THE EFFECT OF CEREBRAL VASCULAR DISEASE ON SKELETAL MUSCLE

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BY

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ABSTRACT

Twenty-five patients with a mean age of 59.7 ± 11.8 (SD) years who were hemiparetic due to a cerebrovascular lesion of the cortex were assessed to determine the degree of neuromuscular dysfunction produced in the affected lower limb. Dysfunction was postulated to be the result of a secondary lower motoneuron lesion precipitated by the primary upper motoneuron lesion. The effects of cerebrovascular disease on skeletal muscle were assessed through an evaluation of the motor unit which involved assessment of excitable muscle mass (M-wave amplitudes), motor unit counts, peripheral nerve conduction velocities, evoked contractile properties of the dorsiflexor muscles (tibialis anterior) of the lower limb and degree of motor dysfunction expressed as a function of motor unit activation and maximum voluntary contraction (twitch interpolation method). Results showed preservation of the skeletal muscle with normal contraction times (108 ± 33 ms and 106 ± 35 ms, affected limb versus unaffected limb) and half relaxation times (119.3 ± 41 ms and 114 ± 32 ms respectively). Twitch torque was maintained and did not show significant differences between limbs (2.3 ± 1.6 N.m and 2.4 ± 1.5 N.m., paretic vs. non-paretic limb). Voluntary force production of the affected limb, (10 ± 12.1 N.m) however,

was 38% of that produced by the unaffected limb (26 ± 1.4 N.m.) and measures of mean percent motor unit activation of the paretic limb were 58% of that produced by the unaffected limb. Interpolated twitch results showed that mean percent motor unit activation was significantly different in the affected limb (46 ± 36%) than the unaffected limb (79 ± 19.6%). These results indicate that some motoneurons in hemiplegic patients were healthy but not readily activated. No effect was seen for age, sex of the subject and time post stroke. No significant difference in the pattern of results was observed between initial and final test results for subjects examined more than once. Conclusions were that skeletal muscle integrity was preserved probably due to spinal reflex activity and force production was depressed due, in part, to an inability to fully activate motor units. The inability to activate motoneurons may occur because some motoneurons are in a dysfunctional state. The following data from the present experimental work revealed several trends suggesting the possibility of a sick motoneuron hypothesis due to transynaptic motoneuron degeneration and the existence of a secondary lower motoneuron lesion in stroke syndrome. These trends are: 1) decreased motor unit counts of a sub-group of the total sample consisting of subjects under 60 years of age approached conventional

levels of significance. Mean values for the affected limb were 73.8 ± 52 and 130.0 ± 61 for the unaffected limb ($P < 0.05$, $F = 5.05$, critical $F = 5.59$) In addition, M-wave amplitudes showed significant differences between limbs in the sub-group (4.0 ± 2.3 mV and 5.7 ± 2.2 mV affected vs unaffected limb $p<0.05$), indicating that transynaptic motoneuron loss may have occurred; 2) decreased nerve conduction velocities and prolonged terminal latencies in the motor nerves of the paretic limbs also suggest sick motoneurons and the possibility of a dying back phenomenon of the terminal nerve endings; 4) normal M-wave amplitudes and twitch torque values of the tibialis anterior muscle coupled with the prolonged terminal latencies may be indicative of collateral sprouting of terminal axons which have taken over previously denervated muscle fibres. Future studies are needed to confirm or refute these observations.

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In Memory of Robin

For Blair

To John

I wish to thank my son Blair for caring for me and encouraging me during these past years. To John, thank you for never failing me, for always being near and for giving me hope. My deep appreciation is given to Eva Marsh, June Fuller and Olga Maximiuk, for keeping my world warm and friendly as I once again became a student.

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I. INTRODUCTION

Traditional approaches to the treatment of stroke syndrome (STROKE) adopt the view that muscle dysfunction in stroke patients is caused solely by an upper motoneuron lesion (UMNL) precipitated by anoxic death of brain cells and neurotransmitter imbalances (Licht 1975). Clinical lower motoneuron (LMN) signs are considered to be due to defective cortical drive on the LMN or simply the result of localized trauma post-stroke (Moskowitz and Porter 1963; Alpert et al. 1971, 1973). Past research studies focused on the vascular disorder producing the UMNL (Wade et al 1985) and little attention has been given to the motor unit.

A second view, based on the existence of trophic interactions in the mammalian central nervous system (CNS) and between muscle and motoneurons (McComas et al. 1971 a b, 1974 and McComas 1977) suggests that lesions of the UMN directly affect the LMN. Loss of functional motor units reported in stroke syndrome (McComas et al. 1973) and attributed to transynaptic motoneuron degeneration support an UMN-LMN hypothesis. Transynaptic motoneuron degeneration is thought to be a process in which the alpha motoneuron is deprived of its cortical trophic input as well as its synaptic drive. As a result, the motoneuron becomes dysfunctional and is unable to make effective synaptic connections with its respective muscle fibers and muscle weakness or flaccidity may occur. Once deprived of its

cortical synaptic connections, the alpha motoneuron then has target sites suitable for collateral sprouting from sensory nerve fibres which serve to restore the integrity of the alpha motoneuron (McCouch et al. 1958). Nerve fibre sprouting also occurs at the axon terminal. The relationship between collateral nerve sprouting on the alpha motoneurons and/or sprouting at the axon terminal and muscle tone is unknown. It is conceivable that collateral sprouting on the alpha motoneuron along with disturbances in corticospinal pathways could result in excessive muscle tone (spasticity). The process of transynaptic motoneuron degeneration proposes additional physiological explanations for the clinical observations of an initial stage of muscle flaccidity, followed by the appearance of muscle spasticity which usually preceeds normal functional muscle return in stroke patients (Twitchell 1951; Brunnstrom 1971). Current investigations of muscle spasticity, demonstrating that drugs such as dantroline act on the sarcoplasmic reticulum to disrupt normal calcium function (Young and Delwaide 1981), also implicate the LMN in concepts (spasticity) which traditionally have been thought to be due, solely to a deficient cortical drive system.

Other studies of myasthenia gravis, muscular dystrophy and spinal muscular atrophy have produced the "sick motoneuron hypothesis" in which impaired muscle function is attributed to functionally dead or to sick motoneurons.

This theory is compatible with the theory of transynaptic motoneuron degeneration because in the latter case, the degree of dysfunction of the alpha motoneurons may be varied. If motoneurons are sick, they show defective neuromuscular transmission when tested during maximum voluntary contraction or repetitive nerve stimulation. These motoneurons may still control a number of normal muscle fibers, but are unable to acquire previously denervated muscle fibers. In addition, impulse conduction along the axons are normal, but slowed at the terminal branches. Dead motoneurons do not exert trophic or excitatory influences on muscle fibers. In stroke syndrome, either state of the motoneuron could occur depending on the extent of the UMNL and subsequent neurotrophic loss. The above process could account for the clinical observation that in some stroke patients, limbs remain in a state of muscle flaccidity or spasticity with little or no normal recovery (Twitchell 1951).

Studies indicating slowing of peroneal nerve conduction velocities (NCV) in the hemiparetic limb (Cowley et al. 1967; Widener et al. 1967; Chokroverty et al. 1978; Cruz Martinez 1983), the presence of fibrillation potentials in the muscle fiber membrane (Goldkamp et al. 1976), specific muscle fiber atrophy (Chokroverty et al. 1976, Cruz Martinez 1982) particularly of type II fibers (Edstrom 1970) and electrophysiological motor unit type changes (Young et al.

1982) in stroke patients also cast doubt on an exclusive upper motoneuron hypothesis in vascular hemiplegia.

Some clinical observations, such as muscle paralysis, flaccidity, atrophy and spasticity seen in stroke patients could be consistent with either viewpoint. However, the question of LMN changes being due to post-stroke trauma is rejected because McComas et al. (1973) report functional loss of motor units not only in the extensor digitorum muscle (EDB) but also in the thenar and hypothenar muscles in vascular hemiplegia. They argue convincingly that the theory of a post-traumatic lesion requires the acceptance of multiple nerve entrapments, which are unlikely, to explain all the muscle dysfunction present in the condition.

Based on the evidence presented, the controversy of whether loss of voluntary strength following stroke is entirely due to loss of volitional drive on motoneurons, or whether it is also the result of changes in the motor unit, such as death of functional motoneurons, loss of excitability of motoneurons, failure of neuromuscular transmission or atrophy of muscle fibers, is still unresolved.

This thesis argues that following stroke, the alpha motoneuron is deprived both of its normal synaptic and neurotrophic input with subsequent secondary effects occurring in the muscle unit. A net decrease in excitation of the alpha motoneuron results in voluntary muscle

weakness, flaccidity and consequent atrophy. A net increase of excitation of the alpha motoneuron results in muscle spasticity. Because both synaptic and neurotrophic input is altered, the integrity of the skeletal muscle is disturbed as indicated by changes in the muscle's contractile properties. However, spinal reflex activity might over-ride cortical trophic disruption and the effects of an UMNL on skeletal muscle fibers are unpredictable.

To assess the effects of deprivation of cortical trophic effects and impulse transmission on the alpha motoneuron and muscle unit, electrophysiological evaluation of the motor unit, the motor unit counts and measures of the motor nerve conduction velocity, excitable muscle mass (M-wave amplitude) and evoked twitch contractile properties were made. Upper motoneuron dysfunction was assessed by measuring the extent of motor unit activation during maximum voluntary contractions (twitch interpolation method). Unlike clinical assessments such as the modified Brünnstrom assessment which relates UMN functioning to specific tasks, the present study, with the measures used,was able to quantitate the extent of the motor unit dysfunction following stroke. The primary purpose of the present thesis was to provide additional evidence of the effects of stroke on motor units. Three main questions were asked.

Purpose of the Experiment:

1) Is there a difference in excitable muscle mass (M-wave), motor unit counts, and peripheral nerve conduction velocity between the affected and unaffected limbs of patients who are hemiplegic due to cerebrovascular disease?

2) What are the evoked contractile properties of the tibialis anterior muscle of the affected and unaffected limb of subjects who have had a "stroke"?

3) What is the degree of motor unit dysfunction between limbs expressed as a function of motor unit activation and maximal voluntary contraction (M.V.C.)?

II. LITERATURE REVIEW:

The literature review focusses on an analysis of pertinent research into LMN dysfunction in stroke syndrome. Research is presented as it relates to the motoneuron and the muscle unit. The relationship between LMN function and muscle flaccidity/atrophy and/or spasticity, common states of skeletal muscle in stroke syndrome, is discussed.

A. THE MOTONEURON

1. Alpha Motoneuron Excitability

Stroke Syndrome is usually characterized by an initial stage of muscle flaccidity. As the hemiplegia evolves, the muscle passes through the states of flaccidity/or spasticity as some normal muscle control returns. Investigations into the status of the motoneuron during these states used two techniques: Analysis of the H-reflex and reflex potentiation.

a) General Characteristics of the H-reflex and Reflex Potentiation.

1. H-response

The H-reflex demonstrates the lower motoneuron's ability to transmit impulses to skeletal muscle and the ability of the muscle to respond independent of input (drive) from higher cortical centers. It represents the algebraic sum of the impulses converging on the alpha motoneuron and is the electrical representation of the monosynaptic stretch reflex. The H-response is elicited

through the application of a single sub-maximal shock to a peripheral nerve and evokes two discrete action potentials. The first response, the M-wave is a measure of the excitable muscle mass produced when the motor efferent fibres of the peripheral nerve are stimulated. The second response, the H-response, is a measure of the excitability of the afferent sensory nerve fibres and the anterior horn cell (AHC) supplying the same muscle from which the M-response was obtained. The H-response is produced by direct stimulation free from any direct influences of the fusimotor system. MNE is expressed as the largest obtainable H-wave/the largest M-wave (H/M ratio) and indicates the degree of spasticity or flaccidity of a muscle (Garcia-Mullin and Mayer 1972; Angel and Hoffman 1963). This measure depicts the number of motoneurons excited monosynaptically, estimated as a fraction of the motoneuron pool (Angel and Hoffman 1963).

Criticisms of this technique are that the H-reflex activates less than 50% of the motoneuron pool and is not reproducible until a small M-wave has occurred and consequently is a limited indicator of MNE. Further, the H-response may be facilitated or inhibited by reflexes from the brain stem as well as being affected by pre and post synaptic inhibition and it is not possible to differentiate between sources of inhibition (Granit and Burke 1973). It is not possible, also, to determine whether impaired

potentiation is due to deficits in the soma, dendrites or axons.

Other studies indicate that the H-reflex may not be a precise measure of MNE independent of cortical drive. Reasons for this view are based on: 1) the role the gamma efferents have been shown to play in reflex excitability (Angel and Hoffman 1963), 2) the fact that active voluntary contractions of agonists facilitate the H-response while contractions of antagonist muscles inhibit the response (Thomas and Lambert 1960; Mayer and Mawdsley 1965; Gotleib and Agarwal 1971) and, 3) the finding that the H-response has been induced in the extensor digitorum communis muscle by passive extension of the wrist as well as being produced in muscles where it was not present at rest (Garcia et al. 1979). The H/M ratio has also been shown to be affected by the intensity of the submaximal stimulus (Angel and Hoffman 1963) because as the stimulus intensity increased, the H-response increased to a point and then progressively decreased while the M-wave rose along a sigmoid curve and levelled off once all the motor nerve fibres had been recruited. Sica et al. (1971) argued that the H/M ratio may be unity due to excitability of the entire motoneuron pool which can occur with an adequate stimulus. This finding suggests that it is possible to excite the entire motoneuron pool without an UMNL. Further, the amplitude of the H-reflex has been shown to be severely depressed with

increasing frequency of stimulation probably because fibres of the motor unit become refractory to another impulse (Goodgold et al. 1953).

2) Reflex Potentiation

To overcome difficulties encountered with the H-response, it was advocated measuring the amount of recovery of excitability after stimulation of the H-response using a technique known as reflex potentiation (Upton, Sica and McComas 1971, McComas, Sica and Upton 1971). This method depends on the amount of background facilitation that can be imposed on the alpha motoneuron during a maximal voluntary contraction. While this technique is primarily a measure of cortical drive, it will be described here to show its relationship to the lower motoneuron.

Studies using the method apply a supramaximal stimulation to a peripheral nerve. A V1 response occurs which is thought to consist of an H reflex potentiated by a voluntary contraction (Upton et al. 1971). The voluntary contraction is facilitatory to the motoneurons. This increased facilitation of the motoneurons is summated with excitation by the Ia afferent nerve fibres which have been activated by electrical stimulation. A reflex response occurs because the volitional impulses and the antidromic impulses produced by the electrical stimulation collide. These colliding impulses cancel each other out and the reflex activity occurs. Without the collision the reflex

would be blocked by antidromic impulses in motor axons.

A second response, the V2 response is said to be caused by a transcortical reflex (Lee and Tatton 1975). The proposed pathway is thought to pass from mechanoreceptors, to the cuneate nucleus to the ventro basal thalamus, to the post-central cortex. It then goes to the pre-central cortex and then on to motoneurons. It is thought that the V2 response is the result of background facilitation of the motoneurons. It can be seen that the precision of the technique depends primarily on an intact upper motoneuron.

Criticisms of the technique offered by Sica et al. (1971) are that in some instances, some motoneurons may discharge impulses too late for collision with the antidromic volley. In other instances, the V1 response actually declined on effort. It was suggested that the motor axons may have been refactory after the volitional output or the effect may have been due to an inhibitory mechanism. In spite of limitations and criticisms both techniques have been used in the assessment of MNE in stroke syndrome and have provided useful information.

b) The H-response and Reflex Potentiation in Stroke Syndrome.

1. H-Response

The state of MNE at various stages of evolving hemiplegia is of interest when considering an UMN-LMN hypothesis. Studies employing analysis of the H-response

show decreased MNE only in the hemiparetic limb, and this response appeared 2 days post onset. MNE increased as subjects became more chronic (Garcia-Mullin and Mayer 1972). Decreased MNE is accompanied by depressed tendon jerks and diminished muscle tone in the affected limb while increased MNE is accompanied by hyperreflexia (Angel and Hoffman 1963) and augmented muscle tone. Increased MNE is directly proportional to the time post stroke. The pattern of results in these studies lacks predictability because some subjects showed no difference between MNE in either limb while others showed increased excitability in the affected limb in the acute stage. Instances in which MNE was increased in the non-paretic limb of some chronic patients were also noted. It has been postulated that in these last instances, subjects either had bilateral lesions which were not manifested clinically or that unilateral lesions have bilateral effects on spinal motoneurons (Angel and Hoffman 1963). While measures of motoneuron excitability reflect deficits in impulse transmission, they do not indicate direct evidence of motoneuron damage.

Magladery et al. (1952) studied the recovery of the H-reflex after conditioning by a preceeding submaximal stimuli (paired stimuli). An index of threshold excitability was developed in which the difference in intensity between stimuli that activate afferent sensory fibres and those that activate motor axons was calculated. Results showed

increased motoneuron excitability with any type of UMNL probably due to a lack of inhibitory effect on AHC from descending cortical pathways. When using a single impulse volley there was a slight increase in MNE in the spastic subjects vs normal control subjects. Miglietta (1969, 1970), using the paired stimuli technique, in which the conditioning volley was supramaximal noted that motoneuron excitability was increased bilaterally in hemiplegic patients suggesting changes in the spinal motoneurons which affect both "affected" and "unaffected" limbs.

