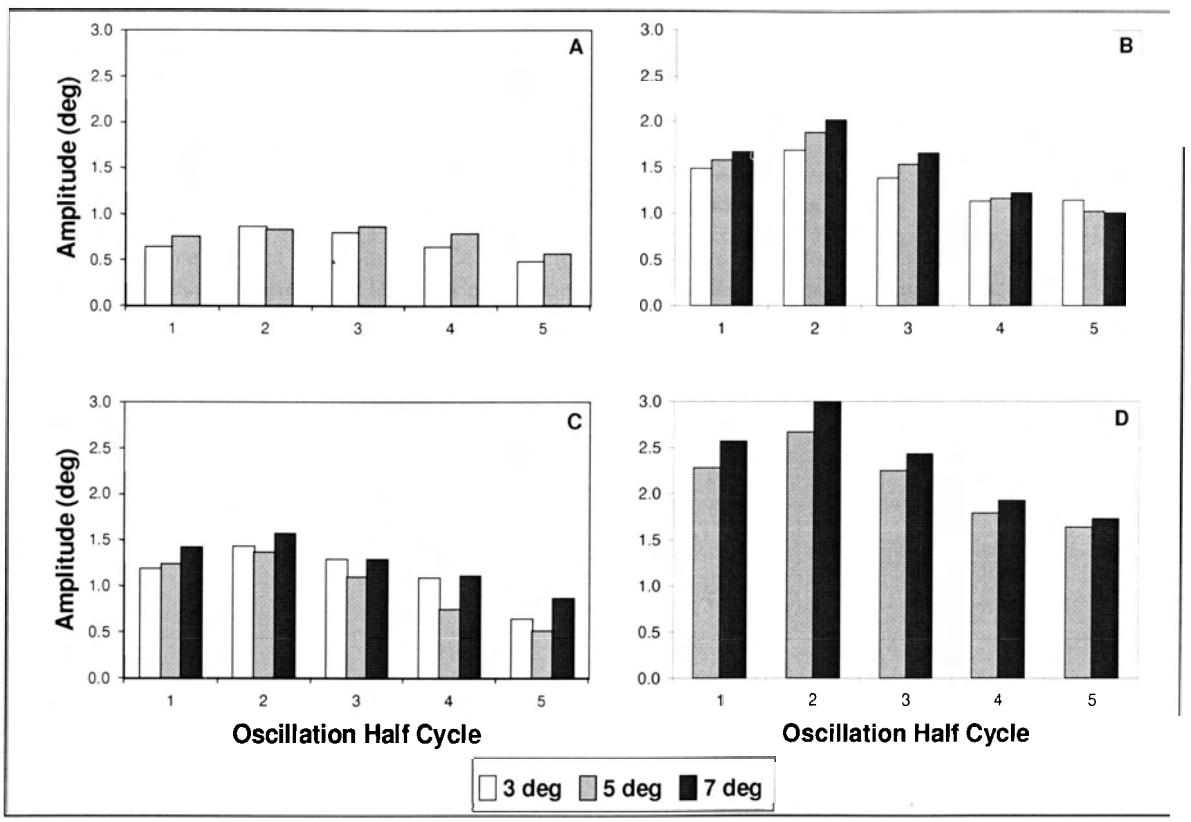

Figure 7. The mean amplitude of oscillation half-cycles for each accuracy target across all subjects and levels of negative viscosity. Trials are designated as follows:

	A	B	C	D
Target Amplitude	20 deg	20 deg	40 deg	40 deg
Velocity Target	120 deg/s	220 deg/s	220 deg/s	420 deg/s

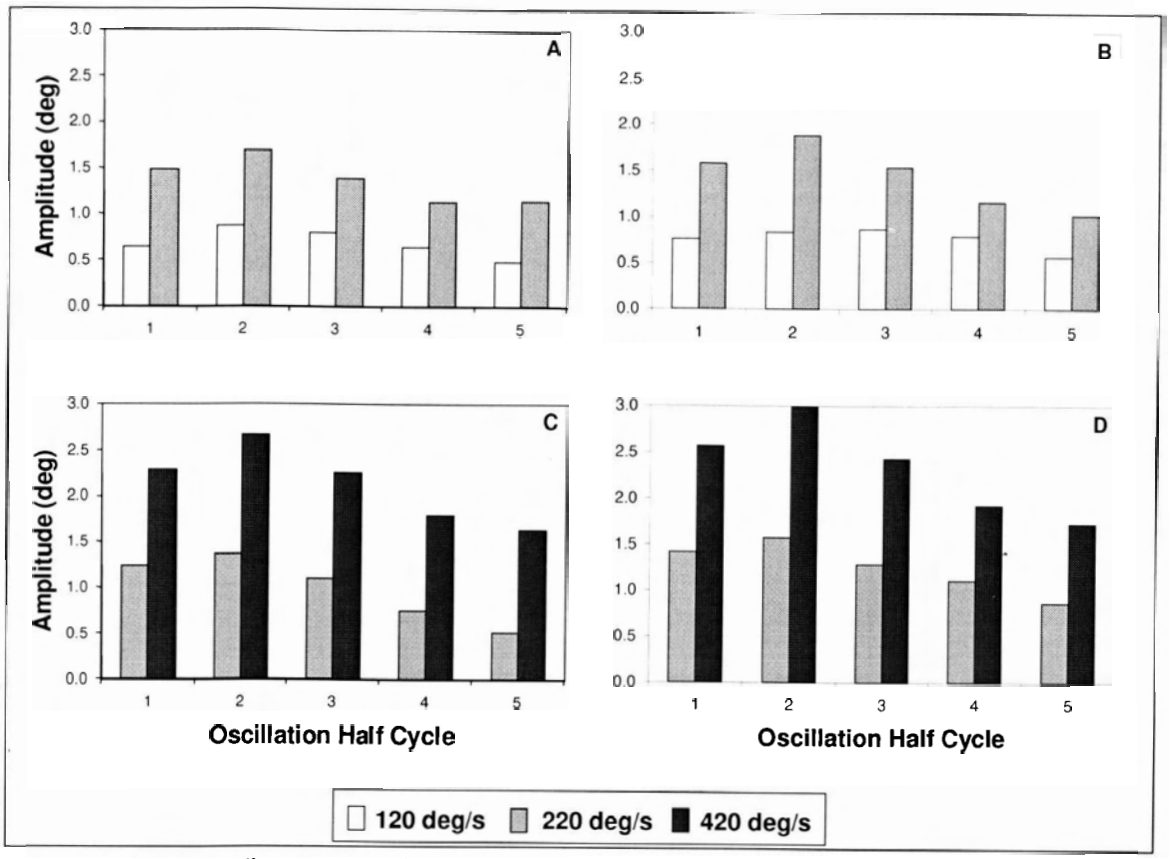

Figure 8. The mean amplitude of oscillation half-cycles at for each velocity target across all subjects and levels of negative viscosity. Trials are designated as follows:

	A	B	C	D
Target Amplitude	20 deg	20 deg	40 deg	40 deg
Target Width	3 deg	5 deg	5 deg	7 deg

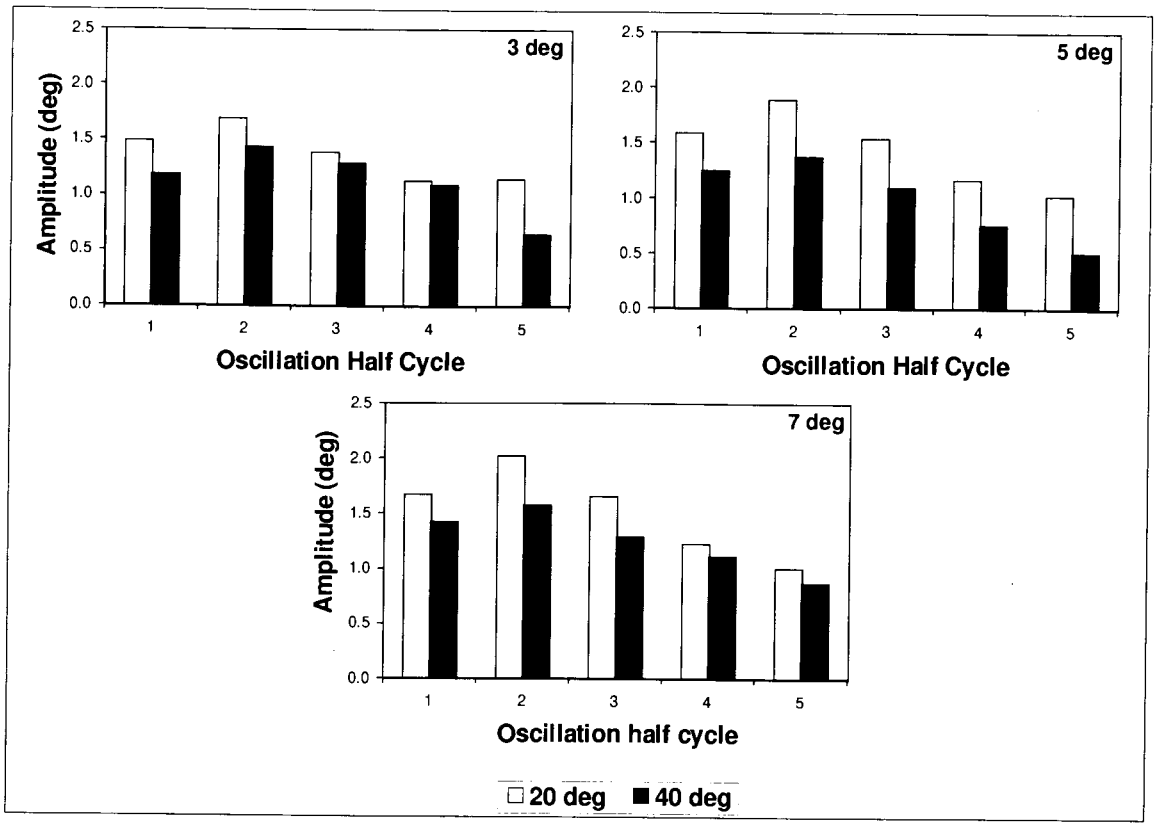

Figure 9. The mean amplitude of oscillation half-cycles at for each target amplitude across all subjects and levels of negative viscosity. Trials were conducted for a target velocity of 220 deg/s to target widths of **A:** 3 deg; **B:** 5 deg and **C:** 7 deg.

