The Objective Assessment of Spasticity in Hemiplegia

by

Bettina von Kampen

A Thesis
Submitted to the Faculty of Graduate Studies
in Partial Fulfillment of the Requirements
for the Degree of

MASTER OF SCIENCE

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THE OBJECTIVE ASSESSMENT OF SPASTICITY IN HEMIPLEGIA

BY

BETTINA VON KAMPEN

A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirements for the degree

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MASTER OF SCIENCE

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Abstract

Spasticity resulting from cerebrovascular accident (CVA) can interfere with normal motor function. The development of objective measurements of spasticity are imperative in order to measure the effectiveness of treatment techniques and medications aimed at reducing spasticity.

Ten subjects with a history of one CVA were recruited for this study. They were age matched to an able bodied control group. Clinical tests included the knee Ashworth scale and ankle clonus. These results were compared with two objective tests: the pendulum test and a passive Ramp and Hold test. The pendulum test was administered with the subjects seated. An electrogoniometer was used to monitor the knee joint angle. EMG electrodes were placed over the quadriceps muscle to measure the stretch evoked reflex activity. The affected limb was released from a fully extended position and allowed to swing freely. Three trials were performed. The parameters derived from this test were: EMG threshold angle, number of oscillations, Relaxation Index (RI) and the decay in amplitude of successive angular excursions. The Ramp and Hold test was also performed with the subjects in a seated position. The EMG electrodes remained configured as they were for the pendulum test. A Kin-Com dynamometer was used to impose the passive knee flexion displacements of 90°. Four test velocities were used: 45°/s, 90°/s, 135°/s and 180°/s. The effect of altering the acceleration of the ramp displacement was also tested. A vibratory stimulus was applied over the ligamentum patellae prior to the ramp displacement at the highest velocity setting. Parameters derived from this test were: EMG threshold angle, EMG latency, and EMG area. A velocity dependent response required an increasing EMG area with velocity and an acceleration dependent response required an increased EMG with acceleration.

There was no velocity dependent response observed in the CVA group, however the CVA group had a lower threshold velocity. There was an acceleration dependency in both the CVA group and the control group. The majority of the control group showed no EMG response during the Ramp and Hold test whereas in the CVA group there was only one subject who showed no EMG response. There was a trend towards vibratory inhibition observed in the control group which was not seen in the CVA group. The results of the pendulum test showed significant differences in threshold angle (lower in CVA), RI (lower in CVA) and number of oscillations (smaller in CVA) between the groups. Phase-plane plots derived from the pendulum test data readily demonstrated the qualitative differences between a spastic and a normal limb.

Significant correlations were found between threshold angle from the pendulum test, threshold velocity from the Ramp and Hold test and the presence of ankle clonus. The Ramp and Hold test and the pendulum test were found to be sensitive objective measures of stretch reflex activity in a CVA population.

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Introduction

Spasticity is often a sequelae to cerebrovascular accident (CVA). It can impair normal motor function, especially gait and upper limb co-ordination. It can interfere with balance and proprioception. The study of spasticity has been ongoing to determine its underlying cause and neurophysiological mechanisms, in the hope of discovering a way of reversing or diminishing its effect. Reducing spasticity due to CVA with the use of medication does not ensure the return of normal tone and function, and therefore such medication is seldom used (Katrak, 1992). Other therapeutic techniques (vibration, positioning, splinting) are more commonly used to decrease tone in spastic muscle. The effectiveness of these techniques is not long lasting. Several definitions of spasticity exist. The most conventional one is the following: "the hyperexcitability of the spinal stretch reflex resulting in a velocity-dependent increase in the tonic stretch reflex with exaggerated tendon jerks" (Lance, 1980). Tonic stretch reflex refers to the reflex contraction of the muscle resulting from the stretch applied to the muscle.

Several mechanisms of spasticity, specifically the component of increased stretch reflexes, have been proposed: an increase in stretch reflex gain (Rack, 1984; Thilmann, 1991), a decrease in stretch reflex threshold (Powers, 1988, 1989; Levin, 1993), increased excitability of the Ia monosynaptic reflex arc (Lee, 1987), decreased presynaptic inhibition of Ia fibers (Iles, 1986; Pierrot-Deseilligny, 1990), intrinsic changes of the muscles (Hufschmidt, 1985) and abnormalities of motor unit discharge (Rosenfalck, 1980; Tang, 1981). Stretch reflexes in spasticity can be elicited in antagonistic muscles during active movement, which is a major factor in the motor disability associated with spasticity (Pierrot-Deseilligny, 1990).

There is a lack of objective, quantifiable measurements for spasticity. Clinically, the tests most often used, such as the Ashworth scale (Ashworth, 1964) require the subjective interpretation of the examiner. These tests measure the change in muscle tone in response to a manually imposed passive movement. The importance of objectively measuring the

increased stretch reflexes associated with spasticity is increasing with the development of new medications and treatment techniques, most of which focus on normalizing muscle tone. This refers to reducing the spasticity to a level which will match that of the unaffected limb. It is presumed that normalizing muscle tone will facilitate normal function of that muscle. Methods to objectively quantify spasticity must be available in order to assess the efficacy of these treatments. The long term assessment of spasticity following CVA will become possible with the development and implementation of such tests.

The assumption has been made by other investigators (Burke, 1970, 1971; Berger, 1988), implied by their choice of subjects, that the underlying mechanisms of spasticity are similar in CVA's, spinal cord injuries, multiple sclerosis, cerebral palsy and other conditions resulting in spasticity. The mechanism of injury or aetiology of the disease is different for each of these diagnoses. The clinical manifestation of spasticity is not the same in these conditions. It was the intention of this study to analyse these responses in a homogenous group of subjects. There is a paucity in the literature of studies using homogenous groups of hemiplegic subjects for analysis. The underlying causes of spasticity may become clearer if the differences in its presentation between patient groups can be determined. The link between the pathology resulting in spasticity and its clinical presentation may help to explain the neurophysiological basis of spasticity.

The purpose of this study was to compare two methods of assessing spasticity (Ramp and Hold test and pendulum test) in subjects with hemiplegia due to cerebrovascular accident (CVA). An objective, repeatable method of measuring spasticity is important in order to study the time course of spasticity following the onset of a CVA and to determine the effectiveness of medication and therapeutic techniques. This study also examined the effect of an acceleration stimulus on the stretch evoked reflex response. The possible underlying neurological mechanisms of spasticity as revealed by the results of this study are discussed.

Review of Literature

Neurophysiological Background

Spasticity has been defined as an exaggerated stretch reflex. The stretch reflex is mediated via a monosynaptic reflex arc involving alpha-motoneurons and Ia afferent fibers (Lloyd, 1943). Muscle spindles are velocity sensitive as they respond to the rate of change in length of the muscle (Matthews, 1972). The stretch reflex is a reflex contraction of the muscle in response to a stretch. The Ia afferent fibers arising from the muscle spindle make direct excitatory connections with the alpha motoneurons in the spinal cord. This loop is subject to excitatory and inhibitory descending control from cortical centres as well as from other spinal cord circuits. Ia synaptic connections from one fiber will send terminals to the entire homonymous motoneuron pool (Burke et al, 1974) The Ia pathways are monosynaptic to the homonymous and the synergist motoneurons (Mendell and Henneman, 1971).

The Ia fibers from the agonist muscle also make synaptic contact with the antagonist via an inhibitory interneuron. This ensures that the antagonist does not respond with a stretch reflex after the agonist reflexively contracts. Reciprocal inhibition of opposing muscles from higher centres allows for the CNS to send only simple commands to a limb. Ia axons make direct excitatory contact with the alpha motoneurons and with the Ia inhibitory interneurons. The Ia inhibitory interneuron also receives excitatory and inhibitory impulses from descending pathways (Jankowska, 1976). Inhibition of the Ia inhibitory interneuron would more readily permit muscle contraction to occur in an opposing muscle. This would result in co-contraction around a joint given an adequate (supra-threshold) excitatory drive to the motoneuron. Excitation of the Ia inhibitory interneuron would augment its inhibitory action on the antagonist muscle group enabling the reciprocal inhibition of the agonist.

The stretch reflex elicited by a tendon tap has become valuable in clinical diagnosis. The local sign of a stretch reflex (e.g. knee jerk) is limited to the muscle from which the stimulus arises (homonymous motoneurons) and the reciprocal inhibition of the antagonist muscle. C. Sherrington in the early 1900's referred to the stretch reflex as the myotatic reflex. Myotatic simply means extended muscle. This reflex was found to be most highly developed in the physiological extensors. The phasic component (tendon tap response) of the stretch reflex is velocity dependent. The weaker tonic component of the stretch reflex was found by Lloyd in 1940, to be a length dependent response.

A self regulatory, negative feedback mechanism is also in place to stabilize the motoneuron firing rate. Renshaw cells arising from the alpha motoneuron make inhibitory connections with an inhibitory interneuron which acts upon the homonymous muscle (Renshaw, 1941). A branch of the Renshaw cell also inhibits the Ia inhibitory interneuron. This augments the activity of the antagonist muscle. As the motoneuron firing rate increases so does the activity of the Renshaw cells. With a decrease in firing rate the inhibition is decreased thus increasing the net excitability of the motoneurons. The effect of recurrent inhibition is distributed to muscles around a joint.

The tendon organ reflex is a disynaptic, negative feedback system to regulate muscle tension. It is mediated by the Ib afferent fibers which arise from the Golgi tendon organs. The Ib afferents make excitatory connections with Ib inhibitory interneurons of the agonist and synergist muscles (Lundberg et al, 1975). This system is referred to as autogenic inhibition. Through this feedback mechanism, the activity of the homonymous muscle is inhibited. A process of reciprocal excitation is also mediated via the Ib afferents. The Ib afferents synapse onto Ib excitatory interneurons which facilitate the activity of the antagonist muscle. This system keeps the discharge frequency of the alpha motoneurons at a safe level and helps to avoid microruptures at the musculo-tendinous tissues. The Ib inhibitory interneuron receives input from the Ia afferent fibers, cutaneous receptors and joint receptors. They are subject to influence from excitatory and inhibitory descending pathways. All of these connections provide a spinal mechanism for the fine control of

movement. Stiffness may be controlled by the nervous system where the stiffness is derived from the combined input of Ia (length information) and Ib pathways (force information). The degree to which spinal cord injury will affect stiffness is not known.

If a neuron makes an inhibitory connection with the cell body of another cell (axo-somatic synapse), it will decrease the likelihood that the cell will fire. This is post-synaptic inhibition. If an inhibitory neuron contacts the axon terminal of a cell (axo-axonic synapse), it can reduce the amount of transmitter released by the second cell onto a third cell. This is presynaptic inhibition. Some researchers have studied decreased presynaptic inhibition as a cause for spasticity (Pierrot-Deseilligny, 1990, Hultborn, 1987).

The excitability of the stretch reflex depends upon descending control of the pathways. Spasticity can result from abnormalities in any one of the spinal pathways controlling stretch reflex activity. Ia impulses reach the alpha motoneurons through non-monosynaptic pathways as well. a number of circumstances can result in an exaggerated stretch reflex such as inhibition or disinhibition of the spinal pathways. Facilitation of these pathways could contribute to spasticity (Pierrot-Deseilligny, 1990). The interneurons of spinal circuitry are largely under the control of descending pathways. As the descending systems degenerate as a result of CVA, for example, vacancies open up on the interneurons, motoneurons and terminals which are replaced by other systems. Spasticity is often a carelessly used term. It can denote transient or steady state reactions to stretch, phasic or tonic flexion and extension reflexes, distorted or dystonic purposeful behaviour and even decreased co-ordination and strength, as in spastic paraparesis.

Dynamometer Tests

A common term used to defined constant angular speed motion imposed by a dynamometer is isokinetic. A precise term would be isovelocity, since the velocity is the only parameter which can really be controlled by a dynamometer. Isovelocity

dynamometers have come to be widely used in the assessment, training and rehabilitation of people with sports related and other orthopaedic conditions. Their use in assessing spasticity has not been extensively reported in the literature.

Bohannon and Larkin (1985) used a Cybex II isokinetic dynamometer to reproduce a pendulum test. Their protocol consisted of seating the subject, either supine or in sitting. The subject was stabilized to the table with straps at the thigh, trunk and pelvis. The knee axis of rotation was aligned with the axis of rotation of the dynamometer. The angular speed of the dynamometer was set at 300°/sec. The examiner flexed the subject's leg until the heel touched the table. The approximate knee joint angle at this point was 115°. The leg was then fully extended. The two positions were recorded and represented full range of motion of the knee joint for that subject. The subject was reminded to relax the leg completely and the leg was dropped by the examiner. The authors made several observations using this technique to compare normal subjects to subjects with spasticity resulting from CNS lesions. Normal subjects averaged five to six peaks corresponding to pendular excursions of the leg and foot in the goniogram. The amplitude of the first backward swing (flexion from full extension) was greater than 115°. This usually resulted in the heel touching the table on the first downswing. Also, the goniograms, which show angular position/time graphs, taken in the sitting and supine positions were similar. Subjects with lower limb spasticity showed a greater magnitude of knee flexion on the first downswing when tested in the sitting as opposed to the supine position. This is likely a reflection of the pre-stretch or change in starting length on the rectus femoris. This would be due to the fact that rectus femoris is a biarticular muscle which also spans the hip as it crosses the hip joint. This means that a greater reflex response was elicited or the passive length/tension relationship of rectus femoris altered the kinematics (joint trajectory) by providing a greater resistance to motion. For this reason the authors recommended testing in the supine position. Comparisons were made between the degree of knee flexion with the first downswing and the degree of knee flexion before the test when the heel was brought into contact with the table. The rate of fall of the limb might have been controlled by the angular speed limit of the dynamometer which was 300°/s.

Peak values for velocity during a pendulum test are usually around the 150°/s mark, so the utilisation of a dynamometer was probably not a negative factor.

A follow up to the above study was done by Bohannon (1987). Thirty patients with intracranial lesions were recruited for the study. The purpose of the study was to determine the between trial variability and reliability of the above protocol for the pendulum test. Criteria was that subjects demonstrated "manually discernible resistance to passive knee flexion" or a stiff legged gait. The history of the subjects was either one of CVA or head injury. The protocol was consistent with the 1985 study, with the subjects positioned supine. Four trials per subject were performed. The most spastic side was tested. A relative angle of reversal (RAR) was calculated as the difference between maximum possible knee flexion and the degree of knee flexion after the first swing. The RAR of a normal subject would be zero. A one way analysis of variance (ANOVA) showed the variance of the RAR between trials not to be significant. An intra-class correlation coefficient (ICC) determined the reliability of repeated pendulum tests to be high. A characteristic of the pendulum test is that the displacement of the limb is represented as a damped sinusoid. Aberrations from this symmetrical but damped trace can indicate abnormalities in muscle tone. The traces derived from the Cybex II for the intracranial lesioned group do not resemble a damped sinusoid. The benefit of using the Cybex II isovelocity dynamometer over a conventional pendulum test, where the limb is released from an extended position and allowed to swing freely, is not apparent.

Firoozbakhsh (1993) used a KinCom isovelocity dynamometer to measure the resistance to passively imposed flexion and extension of the knee at different angular velocities. Eight male subjects with histories of spinal cord injury, CVA and anterior horn cell disease were recruited for testing and compared with an able bodied control group. Subjects were seated with stabilising straps over the thighs and hips. Gravity correction was performed. The more spastic leg was tested. The KinCom was programmed in the training mode. Angular velocities were: 30, 60 and 120°/sec. The knee was taken through a 60° displacement (25° to 85° flexion). Resistance of the limb to the passive

displacement was determined by recording the torque and angular position of the knee at each pre-set velocity. The maximum torque (Nm) was recorded during four consecutive trials. The slope of the moment-velocity curve (Ncm/°stretch) was also derived to assess the sensitivity to the rate of stretch. Each subject was tested once per week for six weeks. For both groups the extensor maximum torque was greater than the flexor maximum torque at velocities greater than 60°/sec. The difference was not significant. Maximum torque values were higher for the spastic group but not statistically significant. When comparing the sum of four consecutive maximum torques, the difference between spastic and normal controls was significant. The values of maximum torque and the sum of maximum torques increased linearly with increasing velocities for both groups. A comparison of the slopes revealed that the spastic group showed a greater sensitivity to the rate of stretch than the normal controls. The latency and duration of the maximum torque response were not taken into account in this analysis. There is also no explanation of how the maximum torque is identified in the trace. The increase from baseline occurs very close to the change in direction of the actuator arm. Depending upon the acceleration used, the KinCom can produce acceleration dependent inertial moments at the time of the turn which can look like increases in torque due to muscle contraction when in fact it is not.