Chaco et al. (1984) studied 12 subjects with vascular hemiplegia to determine evidence of recurrent inhibition in spastic hemiplegia. The method used involved establishing a collision of impulses (conditioning reflex) in the motor axons and subsequently exploring with a test reflex, only those motoneurons which had already discharged in the conditioning reflex. Only motoneurons which fired because of the first volley (conditioning volley) had their excitability evaluated by a second volley (test volley). The strength of the stimulus of the conditioning volley did not activate the alpha motor axons but the second stimulus was supramaximal for the alpha motor axons and provided an antidromic volley at the site of stimulation. The timing between the 2 volleys was 10 ms and permitted the reflex discharge caused by the first afferent volley to collide with the antidromic volley. The reflex activated

motoneurons were not invaded by the antidromic motor responses. These motoneurons were then available for activation by the Ia afferent volley produced by the test stimulus. This stimulus produced another reflex discharge. The amplitude of this discharge was then used to determine excitatory or inhibitory influences on these motoneurons.

The ratio of maximum M wave amplitude and H-reflex amplitude was calculated for both the affected and unaffected limb for both the conditioning volley and the test volley. Results showed no significant increase of the H test reflex amplitude on the affected side indicating no decrease in recurrent inhibition. In 4 patients there was evidence of an increase in recurrent inhibition following the conditioning stimulus. The authors concluded that in some cases there may be a disinhibition of Renshaw cell firing due to suppression of supraspinal inhibitory control.

2) Reflex Potentiation

Impaired potentiation of the H-response using the reflex potentiation method was present in 11 patients who were hemiplegic from various causes (Sica, Upton and McComas 1971). Eight of the patients had defective potentiation even when muscle weakness had returned to "normal" (See Motor Unit Activation Section). However, muscle strength was not quantitated and while "normal" strength may have returned, "normal" strength may not have represented a maximum voluntary contraction. If, in this

last instance, MNE is determined by volitional activity then MNE would be considered normal when strength was "normal", as assessed by potentiation of the H-reflex but the return to normal function would still be abnormal.

2. NERVE CONDUCTION VELOCITY

Two major methods of investigation into the integrity of the peripheral nerve in stroke have been used. Method one (LeQuesne 1971) electromyographically measured conduction velocities of the fastest conducting nerve fibres. Method two involved the Hopf Technique (McComas 1977) and measured conduction velocity in the slowest conducting nerve fibres.

a). Electromyographic Analysis

1). Fast Conducting Nerve Fibres

Reports of evaluations of the "fast" conducting fibres of the peroneal motor nerve indicate slowing of NCV on the hemiparetic side as compared with the non-paretic side or controls (Cowley et al. (1967); Widener et al. 1967; Chokroverty et al. 1978; Cruz Martinez 1983). The cause of this finding is controversial. Cowley et al.(1967) studied the posterior tibial nerve in conjunction with the peroneal nerve and because NCV in the former was not significantly different, decreased NCV of the peroneal nerve was attributed to pressure exerted on the nerve post stroke. Other conclusions were that the reported decreased NCV of

the peroneal nerve is evidence of a LMNL.

McComas et al. (1973) argued that evaluation of axon integrity must involve consideration of the whole motor unit. They cite findings of decreased motor unit counts in both thenar and hypothenar muscles in stroke syndrome to show that a post traumatic hypothesis which would involve multiple nerve entrapments is unlikely. McComas et al. (1973) found no significant evidence of decreased NCV between limbs for the peroneal nerve.

Many studies have been done on the median and ulnar nerves but results are conflicting. Electromyographic measurement of the nerve conduction velocities (NCV) of the fastest conducting fibres of the median and ulnar motor and sensory nerves showed no significant differences between paretic and non paretic limbs (Goldkamp 1967; Sutton et al. 1967; Panin et al. 1967; Widener et al. 1967; Cruz Martinez 1983; Young and Mayer 1981; Namba et al. 1971). Evaluation of the NCV of "fast" conducting median and ulnar nerve fibres also showed no effect for sex, hand dominance, time post stroke, degree of motor recovery and degree of spasticity (Sutton et al. 1967; Panin et al. 1967). Another important finding reported is that the threshold for excitability is constantly elevated on the paretic side. Paul et al. (1968) showed that the rheobase (minimal amount of current needed to stimulate the nerve to fire) is greater on the paretic side in 82% of the cases.

The chronaxie (the minimum time at which an electric current just double the rheobase will excite a muscle contraction) was greater in 54% of the cases studied.

Only one study (Takebe et al. 1975) reported significant differences ($P < 0.001$) between NCV of the affected and unaffected limbs for both the peroneal and ulnar nerves. Irregular patterns of the evoked action potential and hypoexcitability of the nerves were also observed. The reported abnormal shape of the evoked action potentials were thought to be due to abnormal depolarization of either/or both muscle fibre membrane and of nerve fibre membrane at the terminal branches of the axon. Hypoexcitability may be due to segmental demyelination with insignificant damage to the axon. Takebe et al. (1975) also found abnormal evoked muscle potentials and hypoexcitability in the nerves of the unaffected limb. This finding was attributed to the fact that 15% of the limb muscles are supplied by the same side of the brain. This study would support a LMN hypothesis and reject a nerve compression theory.

The studies presented have examined the integrity of the axon of the fastest conducting nerve fibres. Other investigators (Namba et al. 1971), have argued that evaluation of the axon should include assessment not only of the fast conducting motor nerves but also the slow and average conducting fibres.

2). Slow and Average Conducting Nerve Fibres

A study of "average" and "slow" motor and sensory nerve fibres (Namba et al. 1971) of the ulnar nerve in acute hemiplegia (1-50 days post stroke) showed that "average" motor NCV was decreased significantly on the affected side ($p<0.001$). Prolonged conduction velocity in the hemiplegic side occurred in 76% of patients when "slow" NCV was measured and in 16% of patients each for "average" motor and "fast" sensory nerves.

Criticism of this study (Takebe et al. 1975) is that it is difficult to determine the peak of the negative wave, (a criterion for determining average fibres) and, as a result, the measures obtained might not be reliable.

B. Morphometric Analysis

Other investigators point out (Pollock et al. 1984; Gillespie and Stein 1983) that since NCV is affected by axon diameter, cross-sectional area, internodal distance and the ratio of axon to the total fibre diameter, morphometric analyses are of great importance in determining evidence of peripheral nerve abnormalities.

In stroke patients, an important investigation of sural nerve biopsy samples from 12 living hemiparetic subjects with no evidence of muscle atrophy showed a 50% or more reduction in myelin thickness and a significant reduction in the mean diameter of myelinated nerve fibres (Pollock et al.

1984). Abnormal internodes, significantly more collagen pockets and more clusters of denervated Schwann cells were found in the affected nerves than in unaffected nerves. Despite these morphological changes, sural NCV was normal.

The authors postulate that results are due to demyelination and re-myelination secondary to a primary axonal atrophy. Further, there might be transynaptic changes in the soma of the spinal motoneurons or dorsal root ganglion neurons in stroke, which might produce atrophy of the distal part of the nerve with secondary demyelination and remyelination. However, other electromyographic (EMG) studies have found significant slowing of conduction velocities only in the proximal ulnar motor nerve (Panin et al. 1967). The normal NCV found by Pollock et al. (1984) was considered by the authors to occur because of insensitivity of EMG methods used in situations of focal demyelination. This point was supported by Saida et al. (1980) who made serial EMG determinations of motor NCV through a demyelinated lesion produced by intra-neural injection of antiserum into the sciatic nerve of rabbits. It was found that when conduction velocities had returned to normal, myelin thickness had returned to only 1/3 that of the control group. It was hypothesized that changes in the morphology of the peripheral nerve might be present in stroke syndrome but be undetected by current EMG recording techniques.

The significance of the role of myelin thickness and conduction velocity is questionable if the results of animal studies are considered. The relationshsip between axon diameter, myelin thickness, and conduction velocity in normal and atrophic cat sural and medial gastroenecmius nerves, using anatomical, physiological and computer calculations (Gillespie and Stein 1983) demonstrates the process of atrophy in the sectioned nerves. Changes in the axon, seen during the process of atrophy show that the axons begin to shrink and the tube formed by the Schwann cell and myelin collapsed inwards indicating that the changes taking place affect the fibre diameter rather than the axon diameter. Although substantial surface changes occur in the myelin (myelin becomes flattened) there was no evidence that myelin thickness changed. The number of turns of the myelin around the axon and length of each turn was unaltered. The significance of this finding is that NCV is dependent on myelin thickness and internodal distance. However, results of this study showed that reduction in NCV correlated more closely with decreases in axon diameter rather than in nerve fibre diameter (axon and myelin sheath). These findings, while important, cannot be directly applied to the human state because atrophy produced by section of nerves may not be comparable to atrophy occurring in stroke syndrome.

The studies presented have shown that considerable

uncertainty exists concerning the state of the axon in patients with vascular hemiplegia. To this author's knowledge, no studies have been reported specifically on the effects of spasticity and/or atrophy on nerve fibre diameter in vascular hemiplegia. Animal studies provide some information about the effects of hypoactivity and hyperactivity on nerves (possible causes of spasticity or atrophy). Hypoactivity may cause atrophy, whereas spasticity may be a type of hyperactivity.

3). Effects of Hypoactivity and Hyperactivity on Nerves

Many models of hypoactivity have been used (nerve crush, tenotomy) but models of external immobilization most closely parallel the process of stroke recovery in which muscle atrophy may be due to simple disuse. Using an immobilization model (Eisen et al. 1973) found a decrease in nerve fibre diameter in the nerve supplying rat soleus muscle. Tomanek (1968) showed that immobilization produced no change of the intact nerve to the medial gastrocnemius in young or adult rabbits. Therefore, disuse may have specific effects on nerves supplying different muscles.

Eisen et al. (1973) also found that hyperactivity of the soleus muscle on the contralateral side of the immobilized limb produced an increase in mean fibre diameter of intact nerve fibres. These studies indicate that the state of the axon may be influenced by both hypoactivity and

hyperactivity and be reflected by flaccidity and spasticity of muscle.

3. Motor Unit Counts

a) Functional Loss

In a study of 46 patients who were hemiparetic due to cerebrovascular lesions, McComas et al. (1971a) determined the number of functioning motor units in the extensor digitorum brevis (EDB) muscle. Using a non-invasive method McComas et al. (1971a) showed no significant difference in the number of motor units between the two legs, two months after the onset of the stroke. After 2 months, only about one-half the original population of motor units were still functioning in the hemiparetic limb. There was no evidence of continuing loss of motor units beyond the two-month period. Motor unit potentials were significantly ($P<0.001$) larger in the affected limb and this enhancement of potentials did not occur until 20 months post stroke. Conclusions were that since the motor unit potentials reflect the number of muscle fibres in a particular motor unit, enhanced potential amplitudes are probably due to collateral re-innervation of denervated muscle fibres by surviving motoneurons. This study would suggest that in stroke syndrome, some motoneurons functionally die and are lost while others are sick and recover. The authors argue

that loss of motor units is not due to pressure on the peripheral nerve for had that been the case, loss of functioning units would have occurred earlier than the 2 month period to coincide with the period in which the patient was immobilized in bed. Motoneuron loss is thought to occur abruptly because two of the cases studied over a three month period from date of onset showed sudden, marked loss of motoneurons after the two month period. It was postulated that Wallerian degeneration occurred in the cortico-spinal tract in the initial 2 month period and was followed by subsequent change in the soma and axon of the alpha motoneuron.

McComas et al. (1971 b, 1973) reason that neurotrophic substances are known to travel at rates of 1 mm/day in the sciatic nerve of rabbits (slow axoplasmic flow) and they speculate that trophic substances would be depleted in the axon in about 2 days. The soma would then take the rest of the initial 2 month period to degenerate.

b). Anatomical Loss

Histological investigations exist to support the EMG findings of McComas et al. (1973). Anatomical loss of motoneurons has been found in patients who died at various times from cervical cord lesions (Reske-Neilson et al. 1971). A marked loss of motoneurons was found from samples taken well below the lesion and these motoneurons showed

histological evidence of enlarged cell bodies with the Nissl substance pushed to the periphery of the cell. Evidence of axonal sprouting was observed in biopsied specimens of the tibialis anterior and peroneus brevis muscle. Forty-one and 43 days after the lesion, there was evidence of collateral sprouting even though denervation was not seen in the muscle. Collateral re-innervation of nerve fibres occurred by 14 months post injury. This study shows anatomical evidence of the process of trans-synaptic motoneuron degeneration described by McComas et al. (1973).

4. Motor Unit Activation

a) Possible Mechanisms

The mechanisms of motor unit activation and performance in stroke syndrome is not well documented in the literature. The issue has been addressed in part by Sica et al. (1971) and two possible explanations of impaired functioning have been put forth. The first explanation argues that in healthy subjects, Ia endings are normally under the control of pre-synaptic inhibitory mechanisms. These pre-synaptic inhibitory mechanisms receive inputs from higher centres. During a voluntary contraction, in healthy people, the pre-synaptic mechanisms are restrained by descending pathways. In stroke patients, this disinhibition may not occur and stretch reflexes would be increased because of an

overriding influence of increased fusimotor activity. The authors also argue that if enough descending tracts are left, then volitional activity would be unaffected. This explanation would account for the finding that stroke patients may have impaired reflex potentiation even though muscle weakness is not present. Another possibility might be that the stretch reflex increases because of a temporary suppression of post synaptic inhibitory neurons (those inhibitied by Ib Golgi tendon organ fibers).

The other explanation is that during a voluntary contraction, one group of descending fibers normally elicits excitatory post synaptic potentials (EPSP's) which produce a background but sub-threshold depolarization of the motoneuron. Superimposed on this background are large EPSP's from descending pathways which cause the motoneuron to fire an action potential. Further, Ia inputs may also elicit impulses in the motoneuron. It is postulated that in stroke syndrome, the sub-threshold depolarization is reduced. The Ia inputs no longer evoke a reflex discharge but volitional pathways and their resulting EPSP's are still able to produce a voluntary contraction. Again this explanation could explain why "normal" strength has been observed in hemiplegic patients with impaired potentiation.

Upton et al. (1971) also reason that 2 basic mechanisms exist whereby descending pathways could facilitate

motoneurons. Firstly, the effect of the descending fibres may be powerful enough to exert a direct influence and stimulate the motoneurons either monosynaptically or through interneurons. The second explanation might be that descending fibres may terminate on fusimotor neurones and facilitate the motoneurons via the stretch reflex arc. Upton et al. (1971) tested this last hypothesis by decreasing the effect of the reflex loop by blocking the fusimotor fibres with an anaesthetic. Results showed that marked potentiation could still occur. This observation illustrates the powerful drive of the UMN on the LMN.

Studies of MNE and reflex potentiation provide important information concerning possible mechanisms of motor unit activation in vascular hemiplegia. Reported electromyographic studies provide little information on interference patterns, an indicator of the nature of motor unit recruitment during a maximum voluntary contraction. To this author's knowledge only one study (Sequra et al. 1981) reports that motor unit recruitment was back to normal at 24 months post stroke. Coincident with this observation was the observation that there was no spontaneous activity of the muscle membrane at rest and muscle strength had returned to "normal" except in the distal muscles of the hand and foot. No studies have quantified motor unit activation during performance in stroke. Without such quantification,

it is difficult to determine whether MNE merely contributes to the pathological state of muscle spasticity or whether it is a predictor of potential improved muscle function.

The question of motor unit activation in UMNL raises several other issues addressed in the literature. These issues concern recruitment order of motoneurons during voluntary activity, reflex activity, and whether differences in motor unit activation exist between fast and slow twitch motor units.

b). Recruitment Characteristics

1). Voluntary Activity

In healthy subjects recruitment order in phasic activity is very variable while in tonic activity it is more stabilized. Grimby and Hannerz (1972, 1973 a,b.) report that the difference between tonic and phasic recruitment order increases with sustained inhibition of the motoneuron pool prior to activation and decreases with sustained facilitation. Specific types of motor units are activated in an order that is appropriate for the type of movement required. Healthy people are able to pre-program the motoneuron pool for either tonic or phasic recruitment by adjusting the pre-facilitation to a high or low level. It is also reported that discharge from the muscle spindles and indirectly through facilitation of the gamma loop contribute to tonic recruitment order. The degree of dependence of

tonic voluntary activity on support from the muscle spindle and gamma loop is unknown.

In voluntary movement, Grimby and Hannerz (1973 b) showed also that recruitment order in healthy subjects reversed when changing from a tonic to a phasic contraction. If this finding were observed in hemiplegic patients it would be important particularly in assessing the efficacy of proprioceptive neuromuscular facilitation in stroke patients. However, Desmedt and Godaux (1977) have shown that recruitment order is preserved even in brisk ballistic movements. Apparent reversals in motor unit activation are attributed to the fact that occassionally a large motoneuron with a fast conducting motor axon may appear to fire ahead of a smaller unit which has a lower threshold for activation but a slower conducting axon. That is, while the low threshold unit may be activated first, the larger motoneuron is able to transmit the impulse more quickly to the muscle and in fact appear to fire before the slower unit.

Grimby and Hannerz (1970, 1972) report that spastic patients (due to spinal cord lesions) can voluntarily activate a low threshold motor unit for only a few minutes. After this time, the patient loses the ability to drive not only the activated unit tonically but also surrounding units. While spastic patients can fire single motor units they are not able to sustain tonic contraction. It is

postulated that in these cases, the tonic voluntary drive of the motoneuron pool is lost rather than that of single motor units. In view of the work of McComas et al. (1973) who showed functional loss of motor units in stroke syndrome, this issue must be explored more extensively.

