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Effects of Altering Mechanical Properties on Manual Tracking Performance in Humans with Cerebellar Incoordination

by

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled, "Effects of Altering Mechanical Properties on Manual Tracking Performance in Humans with Cerebellar Incoordination" submitted by Betty-Lynn Morrice in partial fulfillment of the requirements for the degree of Master of Science.

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ABSTRACT

The mechanisms producing incoordination in patients with cerebellar lesions are not well understood and attempts to improve function in these patients with current rehabilitation techniques are disappointing. This study examines whether tracking performance of cerebellar patients can be improved by modifying the mehanical properties of a manipulandum used for manual tracking of a visual target.

Five patients with unilateral cerebellar lesions and six age-matched control subjects were presented with a visual display of a target moving horizontally across a screen in an unpredictable pattern at velocities of 0-30 degrees/sec. They were required to track the target by flexing and extending the wrist. After a learning period to familiarize the subjects with the apparatus, the viscosity, stiffness, and inertia of the apparatus were systematically altered. Viscous resistance and stiffness were provided by feeding back the velocity and position, respectively, to the amplifier that powered a torque motor. Inertia was altered by adding weights to the manipulandum. The cumulative difference between the signals representing wrist position and target position provided a measure of error for each 20 second trial. The amount of EMG activity in wrist flexors and extensors was also measured, and the degree of coactivation estimated. Signals representing target and wrist position were differentiated to examine movement velocity.

When patients performed the tracking task with the affected arm, error scores were significantly higher than in the clinically unaffected arm or the control subjects. Increased viscosity improved

tracking accuracy in the affected arms of patients, but not in their clinically unaffected arms nor in control subjects. Increased stiffness produced no consistent change in positional error scores. Addition of inertia either made no change in accuracy, or increased the error. Tracking velocities in the affected arms had higher velocity error scores, more high amplitude velocity peaks, and more zero crossings than the clinically unaffected arms or the control subjects.

In a separate set of experiments, the control subjects' and cerebellar patients' wrists were given a torque perturbation, resulting in an oscillation of wrist position. From these oscillations, the damping factor at the wrist was calculated. The cerebellar patients had less damping in their forearms than the control subjects.

These results suggest that excessively high movement velocities contribute to tracking error in patients with cerebellar lesions, and that applying viscous resistance can dampen these movements to improve tracking accuracy.

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INTRODUCTION

1. RATIONALE

Cerebellar incoordination is a common cause of disability in neurological conditions such as multiple sclerosis, cerebellar neoplasms, and stroke. The mechanisms of this incoordination are not well understood. Patients with cerebellar lesions exhibit motor control disorders which can range in severity from incoordination that is barely perceptible to disabilities which render the individual totally dependent in all activities of daily living. Cerebellar incoordination cannot be ameliorated with current pharmacological treatments. In rehabilitation settings there have been some mixed successes reported in decreasing the ataxia of cerebellar patients by using weighted wrist cuffs during functional activities. This empirical treatment requires further research regarding the mechanisms producing the incoordination, determining effective methods to diminish the ataxia, and quantification to demonstrate the effect of these methods on performance of functional activities.

Modification of the mechanical load acting on a human limb has been shown to affect the amplitude and frequency of tremor and of the position oscillations produced when the limb is perturbed (for example, Joyce and Rack 1974, Stiles 1983). These mechanical loads include viscosity, stiffness, and inertia.

Stiffness and viscosity are properties of a limb that are a result of intrinsic mechanical properties and reflex response. Stiffness can be calculated by dividing the change in force by the change in length produced by an external perturbation. Viscosity is a

friction-like property which can be dependent on velocity. The viscous properties largely determine the damping of the system. The amount of damping, in turn, determines the amplitude of the oscillations, the number of oscillations, and how long it takes to return to a steady state.

Various studies have examined the effects of altering mechanical loads on normal humans and monkeys, and on those subjects with cerebellar dysfunction; but many questions remain unanswered. In order for these results to be of therapeutic benefit to patients with cerebellar dysfunction, we must determine which specific type of mechanical loading condition will diminish the amplitude and frequency of the tremor most effectively. To reflect the functional effect of these modifications of viscosity, stiffness, and inertia, patients should be subjected to these conditions while performing a functional task. Results from further investigation into the mechanisms producing this incoordination may enhance our development of appropriate therapeutic measures.

To address these questions, this research project involved patients with unilateral cerebellar lesions and control subjects performing manual tracking of a visual target. The viscosity, stiffness, and inertia of the apparatus were altered systematically. Wrist position, velocity, and EMG were recorded and quantified during the tracking movements.

2. OBJECTIVES

The general objectives of this research project were: a) to investigate possible therapeutic benefits of altering mechanical loading of the upper extremity of patients with cerebellar lesions while performing a manual tracking task, and b) to enhance understanding of mechanisms producing dysfunction in these patients.

The specific aims of this project were to address the following questions:

a) What are the effects of altering viscosity, stiffness, and inertia on manual tracking performance of normal subjects?

b) What are the effects of altering viscosity, stiffness, and inertia on manual tracking performance of patients with cerebellar lesions?

c) Do patients with cerebellar lesions have errors in the programming of velocity of movement? Do excessively high movement velocities contribute to the tracking inaccuracies which occur as a result of cerebellar dysfunction?

d) Do normal subjects use coactivation of agonist and antagonist muscles to increase limb stiffness and thereby improve accuracy of manual tracking? Does failure to produce an appropriate level of coactivation contribute to tracking inaccuracies which result from cerebellar dysfunction?

e) Are inappropriate corrections for visually detected errors responsible for some of the excessively high velocity tracking movements in cerebellar patients? Does removal of visual feedback result in smoother movement trajectories?

f) Is reduced mechanical damping in the limb a factor which contributes to errors in tracking performance in patients with cerebellar lesions?

3. LITERATURE REVIEW

3.1 CEREBELLAR FUNCTION AND DYSFUNCTION

The cerebellum is involved in control of posture and movement (Brooks, Thach 1981, Eccles et al. 1966). Because of its inputs from the cerebral cortex, the brain stem, and the periphery, the cerebellum may serve a comparator function; comparing the intended act with the actual motor response (Ghez, Fahn 1985). The sequencing and timing of muscle activation during movement is controlled in part by the cerebellum, and this can be appropriately modified during changing conditions (Brooks, Thach 1981). Although the exact mechanism is unknown, the cerebellum is thought to play a role in motor learning, and in associative learning (Gellman, Miles 1985).

The motor task examined in this study is manual tracking of a visual target. It has been suggested by Miall et al. (1987) that the cerebellum is responsible for adjusting the amplitude and velocity of tracking movements by combining measures of the current error with estimates of target speed and direction. "The cerebellum has an inhibitory role in tuning movements during visuo-motor tasks and optimal tuning using feedforward measurements of target motion cannot be made without it" (Miall et al. 1987).

Damage to the cerebellum can result from a variety of causes, such as: degenerative disease, injury, infarct or hemorrhage, toxic conditions, neoplasm, or surgery. Because of its role in motor control, a lesion in the cerebellum can cause movement disorders.

Muscles that normally function together to produce a smooth, coordinated movement lose their synergies. This decomposition of movement results in error of force, velocity, range, and timing, as described by Gordon Holmes (1917, 1939). Hypotonia is often a feature in these patients (Gilman et al. 1981). With cerebellar lesions reaction time is increased (Beppu et al. 1984, Holmes 1917, Meyer-Lohmann 1977, Rothwell 1987), and there can be errors in prompt and uniform decceleration, resulting in dysmetria (Holmes 1939). Another feature of cerebellar dysfunction can be intention tremor, which is absent at rest and present during voluntary movement or maintained postures (Beppu et al. 1984, Gilman et al. 1981, Holmes 1939). A patient with cerebellar incoordination could exhibit both dysmetria and intention tremor, or only one of these signs in isolation.

3.2 MECHANISMS OF DYSMETRIA

The mechanisms producing dysmetria and intention tremor are not well understood; however several theories regarding their origin exist. Possible mechanisms responsible for dysmetria will be discussed first. Holmes (1939) attributed dysmetria to a slowness in the relaxation of the agonist and decreased tone of the antagonist. More recently, EMG analysis of patients performing an elbow flexion task suggested that a prolongation of the initial agonist and/or antagonist components may be responsible for dysmetria (Hallet et al. 1975). Beppu et al. (1984) concluded that "dysmetria may be attributed not only to inadequate consolidation of motor programs in timing and size but also to a defect in the comparator function". A study by Brooks (1984) states that dysmetria is caused by inappropriate selection of a normal motor program which has been

generated by other brain structures. Flament and Hore (1986) concluded that it is unlikely that dysmetria results from inappropriate selection or triggering of a normal motor program; in examining kinematic and EMG characteristics of dentate-cooled monkeys they showed that hypermetric arm movements without oscillations have different properties than those of normal arm movements of similar velocity and amplitude.

3.3 MECHANISMS OF TREMOR

Tremor exists in both the normal physiological state and in pathological conditions. There are various types of pathological tremors; intention tremor is the type which may be present in the patients studied in this project. Many of the studies which have examined the mechanisms of tremor have investigated both physiological and pathological tremor in humans and monkeys. As a result of these investigations three main models have arisen to explain the origin of tremor (Riley, Rosen 1987, Rosen, Adelstein 1984, Stein, Lee 1981), which will be outlined below.

The first hypothesis is that the limb acts as a spring-mass system and the underdamped mechanical and stretch reflex characteristics of this second order system result in increased oscillations when it is perturbed (Elble 1984, Flament et al. 1984, Joyce, Rack 1974, Lakie et al. 1986, Rietz, Stiles 1974, Stiles, Randall 1967, Stiles 1983). This perturbation could be caused by asynchronous contractions of the motor units of the active muscles (Rietz, Stiles 1974, Riley, Rosen 1987), ballistocardiogram, or other body movements (Joyce, Rack 1974, Lakie et al. 1986). The frequency of this tremor is determined by the passive mechanics of the limb

(Joyce, Rack 1974, Lakie et al. 1986), and does not require descending nervous system input (Joyce, Rack 1974, Lakie et al. 1986, Rietz, Stiles 1974, Stiles 1983).

A second theory is that tremor is driven by a central nervous system oscillator which generates abnormal descending signals (Lance 1975, Rosen, Adelstein 1984, Homberg et al. 1987). The tremor in some Parkinsonian patients cannot be reset by external inputs; therefore, this particular tremor may be of central origin (Stein, Lee 1981). Riley and Rosen (1987) suggest that a CNS oscillator plays a dominant role in tremor production. Elble et al. (1987) have recently observed that advanced essential tremor could be disrupted by perturbations, but there was no consistent time-locked resetting of the oscillations. Elble suggests that the central oscillator responsible for essential tremor is coupled to the stretch reflex; this reflex does not produce the essential tremor rhythm, but may govern the amplitude of the oscillations.

The third model of tremor is that it is due to instability in the servo-controlled neuromuscular reflex loop (Lippold 1970, Murphy 1975, Nichols et al. 1978, Stein, Oguztorelli 1976, Vilis, Hore 1977 and 1980). Some supportive evidence for this model was provided by Lippold (1970) and Vilis and Hore (1977) who showed that physiological tremor could be resynchronized by perturbation applied to the limb. In a premamillary cat, also, the oscillations can be reset by stimulation of the muscle nerve, suggesting that the oscillations are not produced by a central generator (Nichols, Stein, Bawa 1978). These authors propose that physiological tremor is maintained by reflexes reinforcing a tendency for oscillation in a mass-spring system. Murphy (1975) concluded that velocity related information in

the multiple feedback loops to the CNS allows phase leads in feedback to compensate for time delays; the cerebellum is a major part of one of these feedback channels, therefore a cerebellar lesion results in an unstable position control system which will oscillate when it is perturbed. Vilis and Hore (1980) found that in dentate-cooled monkeys there was a loss of predictive instruction from the cerebellum to the motor cortex, and in turn to the antagonist muscle, to terminate the return of the limb after being perturbed. Therefore, the servomechanism through the motor cortex is driven only by feedback from the periphery, resulting in delay. This allows an overshoot of the corrective return movement, and a series of alternating stretch reflexes results.

A recent study by Hore and Flament (1986) which studied monkeys with cooled lateral cerebellar nuclei performing a step-tracking task concluded that "discontinuities and tremor in movements during cerebellar dysfunction result from the same mechanism: alternation between disordered stretch reflexes and disordered servo-assistance mechanisms, both partly involving transcortical pathways".

3.4 MODIFICATION OF MECHANICAL PROPERTIES

A limb possesses the properties of inertia, stiffness, and viscosity. Stiffness can be attributed to a deformation of cross-bridges without significant reformation (Rack, Westbury 1974, Morgan 1977, Lacquaniti 1982). Besides forming short-range elasticity, cross bridges can act over a longer distance; the actin-myosin bonds can break and reform to act as a viscosity (Stein, Lee 1981). Stiffness at a joint is increased whenever there is an increase in the activation of agonists or antagonists, and especially with

coactivation (Hasan 1986, Ghez 1983, Lacquaniti et al. 1982, Akazawa et al. 1983). Akazawa et al. (1983) have shown that stiffness increases linearly with coactivation. Joint stiffness has been reported to play an important role in decreasing the effort of a task (Hasan 1986), and to allow better control of the terminal phase of a movement (Ghez 1983). When a subject actively resists an applied perturbation both the stiffness and viscosity of the forearm are increased (Lacquaniti et al. 1982).

The forearm behaves as a second order linear system in response to a perturbation (Lacquaniti et al. 1982, Stiles 1983, Elble et al. 1987, Stein, Lee 1981). In a second order linear model the frequency of oscillations is proportional to the square root of the stiffness, and inversely proportional to the square root of the mass (Elble et al. 1987). Damping of this system increases as the amount of viscosity is increased. Formulae used to describe a second order linear system are found in Figure 4 of the Methods section.

Some studies which have examined the effects of modifying mechanical loads on physiological and pathological tremor in humans and monkeys have revealed the following results. Holmes (1939) observed that if cerebellar patients carried a heavy bar the alternating movements of pronation and supination were improved in rate, rhythm, and range. The addition of mass to attenuate tremor was subsequently examined by Morgan (1975a, 1975b), who showed that the use of weighted wrist cuffs provided a significant decrease in intention tremor in humans. This study also observed that there is an optimal amount of weight for each subject, and that more weight is needed with more severe tremor. Hewer, et al. (1972) also used weighted wrist cuffs to attenuate tremor amplitude; the tremor was

diminished in 58% of the subjects, but was of therapeutic use in only 36% of the subjects. In 1965, Chase applied weight to the finger of a human with intention tremor and found that there was a marked attenuation of tremor when the weight stretched the finger flexor muscles; however, there was increased amplitude of tremor when the weight loaded the extensors. This particular finding has not been reproduced in subsequent research. More recent studies have confirmed that both the frequency of tremor and the frequency of oscillations resulting from a perturbation decrease with mass loading of the limb or apparatus (Elble 1984 and 1987, Flament, Hore 1986, Joyce, Rack 1974, Hore, Flament 1986, Lakie et al. 1986, Stein, Oguztorelli 1975, Stiles 1980 and 1983, Vilis, Hore 1980). It has also been shown that the addition of mass decreases the amplitude of oscillations (Hore, Flament 1986, Vilis, Hore 1977). Elble et al. (1987) observed that mass loading produced a dramatic suppression of essential tremor rhythm; however, Homberg et al. (1987) found that in patients with essential tremor and with Parkinsonian patients, the peak frequency remained stable irrespective of changes in load. These same authors (Homberg et al. 1987) did report a gradual decrease in physiological tremor peak frequency with increased load, as would be expected with a passive mass-spring system. Elble et al. (1984) concluded that since the frequency is altered by inertial loading, tremor must be generated both by feedback loops which consist of peripheral reflex paths and by limb mechanics. Vilis and Hore (1980) found that with these changes in mechanical load the related EMG bursts altered appropriately, therefore the EMG bursts must be reflexively evoked and not from central oscillations. Although the frequency of the oscillation

declines as the mass is increased, the rate at which the oscillation dies away diminishes (Stein, Oguztorelli 1975).

The second mechanical property which can be altered is stiffness. As would be predicted if the limb acts as a second order linear system, actively stiffening the limb increases its resonant frequency (Lakie et al. 1984). External application of stiffness through the use of springs has also been reported to increase the frequency of oscillations (Elble et al. 1987, Joyce, Rack 1974, Vilis, Hore 1977).

The third type of mechanical loading which can be applied is viscosity. Increased viscous resistance has attenuated pathological tremor in humans and monkeys (Adelstein and Rosen 1981, Elble et al. 1984, Riley, Rosen 1987, Vilis, Hore 1977). Vilis and Hore (1977) found that although viscosity decreased the amplitude of oscillations it had little effect on the frequency. A study by Riley and Rosen (1987) showed that the viscous damping modifications must be adapted to the individual subject; no one system modification led to improvement in tracking of all the tremor disabled subjects.

3.5 MANUAL TRACKING ERROR

Normal humans and monkeys track an unpredictable target discontinuously with periodic positional corrections (Miall et al. 1985 and 1987, Rothwell, 1987). Following cerebellar inactivation, monkeys track a target with less accuracy (Miall et al. 1987). Tracking accuracy is also decreased in humans with cerebellar ataxia (Beppu et al. 1984 and 1987).

A corrective response for a visually presented error requires about 200 ms. (Beppu et al. 1987, Keele, Posner 1968, Miall et al. 1985, Poulton 1981). The frequency of positional corrections during

tracking in normal humans is about 1 to 3 per second. The time required for a corrective response is longer in cerebellar patients who have a slowed reaction time, but stepwise pursuit with 1 to 3 Hertz frequency would still be an appropriate range (Beppu et al. 1987).

3.6 MANUAL TRACKING VELOCITY

In normal monkeys performing pursuit tracking the velocity recording had many small peaks which were regular in height; with cooling of the interpositus nucleus the velocity signal became irregular with large individual peaks, but there was little change in the average frequency of movements (Miall et al. 1987). Miall et al. (1987) suggest that the cerebellum plays a crucial role in using velocity information in the feedforward control of movements. Tracking in patients with cerebellar lesions deteriorates because they are unable to calculate the required amplitude of each movement from the speed of the target. The movements become inappropriately large and fast, and the patients must rely on estimates of the positional error to control each movement (Miall et al. 1987). Miall et al. (1986) found that the average amplitude of the intermittent movements increased with target frequency, and that the main influence on the size of each movement is not the magnitude of the current visual error, but the speed at which the target is moving at the start of each movement. They concluded that target velocity is the single most important control signal in tracking, followed by positional error.

Beppu et al. (1984) state that in normal humans performing ramp-tracking the handle movement had a fairly constant velocity, and the size of initial peak velocity (IPV) increased with target velocity and

seemed related to a catch-up reaction. In the patients with cerebellar ataxia that they studied the size of the IPV varied in each trial and the linearity between the IPV and the initial error decreased markedly. During tracking these patients were unable to maintain a constant target velocity but repeated jerky movements at a frequency between 1 and 3 Hertz (Beppu et al. 1984, Miall et al. 1985). Beppu et al. (1987) concluded that the velocity undulation is not caused by a primary defect in the motor system or sensory system, but that "the pursuit pattern with velocity undulation in patients with cerebellar ataxia represents a repetition of voluntary correction responses for errors which are the inevitable outcome of the immediately preceding dysmetric catch-up responses, certainly not a form of involuntary tremor".

3.7 EMG DURING PURSUIT TRACKING

The cerebellum may play an important role in switching between the coactivation and reciprocal activation of antagonist muscles (Smith 1981). The generation of appropriate agonist and antagonist muscle activity to control the magnitude and timing of arm movements requires a normally functioning cerebellum (Flament, Hore 1986). Smith suggests that the Purkinje cells inhibit antagonist muscles during reciprocal activation but are themselves inhibited during antagonist coactivation.

During ramp-tracking at the elbow in normal humans a reciprocal EMG pattern was well maintained throughout the tracking task; in cerebellar patients there was coactivation of the antagonistic muscles in about half of the cases (Beppu et al. 1984). These authors also found that during tracking by patients with cerebellar ataxia the

amount of EMG discharge during tracking showed a marked fluctuation, reflecting the irregular tracking movements. They found no clear correlation between the antagonist coactivation and the clinical severity or type of cerebellar ataxia.

In physiological tremor there is no rhythmic bursting or motor unit synchronisation in EMG recordings (Homberg et al. 1987, Hagbarth and Young 1979). In essential and Parkinsonian tremors the EMG shows strong motor unit synchronisation, and burst repetition rates at a constant frequency (Homberg et al. 1987).

Schieber and Thach (1985a) trained normal monkeys to perform ramp tracking at the wrist. They determined that position dependent factors (length tension characteristics and passive elastic loads) were responsible for modulation of EMG. When they varied target velocities it was found that the average EMG amplitude did not vary appreciably with the wrist velocity. Applying maintained torque loads changed the level of the EMG activity, but did not change the basic pattern.

Hasan (1986) observed that movements with increased inertial loading resulted in increased coactivation.

3.8 PHASE LAG DURING TRACKING

During tracking movements, reaction time cannot be measured accurately because if the limb is moving when the stimulus appears the start of the response cannot be specified exactly (Poulton 1981). If the time and direction of the stimulus are partly predictable, the person may respond without waiting for the stimulus. At the onset of tracking the person's response marker lags a reaction time behind, but soon the cursor catches up with the target (Poulton 1981). After a

few oscillations with the cursor at first behind and perhaps just ahead of the target, it usually moves in line with the target. When the subject maintains alignment he is predicting that the target will continue to move in a straight line at a constant speed (Poulton 1981). With a pursuit display the subject can predict the future movement of the track and reduce his time lag (Poulton 1974). It is possible to measure the lag or lead error in time at each position (Poulton 1981).

Day et al. (1984) have shown that patients with Parkinson's disease are able to use a predictive motor strategy when manually tracking a visual target. Their mean tracking lag was often below visual reaction time. These results are discrepant with the finding of Flowers (1978) who showed that Parkinsonian patients are unable to anticipate target movement to use a predictive strategy when tracking.

In a sinusoidal tracking task, monkeys with and without cerebellar lesions, were able to produce the lowest positional error scores with time lags of 50-100ms (Miall et al. 1987).

3.9 EFFECTS OF LEARNING ON TRACKING BEHAVIOR

Poulton (1981) has made the following observations regarding learning during pursuit tracking by a normal human subject. Over time a person can learn the average frequency and average amplitude of a tracking task. From the current visual position and the rate of the track marker, the subject can predict its position and rate over 0.25 seconds, which enables him to keep the cursor moving in line with the target. If the subject is able to see the nature of the error it provides him with knowledge of the results of the accuracy of his prediction, thus enabling him to learn to predict better. Practice

improves the performance by producing changes in the long-term memory. With practice, the motor program in long-term memory comes to fit the track better, so corrections are not needed as often.

The experimental design and data analysis must take into account the factors of transfer bias and central tendency bias. A transfer bias is created by the order in which stimuli are presented; strategies adopted in the first condition may affect performance in subsequent conditions (Poulton 1981). The only way to avoid transfer bias is to use separate groups of people for each experimental condition. Transfer bias can be diminished by changing the presentation order of the various conditions over the group of subjects. A central tendency bias is created when subjects perform a number of conditions in turn; the strategies they develop are influenced by all conditions performed previously. This bias favors the experimental conditions that are most like the other conditions used in the experiment (Poulton 1981).