Burke et al (1970) assessed nineteen patients with lower limb spasticity. Ten of the patients were spinal cord injured, four had Multiple Sclerosis, one had spasticity due to pseudobulbar palsy, and one suffered from familial spastic paraplegia. Three normal controls were studied. EMG activity of the quadriceps muscle was recorded during passive knee flexion with the patient in prone. Angular velocity was numerically differentiated from the output of a goniometer which was also used to measure knee joint angle. They found that the EMG magnitude increased in a linear fashion as the velocity of passive knee flexion was increased. This relationship was found if the movement started from full extension or if the knee was partly flexed prior to being moved. The minimum velocity required to elicit a reflex response in quadriceps was calculated. This velocity ranged from 5-193°/sec. The value for minimum velocity increased as the starting knee

angle was progressively flexed. With a constant velocity of passive flexion, the magnitude of the EMG response decreased as the start angle was increased (more flexed). The greater the initial degree of flexion the smaller the EMG magnitude. To determine whether this position dependent suppression was arising from knee joint afferents, the authors placed a sphygmomanometer cuff over the distal part of the thigh. A pressure of 200 mmHg was maintained for 30 to 45 minutes to minimize joint proprioception. Under these conditions, spasticity was reduced but the clasp-knife effect was still observed. This could also indicate a decreased responsiveness of muscle proprioceptors. Position dependent suppression of the stretch reflex was also still demonstrated. A fatigue effect of the stretch reflex was also demonstrated in this study. Three separate trials were performed at the same velocity. A clear decrease in reflex EMG activity is seen over successive trials. Other findings were that the faster the velocity of displacement, the longer the duration of the reflex activity. The limb was oscillated in a sinusoidal fashion through 30° of movement at 1 cycle per second. It was observed that the reflex response was greatest when the limb was oscillating around a point near full extension. This reflex response decreased as the centre of oscillation approached a more flexed position. This result likely occurs because fewer Ia fibers were responding when the limb was in a more flexed position. When the limb is displaced from an extended position to a fully flexed position, the magnitude of that stimulus is greater than when the displacement is not as large.

Burke et al (1971) also studied the stretch reflex in the hamstring muscles of subjects with spasticity. Sixteen subjects in total were used in this study. Eight were paraplegics and quadriplegics, five suffered from Multiple Sclerosis and two had familial spastic paraplegia. Subjects were lying in prone for the testing. Surface electrodes placed 10 cm apart over the hamstring muscle belly recorded the response to a passive stretch. A goniometer measured knee joint angle. Its output was differentiated to produce angular velocity data. Data collected included raw EMG, integrated EMG, joint angle and angular velocity. The limb displacement was started at three different degrees of knee joint flexion (from full extension to full flexion). Passive stretches were performed at approximately constant velocities and different velocities were used for each sequence. Velocities of up

to 300°/s were used. Sinusoidal stretching was also performed. The centre of oscillation was varied while the amplitude and velocity were held constant. The amplitude of the oscillations was 20° and 40° and were studied over a range of velocities. The resistance of the hamstring muscle to stretch was highest at full extension. The stretch reflex increased with increasing velocity. The authors do not indicate whether this increase is in the magnitude or the duration of the response. There was no position effect demonstrated with varying the starting angle of the limb. The trials with sinusoidal oscillations of the limb showed a velocity dependence. Doubling the amplitude of the oscillations had no effect. The effect was greatest when the limb was oscillating at a high velocity near the extended position. It is observed in these two papers that the reflex response of muscle to a passive stretch is greatest when the muscle is in a lengthened position. It may be that more muscle spindles are activated when the muscle is stretched from full extension than from a more flexed position. The velocities used in the study on the hamstring muscle exceeded those used for the quadriceps muscle. This may have been necessary in order to elicit a response from the hamstrings muscle as in conditions resulting in lower limb spasticity, the quadriceps muscle is usually most affected. The authors do not report any findings from a control group so no comparisons can be made to an able bodied population.

When examining the stretch reflexes in biceps and triceps in spastic subjects with CVA, high cervical spinal cord lesions, motor neuron disease, Ashby (1971) found a velocity dependence for stretch reflexes of both muscles. The time from onset of disease was three weeks to two years. The test protocol involved four types of stretching movement: 1) full range linear movements, 2) moving the limb in three sequential steps of equal amplitude 3) performing sinusoidal oscillations of the limb (20-30° amplitude) around three different centres of oscillation and 4) sinusoidal oscillations of the limb throughout its full range. The range of velocities employed was from 0°-500°/sec. This study used EMG onset as the threshold angle of the stretch evoked reflex and compared this with varying velocities. The greater the initial length of the muscle when stretched, the greater the magnitude of the response elicited. Surface electrodes were placed over the biceps and triceps muscle groups of the upper limb. Elbow joint angle was measured with

an electrogoniometer. From this the angular velocity of the limb segment was derived. A force transducer measured resistance of the limb during the stretch. When analysing sinusoidal motion, if reflex activity is only related to the velocity of the stretch, then the response would appear in the middle of the stretching phase. If the reflex response is only related to the length of the muscle, then the peak in EMG activity would appear at the end of the stretch when the muscle is at its greatest length. If the reflex EMG is related to both velocity and muscle length, then the response would appear in between these two points. For all subjects a linear relationship between reflex EMG and stretch velocity was established. There was no difference in threshold EMG between biceps and triceps, however the magnitude of the reflex response was greater with higher velocities in the triceps muscle. There was a sustained reflex response in the biceps muscle when it was maintained on a stretch. This response had a larger amplitude when the muscle was at its greatest length. This only occurred in half of the subjects and was not recorded in the triceps muscle of any of the subjects. During the trials when the limb was oscillated around different centres of oscillation the greatest reflex response was recorded when the muscle was at its greatest length. The same effect occurred in the majority of subjects in the triceps muscle. Peak EMG was recorded in the second phase of the sinusoidal motion. As the velocity of motion increased the peak moved towards the middle of the phase. The authors state that this is because the EMG response is proportional to the velocity of the stretch as well as the length of the muscle. These results concur with the results of the previous studies cited, except for the quadriceps muscle where increasing muscle length appears to inhibit the reflex response.

Measures of joint stiffness for spasticity quantify the relationship between motor output and sensory input. Reflex stiffness is a component of total joint stiffness. Reflex stiffness is comprised of: 1. the muscle fibers contracting in response to an imposed stretch (dynamic component) and 2. reflex sources such as motor unit recruitment and rate modulation of active units (Powers, 1988). Increased reflex stiffness would be observed as an increased slope of the torque-angle relationship, where the input (limb displacement) produces a larger than normal output (torque) (Lee, 1987). Ramp and Hold methods are

used to study the dynamic as well as the static output of the stretch reflex. Torque, EMG magnitude and latency during different phases of the ramp displacement are measured. A servo controlled torque motor controls the displacement of the limb at various velocities. The limb is passively moved through a predetermined range of movement. Angular position is normally measured by a potentiometer and velocity is measured by a tachometer. Torque is measured by a torque meter mounted between the motor shaft and the beam upon which the limb is resting.

Dynamic stiffness (torque) is measured during the constant velocity portion of the ramp displacement. The static stiffness is calculated as the difference between EMG amplitude and/or torque output before and after the limb has been moved (Powers, 1989). The threshold angle of the stretch reflex is the angle at which the first increase in EMG is seen during the ramp displacement. In this study the first increase in torque after the ramp displacement is calculated as the torque threshold angle. The acceleration phase (that period of the ramp displacement before constant velocity is attained) is not considered in the analysis. Only the constant velocity portion of the displacement is analyzed. The ramp and Hold method can be used to test resting muscle or pre-activated muscle. Voluntary activation of the motor units can be used to bring motor neurons above activation threshold. It is thought that any changes in torque or EMG activity during the ramp displacement under pre-activated conditions is due solely to increased reflex gain (Powers, 1989). Dynamic torque levels did not vary with increasing levels of spasticity. However, dynamic EMG activity did show an increase with increased spasticity at increasing velocities. When testing passive limbs there was an inverse relationship between velocity (0.25-3.0 rad/s) and EMG threshold angle. The same relationship was found for torque and velocity, but it was not as strong. When the muscles were pre-activated the velocity dependence relationships were similar for spastic and normal limbs. The researchers asked subjects to perform a low level voluntary contraction to activate some of the alpha motoneurons. Torque output was monitored to ensure all subjects generated a flexion torque of 1-2.5 Nm. Stretch reflex gain refers to the larger than normal increase in torque in a spastic limb when taken through a standard joint deflection. Again

acceleration parameters are not included in the analysis. This increased output is a reflection of the joint stiffness. Decreased threshold angle could be a reflection of higher reflex gain (output requires less input than normal) (Lee, 1987). This would appear as an increase in the amount of EMG activity and torque (Powers, 1988).

Powers (1988) used this method to simultaneously measure stretch reflex thresholds and joint stiffness in the spastic arm of hemiparetic subjects. It was found that the predominant source of torque variation in the passive limb was variations in reflex threshold. As the torque response increased with increasing velocities (acceleration not noted), the onset of the response was observed earlier in the limb displacement (decreased threshold angle). After eliminating threshold differences by having subjects perform a low level voluntary muscle contraction, the stiffness measurements for normal and spastic subjects were comparable. It was concluded that changes in stretch reflex gain are not a prominent source of change in stretch evoked torque because there was no evidence of increased total joint stiffness.

Lee et al (1987) investigated increased stretch reflex gain in voluntarily activated spastic muscle as a model for spasticity. They theorized that an increase in gain would be manifested as an increase in reflex stiffness. The slope of the torque-angle relationship (as elicited by varying constant velocity ramp displacements of a limb segment) would be increased. The authors studied the reflex stiffness of the bilateral elbow joints in hemiparetic subjects. Fifteen subjects with hemiplegia (due either to CVA or head injury) and ten able bodied controls were used for this study. Subjects were evaluated for: muscle tone (0-5), muscle strength (0-5), tendon reflexes (0-5) and for passive range of motion. The variables recorded were force, velocity, joint angle and EMG from biceps brachii, brachioradialis and triceps. For the test procedure the subject was seated with the arm at 30° shoulder flexion and 90° elbow flexion. A load was applied for 2.5 s to the subject's wrist causing extension at the elbow. The degree of joint displacement achieved following the load is not mentioned. Loads were selected to match the strength and fatigue limitations of the spastic limb. The dynamic phase of the displacement was centred

around a 50 ms window which encompassed the peak of force. 40 to 50 trials were recorded for each subject. 8 to 10 trials were recorded for each load. A consistent finding of increased dynamic stiffness was found in only 3/15 subjects. The most common finding was that the stiffness measurements were comparable in both the spastic and the contralateral arm of the subjects. It was concluded that changes in motoneuron threshold are responsible for the increased responses of stretch reflexes in spastic subjects.

Thilmann (1991) criticizes the use of voluntarily activated muscle in these experiments because most clinical assessments of spasticity are performed on passive limbs. Using velocities ranging from 35 to 300°/sec, ramp displacements were performed for elbow extension in spastic and normal subjects. Velocities greater than 200°/s elicited stretch reflexes in 50% of normal subjects. Tendon jerk reflexes were elicited from biceps and triceps prior to the ramp and hold series. A reflex hammer was used which contained a microswitch which activated computer sampling. There was no correlation between the size of the tendon reflex and the size of the response to the imposed stretch for either group. The response characteristics measured were integrated EMG, onset latency and duration of the response. Latency was determined from the first deflection in the velocity record. The normal range of response latencies was defined as 61-107 ms. Responses observed outside of these limits were taken to be abnormal. The normal response was characterized by one synchronous burst of EMG activity in a relaxed biceps muscle. As the velocity decreased so did the duration of this burst. There was no change from baseline EMG activity before or after this burst in the normal subjects. In the hemiparetic group there was a late EMG response as well as activity with a "normal" latency. For lower velocities there was often no early response corresponding to the time of early onset latency for "normals". The late activity appeared close to the end of the ramp displacement. This late activity appeared at velocities where there was no longer an early response being elicited. The level of biceps EMG activity is closely linked with displacement velocity but, the time of onset of this activity is less clearly linked with the velocity. In the majority of subjects the onset latency of late activity remained constant irrespective of the position of the arm and velocity of displacement. In subjects tested

early after the onset of their stroke, the responses to imposed stretch differed from those where the spasticity well established. It appeared that early in the stages of spasticity the level of EMG shows no correlation with the velocity of the stretch. Thilmann and co-workers suggest that the reflex gain increases within three months after CVA and then reduces again as spasticity becomes more established. The authors conclude that threshold changes alone cannot be responsible for spasticity. If this were the case then the late response would also appear in normals but at very high velocities. Thilmann and co-workers attribute the late response to polysynaptic pathways which are most likely mediated by muscle spindle afferents. The longer latency components were enhanced and therefor not mediated by a monosynaptic pathway. Physiological latency is difficult to determine. This study used a velocity of 300°/s and did not elicit such a response in the normals. They attribute the results to an increase in gain of the stretch reflex pathway. This contrasts the results of the previously discussed studies which refute the existence of increases in reflex gain. The differences in methodology must be considered when reviewing the conclusions derived from the results. The primary differences in methodology as previously discussed are displacing of passive versus voluntarily pre-activated limbs. The definition of spasticity implies a resistance to passive limb displacement, therefore the experiments of Powers and Lee may not truly be offering a measure of spasticity. This does not preclude other pathophysiological mechanisms which can result in spasticity.

Sinkjaer et al (1993) studied the reflex mediated and non-reflex mediated responses to passive movement in subjects with Multiple Sclerosis. Ankle joint stiffness was studied in 13 subjects. All subjects had evidence of lower limb spasticity and were able to perform voluntary muscle contractions at the ankle joint. Subjects were seated for testing with the knee and ankle joints at 100°. A motor rotated the ankle. Strain gauges measured torque. Skin temperature was maintained by a lamp and a sensor over the skin. A pre-set torque level was displayed on an oscilloscope which the subject tried to match. During the first test session a 50 ms, 4° stretch was applied to the dorsiflexors during the contraction. At the second test session the plantarflexors were stretched 2° for 30 ms. EMG activity

was recorded over the gastrocnemius and tibialis anterior muscles by surface electrodes. Torque, angle and EMG were recorded. Subjects were asked to maintain contraction levels from zero to MVC. These levels were randomly changed. Electrical stimulation was applied to the tibial nerve for the first test session and to the deep peroneal nerve for the second. Stimulation of the nerve innervating the stretched muscle is thought to eliminate the stretch reflex in that muscle, leaving only the non-reflex mediated mechanisms to be observed. The results showed that the MVC for the subjects with Multiple Sclerosis were significantly weaker than for the control group. There was no significant difference in intrinsic muscle stiffness between the two groups in the dorsiflexors. Reflex mediated stiffness was defined as the difference between total stiffness and non-reflex stiffness. Total stiffness was higher in the subject group than for the control group. There was no significant difference in reflex stiffness for the dorsiflexors between groups. The non-reflex stiffness was increased in the spastic group for the plantarflexors. In the subject group the total stiffness did not differ from the non-reflex stiffness, indicating that there was no reflex mediated component. In summary, the reflex mediated response of the dorsiflexors to stretch during a voluntary muscle contraction in spastic subjects is comparable to a control group. In the plantarflexors the reflex mediated response was found to be reduced. If the electrical stimulation of the peripheral nerves serves to eliminate any reflex response to imposed stretch, then it is not clear how the authors measured a reflex component. It is also unlikely that a perturbation of 4° and 2° would provide a sufficient stimulus to elicit a stretch reflex. The only sound conclusion that can be made from this study is that the subject group was significantly weaker than the control group.

Toft et al (1993) performed a similar experiment with Multiple Sclerosis subjects to compare EMG and mechanical stretch responses in spastic and healthy subjects. 13 subjects with lower limb spasticity were tested. The experimental protocol was identical to the previous study. EMG latency of the initial response (M1) and of the two peaks of the initial response (M1, M2) were recorded, as well as total EMG of the initial response (phasic response). A late EMG response was defined as any EMG activity occurring 200-

400 ms after the stretch. These responses were only observed in the dorsiflexors. The plantarflexors showed no reflex EMG activity in the subject group. There was no M2 response at low contraction levels for either group. There was no difference in EMG onset latency between groups. There was no difference in tonic EMG response between groups. Again, the magnitude of the perturbation is probably not enough to elicit a reflex response, which may explain why none was observed in the plantarflexors.

Gottlieb et al (1978) used sinusoidal oscillations about the ankle joint to measure the joint "compliance". They identify three physical processes by which a joint will resist passive displacement: 1) inertia, which produces reaction forces proportional to acceleration, 2) viscoelastic properties which are both a function of muscle length and the rate of change of length and 3) reflex evoked muscle contraction. It is the total of these three properties which characterizes joint compliance. The test group consisted of subjects with clinical spasticity. Only reference to spinal cord injury is made. These results are compared with normal controls. The authors do not specify the size of the groups. Subjects were seated with the thigh horizontal and the knee at 30° of flexion. The foot was strapped to a footplate with the axis of rotation at through the medial malleolus. The ankle angle was adjusted to be perpendicular with the tibia. Surface EMG electrodes were placed over the soleus and tibialis anterior muscle bellies. The subjects were instructed to relax while sinusoidal oscillations were imposed around the ankle joint. Twelve different frequencies between 3 and 12 Hz were used for 11 seconds at a time. For each frequency the angle and torque was computed. The ratio between the two was defined as the joint compliance. Average angle and EMG waveforms were also computed. In the normal subjects the peak amplitude of oscillation occurs at 4 Hz. With large angular displacements the EMG shows some synchronous activation in the soleus muscle. The latency of this muscle activity was 40 ms. When subjects opposed a biasing torque of 2.7 Nm, the large amplitude oscillations occur at 6.5 Hz and the soleus EMG shows rhythmic synchronization to the dorsiflexion phase. At 20 Hz the soleus EMG activity is synchronized with plantarflexion. Tibialis anterior EMG were not as well synchronized. There was always more soleus activity than tibialis anterior. It does not appear that the

authors have controlled for the range of movement. The velocity is set by the oscillating frequency and this controls the amplitude of displacement. The larger the amplitude of oscillation, the lower the frequency. In relaxed subjects with clinical spasticity the tibialis anterior EMG was modulated at all frequencies between 3 and 12 Hz. The tibialis anterior EMG was evoked by dorsiflexion. This EMG response was most active during the 6 and 6.5 Hz frequencies. The soleus EMG did not show any stretch modulation until 6 Hz. The peak soleus activity was recorded at 6.5 Hz. At a frequency of 7.5 Hz the small and large amplitude cycles were alternating. Soleus and tibialis anterior EMG latencies were 30 ms after a large dorsiflexion movement. At an oscillating frequency of 6.5 Hz the tibialis anterior EMG was active only every other cycle whereas the soleus EMG was active throughout each cycle. It is the authors' interpretation that at certain frequencies the EMG response would augment the amplitude of the ensuing oscillation.