On extreme voluntary effort, spastic patients were able to activate units other than the last units fired tonically (Grimby and Hannerz 1973). The new units fired in irregular bursts without a definite order of recruitment. The later finding was said to be due to fatigue and it was argued that normal facilitatory mechanisms, ordinarily used to precipitate tonic voluntary movement, fatigue very quickly in spastic patients and these patients must then use facilitatory mechanisms that are normally employed in phasic voluntary activity. The authors also point out that the original tonic recruitment order can be restored by re-enforcement of the voluntary effort with tonic proprioceptive and exteroceptive reflex activity.

2). Reflex Activity

Grimby and Hannerz (1973a) have also reported that patients with complete spinal cord lesions with resultant loss of cortical drive show that the first motor unit recruited in tonic reflex activity is the same even if the type of stimuli is altered. Patients with incomplete lesions show that recruitment order is the same for both

voluntary and reflex movement. If this situation were the same for stroke patients then physiotherapeutic techniques such as the Bobath method and proprioceptive neuromuscular facilitation techniques should produce physiological change. These methods of treatment have not been assessed from this prospective.

Edstrom et al. (1973) report that the level of tonic spasticity does not influence the recruitment order in tonic exteroceptive reflex activity. They showed that when the level of tonic spasticity was very high, the first unit recruited in tonic activity was also the first unit in phasic activity, and the second unit in tonic activity was also the second unit in phasic activity. They observed that in strong tonic spasticity, recruitment order is stereotyped and only low frequency units are available to act as low threshold units. After a rest period, it was noted that a different unit was recruited initially in phasic than in tonic activity. In phasic activity the first unit fires only once but will fire in short bursts if the stimuli is stronger (subjects were stimulated reflexly by using skin stimuli on the heel). The discharge frequency of this unit was found to be higher than the discharge frequency of the first unit in tonic activity. The authors further report that recruitment order in non-spastic paralysis is flexible and both high and low frequency units are available as low

threshold units. In addition, histochemical evidence showed a selective type II muscle fibre atrophy in muscles with strong tonic spasticity but a non-selective atrophy in muscles with weak tonic spasticity. Conclusions of this work were that the atrophy pattern was related to tonic spasticity and that there was a selective disuse of high frequency units which resulted in selective type II muscle fibre atrophy.

c). Discharge Frequencies

In studies of the discharge characteristics of single motor units in subjects with supraspinal motor disturbances (Freund 1983), there was an inability of the motoneuron to fire at constant intervals. Differences in the temporal order of the spike discharge of the motoneuron were suggested to be related to lesions of different parts of the motor cortex. The authors concluded that the impulse generation at the motoneuron seems to be specifically changed by disturbances of different motor areas. This study did not describe the sample and results presented were for "a patient with minor symptoms of left spastic hemiparesis."

Andreassen (1980) studied firing patterns of motor units in healthy subjects and 10 subjects with cerebral or cerebellar accident. Recording from tibialis anterior muscle was done. Recordings of two hundred consecutive

intervals from 10-20 motor units during a tonic voluntary contraction in which 20% maximum force was generated were made. Results showed reduced firing frequencies of motor units in spastic cases. There was reduced variability between firing intervals in spastic patients even though they were unable to maintain a constant force level. In normal subjects long and short intervals tended to alternate but in spastic patients, long intervals of firing were followed by long intervals and short intervals were followed by short intervals.

Young and Shahani (1980) report that while the mean frequency of discharge of a large number of small motor units tends to be lower than normal, this phenomenon is not the case for all small motor units in any given spastic muscle in vascular hemiplegia. They found that in almost every case studied there was at least one normally behaving small motor unit among all the abnormal ones and that some of these units can be driven to fire at higher frequencies by extra voluntary effort. In stroke subjects, units were seen to fire regularly at low rates of discharge but as impulse frequency increased, the units fluctuated gradually from a slow to a fast discharge rate.

Although not substantiated by the work of Chaco et al. (1984), the above authors suggest that one possible mechanism responsible for control of motoneuron discharge

would be recurrent inhibition by Renshaw cells. The other possibility might be the effects of motoneuron after-hyperpolarization as suggested by Sica et al. (1971). It may also be that the motor system adapts different strategies for maintenance of constant force output in spastic cases or that motoneurons behave differently in different muscles.

The next issue addressed in the literature concerns possible differences in motor unit activation between fast and slow twitch motor units in patients with vascular hemiplegia. This question is of interest in the analysis of neuromuscular function in stroke syndrome because the physiological mechanisms causing muscle atrophy and weakness in patients with UMNL are not fully explained. Edstrom (1970) reports evidence of muscle atrophy of fast twitch muscle fibres in the antigravity muscle of patients with spasticity. Type I (slow twitch) fibres showed no evidence of atrophy and in some cases were hypertrophied. These changes were attributed to selective disuse of high threshold phasic motor units caused by a decrease in voluntary activation and a corresponding increase of discharge of low threshold units due to spasticity.

The work of Mayer and Young (1980) on the isometric contraction of single motor units of hemiplegic patients supports the above hypothesis. They found that in stroke

patients with spastic hemiparesis there was evidence of large slow twitch units which generated large tensions and had low fatigue resistance. These units were not seen in the control group and were originally thought to have been fast twitch units. These units were also thought to occur because of selective disuse of high threshold phasic motor units caused by diminished voluntary activation with an accompanying increase in discharge of low threshold units.

5. Neuromuscular Junction Dysfunction

a). Decremental Responses to Repetitive Stimulation

Studies of patients with vascular hemiplegia have shown decremental responses to repetitive stimulation of the periphral nerve (McComas et al. 1973; Cruz Martinez 1983). These works suggest that in stroke syndrome there may be degeneration of the terminal ending of the axon, and decreased responses to repetitive stimulation may be due to failure of the impulse to reach the pre-terminal axon or perhaps there is not enough acetylcholine released by the impulse. Decremental responses may also be due to a decrease in acetylocholine quanta available for release from the axon terminal or there may also be a functional abnormality in pre-synaptic neuromuscular functioning secondary to impaired axoplasmic flow. Swift and Lambert (1974) studied the effects of acrylomide poisoning in rats

and showed that when the frequency of the miniature end plate potentials fell, the store of acetylcholine quanta dropped to 1/2 or less of the original value.

b). Terminal Latencies

Increased terminal latencies have been reported in the nerves of hemiparetic patients in the affected limb (McComas et al. 1973) but these findings were not confirmed by the work of Mayer and Young (1982). McComas et al. (1973) hypothesized that increased terminal latencies may be caused by a dying back of the terminal axonal endings. The process moves from a period of partial synaptic failure involving only a few synaptic junctions to eventual total synaptic failure. This degenerative process of the axon is thought to spread towards the soma. During partial synaptic failure, impulse transmission is absent but the neurotrophic effect is present and muscle fibres show no features of denervation. In total synaptic failure both impulse transmission and neurotrophic effects are absent and the muscle fibres show electromyographic (EMG) evidence of denervation (fibrillation potentials, and positive sharp waves). The course of the dying back phenomenon is that the soma may disintigrate and muscle atrophy occurs or collateral reinnervation by healthy motoneurons may result. The denervated muscle fibres would be adopted by a different motoneuron. Morphological findings indicating the

presence of axonal sprouting with the formation of complex end-plates have been reported by Cruz Martinez (1982) and support the hypothesis of McComas et al. (1973).

c). Jitter

In a single fibre EMG study of the extensor digitorum communis muscle in patients with vascular hemiplegia, jitter was calculated in patients with good motor recovery in the upper limb (Cruz Martinez 1982). If single fibre potentials are recorded from a muscle after repetitive stimulation of its nerve, the latencies of the elicited responses are almost the same with each stimulus. This latency variability is known as jitter and may be an indication of neuromuscular junction dysfunction (Kimura 1983). Increased jitter was found only in some of the acute patients (less than 10 months post stroke).

B. THE MUSCLE UNIT

1. Membrane Properties

a). Spontaneous Activity

Fibrillation potentials and positive sharp waves are electromyographic evidence of denervation or may simply be indicators of muscle membrane hyperirritability (Johnson et al. 1975). Fibrillation potentials are fully developed action potentials which reflect a spontaneous hyperirritable state of muscle fibre membrane and may be detected 2 weeks

after denervation. These potentials are also reported in inflammatory conditions such as polymyositis, and diseases of muscle fibre degeneration (Duchenne muscular dystrophy). In the latter case, spontaneous activity may be due to functional isolation of muscle fibre segments from the end-plates zones. In the former situation, potentials are thought to arise from the original end-plate (McComas 1977). Positive sharp waves are also thought to originate from the old end-plate zone but have also been recorded from other locations along the muscle fibre membrane. These potentials, unlike fibrillations, are too small to trigger an action potential.

Several works have reported spontaneous activity in the hemiparetic limb of subjects with cerebrovascular disease (Goldkamp 1967; Bhala 1969; Kruger and Waylonis 1973; Zallis et al. 1976; Johnson et al. 1975; Spaans and Wilts 1982; Cruz Martinez 1983). Incidence of fibrillation potentials and positive sharp waves ranged from 56.8% and 69.6% respectively (Goldkamp 1967, n = 116 patients) to 100% (Johnson et al. 1975, n = 20 patients) and were more prominent in the upper extremity than in the lower extremity. Sixty-five percent (65%) of patients had fibrillation potentials and 74% demonstrated positive sharp waves in the upper limb as compared with 30% fibrillations and 46% positive sharp waves in the lower limbs (Kruger and

Waylonis 1973; Cruz Martinez 1983; Bhala 1969).

Spontaneous activity was most pronounced in the distal muscles but was also found in proximal muscles. It was also most prominent and persistent in cases of flaccid hemiplegia. This observation coupled with the observation that spontaneous activity disappeared first in the antigravity muscles (biceps, wrist flexors and finger flexors in the upper limb and quadriceps and gastrocnemius - soleus muscles of the lower limb) support an argument in favour of a trophic interaction (greater decreased axoplasmic flow distally) between the UMN and LMN. It may also be postulated that since spontaneous activity recovers first in those muscles involved in the flexor synergy of the upper limb and extensor synergy of the lower limb, it may be that these spinal reflex postures provide a neurotrophic influence on the LMN.

All studies noted that fibrillation potentials did not appear before 2 weeks and rarely past 6 months and disappeared when voluntary movement and spasticity appeared. It was also observed that in 78% of subjects who were able to voluntarily activate motor units, fibrillations were absent (Zallis et al. 1976). Fibrillation potentials were often associated with decreased evoked muscle potential amplitudes (Zallis et al. 1976; Spaans and Wilts 1982).

These findings must be placed against the work of Alpert et al. (1973) who studied 128 muscles of hemiplegic (stroke) patients testing 1-2 weeks post stroke with a minimum of three sites in each muscle. They found fibrillation potentials in only 2.3% of the muscles in the paretic limb. Conclusions were that any signs of LMN dysfunction in vascular hemiplegia are due to a compression neuropathy.

b). M-wave amplitudes

M-wave amplitudes are the electrophysiological measures of excitable muscle mass. These amplitudes are proportional to the total cross-selectional areas of the active muscle fibres in each muscle. The accuracy of this technique depends on all the muscle fibre action potentials being averaged by the recording electrodes. M-wave amplitudes indicate the integrity of the muscle fibre membrane and are an effective way of assessing muscle fibre atrophy.

Zallis et al. (1976) showed that M-wave amplitudes recorded from the affected limb in stroke syndrome were 1/3 that of the unaffected limb and demonstrated an inverse correlation between the presence of fibrillation potentials and M-wave amplitude. Other investigators (McComas et al. 1973; Spaas and Wilts 1982) also found reduced evoked potential amplitudes on the paretic side. In the McComas

study, it was shown that after 19 months post stroke, there was an increase in M-wave amplitudes on the affected side.

c). Motor Unit Potential Amplitudes

The size of the motor unit potentials are an indication of the functional status of the motoneurons. Very large motor unit potentials are indicative of denervation and re-innervation while small motor unit potentials suggest muscle atrophy. Changes in motor unit potentials are associated with changes in muscle fibre size and muscle fibre grouping. Mean motor unit potential amplitudes in the affected limb were significantly greater than those in the unaffected limb (McComas et al. 1973). No evidence was found to suggest that a loss of functioning motor units was related to a lesion in a particular hemisphere. In the McComas study, it was observed that little change in mean size of motor unit potentials occurred before 20 months. This finding in conjunction with decreased responses to repetitive nerve stimulation and prolonged terminal latencies, prompted the authors to postulate that the motoneurons were in a dysfunctional state. After 19 months, the mean motor unit amplitudes increased as did the M-wave amplitudes indicating that axonal sprouting and the adoption of denervated muscle fibres by surviving motoneurons occurred or perhaps muscle fibres hypertrophied. Results from this study seemed to indicate that previously dysfunctional muscle fibres were

restored. This restoration may have been due to increased trophic effects from spinal neurons which, by axonal sprouting, may have taken over synaptic sites abandoned by degenerating corticospinal fibres on alpha motoneurons (McCough et al. 1958).

Sequra et al. (1981) report evidence of small polyphasic motor unit potential (MUP's) in some hemiparetic muscles (EDB, biceps and vastus medialis) as early as 1 month post stroke. At 5 months there was an increase in large and polyphasic voluntary motor unit potentials but these units were reduced in number. At 24 months, MUP's showed an increase in large or slightly large voluntary MUP's which coincided with normal strength except in distal muscles of the hand and foot.

The above studies attempted to determine origin of the LMN signs observed electromyographically (EMG). The arguments are as follows. If the findings reported were due to a brachial plexus traction lesion caused by subluxation of the shoulder or a compression neuropathy due to either the upper limb or lower limb being pressed against a wheelchair, the muscle atrophy should have had a segmental distribution in accordance with the peripheral nerve involved. However, muscle atrophy was usually profound and diffuse (Bhala 1969; Cruz Martinez 1982; 1983). Further, if a radiculopathy (nerve root involvement) were the source of

the abnormality then the paraspinal muscles should have been involved. Kruger and Waylonis (1973) report normal paraspinals in 90% of cases studied and argue that this finding and the LMN signs noted in limb muscles are consistent with a trophic influence on the LMN because a cortical lesion of area 4 (motor area) has more corticospinal fibres and would affect the limb muscles more severely than destruction of area 6 (pre-motor area) which has fewer, high threshold fibre projections which supply the paraspinal muscles. These authors also argue that a lesion of the nerve roots is improbable because voluntary motor unit potential amplitudes were normal even in muscles of flaccid limbs. The fact that nerve conduction velocities were normal, spontaneous activity was not bilateral and the diabetic, hemiplegic patients studied had fewer abnormal E.M.G. findings than the other patients or controls seem to discount a peripheral neuropathy (Kruger and Waylonis 1973; Lawrence and Locke 1961).

Spaas and Wilts (1982) also argue against a peripheral nerve lesion. Their argument is based on evidence of normal sensory potentials. They believe that spinal sensory ganglion cells and their peripheral axons are not dependent on a CNS effect. Reduced or absent motor unit potentials on maximum effort and a normal evoked muscle mass action

potential elicited with electrical stimulation are all suggestive of a cortical lesion as the source of spontaneous activity. They also report a steady recovery pattern in serial studies in which the number of fibrillation potentials decrease with time and the density of the motor unit pattern on maximal effort increases. There may also be an increase in the amplitude of the M-wave response. These authors also argue that in their subjects the finding of profuse fibrillation potentials with normal tendon reflexes rule out spinal root avulsions.

Cruz Martinez (1983) points out that while some hemiplegic patients may have plexus lesions, those studied showed early decremental responses to repetitive nerve stimulation and slowing of nerve conduction velocities of the peripheral nerve as well as plurisegmental evidence both proximally and distally of spontaneous activity, all supporting an UMN-LMN hypothesis.

The other issue explored is that of vasomotor disorders. It is postulated that blood in the cerebrospinal fluid (CSF) would irritate nerve roots. If vasomotor disorders were the cause of spontaneous activity, then there should be involvement of paraspinal muscles and fibrillation potentials should be found bilaterally. This was not the case (Kruger and Waylonis 1973; Bhala 1969). These studies have all concluded that LMN signs in stroke syndrome are due to a direct cortical trophic influence.

2. Contractile Properties

a). Factors Affecting Contractile Properties

Three features are of prime interest in considering the contractile properties of skeletal muscle in stroke syndrome. They are the length of time it takes the muscle to contract to peak tension - contraction time (CT), how long it takes the muscle to relax - relaxation time (RT), measured as half relaxation time (1/2 RT), and how much force the muscle produces at full contraction - twitch torque (T.T.). The time course of skeletal muscle contractions is not fixed (Buller and Eccles 1960; Vrbová 1963) and two main theories exist concerning factors which affect the speed of muscle contraction. These theories focus on axoplasmic transport and impulse transmission.

1). Axoplasmic transport

It has been postulated that the speed of contraction of muscle is determined by the trophic substances travelling down the motoneuron (Buller et al. 1960). This theory was tested in cross-innervation experiments on kittens in which the nerve to the soleus muscle was grafted to the flexor digitorum longus (FDL) muscle and vice versa. The purpose of these studies was to determine the effects of a slow motoneuron on fast muscle and fast motoneurons on slow muscle (Buller et al. 1958; 1960). Results showed that slow soleus developed faster CT and 1/2 RT became shorter.