Viscoelastic Behavior

There was a considerable amount of trial to trial variability in the parameter estimates for all of the viscoelastic parameters. However the data do show some very clear patterns. Stiffness tended to be high for the first half-cycle of oscillation as the wrist moved in extension. In all cases the wrist stiffness dropped over the second half-cycle of oscillation and then progressively increased for the next three half-cycles. Viscosity tended to be low and was very often negative for the first half-cycle oscillation. It increased until the third or fourth half-cycle and then decreased slightly over the fourth and fifth half-cycles. The damping ratio followed a very similar pattern to that of viscosity. The coefficient of determination (variance accounted for) was very high (on average $R^2 = 0.987$) indicating that the model fit the data extremely well.

Effect of Oscillation Amplitude and Velocity

For all trials, both the oscillation amplitude and velocity decreased over the course of the stabilization phase. In order to determine the effects of these decreases on the viscoelastic parameters, the trials separated into three groups based on amplitude or velocity and compared with stiffness and viscosity respectively (Figures 10 and 11). For each comparison, a one-way ANOVA was conducted to determine if the changes in the viscoelastic parameters were statistically significant. Stiffness increased nonlinearly as the amplitude of the oscillations decreased ($P < 0.001$). In addition, viscosity increased nonlinearly as the velocity of the oscillations decreased ($P < 0.001$).

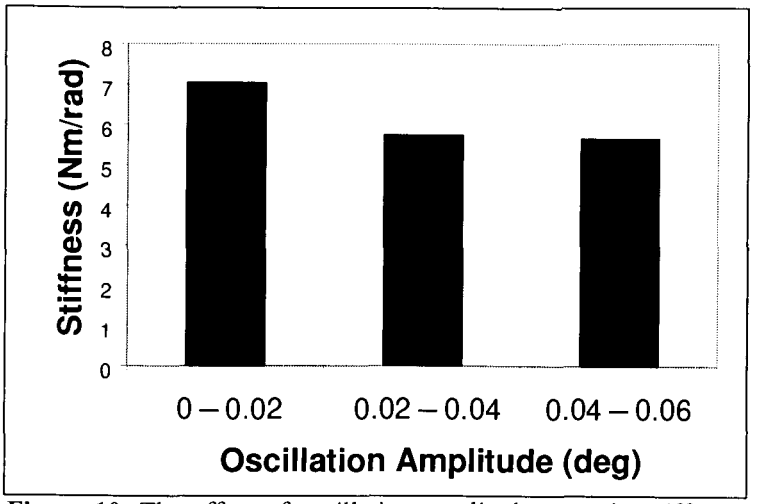

Figure 10. The effect of oscillation amplitude on wrist stiffness. Each bin is an average of all oscillations with that particular amplitude.

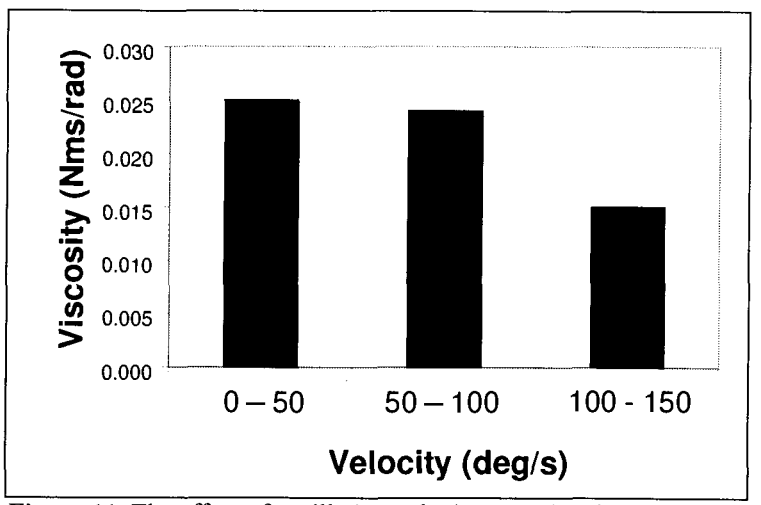

Figure 11. The effect of oscillation velocity on wrist viscosity. Each bin is an average of all oscillations with that particular velocity.

Effect of Accuracy

When the peak velocity was 220 deg/s wrist stiffness increased as the width of the target window was decreased (average $P<0.039$). However, this was not the case for the 120 deg/s or 420 deg/s trials. Statistical comparisons were made to determine the effect of accuracy on the viscoelastic parameters by conducting a series of one-way ANOVA tests. Trials were matched for target amplitude and peak target velocity. Statistically significant differences were observed for seven of the ten tests when the peak velocity was 220 deg/s. (Table 5). The accuracy constraint had no effect on either the wrist viscosity (average $P=0.554$) or the damping ratio (average $P=0.505$). The mean values across all subjects for each comparison are shown in Figures 10-12.

	A	B	C	D
Oscillation	20 deg	20 deg	40 deg	40 deg
Half-Cycle	120 deg/s	220 deg/s	220 deg/s	420 deg/s
		Stiffness		
1.000	0.019	0.116	0.065	0.778
2.000	0.571	0.001	0.013	0.645
3.000	0.913	0.012	0.003	0.253
4.000	0.398	0.018	0.038	0.280
5.000	0.421	0.086	0.030	0.312
		Viscosity		
1.000	0.851	0.977	0.306	0.631
2.000	0.176	0.270	0.363	0.606
3.000	0.739	0.163	0.482	0.475
4.000	0.974	0.976	0.609	0.767
5.000	0.267	0.344	0.324	0.790
		Damping Ratio		
1.000	0.946	0.993	0.178	0.457
2.000	0.140	0.237	0.454	0.828
3.000	0.952	0.031	0.216	0.189
4.000	0.765	0.822	0.373	0.686
5.000	0.239	0.338	0.408	0.841

Table 5. P-value results for one-way ANOVA tests on the effect of an accuracy constraint on wrist stiffness, viscosity, and damping ratio.

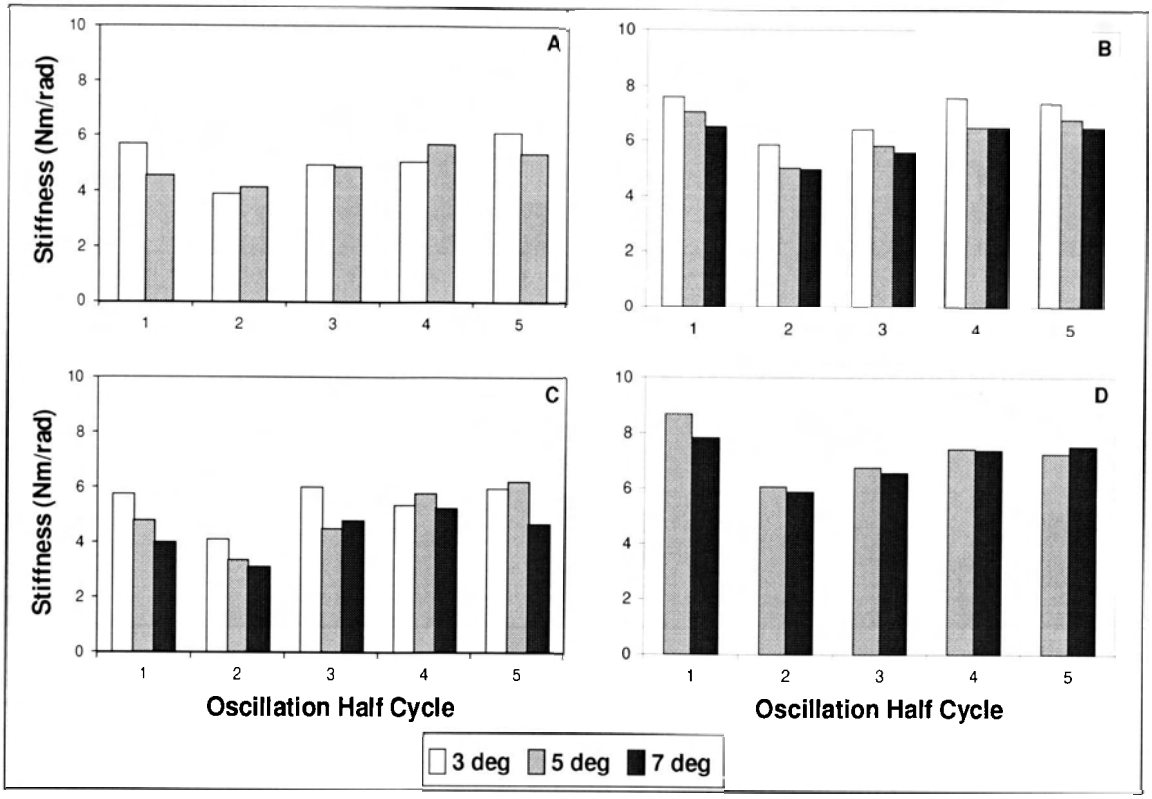

Figure 12. The effect of an accuracy constraint on wrist stiffness. Trials are labelled as designated in Table 5.