Verrier at al (1984) studied the short and long latency responses to imposed displacement of the wrist musculature in patients with cerebrovascular lesions. 18 patients with unilateral infarcts were used as subjects for this study. 17 age matched normal subjects served as controls. Subjects were seated with the arm supported and the elbow at 90° of flexion. Surface EMG recordings were taken over the flexor carpi radialis (FCR) and the extensor digitorum communis (EDC). Five different displacement conditions were randomly presented to the subject. The amplitude of the displacement, the direction (flexion/extension), the duration and the delay between trials were all randomly calculated by a computer. The EMG response was normalized against a maximum M response as elicited by electrical stimulation. Angular displacements in the hemiparetic group were limited to their passive ROM. The amplitude of displacement was consistent at 50° in either direction. The responses of the hemiparetic group were recorded at their spontaneously occurring background activity. A continuous pre-load was applied to the control group in the cases where no EMG response was elicited. These inconsistencies make the results of this study unreliable. The mean M1 latency response in control subjects was 35.4 ms. The onset latencies in the hemiplegic group (mean = 36.5 ms) were not significantly different from the control group, however the duration of this response

was longer in the hemiplegic group. The long latency responses in both groups were also comparable. The authors describe an additional late response not observed in the control group. This late response seemed to demonstrate a velocity dependence, as the latency increased with decreasing velocities. These authors also report an increased sensitivity of the hemiplegic group to lower test velocities. The M1 response was more consistent in the hemiplegic group than in the control group. The variability in the control group may have arisen from different test conditions. Some subjects generated a constant pre-load prior to the displacement whereas other control subjects experienced a purely passive movement.

Rack and Thilmann (1984) examined force and EMG responses during sinusoidal oscillation of the ankle in 17 subjects. The diagnoses for the subjects included CVA, multiple sclerosis and motor neuron disease. The control group consisted of 13 able bodied subjects. Subjects were seated with the knee immobilized and the ankle secured so that only flexion and extension movements could be elicited at the joint. EMG activity was recorded from the soleus and tibialis anterior muscles. Subjects were instructed to relax the limb. The ankle was then driven through sinusoidal displacements at varying frequencies. The results showed that bursts of EMG activity occurred during the later portion of the dorsiflexion phase at lower frequencies. The EMG activity also appeared to be "locked in" to the frequency of the cycle. As the frequency of the movements increased, the bursts were observed later in the cycle. This eventually led to the EMG burst being present at the maximum dorsiflexed position. At frequencies higher than 6.5 Hz the EMG bursts were present only in alternate cycles. At low frequencies, the peak of force followed the EMG burst. As the frequency of the cycles increased the force peak was observed during the plantarflexion phase of the movement. The EMG bursts at the higher frequencies were large but resulted in smaller force peaks, possibly due to the shortening of the muscle. The peaks were larger when they occurred as the muscle was lengthening. At the higher frequencies the effect of the stretch reflex was observed nearly a full cycle after the movement by which it was elicited. For example at 5 Hz the soleus reflex response acted to resist the extension movement. At 7 Hz the soleus reflex response acted during the lengthening phase of the following cycle. For the control group the reflex

response was rarely detected during the lower frequency trials. At higher frequencies the EMG response of the soleus muscle appears locked in to the phase of the movement. Usually the amount of EMG activity is small, reflecting activity in only a few motor units. With prolonged cycling however, the responses of the normal group began to resemble those of the spastic group. It was concluded that the reflexes of the spastic group are set at a higher level of excitability, but within a normal range. When the testing was continued for a prolonged period (30 minutes) and the subject had undergone thousands of cycles of sinusoidal driving, the normal subjects showed these same responses. Reflex activity was further enhanced by "facilitation" of the reflex pathway. This was achieved by having the subject exert a small plantarflexing force (10-20% maximum voluntary contraction (MVC). The activity in the soleus muscle was observed in each cycle, even at low frequencies. These results were similar to those obtained from the spastic subjects. The authors concluded that under appropriate conditions the responses found in spastic subjects may also be elicited in normal subjects. The responses elicited in the normal subjects required higher frequencies than for the spastic subjects. The responses in the spastic subjects may be set in an exaggerated state of excitability. This merely reflects one end of a normal range of stretch reflex activity.

Thilmann (1991) further investigated the early and late reflex responses in subjects with Huntington's disease. The first short latency response (M1) has generally been accepted as arising from monosynaptic connections from muscle spindle afferents onto homonymous motoneurons. The longer latency response (M2) has many proposed origins: slower conducting afferents, longer pathways, transcortical reflex loops, polysynaptic pathways. Thilmann suggests that the transcortical reflex loop may be present for muscles in the hand which execute complex movement patterns and have many cortical connections, but that other larger muscles responsible for posture and gait do not require these extensive cortical connections. If the mechanism for M2 is the same for all muscles (transcortical reflex loop) then it should uniformly disappear for all muscles tested in subjects with Huntington's disease where the CNS damage is supraspinal. Ramp and Hold testing was used for the following muscles: first dorsal

interosseus muscle, biceps brachii, triceps brachii and triceps surae. Ramp displacements were imposed upon passive and pre-activated muscles. The M2 response was only absent when the first dorsal interosseus muscle was tested. For all the other muscles tested the M2 response was present. The authors concluded that the M2 response is not universally mediated by a transcortical pathway.

Ibrahim (1993) studied the response in elbow joint muscles during ramp displacements for active and passive muscles in patients with spastic hemiparesis. The active condition consisted of a 0.3 Hz sinusoidal torque applied to the forearm while the subject held the joint angle constant at 90°. For the passive condition the applied torque was the same, but the subject was instructed to relax the upper limb completely. Also a 30° ramp displacement was passively imposed at 200°/sec. Generally the torque and EMG responses were larger during the active tasks for elbow flexors and extensors. A more distinct delineation between M1 and M2 responses was observed during the active task for elbow flexors. Displacement velocity appeared to have the greatest effect on EMG response for both the active and passive conditions. There was no difference in response latency of M1 observed between the affected and unaffected sides under active conditions. The amplitude of the M1 response was larger on the affected side during the active conditions. The duration of the EMG response was related to the displacement amplitude (Large displacement=large response). The conclusion drawn from these results are that the reflex gain is higher in spastic than unaffected muscles. The authors feel that the wide range of responses seen in passive and active muscles argues against a single mechanism underlying the stretch evoked EMG activity in spasticity.

Electrophysiological Studies and Vibratory Inhibition Studies

Levin and Hui-Chan (1993) examined the reproducibility of several reflex measurements and the correlation of the H-reflex with clinical measurements of spasticity. Ten subjects with spastic hemiparesis and seven control group subjects were used in this

study. The following clinical measurements were taken: Achilles tendon jerks (4 point scale), 5 point Ashworth test at the ankle joint and ankle clonus (4 point scale). From these measurements the authors came up with a composite spasticity score where 0-9 indicated mild spasticity, 10-12 was moderate spasticity and 13-16 indicated severe spasticity. Subjects were tested 3 to 5 times at one week intervals. Three physiological measurements were also taken: a) H/M ratio, b) the amount of inhibition during vibration (H_{vib}/H_{cnt}) and c) the excitability of the soleus stretch reflex in terms of latency, duration and magnitude. Subjects were positioned in a semi-supine position with the thigh and calf supported. The knee was fixed at 30° flexion. The foot was positioned in neutral and attached to a footplate. H reflexes were elicited by stimulation of the posterior tibial nerve. A vibratory stimulus was applied to the inferior third of the Achilles tendon for a one minute accommodation period prior to recording the H-reflexes. The vibratory stimulus remained on for the duration of the recording. Soleus stretch reflexes were recorded during a rapid 30° dorsiflexion. The ankle was displaced from 20° plantarflexion to 10° of dorsiflexion. 30 seconds rest was provided between trials. The velocities of the ramp displacements were different on different test days ranging from 45°/s to 77.5°/s. These were not found to be significantly different. The H/M ratios were significantly increased in the hemiparetic group when compared with the control group. The authors report the presence of vibratory inhibition but no significant difference between the groups was present. They do not report whether there was any inhibition resulting from the vibratory stimulus. The stretch reflex latency and onset angle were smaller in the hemiparetic group. The duration of the EMG response was longer and the magnitude of the response were larger in the hemiparetic group. The clinical ratings of spasticity (H/M ratio, vibratory inhibition, stretch reflex onset angle and area) showed a high degree of reproducibility. Other variables (stretch reflex latency and duration) were not reproducible from one session to the next. This result is surprising since the stretch reflex latency and onset angle were found to be highly correlated. Correlations were found between the composite spasticity score and the ankle Ashworth and clonus. The Achilles tendon reflex was not correlated with any of the clinical or physiological measures.

Katz et al (1992) conducted a similar study to determine the correlation of objective measures of spasticity with clinical tests. Ten subjects with spastic hemiplegia were used in this study. Subjects participated in three test sessions, one week apart. The Fugl-Meyer scale of motor function was used to assess the subjects. A modified 5 point Ashworth scale was used to test the upper and lower extremities of each subject. The objective measures included the pendulum test and H reflex studies. For the pendulum test the subject was positioned in supine with the hemiparetic limb extended over the edge of the table. An AFO was applied to the limb to minimize ankle motion during the test. A goniometer recorded the angular displacement of the limb. EMG recording were taken over the quadriceps and hamstring muscle groups. Ten trials were performed on each limb with a thirty second rest in between. Lower and upper extremity H reflex tests were performed on both the affected and the unaffected limb. Finally, a Ramp and Hold test was performed on the upper extremities to determine torque and threshold angle. EMG activity was recorded from the biceps, brachioradialis and lateral triceps during passive extension. The amplitude of the displacement was one radian (57.3°) from 90° of elbow flexion. The two stretch velocities were 30°/sand 60°/sfollowed by a 1.5 to 3.0 second hold phase. Threshold angle was calculated from the torque onset and the onset of the EMG. Torque output was measured at 45° for each trial. There were 10 trials at each speed per subject. The pendulum test was analysed using a Corrected Relaxation Index (CRI). This was calculated as the amplitude of the first swing/amplitude of the final resting angle. The results indicated that the CRI was accurate in identifying a spastic limb. Threshold angles for the biceps varied from 4.5° to 40.4°. A value of 60° was assigned during the trials in which no EMG activity was recorded, assuming that the threshold angle was beyond the measured limits of extension. These values are misleading because it is possible that there would be no reflex activity in these subjects, especially given the low velocities of the ramp displacements. It would be more accurate to record no data for these trials. Significant correlations were found between the Ashworth test and the Ramp and Hold threshold angle at both speeds. The Fugl-Meyer test also correlated with the Ramp and Hold threshold angle. The Ashworth and the Fugl-Meyer test were also correlated. The pendulum test was correlated with the lower limb Ashworth test. The H/M

ratio did not correlate with either clinical scale in this study, as was the result of the previously discussed study. H reflex tests are not easily reproduced over successive test sessions, which may be the reason for this result. The authors do not report their findings on the reproducibility of these tests, including the reproducibility of the Ashworth test and the Fugl-Meyer which rely on subjective interpretation.

The spasticity that results from a CVA is likely the result of altered descending control onto the spinal cord circuits. Katz and Pierrot-Deseilligny (1982) investigated recurrent inhibition of the alpha motoneurons in 95 patients. They hypothesized that recurrent inhibition was decreased in the subject group. Eighty-seven had suffered a stroke and 8 had spinal cord injuries. The duration of illness was from six weeks to ten years. All subjects had a hyperreactive ankle jerk and clonus at the ankle. Subjects were seated with the knee semi-flexed and the ankle at 110° of plantarflexion. Surface electrodes were used for both stimulation and recording. H-reflexes were recorded from the posterior tibial nerve. The Renshaw cells were activated by a conditioning H-reflex volley. The resulting recurrent inhibition was assessed by a second H-reflex discharge. By making the test stimulus large enough to activate the alpha motoneurons, an antidromic motor volley is produced at the site of stimulation. The conditioning H-reflex discharge will collide and eliminate the antidromic motor volley. Therefore the alpha motoneurons will not be affected by the impulses from the antidromic motor volley. They will be available to fire in response to the input of the Ia afferents elicited by the test stimulus. The H test reflex has been generated by the alpha motoneurons which have already given rise to the conditioning stimulus. The excitatory postsynaptic potentials (EPSP) created by the Ia conditioning volley are eliminated by the action potentials they have elicited. The amplitude of the H test reflex is not influenced by these EPSP's and can be used to estimate the inhibitory effects of the conditioning volley. These authors found that at rest recurrent inhibition was increased in the subject group. During weak activation of the soleus muscle in the control group the recurrent inhibition increased whereas during strong contractions the recurrent inhibition decreased (due to dysfacilitation of the Renshaw cells). During weak contractions the increase in recurrent inhibition was less in

the spastic subjects than in the normal controls. The authors conclude that the supraspinal facilitation of the Renshaw cells observed in the control group has disappeared in the spastic group.

Iles and Roberts (1986) examined the contribution of presynaptic inhibition of monosynaptic reflexes in 14 subjects with upper motoneuron disease. Their diagnoses included CVA, Multiple Sclerosis, cervical spondylosis and meningioma. Lower limb spasticity was assessed using the Ashworth scale. The examiners also assessed muscle power and the presence or absence of clonus. Test H reflexes were elicited in the soleus muscle. Subjects were tested both at rest and during voluntary contractions. The test reflex was conditioned by a maintained vibration over the gastroc-soleus, tibialis anterior, quadriceps or biceps femoris muscles. According to these authors any postsynaptic activity of afferents activated by the vibratory stimulus would lead to a change in firing of motoneurons and therefore a change in torque. If the subjects are instructed to maintain a constant torque level then they would have to respond to the increase in torque produced by the vibration. This would be accomplished by a decrease in effort (descending control) to decrease the torque. If the H reflex elicited is smaller during the period of vibration, then a presynaptic mechanism must be active. These authors found that the inhibition of the H reflex decreased as the voluntary contraction became stronger in the control group. The strength of the inhibition in the spastic group was less than that seen in the control group, but it also had a tendency to decline with increasing plantar flexion force. Subjects with the severest spasticity and the least power demonstrated the least inhibition of the H reflex.

Vibrating muscle fibers percutaneously over the muscle belly is known to have a paradoxical effect on the alpha motoneurons supplying that muscle. Vibration produces a powerful excitation of alpha motoneurons known as the tonic vibratory reflex. The long latency of this reflex response suggests a polysynaptic pathway (Desmedt, 1980). It also inhibits phasic myotatic reflexes involving motoneurons from the same motoneuron pool (Desmedt, 1980). The latency response to vibration of the primary afferent fibers is short

and confined to the vibration period. The latency of the inhibitory response to vibration is approximately 100 ms post stimulus onset and can outlast the stimulus by several seconds (Desmedt, 1980).

The excitatory effect of a vibratory stimulus is greater for the Ia fibers than for the Group II fibers (Matthews, 1986), especially with vibratory frequencies greater than 1 Hz (Desmedt, 1980). A stretch stimulus tends to excite both Ia's and Group II afferents. A Group II response will likely not be seen with a vibratory stimulus (Matthews, 1986). The reflex response to vibration falls away after 40-50 ms whereas the reflex response to stretch continues throughout the 40-50 ms period (Matthews, 1986). This late response must be due to the Group II fibers as the Ia fibers are locked into the vibratory stimulus.

Vibratory stimulation has been used to examine presynaptic inhibition onto the Ia fibers (Hultborn, 1987). When testing subjects with spasticity, it has been shown that the depression of the H reflex is not as great in these subjects as in normal controls. This indicates a decrease in presynaptic inhibition in people with spasticity (Pierrot-Deseilligny, 1990). The effect of applying a vibrating stimulus during a Ramp and Hold test has not been investigated in human spasticity.

Pendulum Test

Wartenberg (1951) developed the pendulum test as a clinical tool for assessing muscle tone for the purpose of neurologic diagnosis. His method requires the patient to be seated. Both legs are lifted to the horizontal level and released, allowing them to swing freely. He observed that patient interference with the test was common. However, in assessing hemiparetic patients it was observed that they tended to interfere more so with the unaffected leg than with the leg on the paretic side. The damped sinusoid is described although there is no method used to record the trace and diagnosis is based on what is observed. It is simply stated that if one leg swings longer than the other then the presence

of some abnormality must be assumed. Any abnormality of movement usually occurs in the extension phase after the initial drop of the legs. A decreased initial drop into knee flexion is also described in states of increased extensor tone. The emphasis is on observing the quality of the resulting movement rather than the quantity.