The fast FDL re-innervated by slow soleus nerve had a slower CT and 1/2 RT became lengthened. These studies support either a trophic effect or impulse activity effect on contractile properties of muscle.

2). Impulse Transmission

Later studies stated that the contractile properties of skeletal muscle are dependent on the pattern or frequency of impulses to the muscle (Vrbovā 1963; 1966; Eccles and Eccles 1962; Lømo et al. 1979; Salmons and Vrbová 1969). All muscles are thought to become fast unless subjected to the electromechanical effects of activation at a frequency of 10/s. This frequency parallels the normal frequency of discharge of slow motoneurons (Eccles and Eccles 1962). Vrbovā (1963) studied spinalized rabbits in which the achilles tendon was transected and showed that nerve stimulation at 10/sec stopped the soleus muscle from becoming fast. Lømo et al. (1979) studied the denervated soleus muscle in the rat and demonstrated that intermittent high frequency (100 Hz) stimulation makes slow muscle fast while low frequency stimulation (10 Hz) makes slow muscle slower. However, high frequency stimulation (intermittent or infrequent) always produced fast contractile properties. It may be theorized that regardless of whether stroke syndrome is characterized solely by an UMNL or by an UMNL with a secondary LMNL, cortically induced impulse

transmission in motor axons would be altered and reflected in the contractile properties of skeletal muscle.

3). Muscle atrophy

The above theories are of importance in the study of neuromuscular dysfunction in stroke syndrome. As patients regain more motor control, clinical observations of changes in the skeletal muscle have been well documented (Twitchell 1951; Brunnstrom 1971). Initially, the muscle is in a state of flaccidity which may evolve to stages of spasticity followed by a subsequent decrease in spasticity and the appearance of coordinated voluntary movement. In other instances, flaccidity may result in muscle atrophy. Much speculation surrounds the cause of muscle atrophy in cerebrovascular hemiplegia. Lesions of the UMN may produce an arthropathy, disuse, or vascular changes which in turn may result in muscle atrophy. Transynaptic motoneuron degeneration, pressure neuropathies and parietal lobe lesions are also thought to be causes of muscle atrophy in stroke syndrome (Segura and Sahgal 1981; Chokroverty and Reves 1976; Solandt and Magladery 1942; McComas et al. 1973, Fenichel et al. 1964). If muscle atrophy is viewed as having occurred because of a net decrease in excitation of the alpha motoneuron with subsequent alteration in axoplasmic transport and normal impulse activity then histological evidence should parallel findings of abnormal

electrophysiological measures of skeletal muscle contractile properties. It may be reasoned that flaccid muscle would have prolonged contraction times and either normal or slightly prolonged relaxation times (McComas 1977). There would probably be evidence of decreased twitch torque or evidence of an inverse relationship between peak tension and CT; that is, it might take longer for the muscle to produce its peak tension.

4). Spasticity

If spasticity is viewed as a net increase in excitation of the alpha motoneuron, (Herman et al. 1973; Rushworth 1960) and if excitation results in a chronic stimulation pattern of muscle, then based on work of Vrbová, (1963, 1966) one might expect the contraction time of the muscle to be normal or shortened but relaxation time could be prolonged because of delayed re-absorption of calcium into the sacroplasmic reticulum (Young and Delwaide 1981).

b). Stroke Syndrome and Muscle Contractile Properties.

1). Whole Muscle Studies

Analysis of muscle contractile properties in patients who are hemiparetic due to cerebrovascular disease (McComas et al. 1973) showed a significant decrease in twitch torque in the paretic limb. This finding is consistent with histological studies of skeletal muscle in hemiplegia which showed evidence of extreme variation in fibre size and

grouping of small atrophic muscle fibres (Segura and Sahgal 1981). Reports of decreased mean fibre cross-sectional area of type II muscle fibres (Edstrom 1970) reported are compatible with decreased twitch torques. Edstrom (1970) postulated that the cause of muscle atrophy in hemiplegia is due to selective disuse of high threshold motor units which results in increased firing of low threshold units.

Mean contraction times on the affected side were significantly slower than those on the normal side (McComas et al. 1973). Half-relaxation times showed no significant difference between limbs. McComas et al. (1973) argue that surviving motoneurons may have been motoneurons which received the smallest corticospinal inputs and the largest segmental ones. These units may also have innervated muscle fibres having relatively slow isometric twitches. They also argue that it is unlikely that surviving motor units are all slow twitch units.

The contractile properties of spastic elbow flexors of 7 hemiparetic subjects (Ismail and Ranatunga 1981) were assessed. In this work, spasticity was described as "marked". There was no significant difference in twitch torque, contraction time or half relaxation time between the affected and unaffected limbs. There was also no effect for sex or time post stroke. Results from patient data were not significantly different from a control group.

2). Single Motor Unit Studies

Young and Mayer (1979) studied the mechanical properties of single motor units and found that fast twitch units had significantly shorter mean contraction times but showed no change in twitch torque when compared with controls. Slow twitch units showed no change in mean contraction time but twitch torque values were similar to those of fast twitch units. Normally, fast twitch fatigable (FF) units have the largest twitch and tetanic tensions and fatigue on repetitive stimulation. Fatigue resistant (FR) units have intermediate size twitch and tetanic tensions and are resistant to repetitive stimulation. Slow twitch motor units are found to have the smallest twitch and tetanic tensions. This work showed that after a short period of flaccid hemiplegia, all motor units have slower contraction times and slow twitch units generate more twitch tension.

Isometric contractions of single motor units in the first dorsal interrosseous muscle were studied more intensively in 24 hemiplegic patients (Young and Mayer 1982). Subjects were divided into an acute group (2-31 days post stroke) and a chronic group (10 weeks - 10 years post stroke). The acute group was further divided into those subjects whose muscles were flaccid and those demonstrating spasticity. Results showed that the mean contraction time

of all motor units was slightly greater in hemiplegic patients than controls. In addition, the mean contraction time of fast twitch motor units was slightly longer in spastic hemiplegia. Slow twitch units generated a greater twitch tension in spastic hemiplegia as well. Evidence of fast twitch (FF) fatigable, fast twitch fatigue resistant (FR), slow twitch fatigue (S) resistant units were found in all groups but a fourth type of motor unit based on its contractile properties, termed slow twitch fatigable (SF) was also identified. These units represented 21% of the units assessed and had slow twitch contractile times. This study also demonstrated that the SF units demonstrated the property of "sag", a feature of fast twitch units. The authors concluded that these motor units must have originally been fast twitch units. Further, all motor units in the hemiplegic group showed an increase in fatigability. This finding is compatible with the work of Lømo et al. (1979) who found that resistance to fatigue depends on the sum of impulse activity. More slow twitch units were found in the chronic than in the acute hemiplegic cases. Conclusions of this study were that, because increases in twitch tension were observed in all units in spastic hemiplegia, their appearance could not be explained on the basis of hypertrophy of type I muscle fibres (Edstrom 1970). It may be argued that increased twitch torque is due to

collateral reinnervation (McComas et al. 1973). It was postulated that the muscle weakness in chronic cases may be due to increased fatigability of all motor units and the appearance of SF units. Prolonged contraction times were thought to be due to a prolonged active state in hemiplegia (re-absorption of calcium into the sarcoplasmic reticulum), which would increase twitch torque. Prolonged CT may occur even if the muscle was atrophied and subsequently twitch torque would be increased.

III. MATERIALS AND METHODS

A. SUBJECTS

1). Population

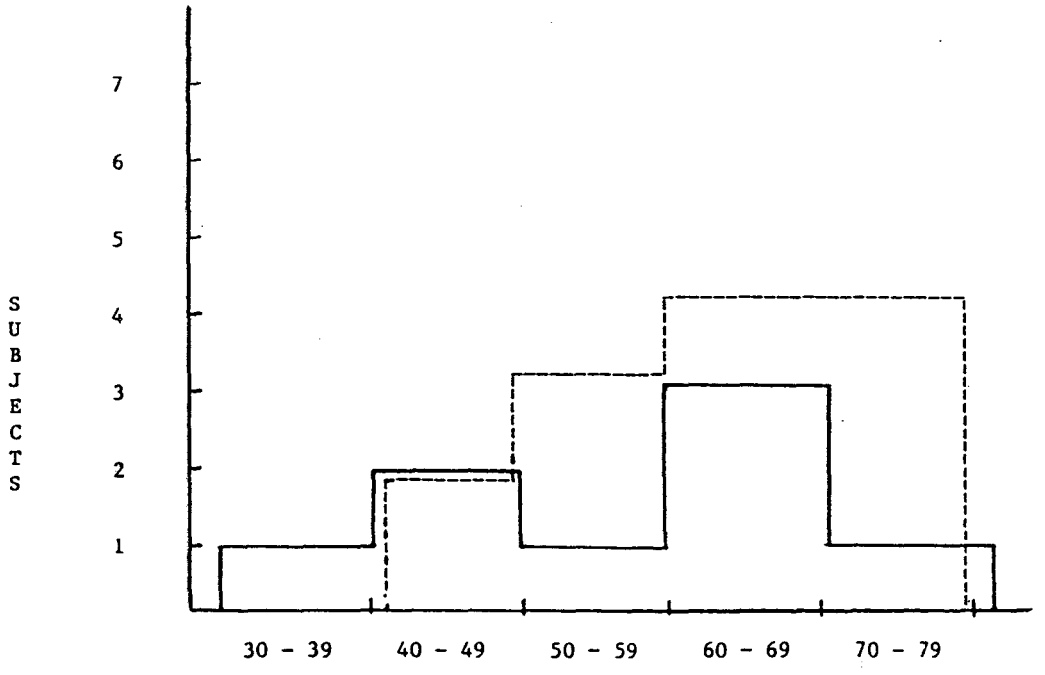
Subjects were recruited from the Stroke Units at the McMaster University Medical Centre, the Chedoke-McMaster Rehabilitation Centre and the Hamilton Stroke Recovery Association. The purpose and procedures to be applied were explained and consent was obtained from either the patient and/or his family. The study was approved by the Ethics Committee at the McMaster University Medical Centre. Permission to review the charts of subjects was obtained from the medical directors of St. Joseph's Hospital, Hamilton, Ontario and the Hamilton Civic Hospitals. From the above populations, a sample was selected which had the following characteristics.

2). Sample

The sample consisted of 22 subjects of which 14 were males and 8 were females. The mean age was 59.7 $\pm$ 11.8 years (range 36-73 years). The mean age for females was 55.5 $\pm$ 15 (SD) years and $\pm$ 9.6 62.1 (SD) years for males. Twelve subjects were between 59-79 years, 8 of which were males and 4 were females. (See figure 1). All testing was done on both the hemiplegic and non-hemiplegic limb with the non hemiplegic limb being considered the control limb. Bilateral reflex testing and a careful review of CAT scan reports and histories of all subjects were done to make a reasonable determination that the unaffected limb was, in fact, "normal".

Figure 1. The Relationship Between Number of Subjects and Age

The continuous line indicates the number of females in a particular age category and the broken lines depicts the number of males in each group.


SUBJECTS
7
6
5
4
3
2
1
30 - 39
40 - 49
50 - 59
60 - 69
70 - 79
AGE (YEARS)

While all subjects were hemiparetic due to cerebrovascular disease, and all strokes were complete, 6 males and 4 females had vascular lesions involving the territory of the brain supplied by the left middle cerebral artery and 8 males and 4 females had lesions involving the right middle cerebral artery area. Lesion site documentation was unavailable on 2 subjects and was deduced from the patients' verbal history.

Of the total sample, 3 subjects had previous transient ischemic attacks (T.I.A.'s) or a previous stroke, 1 subject had a 10 year old history of Guillian-Barre Syndrome, 5 subjects were diabetic, and 1 patient had an old sciatic nerve injury and presented with marked atrophy of the tibialis anterior (TA) muscle on the hemiparetic side. This latter subject had received no previous physiotherapy and had been braced immediately post stroke. It was uncertain whether the muscle atrophy was due to disuse, the nerve injury or the site of the cerebrovascular lesion. Because of this uncertainty, the subject was excluded from the study. Two additional subjects had arteriosclerosis, another had a history of peripheral vascular disease and a third had a very short attention span. Of those subjects who had previous T.I.A.'s, there was no clinically discernable motor loss in the unaffacted limb.

3). Classification of Subjects

a). Major Classification System

Ten subjects were classified as being "acute" (less than one year post-stroke) and 12 subjects were "chronic" (greater than one year post stroke). Total time post stroke ranged from 9 days to 22 years.

b). Sub-Classification System

Subjects were further classified according to the stage of motor recovery of their leg and foot according to a modified version of the Brünnstrom Assessment (See Appendix A). This assessment technique is based on the work of Twitchell (1951) who showed that patients who have had a cerebrovascular accident (CVA) or "stroke" regain motor control in a step-wise fashion and that while a patient may plateau at any level, none of the stages are ever skipped. Because the assessment form is a recognized indicator of motor recovery over time, it was selected for use in this study in an attempt to assess neuromuscular physiological return over time.

Main features of the Brünnstrom Assessment are that the patient must be able to perform a specified number of activities in order to qualify for any particular "stage". The rating scale ranged from Stage 1 in which there is no active movement to Stage 7 in which motor control was considered normal (See Appendix A). In the work being reported, the classification of the subjects was as outlined in Tables 1, and 2. To determine the effects of a stroke on

Table 1

The Relationship Between Number of Subjects and Stage of Motor Recovery of the Foot and Leg

Stage of Motor Recovery Foot and Leg	Number of Subjects Foot	Leg
1	3	1
2	9	0
3	2	6
4	2	3
5	1	3
6	5	7
7	0	2
	n = 22	n = 22

TABLE 2

The Relationship Between the Number of Subjects, Stage of Motor Recovery, Time Post Stroke and Sex*

Stage of Motor Recovery	Number of Subjects				
	Acute		Chronic		TOTAL
1	1	(0)F	0		1
2	0		0		0
3	2	(1)F	4	(2)F	6
4	1	(0)F	2	(1)F	3
5	2	(2)F	1		3
6	4	(1)F	3	(1)F	4
7	0		2		0

* Indicates the number of females in each group

muscle over time, two sub-groups were devised.

c). Sub Groups

Six acute subjects were tested on two separate occasions, usually within one month post stroke in an attempt to determine physiological change of the neuromuscular system over time in the acute stage of motor recovery. The mean age of these subjects was 66.3 ± 8 years (range 57-73 years). Four subjects were males and 2 were females.

For the purpose of data analysis, the data from 9 subjects (acute and chronic) under the age of 60 years were also examined. The mean age of these subjects was 47.4 ± 7 years (range 36-57 years). Five subjects were males and 4 subjects were females. This sub group was assessed in order to compare the results of this thesis work with a comparable sample studied by McComas et al. (1973).

B. MEASUREMENTS

Measurements were taken of the twitch contractile properties, maximum voluntary strength, and motor unit activation of the tibialis anterior muscle. Motor unit counts were recorded for the extensor digitorum brevis muscle. Peroneal nerve conduction velocities and measures of excitable muscle mass of both muscles were determined.

1. Twitch Contractile Properties

a). Purpose of Measurement

To assess muscle fibre integrity, measurements were made of the evoked twitch contractile properties (twitch tension, contraction time and half relaxation time), and the maximum isometric voluntary contraction of the tibialis anterior muscle.

b). Muscle Group

Tibialis Anterior Muscle

This muscle arises from the lateral condyle of the tibia, the interosseous membrane and the upper two thirds of the lateral aspect of the tibia. At its distal end, this muscle forms a long tendon which passes medially as it crosses under the extensor retinaculae at the ankle joint. It inserts on the medial and inferior surfaces of the medial cuneiform and the base of the first metatarsal bones.

This muscle has been shown to be comprised of 73% type I slow twitch muscle fibres and 27% fast twitch type II muscle fibres (Johnson et al 1973). Tibialis anterior produces maximum voluntary torque when stretched to 30° plantarflexion. It has also been shown to produce 42% of the force produced by all the dorsiflexor muscles (extensor digitorum longus, extensor hallucis brevis and peroneus tertius) when subjected to a 100 Hz tetanus (Marsh et al 1981). It was selected for the study for the following reasons.

1) In the leg, the tibialis anterior muscle shows regression poststroke before the gastrocnemius, peroneus and other extensor muscles of the toes (Licht 1975). Further, increased muscle tone in the lower limb, occurs first in the plantar flexor muscles (Licht 1975) rather than the dorsiflexor muscles. Therefore, the tibialis anterior muscle is suitable for evaluation over time.

2) Because tibialis anterior is a superficial dorsiflexor muscle of the leg, it is amenable to surface electromyographic recording (Goodgold 1983). During both strength testing and electrophysiological evaluation, the functioning of the muscle is easily observed.

3) Because the muscle action of tibialis anterior occurs in one plane, with one articulation (even though the dorsiflexor action also involves inversion of the forefoot), it is an appropriate muscle for evaluating isometric maximum voluntary contractions (Twitch). These features were recognized by Marsh et al. (1981) who developed a torque measuring device to study this muscle.