Schieber and Thach (1985b) observed that normal monkeys coactivated their wrist flexors and extensors while learning a tracking task for up to 6 months. Once the monkeys were trained they did not contract the antagonist.

3.10 CONTRIBUTION OF VISUAL FEEDBACK TO TRACKING ERROR

Humans can follow sinusoidal targets accurately and make little use of visual feedback (Miall et al. 1986). However, during tracking tasks of an unpredictable pattern, limb control is dominated by visual information (Poulton 1974 and 1981, Beppu et al. 1987).

Beppu et al. (1987) found that the undulation present during pursuit movement in cerebellar ataxia is a result of repeated

visually-guided error correction responses. They erased visual cues during pursuit in patients with cerebellar degeneration and there was a resultant signficant reduction of the rapid irregularities in the velocity recording.

Miall et al. (1985) delayed the visual feedback during tracking by normal humans and monkeys. The introduction of these visual delays decreased the number of corrections of positional error and increased their amplitude.

Contrary to these results, Flament et al. (1984) observed that the removal of visual feedback did not change the characteristics of tremor in monkeys with cerebellar lesions. They concluded that cerebellar intention tremor is driven by stretch-evoked peripheral feedback and not by voluntary correction based on vision.

4. SUMMARY OF METHODOLOGY

Patients with unilateral cerebellar lesions and control subjects were presented with a target moving horizontally across a screen in an unpredictable pattern at velocities of 0 - 30 degrees per second. Subjects were required to track the target by flexing and extending the wrist. After a learning period to familiarize the subjects with the apparatus, the viscosity, stiffness, and inertia of the apparatus were systematically altered. Position, velocity and EMG were recorded; from these were derived positional error and velocity error scores, and a coactivation index.

Some of the subjects also performed three self-paced sinusoidal movements without visual feedback to examine the contribution of visual input to discontinuities of movement.

In a separate experiment, subjects were instructed to not intervene as their wrist was perturbed by a torque motor. From the resultant oscillations of position the damping factor at the wrist was calculated.

5. PRINCIPAL RESULTS

Increased viscosity improved tracking accuracy in the affected arms of patients, but not in their clinically unaffected arms nor in control subjects. Increased stiffness produced no consistent change in error scores. Addition of inertia either made no change in accuracy, or increased the error.

Tracking velocities in the affected arms of the patients had more high amplitude velocity peaks and more zero crossings than the clinically unaffected arms or control subjects, and no significant change in the number of reversals.

There was no significant difference between subject groups in the amount of coactivation that was used during tracking; however, further investigation of this question is required.

In both the normal subjects and the cerebellar patients there were fewer irregularities of position during a self-paced sinusoidal movement than during tracking of an unpredictable target.

There was less damping in the wrists of cerebellar patients than in the normal control subjects.

METHODS

1. SUBJECT SELECTION

Subjects with cerebellar lesions were referred from the Neurology and Neurosurgery services at the Foothills and Calgary General Hospitals. The ideal patient for this project was one with a unilateral lesion involving the cerebellum only, thus having one clinically unaffected arm to be used for comparison against the arm with incoordination. Five patients meeting these criteria were available for testing over a one year period. Recordings were also obtained from twelve other patients with motor control disorders, including patients with spinocerebellar degeneration, Multiple Sclerosis and Parkinson's Disease.

Normal subjects were age matched (plus or minus three years) to the subjects with unilateral cerebellar dysfunction.

The protocol was approved by the Ethics Committee of the Faculty of Medicine and informed consent was obtained from all subjects after an explanation of the purpose of the experiment and the procedures involved. (appendix A)

2. SUBJECT EXAMINATION

A clinical assessment was performed on each patient. This included evaluation of muscle tone (as determined by resistance to passive movements), tendon reflexes, strength of major muscle groups of the upper extremities, and coordination (finger-to-nose test, rapid supination/pronation of forearm, and hand and finger tapping in 10 seconds).

The medical charts and radiographic findings for each patient were reviewed.

3. TRACKING TASK

Subjects were seated with one forearm secured in neutral pronation/supination on a horizontal base with the hand enclosed in a sansplint hand mold with fingers extended to prevent gripping. The wrist joint was positioned exactly above the axis of movement of the manipulandum, which was attached by a vertical shaft to the torque motor (Aeroflex wide angle brushless DC torque motor with a maximum output of 1.8 Newton-meters). The arm tested was the affected limb in the case of unilateral deficit (using the other limb as a control), or the more clinically affected limb if the deficit was bilateral. The right or left arms of the normal subjects were selected randomly. The starting position of the wrist for the tracking task was in a neutral position; midway between flexion and extension. (Figure 1)

Surface EMG electrodes (0.9 cm. diameter gold disc electrodes) were placed 2.5 centimeters apart over the muscle bellies of flexor carpi radialis and extensor carpi radialis.

Target movements and data collection were controlled by a Digital PDP 11-40 computer. The subject was presented with a visual display on a CRT screen at eye level and approximately one meter from the subject's eyes. The visual display consisted of a target depicted by an open 2 cm square, and a cursor which appeared as a solid 1.25 cm square within the open square of the target.

The cursor was controlled by the output of a potentiometer monitoring the position of the wrist joint. The subjects were

required to track the target by performing carefully controlled flexion and extension movements at the wrist.

The target moved horizontally across the screen in an unpredictable pattern for twenty seconds at handle velocities of 0 to 30 degrees/sec. Thirty-five degrees of handle movement corresponded to 12.6 cm excursion on the CRT screen. The tracking pattern was created by the sum of two sinusoidal waves to create a maximum range for the task of 35 degrees of handle movement. One wave contributed the equivalent of 19 degrees of handle movement, while the other wave contributed 16 degrees. The first wave had a frequency of 0.15 Hertz, and the second was 0.345 Hertz.

4. ALTERATION OF MECHANICAL LOADING

To determine whether accuracy of performance in a tracking task can be improved by altering the mechanical properties of the limb and the apparatus, the tracking task was performed with a presentation of the various resistive conditions. These conditions were the following: 2 levels of viscosity (0.022 and 0.044 Newtons/sec/degree), 2 levels of stiffness (0.4866 and 0.9265 Newtons/degree), and 2 inertial masses (0.5 kg and 1.0 kg). Each of the subjects performed the task with these same levels of viscosity, stiffness, and mass. Viscous resistance was provided by feeding back the velocity of movements through the amplifier of the torque motor; stiffness was created by feeding back the position of the handle through the torque motor. Inertia was altered by adding lead shot weights to the manipulandum.

As the velocity of the handle movement increased, the torque produced by the feedback device increased in a linear manner, at all

gains tested, thus producing viscosity which consistently varied with velocity. (Figures 2a and 2b) Similarly, the stiffness created by feeding back the handle position increased as a function of the distance from the neutral flexion/extension handle position. (Figure 3)

5. PROTOCOL

Subjects initially performed 10 trials in an unloaded condition to familiarize themselves with the tracking task. Following these learning trials, the subjects performed 6 trials for each of the 6 resistive conditions. The two levels of each resistive condition were performed in succession, with the lowest level being presented first. Following each resistive condition were 3 unloaded trials. A Student's t-test was done to compare results during the 6 trials of each resistive condition to the total of 6 unloaded trials immediately preceding and following them.

The experimental protocol was designed to avoid biasing the results from the effects of learning in the following manner:

- 10 unloaded trials were performed at the outset to familiarize the subjects with the apparatus and the task.
- Subjects were instructed to try several movements against each imposed load before the tracking trials with that load began.
- Each set of tracking trials with a load were compared with the unloaded trials immediately preceding and following it.
- The order in which the altered viscosity, stiffness, and inertia were presented to each subject in one population was varied.

6. DATA COLLECTION

6.1 POSITIONAL ERROR

At each sampling point on the position tracing, the distance between the cursor and the target was measured and summed for each 20 second trial to create a quantitative measure of error or accuracy, which is referred to as the "positional error score". This score was used to provide comparisons of the various trials of an individual to determine which testing conditions produced optimal accuracy of performance. The positional error score was also compared between subjects in each population. The amount of error depicted on the plot of each trial could be measured during various phases of movement to determine if there was a consistent pattern in an individual's error or accuracy.

6.2 MOVEMENT VELOCITY

To determine if the errors in programming velocity of movement contribute to the irregularities and overshoot which occur when a subject attempts to manually track a moving visual target, the signals representing target and cursor position were differentiated to examine movement velocity. A "velocity error score" was derived in the same manner as that of the positional error score; that is, at every data point on the velocity trace the distance of the cursor relative to the target was measured and summed for each 20 second trial. Three other measurements were made for the velocity tracings for each trial: a) the number of velocity peaks greater than 30 degrees/sec., b) the number of "reversals" in the velocity trace, and c) the number of times the velocity trace crossed zero degrees/second. A computer program was used to display each velocity trace and to keep a tally as

the investigator manually counted each of these measurements. The measurements were done only for the patients with the unilateral cerebellar lesions and the control subjects.

6.3 ELECTROMYOGRAPHY

The EMG activity for each subject was calibrated by having the subject hold their wrist in a neutral flexion/extension position while a 0.45 Nm torque was imposed on the wrist flexors and extensors, alternately, for 5 seconds each. The EMG sampling rate was 100 per second. The EMG activity from the wrist flexors and wrist extensors was amplified, and passed through a full wave rectifier-averager with a time constant of 66 ms. An index of the amount of coactivation between wrist flexors and extensors was calculated by obtaining from the plot at each data point the amount of flexor and extensor EMG during the twenty second trial and using the following formula:

$$\text{Coactivation index} = (A + B) \times A/B$$

Where: A = flexor EMG

B = extensor EMG

When: There is a smaller amount of flexor EMG than extensor EMG.

If: The flexor EMG is larger than the extensor EMG then A and B are reversed.

This formula for the coactivation index provides a value which indicates the relative magnitude of coactivation, and not just a ratio of activity present in the 2 muscles.

The coactivation index was examined during the learning trials to determine whether subjects changed the amount of coactivation they used while improving their accuracy of tracking performance. The coactivation index allowed a comparison of the relative amounts of coactivation in both subject groups, and also during the various resistive conditions imposed on the tracking task.

7. PHASE LAG BETWEEN TARGET MOVEMENTS AND SUBJECT'S RESPONSE

To determine what effect the length of time the subjects take to respond to the moving target has on the positional error score, the cursor tracing was shifted horizontally with respect to the target tracing to obtain the lowest possible error score.

By obtaining the phase lag value that produces the lowest error scores, one could determine the following: the existence of any consistent differences in this lag time between subject populations, differences which occur during the various resistive conditions, and any changes that may occur with the lag time during learning of the tracking task.

The amount of decrement in positional error score which can be obtained by this time shift was also examined in all subject populations.

In order to interpret these results, it is helpful to know the time that it takes for the subjects to respond to a visual cue with a wrist movement. The reaction time of 2 control subjects and 2 patients (affected arms) was determined. This was accomplished by positioning the subjects in the same apparatus as for the tracking task, and displaying a target and a cursor on the screen. The target would jump horizontally to one side or the other at unpredictable

intervals, and the subject was instructed to move the cursor in line with the target as soon as possible. The target moved 10 times to the same location at each side of the screen. The range and mean of the reaction times were provided by the computer program.

8. EFFECTS OF ELIMINATING VISUAL FEEDBACK

Some of the subjects (2 with unilateral cerebellar lesions, 7 patients with other diagnoses listed above, and 3 control subjects), were required to perform a self-paced sinusoidal wrist movement. Without a target on the CRT screen, they were asked to rhythmically flex and extend the wrist over approximately the same range of motion and at a similar velocity to that of the tracking task. The subjects were instructed not to look at their wrist. They performed this task for 3 trials of 20 seconds duration each, immediately following the initial 10 learning trials of the tracking task. These self-paced sinusoidal movements were performed to observe the differences between this movement without visual input, and the movements while tracking a visual target.

9. DETERMINATION OF DAMPING FACTOR AT THE WRIST

To understand results from these experiments, it is important to examine not only the mechanical properties of the apparatus, but also to know whether there are any consistent differences between subjects with regard to the amount of mechanical damping which exists at the wrist joint.

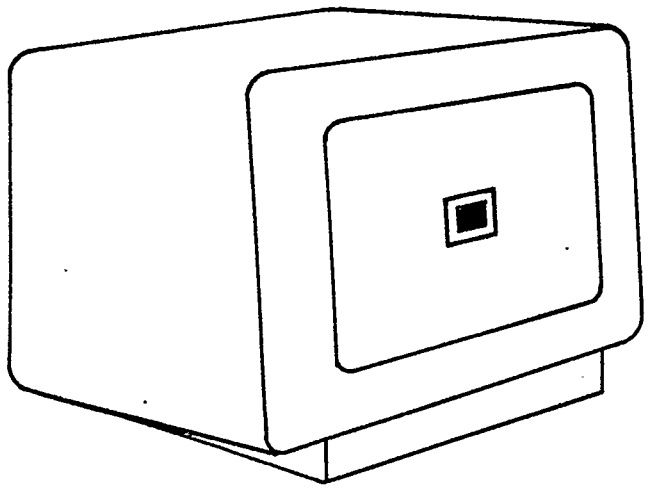
Subjects were positioned as described for the tracking task. A light emitting diode indicated to the subject when their wrist was in the neutral (90 degree) position of relative flexion/extension. The

subject resisted varying preloads to the wrist flexors, and a 50 msec. pulsed torque perturbation at 1.8 N-m was delivered through the manipulandum to produce extension at the wrist. Subjects were given the instruction "do not intervene" (Asatryan, Feldman 1965). This produced an harmonic oscillation of the wrist position which could be described by a second order linear system. Force, velocity, and EMG of the wrist flexors and extensors were also recorded for each perturbation trial.

From measurements and calculations of these oscillations of wrist position, the damping factor could be determined as outlined in Figure 4.

FIGURE 1

Position of subject in apparatus for manual tracking task of a visual target.



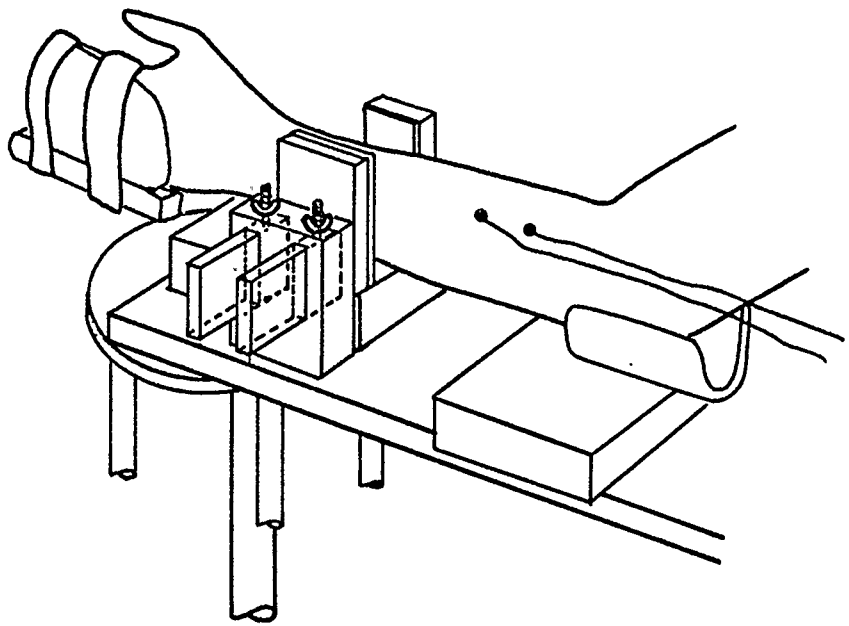


FIGURE 2a

A function generator was used to produce ramp inputs of constant duration and variable amplitude. This resultant velocity was fed through the amplifier to the torque motor. The motor produced a torque of 1.8 Nm with an input of 5 volts, so the torque produced by these inputs could be calculated. This was done at 4 gains. There was a linear relationship between the increase in velocity and the increase in torque at all gains tested.

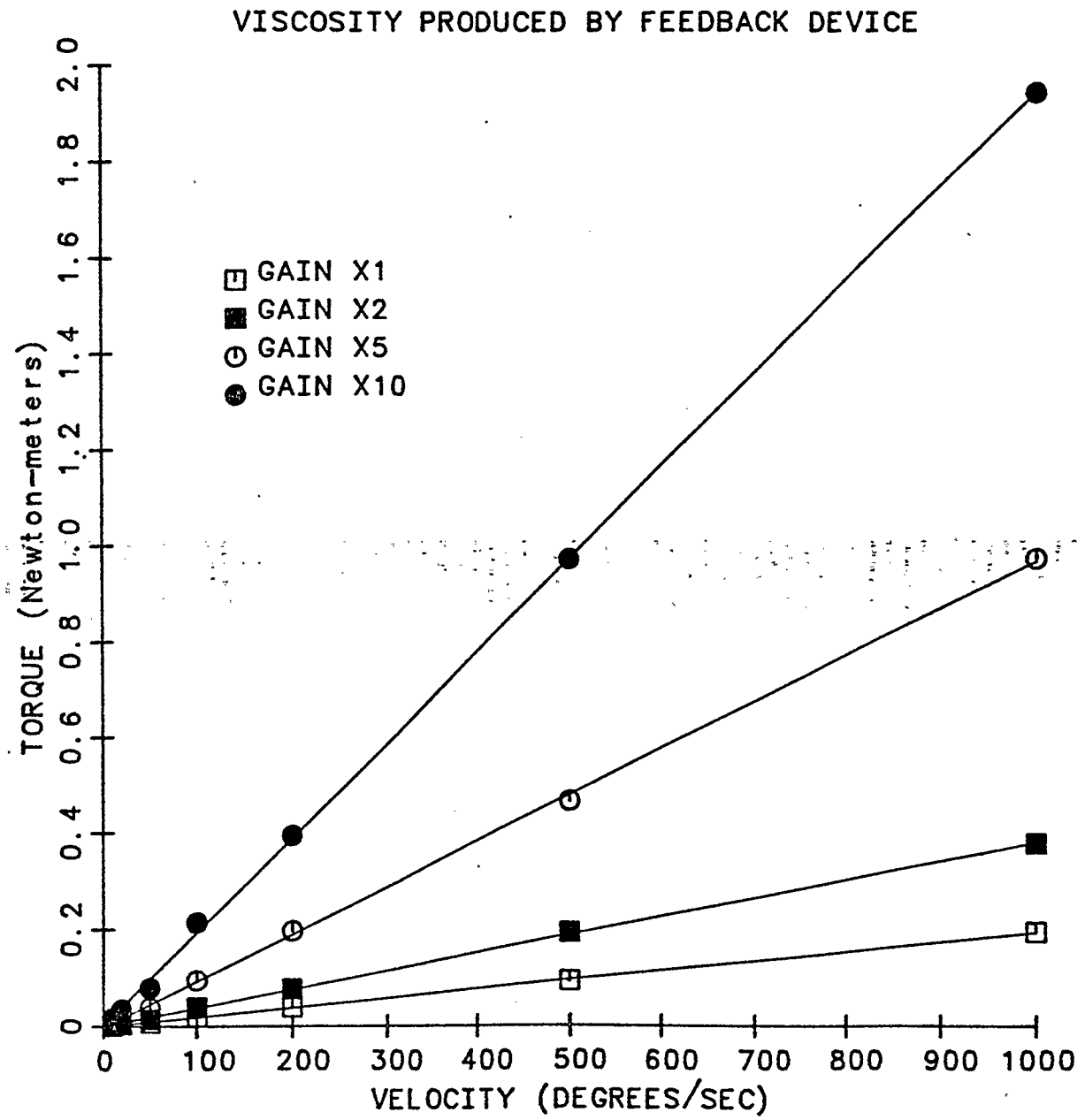

VISCOSITY PRODUCED BY FEEDBACK DEVICE
GAIN X1
GAIN X2
GAIN X5
GAIN X10
TORQUE (Newton-meters)
0
0.2
0.4
0.6
0.8
1.0
1.2
1.4
1.6
1.8
2.0
0
100
200
300
400
500
600
700
800
900
1000
VELOCITY (DEGREES/SEC)

FIGURE 2b

The viscosity produced at 4 gains was plotted on a log scale to show that the changes in torque produced by the velocity inputs at one gain varied in parallel to the changes in torque at the other gains.

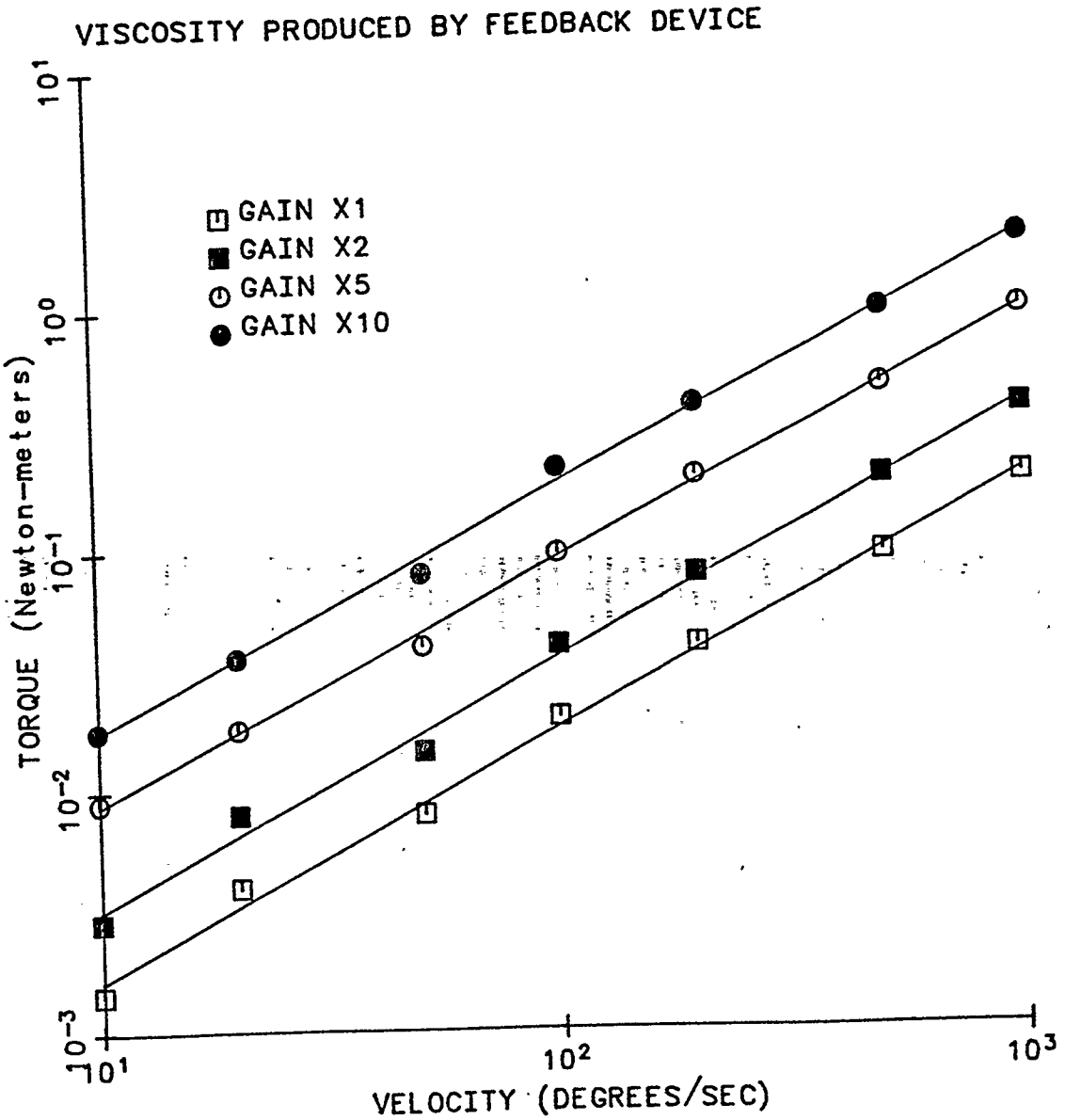

VISCOSITY PRODUCED BY FEEDBACK DEVICE
GAIN X1
GAIN X2
GAIN X5
GAIN X10
TORQUE (Newton-meters)
10^{1}
10^{0}
10^{-1}
10^{-2}
10^{-3}
10^{1}
10^{2}
10^{3}
VELOCITY (DEGREES/SEC)

FIGURE 3

Using the feedback device for the handle position, as the handle is moved from the position of neutral flexion/extension, the torque produced increased linearly at both gains used in this study.