Boczko and Mumenthaler (1958) modified the pendulum test developed by Wartenberg. In their protocol the subject is seated and the leg to be tested is supported by a board resting at the back of the ankle. The position of the knee joint is at 45° of flexion. The room in which the test is conducted is darkened because the motion of the leg is monitored by a light attached to the big toe of the subject. A camera records the trial. It moves in a plane of movement perpendicular to the plane of motion of the leg. They describe a sinusoidal movement recorded by the camera. The overall swinging time of the leg is deduced from the known rotational speed of the camera. The authors tested six healthy subjects and thirteen subjects with lower limb spastic paralysis. The parameters which were measured were both qualitative and quantitative: total wave count, amplitude of the first backswing (A1). A normal record was one in which the trace showed a damped sinusoidal pattern with no loops and brisk changes in direction. The healthy subjects showed six to seven waves and never less than five. The pendulous movement can last more than twelve seconds. 15% of the subjects with spasticity showed traces similar to those of the healthy subjects. Characteristic deviations were described as arches and loops in the trace. These are the result of a lateral displacement of the limb away from the saggital plane of motion. The authors describe a jerky extension movement on the first backswing of the leg, but they do not attribute this to an exaggerated stretch reflex. The number of swings in the spastic subjects was five or less. Mean swinging time in the healthy subjects was 10.82 seconds. The mean swinging time for the spastic subjects was 7.09 seconds. A1 was measured in millimetres on the camera trace. Mean A1 in healthy subjects A1 was 14.09 mm. In the spastic group the mean A1 was 9.33 mm. The extent of the damping effect was calculated as the relation of A1 to the difference between A1 and the amplitude of the first forward swing (A2): A1/A1-A2. This was referred to as the amplitude ratio. The mean value for the healthy subjects for the amplitude ratio was 6.41.

For the subjects with spasticity the mean value was 2.61. The lower the amplitude ratio the less displacement is seen after the first downswing. This study represents the first attempt to objectively quantify the degree of spasticity in a patient population using the pendulum test.

Badj and Bowman (1982) added electromyographic (EMG) recording to the pendulum test to determine the beginning and duration of knee extensor activity. Fifteen spinal cord injured subjects were tested. They were positioned in supine with both legs dangling over the side of the bed. Surface electrodes were placed over the quadriceps muscles. An electrogoniometer was placed over the knee joint. A switch was located at the ankle, operated by the examiner to determine the onset of the movement. A visicorder light oscillograph recorded the signals from the switch, EMG and the electrogoniometer. The limb was manually brought to the horizontal and released after the subject had been asked to be as relaxed as possible. The limb was allowed to fall freely as knee joint angle and quadriceps EMG were recorded. Testing was done on five consecutive mornings, before any physiotherapy exercises had been done. The results show an increase in EMG activity and a decrease in excursion of the limb with increasing degrees of spasticity. Their method of rating the degree of spasticity is not defined. It is stated that all of the tests are performed from a starting position of full knee extension, however all of the traces pictured show a starting angle of 55-65° of knee flexion. There is a clear shift in the quadriceps latency with an increasing severity of spasticity. Subjects were also tested in both lying and sitting positions. The authors state that the results support using the supine position for a more sensitive assessment of spasticity, although these results are not shown, nor are they compared with the results obtained in a seated position. When the authors performed the tests at thirty second interva

Badj and Vodovnik (1984) repeated the protocol of the 1982 paper and added numerical analysis. One limb of the electrogoniometer was attached to the tilt table upon which the subject was lying. 90° on the goniometer does not correspond to 90° of knee flexion with this set up. The potentiometer was placed at the approximate centre of joint rotation. The second leg of the goniometer was secured to the calf of the subject. A ball joint allowed free movement of the limb out of the saggital plane. A tachometer measured the velocity of limb movement and EMG recording was used to assess quadriceps activity during the test. A Relaxation Index (R2) is derived from the results. R2 is defined as the ratio between the magnitude of the first drop (A1) and the magnitude of the initial angle (A0). Normal subjects show an R2 of 1.6 or more. For this reason the R2 is normalized by calculating R2/1.6. With this calculation an R2 greater than 1 would indicate a non spastic limb and an R2 less than 1 would quantify various degrees of spasticity. A value of 0 would indicate that no movement occurred when the limb was released from the starting angle. The authors recorded eight parameters from the pendulum test: Relaxation Index, number of swings, the area below the goniogram prior to the first crossing over the resting angle value, the first maximum of the goniogram, the Relaxation Index at the half swing (test is performed from a 45° angle, not full extension), the average Relaxation Index of ten successive swings, the first maximum and the first minimum of the tachogram. Tests were performed on ten spinal cord injured subjects and five hemiplegic subjects. Subjects showed at least moderate spasticity on manual testing. Ten successive tests were performed at thirty second intervals. Subjects were tested on five consecutive days. The authors report a plateau in the Relaxation Index after two or three pendulum tests when performed at this rate. The authors determined that the first maximum displacement alone is insufficient to measure spasticity as these values approached normal even when the presence of spasticity was evident. When the test was performed beginning with the limb at 45° flexion, a smaller degree of spasticity, as measured by the Relaxation Index, was evident than when the test was performed with the limb at a fully extended position. Velocity as recorded by the tachometer was not a reliable measure of spasticity. The authors also found that the Relaxation Index was variable over the five day test period

reflecting variations in the level of a subject's spasticity. The authors found high correlations between the Relaxation Index derived from a test performed with the limb in full extension and the first maximum displacement of the limb. These parameters were only highly correlated with each other with the spinal cord injured subjects and not with the hemiplegic subjects. For the hemiplegic subjects the parameters with the highest degree of correlation were the average Relaxation Index of ten swings and the minimum and the maximum of the tachogram. The area under the goniogram is not included as a measure with a high degree of correlation for either group. It is unusual that the parameters of displacement amplitude are not associated with the parameters of displacement velocity since spasticity is a velocity dependent phenomenon. The stimulus parameters are not controlled during the pendulum test except for the start angle. As such, displacement is not a controlled parameter. One would predict that a greater velocity attained during the first displacement would be reflected in a smaller amplitude of displacement due to the increased stimulus for the stretch reflex response. This point is not included in the author's discussion. The authors recorded quadriceps EMG but did not include it in the analysis. They state that gross surface EMG recording does not give any additional information. They did not use the EMG recording to report differences in onset angle of the stretch reflex, not did they report the differences in magnitude of the EMG response between normal and spastic subjects.

Brown et al (1988) ran a reliability study of the pendulum test in a random sample of healthy, elderly subjects. One hundred subjects volunteered for the study. They were randomly divided into three groups: S1 - tested in the supine position, S2 - tested in semi-supine with the trunk supported at 30° and S3 was tested in sitting. The limb to be tested was released manually after the investigator determined that the subject had adequately relaxed the limb. Output was recorded for a period of ten seconds. Six to seven tests were performed on each leg. The investigators also tested a group of younger subjects ages nineteen to twenty four. The test protocol was the same but each subject was tested in all three positions. The measurements obtained from the test were the Relaxation Index, the first maximum velocity, the first maximum acceleration and the maximum acceleration on

the first rebound swing. Statistical tests of the data were by multi factor analysis of variance. Not only were the plots of the damped sinusoid obtained but also a phase-plane plot which resembles a whirlpool. This display plots velocity data against angle data. These plots allow for quick identification of any aberration from the norm such as voluntary input on the part of the subject or pathological responses found in the affected limb of stroke patients. The results demonstrated that the greatest variation was found in the group tested in sitting, indicating that the test is most difficult to regulate in this position. They also state that by testing the younger subjects in all three positions they found that the effect of position did not contribute to measurement variability. The acceleration data showed that in all cases the maximum acceleration was greater for the first rebound swing than for the initial swing. The authors stated that after performing a few tests they became proficient at recognising when a subject had adequately relaxed his or her leg. The use of EMG recording would be of benefit to ensure a relaxed state in all subjects and would perhaps decrease the large degree of variability that the authors found between subjects. A reliable method of assessing relaxation is especially important for research purposes.

An additional study was undertaken by Brown et al (1988) to determine whether the pendulum test can differentiate between spasticity and rigidity. Thirty subjects were recruited who had suffered strokes within three to nine months of the experiment. Twenty subjects with Parkinson's disease also participated in the study. All of these subjects were ambulatory and living at home. The protocol was the same as described in the previous experiment except that all subjects were tested in a semi-supine position. Again, relaxation was determined manually by the investigator. The results of the stroke subjects showed that the unaffected leg revealed results similar to those previously recorded from healthy subjects. The affected leg does not overshoot the final resting angle as does the unaffected leg. The authors also describe a characteristic inflection in the trace seen only on the affected side. This inflection is seen early in the initial downswing of the leg. It represents a slight change in direction of the limb as it descends. This may be the result of a reflex contraction of the quadriceps muscle. The Relaxation Index for the spastic

subjects was calculated as the ratio of the angle change to the point of inflection and the difference between the start angle and the rest angle. The mean Relaxation Index for the affected leg was 0.99. For healthy elderly the mean was 1.54. The mean Relaxation Index for the unaffected leg of the stroke group was 1.21. The traces in the paper show that the pendulum test for the affected and unaffected legs of the stroke group were not released from a horizontal position. This may have confounded the results if all of the subjects had a different start angle for the test. The group tested with Parkinson's disease showed a reduced number of oscillations when compared with normal controls. Their relaxation indices were also lower than those of the control group but greater than the group with spasticity. There was no evidence of an inflection in the traces of the Parkinson's group. The maximum velocity of leg swing was lower in the group with Parkinson's Disease than in the group with spasticity.

The Relaxation Index, as calculated by these authors, does not consider the entire resultant trace from a pendulum test. When comparing traces of normal and spastic subjects, it is apparent that the total displacement of the spastic subjects is approximately half that of the normal subjects. Rather than calculating a Relaxation Index, other parameters such as total displacement or number of oscillations can be used to gauge abnormal neuromuscular responses in this group. It is uncertain how variations in these responses would permit a greater understanding of their neural mechanisms.

Summary of Literature

One of the gaps in the literature is a lack of homogeneity in subject groups. Previous authors have used combinations of subjects with various diagnoses. It is difficult to make comparisons of results when the subject groups across a body of literature are not homogeneous. If researchers start to use groups of subjects with the same diagnosis then subtle differences in the presentation of spasticity may be observed.

Many studies of spasticity have been conducted with spinal cord injured patients. The underlying neurophysiological mechanisms of spasticity as it presents in hemiplegia has not been as extensively studied. Many of the studies of hemiplegia due to CVA focus on assessment of function rather than the objective quantification of spasticity. The studies that have tested objective measures of spasticity have required elaborate laboratory set-ups and would not be easily reproduced in a clinical setting.

Many of the Ramp and Hold tests have been conducted on joints such as the elbow and the ankle. Spasticity around the knee joint has not been extensively investigated using the Ramp and Hold method. Understanding the functional contribution of knee extensor musculature in stroke patients is important when analyzing abnormal responses in that muscle group.

The quantifiable parameters derived from the pendulum test have focused on the calculation of a Relaxation Index. The total number of oscillations and the pattern of decay of the sinusoidal waveform have not been considered. The inclusion of these parameters will help to more clearly distinguish between a spastic and a normal limb when analysing pendulum test results. These parameters are all indicators of velocity dependent stretch evoked reflex activity.

Past studies using Ramp and Hold testing have not altered the acceleration of the imposed displacement. The velocity was altered and the acceleration remained constant. This study will examine the effect of velocity as well as acceleration on the stretch evoked reflex response in hemiplegia.

Objectives

To examine the stretch evoked reflex response in the quadriceps muscle in CVA patients as compared to a group of able-bodied controls.

To examine the effect of altering the acceleration of the imposed ramp displacement on the quadriceps stretch evoked reflex response.

To compare the sensitivity of commonly used clinical tests for spasticity (Ashworth test, ankle clonus) to objective tests (pendulum, ramp and Hold). Clonus is included in the clinical tests because it is likely a velocity dependent response and it is a sensitive measure of abnormal stretch reflex activity, even though it is tested at the ankle and not at the knee.

To observe the effect of the application of a vibratory stimulus prior to the ramp displacement on the quadriceps stretch evoked reflex response.

Hypotheses

Ramp and Hold Test

The CVA group will have lower threshold angles and lower threshold velocities than the control group.

A velocity dependent response of the quadriceps electromyogram will be observed for the CVA group during the Ramp and Hold test. A velocity dependent response will not be as great in the control group.

An acceleration dependent response of the quadriceps EMG will be demonstrated for both groups during the Ramp and Hold test. This effect will be greater in the CVA group.

There will be a positive correlation between the subjective clinical assessment (ankle clonus and knee Ashworth Scale) and measures derived from the Ramp and Hold test.

The vibratory stimulation will result in a decreased EMG response during Ramp and Hold test for both groups. This effect will be greater in the control group than for the CVA group.

Pendulum Test

The CVA group will have lower threshold angles and lower threshold velocities than the control group.

When compared to the control group, the CVA group will have abnormal (non-symmetric, spiral pattern) phase-plane plots derived from the pendulum test.

There will be a positive correlation between the subjective clinical assessment (ankle clonus and knee Ashworth Scale) and measures derived from the the pendulum test.

Clinical Relevance

An objective method of measuring spasticity in CVA patients is important to monitor the effectiveness of therapeutic and pharmaceutical interventions used to control spasticity. An area of research that has not been well developed to date is the time course of spasticity following a cerebrovascular accident. The tests protocols used in this study could be repeated over time to determine how the stretch evoked reflex response changes over time. The pendulum test in particular is relatively inexpensive and easy to administer. The results are easy to interpret at a glance and are also quantifiable. This test would be an important addition to a routine physical examination of the CVA patient.

The results of this study will also validate or refute the use of subjective clinical tests such as the Ashworth test or ankle clonus. If the results of these tests do not correlate with the objective results of the pendulum test and the Ramp and Hold test then their reliability should be questioned.

Assumptions

The control group would be representative of a larger population of age-matched, able-bodied individuals.

The CVA group would be representative of the CVA population.

The pendulum test and the Ramp and Hold tests would be sensitive enough to detect spasticity in the CVA group subjects.

The range of velocities for the Ramp and Hold test would be sufficient to elicit a velocity dependent response in the CVA group.

The earliest observed EMG response following a ramp displacement or in the pendulum test is assumed to be a Ia mediated or M1 response.

Limitations

Due to the small sample size and possibly different lesion sites, the generalizability of the results to the CVA population will be limited.

The Ashworth test was only performed at the knee joint. The scores from other joints may have been useful in gaining a broader picture of the manifestation of spasticity in each subject. By limiting the Ashworth test to the quadriceps muscle group its application to the upper limb is unknown.

Only the quadriceps EMG was recorded. Recording from the hamstrings and other lower limb muscle groups could have increased our understanding of the interaction of all the lower extremity muscle groups in a patient with spasticity due to CVA.

Methodology

General Design

This was a comparison study between subjects with a history of cerebrovascular accident and age matched able bodied controls. Two tests of spasticity were used: the pendulum test and the Ramp and Hold test. The pendulum test consists of the examiner passively extending the knee of the subject and then releasing it. The resultant pendular motion of the limb is observed. The Ramp and Hold test consists of passively imposed ramp displacements of the lower limb. A dynamometer is used to impose the displacements at different angular speeds and accelerations.

Subjects

Informed consent was received from each subject (Appendix A). This study was approved by the Human Ethics Committee (University of Manitoba) prior to gathering the data. A brief history was obtained from each subject including information regarding medication, previous orthopaedic surgery and any knee pathology (Appendix B). Ten subjects with a history of cerebrovascular accident were recruited at the Health Sciences Centre, Winnipeg, MB. An equal number of able bodied individuals from the population of Winnipeg volunteered to serve as an age matched control group. The control group subjects were asymptomatic with no known orthopaedic or neurological conditions which would otherwise affect the results.

Males or females with a history of one CVA were included in this study. The age range was forty to seventy-five years of age. Subjects with injuries between six months and five years since onset were sought. The CT scan reports of all subjects were obtained to determine the site of the lesion. Subjects with a middle cerebral artery CVA were

preferred as this is the most common site of CVA and to ensure homogeneity in the test group. Subject's were chosen if they demonstrated near full passive knee extension of the affected side, if they were able to transfer with minimal assist and if they were able to comprehend and follow through on instructions.

Subjects were excluded from the study if they were taking any medication known to control or influence spasticity. Other medications were recorded. If subjects had a history of lower limb orthopaedic surgery or any knee pathology they were excluded from the study. If severe flexion contractures of the knee were present or if the subject showed signs of unilateral neglect they were excluded from the study. Subjects with other serious medical conditions were also excluded.

An evaluation of the subjects' lower extremity tone was performed. The Ashworth test is a way of grading spasticity. The following scale was used:

0 = no spasticity

1 = slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part is movedin flexion or extension.

2 = slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of motion.

3 = more marked increase in maucle tone through most of the range of motion, but the affected part is easily moved.

4 = considerable increase in muscle tone through most of the range of motion, passive movement difficult.

5 = affected part is rigid in flexion or extension. (Bohannon and Smith, 1987).

The test is a manual test where the examiner passively extends and then flexes the leg at the knee joint. The amount of resistance perceived during the test is assigned a score

from zero to five. Clonus around the ankle was assessed by a quick passive dorsiflexion of the foot. Clonus was recorded as present or absent.