4) Tibialis anterior is innervated by one nerve, the common peroneal nerve, which also supplies the extensor digitorum brevis muscle. This nerve is easily stimulated as it crosses the neck of the fibula as well as being reasonably accessible at the ankle. The peroneal nerve also has a low threshold for stimulation and may be activated

FIGURE 2

COURSE OF THE PERONEAL NERVE

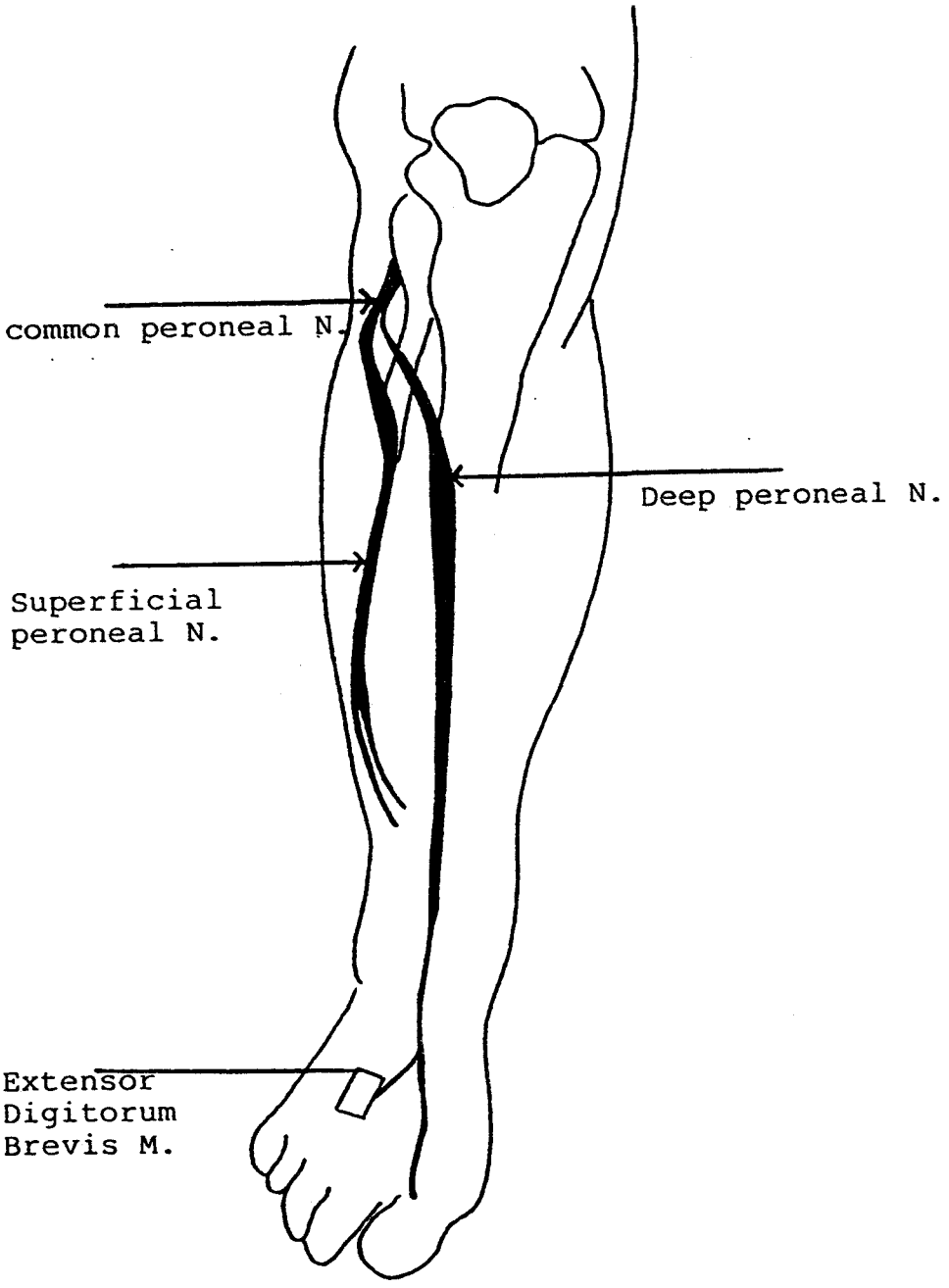
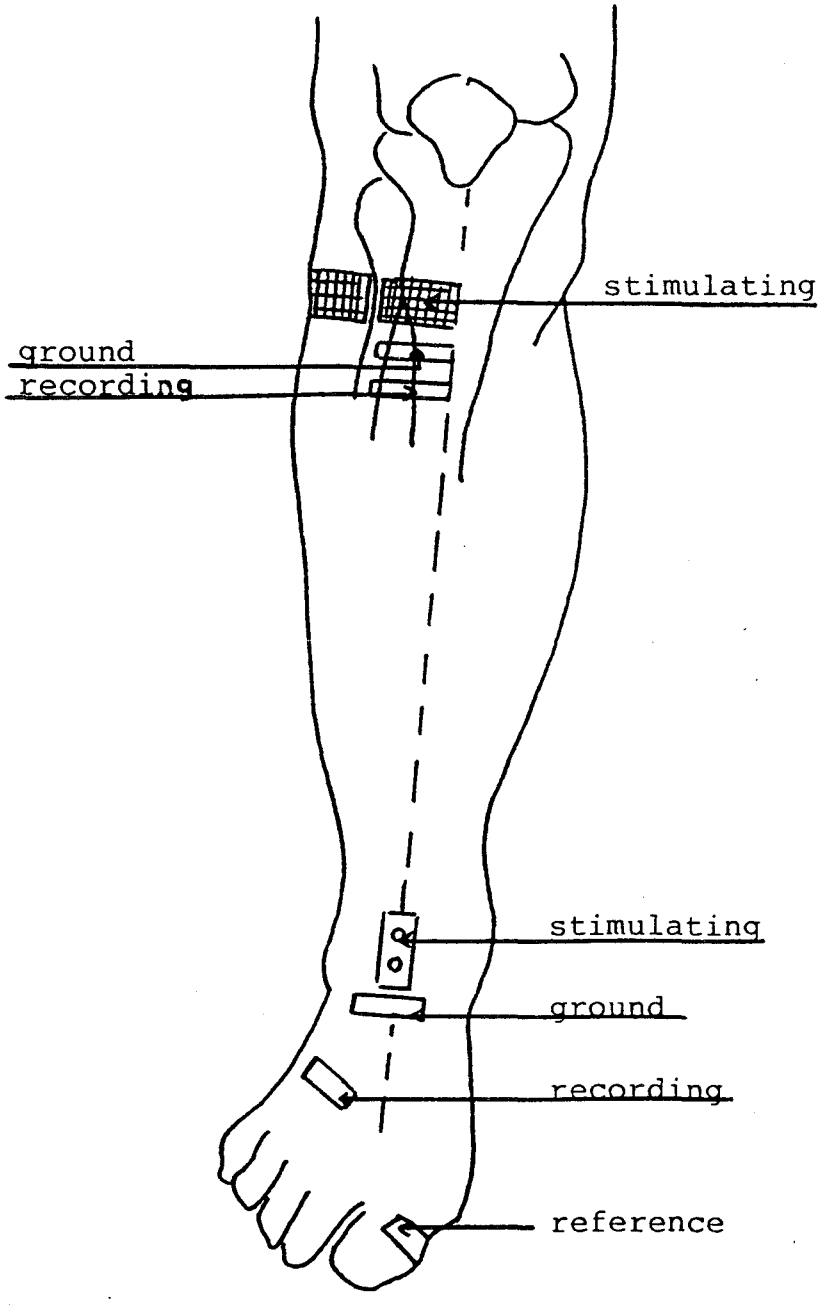


FIGURE 3

ELECTRODE PLACEMENT FOR STIMULATION OF THE PERONEAL NERVE, RECORDING FROM TIBIALIS ANTERIOR AND EXTENSOR DISITORUM BREVIS MUSCLES.



with little discomfort to the patient and with minimum chance of stimulating cutaneous pain reflexes. This common nerve supply to both tibialis anterior and extensor digitorum brevis muscle permits study of both muscles with minimal disruption to the patient (see figure 2 and 3).

c). Apparatus

Leg Holding Device

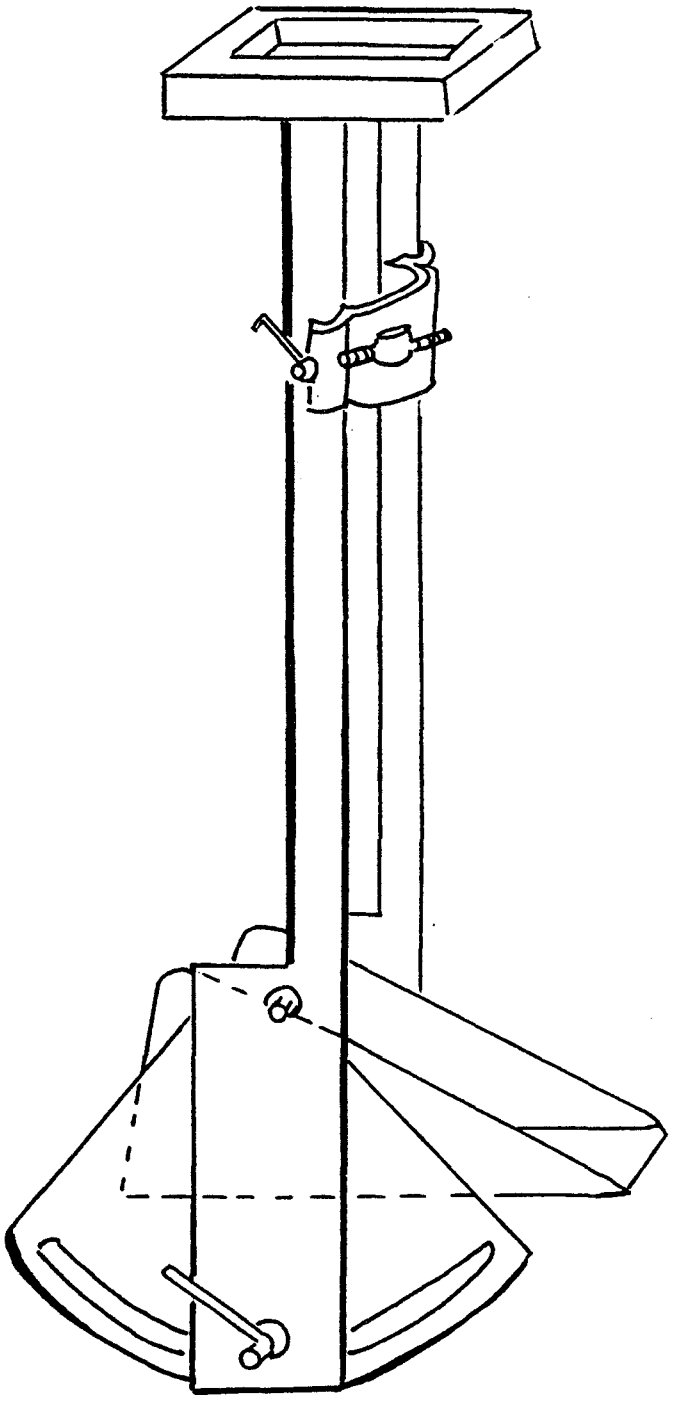
A leg-holding device (Figure 4) with an adjustable foot plate was used to determine the amount of tension produced by the tibialis anterior muscle. Pressure from torques acting on the foot plate was transmitted to strain gauges which were mounted on a rigid bar attached to the foot plate of the leg-holding apparatus. An electrical signal was passed via the strain gauges to a preamplifier and then to a variable-persistence cathode ray oscilloscope (Hewlett-Packard 141B). Calibration of the system was done with known torques of up to 74.53 N.m.

Rectangular voltage pulses, 50-100 μs were delivered from a Devices Limited Model 3072, constant voltage stimulator to the peroneal nerve at the neck of the fibula. The stimulator received a triggering pulse from a digital timing device (Devices Limited Digitimer, Model 3290).

The E.M.G. signal from the tibialis anterior muscle was passed through an amplifier with a band pass of 10 Hz - 1 KHz and was displayed on a variable persistence cathode ray

FIGURE 4

A LEG HOLDING DEVICE



oscilloscope (Hewlett-Packard Model 141B).

Position of Subjects

Four criteria for selecting the test position were determined. Firstly, the position selected had to facilitate the development of twitch torques in order to compare the contractile properties of the affected and control limbs. Secondly, the influence of the plantarflexors had to be eliminated. Thirdly, the position had to permit evaluation of the EDB muscle. Fourthly, the position had to provide maximum safety, stability and comfort for the subjects, some of whom were acutely ill.

To achieve these aims, the patient sat either in his/her wheelchair or in a "barber's chair" which could be raised or lowered according to the length of the subjects tibia. The knee joint angle was set at 90° by either adjusting the height of the "barber's chair" or by positioning the limb with pillows when the subject was tested in a wheelchair. A clamp over the knee was used to prevent raising of the heel. The limb was maintained in mid-position (internal-external rotation of the hip) with velcro straps which were attached around the vertical side rails of the leg-holding device at mid-calf level, anteriorly and posteriorly. The foot was stabilized on the foot-plate at an angle of 15° plantarflexion by securing velcro straps over the top of the foot. Positioning

subjects in the described manner satisfied the experimental criteria. Physiological justification for the above position is as follows.

Firstly, the work of Marsh et al. (1981) who showed that maximum twitch torque of tibialis anterior occurred when the muscle was in a lengthened position (30° plantarflexion) is recognized. Hemiplegic subjects could not tolerate 30 degrees of plantarflexion because the acute subjects were severely debilitated and had very poor or no sitting balance. This handicap resulted in the subject increasing the extension angle of the hip when sitting, producing not only discomfort in the forefoot but also excitation of the plantar flexor muscles. Marked flaccidity of the lower limb also exerted a significant influence on the discomfort felt by these subjects. Chronic subjects were often severely spastic and could not tolerate 30° of plantarflexion. While it is evident that the position selected produced satisfactory torque, it is likely that the position selected did not represent the optimal muscle length for force development.

Secondly, while it is also recognized that Marsh et al. (1981) avoided exciting the peroneus muscles by stimulating directly over the dorsiflexor muscles, it was felt that the chances of producing cutaneous pain reflexes in hemiplegic patients with this technique were greater than by

stimulating the peroneal nerve.

d) Procedure

Limb Temperature

It has been shown (LeQuesne 1971) that nerve conduction velocity decreases by 2.4 m/s per degree fall in intramuscular temperature and by 1.2 m/s per degree Celsius fall in skin temperature. In the present investigation, an attempt was made to control the skin temperature by either, a) immersing both limbs in a bucket of very warm water, (Geerling and Marchese 1985) or b) by warming the limb with an electric blanket. The mean skin temperature of the affected limb was 31.9 $\pm$ 2° C and the unaffected limb was 32.2 $\pm$ 2.4 degrees. Skin temperature could not be maintained throughout the experiment even with the use of an infrared lamp and a room temperature of 25° C.

Skin Preparation

To decrease tissue impedence, the skin over which the electrodes were to be placed (see figure 3) was rubbed with alcohol swabs, but was not debrided for fear of causing abrasions which might heal poorly because of peripheral vascular deficits common to hemiplegic patients.

Electrode Preparation and Placement

All electrodes used in this project were surface electrodes.

i). Stimulating Electrodes

Stimulating electrodes consisted of two, 4 x 4 cm 8 ply gauze pads soaked in water and wrapped around 3 x 5 cm lead plates. These electrodes were prepared with conducting cream and fastened to the skin with skin tape. A velcro strap placed over the electrodes held them securely. The cathode was placed over the common peroneal nerve just below the head of the fibula. The anode was placed anteriorly to the cathode. While unconventional, this method of electrode application was chosen because it decreased the possibility of activating the tibial nerve and because lower stimulus voltage could be used to elicit the maximal muscle twitch in the tibialis anterior muscle. Anodal block was avoided because brief, rectangular voltage pulses (50-100 μs) were applied by the stimulator.

ii). Recording, Reference and Ground Electrodes

Recording, reference and ground electrodes were rectangular silver strips 5 mm x 4 cm prepared with electrode conducting cream. The recording electrode was placed mid-way over the belly of the tibialis anterior muscle and the ground electrode was placed between the stimulating and recording electrodes. The reference electrode was taped over the great toe (see figure 3).

iii). Stimulus Application

A supramaximal stimulus was given to the peroneal nerve

to elicit a total muscle response (maximum M-wave). The latency, peak-to-peak amplitude of the compound muscle action potential and voltage required to produce the M-wave were recorded. Once the M-wave had been established, another supramaximal twitch torque of the muscle. The maximum stimulus intensity was 400 volts (Mean 260 $\pm$ 70 V). The affected limb often required a greater stimulus intesity and longer pulse width (100μs) to evoke a response than did the unaffected limb.

The following measurements were taken of the twitch contractile properties and were estimated from a display on the variable persistence cathode ray oscilloscope.

a) peak-level of the twitch torque

b) twitch contraction time

c) half relaxation time

Simultaneous to the above procedures, the M-wave response and EMG activity of the muscle was recorded on separate channels, displayed and photographed.

The twitch torque, expressed in newton metres is equal to the amplitude of the response seen on the oscilloscope. Contraction time is the time of the first deflection of the signal from the baseline on the oscilloscope to maximum torque and is expressed in milliseconds. Half relaxation time is the time from maximum torque to one-half maximum torque expressed in milliseconds.

2. Voluntary Muscle Strength

a) Purpose of Measurement

This measure was used to quantify the amount of force produced by the dorsiflexor muscles of the paretic and non-paretic lower extremities during a maximum voluntary contraction. It is recognized that the tibialis anterior muscle only produces 40% of the force produced by the dorsiflexor muscle group (Marsh et al. 1981).

b) Apparatus

Stimulus parameters were as previously described. The apparatus used, skin preparation, and electrode placement were the same as those used in the isometric twitch studies.

c) Procedures

After the isometric twitch studies were completed, the subject was permitted a 30 second rest. A second stimulus was then applied and the evoked twitch characteristics were measured. Next, the subject performed a maximum voluntary contraction (MVC). During the MVC, a single twitch stimulus was applied in an attempt to determine whether complete muscle activation had occurred. Three trials were attempted and the best results were recorded.