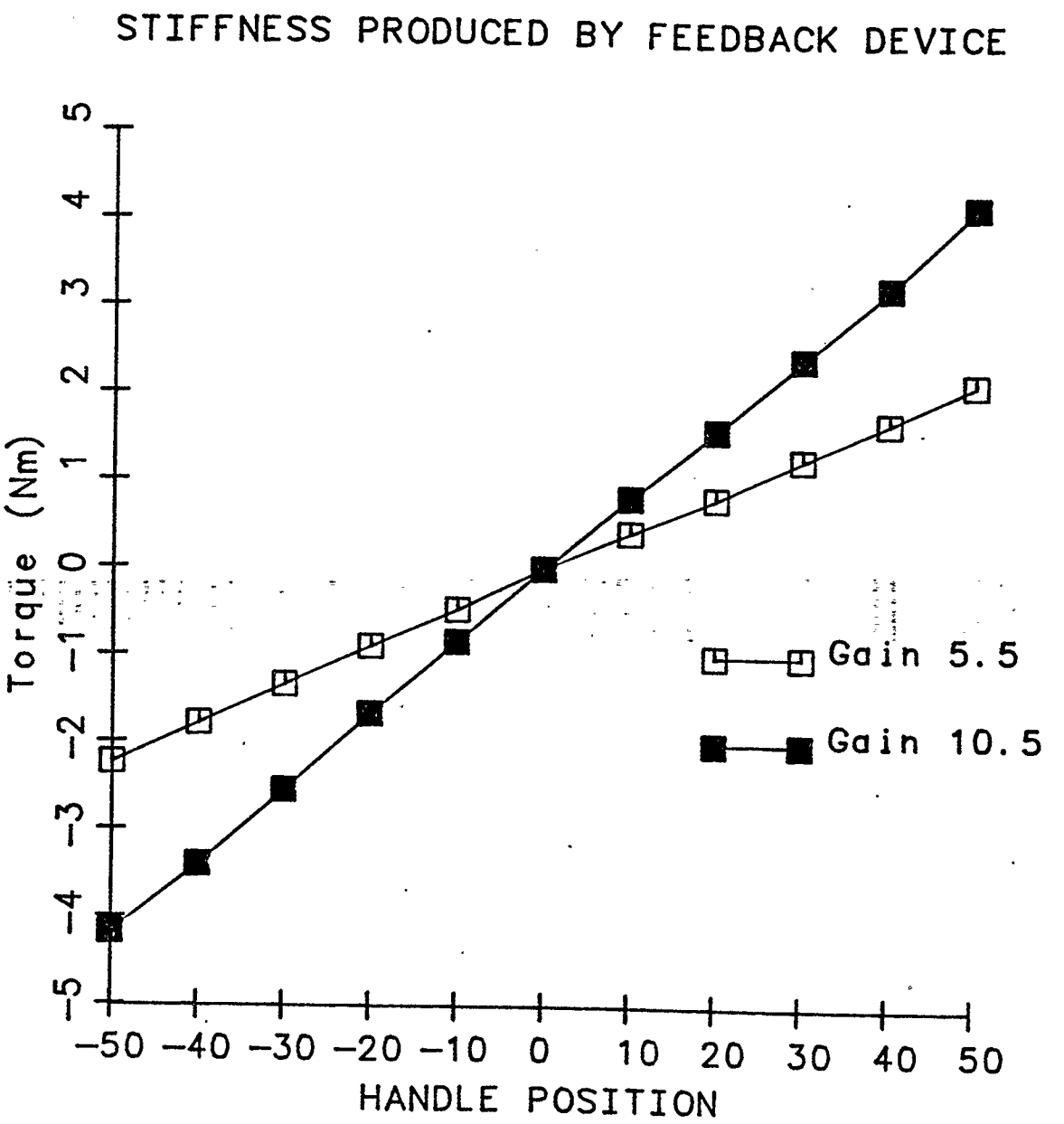
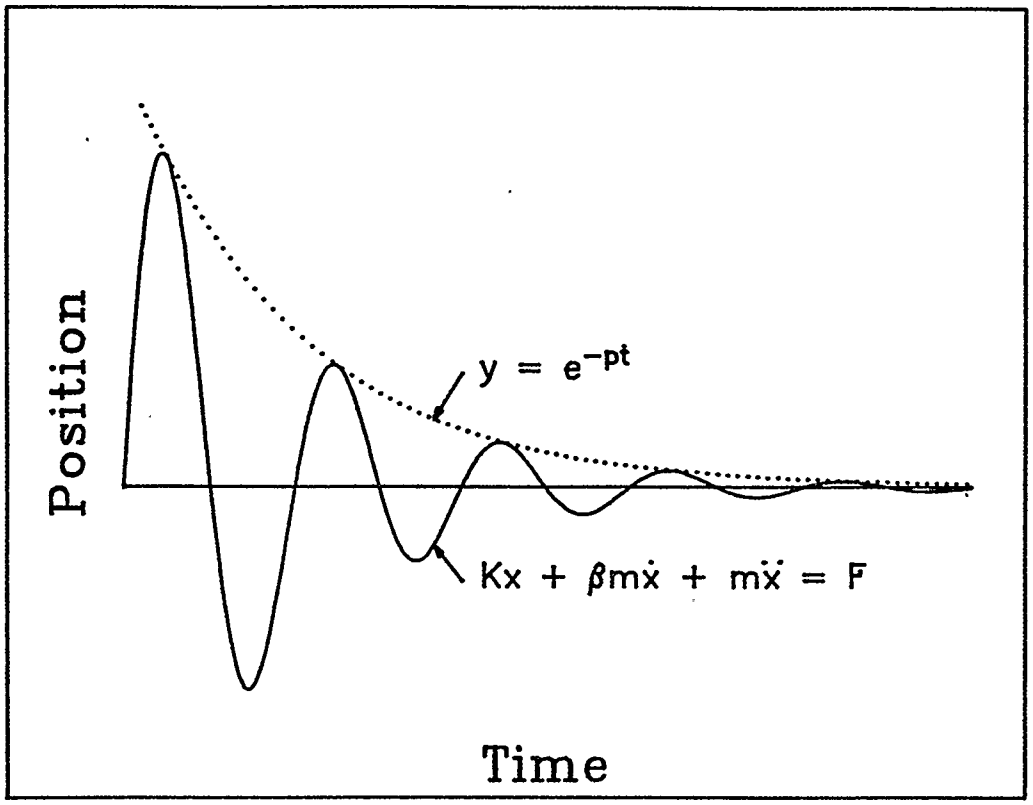

STIFFNESS PRODUCED BY FEEDBACK DEVICE
Torque (Nm)
5
4
3
2
1
0
-1
-2
-3
-4
-5
-50 -40 -30 -20 -10 0 10 20 30 40 50
HANDLE POSITION
Gain 5.5
Gain 10.5

FIGURE 4. MATHEMATICAL MODELLING



1) Determine p $\quad p = 1/\tau$

2) Measure f_D $\quad f_D = 1/t_D$

3) Calculate f_N and ζ $\quad f_N = \sqrt{f_D^2 + p^2/4\pi^2}$

$\zeta = p/2\pi f_N$

4) Calculate K and β, assuming constant m

$f_N = 1/2\pi\sqrt{K/m} \rightarrow K = 4f_N^2\pi^2 m$

$\zeta = \beta/2\sqrt{Km} \rightarrow \beta = 2\zeta\sqrt{Km}$

τ = time constant
p = rate constant of decay
f_D = damped frequency
t_D = period
f_N = natural frequency

ζ = damping factor
K = stiffness
β = viscosity
m = mass
F = force

RESULTS

Manual tracking of a visual target was performed with various resistive conditions by normal control subjects and by patients with motor control disorders. The following aspects of tracking performance were investigated: positional error, velocity of movement, EMG of the wrist flexor and extensor muscles, time-shift plots producing the lowest positional error, learning effects on error and EMG, and self-paced sinusoidal tasks.

Relevant clinical findings of the patients studied are summarized in Table 1. Computed tomography (CT) scans of the cerebellar lesions in the patients with unilateral cerebellar dysfunction are shown in Figure 5, excluding the patient with a cerebellar infarct whose CT scan was unremarkable.

1. WRIST POSITION DURING TRACKING MOVEMENTS

Position recordings during tracking are shown in Figure 6. The control subject did not follow the target smoothly, but made corrective adjustments while tracking. The patient with a cerebellar lesion made higher amplitude corrective movements than the control subject. A recently published report by Miall, Weir and Stein (1987) reveals similar tracking patterns to these in normal monkeys and in monkeys with cerebellar lesions.

1.1 POSITIONAL ERROR SCORE

For each 20 second tracking trial a positional error score was derived, as described in the Methods. The mean positional error score during all unloaded trials was obtained for each subject. These mean

positional error scores were averaged for all of the subjects in each population studied (Figure 7). Tracking with the affected arms of the patients with unilateral cerebellar lesions produced greater error ($p<0.005$) than did tracking with the clinically unaffected arms and by the control subjects. The group of patients with "other" cerebellar lesions tracked with greater error than the control subjects ($p<0.005$) and than the affected arms of the patients with unilateral cerebellar lesions ($p<0.025$). The tracking error of the Parkinsonian patients was not significantly different from the control subjects.

1.2 EFFECTS OF ALTERED VISCOSITY, STIFFNESS AND INERTIA ON POSITIONAL ERROR

Figure 8 shows the positional error scores of a normal subject during each of the experimental and unloaded conditions, in the order in which they were presented. The only significant change in positional error during the various conditions is an increase the error during tracking with the 1.0 kg mass added to the manipulandum ($p<0.050$). Note that the error score during each of the sets of unloaded trials remains relatively constant. In Figure 9 are the positional error scores during the various conditions in a patient with a unilateral cerebellar hemangioblastoma. The positional error scores for this patient are more than twice as high as the scores for the normal subject. The error score is almost halved during the trials performed with the highest level of stiffness and with both levels of viscosity ($p<0.005$).

Table 2 summarizes the effects of the various loading conditions on the positional error scores in subjects with unilateral cerebellar

lesions and control subjects. Addition of either level of viscosity improved tracking accuracy in the patients' affected arms; however, in their clinically unaffected arms and in the control subjects the altered viscosity usually made no change or occasionally improved the tracking accuracy. Addition of stiffness did not produce any consistent change in the tracking accuracy in these subjects. Application of both amounts of mass produced either no change in the error score or made it higher in the unilateral cerebellar patients and control subjects.

Unlike the patients with unilateral cerebellar lesions, the group of subjects with various cerebellar pathologies (not shown in table) did not show a significant change in error score when either level of viscosity was added. As well, no significant change in the error score was produced by adding either level of stiffness. With the exception of one subject whose error score remained unchanged, these patients had an increased error score with the addition of inertial mass of 0.5 kg. or 1.0 kg.

The 2 patients with Parkinson's Disease did not have significant change in their positional error scores with the addition of viscosity, nor with increased inertia. One of these 2 subjects had increased positional error ($p<0.050$) when the highest level of stiffness was added, while the other subjects' positional error score did not change with increased stiffness.

2. VELOCITY OF TRACKING MOVEMENTS

Typical movement velocity traces during a portion of the tracking task are shown in Figure 10 for a control subject and a subject with a unilateral cerebellar lesion. The amplitude of the velocity peaks was

greater in the movements performed by the patients than by the control subjects.

2.1 VELOCITY ERROR SCORES

A velocity error score was derived from the velocity trace in the same manner as the positional error score was obtained from the position recordings. The mean velocity error scores of the different subject groups are given in Figure 11. The velocity error score was higher ($p<0.005$) during tracking with the affected arms of the patients than with the unaffected arms or control subjects.

The group of patients with other cerebellar lesions produced higher velocity error scores ($p<0.050$) than the control subjects, and the velocity error scores in the Parkinsonian patients were not significantly different from the control subjects.

2.2 EFFECTS OF ALTERED VISCOSITY, STIFFNESS, AND INERTIA ON THE VELOCITY ERROR SCORES

Table 3 summarizes the effects of the loads on the velocity error scores. When the highest level of viscosity was applied the velocity error score became lower for every patient with unilateral cerebellar lesions and for each control subject. With moderate viscosity the velocity error score produced was lower for all but 2 subjects (one patient's unaffected arm and one control subject). There was no consistent change in the velocity error scores for these subjects when either stiffness or inertia were altered.

Similar results were found for the other cerebellar patients and the patients with Parkinson's Disease; viscosity decreased the

velocity error score, and stiffness and inertia produced no consistent effect on the velocity error score.

2.3 VELOCITY REVERSALS, AMPLITUDE PEAKS, AND ZERO CROSSINGS

To extract further information from the velocity traces, the number of reversals, amplitude peaks > 30 degrees/second and zero crossings were counted. Figure 12 describes how these measurements were made.

In the unloaded condition the velocity recording of the affected arms of the patients had more high amplitude velocity peaks ($p<0.005$) than their clinically unaffected arms or the control subjects (Figure 13).

There tended to be fewer reversals in the velocity recording of the control subject than the patients affected arm, but this difference was not statistically significant (Figure 14).

The patients had more zero crossings of their tracking velocities with their affected arm ($p<0.010$) than with their unaffected arm or than the control subjects (Figure 15).

The number of reversals, amplitude peaks > 30 degrees/second and zero crossings were also counted during the trials in which the mechanical loads were altered. These results are summarized in Tables 4 to 6.

The number of high amplitude velocity peaks was usually diminished with the application of viscosity, but in a few subjects there was no significant change. Increased stiffness produced no consistent effect on the number of peaks > 30 degrees/second, and increased inertia resulted in either fewer high amplitude peaks or no significant change (Table 4).

The number of reversals in the velocity trace was either decreased or unchanged when viscosity was added. Increased stiffness resulted in more reversals or no significant change. Increased inertia caused either fewer reversals or no significant change to the number of reversals (Table 5).

In all but one patient's affected arm the application of viscosity resulted in fewer zero crossings of the velocity trace. The added viscosity tended not to change the number of inappropriate zero crossings in the control subjects. This is probably due to the fact that the control subjects had few inappropriate zero degree crossings in the unloaded condition (Figure 15). Increased stiffness resulted in either a greater number of zero crossings or produced no significant change. With the exception of 2 patients' affected arms, increased inertia produced no change in the number of zero crossings (Table 6).

3. ELECTROMYOGRAPHY

3.1 COACTIVATION INDEX

Normal subjects may improve tracking accuracy by coactivating opposing muscles acting at the wrist joint, thus increasing the stiffness of the joint. A contributing factor to the tracking difficulties encountered by cerebellar patients may be an inability to generate sufficient coactivation. To address this question, an estimate of the amount of coactivation present was estimated by using a coactivation index, as described in the Methods.

Further investigation is necessary to determine the validity of this coactivation index. Initial studies where the subject was instructed to coactivate to varying degrees have revealed an

appropriate adjustment in the coactivation index. Figure 16 shows an example of the EMG recording for 2 control subjects who coactivated to different degrees. This is reflected by their coactivation indices.

In Figure 17 are EMG recordings from a control subject and a patient, with their corresponding position recordings and coactivation indices. In this example there was less coactivation during tracking by the patient than by the control subject. Bursts of EMG in one muscle of the patient are accompanied by deactivation in the antagonist. This results in an unstable position trace which tends to oscillate about the target.

When the mean coactivation index of all 28 unloaded trials for each subject was plotted, there was a tendency for the control subjects to coactivate more than the patients, but this difference was not significant (Figure 18). To rule out the possibility that changing the loading conditions interfered with the amount of coactivation during the interspersed unloaded trials, we also examined the 10 initial unloaded trials only, and the 3 unloaded trials performed immediately prior to the first condition change. In both of these cases there continued to be no significant difference in the coactivation indices between these subject groups.

Further studies with a greater number of successive unloaded trials should be done to adequately test this hypothesis.

3.2 EFFECTS OF ALTERED VISCOSITY, STIFFNESS, AND INERTIA ON THE COACTIVATION INDEX.

Table 7 summarizes the effects of the applied loads on the coactivation index. Increased viscosity produced no consistent change

in any of the subjects. Increased stiffness and inertia tended to produce either higher coactivation indices or no significant change.

4. PHASE LAG BETWEEN TARGET MOVEMENT AND SUBJECT'S RESPONSE

Increased reaction time is often a feature of patients with cerebellar lesions (Holmes, 1917, Beppu, 1984, Rothwell, 1987). To examine if a time lag in tracking contributes to the inaccuracies seen in cerebellar patients, we shifted the target position on the cursor position to produce the lowest positional error score (Figure 19).

4.1 REACTION TIME

The time required to respond to a visual cue with a wrist movement in one of two directions was tested in the affected arms of 2 patients and 2 normal subjects. The mean reaction time for the patients was 366.50 ms., and for the controls was 258.70 ms. The difference between these 2 reaction times is significant ($p<0.005$), using Student's t-test.

4.2 POSITIONAL ERROR SCORE

To acquire the lowest positional error score required a greater time shift in the position recordings of the patients' affected arm ($p<0.025$) than in their clinically unaffected arm or in control subjects (Figure 20). The patients with other cerebellar lesions also had a greater phase lag ($p<0.025$) than the control subjects. Although the phase lag in the Parkinsonian patients was greater than the control subjects, this difference was not significant.

The amount of improvement in the error score which was produced by this time shift was greater in the affected arms of the patients

($p<0.010$) than in their unaffected arm or in the control subjects (Figure 21). Patients with other cerebellar lesions also showed greater improvement in their error score ($p<0.005$) with the time shift than did the control subjects. The improvement in the error score of the Parkinsonian patients with the time shift was greater than the control subjects, but again this difference was not significant.

4.3 EFFECTS OF ALTERING VISCOSITY, STIFFNESS, AND INERTIA ON PHASE LAG PLOTS

These phase lag plots were examined during the trials in which viscosity, stiffness, and inertia were altered. No significant change was found for the amount of time shift necessary to produce the lowest error score with any of these conditions in any of the subject groups.

5. LEARNING EFFECTS

5.1 LEARNING TRIALS

To determine the number of trials which were necessary to familiarize subjects with the tracking task, 4 normal subjects performed a series of trials in the unloaded condition. In each of these subjects there was a decrement in the positional error score during the first 10 trials, after which the error score levelled off. This data for one of the subjects is displayed in Figure 22.

5.2 CORRELATION BETWEEN COACTIVATION INDEX AND POSITIONAL ERROR SCORE

It is possible that normal subjects increase the amount of coactivation between their wrist flexors and extensors as they learn the tracking task and their positional error decreases. Patients with

cerebellar lesions may be unable to alter the amount of coactivation sufficiently to bring their positional error score down.

To investigate the relationship between error and EMG activation patterns, the positional error scores were plotted against the coactivation index for the initial 10 unloaded trials. The correlation coefficient was obtained for each subject. There was no trend found in the correlation between positional error and coactivation in any population.

It is believed that the experimental design used did not adequately address this question. A greater number of succesive trials in the unloaded condition would be necessary to determine any correlation between the coactivation index and error score. Furthermore, the usefulness of the coactivation index, as defined here, needs to be ascertained.

6. EFFECTS OF ELIMINATING VISUAL FEEDBACK

To determine whether visual feedback contributes to errors made during a tracking movement, subjects performed a self-paced sinusoidal wrist movement without a target or cursor display. Figure 23 shows typical position and EMG recordings for a control subject and a cerebellar patient performing this task. The control subject had only rare corrective movements, as compared to slightly more frequent discontinuities while tracking a pseudo-random target with visual feedback. The cerebellar patient's position recording also has markedly fewer corrective movements during this self-paced sinusoidal task than while tracking the unpredictable pattern.

All subjects had less coactivation of their wrist flexor and extensor muscles during this task than while performing the tracking

task. As seen in Figure 23, the muscles activate reciprocally corresponding to the handle position.

7. DETERMINATION OF DAMPING FACTOR AT THE WRIST

Patients with cerebellar lesions often exhibit hypotonia (Gilman et al. 1981). If the hypotonia resulted in decreased damping of the limbs this could explain why increasing the viscosity through an external device can assist these patients in tracking accuracy.

The oscillations of position produced in the human forearm in response to a perturbation can be modelled by a second order linear system (Lacquaniti et al. 1982, Lakie 1984, Nichols et al. 1977, Stein and Lee 1981, Oguztorelli and Stein 1976).

Figure 24 shows that it can be appropriate to use a second order linear system to model the response of a normal subjects' wrist to a perturbation. This subject resisted a 0.9 Nm preload to the wrist flexors, and was instructed not to intervene, as a 1.8 Nm torque perturbation was delivered to the wrist extensors. These results are representative of those of the 6 other subjects tested in this manner.

Five control subjects and 2 subjects with cerebellar dysfunction received torque perturbations to their wrist to produce oscillations similar to those in Figure 25. From these recordings, the damping factor was calculated, as described in the Methods. The damping at the wrist of the cerebellar patients was less than that at the wrists of the control subjects ($p<0.010$). The range of damping factors for the control subjects was 0.24 to 0.36, and for the patients was 0.21 to 0.23.