Electromyography

The electrical activity of muscle can be studied using either surface or indwelling needle electrodes. For the purpose of this study it was not necessary to observe the contribution of individual motor units. In order to study the response of a large muscle group like the quadriceps, several indwelling electrode would have been required to get a cross sectional sample of muscle fibers. By using surface electrodes, the muscle fibers near the electrodes are represented in the EMG trace.

The skin was rubbed with an alcohol swab to decrease the input impedance. Adult silver/silver chloride EKG electrodes were used (Medtronic-Cleantrace 1700-005). The diameter of the gelled portion of the electrodes was 19 mm. A differential recording method was used with three electrodes; two active and one reference. The active electrodes were placed in a proximal-distal configuration over the quadriceps muscle at mid-belly level. Electrodes were separated by approximately 2-4 cm. The reference electrode was placed over the medial aspect of the knee joint. The leads of the electrodes were twisted in order to decrease the reception to 60 Hz line noise.

The three EMG electrode leads (short wires to minimize pick-up of unwanted noise) were connected to small preamplifiers and the preamplifiers taped to the velcro strap which secured the upper thigh to the dynamometer. These preamplifiers amplified the raw signal 100 times and had a 44 MΩ input impedance. Nominal skin impedance is known to range from 200 to 10,000Ω. The input impedance of the preamplifier is recommended to be at least 100 times that of the skin resistance (Winters, 1980). The initial preamplification serves to improves signal to noise ratio for transmission to the main amplifier. With differential recording, a triphasic EMG waveform is most commonly observed when a single unit is recorded. Common mode rejection of the amplifier is used

to eliminate any signals which are generated within the room from electrical sources such as appliances connected to wall plugs or fluorescent lights. The common mode rejection ratio was greater than 90 dB, adequate for rejection of common 60 and 120 Hz line noise. This is also the recommended common mode rejection ratio (Winters, 1980).

The preamplifier output was connected to the main amplifier where the signal was band pass filtered (30-1000 Hz, ±3 db). The low pass setting of the band pass filter is used to eliminate unwanted high frequency noise and the high pass setting is used to reduce motion artefact. While the subject produced a low level of quadriceps muscle isometric contraction, the gain of the main amplifier was adjusted to full scale. This method of gain adjustment resulted in high overall system gain.

The data acquisition sampling rate was 2000 Hz (sample every 0.0005s) for the Ramp and Hold test and 500 Hz (sample every 0.002 seconds) for the pendulum test. A 12 bit data acquisition board (Advantech PCL-812 PG) controlled by custom acquisition software was used for data collection. EMG, force, angle and velocity signals were collected by the data acquisition computer. Custom software numerically derived additional parameters of angular acceleration and moment. These signals were then stored to file. The files were analysed in a spreadsheet (Quatro Pro, Version 5.0) and other custom software. Raw EMG data should not be compared because of variations in gain, electrode placement, subcutaneaous fat, etc between subjects.

Bandpass filtered EMG signals from quadriceps muscle revealed clean, readily observable EMG activity when present. Amplitude normalization of the EMG signals would be performed by using the EMG response recorded for the highest velocity and high acceleration condition of the Ramp and Hold test. The EMG data should be normalized using the follwing normalization procedure:

$$\frac{\text{EMG area}}{[\text{EMG area } (180°/s,\text{high})] + [\text{EMG area } (180°/s,\text{low})]}$$

Pendulum Test

Goniometer

An electrogoniometer was fabricated using two formed plexiglass arms approximately 30 cm in length. A potentiometer (Bourns, 10 KΩ, linear ±0.25%) was situated at the axis of the goniometer between the two arms. This device was used to monitor joint position during the pendulum test. The goniometer was secured to the limb with the potentiometer axis at the knee joint axis approximated by the knee joint line. One arm of the goniometer was aligned with the greater trochanter and the other was secured in line with the lateral malleolus.

A precision 5V DC power supply was attached across the potentiometer and the voltage signal between the ground and slider was recorded. The output of the electrogoniometer was numerically differentiated to obtain angular velocity. The voltage output was collected at 500 Hz. Calibration was performed with a protractor. The calibration revealed that the output of the potentiometer corresponded to 15 mV per degree of motion.

Protocol

Subjects were seated on the dynamometer with the thighs and trunk supported. Subjects were required to wear an custom fabricated ankle foot orthosis (AFO) on the affected leg to eliminate any artefact due to ankle motion. The AFO was fabricated with an angle of 90°. The thigh was stabilized with a padded velcro strap and a seat belt was fastened across the hips. An electrogoniometer was attached with velcro straps to the proximal and distal thigh and to the proximal shank.

The subject's leg was passively extended by the examiner to full knee extension. The subject was asked to completely relax the leg. This was verified by EMG recording. The subject's leg was supported by the examiner until it was quickly released, allowing the limb to swing freely. A total of three trials were conducted with a rest period of thirty seconds between trials. If it was obvious during the trial that the subject did not or that the subject had in any way actively interfered with the free swinging motion of the limb, then the trial was repeated. No more than six trials in total were perfomed on any subject.

Data Analysis

EMG and electrogoniometer data was sampled at 500 Hz. Each trial was recorded for ten seconds. Parameters measured from the pendulum test were: threshold angle, Relaxation Index, total number of oscillations and the amplitude of each successive oscillation (decay). The threshold angle was determined by observing the point at which the EMG signal departed from the resting baseline after the release of the limb. Relaxation Index was calculated as the amplitude of the initial displacement divided by the final resting angle. The total number of oscillations were counted from the angular position waveform. The decay of the sinusoidal waveform was determined by calculating the amplitude of each successive leg swing.

Ramp and Hold Test

Dynamometer

The KinCom 500H (Chattex, Hixon, TN) isovelocity dynamometer was used to perform the Ramp and Hold testing. The dynamometer is a computer assisted feedback device which allows for a period of constant velocity angular ramp displacements to be passively imposed to the lower limb of the subject. The subject was seated on the dynamometer with the thighs and trunk supported. The thigh was secured with a padded velcro strap. A seatbelt was fastened across the subject's hips. The trunk-hip angle was constant at approximately 100°. The leg was attached by a padded velcro strap to the actuator arm which is equipped with a force transducer at the site of attachment. The leg was aligned with the actuator arm of the dynamometer using the following landmarks: greater trochanter, lateral femoral condyle of the knee and the lateral malleolus of the ankle. The angular velocities used were: 45°/s (0.78 rad/s), 90°/s (1.57 rad/s), 135°/s (2.35 rad/s) and 180°/s (3.14 rad/s). The actuator arm with the limb segment attached is accelerated to the pre-set velocity by the device. The magnitude of the acceleration can be selected from three levels (low, medium and high). High acceleration is approximately equal to 87.26 rad/s^2 (3000°/s^2). Low acceleration is approximately equal to 35 rad/s^2.

Protocol

The dynamometer passively moved the limb to 90° of knee flexion at the following velocities: 45°/s, 90°/s, 135°/s and 180°/s. The acceleration parameters were set at "high" for each velocity. At 180°/s there was also a trial with "low" acceleration which preceded the trial with high acceleration. Prior to recording the first trial, a warm up of three repetitions at 45°/s was performed. This enabled the subject to become familiar with the

device. It also allowed the examiner to determine whether or not the subject was adequately relaxing the limb during the trial.

There were six conditions for the Ramp and Hold tests which were as follows:

Condition 1	45°/s (high acceleration)
Condition 2	90°/s (high acceleration)
Condition 3	135°/s (high acceleration)
Condition 4	180°/s (low acceleration)
Condition 5	180°/s (high acceleration)
Condition 6	180°/s (high acceleration with vibration)

The order of the trials was from lowest velocity to highest. The limb was held in full extension for 4 seconds prior to being passively displaced to 90° of knee flexion. After each ramp displacement, the limb was returned to 0° extension at a velocity of 25°/s. There was a 60 second rest between each different velocity as well as a 10 second rest between repetitions. The limb was resting in an extended position during these periods.

There were a total of three repetitions at each test velocity. Only the involved limb was tested for the stroke group and the right lower limb for the control group.

For the vibration trial the speed was 180°/s with high acceleration. A vibrator (Wahl 2 speed Vibrator, Toronto) was used to deliver a stimulus over the quadriceps tendon, inferior to the patella (ligamentum patellae). The Wahl vibrator has the capacity to deliver a vibratory stimulus of 60 Hz. The amplitude of the vibration is approximately 1.5 mm (peak to peak). The examiner manually land-marked the ligamentum patellae and placed the vibrator over the tendon for the three second duration which ended just as the displacement was initiated. This was under manual control and therfor precise quantification of time was not possible.

Data Analysis

Recording began prior to limb displacement (250-500 ms) to determine the baseline EMG level for the subject. Each trial was recorded for a total of three seconds. Spreadsheet analysis was used to determine displacement onset, EMG onset (latency) and threshold angle. EMG responses were also analysed graphically to detect any motion artefact. The duration of the EMG response was derived by visual analysis of the raw plots. The presence of acceleration dependency, velocity dependency and adaptation were determined. EMG threshold angle was determined through spreadsheet analysis. It was defined as the angle at which the EMG signal departs from the resting baseline. Latency was defined as the time from the onset of motion to the beginning of the reflex EMG signal. Acceleration dependency was said to be present if the EMG response was greater at 180°/s with high acceleration that it was at 180°/s with low acceleration. Velocity dependence was present if the magnitude of the EMG response was greater with increasing velocities. Adaptation was present if a reflex response was present during the first of three trials only. If no reflex response was seen during the subsequent two trials then adaptation of the response had occurred. The effect of vibratory inhibition was determined by comparing the EMG duration for the 180°/s, high acceleration conditions with and without vibration. The window for analysis for all trials included the total EMG response. This window did not exceed the end of the displacement and therefore includes the total stretch reflex response.

Statistical Analysis

All statistical analysis was performed using the statistical software programme, Systat (version 5.0 for Windows). Mean, standard deviation (SD), standard error of the mean (SE) and range were determined for all parameters. When required, independent t-

tests were performed to compare responses between groups. Pearson correlation coefficient was used to compare the clinical tests to the results of the Ramp and Hold test and the pendulum test, even though the clinical tests were ordinal data.

Results

Subjects

The CVA subjects were recruited from the Health Sciences Centre. Table 1 summarizes the demographics of the CVA subject group. 10 control subjects were recruited with a mean age of 60 years (SD=11.05). Examination revealed that ankle clonus was present in subjects who had Ashworth scores greater than 0, as well in subjects with no apparent spasticity about the knee.

CVA	Age (years)	CVA Age (months)	CVA Side	Ashworth Score	Ankle clonus
1	62	14	R	0	no
2	76	19	L	0	yes
3	59	10	R	3	yes
4	36	7	L	0	no
5	59	30	R	0	yes
6	43	26	R	2	yes
7	48	21	R	0	no
8	77	168	R	0	no
9	49	11	L	0	yes
10	55	36	L	2	yes
mean	56	34			
	(SD=12.55)	(SD=45.46)			

Table 1. CVA subject demographics. The duration of the CVA and the lesion side are shown. A clinical evaluation of the subject's knee spasticity was performed (5 point Ashworth scale). The presence of ankle clonus was recorded which was evoked by a quick passive dorsiflexion about the ankle.

Pendulum Test

Typical pendulum test plots are shown in Figure 1. Of the thirty plots derived from the CVA group, all thirty demonstrated abnormal features when compared with the plots from the control group. None of the thirty plots from the control group showed abnormal features. It is apparent when comparing the two plots that the EMG response is greater in the CVA subject than in the control subject. The highly damped sinusoidal waveform of the CVA subject's pendulum test is strikingly different from the symmetrical, pendular waveform derived from the pendulum test of the control subjects. The number of oscillations is also fewer for the CVA subject when compared with the control (Figure 4). As well the amplitude of the initial oscillation is less for the CVA subject (Figure 4).

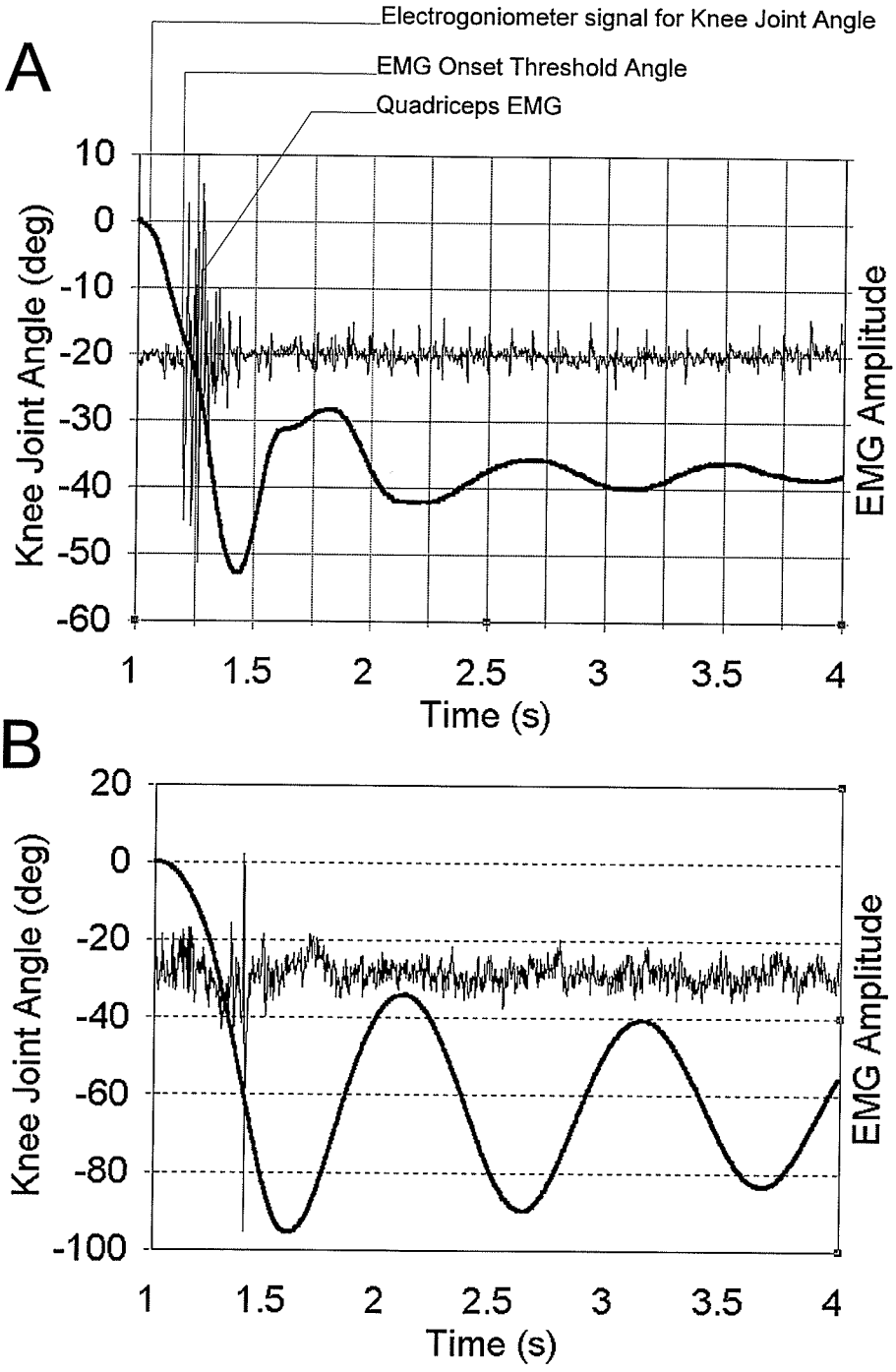

Figure 1: Typical pendulum test plots for a CVA patient (top panel) and an able bodied control subject (bottom panel). The EMG (mV) activity for the CVA subject is greater than for the control subject. The amplitude of the limb excursions is greater in the control subject and highly damped in the CVA subject. The threshold angle is also greater in the control subject. The resting angle for the control subject does not appear in the trace.

Figure 2 shows typical phase-plane plots derived from the velocity and angle data from the pendulum test. Again the differences between the CVA subject and the control subject were clear for all subjects. The CVA subject's trace is asymmetrical when compared with the control subject. The trace for the control subject resembles a whirlpool. The phase-plane plots were capable of identifying the CVA group by demonstrating the qualitative differences between a spastic and a normal limb. The mean values observed for the pendulum test for threshold angle, Relaxation Index, latency, and number of oscillations is shown in Table 2.

		Mean	SD	SE	MIN	MAX
TA	CVA	20.53	6.94	2.19	13.12	20.53
	control	36.55	16.44	5.81	19.8	64.37
RI	CVA	1.32	0.173	0.055	1.04	1.54
	control	1.52	0.145	0.046	1.39	1.8
LAT	CVA	180	52	16	95	250
	control	256	79	28	147	384
OSC	CVA	4.67	1.025	0.324	3.3	6.6
	control	6.29	1.148	0.363	4.6	8.0

Table 2. Comparison between the CVA group and the control group of pendulum test parameters. TA - threshold angle (°), RI - relaxation index, LAT - latency (ms), OSC - number of oscillations.

The mean threshold angle for the pendulum test for the stroke group was 20.53° (SD = 6.94°, SE= 2.197°). The range was from 13.12° to 30.59°. The mean threshold angle in the control group was 36.55° (SD = 16.44°, SE= 5.816°). The range of responses was from 19.8° to 64.37°. This difference is significant at a p value of 0.013.