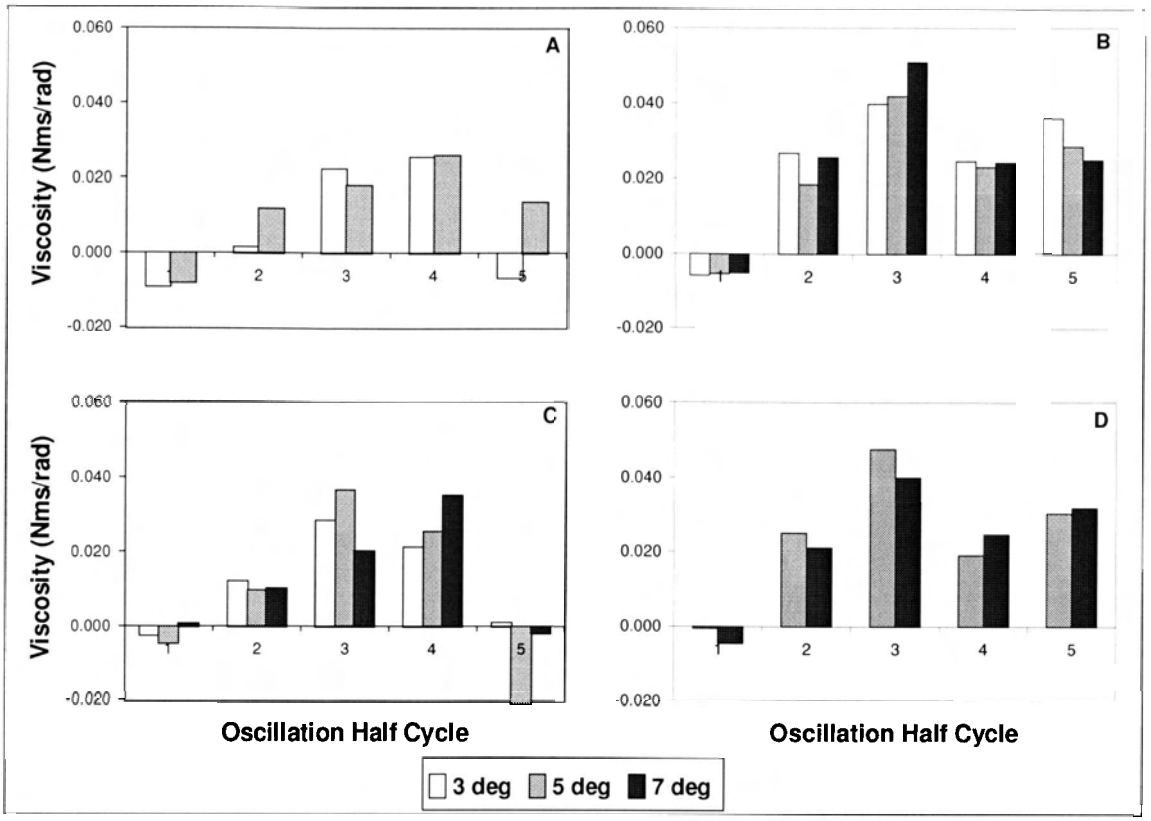

Figure 13. The effect of an accuracy constraint on wrist viscosity. Trials are labelled as designated in Table 5.

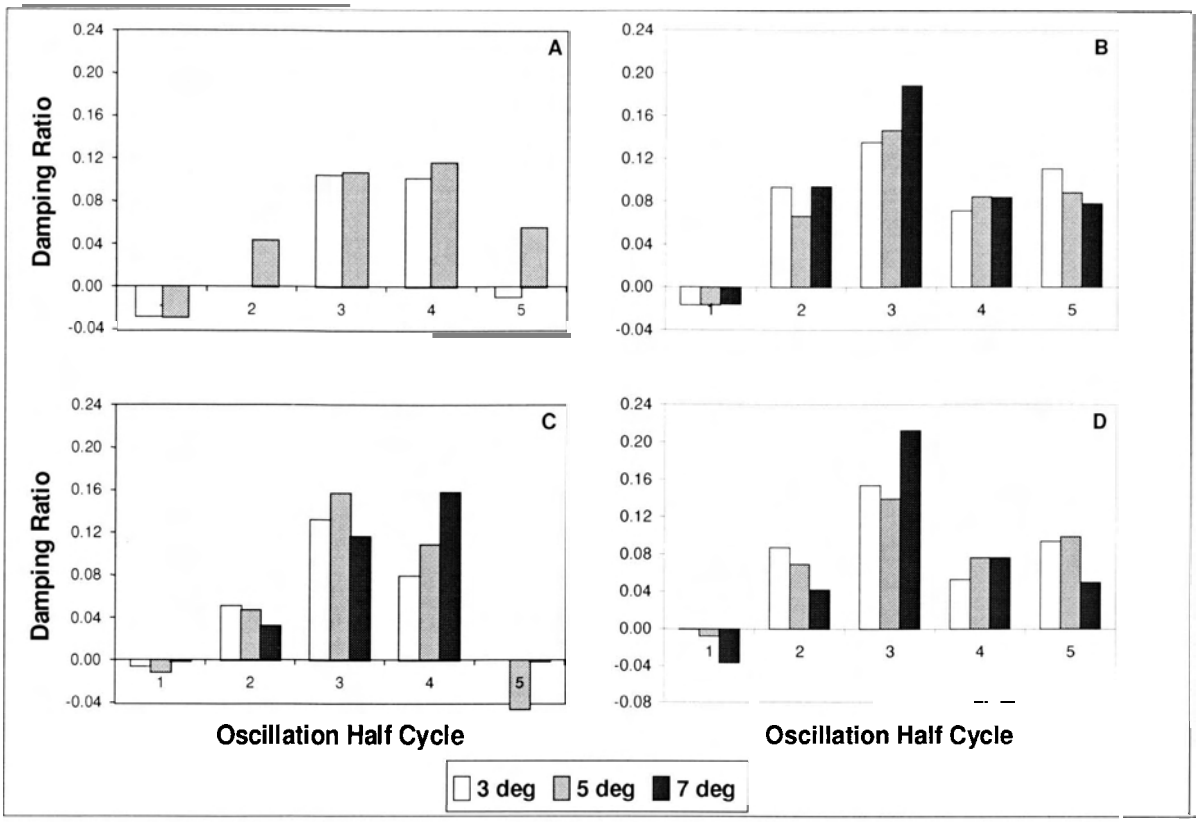

Figure 14. The effect of an accuracy constraint on the damping ratio of the wrist. Trials are labelled as designated in Table 5.

Effect of Velocity

Wrist stiffness increased as the target velocity increased (average $P<0.022$). Statistical comparisons were made with one-way ANOVA tests as described above. Trials were matched for target amplitude and target width. Statistically significant differences were observed for seventeen of the twenty one-way ANOVA tests (Table 6). Although not statistically significant, the wrist viscosity and damping ratio almost always increased for the faster movements (averages $P<0.226$ and $P<0.235$). The mean values across all subjects for each of the parameters are shown in Figures 13-15.

Oscillation Half-Cycle	A 20 deg 3 deg	B 20 deg 5 deg	C 40 deg 5 deg	D 40 deg 7 deg
		Stiffness		
1	0.004	<0.001	<0.001	<0.001
2	<0.001	0.003	<0.001	<0.001
3	0.008	0.044	<0.001	0.002
4	<0.001	0.194	0.004	0.003
5	0.007	0.032	0.074	<0.001
		Viscosity		
1	0.509	0.466	0.295	0.056
2	<0.001	0.301	0.012	0.109
3	0.067	0.009	0.450	0.011
4	0.939	0.780	0.449	0.151
5	<0.001	0.210	<0.001	0.003
		Damping Ratio		
1	0.412	0.257	0.301	0.313
2	<0.001	0.345	0.104	0.144
3	0.322	0.175	0.903	0.285
4	0.430	0.338	0.029	0.002
5	<0.001	0.341	<0.001	0.004

Table 6. P-value results for one-way ANOVA tests on the effect of target peak velocity on wrist stiffness, viscosity, and damping ratio.