TABLE 1

RELEVANT CLINICAL FINDINGS OF PARTICIPATING PATIENTS

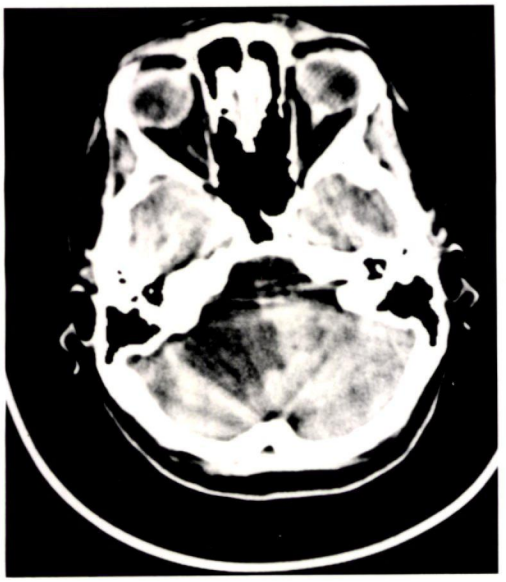
DIAGNOSIS	AGE	DYSMETRIA	TREMOR	OTHER FINDINGS
R. post fossa epidermoid	29	mild	mild	hyporeflexive
R. cerebellar hemangioblastoma	30	minimal	none	only fine motor problems R. hand
R. cerebellar astrocytoma	20	mild	mild	
L. cerebellar astrocytoma	16	mild	mild	
R. cerebellar infarct	25	mild	mild	sl. weakness of med interossei, slight proprioceptive loss right fingers
Spinocerebellar degeneration	33	mild	mild	hyporeflexive
Spinocerebellar degeneration	37	moderate	mild	dysarthria, balance difficulties, hyperreflexive
Spinocerebellar degeneration	58	mild	mild	
Head injury	25	severe	moderate	hyperreflexive
Demyelinating encephalomyelitis	38	none	moderate	hyporeflexive, dysarthric
Multiple Sclerosis (Left cerebellar involvement)	54	moderate	none	sl. dysarthria
Diffuse cerebellar degeneration-cause undetermined	49	moderate	mild	In W/C, requires assistance with ADL
Brain stem astrocytoma	17	mild	mild	mild weakness of L. interossei, marked weakness of L. tibialis ant.
Parkinson's disease	68	none	moderate	more problems with L. than R.
Parkinson's disease	40	none	mild	more problems with L. than R.

FIGURE 5

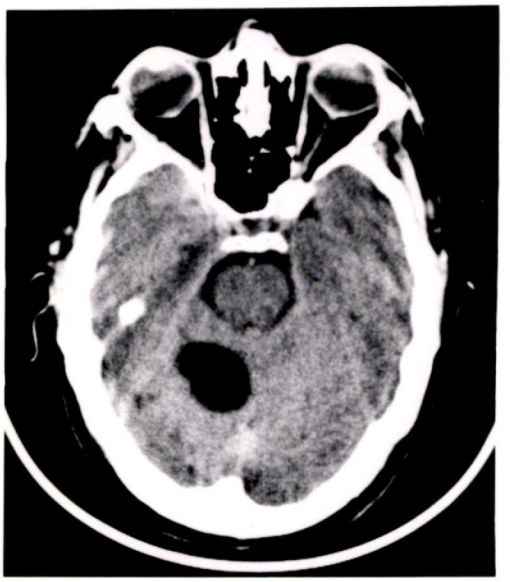
CT scans of 4 of the 5 patients with unilateral cerebellar lesions. The relevant clinical findings of these patients are outlined in Table 1. The CT scan of the fifth patient, with a unilateral cerebellar infarct, is not shown as it was unremarkable.

FIGURE 5

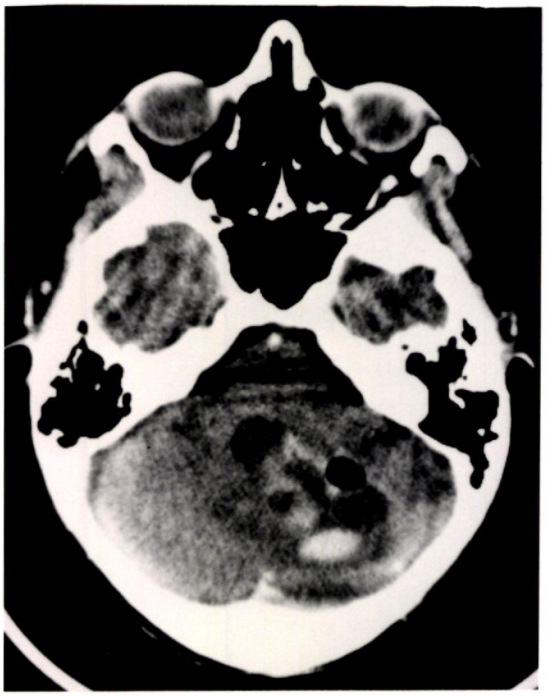
A. EPIDERMOID



B. HEMANGIOBLASTOMA



C. ASTROCYTOMA



D. ASTROCYTOMA

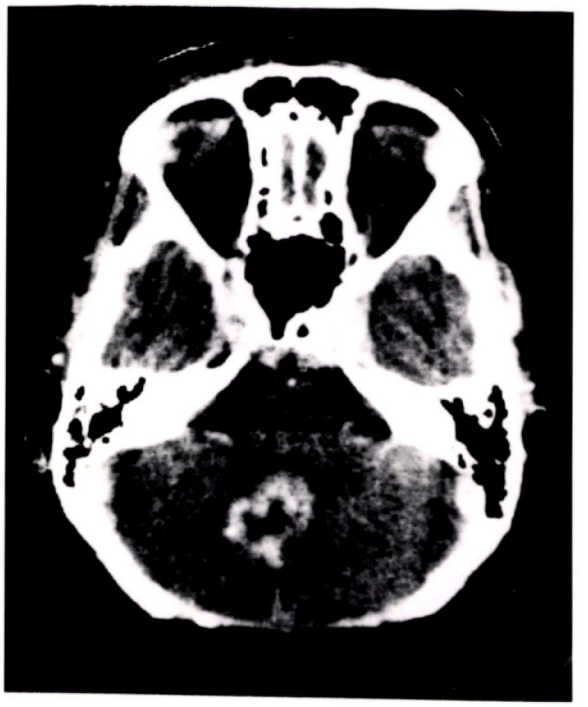


FIGURE 6

Position of the target and the cursor (controlled by the the handle) during a portion of a 20 second tracking trial in the unloaded control condition. The top panel is a typical position trace of a normal control subject and the lower panel is that of a patient's affected arm. At each data point the difference between the cursor and target positions was measured and summed for 20 seconds to create the positional error scores given in this figure.

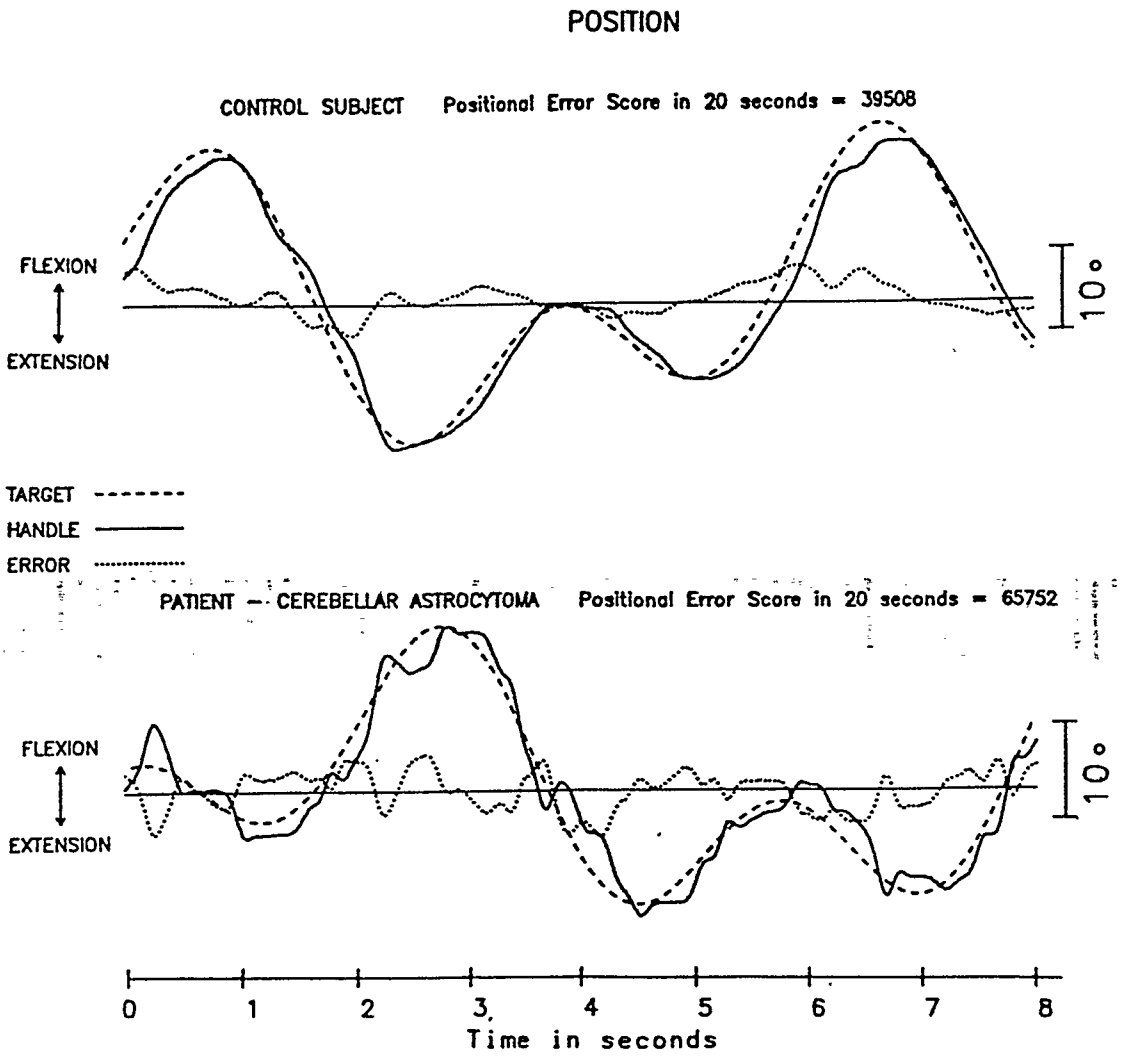

POSITION
CONTROL SUBJECT Positional Error Score in 20 seconds = 39508
FLEXION
EXTENSION
10°
TARGET
HANDLE
ERROR
PATIENT — CEREBELLAR ASTROCYTOMA Positional Error Score in 20 seconds = 65752
FLEXION
EXTENSION
10°
0 1 2 3 4 5 6 7 8
Time in seconds

FIGURE 7

Each subject performed a total of 28 unloaded trials (ie. no resistive condition imposed). For each subject population the mean positional error scores were averaged to produce these bars with their standard error. The results of the patients' arms were compared to the control subjects.

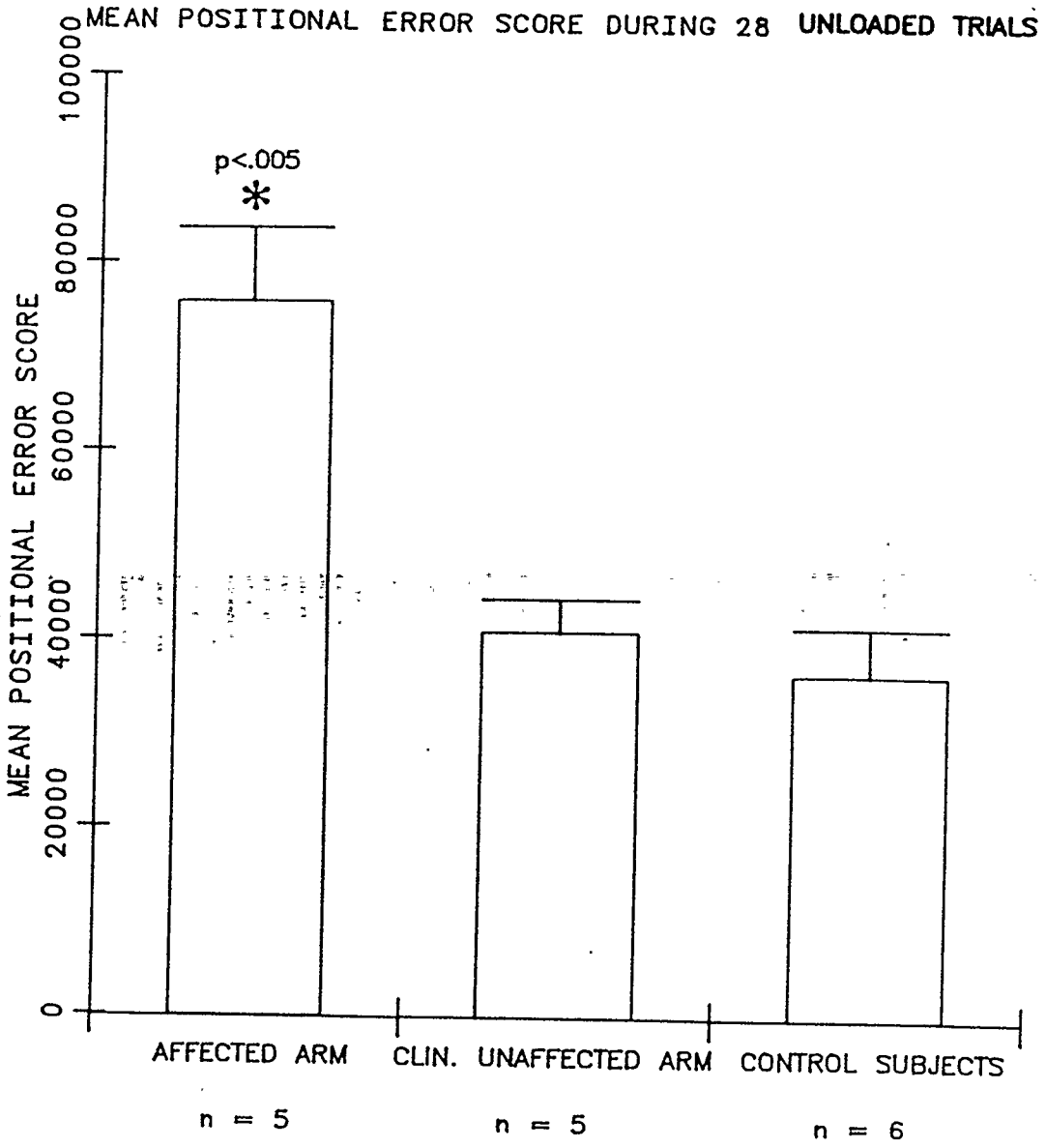

MEAN POSITIONAL ERROR SCORE DURING 28 UNLOADED TRIALS
MEAN POSITIONAL ERROR SCORE
0
20000
40000
60000
80000
100000
p<.005
*
AFFECTED ARM
CLIN. UNAFFECTED ARM
CONTROL SUBJECTS
n = 5
n = 5
n = 6

FIGURE 8

The mean positional error score during each set of unloaded and experimental conditions is shown, with standard error bars, for a normal subject. Each set of the unloaded condition consisted of 3 trials and each set of experimental conditions had 6 trials. A Student's t-test was used to compare results during the resistive condition trials to the unloaded trials immediately preceding and following them.

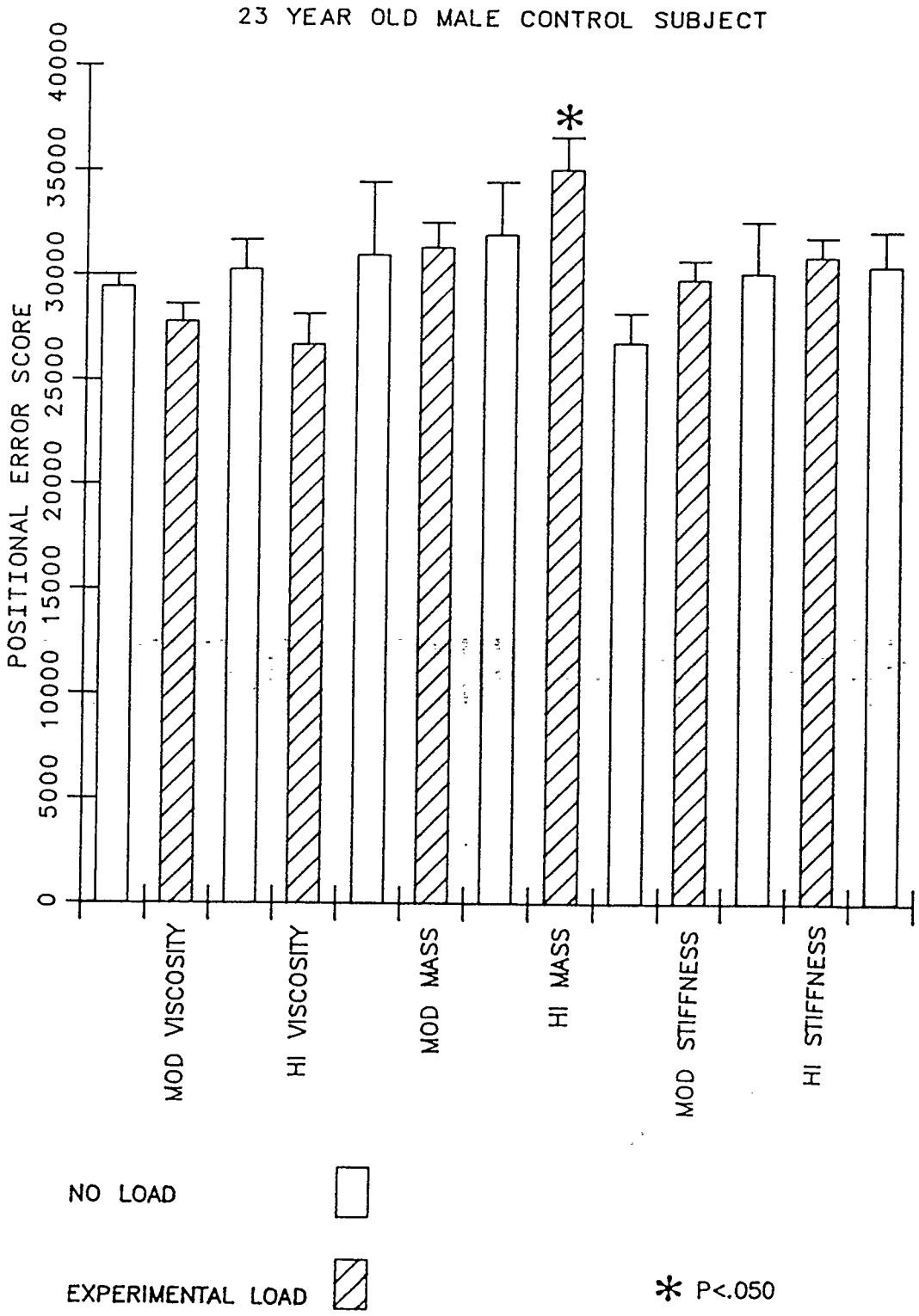

23 YEAR OLD MALE CONTROL SUBJECT
POSITIONAL ERROR SCORE
0
5000
10000
15000
20000
25000
30000
35000
40000
*
MOD VISCOSITY
HI VISCOSITY
MOD MASS
HI MASS
MOD STIFFNESS
HI STIFFNESS
NO LOAD
EXPERIMENTAL LOAD
* P<.050

FIGURE 9

The mean positional error score during each of the tracking conditions is shown for the affected arm of a patient. See the legend of Figure 8.

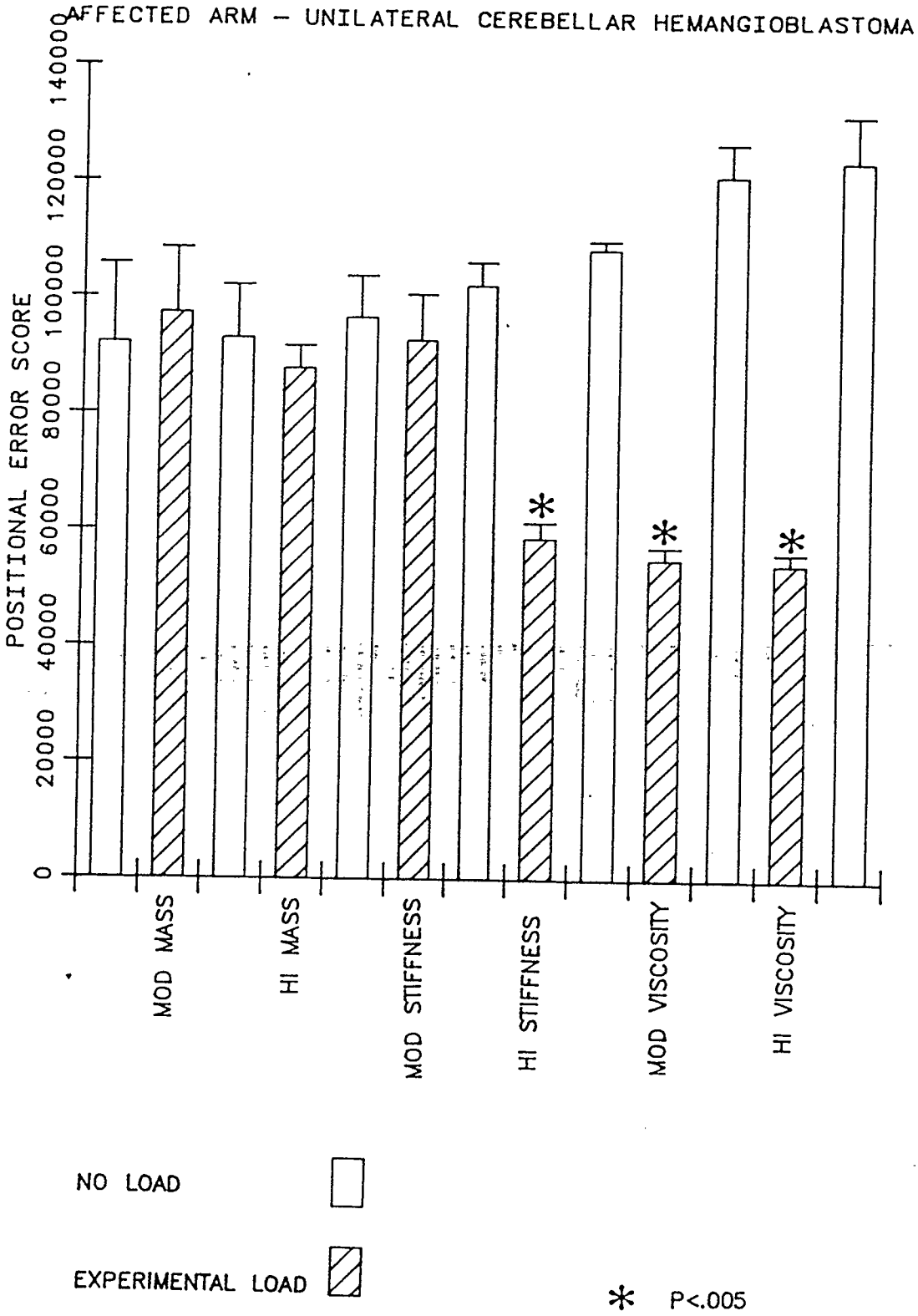

AFFECTED ARM – UNILATERAL CEREBELLAR HEMANGIOBLASTOMA
POSITIONAL ERROR SCORE
0
20000
40000
60000
80000
100000
120000
140000
MOD MASS
HI MASS
MOD STIFFNESS
HI STIFFNESS
MOD VISCOSITY
HI VISCOSITY
*
*
*
NO LOAD
EXPERIMENTAL LOAD
* P<.005

TABLE 2

This summarizes the effects of each of the loading conditions on the positional error score of the unilateral cerebellar patients and control subjects. Each asterisk represents one subject. For a change to be considered significant p must be < 0.050.