3. Motor Unit Activation

a) Purpose of Measurement

The measurement technique used was the interpolation twitch technique (Bēlanger and McComas 1981, Chapman et al.

1985). This method involves measuring the maximum voluntary contraction produced. While the subject produces a MVC, a supramaximal stimulus is applied to the appropriate peripheral nerve. If all the motor units are being utilized optimally to produce the MVC, no interpolated response will be observed on the force display on the oscilloscope. If all the motor units are not being recruited, a twitch will be superimposed on the force display.

b) Apparatus

Apparatus and stimulus parameters are as previously described.

c) Procedures

During the MVC procedures previously described, a single twitch stimulus was applied to determine whether full muscle activation had occurred (interpolated twitch). Maximum voluntary contraction was ensured by having the subject dorsiflex strongly against the footstrap. Encouragement was given verbally by the tester and visually from the oscilloscope. The length of time the MVC could be maintained varied with each subject and the twitch stimulus was timed accordingly. The amplitude of the MVC and interpolated twitch was read from the oscilloscope. The interpolated twitch technique was followed by a rest period lasting for several seconds. Following the rest another single twitch stimulus was applied. This procedure was followed to account for the possibility that potentiation of the twitch from the original twitch study would likely occur. The interpolated twitch technique involved three

trials. Mean values were used to calculate mean percent motor unit activation.

Calculations of the percent motor unit activation were done by calculating the difference between the twitch torque, as determined in the isometric twitch study when the muscle was at rest following the MVC, and the interpolated twitch torque. The value obtained was then multiplied by 100.

$$\% \text{ M.U.A.} = \frac{\text{T.T. (Rest)} - \text{I.T.T.}}{\text{T.T. (REST)}} \times 100$$

% M.U.A. is the percent motor unit activation, T.T. (Rest) is the value of the twitch torque measured at rest and I.T.T. is the value of the interpolated twitch torque.

4. Motor Unit Counts

a) Purpose of Measurement

The purpose of this measurement technique was to assess the number of functional motor units in both acute and chronic patients following a cortical vascular lesion.

b) Muscle Group

Extensor Digitorum Brevis Muscle

Extensor digitorum brevis muscle (EDB) arises from the fore part of the upper and lateral surface of the calcaneum. It passes obliquely forwards and medially across the dorsum of the foot and ends in four tendons. The most medial

portion of the muscle inserts into the dorsal surface of the base of the proximal phalanx of the great toe. The other three tendons are inserted into the lateral sides of the tendons of the extensor digitorum longus muscle of the second, third and fourth toe. Its nerve supply is a branch of the deep peroneal nerve.

This muscle is reported to consist of 45% type I slow twitch muscle fibres and 55% (mean values) type II fast twitch muscle fibres Johnson et al. (1973). Its prime action is extension of the toes at the metatarsophalangeal joints.

The EDB muscle was selected for study because it is regarded as the only muscle supplied by the deep peroneal nerve below the ankle and the possibility of electrical interference from other muscles is eliminated. Controversy exists concerning the use of this muscle. Jennekens et al. (1972) and Rosselle and Stevens (1973) report evidence of muscle atrophy and abnormal spontaneous activity of EDB in healthy young adults. Ballantyne and Hansen (1974), Campbell, McComas and Petito (1973) found no fibrillation potentials but did find evidence of decreased interference pattern. McComas et al. (1971) argued that denervation may occur normally in EDB, but it is not likely to cause a significant decrease in motor unit population before age 60. This feature of EDB is important in assessing the results of

motor unit counts of patients who are usually older than 60 years.

Another potential problem when recording motor unit potentials from EDB is the possibility of interference occurring from muscles supplied by an accessory branch of the peroneal nerve. McComas et al. (1971) found considerable variation in their subjects who had this type of innervation. It is reported that when potentials were recorded from the interossei, they were inverted and smaller than potentials recorded from EDB. It is believed that the accessory nerve did not contribute significantly to EDB recordings (only one out of 23 subjects displayed the anomaly and only 10% of the motor unit counts could be attributed to the accessory nerve). A pilot study of 35 normal subjects done in preparation of this thesis found no evidence of this phenomenon.

Despite the above limitations this muscle was selected because of the reasons discussed and because the muscle has one motor end plate zone and studies have shown that the motor units obtained from this muscle summate algebraically.

c) Apparatus

The apparatus and position of the subjects has been previously described. However, some subjects were tested lying on a bed rather than sitting with the leg in the leg-holding device. The evoked twitch responses of these

subjects were performed as previously described, but on another day. Due to equipment restraints, the subjects tested in this manner were all from the Chedoke-McMaster Rehabilitation Centre.

d) Procedure

Limb Temperature and Skin Preparation

Limb temperature and skin preparation were as described, with the exception that those subjects tested in supine lying had their limbs warmed with a heating blanket.

Electrode Placement

The stimulating electrodes were silver chloride discs 10 mm in diameter, mounted in a Perspex holder so that their centers were 3 cm apart. The electrodes were applied with electrode jelly and fastened with a velcro strap. (see figure 3).

The recording or stigmatic, ground and reference electrodes were strips of silver foil 6 cm x 6 mm. The recording electrodes were taped over the motor-end plate region of EDB while the reference electrode was placed at the metacarpophalangeal joint (MP) of the great toe. The ground electrode was placed over the forefoot, proximal to the recording electrode.

Stimuli of 50 or 100 μs duration were delivered at a frequency of 20/min. The intensity of stimulation was gradually increased, from a sub-threshold level until the

first response was observed. This response of the motor units was seen to enlarge in discrete steps, as additional units were stimulated with increased intensity of stimuli. After 10 increments were obtained, their mean amplitude was calculated. A supramaximal stimulus was given to the peroneal nerve to elicit a maximum M-wave. The amplitude of the M-wave was then recorded. The motor unit estimate (McComas et al. 1971) was calculated in the following way:

$$\text{Motor Unit Estimate} = \frac{\text{Amplitude of the Maximum Muscle Compound Action Potential}}{\text{Mean Motor Unit Potential Amplitude}}$$

5. Nerve Conduction Velocities

a) Purpose of Measurement

The purpose of the application of this measure was to distinguish between "central" impulse transmission deficits and those abnormalities due to peripheral nerve damage which may have been caused by the CVA. Peripheral nerve conduction velocity measurements were done on the peroneal nerve. The technique used measured the impulse transmission of the fastest conducting nerve fibres in the peripheral nerve.

b) Apparatus

The apparatus and position of the subject remained unchanged. Limb temperature, skin preparation and electrode placement have been previously described.

c) Procedure

Stimulation and Recording

A supramaximal stimulus was applied at the ankle as well as on the lateral aspect of the lower limb distal to the head of the fibula. The nerve was also stimulated above the knee posteriorly at a point medial to the biceps femoris muscle. The amplitude of the response and the time interval between the stimulus and the onset of the muscle action potential was recorded in both instances. The difference in latency of the two responses is the conduction time in the fastest conducting fibres in the respective nerve trunks. Latency is the time between application of the stimulus and the point of negative deflection of the muscle action potential. Conduction velocity was calculated using the following formula.

$$\text{Conduction Velocity} = \frac{D}{t1 - t2}$$

D is the distance measured from the point of stimulation at the knee (distal to the head of the fibula) to the point of stimulation at the ankle or from a point medial to the biceps femoris muscle to point of stimulation

at the head of the fibula, t1 is the latency of the response read from the oscilloscope and obtained from stimulation of the peroneal nerve at the knee or at a point medial to the biceps femoris muscle and t2 is the response obtained at the point of stimulation at the ankle.

C. DATA ANALYSIS

Complete data sets were collected for 22 subjects. In some instances, individuals were not able to complete the required number of trials at one session. Testing was completed as soon as possible following the initial test.

Data were analyzed using the Ohio State Computer package. A three-way mixed analysis of variance was used to determine the effects of gender, time post stroke and limb status. Post Hoc tests were done to determine which specific means differed from each other when significant effects ($P = .05$) were found. In assessing the degree of functional loss in the affected limb, dysfunction was expressed as a ratio of the affected limb/unaffected limb. These ratios were analyzed using a two factor (gender and time) analysis of variance. The Pearson-product correlation method was used to determine whether there were significant correlations between age and stage of motor recovery, and variables which significantly differentiated between the affected and unaffected limbs.

In order to compare the results of this study with previous work (McComas et al. 1973), similar but separate analyses were conducted on the data of subjects under 60 years of age.

A Student correlated "T" test was used to determine statistically significant differences between first and second test situations for acute subjects. This method of analysis was also used to determine whether there was a learning effect when assessing motor unit activation and whether twitch torque had been potentiated between trials. In most instances, probability values for the various significant effects are reported in the text while the relevant statistics are reported in Appendix C.

IV. RESULTS

A. MAXIMUM VOLUNTARY CONTRACTION (M.V.C.)

The analysis of variance (ANOVA) for maximum voluntary contraction revealed a main effect for limb ($P < 0.01$) (Table 3) and a sex by limb interaction ($P < 0.05$) (Table 4). As expected, subjects demonstrated the ability to produce more force with the unaffected limb (26.2 $\pm$ 14 N.m.) than with the affected limb (10.0 $\pm$ 12.1 N.m.). Table 5 illustrates that these limb differences were more pronounced in the male subjects than the female subjects. This finding could be due to the fact that male subjects in this study were generally older and more severely disabled than the female subjects. As well as the sex by limb interaction, the interaction between time post stroke and limb approached conventional levels of significance ($P < 0.10$) (figure 5). Specifically, there were greater differences in force production between the affected and unaffected limb in acute patients than in the chronic patients (Table 6).

Further statistical analysis of variance in which impairment was expressed as a ratio of affected limb to unaffected limb yielded similar results. There was a significant influence of time post stroke ($P < 0.01$), once again indicating greater impairment in acute cases (Table 7, 8). F-ratios for the affected/unaffected limb analyses are reported in Appendix C.

TABLE 3

Mean Twitch Torque (N.m), Mean Maximum Voluntary Contraction (N.m), Mean Percent Motor Unit Activation and Percent Motor Unit Activation as a Function of Limb (N = 22)

Variable	Limb	
	Affected	Unaffected
Twitch torque (N.m) (tibialis anterior muscle)	2.3 ± 1.6	2.4 ± 1.5
M.V.C. (N.m)	10 ± 12.1	26.2 ± 1.4
$\bar{X}$ % M.U.A.	46 ± 36	79 ± 19.6
% M.U.A. (1st trial)	42.4 ± 33.6	86.6 ± 16.5

$\bar{x}$ ± SD

M.V.C. - maximum voluntary contraction

% M.U.A. - percent motor unit activation

$\bar{X}$ % M.U.A. - mean percent motor unit activation

TABLE 4

Mean Maximum Voluntary Contraction (N.m), Mean Percent Motor Unit Activation and Percent Motor Unit Activation as a Function of Sex and Time Post Stroke

Variable	N	Sex	Acute	Chronic
MVC	8	F	(4) 10.7 ± 8.1	(4) 16.6 ± 7.9
	14	M	(6) 15.8 ± 21.5	(8) 23.3 ± 14.9
$\bar{X}$ % M.U.A.	8	F	(4) 54.5 ± 34	(4) 71.1 ± 37.4
	12	M	(4) 37.4 ± 31	(8) 75.0 ± 31
% M.U.A.* (1st trial)	7	F	(3) 64.6 ± 36	(4) 66.4 ± 4
	13	M	(6) 44 ± 38	(7) 81 ± 21

$\bar{x}$ ± SD

TABLE 5

Mean Maximum Voluntary Contraction Percent Motor Unit Activation and Mean Percent Motor Unit Activation as a Function of Sex and Limb

Variable	N	Sex	Affected Limb	Unaffected Limb
M.V.C. (N.m)*	8	F	10.7 ± 10.3	16.8 ± 4.7
	14	M	9.3 ± 13.1	30.8 ± 15.4
% M.U.A.	7	F	47.7 ± 27.1	87.9 ± 14
	13	M	43 ± 36	90 ± 17.8
$\bar{X}$ % M.U.A.	8	F	50.5 ± 34	75 ± 35
	12	M	43 ± 37	81 ± 20

$\bar{x}$ ± SD

Figure 5 - Maximum Voluntary Contraction as a Function of Limb and Time.

MAX. VOLUNTARY CONTRACTION, LIMB & TIME

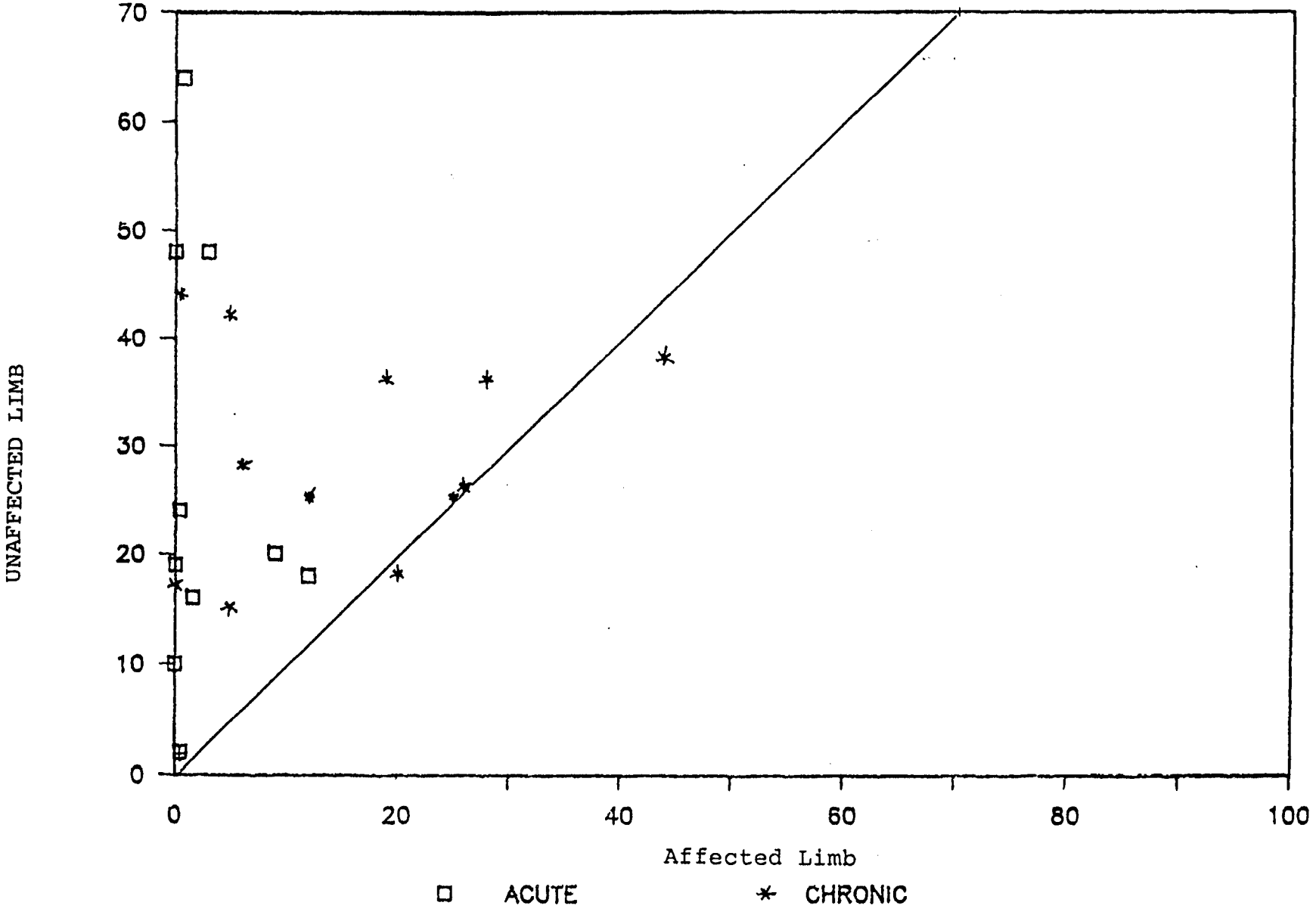


TABLE 6

Mean Maximum Voluntary Contraction and Mean Percent Mean Motor Unit Activation as a Function of Time Post Stroke and Limb

Variable	Time Post Stroke		Affected Limb	Unaffected Limb
M.V.C. (N.m)	Acute	(M & F)	2.55 ± 4.0	25.14 ± 18.4
	Chronic	(M & F)	15.9 ± 13.1	26.17 ± 10.3
$\bar{X}$ % M.U.A.	Acute	(M & F)	35.0 ± 31.8	57.0 ± 31.8
	Chronic	(M & F)	53.3 ± 37.4	93.1 ± 1.3

Values are $\bar{X}$ ± SD

TABLE 7

Maximum Voluntary Contraction and Percent Motor Unit Activation Ratios* for Acute and Chronic Hemiparetic Patients

Variable	N	Acute	Chronic
M.V.C.	21	(n = 10)	(n = 11)
Ratios		.22 ± 26	.62 ± .42
% M.U.A.	20	(n = 9)	(n = 11)
Ratios		.39 ± .39	.66 ± .27

$\bar{x}$ ± SD

* Ratios calculated: $\frac{\text{affected limb}}{\text{unaffected limb}}$

TABLE 8

Mean Maximum Voluntary Contraction and Percent Motor Unit Activation Ratios as a Function of Sex and Time Post Stroke

Variable	N	Sex	Acute	Chronic
M.V.C.	7	F	(4) .30 ± .3	(3) .87 ± .30
Ratios	14	M	(6) .17 ± .24	(8) .51 ± .42
% M.U.A.*	6	F	(3) .72 ± .35	(3) .64 ± .02
ratio	14	M	(6) .22 ± .31	(8) .66 ± .33

$\bar{x}$ ± SD

* Ratios:

$$\frac{\%\ \text{M.U.A. affected limb}}{\%\ \text{M.U.A. unaffected limb}}$$

$$\frac{\text{M.V.C. affected limb}}{\text{M.V.C. unaffected limb}}$$

Correlational analysis indicated a significant difference between the MVC affected limb to unaffected limb ratio and stage of motor recovery of the foot and the leg (Figure 6 and 7). A significant relationship between MVC ratios and age was not found (Figure 8). Subjects examined more than once showed that there was no difference between initial and final testing (Figure 9). This result should be interpreted cautiously due to the small sample size.