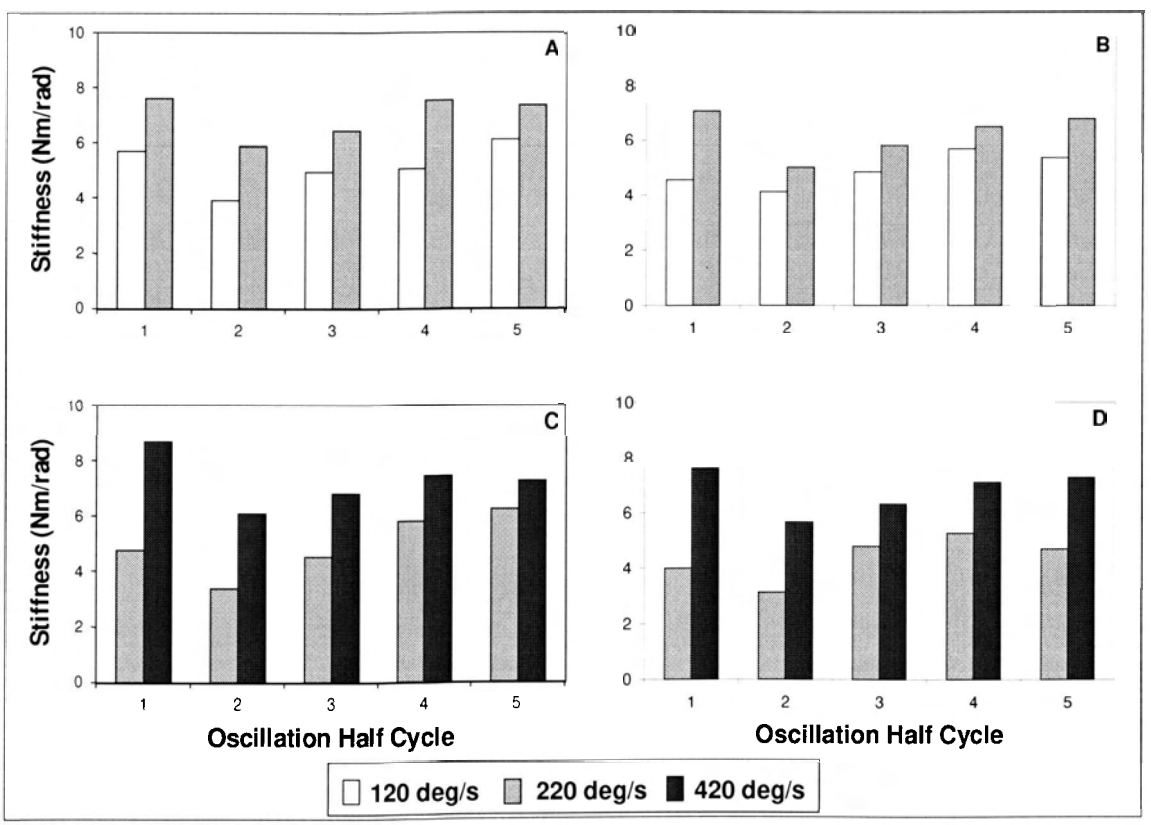

Figure 15. The effect of target velocity on wrist stiffness. Trials are labelled as designated in Table 6.

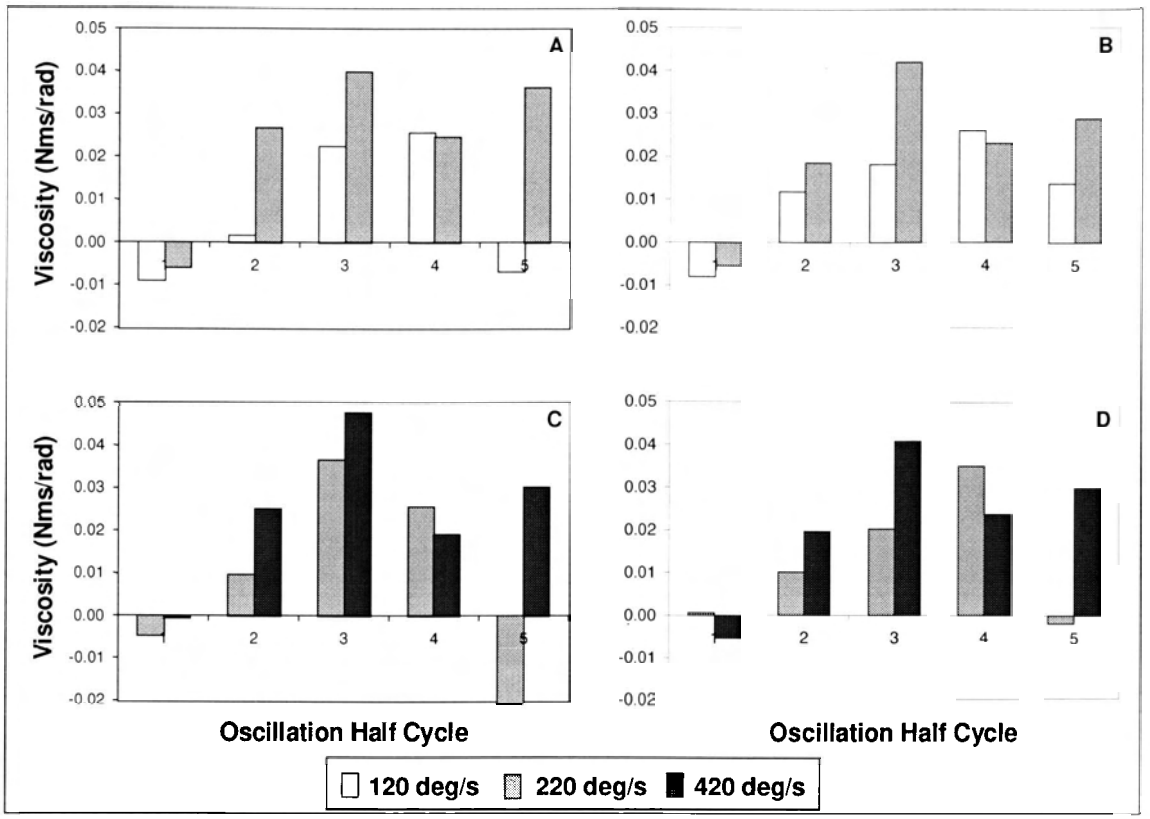

Figure 16. The effect of target velocity on wrist viscosity. Trials are labelled as designated in Table 6.

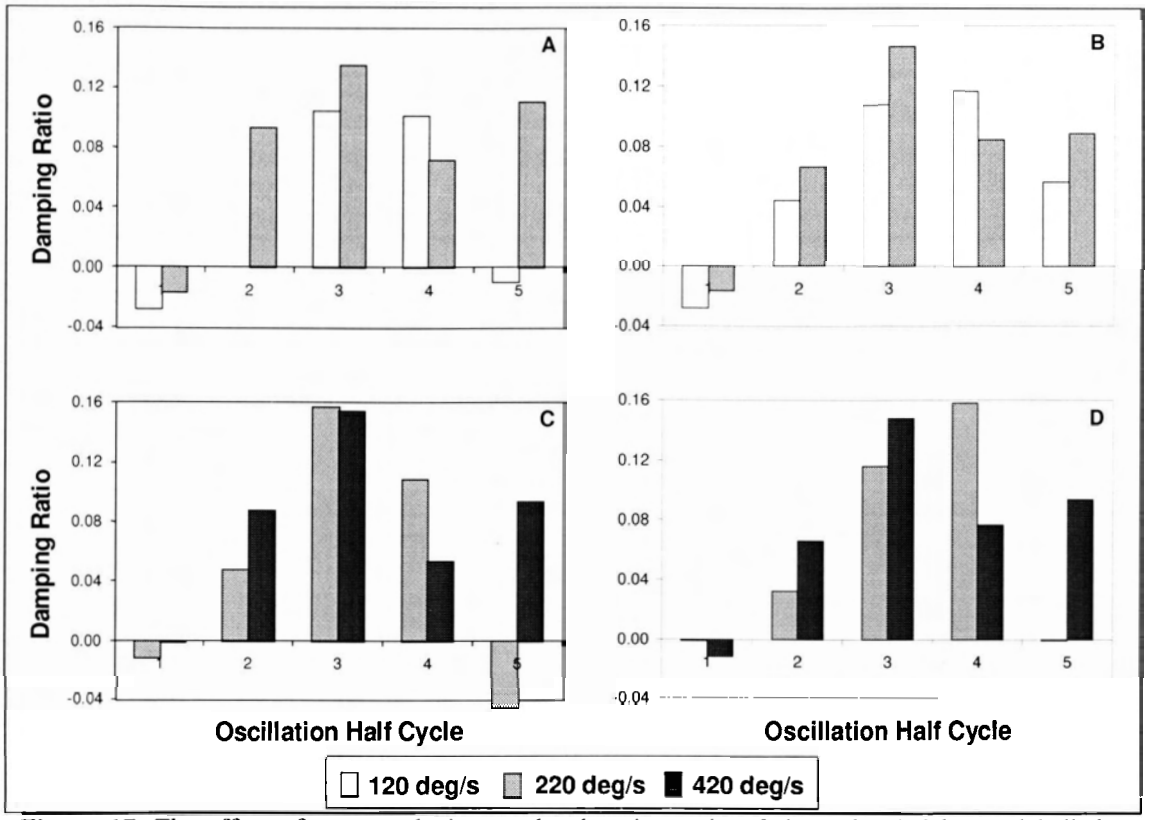

Figure 17. The effect of target velocity on the damping ratio of the wrist. Trials are labelled as designated in Table 6.

Effect of Target Amplitude

Wrist stiffness decreased as the target amplitude was increased (average P=0.030). Statistical comparisons were made with one-way ANOVA tests as described above for a target velocity of 220 deg/s. Statistically significant differences were observed for thirteen of the fifteen one-way ANOVA tests (Table 7). Wrist viscosity and the damping ratio changes were not statistically significant (average P=0.268 and P=0.238). The mean values across all subjects for each of the parameters are shown in Figures 16-18.

Half Cycle	Stiffness			Viscosity			Damping Ratio		
Oscillation	3 deg	5 deg	7 deg	3 deg	5 deg	7 deg	3 deg	5 deg	7 deg
1	0.007	<0.001	<0.001	0.451	0.797	0.041	0.343	0.550	0.130
2	<0.001	<0.001	<0.001	0.035	0.134	0.012	0.116	0.407	0.011
3	0.005	<0.001	0.004	0.288	0.464	<0.001	0.925	0.685	0.017
4	0.003	0.139	0.014	0.782	0.793	0.187	0.835	0.404	0.008
5	0.040	0.242	<0.001	0.004	<0.001	0.033	0.003	<0.001	0.042

Table 7. P-value results for one-way ANOVA tests on the effect of target amplitude on wrist stiffness, viscosity, and damping ratio.