Subsequent tables will use the same format and require the same level of significance.

POSITIONAL ERROR SCORES

	BETTER	NO CHANGE	WORSE
MOD. VISCOSITY			
PATIENTS — AFFECTED ARM	*****		
PATIENTS — CLINICALLY UNAFFECTED ARM	*	****	
CONTROL SUBJECTS	*	*****	
HIGH VISCOSITY			
PATIENTS — AFFECTED ARM	*****		
PATIENTS — CLINICALLY UNAFFECTED ARM	**	***	
CONTROL SUBJECTS	**	****	
MOD. STIFFNESS			
PATIENTS — AFFECTED ARM	**	***	
PATIENTS — CLINICALLY UNAFFECTED ARM	**	*	**
CONTROL SUBJECTS		******	
HIGH STIFFNESS			
PATIENTS — AFFECTED ARM	***	*	*
PATIENTS — CLINICALLY UNAFFECTED ARM		****	*
CONTROL SUBJECTS		******	
MOD. INERTIA			
PATIENTS — AFFECTED ARM		****	*
PATIENTS — CLINICALLY UNAFFECTED ARM		**	***
CONTROL SUBJECTS		*****	*
HIGH INERTIA			
PATIENTS — AFFECTED ARM		****	*
PATIENTS — CLINICALLY UNAFFECTED ARM		**	***
CONTROL SUBJECTS		**	****

FIGURE 10

Velocity of the target and of the cursor during a portion of a 20 second tracking trial in the control condition. The top panel is a typical velocity trace of a normal subject, and the lower panel is that of a patient's affected arm.

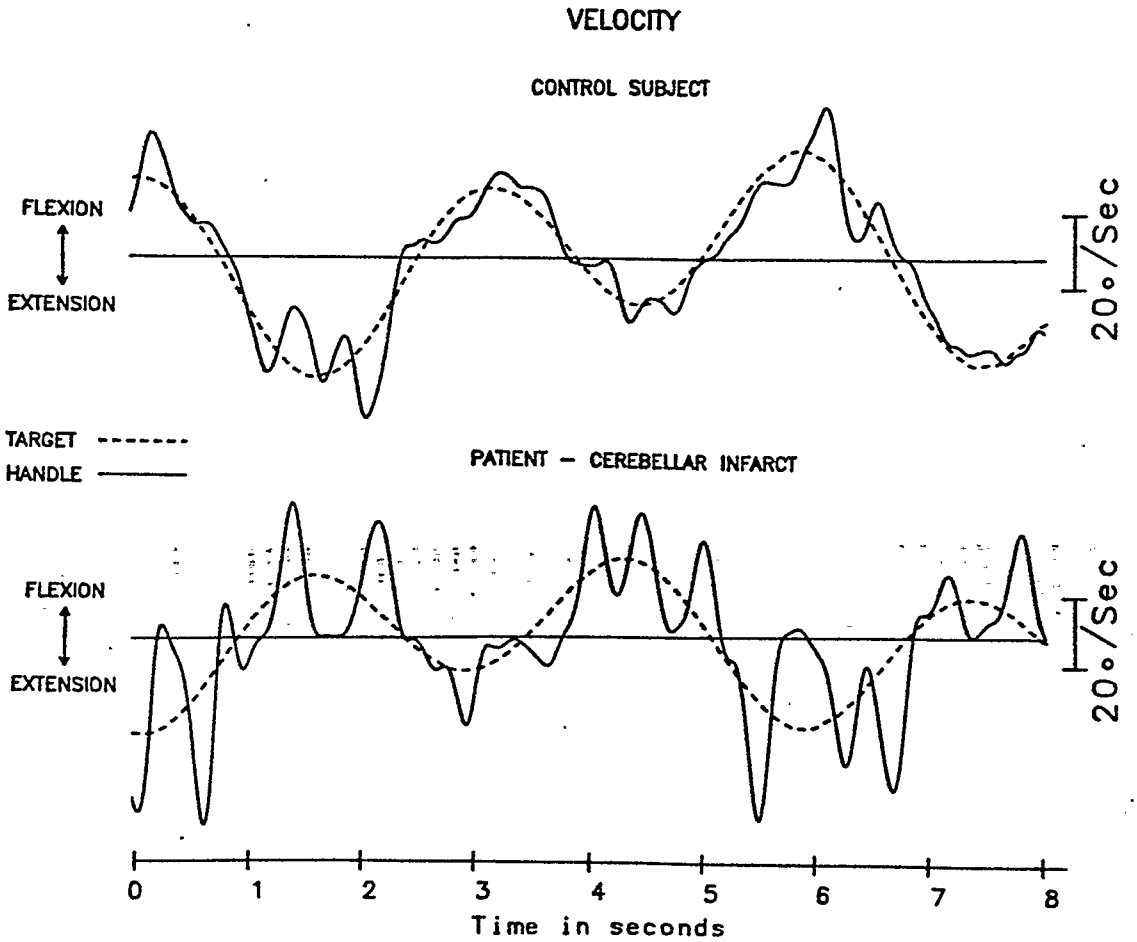

VELOCITY
CONTROL SUBJECT
FLEXION
EXTENSION
20°/Sec
TARGET
HANDLE
PATIENT – CEREBELLAR INFARCT
FLEXION
EXTENSION
20°/Sec
0
1
2
3
4
5
6
7
8
Time in seconds

FIGURE 11

At each data point the difference between the target and cursor velocities was measured and summed for 20 seconds to create a velocity error score. For each population the mean velocity error score during each subjects' 28 unloaded trials were averaged to produce these bars with their standard error. The results of the patients' arms are compared with the control subjects.

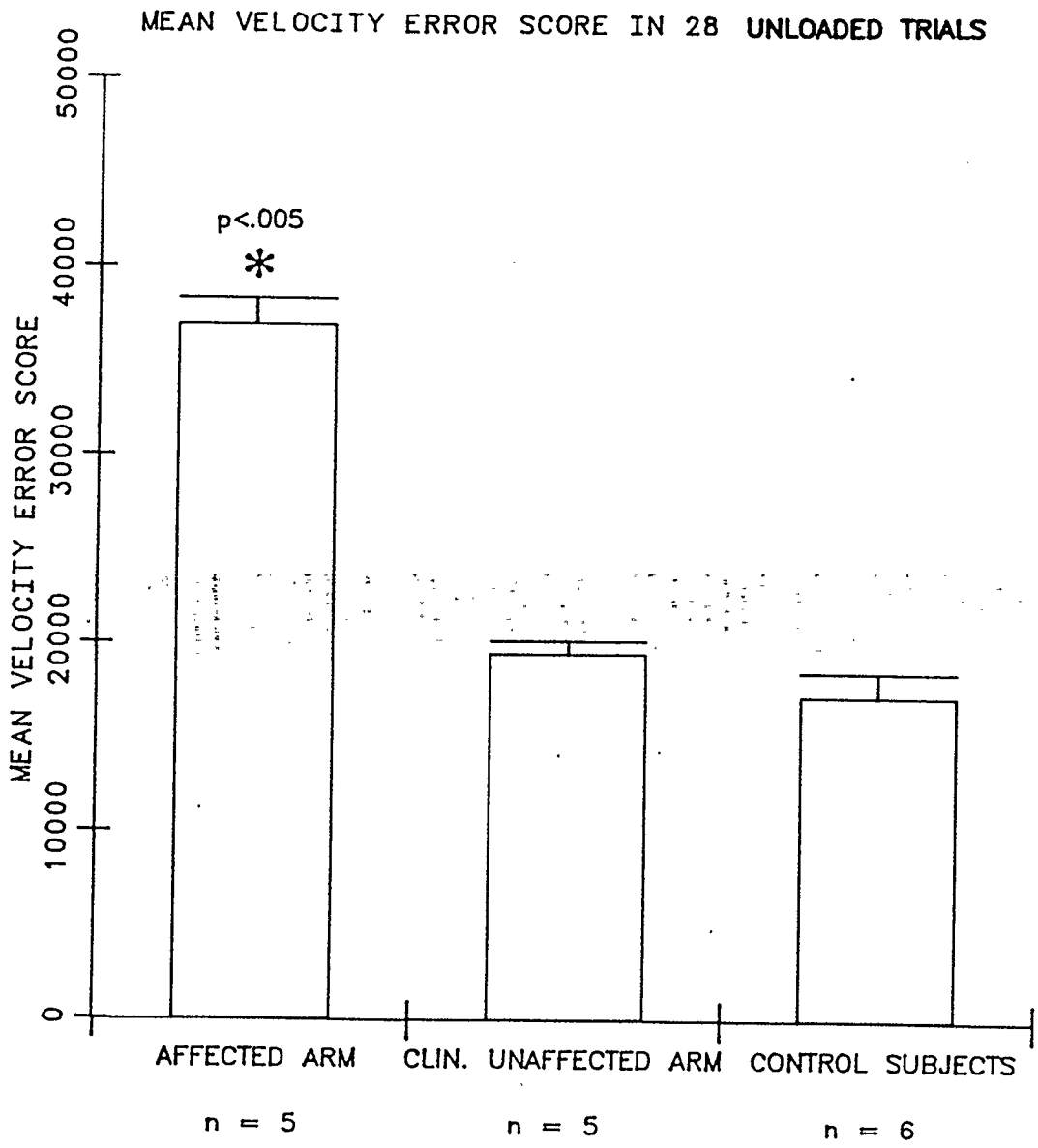

MEAN VELOCITY ERROR SCORE IN 28 UNLOADED TRIALS
MEAN VELOCITY ERROR SCORE
0
10000
20000
30000
40000
50000
p<.005
*
AFFECTED ARM
CLIN. UNAFFECTED ARM
CONTROL SUBJECTS
n = 5
n = 5
n = 6

TABLE 3

The effects of the various resistive conditions on the velocity error score. See Table 2 legend.

VELOCITY ERROR SCORES

	LOWER	NO CHANGE	HIGHER
MOD. VISCOSITY			
PATIENTS — AFFECTED ARM	*****		
PATIENTS — CLINICALLY UNAFFECTED ARM	****	*	
CONTROL SUBJECTS	*****	*	
HIGH VISCOSITY			
PATIENTS — AFFECTED ARM	*****		
PATIENTS — CLINICALLY UNAFFECTED ARM	*****		
CONTROL SUBJECTS	******		
MOD. STIFFNESS			
PATIENTS — AFFECTED ARM	**	***	
PATIENTS — CLINICALLY UNAFFECTED ARM	**	*	**
CONTROL SUBJECTS	*	*****	
HIGH STIFFNESS			
PATIENTS — AFFECTED ARM	**	***	
PATIENTS — CLINICALLY UNAFFECTED ARM	*	***	*
CONTROL SUBJECTS		******	
MOD. INERTIA			
PATIENTS — AFFECTED ARM		*****	
PATIENTS — CLINICALLY UNAFFECTED ARM		****	*
CONTROL SUBJECTS	*	*****	
HIGH INERTIA			
PATIENTS — AFFECTED ARM	**	**	*
PATIENTS — CLINICALLY UNAFFECTED ARM		****	*
CONTROL SUBJECTS		*****	*

EXPLANATION OF MEASUREMENTS FROM VELOCITY RECORDINGS

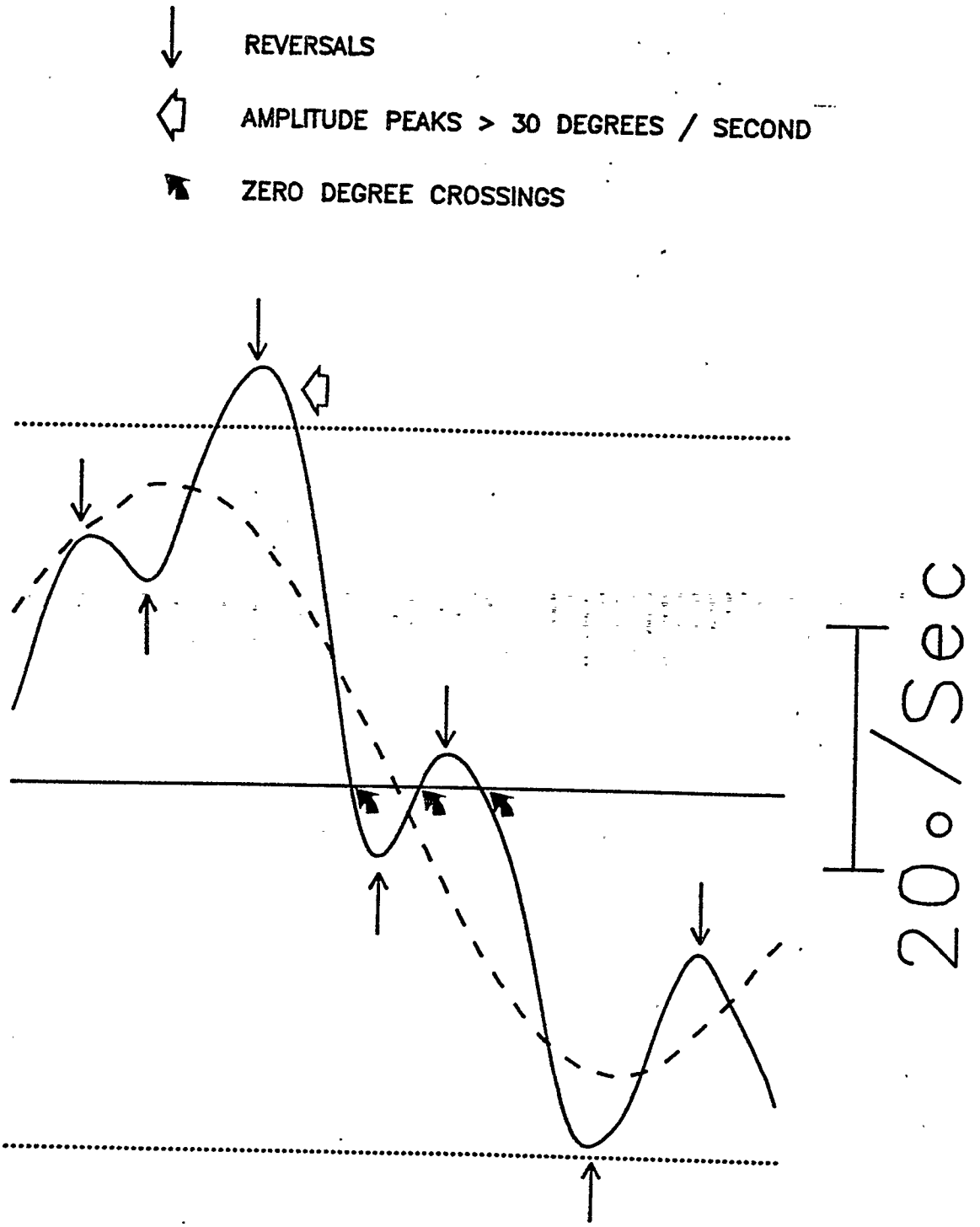


FIGURE 13

As described in figure 12, the number of velocity peaks with an amplitude greater than 30 degrees from zero were counted for each 20 second trial. For each subject population the mean number of high amplitude peaks for each of the subjects' 28 unloaded trials were averaged. Standard error bars are shown. The results of the patients' arms were compared to the control subjects.

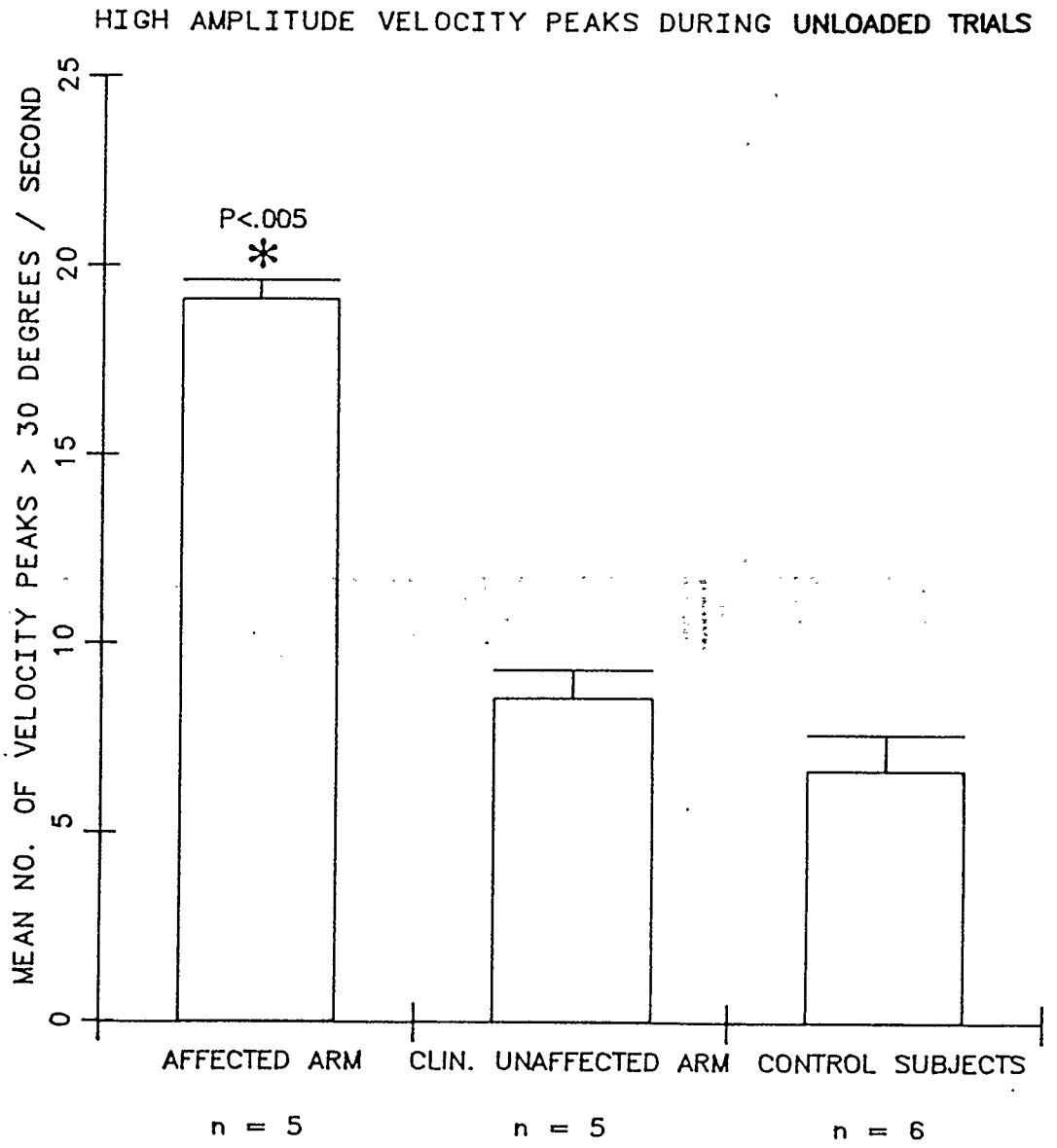

HIGH AMPLITUDE VELOCITY PEAKS DURING UNLOADED TRIALS
MEAN NO. OF VELOCITY PEAKS > 30 DEGREES / SECOND
0
5
10
15
20
25
P<.005
*
AFFECTED ARM
CLIN. UNAFFECTED ARM
CONTROL SUBJECTS
n = 5
n = 5
n = 6

FIGURE 14

As described in Figure 12, the velocity reversals were counted for each 20 second trial. The mean number of reversals for each subject during the 28 unloaded trials were averaged with the results from the other subjects in the same population. Standard error bars are used. Although there was a tendency for fewer reversals in the velocity recording of the control subjects, this difference was not significant.

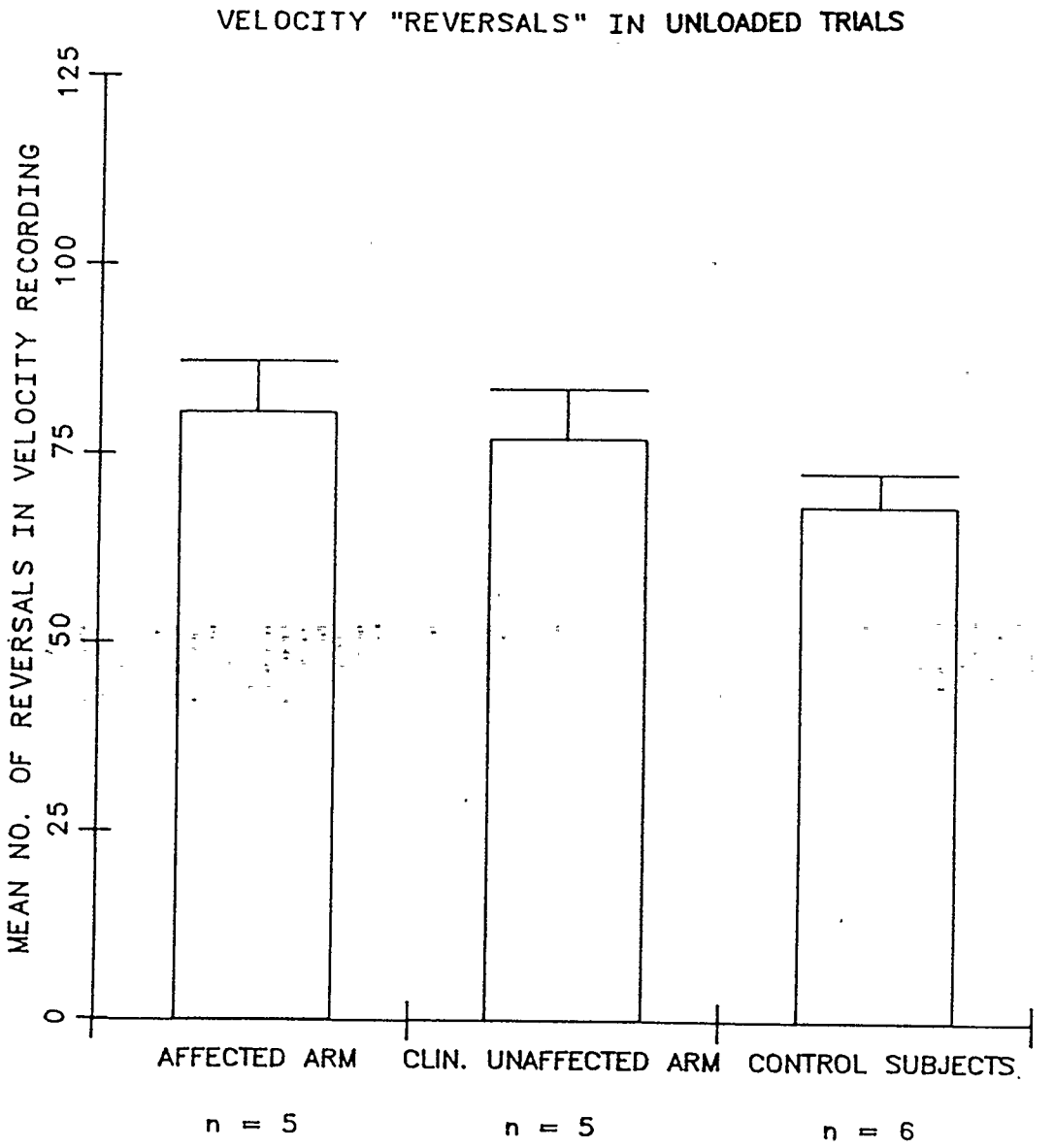

VELOCITY "REVERSALS" IN UNLOADED TRIALS
MEAN NO. OF REVERSALS IN VELOCITY RECORDING
0
25
50
75
100
125
AFFECTED ARM
CLIN. UNAFFECTED ARM
CONTROL SUBJECTS
n = 5
n = 5
n = 6

FIGURE 15

As described in figure 12, the number of zero crossings were counted during each 20 second trial. For each tracking trial, the target crossed the zero degree line 14 times; this number was subtracted from the total number of zero crossings of the cursor for each tracking trial to determine the number of inappropriate zero crossings. The mean number of inappropriate zero crossings for each subject during the 28 unloaded trials were averaged with the means from the other subjects in the same population.