Relaxation index (RI) was calculated as the amplitude of the initial swing dividied by final resting angle. The mean RI for the stroke group was 1.322 (SD = 0.173, SE = 0.055) the range was from 1.04 to 1.54. The mean RI for the control group was 1.52 (SD = 0.145,

SE = 0.046) The range was from 1.39 to 1.8. These differences are significant at a 0.01 level.

The mean latency of the EMG response for the stroke group was 180 ms (SD = 52 ms). The range was from 95 ms to 250 ms. For the control group the mean latency was 256 ms (SD = 79 ms, SE = 28 ms). The range was from 147 ms to 384 ms. These values are significantly different at a 0.025 level.

The mean number of oscillations per trial for the stroke group was 4.67 (SD = 1.025, SE = 0.324). The mean number of oscillations for the control group was 6.2 (SD = 1.14, SE = 0.363). These results are significantly different at a 0.004 level.

Figure 3 summarizes the data in bar graph format from the pendulum test. Significant differences were found between the groups for threshold angle, Relaxation Index, EMG response latency and the number of oscillations.

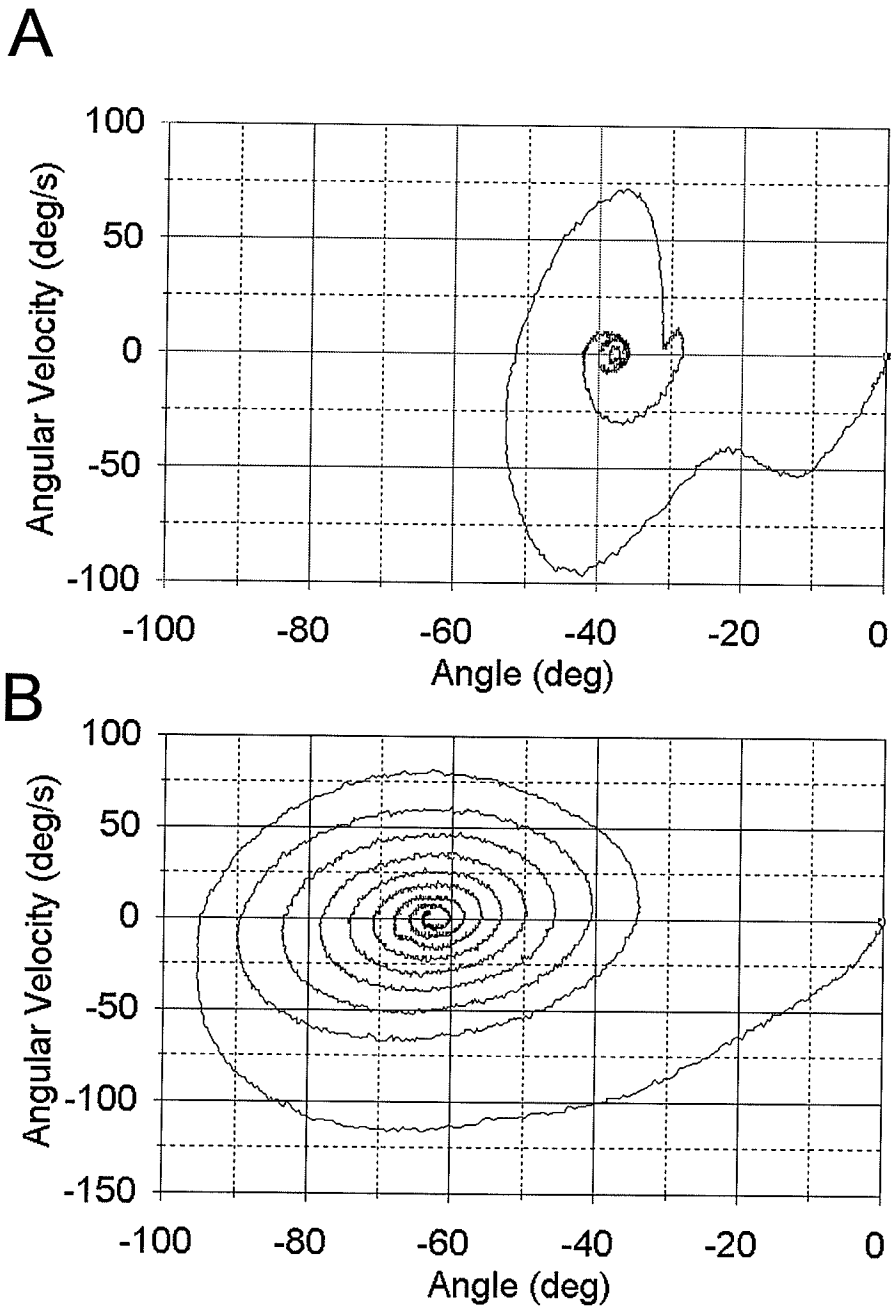

Figure 2: Phase-plane plots for a typical CVA subject (A, top panel) and typical control group subject (B, bottom panel). The asymmetrical plot for the CVA subject is easily distinguished from the symmetrical plot of the control subject.

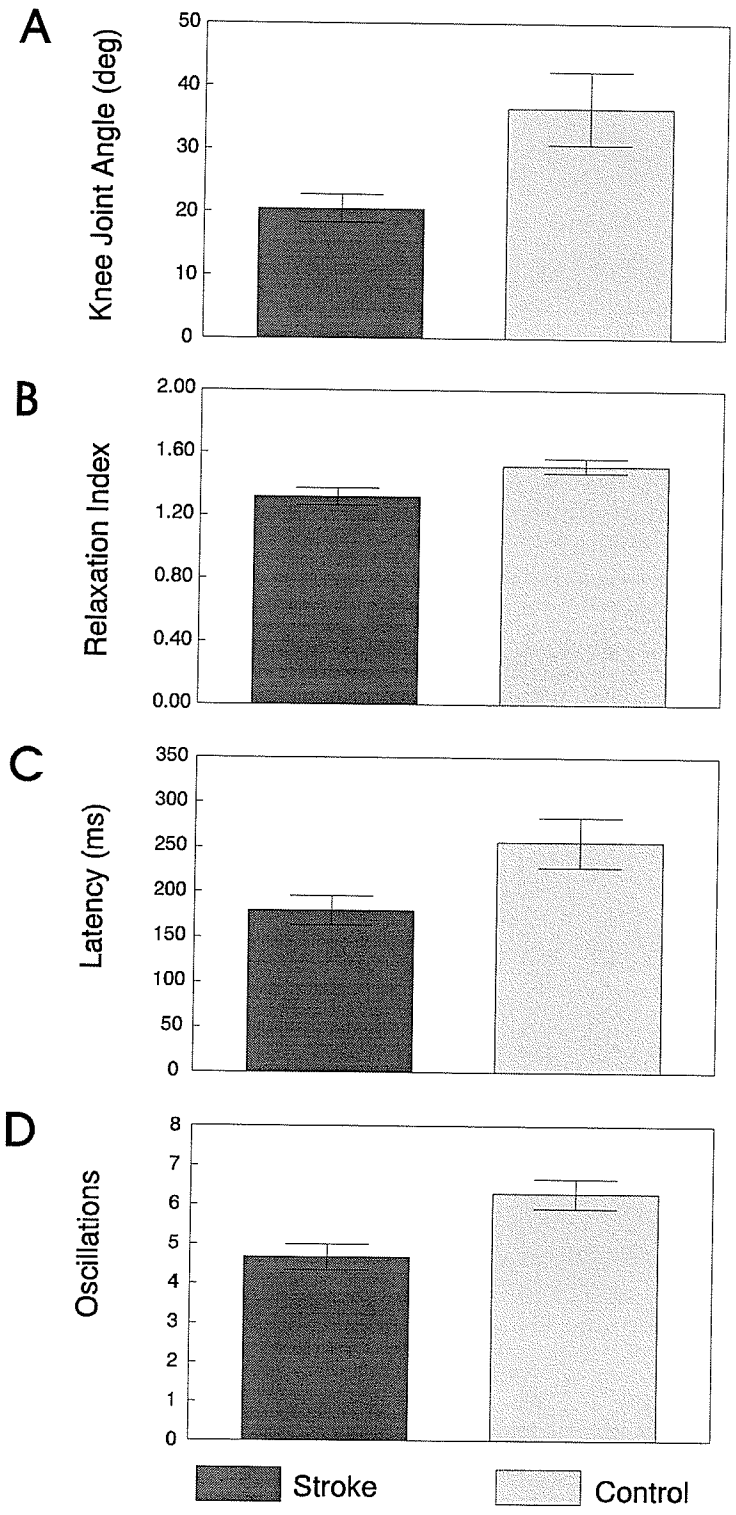

Figure 3. Mean pendulum test results with standard error shown for A. threshold angle, B. Relaxation Index C. latency and D. number of oscillations. The difference between the groups is significant ($p>0.05$) for each measure.

Figure 4 depicts the decay of the angular displacement waveform for both groups. The mean amplitude of oscillations is initially less for the CVA group. The control group shows a gradual decay of the waveform as the leg gradually stops swinging. The CVA group shows a more abrupt decay. The damping effect of the limb decreases the amplitude of the oscillations, resulting in a smaller displacements for each excursions and fewer total swings.

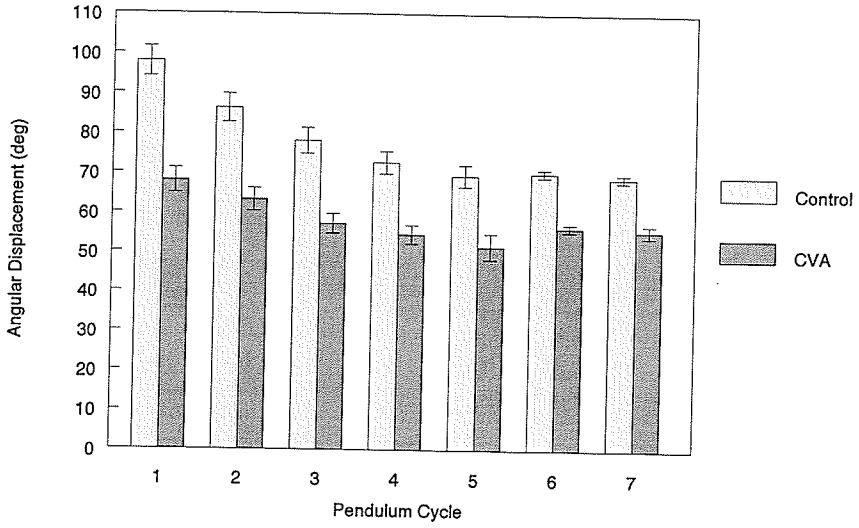

Figure 4. Pendulum test decay. The decay of the amplitude of successive swings in the pendulum test. The curve for the CVA group decays more abruptly than the curve for the control group, indicating an increased damping effect. All control subjects had swings equal to or greater than 7. The number of CVA subjects demonstrating swings greater than 3

Ramp and Hold Test

Figure 5 and figure 6 show raw plots at 180°/s with high acceleration for a control subject and a CVA subject respectively. An EMG response was not observable for the control group subject. The baseline EMG remains constant throughout the duration of the stimulus. The CVA subject shows a large EMG response following the onset of the ramp displacement. These results were typical of those observed for all subjects in each group.There were 3 trials for each subject at each test velocity which equals a total of 90 trials per group. In the control group there was a totalof 7/90 responses for all trials. In the CVA group there was a total of 54/90 responses.

The Ramp and Hold test revealed several differences between the CVA group and the control group. There were no observable EMG responses in either group at velocities of 45°/s or 90°/s. The most notable was the absence of responses in the control group at the higher velocities. Threshold angle for the CVA group was smaller than for the control group. At 135°/s the mean threshold angle was 28.9° (SD=14.63°, SE=5.8°) for the CVA group. There were no responses among the control group at this setting. The mean latency at this setting for the CVA group was 234 ms (SD=115 ms, SE=66 ms). Mean duration of the EMG response was at 135°/s for the CVA group was 338.6 ms (SD=178.0).

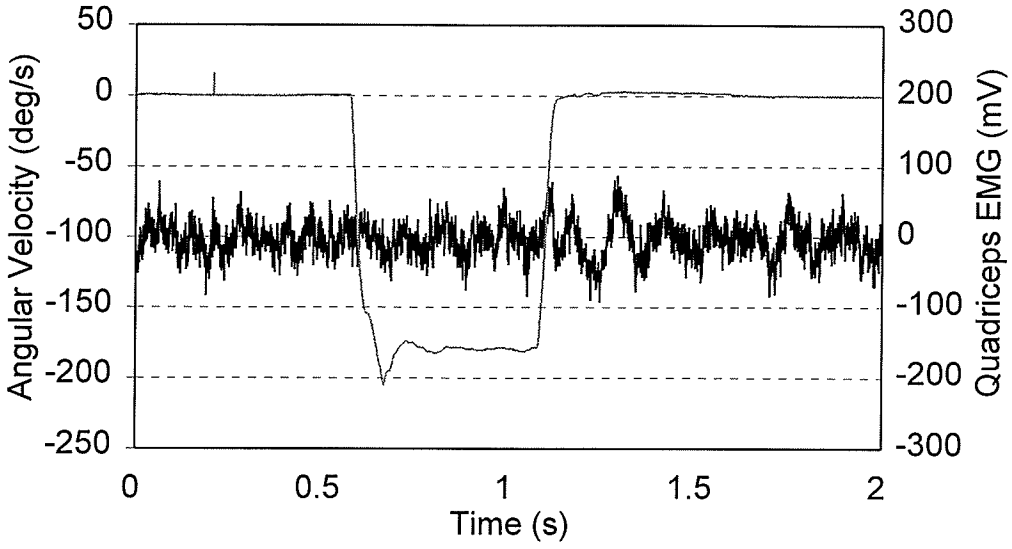

Figure 5 Typical Ramp and Hold test raw plot for a control group subject. The test velocity is 180°/s and the acceleration setting is high. There is no discernible increase in quadriceps EMG from baseline following the onset of displacement. The EMG level is non gain corrected.

At 180°/s with low acceleration the mean threshold angle for the CVA group was 32.65° (SD=12.21°, SE=5.8°). Only one of the control group subjects had an EMG response at this test velocity. The threshold angle was 31°. The mean latency for the CVA group at this setting was 216 ms (SD=72 ms, SE=29 ms). For the control subject the latency was 171 ms. The mean duration of the EMG response for the CVA group was 174.03 ms (SD = 82.03). EMG duration for the control subject was 186 ms.

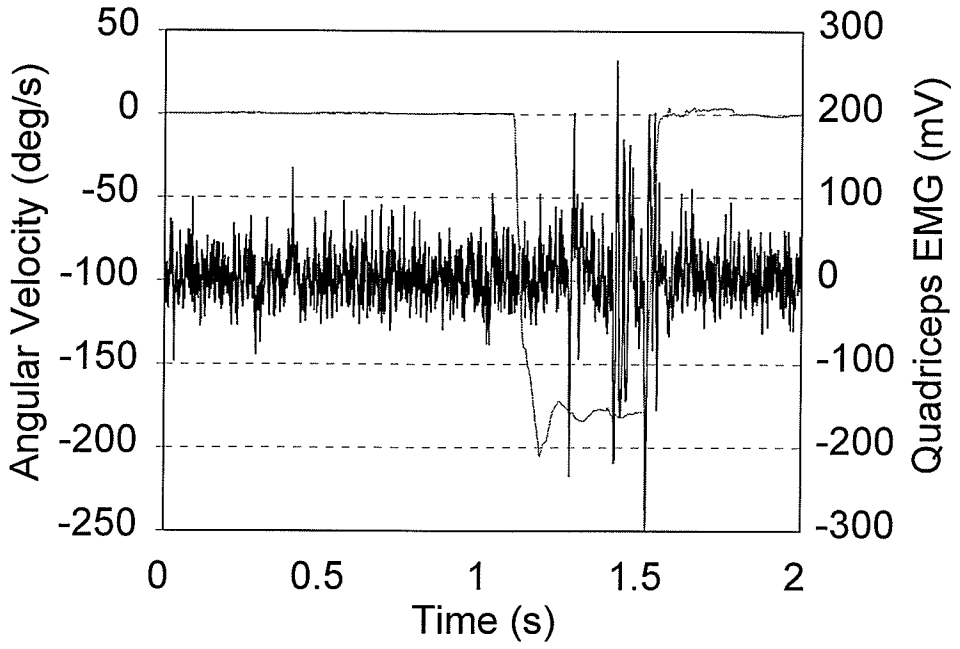

Figure 6 Typical Ramp and Hold test plot for a CVA subject. The test velocity is 180°/s and the acceleration setting is high. The stretch evoked reflex response of the quadriceps is seen shortly after the onset of the displacement. It lasts until the limb reaches 90° of knee flexion.

For the setting at 180°/s with high acceleration the mean threshold angle for the CVA group was 29.42° (SD=11.96°, SE=3.98°). Four of the control group subjects showed small, short duration EMG responses at this setting. The control group, mean threshold angle was 53.17° (SD=12.24°, SE=6.12°). The mean response latency for the CVA group was 170 ms (SD=64 ms, SE=21 ms). For the control group the mean latency was 186 ms (SD=50 ms, SE=25 ms). Mean duration of the EMG response for the CVA group at this setting was 204.34 ms (SD=105.86). The mean duration for the control group was 79.82 ms (SD=42.26, n = 4).