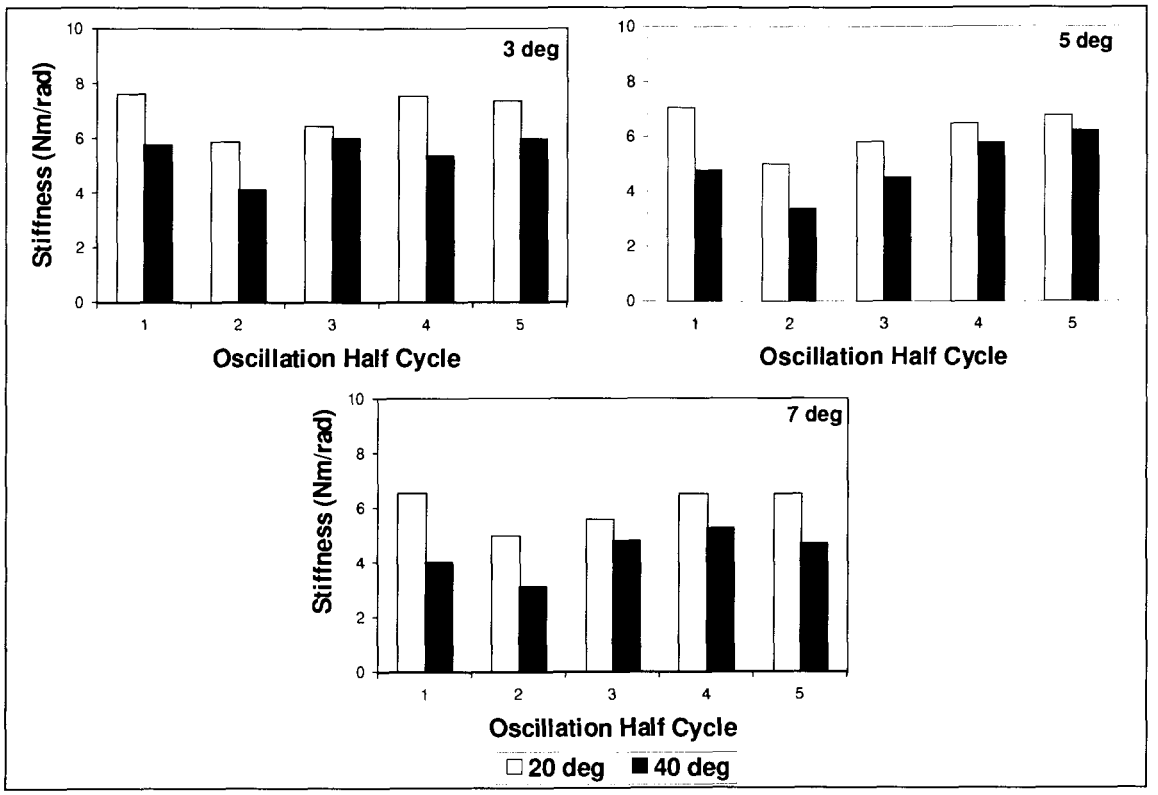

Figure 18. The effect of target amplitude on wrist stiffness.

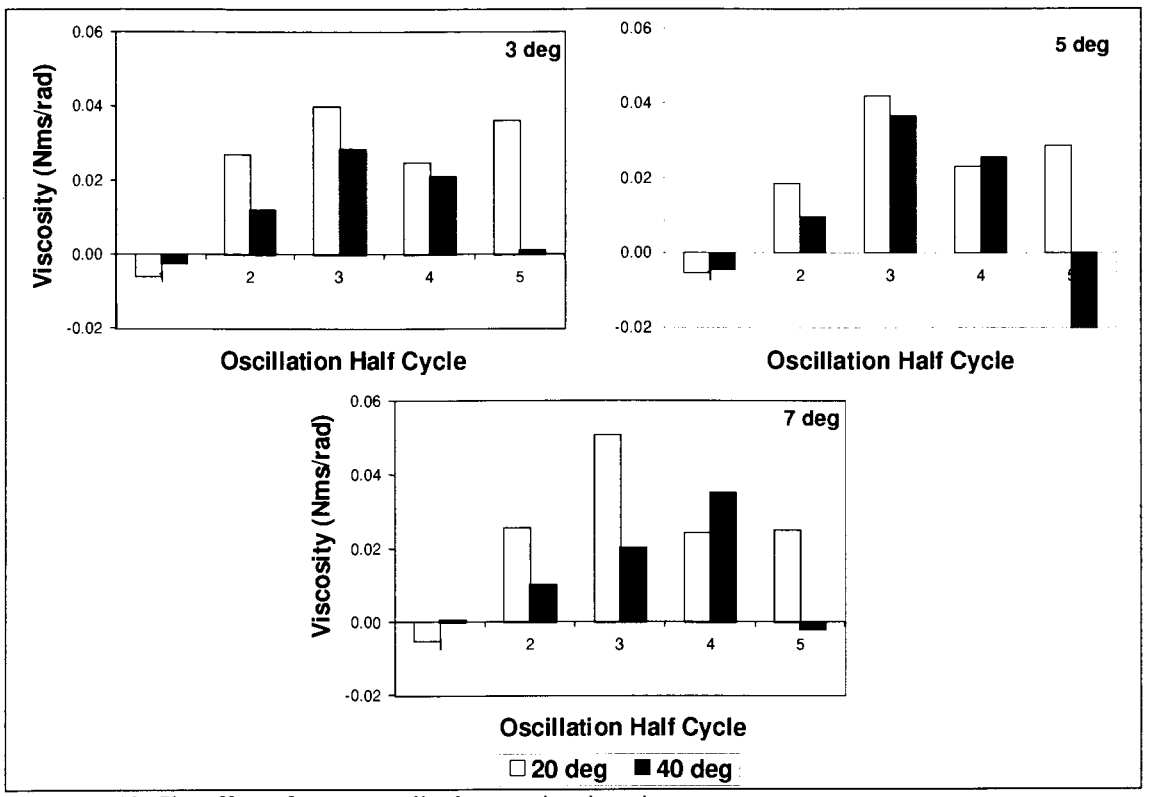

Figure 19. The effect of target amplitude on wrist viscosity.

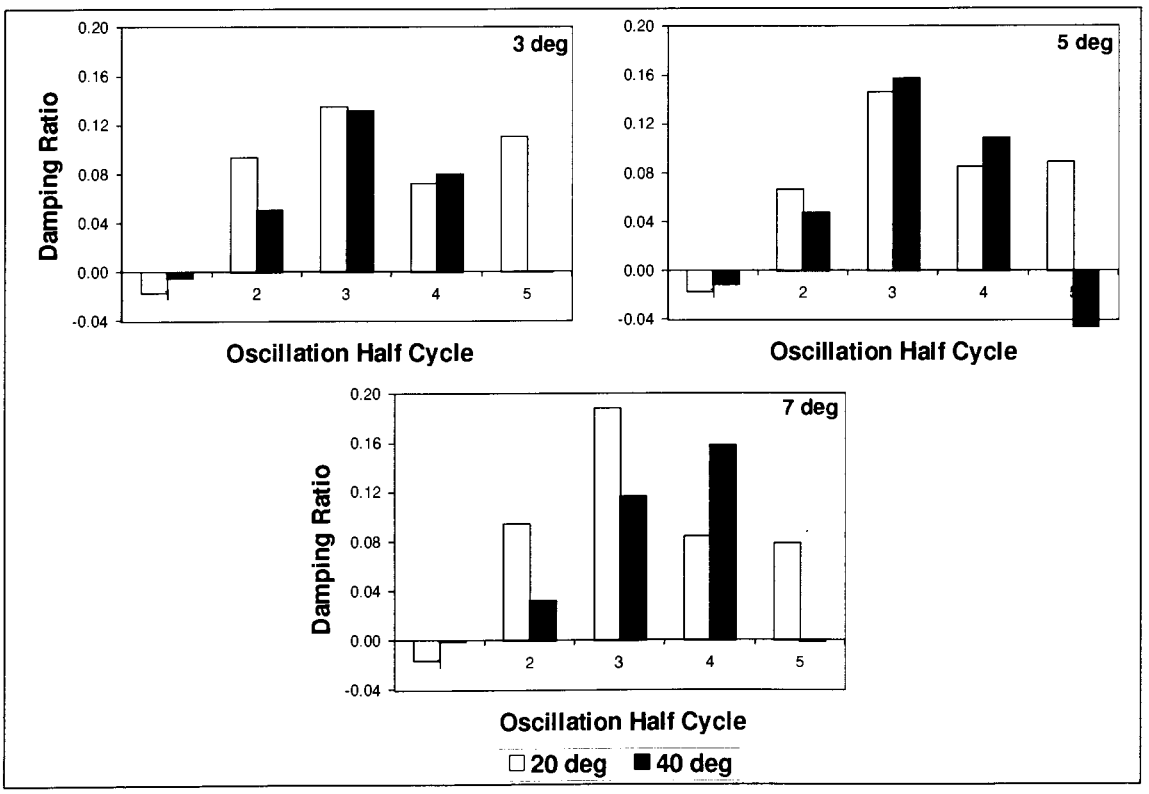

Figure 20. The effect of target amplitude on the wrist damping ratio.