When considering subjects under 60 years of age, analysis of variance for maximum voluntary contraction demonstrated a main effect only for limb ($P < 0.01$, Table 9). For these individuals, force production was uninfluenced by time post stroke. Differences between the sexes were not examined in this analysis because of the small sample size.

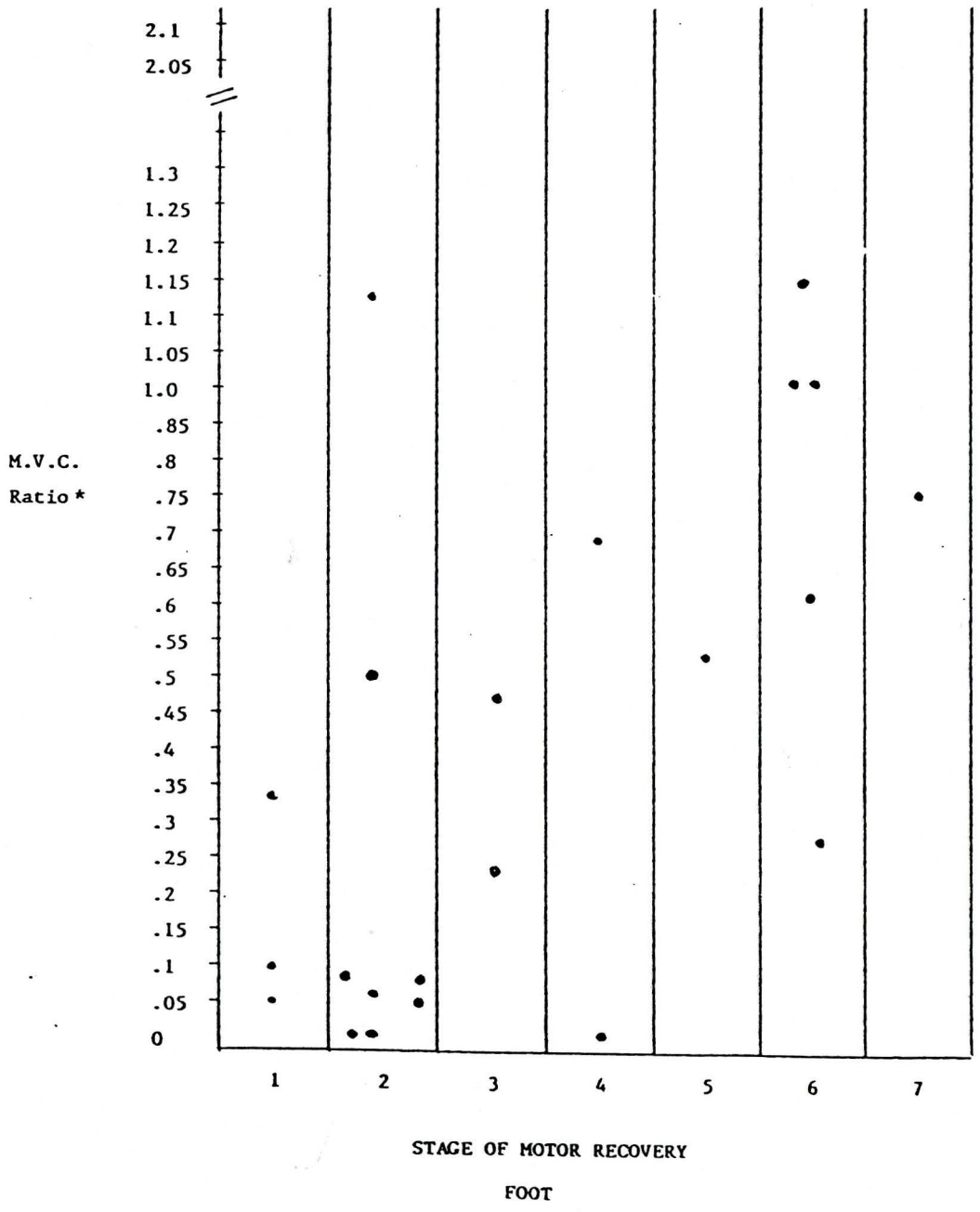
B. MOTOR UNIT ACTIVATION: (M.U.A.)

Two approaches were used to determine motor unit activation. In one instance, the motor unit activation calculation measures were obtained from the first trial only (per cent motor unit activation - % M.U.A.) while in the second instance, the motor unit activation measure was determined from the mean value for three trials (mean percent motor unit activation, $\bar{x}$ % M.U.A.)

Figure 6 - The Relationship Between Stage of Motor Recovery and Impairment of the Foot (N = 22)

The Relationship between stage of motor recovery and MVC Ratios of the Foot showed a significant r (20 df) = .61 ($P > 0.05$)

Figure 6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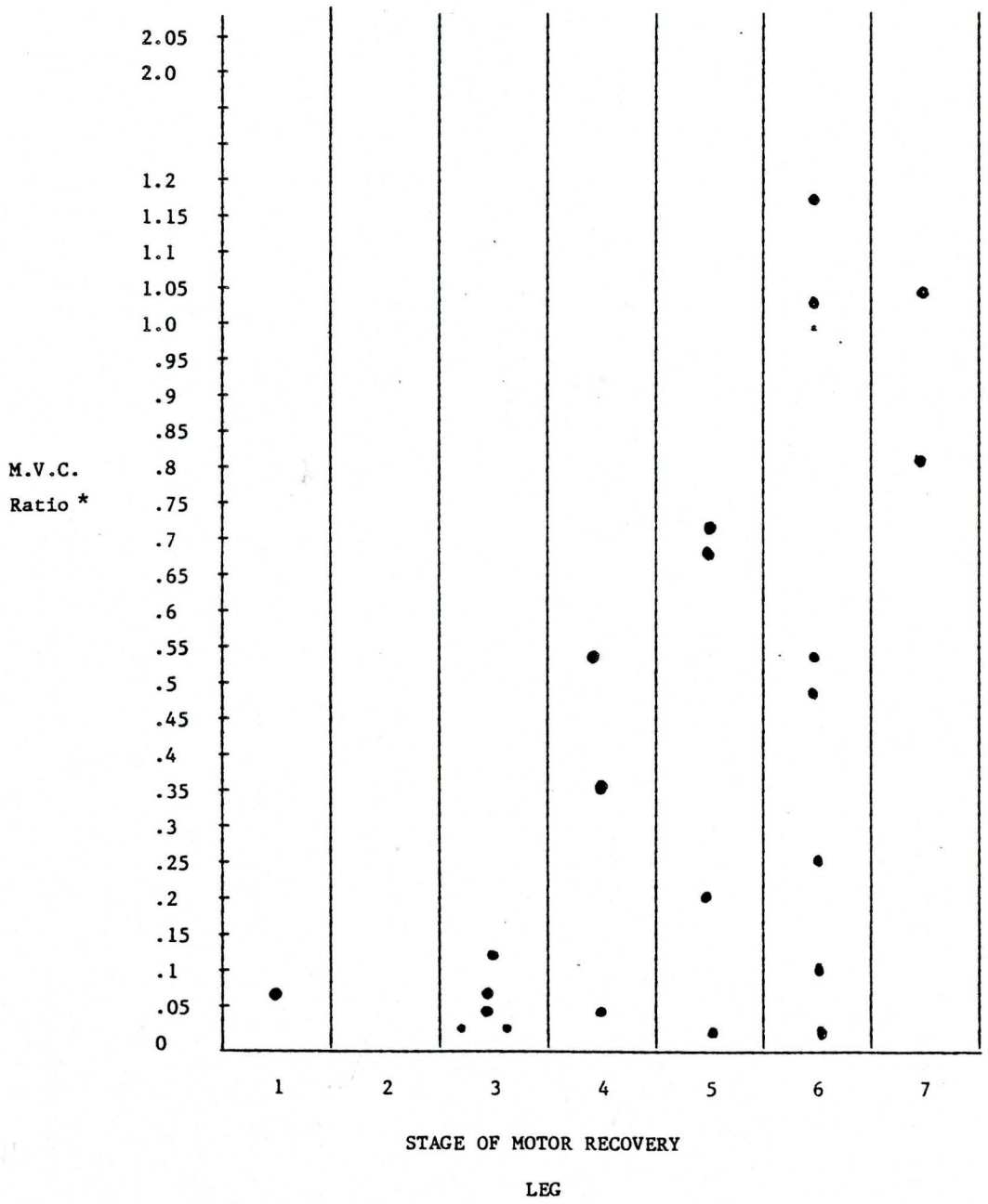
STAGE OF MOTOR RECOVERY
FOOT

Figure 7. The Relationship Between Stage of Motor Recovery and Impairment of the Leg. (N = 22)

The relationship between stage of motor recovery and MVC ratios of the leg was significant $r(20\ df) = .51$ ($P > 0.05$).

Figure 7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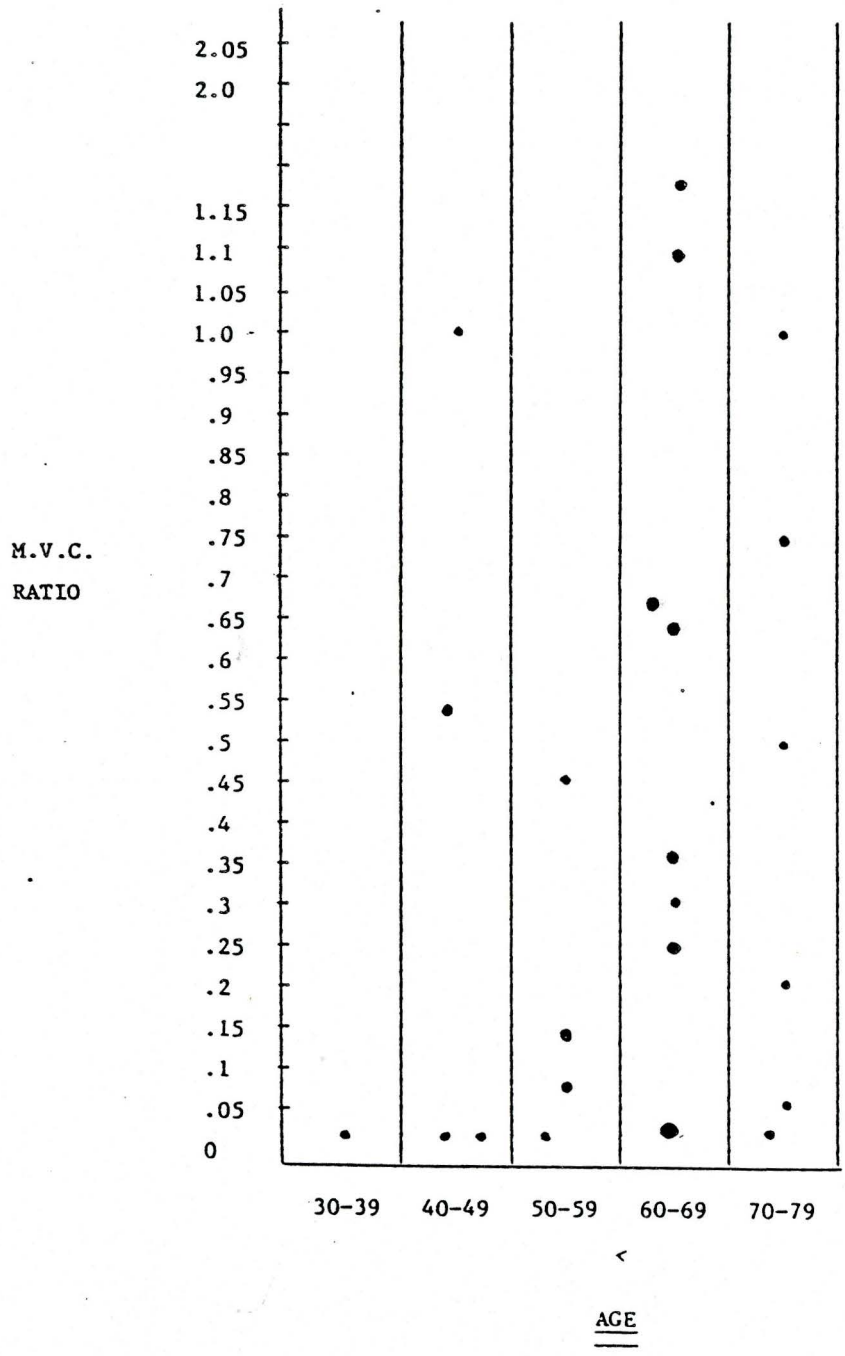
STAGE OF MOTOR RECOVERY
LEG

Figure 8. The Interaction Between Maximum Voluntary Contraction Ratios and Age (N = 22)

The relationship between maximum voluntary contraction ratios and age was not significant $r(20\ df) = .19$ ($P > 0.05$).

Figure 8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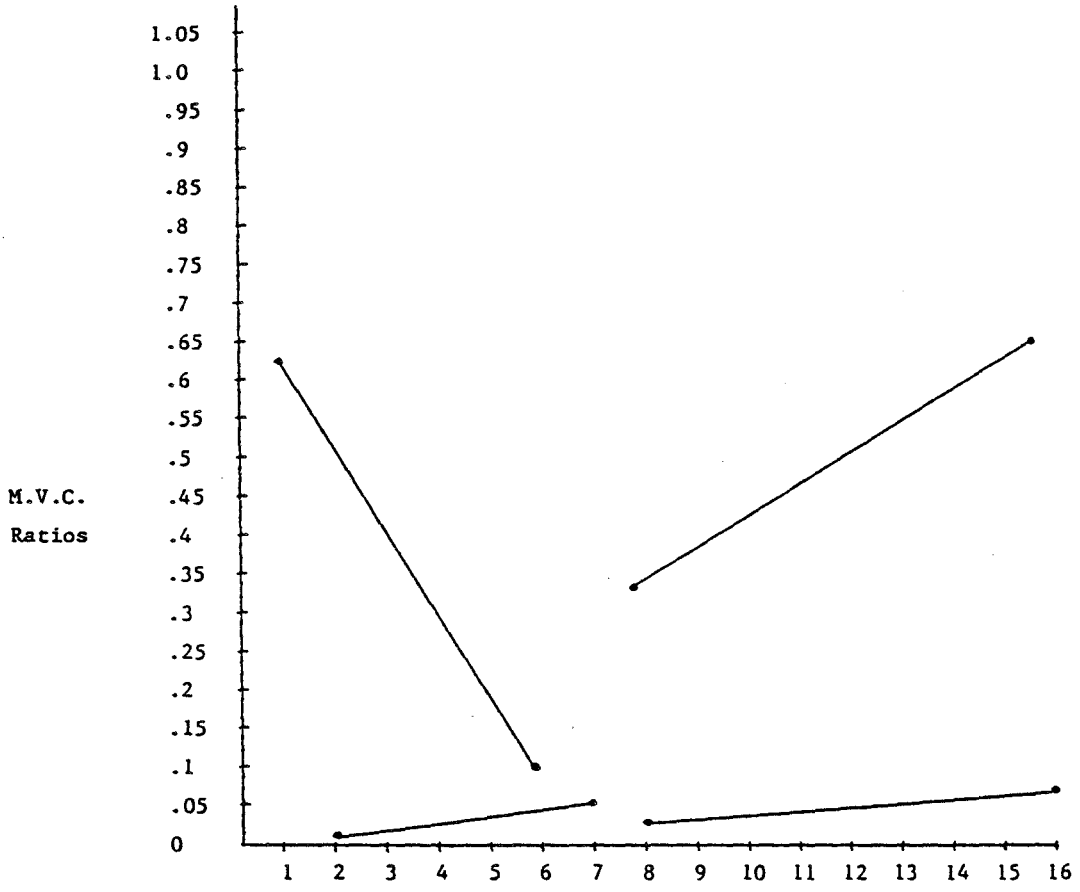
M.V.C.
RATIO
30-39
40-49
50-59
60-69
70-79
AGE

Figure 9. Maximum Voluntary Contraction Ratios and Time Post Stroke in Subjects Tested More Than Once (N = 4). The time period is from 1 week post stroke to 16 weeks post stroke. No effect was found in subjects tested more than once, and 3 subjects showed improvement while 1 subject regressed with time.