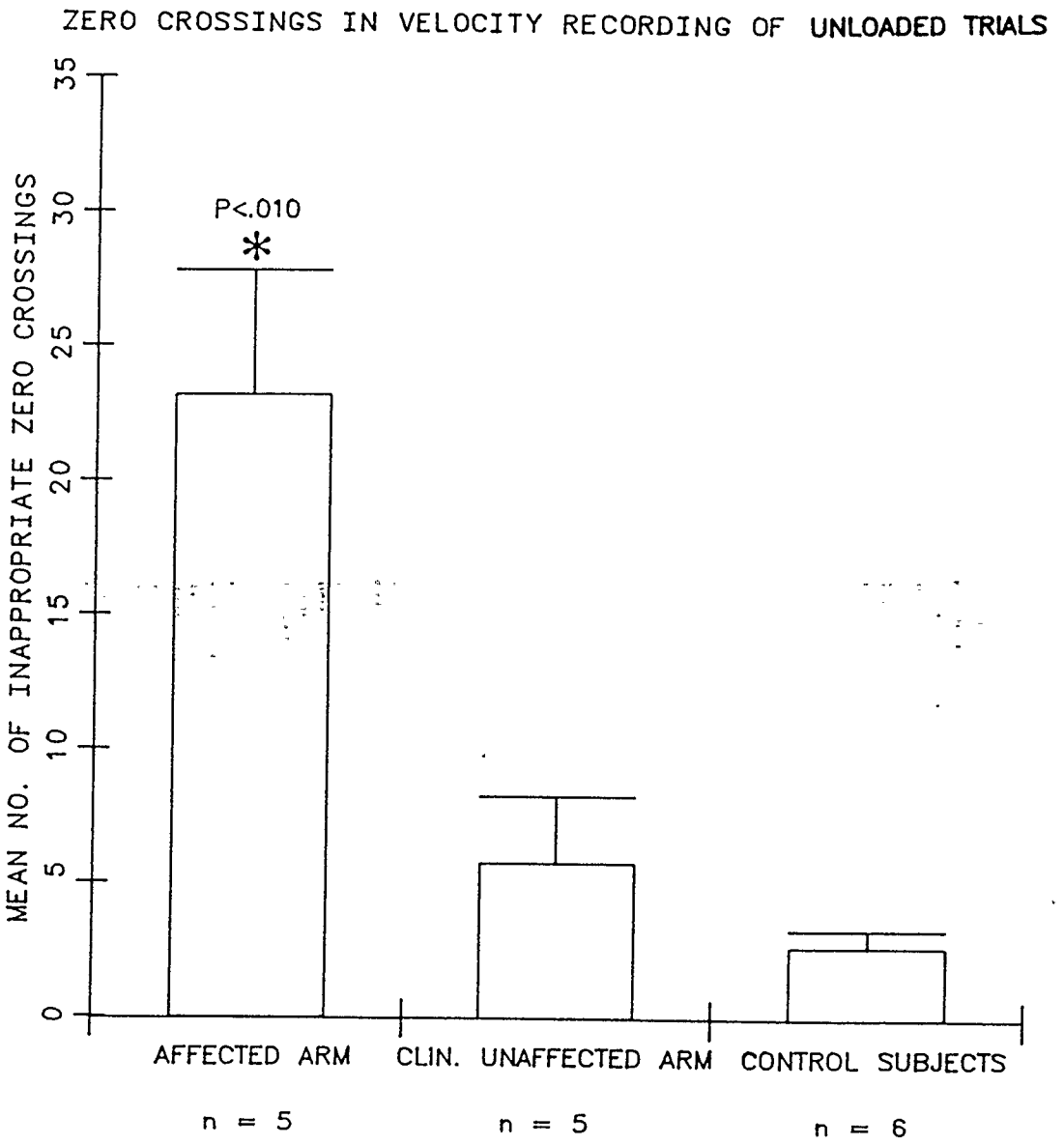

ZERO CROSSINGS IN VELOCITY RECORDING OF UNLOADED TRIALS
MEAN NO. OF INAPPROPRIATE ZERO CROSSINGS
0
5
10
15
20
25
30
35
P<.010
*
AFFECTED ARM
CLIN. UNAFFECTED ARM
CONTROL SUBJECTS
n = 5
n = 5
n = 6

TABLE 4

The effects of the various resistive conditions on the number of velocity peaks > 30 degrees/second. See Table 2 legend.

NO. OF VELOCITY PEAKS GREATER THAN 30 DEGREES/SECOND

	GREATER	NO CHANGE	FEWER
MOD. VISCOSITY			
PATIENTS — AFFECTED ARM			*****
PATIENTS — CLINICALLY UNAFFECTED ARM		***	**
CONTROL SUBJECTS		**	***
HIGH VISCOSITY			
PATIENTS — AFFECTED ARM			*****
PATIENTS — CLINICALLY UNAFFECTED ARM			*****
CONTROL SUBJECTS		*	*****
MOD. STIFFNESS			
PATIENTS — AFFECTED ARM	*	***	*
PATIENTS — CLINICALLY UNAFFECTED ARM	*	**	**
CONTROL SUBJECTS		*****	*
HIGH STIFFNESS			
PATIENTS — AFFECTED ARM	**	**	*
PATIENTS — CLINICALLY UNAFFECTED ARM		****	*
CONTROL SUBJECTS		******	
MOD. INERTIA			
PATIENTS — AFFECTED ARM		**	***
PATIENTS — CLINICALLY UNAFFECTED ARM		***	**
CONTROL SUBJECTS		**	***
HIGH INERTIA			
PATIENTS — AFFECTED ARM		**	***
PATIENTS — CLINICALLY UNAFFECTED ARM			*****
CONTROL SUBJECTS		**	****

TABLE 5

The effects of the various mechanical loading conditions on the number of reversals in the velocity trace. See Table 2 legend.

NO. OF REVERSALS IN VELOCITY TRACES

	GREATER	NO CHANGE	FEWER
MOD. VISCOSITY			
PATIENTS — AFFECTED ARM		*	****
PATIENTS — CLINICALLY UNAFFECTED ARM			*****
CONTROL SUBJECTS		****	**
HIGH VISCOSITY			
PATIENTS — AFFECTED ARM		*	****
PATIENTS — CLINICALLY UNAFFECTED ARM		*	****
CONTROL SUBJECTS		**	****
MOD. STIFFNESS			
PATIENTS — AFFECTED ARM	*	****	
PATIENTS — CLINICALLY UNAFFECTED ARM	*****		
CONTROL SUBJECTS	**	****	
HIGH STIFFNESS			
PATIENTS — AFFECTED ARM	****	*	
PATIENTS — CLINICALLY UNAFFECTED ARM	**	***	
CONTROL SUBJECTS	****	**	
MOD. INERTIA			
PATIENTS — AFFECTED ARM		**	***
PATIENTS — CLINICALLY UNAFFECTED ARM		**	***
CONTROL SUBJECTS		****	**
HIGH INERTIA			
PATIENTS — AFFECTED ARM		*	****
PATIENTS — CLINICALLY UNAFFECTED ARM		**	***
CONTROL SUBJECTS		*****	*

TABLE 6

The effects of the various loading conditions on the number of zero crossings of the velocity trace. See Table 2 legend.

NO. OF ZERO CROSSINGS IN VELOCITY TRACE

	GREATER	NO CHANGE	FEWER
MOD. VISCOSITY			
PATIENTS — AFFECTED ARM		*	****
PATIENTS — CLINICALLY UNAFFECTED ARM		**	***
CONTROL SUBJECTS		******	
HIGH VISCOSITY			
PATIENTS — AFFECTED ARM			*****
PATIENTS — CLINICALLY UNAFFECTED ARM		*	****
CONTROL SUBJECTS		****	**
MOD. STIFFNESS			
PATIENTS — AFFECTED ARM	***	**	
PATIENTS — CLINICALLY UNAFFECTED ARM	*	****	
CONTROL SUBJECTS	*	*****	
HIGH STIFFNESS			
PATIENTS — AFFECTED ARM	****	*	
PATIENTS — CLINICALLY UNAFFECTED ARM	*	****	
CONTROL SUBJECTS	**	****	
MOD. INERTIA			
PATIENTS — AFFECTED ARM		****	*
PATIENTS — CLINICALLY UNAFFECTED ARM		*****	
CONTROL SUBJECTS		******	
HIGH INERTIA			
PATIENTS — AFFECTED ARM		***	**
PATIENTS — CLINICALLY UNAFFECTED ARM		*****	
CONTROL SUBJECTS		******	

FIGURE 16

The EMG activity for each subject was calibrated by having the subject hold their wrist in a neutral flexion/extension position while a 0.45 Nm torque was imposed on the wrist flexors and extensors, alternately, for 5 seconds each.

These portions of the EMG recordings of 2 control subjects are shown with their coactivation indices (calculated over 20 seconds) to demonstrate the appropriateness of the coactivation index for the amount of EMG activity present. The dashed line is the point of reference for both the flexor and extensor EMG.

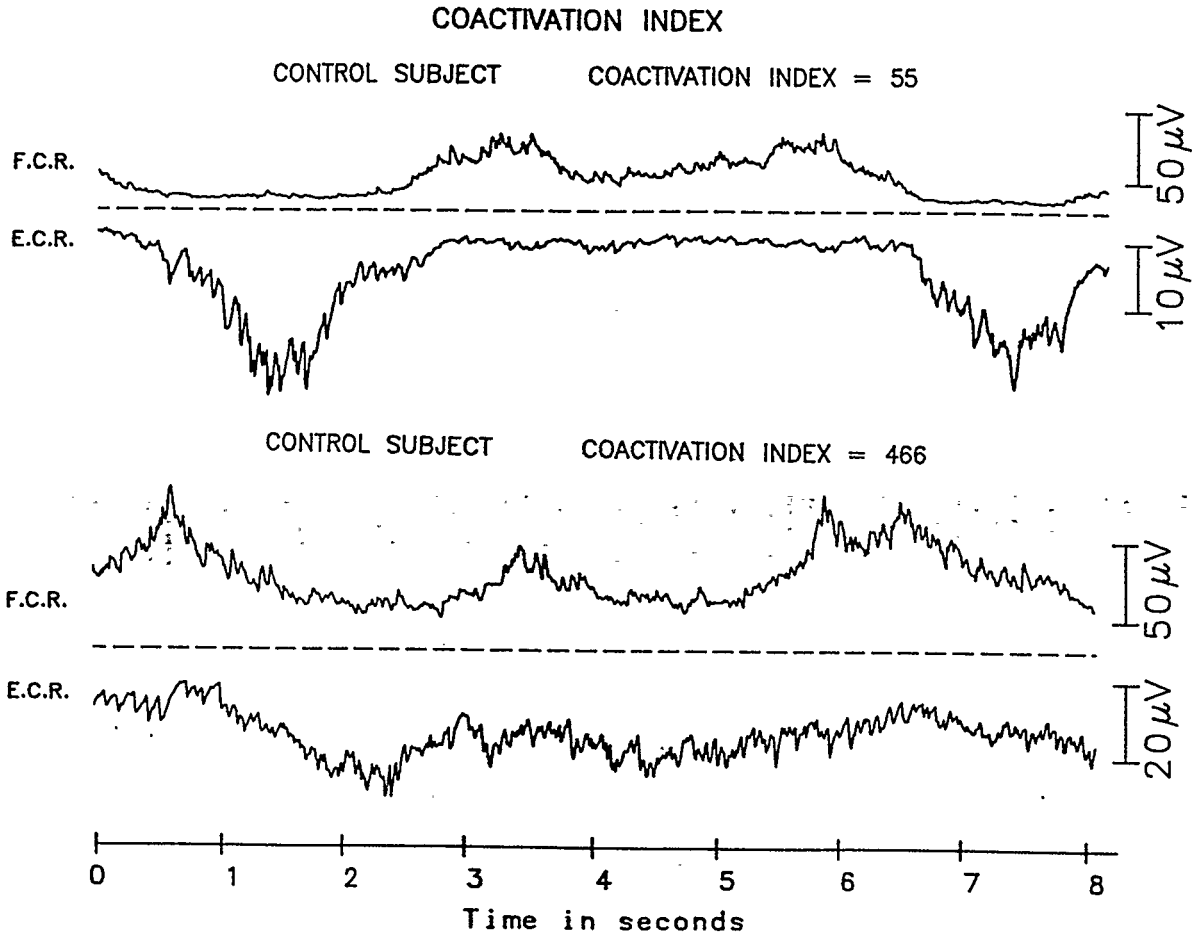

COACTIVATION INDEX
CONTROL SUBJECT
COACTIVATION INDEX = 55
F.C.R.
E.C.R.
50 μV
10 μV
CONTROL SUBJECT
COACTIVATION INDEX = 466
F.C.R.
E.C.R.
50 μV
20 μV
0
1
2
3
4
5
6
7
8
Time in seconds

FIGURE 17

A portion of the 20 second electromyography recordings for a control subject and the affected arm of a patient with a right cerebellar astrocytoma. The corresponding cocativation index and position recordings are shown. The dashed line between the flexor and extensor EMG's is the point of reference for both of these traces; the point of reference for the coactivation index is the time line.

ELECTROMYOGRAPHY

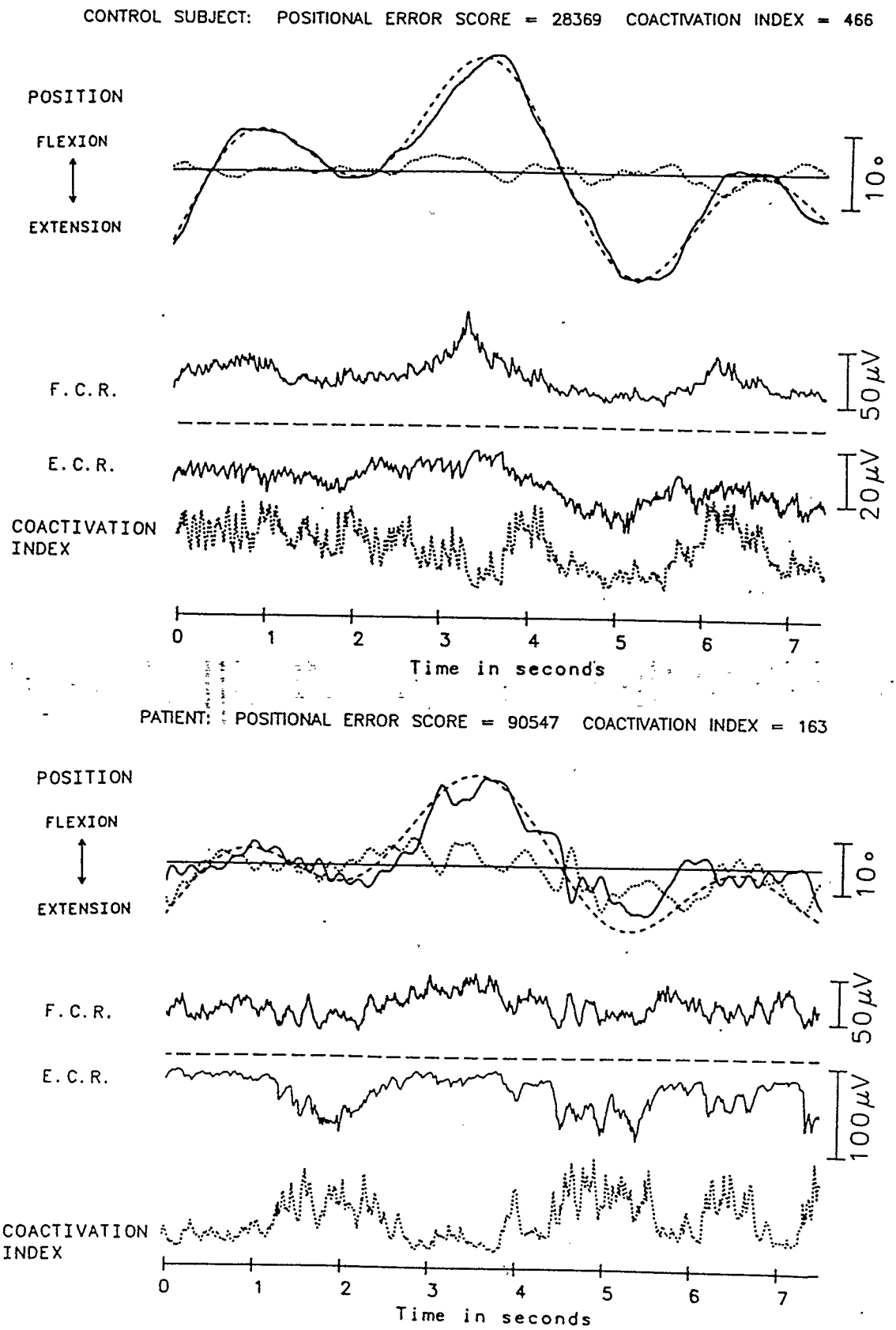


FIGURE 18

The mean coactivation index for each subject during the 28 unloaded trials were averaged with the means from the other subjects in the same population to produce these bars with their standard error. Although there was a tendency for less coactivation in the patients' affected arms, this difference was not significant.

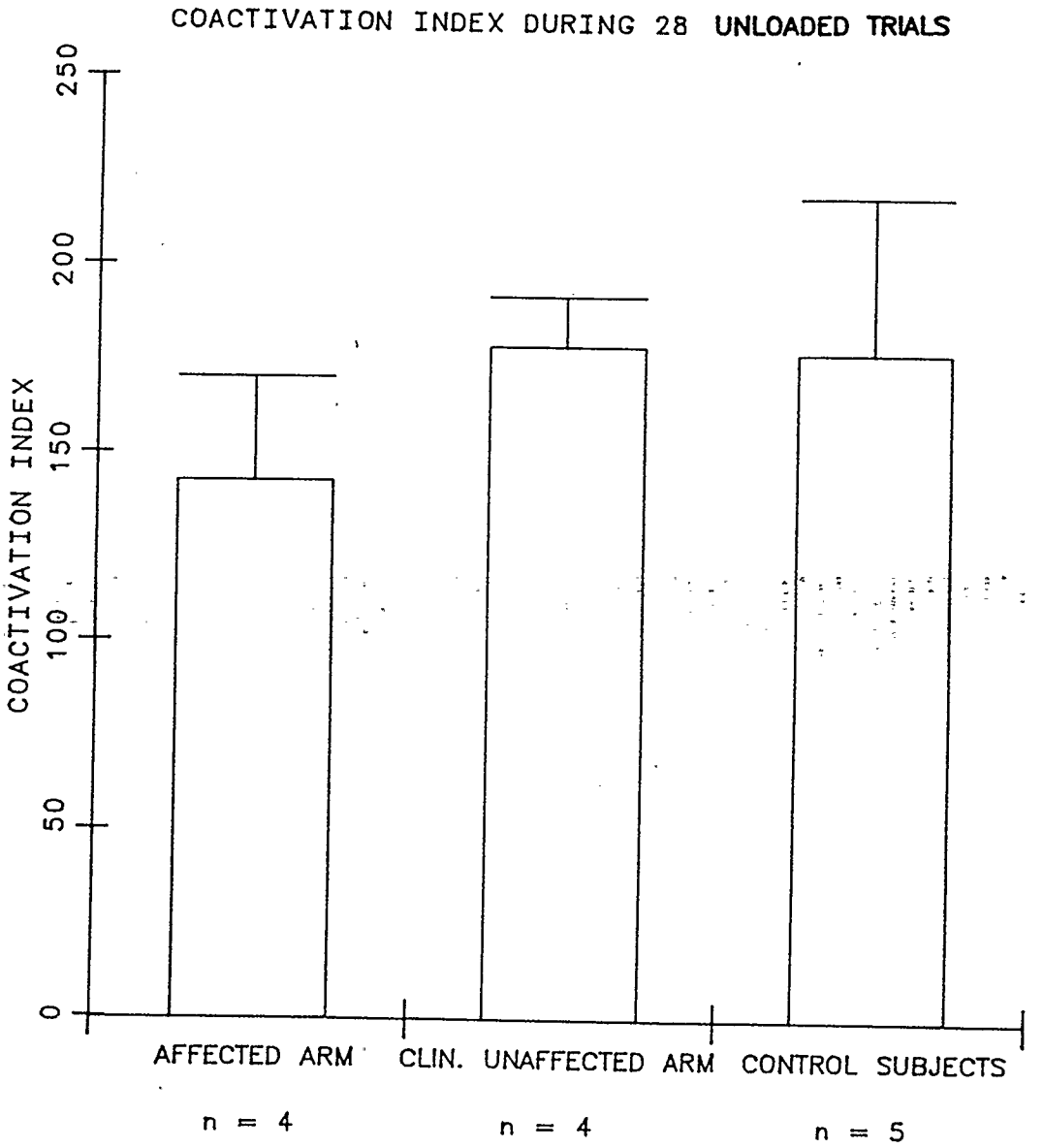

COACTIVATION INDEX DURING 28 UNLOADED TRIALS
COACTIVATION INDEX
0
50
100
150
200
250
AFFECTED ARM
CLIN. UNAFFECTED ARM
CONTROL SUBJECTS
n = 4
n = 4
n = 5

TABLE 7

The effects of the various loading conditions on the Coactivation Index. See Table 2 legend.