Figure 7 summarizes the results for the Ramp and Hold test. Significant differences were found for the EMG area and the threshold angle. The latency of the EMG response was not significantly different between groups. A general shift in latency toward shorter values was present for the CVA group as the velocity and acceleration is increased. Velocity dependence, as determined by visual inspection, was only observed in three subjects in the CVA group.

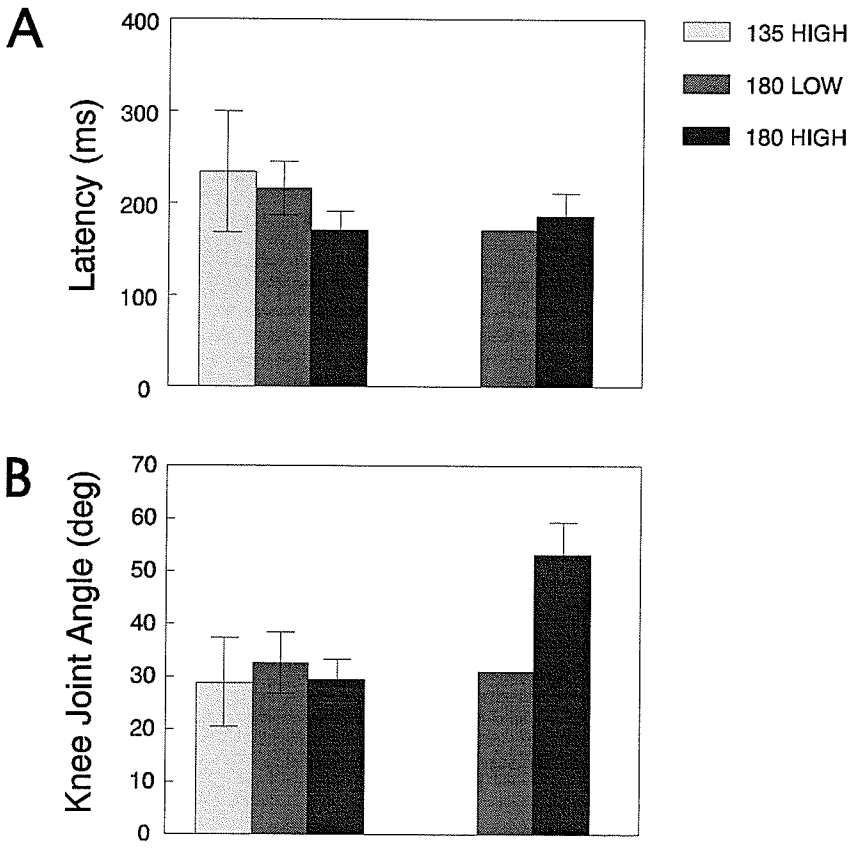

Figure 7: Mean Ramp and Hold test results with standard error bars shown for A. latency and B. threshold angle.

Table 3 summarizes the general responses of the two groups to the Ramp and Hold tests. The most striking observation resulting from the Ramp and Hold test is the lack of response to the stimulus in the control group. Only four of the ten control subjects had any EMG response in all of the Ramp and Hold tests and this occurred at the highest velocity and acceleration setting. In the stroke group the greatest number of responses was observed at the high velocity and acceleration condition, but the EMG response was seen in nine of the ten subjects. There were also more CVA subjects who showed an EMG response at the lower velocity and acceleration settings. The CVA group showed an acceleration effect. The control group showed this effect in 50% of the subjects who showed an EMG response. The control group showed adaptation of the stretch reflex response whereas the CVA group did not.

Another important observation is the variability of the responses in the stroke group. This was a relatively homogeneous group in terms of their diagnoses, yet this is not reflected in the responses. The variability is seen mainly in the magnitude and duration of the EMG response. This may be due to differences in the site of the CNS lesion for each subject, resulting in differences in descending control.

	Threshold Velocity	Acceleration Dependency	Velocity Dependency	Adaptation
CVA				
1	135°/sec	yes	yes	no
2	180°/s (low)	yes	no	no
3	180°/s (low)	yes	no	no
4	-	-	-	-
5	180°/s (high)	yes	no	no
6	135°/sec	yes	yes	no
7	180°/s (low)	yes	no	no
8	135°/sec	yes	yes	no
9	180°/s (high)	yes	no	no
10	180°/s (high)	yes	no	no
Control				
1	-	-	-	-
2	180°/s (high)	yes	no	yes
3	-	-	-	-
4	-	-	-	-
5	-	-	-	-
6	180°/s (high)	yes	no	yes
7	180°/s (low)	no	no	no
8	180°/s (high)	yes	no	no
9	-	-	-	-
10	-	-	-	-

Table 3. General responses for the Ramp and Hold test for the CVA group (top table) and the Control group (bottom table). The threshold velocity corresponds to the angular velocity at the onset of the quadriceps EMG. "High" and "low" refer to the acceleration setting. Acceleration dependency was determined by evaluation of the quadriceps EMG area at the 180°/s test speed (if the EMG response was greater with increased acceleration). Velocity dependence required an increasing quadriceps EMG with increasing velocity. Adaptation was determined by the variability of the EMG response to consecutive ramp stimuli (if the EMG response diminished with consecutive stimuli, then response adaptation was present).

The mean duration of the EMG response following the vibratory stimulation for the CVA group was 187.58 ms (SD=115.35) which was slightly less (16.76 ms) than the duration for the trials without vibration. For the control group the mean duration was 48.15 (SD=23.34) for the vibration trials. The difference in EMG duration for the control group between trials at 180°/s, high acceleration and trials with vibration was 31.67 ms. This difference was not statistically significant.

EMG duration was lower in the control group with vibratory stimulation but the difference was not statistically significant. It appears that in the control subjects where an EMG response was elicited, that the vibration diminished the area of the response. In the CVA group there was no significant change with the vibration. Given the small number of responses evoked in the control group, the trend observed must be considered with caution and further work in this area is indicated.

Using cross-correlation analysis, a positive, significant correlation existed between threshold angle from the pendulum test and threshold velocity from the Ramp and Hold test and the presence of clonus ($p < 0.005$). A lack of correlation existed between the Ashworth test and any other measure from the Ramp and Hold test or the pendulum test.

Discussion

The purpose of this study was to examine the stretch evoked reflex response in the quadriceps muscle in CVA patients. In the present study two methods of objectively quantifying spasticity were applied to a group with a single diagnosis, hemiplegia after CVA. The term spastic hemiplegia is called into question when observing the clinical signs of these subjects. However the objective data would indicate that these subjects indeed have spasticity. Using EMG recording with the pendulum test and the Ramp and Hold test, the stretch evoked reflex activity of the quadriceps muscle was observed. Significant differences in EMG response between the CVA group and the control group were detected with both tests. The responses between the groups differed in both qualitative and quantitative ways. Although the anticipated velocity dependent response did not exist in the control group and was not pronounced in the CVA group, the EMG activity between groups differed in other ways. For instance, the velocity effect was manifested by the earlier threshold velocity for the CVA group.

One objective was to compare commonly used clinical tests for spasticity (Ashworth test, ankle clonus) to objective tests (pendulum, Ramp and Hold). The correlations between these tests was determined. Only ankle clonus was positively correlated to threshold angle in the pendulum test and threshold velocity in the Ramp and Hold test. The Ashworth test did not correlate with any of the objective measures. The multi-factorial nature of spasticity may account for this observation. These tests may be measuring different physiological components of spasticity.

Pendulum test

The pendulum test displayed enough sensitivity to differentiate between the CVA group and the control group when the following parameters were measured: threshold

angle, number of oscillations, Relaxation Index, EMG latency and the amplitude and decay of the swing oscillations. These are all indicators of initial stretch reflex activity. It is not possible to determine whether the response is monosynaptic or otherwise. The parameters measured for the pendulum test were significantly different when the groups were compared. The subjective measure of the Ashworth test was sensitive in detecting spasticity in only a few CVA subjects. However, abnormal neuromuscular activity was observed with the pendulum test in subjects who had Ashworth scores of zero. The presence of clonus was well correlated with the threshold angle of the pendulum test. Ankle clonus is a velocity sensitive test detected by a brisk passive foot dorsiflexion. It is likely a maesure of altered reflex gain in gastrocnemious and soleus. The correlation of ankle clonus with the threshold angle from the pendulum test indicates that the stretch dependent reflex responses about the knee and ankle are altered in both groups. This may be an indication of altered pathophysiology for that region of the spinal cord level.

The quantifiable information which can be gleaned from the pendulum test is the number of oscillations, amplitude and decay of oscillations, Relaxation Index, the threshold angle and the EMG latency. The number of oscillations is calculated by counting the number of swings from release of the limb until its final resting point. The amplitude of the oscillations can also be measured from the pendulum data. By calculating the amplitude of each successive oscillation, the decay of the sinusoidal waveform can be graphically represented. The decay of the oscillations reflects the rate of damping on the limb after release. The rapid decay of the spastic limb can be attributed to an enhanced quadriceps stretch relfex but may also be due to co-contraction of the quadriceps and hamstring muscles. Although this was not measured in this study, previous studies have detected hamstring activity during a pendulum test (Lin and Rymer, 1991).

Threshold angle and EMG latency are parameters which relate to the time of onset of the stretch reflex relative to the onset of limb motion. The onset reflects the synchrony of discharge of Ia fibers in response to the stretch stimulus and the resulting excitatory

effects on the homonymous and synergistic motor units. Previous studies on the pendulum test have not measured these parameters (threshold angle and EMG latency). Threshold angle was smaller and latency was earlier in the CVA group reflecting that the contribution of the Ia fibers to motoneuron excitation resulted in an earlier activation of motor units than in controls.

If the above parameters are calculated, then the calculation of a Relaxation Index become redundant. The studies using RI in the literature all seem to use a different formula to make the calculation so comparison of results is difficult. In some cases, the definition of joint angles was absent (e.g. whether full knee extension is equal to 180° or 0°) making comparisons difficult. Katz et al (1992) describes RI as the amplitude of the initial swing (A1) divided by final resting angle (A0). He observed that in the control group this value usually exceed 1.6, so he introduced a corrected RI. This value is calculated as A1 divided by 1.6 times A0. Using this equation, an RI of 1 would indicate a non-spastic limb and RI of less than 1 would indicate varying degrees of spasticity. Brown et al (1988) calculated the RI as the amplitude of the first swing divided by final resting angle. If there was a slight inflection (an incomplete reversal due to reflex activity) observed in the trace of the CVA group, the calculation was altered to account for this. Bajd and Vodovnik (1984) calculated the RI as the magnitude of the first drop divided by the magnitude of the initial angle. They also used a corrected value of 1.6 to normalize the index. In all previous studies, all of the oscillations occurring during the pendulum test are not considered and any additional information that could be gleaned is therefore lost. The phase-plane plot developed by Brown et al (1988) gives a qualitative picture of the whole test. It is not quantified in any way but the distinction between a spastic and normal limb can be made quickly through visualization. In this study, the CVA group phase-plane plots were all readily identifiable as abnormal by deviating from the symmetrical whirlpool shape reflecting the potential clinical utility of the plot. Further study into the quantification of the shape of the phase plane plot is warranted, such as an index of symmetry.

Several researchers also calculated the maximum velocity of the initial swing (Bajd and Vodovnik, 1984; Brown et al, 1988). A difference in peak velocity was demonstrated between groups, with the control group achieving higher velocities. Higher velocities are likely obtained due to a smaller damping effect resulting from the enhanced contribution of the stretch reflex pathways in the 'spastic' groups. In this study, the threshold angle was significantly earlier (mean difference of 16 degrees) in the CVA group likely reflecting an enhanced gain of the stretch reflex (lower velocity input for a larger EMG activation) in comparison to the control group.

Some subjects (approximately 3) had difficulty adequately relaxing the limb (both CVA and control). This may be a limitation of this test, however through careful observation the trials during which the subject actively assisted the swinging of the limb were not used. It is important that in future studies or in clinical applications using the pendulum test that the evaluator be aware of the state of relaxation of the individual. Fewer oscillations and a greater damping effect in the CVA group may be due to increased in the quadriceps and hamstring muscle groups stiffness due to enhanced stretch reflex gain. The normal damping effect observed in the control group results in a symmetrical waveform whereas in the CVA group there is an obvious asymmetry. The asymmetry seen in the CVA group may be explained by the enhanced gain of the quadriceps stretch reflex and possibly due to over-lapping activity in the quadriceps and hamstrings resulting in a brief period of co-contraction. The co-contraction would interfere with the natural swinging motion of the limb. This co-contraction could be partially explained by a decrease in reciprocal inhibition to the antagonist muscle group, however a decreased reciprocal inhibition would not alone account for the activation of hamstring muscles. Likely the returning swing (from knee flexion to extension) results in a stretch evoked reflex response in hamstring muscles which may overlap with the quadriceps activity if reciprocal inhibition is diminished in the CVA group. Katz and Pierrot-Deseilligny (1982) were able to demonstrate decreased reciprocal inhibition when testing patients with upper motor neuron lesions. Further research into the role of reciprocal inhibition in the CVA group is warranted.

Generally, for the control group there was a very brief burst of quadriceps EMG during the first oscillation. For several of the CVA group there was a quadriceps reflex response observed for the first few oscillations. Where there was only an initial burst in the CVA group, it tended to be of longer duration than that seen in the control group. For these subjects the amplitude of the displacement was greater. So, in the CVA group the stretch evoked reflex activity would result in impeding the free swinging motion of the limb, causing increased damping effect and asymmetry in the phase-plane plot. In the control group the damping effect is arising primarily from the passive properties of muscles and ligaments which span the knee joint (intrinsic 'friction') with a small reflex contribution, if any. As such, the reflex activity did not interfere with the free swinging pendular motion of the limb.

Another limitation of only recording from the quadriceps is that muscle activity around other joints, such as the hip and ankle, is not considered. Two points are necessary to consider here. First, the contribution of biarticular muscles, such as rectus femoris, to the action about the knee. The starting joint angle of the knee and hip play a role in the potential reflex contributions from a muscle like rectus femoris. Control of two joint angles during testing is necessary in order that one does not confound the results. This would be applicable to the pendulum test, and the Ramp and Hold test, as well as Ashworth evaluations. Second, considering that the lesion site in all of the subjects is variable, the manifestation of the lesion on descending control of reflex pathways may be observed about joints other than the knee (hip and ankle). Interestingly, the correlation of ankle clonus and the result of both objective tests may reveal that the manifestation of spasticity may not be joint specific. Further research which evaluates the spasticity about multiple joints (e.g. Ramp and Hold tests of ankle, knee and hip muscles) on the same patient would provide insight into this area.

The pendulum test provides clinicians with an objective measure of spasticity which is relatively inexpensive and easy to administer and interpret. A number of quantifiable

parameters can be readily derived, as well as the immediate visual, qualitative feedback provided by the phase-plane plots. Given the current microprocessor technology, a real-time, hand-held pendulum testing apparatus could be developed. The pendulum test can be used to assess the effectiveness of medication used to control spasticity or to determine the time course of spasticity after CVA or spinal cord injury.

Ramp and Hold Test

It was hypothesized that a velocity dependent stretch evoked reflex would be observed in the CVA group. For the CVA group there was a tendency for more responses at the earlier velocities. This required that the EMG response would increase with an increase in velocity. This effect was seen in only three of the ten CVA subjects. The threshold velocity at which the stretch evoked reflex was first observed was lower for the CVA group than for the control group. Threshold velocity is defined as the velocity at which the first EMG response occurs. Had higher velocities been used for the study, a velocity dependence may have been elicited. The velocities used were chosen to ensure that the subjects could tolerate the highest setting without risk. It is now known that CVA subjects would be able to tolerate higher velocity settings. A pilot study on a small group of CVA subjects which examines the range of velocities needed to evoke larger responses from the quadriceps is warranted. However, the Kin-Com 500H dynamometer has maximum velocity of 250°/s which is only one step greater than the maximum used in this study.

The effect of altering the acceleration was observed in all CVA subjects, even those with Ashworth scores of 0. This acceleraton dependent response appears to be a useful method to distinguish the CVA subjects from control in terms of the duration of stretch evoked responses. In these subjects, increasing the acceleration from low to high settings while retaining the maximum velocity of 180°/s resulted in an increased quadriceps EMG response. This acceleration effect can be interpreted as a modified form of velocity

dependence since dynamic responsiveness of the primary endings of muscle spindles would correspond to acceleration detection. Also, high acceleration means that the passively displaced limb reaches the constant velocity portion of the ramp displacement sooner. As such the Ia discharge may become more synchronous resulting in a greater likelihood of temporal and spatial summation of excitatory post-synaptic potentials in the homonymous and synergistic motoneurons. In the CVA group the EMG duration was greater at 135°/s with high acceleration than it was at 180°/s with low acceleration. This clearly demonstrates an acceleration dependency of stretch evoked reflex activity. The earliest component of stretch reflex response has been demonstrated to be due to Ia fibers. The response of the Ia fibers will appear earlier as higher accelerations. The more robust EMG response observed with the high acceleration would indicate that the spindles (and the resulting Ia activity) have some acceleration sensitivity. Many receptors have been shown to have dynamic response characteristics. Dynamic responsiveness of spindle velocity sensors would correspond to acceleration sensitivity. Another explanation for the enhanced response may be that the Ia fibers respond in a more synchronized burst with high acceleration. The brisk, high acceleration stimulus coordinates and synchronizes the activity of the Ia fibers likely resulting in greater summation of excitiatory events in the motoneurons.