Muscle Activation

In all cases, electromyographic activity (rms EMG) was greater for the ulnar muscles than for the radial muscles during the stabilization and post-movement epochs. FCU EMG was approximately one order of magnitude greater than that of FCR EMG, and ECU EMG was almost twice the magnitude of ECRL EMG. As would be expected, muscle activation was always greater during the stabilization epoch than during the post-movement epoch.

Effect of Accuracy

In general, subjects increased the muscle activity of both flexor and extensor muscles when moving to a smaller target. However, this was not always the case. Statistical comparisons were made for the effect of target width on muscle activity by conducting one-way ANOVA tests on the rms EMG for each subject and muscle during the stabilization and post-movement epochs of the task. Each comparison was matched for target amplitude and target peak velocity. A summary of these statistical tests is presented in Table 8. Each P value in Table 8 is the average value across all subjects. Statistically significant differences were more prevalent during the stabilization epoch than the post-movement epoch. The mean rms EMG results across all subjects for each target width are diagramed in Figure 21.

	Stabilization Phase				Post-Movement			
	A	B	C	D	A	B	C	D
	20 deg	20 deg	40 deg	40 deg	20 deg	20 deg	20 deg	20 deg
Muscle	120 deg/s	220 deg/s	220 deg/s	420 deg/s	120 deg/s	220 deg/s	220 deg/s	420 deg/s
FCR	0.224	<0.001	0.273	0.029	0.1240	0.0020	0.1820	0.0590
FCU	0.246	<0.001	<0.001	0.003	0.0510	0.0720	0.0190	0.0200
ECRL	0.057	<0.001	<0.001	0.014	0.4310	0.0010	<0.001	0.1350
ECU	0.006	<0.001	0.002	0.009	0.0030	0.0010	0.0004	0.0020

Table 8. Statistical significance for the effect of target width on rms EMG activity.

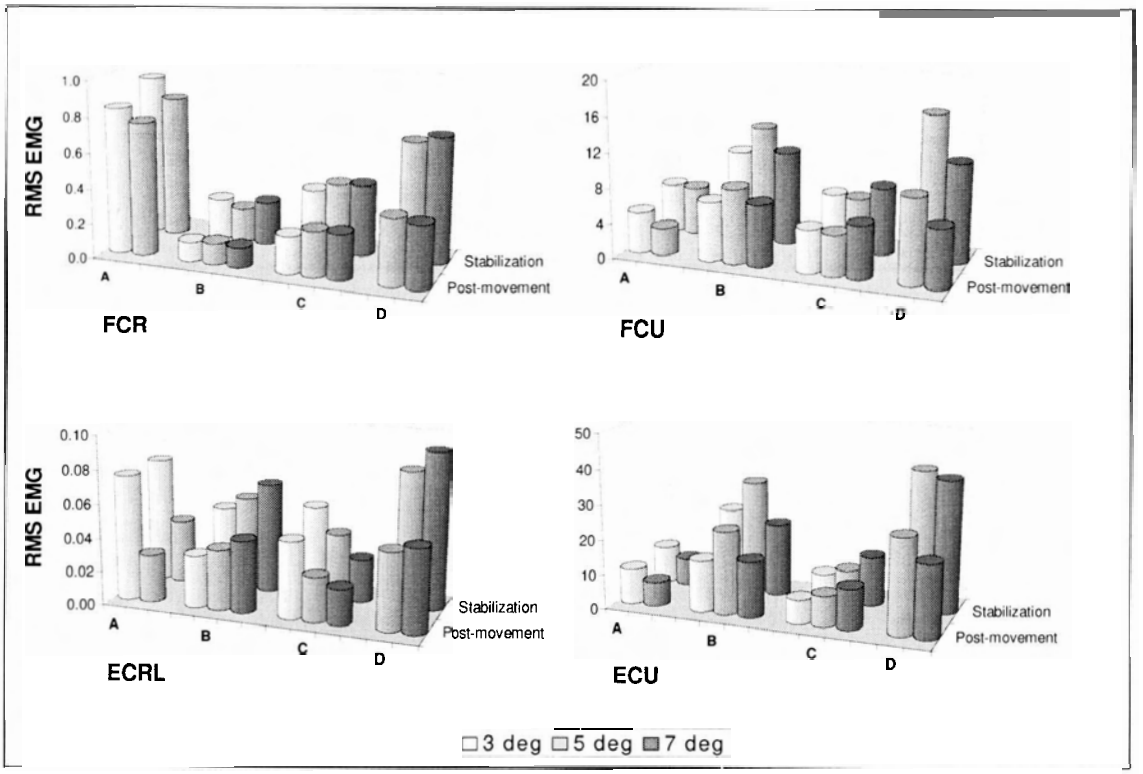

Figure 21. The effect of an accuracy constraint on flexor and extensor rms EMG activity during the stabilization and post-movement phases of the movement. Trials are labelled as designated in Table 8.

Effect of Velocity

Muscle activity increased during both the stabilization and post-movement epochs for faster movements in all but one of the comparisons. Statistical comparisons were made using one-way ANOVA tests as described above. Each comparison was matched for target amplitude and target width. These tests are summarized in Table 9. Each P value in the table is the average value across all subjects More statistically significant differences were observed during the stabilization epoch than the post-movement epoch. The mean rms EMG results across all subjects for each peak target velocity are diagramed in Figure 22.

	Stabilization Phase				Post-Movement			
	A	B	C	D	A	B	C	D
	20 deg	20 deg	40 deg	40 deg	20 deg	20 deg	40 deg	40 deg
Muscle	3 deg	5 deg	5 deg	7 deg	3 deg	5 deg	5 deg	7 deg
FCR	<0.001	0.0710	<0.001	<0.001	0.0460	0.2220	<0.001	<0.001
FCU	<0.001	<0.001	<0.001	<0.001	0.0500	0.0720	0.0160	0.0300
ECRL	<0.001	<0.001	<0.001	<0.001	0.1550	0.0010	<0.001	<0.001
ECU	<0.001	<0.001	<0.001	<0.001	<0.001	0.0030	<0.001	<0.001

Table 9. Statistical significance for the effect of target peak velocity on rms EMG activity.

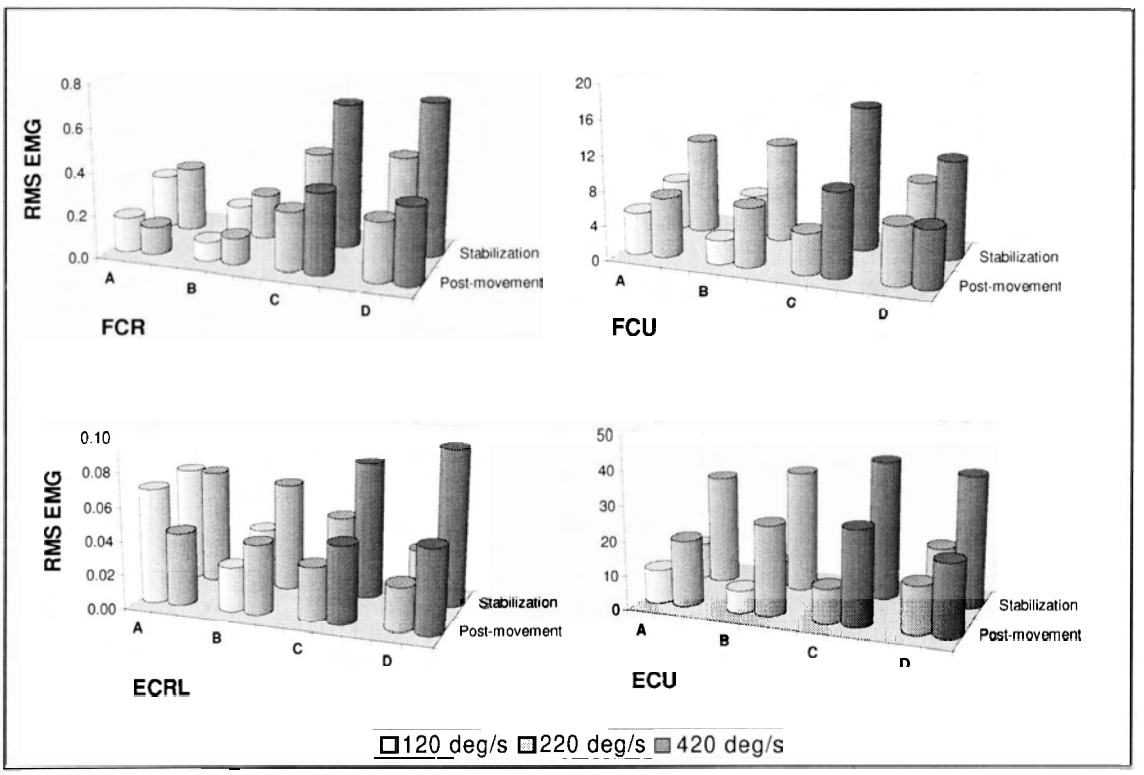

Figure 22. The effect of target velocity on flexor and extensor rms EMG activity during the stabilization and post-movement phases of the movement. Trials are labelled as designated in Table 9.

Effect of Target Amplitude

Electromyographic activity for the ulnar muscles decreased with increasing target amplitude. Activity of the radial muscles, however, did not change uniformly. In particular, FCR activity was greater for the larger amplitude (40 deg) movement to all targets whereas ECRL activity less for the larger amplitude movement when the target width was 5 deg or 7 deg. Statistical comparisons were made with one-way ANOVA tests as described above. For each comparison, trials were matched for target width; in all

cases the target velocity was 220 deg/s. The statistical tests are summarized in Table 10. As in the previous comparisons more statistically significant differences were observed for the stabilization epoch than the post-movement epoch. The mean rms EMG results across all subjects for each target amplitude are diagramed in Figure 23.