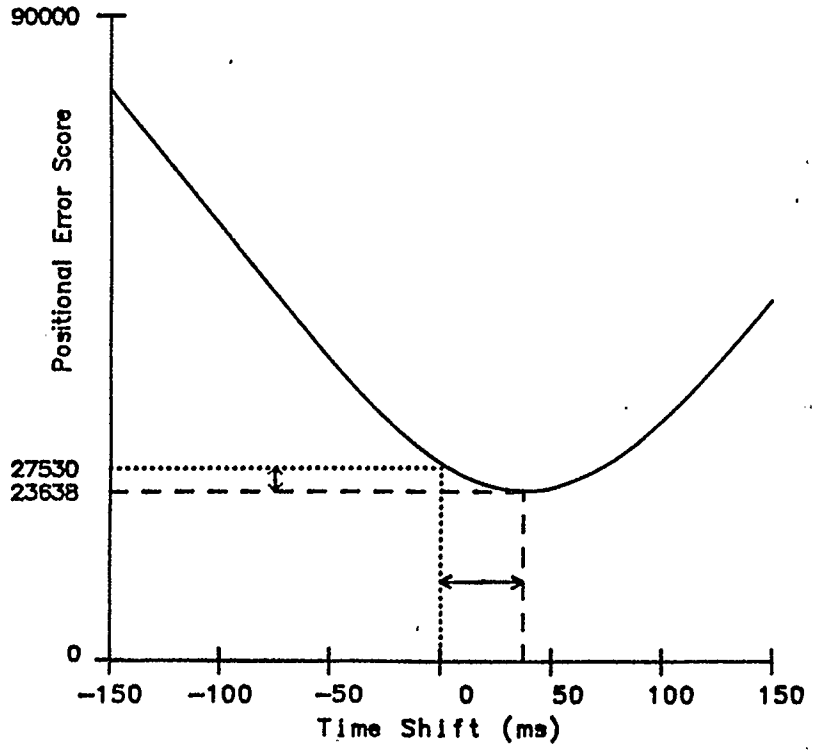
COACTIVATION INDEX

	LOWER	NO CHANGE	HIGHER
MOD. VISCOSITY			
PATIENTS — AFFECTED ARM	*	*	***
PATIENTS — CLINICALLY UNAFFECTED ARM	**	***	
CONTROL SUBJECTS	***	**	*
HIGH VISCOSITY			
PATIENTS — AFFECTED ARM	*	*	***
PATIENTS — CLINICALLY UNAFFECTED ARM	**	*	**
CONTROL SUBJECTS	***	*	**
MOD. STIFFNESS			
PATIENTS — AFFECTED ARM		***	**
PATIENTS — CLINICALLY UNAFFECTED ARM		****	*
CONTROL SUBJECTS	*	**	***
HIGH STIFFNESS			
PATIENTS — AFFECTED ARM		*	****
PATIENTS — CLINICALLY UNAFFECTED ARM	*	***	*
CONTROL SUBJECTS	*	**	***
MOD. INERTIA			
PATIENTS — AFFECTED ARM	**	*	**
PATIENTS — CLINICALLY UNAFFECTED ARM		****	*
CONTROL SUBJECTS		*****	*
HIGH INERTIA			
PATIENTS — AFFECTED ARM		****	*
PATIENTS — CLINICALLY UNAFFECTED ARM		***	**
CONTROL SUBJECTS		****	**

FIGURE 19

To examine the amount of phase lag present during the tracking trials, the target position was shifted horizontally on the cursor position to produce the lowest positional error score. An example of the time shift required for a control subject and a patient are shown.

Control Subject



Cerebellar Hemangioblastoma

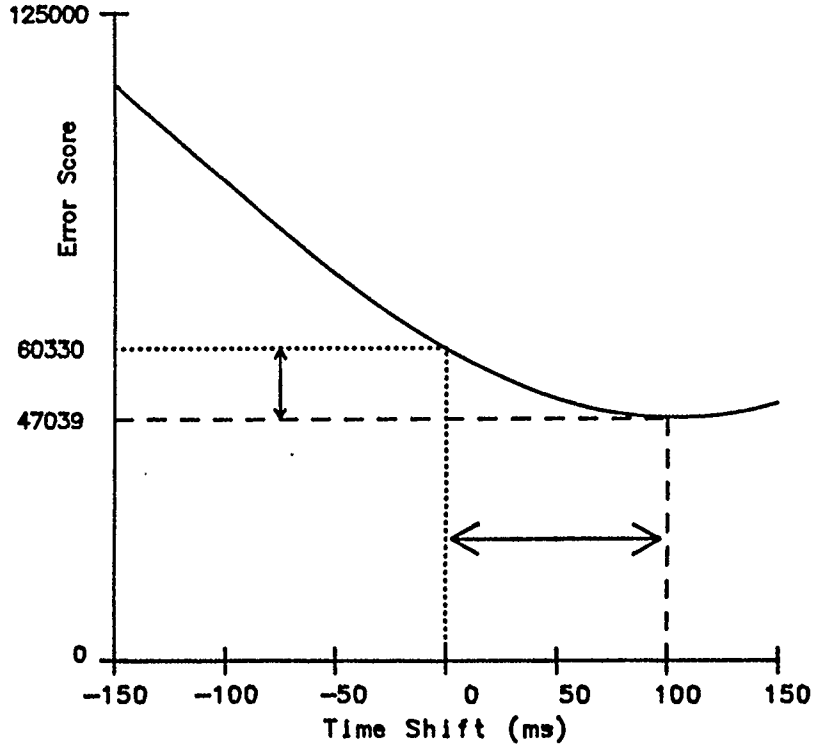


FIGURE 20

The mean time shift required to produce the lowest positional error scores during unloaded trials in each subject were averaged for the subjects in each population. The results of the patients' arms are compared with the control subjects, and standard error bars are used.

TIME SHIFT REQUIRED TO PRODUCE LOWEST POSITIONAL ERROR SCORES

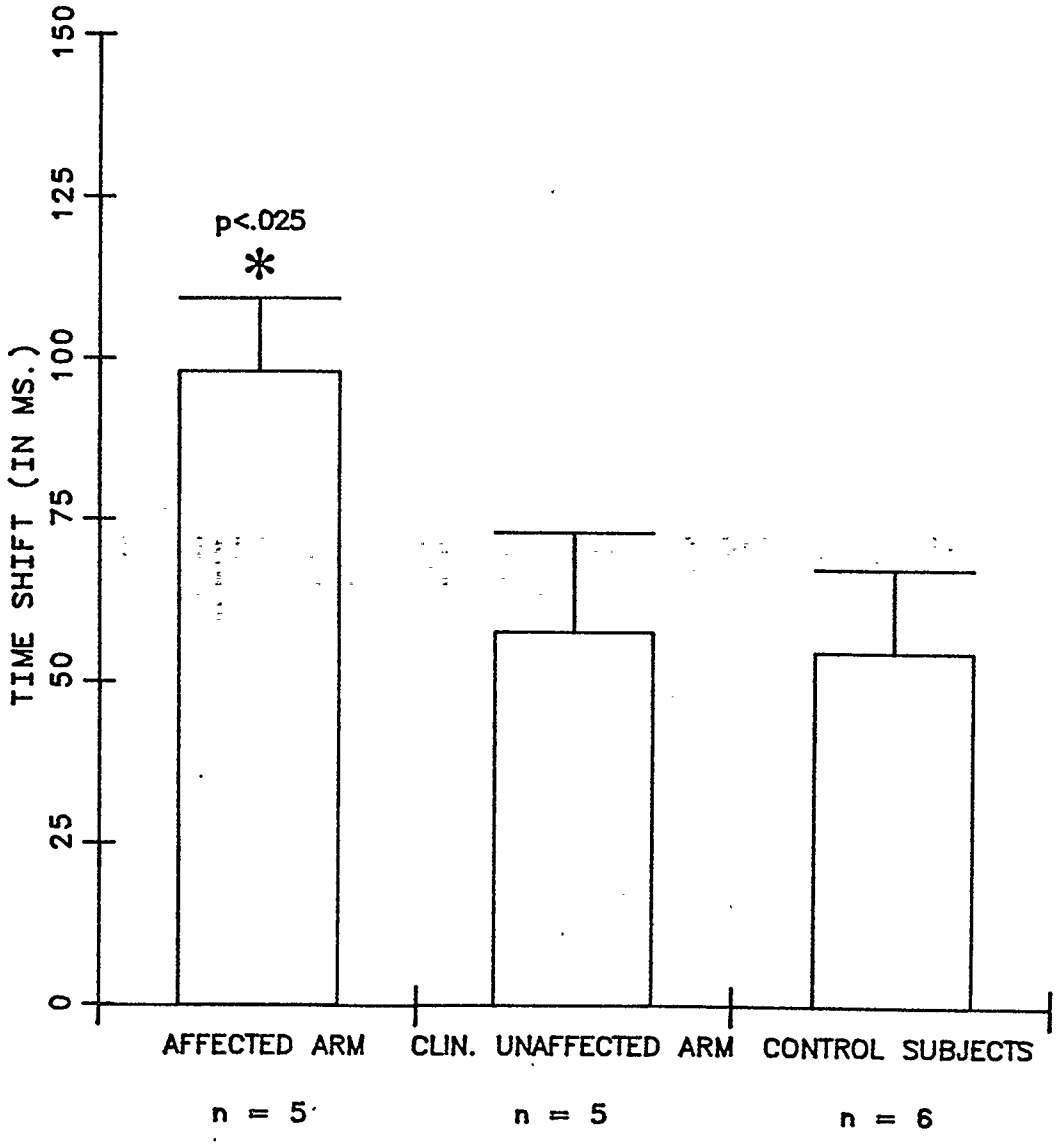


FIGURE 21

The mean amount of improvement in the positional error score produced by the time shift during the unloaded trials for each subject were averaged for the subjects in each population.

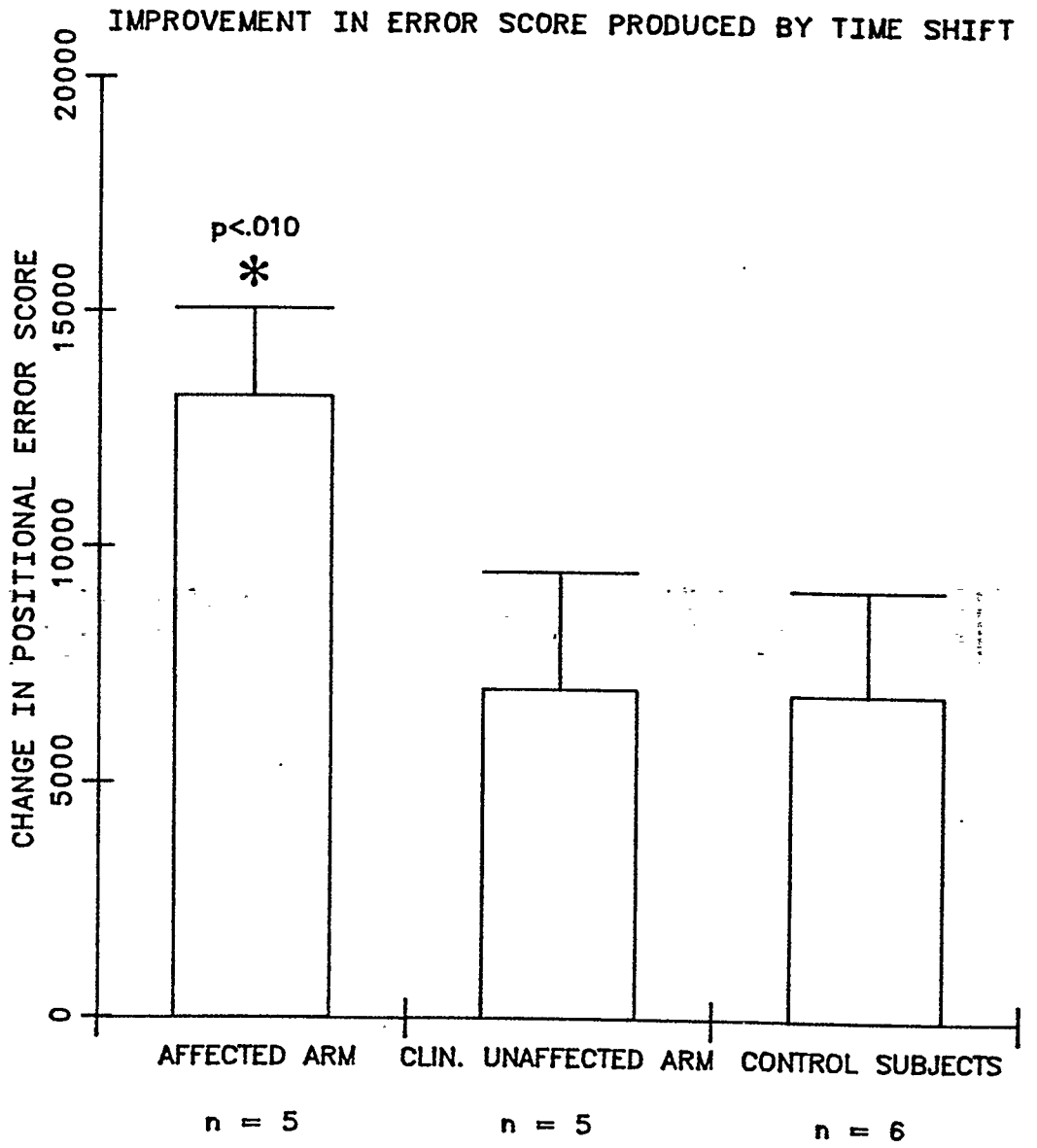

IMPROVEMENT IN ERROR SCORE PRODUCED BY TIME SHIFT
CHANGE IN POSITIONAL ERROR SCORE
20000
15000
10000
5000
0
p<.010
*
AFFECTED ARM
CLIN. UNAFFECTED ARM
CONTROL SUBJECTS
n = 5
n = 5
n = 6

FIGURE 22

A normal 29 y.o. male subject performed 45 tracking trials in the unloaded condition to examine the effects of learning on the positional error score.

UNLOADED TRACKING TRIALS BY A NORMAL MALE

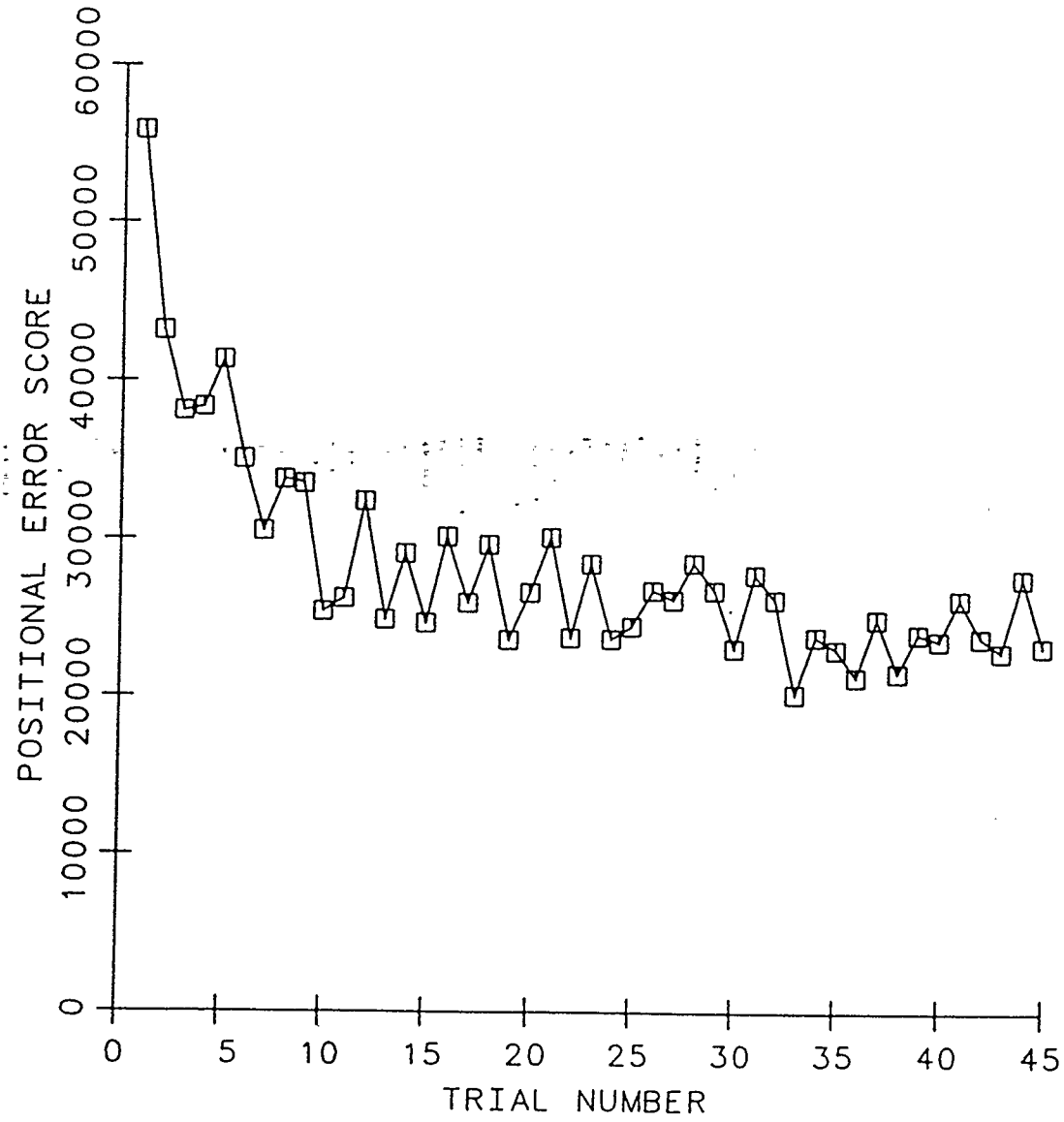


FIGURE 23

Self-paced sinusoidal tasks, without a visual display of the target or cursor, were performed by the subjects in the unloaded condition. A portion of a 20 second trial for a normal subject and a patient with a unilateral cerebellar infarct are displayed.

SELF–PACED SINUSOIDAL TASK WITHOUT VISUAL FEEDBACK

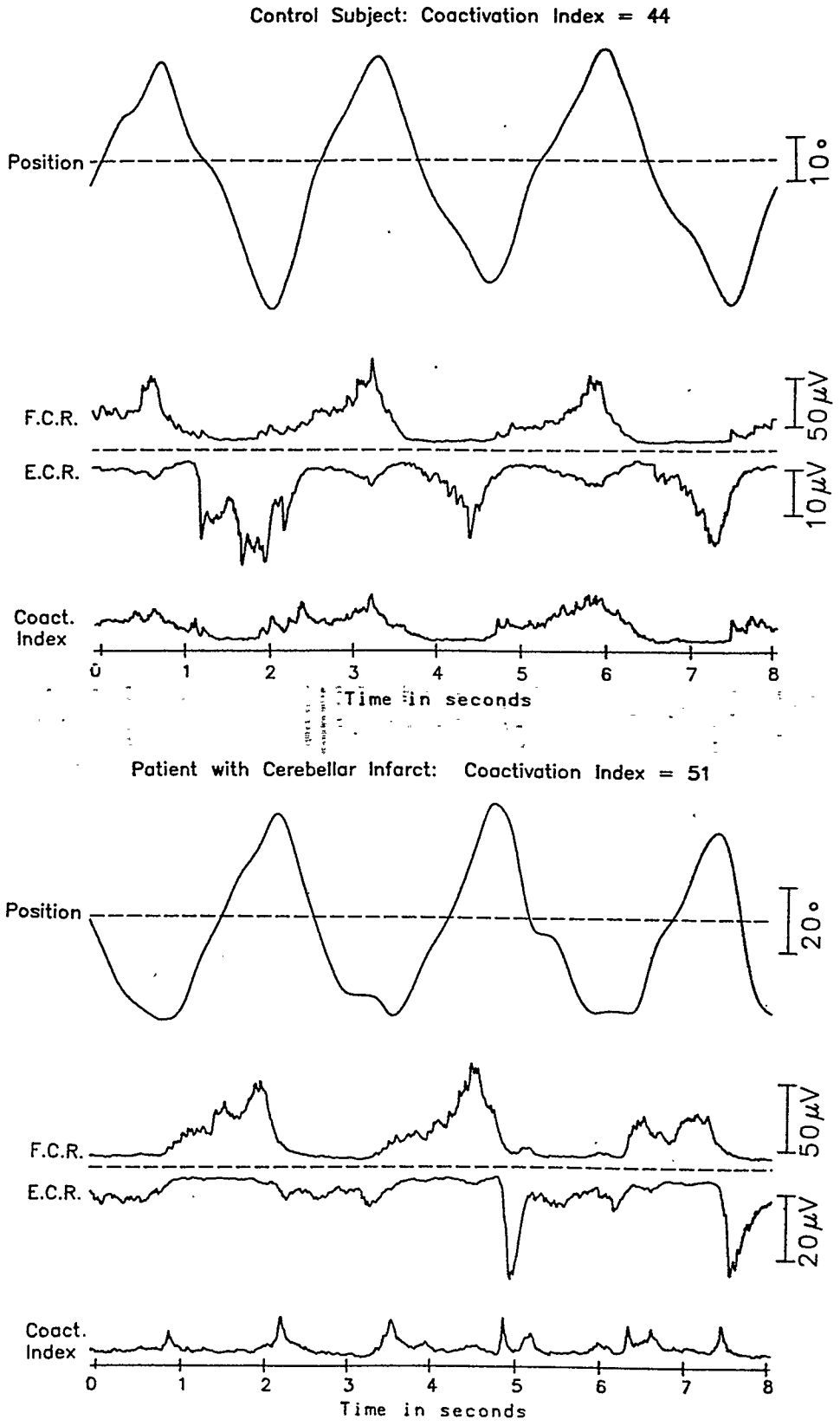


FIGURE 24

The wrist of a control subject was perturbed with a torque pulse while the subject was instructed to not intervene. The amplitude peaks of the oscillation produced in the position trace are plotted against time to show that this oscillation can be modelled by a second order linear system.