There was no significant difference in response latency between groups, however the area of the EMG response in the CVA group was larger. It is possible that Ia fibers are activating a greater amount of or different excitatory, pre-motoneuronal circuits due to altered descending control, resulting in more motoneurons reaching threshold to the velocity stimulus in the CVA group than in the control group. This would be consistent with either descending pathways disfacilitating the inhibitory pathways which influence stretch reflex pathways in the CVA subjects or enhanced descending excitatory effects on stretch reflex pathways. One potential difference could be the difference in presynaptic inhibition of Ia input to motoneurons. In this study, the trend observed for the vibratory inhibiton (no vibratory inhibition in the CVA group) is consistent with the notion that there may be a decreased amount of presynaptic inhibition contrbuting to the enhanced

stretch reflex response in the CVA group. Since the control group demonstrated a mean decrease in quadriceps EMG with vibration and this difference was not statistically significant, the speculation that the CVA group has diminished presynaptic inhbition must be considered with caution. Further study into this potential mechanism for enhanced spasticity is warranted. In order to have definitive information on this mechanism, vibratory inhibition in the control group must be conclusively demonstrated which would require a greater stimulus to evoke a consistent EMG response during the Ramp and Hold test.

Lee et al (1987) used force measurements of voluntarily activated muscles during the dynamic phase of a passive stretch to evaluate reflex stiffness in the biceps muscle of hemiplegic subjects. Voluntary activation of the muscle facilitates the stretch reflex pathway, in effect setting it at a higher gain. A higher loop gain means that the input (passive joint displacement) produces a larger than normal output (higher peak force). The parameters measured were time to peak force and the magnitude of the peak force. The parameter of peak force was thought to reflect the joint stiffness of the biceps muscle. Joint stiffness includes: passive tissues, muscle stiffness and reflex stiffness. When recording EMG responses they found that the magnitude of the response was in fact greater on the less affected side in the hemiplegic subjects. They also found that the stiffness was comparable from side to side. There were no measurements of threshold made during this study. A system operating at a higher gain, where there is a greater baseline level of motoneuron depolarization, would result in earlier response latencies as well as greater response magnitude. The results of this study, in terms of the pendulum test and Ramp and Hold test are consistent with a higher gain, as well as a lower threshold for activation of the stretch reflex pathways.

Thilmann et al (1991) also found no difference in stretch reflex latency in the biceps muscle of hemiparetic subjects when compared to normal controls. They did however observe a prolonged EMG response with increasing velocity. The present study also found a prolonged duration of the EMG response at the highest setting in the CVA group.

The prolonged response was only present at settings with high acceleration. The mean duration at 180°/s with high acceleration for the CVA group was 34.8 ms and for the control group it was 12.9 ms. Thilmann et al (1991) also demonstrated a shift in EMG latency in the biceps muscle of CVA subjects with increasing velocities. He used velocities up to 240°/s to elicit this effect. In this study, a shift in latency was also observed in the control group, but the latencies were generally later (though not significantly) than for the CVA group.

Lee et al (1987) compared two groups of CVA subjects, each at different stages after stroke. They found that the response latencies and durations were different in the two groups. The late response activity was seen primarily in subjects where the spasticity was well established. The authors attribute these changes to a change, over time, in the gain of the reflex pathways. The significant difference in latency observed by Lee and coworkers was not observed in this study or in the work reported by Thilmann and coworkers. This may be due to the difference in their protocol. Lee et al (1987) used an active test whereas Thilmann and coworkers (1991) and this study used a passive technique.

Powers et al. (1988) tested the biceps muscle of CVA subjects. Their protocol tested both passive and initially activated muscles. The velocities ranged from 0.25 rad/s to 2.0 rad/s (15°/s to 115°/s). They recorded torque, stiffness and EMG response onset. The results were not quantified in any way. This is because they did not feel that the EMG onset was related to the mechanical reflex threshold of the joint. They observed that an increase in torque and stiffness in the elbow flexors was accompanied by the onset of EMG activity. No such increase was observed in the control group. The authors attribute these differences to a decrease in stretch reflex threshold in the CVA group. A similar study done by Powers et al (1989) recorded torque and EMG activity responses following passive displacement of pre-activated elbow flexor muscles in hemiparetic subjects. They observed that the stretch reflex characteristics are similar under the condition of pre-activation in normal and spastic muscles. They did not observe a velocity dependent response in initially pre-activated muscle. Velocity dependence was observed with passive

muscle displacement. In this study, the angular velocities imposed were higher than those used to demonstrate a velocity dependent response in the biceps. This could be due to the difference in geometry and passive qualities (elasticity) of the muscles which span the knee and elbow. The different geometry and passive properties would result in different linear velocities at the muscle fiber level. It is the linear velocity which is detected by the primary spindle afferents. The perturbations imposed in this study correspond to an acceleration (increase in angular velocity) to achieve a near constant angular velocity for the period followed by a deceleration (slowing down). The linear velocity of the muscle fibers is not readily determined but certainly would increase with increasing angular velocity. As such it is difficult to compare angular velocities used about different joints since the corresponding linear velocities are indeterminate.

Rack et al (1984) used passively imposed sinusoidal displacement of the ankle to observe the stretch reflex characteristics in hemiparetic subjects. They found that under prolonged testing conditions the normal subjects displayed the same characteristic responses as the hemiparetic subjects. In the present study, adaptation to the stimulus was observed in the control group over the course of three repetitions. The differences observed between this study and the work of Rack and colleagues could be attributed to the difference in the duration of testing. Under normal test conditions the normal subjects did not display any reflex response. Another observation from this study was that the individual CVA subjects demonstrated more consistent EMG responses over the course of three repetitions. However, the differences in the overall duration of responses in the CVA group were quite variable.

The present study also found that the stretch evoked reflex response in the control group showed adaptation to the stimulus. When a reflex response was observed in a control group subject it occurred only during the first of three trials at the test velocity. This variability for the control subjects from one repetition to the next could indicate that the highest velocity stimulus used (180°/s, high acceleration) was just at threshold for stretch reflex activation or that an adaptive circuit was involved which lowered the stretch

reflex loop gain with successive stimuli. In the CVA group, if a reflex response was present it occurred during all three trials. Stretch evoked reflex responses are mediated by the Ia afferents via a monosynaptic reflex loop. There are reflex circuits within the spinal cord which can alter the excitability of the Ia fibers. These circuits are subject to inhibitory or facilitatory input from the higher centres. It is likely that the absence of adaptation in the CVA group is due to altered descending control as the injury is in the cerebral cortex. If the adaptation seen in the control group is due to an inhibitory mechanism, then it would follow that the lack of adaptation in the CVA group would be due to decreased inhibitory input on stretch reflex pathways. Burke et al (1970, 1971) did find adaptation in subjects with lower limb spasticity using passive displacement of the lower limb. They do not report the number of repetitions nor the velocities used in their study. They also do not compare the results to a control group.

Katz et al (1992) measured threshold angle and torque in the biceps muscle of hemiparetic subjects during ramp displacements. He found a strong correlation between threshold angle and spasticity as measured by the Ashworth test. The present study failed to find such a correlation. Katz however was studying the elbow joint. Perhaps the Ashworth test is adequate to detect varying degrees of spasticity in the elbow. We found the presence of ankle clonus to be a more reliable indicator of abnormal stretch evoked responses in the quadriceps. The quadriceps is also a much larger muscle group with different muscle geometry and passive tissue qualities to try to assess with a manual test.

The trend toward vibratory inhibition that was observed for the control group is consistent with previous studies. Vibratory inhibition is thought to operate by "locking in" the Ia fibers to the frequency of the vibration so that they will not respond to any other stimulus, including stretch. It has been demonstrated that presynaptic inhibition of the Ia pathways occurs for hundreds of milliseconds after the termination of vibration-evoked Ia activity (Pierrot-Deseilligny, 1990). By "locking in" the Ia response through vibration and then terminating the vibration just prior to the ramp displacment, it was anticipated that the control group would exhibit vibratory inhibition while the CVA group did not. The

absence of vibratory inhibition in the CVA group would concur with the postulation that spasticity is in part due to a decrease in presynaptic inhibition. This would be a result of altered descending control due to CVA. If the presynaptic inhibitory mechanisms were functioning in the CVA group the relative degree of inhibition may have been greater in this group than in the control group, because their response to the stimulus is greater. The results of this study permit the speculation that presynaptic inhibition in the CVA group is lost in the Ia stretch reflex pathway. The vibratory inhibition is only weakly demonstrated in the control group because the EMG response was not large. Had higher velocities been used the effect may have been more apparent. This effect of a vibratory stimulus on passive displacement of spastic limbs has not been previously documented.

One observation is that there were more stretch evoked reflex responses of the quadriceps muscle during the pendulum test than during the Ramp and Hold test. This may be due to the method of administration of the tests. The peendulum test is a manual test. The limb is held by the examiner and released. It then swings freely whereas during the Ramp and Hold test the limb remains attached to the actuator arm of the dynamometer. The amplitude of the excursion is controlled during the Ramp and Hold test whereas during the pendulum test the amplitude of the swings is not controlled. It was observed during the pendulum test that the first oscillation often exceeded 90°, especially in the control group. A greater angle of excursion may be the reason for the more frequently observed quadriceps EMG activity. There is a greater degree of stretch on the muscle.

Conclusions

The CVA group demonstrated significantly smaller threshold angles and threshold velocities than the control group during the pendulum test and the Ramp and Hold test. The CVA phase-plane plots derived from the pendulum test data revealed asymmetrical, highly damped sinusoidal behaviour characteristics of exaggerated stretch reflex. The number of oscillations for the CVA group was less than for the control group. The quadriceps EMG activity was observed to be present in the first few swings for the CVA group subjects. This type of prolonged response was not observed in the control group.

The Ramp and Hold test data did not reveal a velocity dependent response in the CVA group as anticipated. This Ramp and Hold test did, however, have sufficient sensitivity (velocity threshold and acceleration dependency) to demonstrate the enhanced stretch reflex in the CVA group when compared to the control group.

The EMG response for the pendulum test differed from the response elicited during the Ramp and Hold test. The response during the pendulum test seemed to outlast the stimulus or be renewed with each successive swing. During the Ramp and Hold test only an early response (response during the ramp displacment) was observed. There was no response which outlasted the stimulus, eg. no length dependent response.

An acceleration dependent change in the quadriceps EMG was observed in the CVA group, as well as in two of the control subjects. Given the acceleration dependent increase in the quadriceps EMG, with no shift in the EMG response latency, an acceleration response of the Ia spindle afferents may be surmised. Another explanation for the enhanced EMG response may be that the Ia fibers are firing more synchronously with higher accelerations.

The adaptation of the EMG response in the control group and its absence in the CVA group indicate a decrease in inhibition (lack of an adaptive inhibitory mechanism) for the CVA group.

This study has shown a trend toward vibratory inhibition in the control group and the absence of such inhibition in the CVA group. Decreased presynaptic inhibition may be partially responsible for the enhanced stretch reflex response by increasing reflex gain and dcreasing reflex threshold. It is likely that angular velocities approaching 250°/s would be needed to defintively demonstrate that the vibration resulted in inhibition of the stretch reflex in controls and did not have an effect in the CVA group.

The variability in the EMG response in the CVA group may be a reflection of differences in CNS lesion site. Different lesions would alter the descending pathways in different ways. The manifestation of these differences is apparent in the variability of the responses.

Correlation of the derived measures of the spasticity tests revealed a positive correlation between threshold angle from the pendulum test, threshold velocity from the Ramp and Hold test and the presence of clonus. A lack of correlation was observed between the Ashworth test and any other measure (threshold angle, threshold velocity, EMG latency, EMG magnitude, number of oscillations and Relaxation Index). The imposed velocity in an Ashworth test is neither controllable nor repeatable. Spasticity is multi-factorial in origin and these tests may be measuring different components of spasticity. This may explain the correlation of ankle clonus with the Ramp and Hold test data and the pendulum test data.

The Ramp and Hold test and the pendulum test have been demonstrated to be sensitive and valid measures of stretch reflex activity in a CVA population. These tests are relatively easy to administer and interpret. This makes them suitable for clinical assessment and longitudinal study.

These results are consistent with a decreased threshold for stretch-evoked responses, as well as an increase in reflex loop gain in CVA subjects. A decrease in presynaptic inhibition may be partially responsible for the enhanced stretch reflex observed in the CVA group.

Future Considerations

In future studies of spasticity in hemiplegia it would be helpful to record the EMG activity of the hamstring muscles during the pendulum test. If it is found that there is a co-contraction of the muscle groups occurring as a result of the ramp displacement-evoked activity this would allow contribute to our understanding of reciprocal inhibitory pathways as a contributor to abnormal motor activity in hemiplegia.

A hand-held microprocessor controlled system could be developed for real time analysis of the pendulum test. This would include threshold angle, the number of oscillations and the decay of the sinusoidal waveform, as well as a graphical display of the phase-plane plot. This information could be used to compare tests over time in the same subject or comparisons could be made to an able bodied subject.

The range of velocities for the Ramp and Hold test should be increased. Including higher velocities in the protocol would likely demonstrate a definitive velocity dependence. Higher velocities would also help to clearly demonstrate vibratory inhibition in the control group.

Selection of a test group for a future study should include subjects with a greater degree of spasticity as detected by the Ashworth test. The majority of subjects for the current study did not demonstrate any spasticity upon manual passive displacement of the lower limb. Exaggerated stretch reflex activity was present even in subjects whose Ashworth score was 0. Are these truly tests of spasticity as it is commonly defined?

A future study should also document the natural time course of spasticity over a one year period beginning from the onset of CVA. It would be helpful to be able to report a prognosis to patients and health care providers with regards to the pattern of spasticity over time. It would be of benefit to know the rate and duration of CNS changes following a CVA. As well drug therapies can be compared using the objective tests presented in this study.

Other muscle groups should be tested and their responses compared. The ankle, hip or elbow could be included in a future study. Is the time course of spasticity different for different muscle groups? Is it affected by lesion type, size or location?

A more precise method of testing vibratory inhibition should be developed. Vibration is one of the physiotherapy techniques used to decrease spasticity. A method to quantify the effect of vibration on spasticity can help to assess its value as a therapeutic technique.

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Appendix A - Paraphrase & Informed Consent

Study Description
"The Objective Assessment of Spasticity in Hemiplegia"
University of Manitoba
Page 1 of 3

There are a number of consequences of cerebrovascular accidents (stroke) including altered sensory ability and motor capability. One possible consequence is the development of spasticity where limb motion is impeded by increased resistance from the muscles. We are conducting a study to better understand the neural mechanism responsible for spasticity and to provide a method of evaluating the changes in spasticity that occur with time and with drug therapy. The information provided from this study will aid in the techniques used to document patient progress with treatment and will help to develop better treatment strategies for patients with stroke.

The primary objective of this study is to develop and evaluate measurement techniques which are designed to measure the degree of spasticity. Two tests currently available are 1) the pendulum test and 2) Ramp and Hold tests using a dynamometer. The pendulum test involves releasing the limb from an extended position and observing the pendulum-like motion of the limb. The Ramp and Hold test uses a machine called a dynamometer to passively impose limb motion through a set range of motion at various speeds.

The purpose of this study is to evaluate these two tests (the pendulum test and the Ramp and Hold test). Two groups will be tested: 1) a group with a history of stroke and 2) an able bodied group.

Procedure

You will be requested to attend one test session. The duration of the test session will be approximately 60 minutes.

You will be administered a fifteen minute initial assessment. The assessment will include passive range of motion of your legs. A brief history will also be taken at this time.

You will be administered each of the following tests:

a) Lower limb pendulum test on the affected side.
b) Lower limb Ramp and Hold test on the affected side.

Study Description

"The Objective Assessment of Spasticity in Hemiplegia"
University of Manitoba

Risks

There are no documented risks associated with the pendulum test.

There are no documented risks associated with the Ramp and Hold tests using a dynamometer.

There is a potential risk of muscle damage or bone fracture associated with this test if you should experience a severe muscle spasm during the test procedure. There are no documented incidences of this occurring. However, a cut out switch is available on the dynamometer which can be used to deactivate the machine, as well the dynamometer will automatically shut-down if it detects high force levels.

You will not be identified in any published report of the results of this study. Your participation in this study is voluntary, and you are free to withdraw at any time. If you decide to withdraw your future medical care will not be prejudice in any way.

Participation may be terminated if the researchers feel that you are not complying with the protocol, or if information becomes available to indicate that the protocol is ineffective or dangerous. You do not forfeit any legal rights in the event of an adverse outcome. Your will not receive reimbursement for participation in this study, nor will you be responsible for any costs directly related to this study.

Consent Form
"The Objective Assessment of Spasticity in Hemiplegia"
University of Manitoba

If you have any questions or do not understand any aspect of this form please contact Bettina von Kampen ( ) or Dr. Dean Kriellaars ( ).

I have read and understand this form and the nature of the study including potential benefits and risks. I have had any questions answered which arose during discussion of participation in this study. I agree to participate in this study and abide by the procedural requirements. I understand that I may withdraw from this study at any time.

_____ _____
Signature of Subject Date

_____ _____
Signature of Witness Date

_____ _____
Signature of Investigator Date

Appendix B - Subject Questionnaire

Name:

DOB:

Date of CVA:

Location of CVA:

Independent transfers:

Ashworth:

Quads -
Hamstrings -

Passive ROM:
Knee flexion -
Knee extension -