	Stabilization Phase			Post-Movement Phase		
Muscle	3 deg	5 deg	7 deg	3 deg	5 deg	7 deg
FCR	0.100	<0.001	<0.001	0.005	<0.001	0.258
FCU	<0.001	<0.001	<0.001	0.190	0.103	0.136
ECRL	<0.001	<0.001	<0.001	0.001	<0.001	<0.001
ECU	<0.001	<0.001	<0.001	0.001	<0.001	<0.001

Table 10. Statistical significance for the effect of target amplitude on rms EMG activity

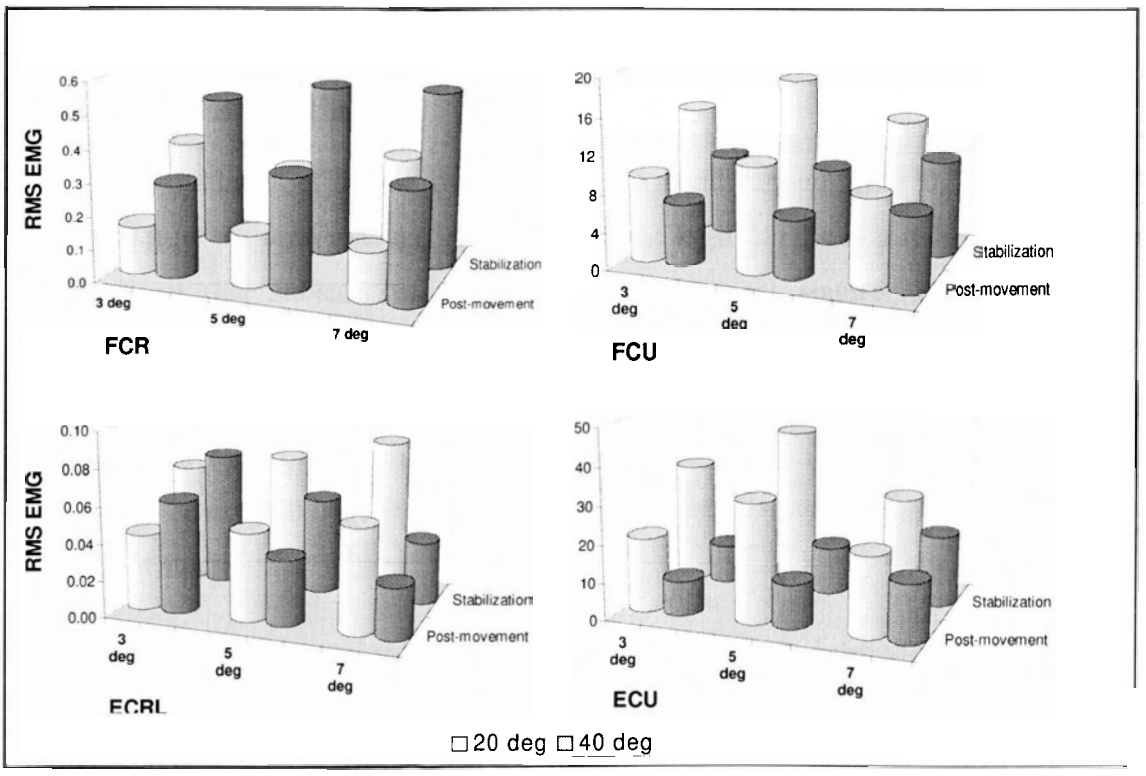

Figure 23. The effect of target amplitude on flexor and extensor rms EMG activity during the stabilization and post-movement phases of the movement. The target peak velocity was 220 deg/s.

DISCUSSION

The purpose of this study was to determine how target velocity, target amplitude, and target width affected the viscoelastic properties of the wrist as the hand stabilized following a voluntary targeted movement. Viscoelastic parameters were estimated using a second order linear model. Second order models have been successfully used to describe joint mechanics in many other studies (Gottlieb and Agarwal, 1978; Zahalak and Heyman, 1979; Kearney and Hunter, 1982; Lacquaniti *et al.*, 1982; Becker and Mote, 1990; Bennett *et al.*, 1992). It was assumed that because the hand was restricted to rotation about the flexion/extension axis of the wrist, that its moment of inertia would be constant. Furthermore, it was assumed that during stabilization at the target position, the viscoelastic properties of the wrist would change slowly enough that they could be presumed were constant over half a cycle of oscillation

Parameter Estimation

Our moment of inertia estimates of the hand ranged from 0.00195 kg-m^2 to 0.00414 kg-m^2 with an average value of 0.00342 kg-m^2. This is a reasonable range of values given that subjects varied a great deal physical stature. The only reported measurements of wrist moment of inertia to which our results can be compared are those of Lakie *et al.* (1980). They used rhythmic torques to determine the resonant frequency, inertia, and stiffness of the hand. They reported a moment of inertia for the hand for three subjects in the range 0.0031-0.0037 kg-m^2. Their results are very similar to the results of the present study.

Across all subjects, trials, and movement conditions, joint stiffness varied from approximately 4-8 Nm/rad. This range is corresponds well with stiffness values in other studies. Lakie *et al.* (1980) reported the resting wrist stiffness to be approximately 1.2 Nm/rad. Gielen and Houk (1984) reported a somewhat higher range of 2-5 Nm/rad for the relaxed wrist using a nonlinear model. De Serres and Milner (1991) found values of static stiffness in the 1.5-4 Nm/rad range. In an experiment with a task very similar to that of the present study, Milner and Cloutier (1997) reported a comparable range for

stiffness (4-6 Nm/rad).

The observation of negative wrist viscosity during the first half-cycle of oscillation of the wrist was unexpected. However, a similar finding has been reported previously. Bennett (1994) reported that the elbow viscosity became negative during movement to a target just as the target was reached. The viscosity then increased as the elbow stabilized at the target and subsequently decreased in a similar manner to that observed in the present experiment. Bennett reported this effect for four out of five subjects. In our study negative wrist viscosity during the first half-cycle of oscillation was observed for all ten subjects.

Negative viscosity could potentially arise from reflex effects. Muscle forces due to reflex feedback originating from changes in muscle length can be phase shifted with respect to the movement. For example, if reflex torque is modulated in phase with the angular velocity, it will oppose the motion and increase the effective viscosity of the joint. If the reflex torque is delayed, it may be phase shifted so that it assists rather than opposes motion, thereby decreasing the effective viscosity of the joint. Destabilizing effects due to reflexes have been reported. However, as will be discussed later, reflexes could not possibly be responsible for any mechanical effects in the first oscillation due to the time delay between the triggering of the reflex and the resulting force output. Alternatively, the effect could be due to an underestimation of the amount of positive velocity feedback. Although this must be considered as a possibility, Bennett (1994) found a similar result using a very different apparatus and data collection system. Oscillation amplitude was observed to increase from the first to second half-cycle. This supports the finding of negative viscosity in the first half-cycle. A possible explanation for why the viscosity might be negative is the timing of the antagonist muscle burst responsible for the deceleration of the hand as the target is approached. The torque from an extensor muscle could have been phased shifted as discussed so that it assisted motion at the time the target was reached rather than resist motion.

Our results indicate that the damping ratio varied from –0.04 to 0.16 and closely followed the viscosity. In contrast, Milner and Cloutier (1997) reported that the damping

ratio remained relatively constant at the higher value of 0.375. It is possible that this discrepancy may be accounted for by the fact that our viscosity estimates are lower than their estimates and that they used a constant moment of inertia of 0.002 kg-m^2 which was lower than our inertia estimates. The combined effect of lower viscosity and higher inertia would decrease the damping ratio.

Several investigators have also reported higher damping ratio estimates for other joints including 0.2-0.4 for the finger abduction/adduction (Becker and Mote, 1990), approximately 0.6 for finger flexion/extension (Hajian and Howe, 1994), and 0.25-0.45 for the ankle (Gottlieb and Agarwal, 1978; Kearney and Hunter, 1990). Estimates in this range have also been reported for the elbow. Bennett estimated the damping ratio of the elbow to be 0.2-0.6 and Lacquaniti *et al.* (1982) estimated it in the 0.08-0.21 range

Effect of Oscillation Amplitude and Velocity

Wrist stiffness increased for smaller amplitudes of oscillation and wrist viscosity increased for slower velocities. The negative correlation with stiffness on amplitude and with viscosity is very likely due to nonlinearilities within the muscles. Similar results have been reported in other studies involving passive and active joints. For example, Kearney and Hunter (1982) reported nonlinear decreases in both stiffness and viscosity of the ankle with smaller displacement amplitudes (0-14 deg) than those used in the present study. MacKay *et al.* (1986a) also reported a decrease in stiffness and viscosity with frequency response and static displacement measurements of the elbow. Milner and Cloutier (1997) reported that joint viscosity was positively correlated with velocity. They suggested that muscle viscosity could have been higher for lower velocity oscillation half-cycles due to increased muscle activation. Because the negative viscosity was presented at the start of the movement rather than at the beginning of the stabilization phase, their subjects' level of muscle activation was much higher than those in the present study.

Effect of an Accuracy Constraint

Wrist stiffness increased during the stabilization phase when the target width was

decreased. This result was expected. If a movement is made to a narrow target compared to a wide target, the flexor and extensor muscles should be activated more strongly in order to constrain the amplitude of the oscillations as the hand is stabilized. The increased cocontraction of flexor and extensor muscles would increase the wrist joint stiffness.