Oscillations Produced by Torque Perturbation at Wrist

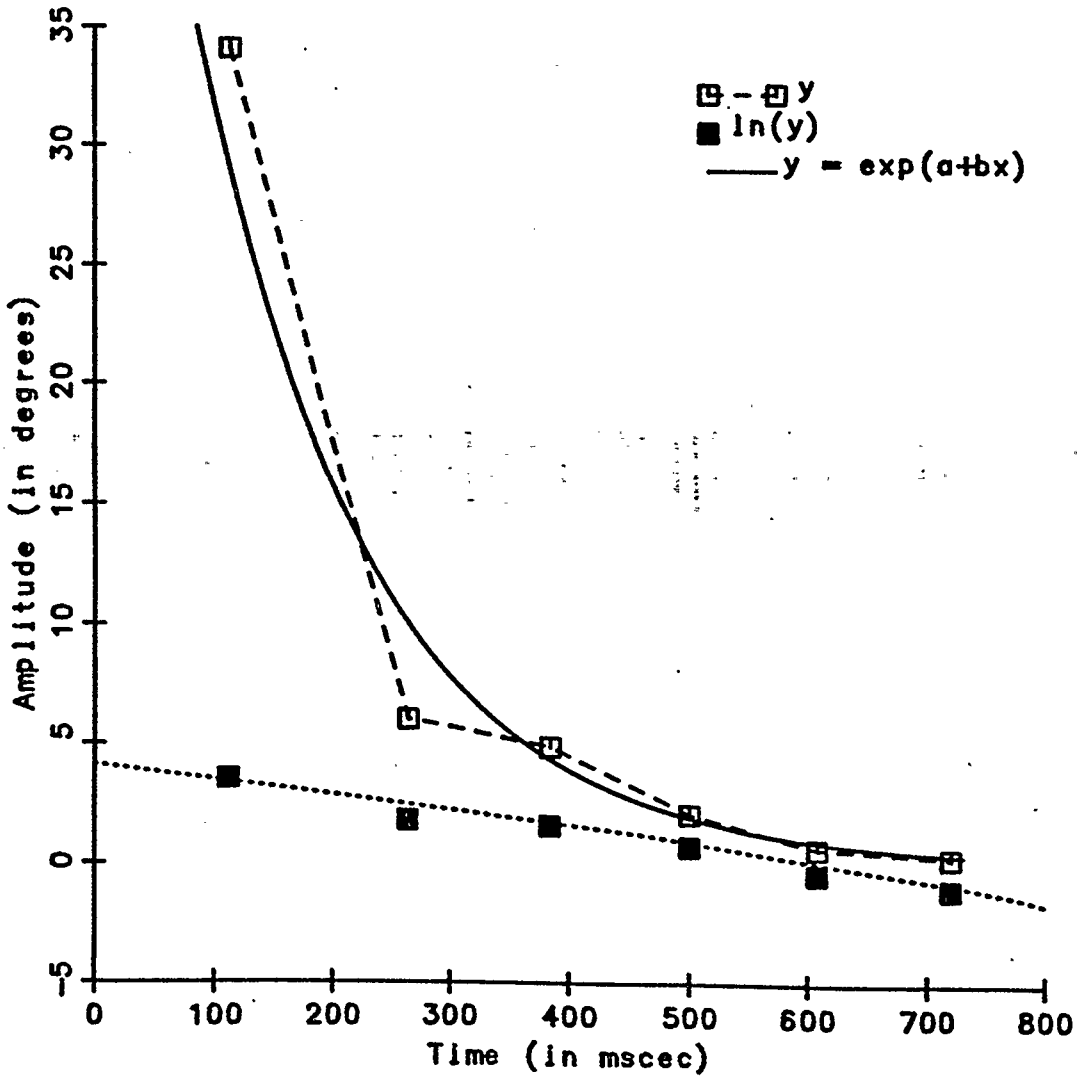
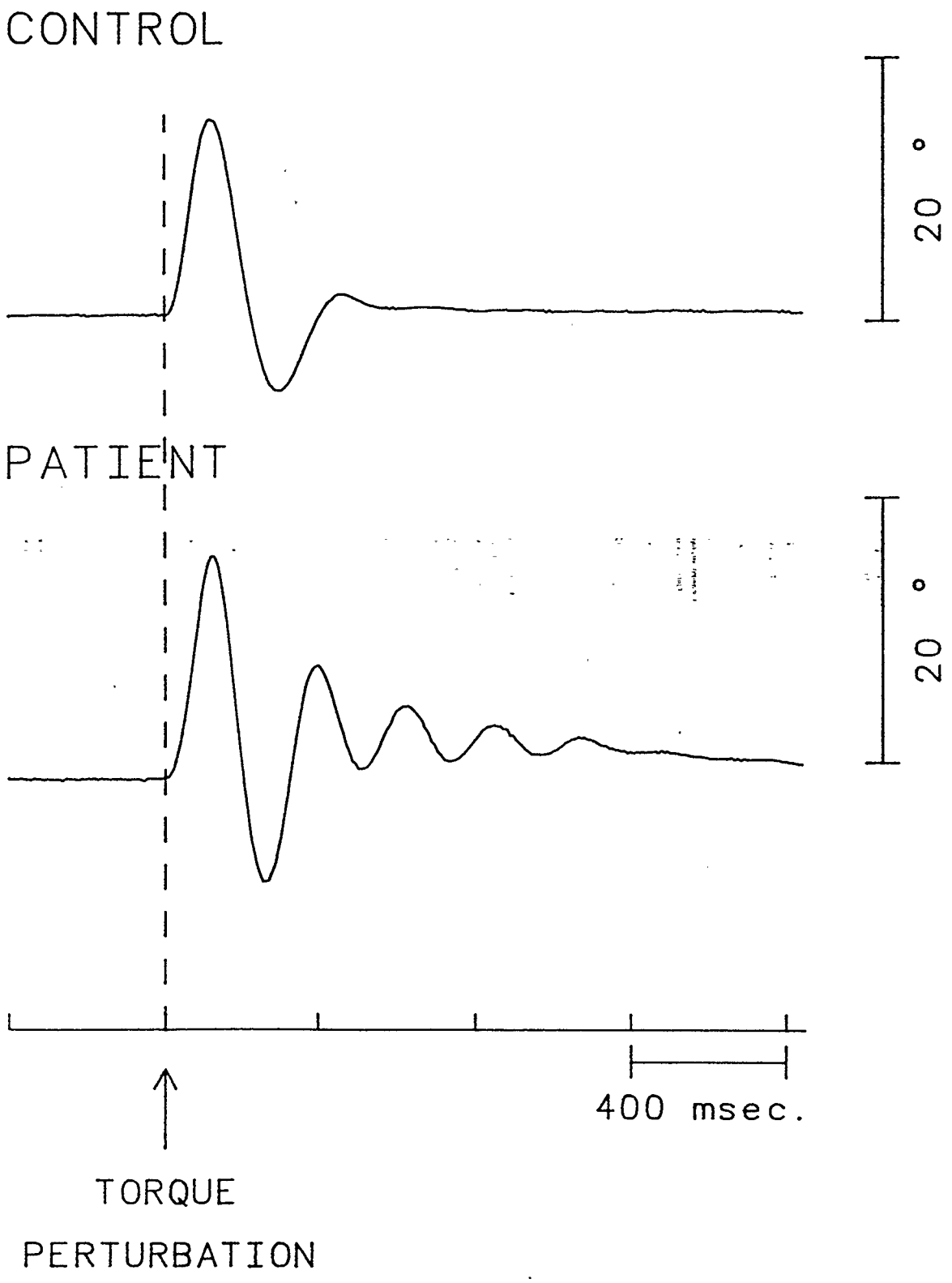


FIGURE 25

Typical oscillations of the position recording produced by a torque perturbation of the wrist in the extensor direction. A single sweep is shown for a control subject and a patient with cerebellar dysfunction.


CONTROL
20 °
PATIENT
20 °
400 msec.
TORQUE
PERTURBATION

DISCUSSION

1. SUMMARY OF PRINCIPAL RESULTS

Manual tracking performance was examined in patients with cerebellar lesions and normal subjects as the mechanical properties of the system were altered. The purpose of this project was to determine if altering the mechanical loading of the wrist might provide therapeutic benefit to these patients, and also to enhance understanding of the mechanisms responsible for cerebellar incoordination.

The principal findings of this research project are as follows:

1. Increased viscosity improves tracking accuracy in the affected arm of patients with unilateral cerebellar lesions.
2. When cerebellar patients track a target, the velocity of their movement changes more frequently and reaches higher velocity peaks than control subjects.
3. There is no significant difference in the amount of coactivation of agonist and antagonist muscles employed by cerebellar patients and control subjects during tracking.
4. Cerebellar patients track a pseudo-random target with a greater time lag than control subjects.
5. When visual feedback is eliminated and subjects are required to perform movements of similar amplitude and velocity as the tracking task, there are fewer irregularities of position and velocity.
6. In response to a torque perturbation, there is less damping in the wrist of cerebellar patients than control subjects.

1.1 TRACKING ACCURACY

Both the control subjects and cerebellar patients tracked the pseudo-random target discontinuously, with corrective positional adjustments. This pattern of tracking has been described previously (Miall et al. 1985 and 1987, Beppu 1984). Patients with unilateral cerebellar lesions produced greater positional error during tracking with their affected arms than with their clinically unaffected arms. The error was also greater than what was observed in the control subjects.

Addition of moderate or high levels of viscosity consistently improved the tracking accuracy in the affected arms of these patients, but not in their unaffected arms nor in the control subjects. Application of viscosity during a functional activity by patients with cerebellar dysfunction has also been investigated by one other group (Riley and Rosen 1987), who did not find that it consistently led to improvement in tracking. The tremor-disabled subjects that were used in their study had cerebellar dysfunction from a variety of different causes, including head injuries, multiple sclerosis, and cerebral palsy. The ten patients examined in our study who had diffuse cerebellar dysfunction due to various causes (besides the 5 patients with lesions restricted to one cerebellar hemisphere) also did not show improvement in tracking accuracy with increased viscosity.

A possible explanation for the differences found between these two groups of patients in their response to increased viscosity could be that the mechanisms responsible for their incoordination are not the same. On clinical examination, the patients with unilateral cerebellar lesions exhibited hypotonia or normal tone; whereas the other patients with cerebellar dysfunction often had concomitant signs

such as spasticity and/or weakness, as their pathology was not restricted to the cerebellum. As well, the types of tremor exhibited in these other conditions could be of a different origin than the intention tremor of the patients with lesions restricted to one cerebellar hemisphere. For example, some of the patients had essential tremor which has been shown by Elble (1987) to be generated by a central oscillator. This research project was not designed to determine the origin of tremor in patients with cerebellar lesions; however, from results which will be discussed below, it is possible that the passive mass-spring limb properties do play a role in maintaining intention tremor (Joyce, Rack 1974, Lakie et al. 1986, Rietz, Stiles 1974, Stiles 1983).

As stated, the addition of viscosity did not improve accuracy in the patients' clinically unaffected arms nor in the control subjects. As will be discussed in section 1.6, Figure 25 reveals that there is less damping in the wrist of the cerebellar patient than in the control subject. Therefore, the normal wrist is probably adequately damped, and increasing this damping further is of no benefit to tracking accuracy. Some of the control subjects did report that the tracking task seemed easier with the viscous resistance. This could mean that less antagonist muscle activity was required when tracking with viscosity; however the coactivation index does not consistently decrease in the control subjects when viscosity is applied.

Previous studies (Hewer et al. 1972, Morgan 1975a,b) have shown that the use of weighted wrist cuffs decreased intention tremor in humans. In this research project, the addition of inertia to the manipulandum either had no effect on tracking accuracy, or it increased the amount of positional error. Angular frequency is

inversely proportional to mass (Elble 1987), therefore, an increased mass should improve tracking performance; however, an increase in mass also results in decreased damping of a second order linear system (Figure 4, equation 4b). Differences in the experimental set-up could contribute to these discrepant findings. Gravity opposed limb movements in these previous studies whereas, in this research project, the limb moved in only one plane where gravity was not a factor. It would be of interest to use a 3-dimensional movement analysis system to examine the effects of increased inertia on freely moving limbs.

1.2 TRACKING VELOCITY

Velocity error scores were significantly higher for the affected arms of the patients than for their clinically normal arms or for the control subjects. By increasing the viscosity of the system the velocity error score was reduced in all subject groups. In order to ascertain the nature of the higher velocity error scores in the patients, other measurements of the velocity recording were obtained. The number of velocity peaks over 30 degrees/sec, reversals, and zero crossings were counted. In his book "Tracking Skill and Manual Control", Poulton (1974) describes counting reversals and zero error crossings as effective quantitative measures of tracking performance.

The velocity recordings for the patients' affected arms had more high amplitude velocity peaks than what was seen in their other arms or in the controls. This is consistent with observations of Miall et al. (1987) during tracking by monkeys with experimental cerebellar lesions. Addition of viscosity decreased the number of high amplitude velocity peaks, which is to be expected. Increased inertia also tended to decrease the number of high amplitude velocity peaks, a

finding which has been previously reported (Hore, Flament 1986, Vilis, Hore 1977). Mathematically, one could predict this relationship between inertia and the peak velocity. In a linear system, when the force driving the system is fixed, the magnitude of the oscillation is inversely proportional to the mass.

The number of reversals in the velocity trace was not significantly different between the patients and the controls. This same finding has been reported by Miall et al. (1987) in a monkey with cooling of the nucleus interpositus. Qualitative observations of the data and a review of related studies suggest that these reversals in the velocity trace are probably due to voluntary corrections of the motor program, and not a result of tremor (Beppu et al. 1987, Miall et al. 1987). Since frequency is directly proportional to stiffness, and inversely proportional to mass (Elble 1987), it is expected that increased stiffness should increase the number of reversals, and increased inertia should decrease the number of reversals. Our results, indeed, show these relationships to be true. Increased viscosity caused a fewer number of reversals of velocity.

The number of times that the velocity trace crossed the zero line was greater in the patients' affected arms than in their unaffected arms or in control subjects. This could have been caused by patients incorrectly predicting the direction of target movement and having to make rapid readjustments, or by patients making inappropriately large corrections for positional errors. Miall et al. (1987) have found that in monkeys with cerebellar lesions the movements become inappropriately large and fast and they must rely on estimates of positional error to control each movement.

1.3 ELECTROMYOGRAPHY

The objectives of the analysis of EMG recording were to determine the following: a) the amount of coactivation used by patients as compared to control subjects during tracking, b) the effects that altered loads have on the amount of coactivation, and c) any changes in the amount of coactivation used while learning the tracking task.

To meet these objectives an index of the amount of coactivation was needed. Other studies have simply used a ratio of the EMG activity of the agonist to the antagonist. In this study we wanted to obtain a value which would express the total amount of EMG activation as well as the level of coactivation, so the formula described in the methods was derived. Interpretation of the results is limited by the fact that the validity of this index has not yet been fully ascertained; however, initial results show that the index varies appropriately with the amount of coactivation present.

Coactivation of the forearm muscles results in an increase in the stiffness and the viscosity of the limb (Lacquaniti et al. 1982). Part of the problem in the cerebellar patients might be reduced viscosity in the limb due to insufficient coactivation of opposing muscle groups, and this could explain why externally applied viscosity improved tracking performance. Although our results showed that the patients did tend to coactivate less than normals during unloaded trials, this difference was not significant. The experimental design which was used to determine the effects of altering mechanical properties on tracking accuracy was not appropriate to address the question of the amount of coactivation used when tracking in the unloaded condition. There was an inadequate number of successive unloaded trials. Unloaded trials were interspersed between loaded

trials, which may have affected the subjects' tracking strategy and EMG activation patterns. Further investigation is required to determine the amount of coactivation used by cerebellar patients and normal subjects during tracking tasks.

The addition of viscosity did not have any consistent effect on the amount of coactivation used by any of the subjects. Increased stiffness or inertia caused either an increase in the coactivation index or no significant change. Hasan (1986) has also observed increased coactivation with inertial loading. Intuitively, one might expect that increased stiffness and inertia might cause more coactivation. Since increased stiffness causes a higher frequency of oscillations and increased inertia causes a decrease in the damping of the system, the subject may coactivate more in these situations in an attempt to dampen the limb.

As stated above, we were interested initially in determining if there was any correlation between the amount of coactivation and learning of the tracking task. However only 10 initial unloaded trials were performed and this was probably an insufficient number to reveal any correlation that might exist between these two factors. To adequately address this question would require a larger number of successive tracking trials, and a training period over a number of days.

Qualitative analysis of the EMG recordings revealed that the patients with cerebellar lesions had more short "bursts" of EMG activity than the control subjects. This was especially evident in the patients with spino-cerebellar degeneration, and in those patients with tremor. These bursts of EMG activity were accompanied by deactivation of the antagonist, causing an unopposed brief fluctuation

in the position and velocity recordings. These EMG bursts have been described in patients with essential and Parkinsonian tremors by Homberg et al. (1987) who attribute them to strong motor unit synchronisation. As a possible basis for tremor, Vilis and Hore (1980) suggest that a disordered servo-controlled neuromuscular reflex loop results in the motor cortex being driven only by feedback from the periphery resulting in a series of alternating stretch reflexes. A greater time resolution of the EMG recordings than was used in this study would be necessary to investigate these EMG bursts more thoroughly.

1.4 PHASE LAG BETWEEN TARGET MOVEMENT AND SUBJECT'S RESPONSE

The time lag between a moving target and the tracking cursor is determined by two factors: a) the ability of the subject to predict the movement of the target (Poulton 1981), and b) the reaction time of the subject. The time it took to react with a wrist movement to a visual cue in cerebellar patients was slower than the control subjects tested. This concurs with the findings of Beppu et al. (1984), Holmes (1917), and Rothwell (1987). In this study, the tracking phase lag was greater in the affected arms of the patients than in their other arms or in the control subjects. Miall et al. (1987) suggest that patients must rely on feedback of the positional error to control each movement, rather than being able to use velocity information in the feedforward control of movements. It is not known to what extent either the increased reaction time or the decreased ability to predict the target movement contribute to creating this increased tracking phase lag.

There is controversy in the literature regarding whether or not patients with Parkinson's disease are able to use a predictive strategy when tracking (Day et al. 1978 vs. Flowers 1978). In this research project there was no significant difference in the phase lag by the Parkinsonian patients as compared to the normal subjects. However, there was a definite trend for the patients with Parkinson's disease to have a greater phase lag; the lack of significance may be due to the fact that only 2 of these patients were tested. This study does not provide sufficient evidence to fully resolve this controversy.

1.5 ELIMINATION OF VISUAL FEEDBACK

It has been suggested that the irregularities which are present during tracking by cerebellar patients are caused by repeated visually-guided error correction responses (Beppu et al. 1987), and that delaying visual feedback can decrease the number of corrective movements (Miall et al. 1985). Contrary findings have been reported by Flament et al. (1984) who observed that removal of visual feedback did not change the tremor in monkeys with cerebellar lesions. To examine the role that visual feedback plays in these discontinuities of position, we had the subjects perform self-paced sinusoidal movements without visual feedback. In this situation, the patients moved their wrists with markedly fewer irregularities of position than during tracking of the pseudo-random target. The control subjects also showed slightly fewer discontinuities of movement, but not to the same extent as the patients. It would be interesting to compare sinusoidal movements without visual feedback to sinusoidal tracking using vision. Humans can follow sinusoidal targets accurately with

little use of visual feedback (Miall et al. 1986), but during pseudo-random tracking tasks limb control is dominated by visual information (Poulton 1981).

These results support the findings of Beppu et al. (1987) and Miall et al. (1985); visual feedback seems to contribute to the positional irregularities during tracking movements.

1.6 DAMPING FACTOR AT THE WRIST

The oscillations produced by perturbing the wrists of control subjects and patients can be described by a second order linear system. Many other studies have also modelled these responses by a second order linear system (Lacquaniti et al. 1982, Lakie 1984, Nichols et al. 1977, Stein and Lee 1981, Oguztorelli and Stein 1976).

Demonstrating that the limb behaves as a second order linear system does not allow any conclusions to be made regarding the origin of tremor. A mass-spring system could maintain an oscillation, but this study has not determined what initiates this perturbation in the system. It has been suggested that the perturbation could be caused by asynchronous contraction of motor units (Rietz, Stiles 1974), ballistocardiogram, other body movements (Joyce, Rack 1974, Lakie et al. 1986), or servo-controlled reflex loop instability (Lippold 1970, Murphy 1975, Nichols et al. 1978, Stein, Oguztorelli 1976, Vilis, Hore 1977 and 1980).

In response to a torque perturbation at the wrist, the cerebellar patients we studied had significantly less damping of their forearms than did the control subjects. Patients with cerebellar lesions often exhibit hypotonia (Gilman et al. 1981), which could explain why their system is underdamped. Since viscosity creates damping, it follows

that when viscosity is applied to an underdamped system this system becomes more critically damped. This is likely the mechanism which causes tracking accuracy in cerebellar patients to be improved with the addition of viscosity.

It would be of interest to apply viscosity to the apparatus in which a cerebellar patient is positioned and then produce a torque perturbation to the wrist. One could expect that the resultant oscillation might approximate that of a control subject. It would also be interesting to apply negative viscosity to the system with a normal subject and observe whether tracking and the response to a perturbation mimic the results of a cerebellar patient. Another study which could extend from this one is to determine the combinations of viscosity and stiffness used by control subjects and by cerebellar patients when tracking. The difference between these values could be added to the apparatus through an external device while the patient is tracking, in order to approximate the mechanical properties of the system of a normal subject.

2. CONCLUSIONS

2.1 In attempting to track a slowly moving target, patients with cerebellar lesions generate excessively high movement velocities.

2.2 Patients with cerebellar lesions exhibit decreased damping of their forearms in response to a perturbation at the wrist. The decreased damping could contribute to their difficulties in controlling velocities of movement.

2.3 Increased viscosity, applied through an external device, improves manual tracking accuracy in patients with

cerebellar lesions. Increased viscosity does not affect tracking performance in normal subjects. This externally applied viscosity could be compensating for inadequate damping in the system of patients with cerebellar lesions.

2.4 Neither increased inertia nor increased stiffness improve tracking accuracy in patients or control subjects.

2.5 When visual feedback was eliminated while subjects performed movements of similar amplitude and velocity as the tracking task, there were fewer undulations of position and velocity. This suggests that faulty corrections for visually detected error contributes to tracking irregularities.

3. SIGNIFICANCE OF THIS RESEARCH PROJECT

The objectives of the research project were to investigate possible therapeutic measures for patients with cerebellar incoordination and to enhance understanding of mechanisms producing dysfunction in these patients. Several factors which may contribute to cerebellar incoordination have been identified.

It has been shown that by increasing the viscosity of a system the tracking accuracy of a cerebellar patient can be improved. There are many functional activities with which these patients have difficulty, which might be improved with the application of a viscous interface. The addition of viscosity to assistive devices already in use by some of these patients might enhance functional abilities, for example: feeding devices, environmental control systems, or an electric wheelchair joystick. Alternatively, an orthosis for the upper extremity could be designed which would provide viscosity at the joints.

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APPENDIX A

CONSENT FORM

RESEARCH PROJECT - Motor control mechanisms in normal humans and patients with motor disorders.

INVESTIGATORS - Dr. R.G. Lee, Dr. W.J. Becker, Dr. G.W. Jason, B.L. Morrice

FUNDING AGENCY - Medical Research Council of Canada, Alberta Heritage Foundation for Medical Research

This consent form is only part of the process of informed consent. It should give you the basic idea of what the research project is about and what your participation will involve. If you would like more details about something mentioned here, or information not included here, you should feel free to ask. Please take the time to read this carefully.

The objectives of the research performed in this laboratory are to study the mechanisms of normal motor control, to determine how these mechanisms are altered by disease or injury of the nervous system, and to use these results in developing more effective methods of rehabilitation of patients with motor dysfunction.

The procedures may include one or more of the following outlined below. The arm may be positioned in an apparatus which provides resistance or disturbance to arm movements. Subjects may be asked to follow a target on a screen by moving their arm. To measure the electrical activity of the muscles, recording discs may be taped onto the surface of the skin; alternatively, sterile needle electrodes may be used for this purpose. This experiment will/will not use needle electrodes. There is a small chance of infection with the needle electrodes, but precautions will be taken to eliminate this problem. Some experiments may involve electrical stimulation of the nerve(s) with a mild current; in this study, electrical stimulation will/will not be used. Movement may be monitored by a camera which records from receivers taped onto the skin. There are no other known risks associated with any of the experimental procedures which will be used.

Participation in this research will involve one attendance, for a maximum of two hours. I understand that I may be asked to participate in subsequent research.

It is not expected that subjects participating in this research will attain any immediate direct benefit from their participation; however, knowledge gained from this research should result in a better understanding of motor dysfunction and methods of its rehabilitation.

The information collected in this research will remain confidential, and only the principle investigators will have access to the subjects name, and their results. Should any of the data be used in presentations or publications, the subjects name will **not** be used.

...2

Your signature on this form indicates that you have understood to your satisfaction the information regarding your participation in the research project and agree to participate as a subject. In no way does this waive your legal rights nor release the investigators, sponsors, or involved institutions from their legal and professional responsibilities. You are free to withdraw from the study at any time without jeopardizing your health care. Your decision regarding participation in this research will not in any way affect your medical treatment. If you have further questions, please contact Dr. R.G. Lee, phone number 270-1260.

(Name)	(Name of Witness)
(Signature of subject, or proxy)	(Signature of Witness)

(Date)