Some subjects reported that greater effort was required when they performed movements to a smaller target. This suggests that they perceived an increase in muscle cocontraction. Despite this perception, the EMG results do not, in all cases, support the supposition that agonist and antagonist muscle cocontraction increased for all muscles as greater accuracy was required. Examining Figure 21, increased muscle activity for the more narrow target window was most notable for FCU and ECU in all but one of the comparisons (C). The results for FCR and ECRL were less consistent. However, the magnitude of the EMG was much smaller for these muscles than that of either FCU or ECU. It can be assumed then, that the major muscles contributing to joint viscoelasticity during stabilization were FCU and ECU.

Varying the target width had no significant measurable effect on either joint viscosity or damping ratio. Although other experiments have shown that viscosity increases with muscle activation under isometric conditions (Hunter and Kearney, 1982; Becker and Mote, 1990) the increase in activation observed in our study was possibly not large enough to produce a significant increase in joint viscosity. It is also notable that the variability in the viscosity and damping ratio measurements was high compared with that of stiffness. The absence of significant differences for viscosity and the damping ratio may have been due to this high variability.

When the target width was decreased, the amplitude of the oscillations during stabilization were also reduced (Figure 7). Despite the fact that this effect was significant ($P<0.001$), in all cases the differences in the magnitude of the oscillations between trials matched for target amplitude and peak velocity target were less than 0.5 degrees. Although viscosity has been reported to increase with decreasing amplitude of oscillation this effect is not as strong as that shown for stiffness (Kearney and Hunter, 1982; MacKay *et al.,* 1986a). In addition, the range of oscillation amplitudes used for both of

these studies were considerably larger than the range observed in the present study. It is possible, therefore, that the differences in oscillation amplitude were not large enough to show a decrease in viscosity.

Effect of Target Peak Velocity

Both stiffness and viscosity increased with the target peak velocity. The increases in viscoelastic impedance were paralleled by an increase in muscle activation for both flexors and extensors. Although the muscles remained tonically active during the post-movement phase, this activity dropped by an average of 32% from the activity levels during stabilization.

When the target peak velocity was increased, the amount of momentum in the system was also increased. This momentum had to be counteracted by muscle activation during the subsequent deceleration and stabilization of the hand. The increased cocontraction of agonist and antagonist muscles needed to effect stabilization resulted in greater stiffness and viscosity of the joint. In addition, when movements are made at different velocities to a target of fixed amplitude, the acceleration profiles will change. A faster movement will require greater acceleration and deceleration and, therefore, increased muscle activation during movement and stabilization.

Increased stiffness has the desirable effect of limiting oscillation amplitude. However, without a concurrent increase in viscosity, the damping constant of the system would not increase. The effect of increased viscosity, therefore, is to increase the damping in the system and allow the oscillations to cease more rapidly.

Gielen and Houk (1984) reported that the resistive force measured at the hand increased with velocity for a set of five different constant velocity ramp stretches. Bennett (1993b) reported that stiffness increased with increased velocity for the elbow. His subjects executed a 60 degree elbow flexion movement with random exposure to a position perturbation at the onset of the movement. He reported that when subjects moved more rapidly, stiffness increased with background torque and that background torque was approximately proportional to velocity squared. There are no experimental

data involving voluntary movements of the wrist to which our viscosity estimates can be directly compared.

Effect of Target Amplitude

Wrist stiffness and viscosity increased when the target amplitude was decreased from 40 degrees to 20 degrees. The muscle activity also increased in both flexor and extensor muscles. However, this was not the case for all muscles. Like the effect noted in response to a change in target width, the confounding EMG results for target amplitude occurred for the two muscles which have the least overall activity and therefore have the least effect on joint stabilization.

For a movement with a given peak velocity target, the peak velocity had to be reached more quickly and the movement stopped more quickly for the 20 degree amplitude target than for the 40 degree amplitude target. The wrist musculature, therefore had to produce larger bursts of activity for the smaller amplitude movements in order to accelerate and decelerate the hand more rapidly. The increased activation of agonist and antagonist muscle during the movement was led to greater cocontraction of agonist and antagonist muscles during the stabilization phase. The increased muscle activity accounts for the observed increase in both joint stiffness and viscosity when the target amplitude was decreased while the target velocity was held constant. The increased stiffness and viscosity combined to rapidly damp out the oscillations about the target.

Given this argument, and the fact that acceleration must also increase as the speed of movement increases, one could argue that the results of this study could be modeled based on the peak acceleration alone. As the peak acceleration increases, the joint stiffness and viscosity should also increase due to increased muscle activation. This method, however, cannot account for changes in muscle activation due to changes in the size of the target. For example, if a series of movements were made at a fixed peak velocity and fixed amplitude, the acceleration profiles for all of these movements would be the same. This method could not, therefore, differentiate the effects of varying the target width as discussed in the previous section.

Effect of Reflexes

The positive velocity feedback applied by the torque motor to effect negative viscosity was turned on when the hand velocity reached zero within the target window. The reduced viscosity of the wrist has the result of stretching the extensor muscles more than they would have been stretched due the momentum of the hand-manipulandum system alone. The effective torque response attributable to the stretch reflex is delayed due to conduction through neural pathways and to the low-pass filter properties of muscle. De Serres (1990) measured the delay due conduction through neural pathways and the duration of excitation-contraction coupling for the wrist flexor and extensor muscles. She reported the shortest period of time for the conduction delay and excitation-contraction to be approximately 22 ms. Furthermore, she reported that the mean time to peak EMG activity was 20 ms. Riek and Bawa (1992) stimulated single motor units for two wrist extensor muscles and recorded the isometric twitch profiles. Their results suggest that time to peak force production is approximately 60 ms. These estimates suggest that from the onset of stretch, the time to peak force production from reflexes is approximately 100 ms. Given these estimates, it is reasonable to assume that the earliest that the reflex generated torque could actually affect the mechanics of the wrist would be 80-100 ms.

The natural frequency for a second order system is

$$f_n = \frac{1}{2\pi}\sqrt{\frac{K}{I}} \qquad [21]$$

The average natural frequency for each subject during the first half-cycle of oscillation across all trials was calculated from the corresponding stiffness values for the estimated inertia. The natural frequency estimates for all subjects ranged from 6.1 to 8.7 Hz with an average value of 7.15 Hz. The corresponding cycle duration for this frequency is 140 ms. Comparing the duration for the first half-cycle of oscillation (70 ms) with the delay for reflex effects, any effect on the estimated mechanical parameters of the wrist due to the addition of positive velocity feedback could not occur until the second half-cycle of oscillation.

The results of the present study indicate that a statistically significant increase occurred in the oscillation amplitude from the first to the second half-cycle (Figures 7-9). In a similar experiment Milner and Cloutier (1997) reported that the oscillation amplitude decreased progressively over all half-cycles. The most significant difference between the two studies is in the timing of the negative viscosity. In their experiment negative viscosity was presented at the onset of the movement. In the present experiment, the positive velocity feedback was added when the hand reached the target. Therefore, it is probable that the increased oscillation amplitude for the second half-cycle was due to the reflex torque generated by this perturbation.

The increased amplitude over the second half-cycle of oscillation and the simultaneous decrease in stiffness (Figures 10, 13, and 16) suggests that the contribution of the reflex torque was sufficiently out of phase with the torque generated from intrinsic muscle mechanics that it contributed to destabilizing the system. Reflex generated destabilizing effects have been reported by other researchers for the wrist (Milner and Cloutier, 1993) and elbow (Bennett, 1994). In both of these experiments the reflexes were induced by perturbations.

CONCLUSION

The purpose of this thesis was to gain a better understanding of the mechanical properties of the human wrist during the stabilization phase of voluntary targeted movements. The results of this study suggests that although both joint stiffness and viscosity are important for stabilization of the limb following a movement, stiffness may be the more important property controlled by the neuromuscular system. In addition, our findings suggest that joint stiffness can be estimated more reliably than viscosity and that viscosity is modulated over a much smaller range. The damping of the wrist appears to be small due to the limited amount of viscosity. The damping is, however, always sufficient to rapidly reduce oscillations about the target.

Between the neural and muscular systems, the intrinsic properties of the muscular system may have the most important influence on motor control during the stabilization phase of a task involving voluntary movement. We have shown that intrinsic muscle mechanics are well suited to stabilization following motion. We have also provided further evidence reflex feedback may not be an effective means of stability control, especially if the limb is operating in a rapidly changing environment where it may be exposed to unexpected disturbances.

This work has applications in a wide range of areas. Knowledge of how the human neuromuscular system controls the mechanical impedance of a joint during stabilization enables more accurate modeling of the dynamics of human movement. An understanding of neural control and joint dynamics may clarify the mechanical changes underlying neuromuscular disease. This may lead to an improved ability to quantitatively describe and objectively analyze neuromuscular disorders. Stability is critical in many aspects of rehabilitation engineering. The design of prostheses and orthoses can be improved by better simulating the dynamics of the joints they replace. Similar improvements may be observed with functional electrical stimulation systems.

This work also has potential applications in the design of telerobotic systems in which the human operator controls a robot at some distance by moving a manipulandum

such as a joystick. If the dynamics of the human system can be predicted, the closed loop stability of the overall human-robot system can be better controlled under all operating conditions. Human motor control is far more sophisticated and is better able to adapt to its environment than are current robotic control systems. Therefore, it may be advantageous to control robotic systems in a similar manner to that of the human neuromuscular system.

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