Figure 9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M.V.C.
Ratios
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
TIME POST STROKE
(weeks)

TABLE 9

Mean Maximum Voluntary Contraction and Percent Motor Unit Activation for Subjects Under 60 Years of Age as a Function of Limb

Variable	N	Limb Affected	Unaffected
M.V.C. (N.m)	9	7.1 ± 9.6	33.3 ± 17.0
% M.U.A.	9	29.9 ± 30.4	92.4 ± 2.4

$\bar{x}$ ± SD

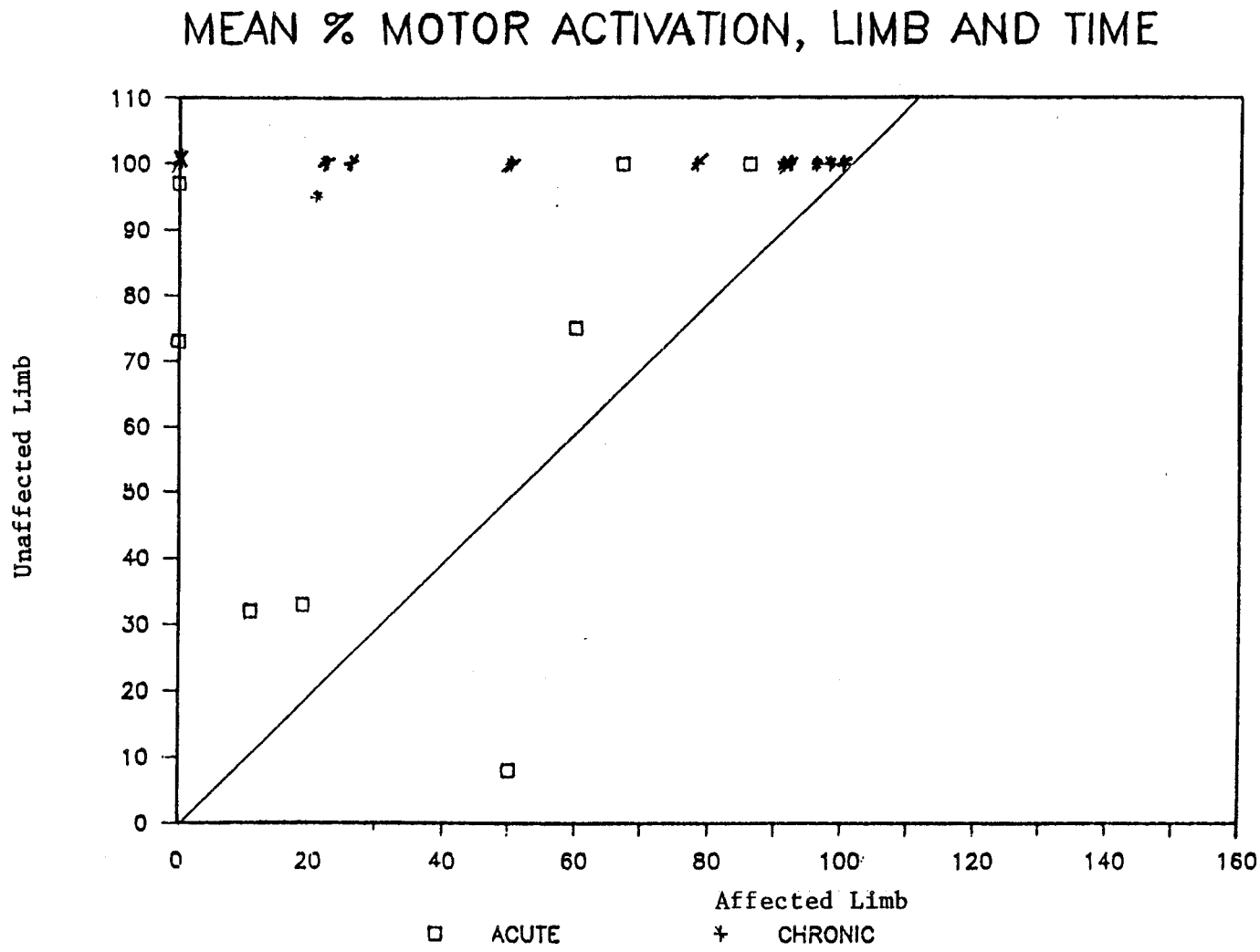
The mean percent motor unit activation data analysis (ANOVA) showed significant main effects for limb ($P < 0.01$) and time post stroke ($P < 0.05$). In accordance with maximum voluntary contraction results, the affected limb revealed significantly less motor unit activation than did the control limb (Table 3, Figure 10). Further, chronic patients activated more units than the acute patients (Tables 4 and 6). This pattern of results was consistent for both genders (Table 5).

Analysis of variance of the percent motor unit activation data showed that in addition to main effects for limb ($P < 0.01$) (Table 3) and time post stroke ($P < 0.05$) (Table 4), several other results are of interest. These findings include a sex by time post stroke interaction ($P < 0.01$) (Table 8) as well as a three way interaction involving gender, time post stroke and limb ($P < 0.05$) (Table 10). Table 10 shows that acute females activated a greater percentage of their motor units in the affected limb than did the males. In chronic cases, the reverse situation occurred (Tables 4 and 5). As previously discussed, acute male subjects were generally older and more severely disabled than females while the chronic females were more disabled than the males.

Data analysis (ANOVA) in which impairment was expressed as a ratio of the values for the affected and unaffected limb (percent motor unit activation ratio - % M.U.A. ratio)

Figure 10: Mean Percent Motor Unit Activation ($\bar{X}$ % M.U.A.), As a Function of Limb and Time.

The squares indicate acute patients and the * depict chronic patients.


MEAN % MOTOR ACTIVATION, LIMB AND TIME
Unaffected Limb
110
100
90
80
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60
50
40
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20
40
60
80
100
120
140
160
Affected Limb
ACUTE
CHRONIC

TABLE 10

Mean Percent Motor Activation as a Function of Sex, Time Post Stroke and Limb in Hemiparetic Patients

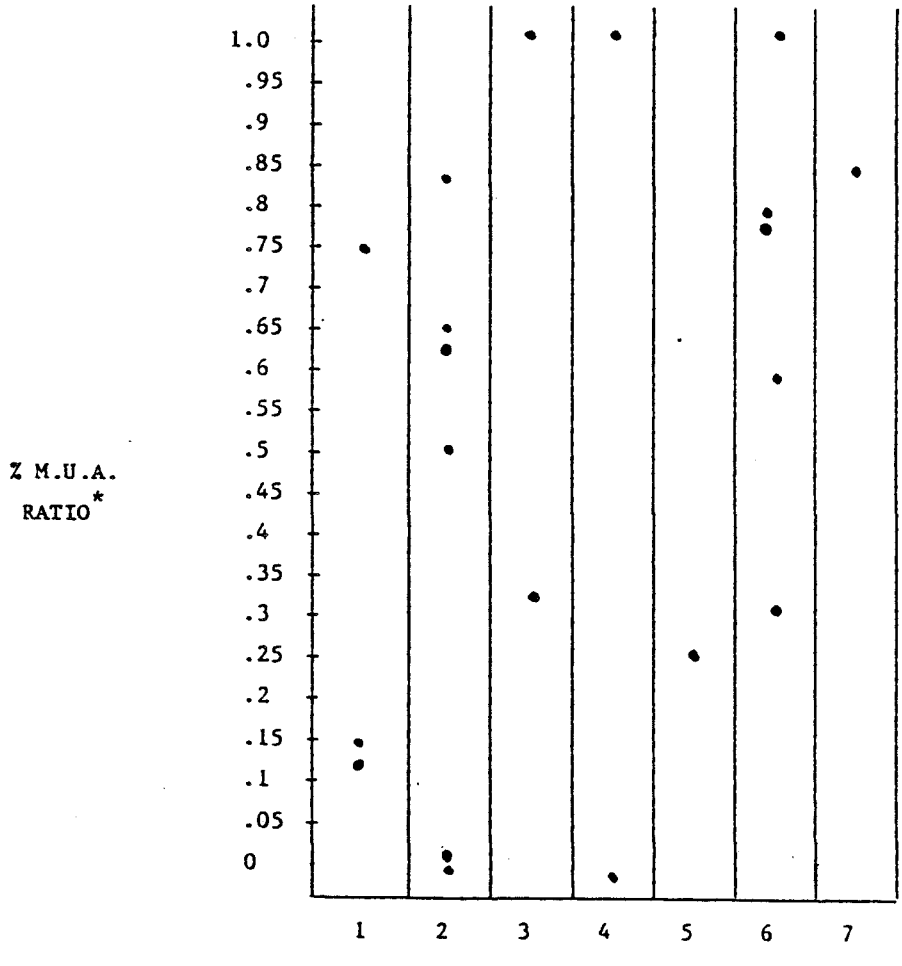
Gender	Time	Limb	
		Affected $\bar{x}$ % M.U.A.	Unaffected $\bar{x}$ % M.U.A.
Males	Acute	14.2 ± 2.3	79.5 ± 25
	Chronic	74 ± 28	100 ± 0
Females	Acute	69.8 ± 29	74 ± 28
	Chronic	45.8 ± 41	90 ± 20

$\bar{x}$ ± SD

Figure 11: Stage of Motor Recovery as a Function of Impairment of the Foot. (N = 20)

A significant correlation existed between stage of motor recovery of the foot and percent motor unit activation ratio $r(18\ df) = .37$ ($P > 0.05$)

Figure 11

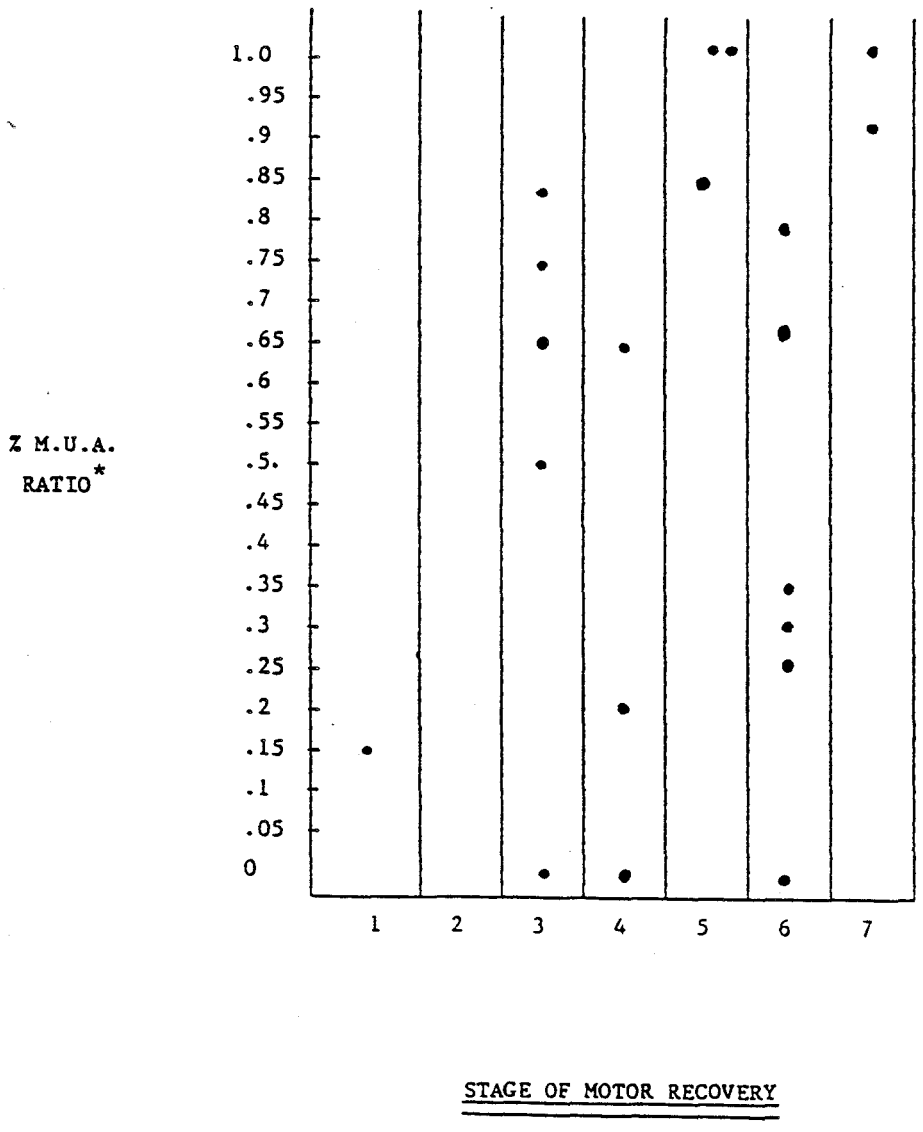


Foot

Figure 12: Stage of Motor Recovery as a Function of Impairment of the Leg (N = 20).

No discernable pattern of results was observed between stage of motor recovery of the leg and percent motor unit activation (first trial) $r(18\ df) = .27$ ($P > 0.05$)

Figure 12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STAGE OF MOTOR RECOVERY

revealed comparable, but not statistically reliable, results (Table 7, 8). Correlational analysis of percent motor unit activation ratios revealed that impairment was related to stage of motor recovery of the foot (Figure 11) but showed no significant correlation with the leg (Figure 12). There was also no relationship between age and impairment (Figure 13). A T-test conducted on M.U.A. ratios for subjects examined more than once revealed no difference between test situation one and test situation two (Figure 14).

For subjects under 60 years of age, a main effect (ANOVA) for limb ($P < 0.01$) was demonstrated. The subjects in the subgroup were able to activate fewer motoneurons with the affected limb (29.9 $\pm$ 30.4%) than with the unaffected limb (92.4 $\pm$ 2.4%). These findings corresponded with mean MVC results (Table 11) in which the affected limb produced 7.1 $\pm$ 9.6 N.m of force and the unaffected limb produced 33.3 $\pm$ 17.0 N.m. of force.

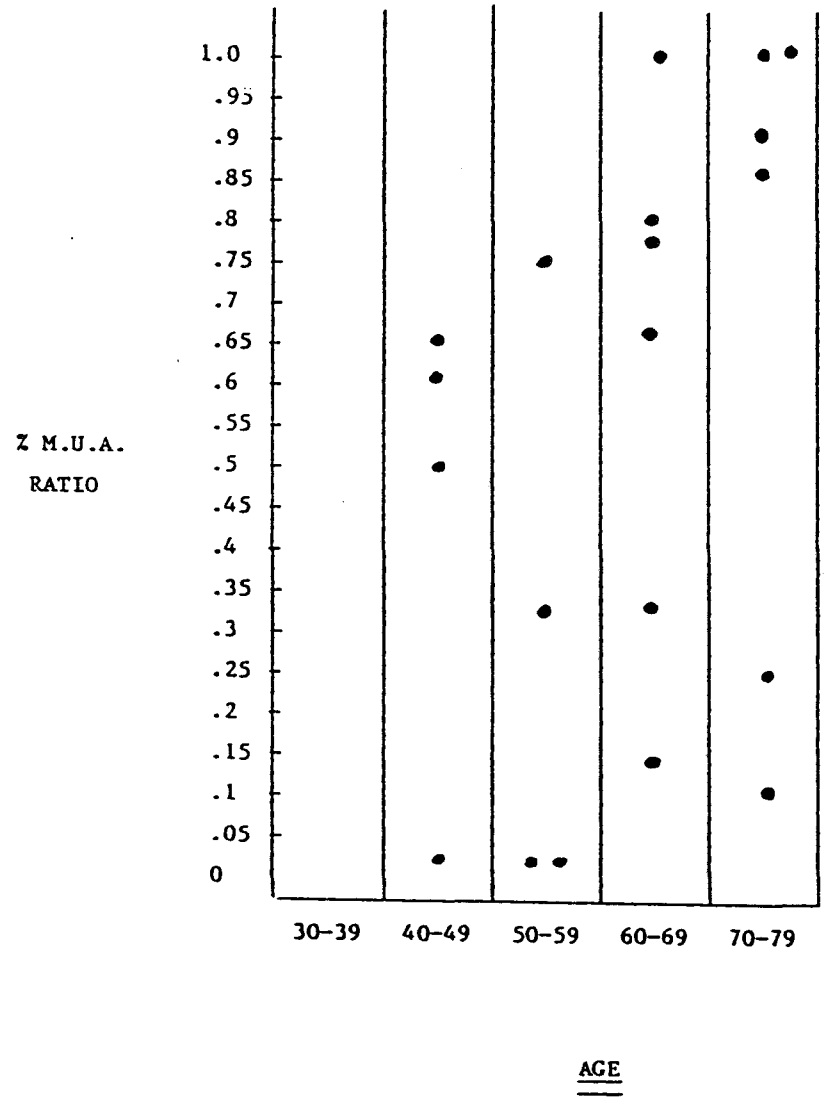
C. MOTOR NERVE CONDUCTION VELOCITIES

Motor nerve conduction velocities recorded above the knee, revealed an effect (ANOVA) for limb that approached conventional levels of significance ($P = .06$). Conduction velocities were slower in the affected limb (41 $\pm$ 17 ms) than the control limb (53.5 $\pm$ 23 ms) (Table 12). This pattern of results did not vary with a subject's sex or time post stroke.

Figure 13: The Relationship Between Percent Motor Unit Activation Ratios and Age. (N = 20)

The correlation was not significant between age and percent motor unit activation $r(18\ df) = .26$ ($P > 0.05$)

Figure 13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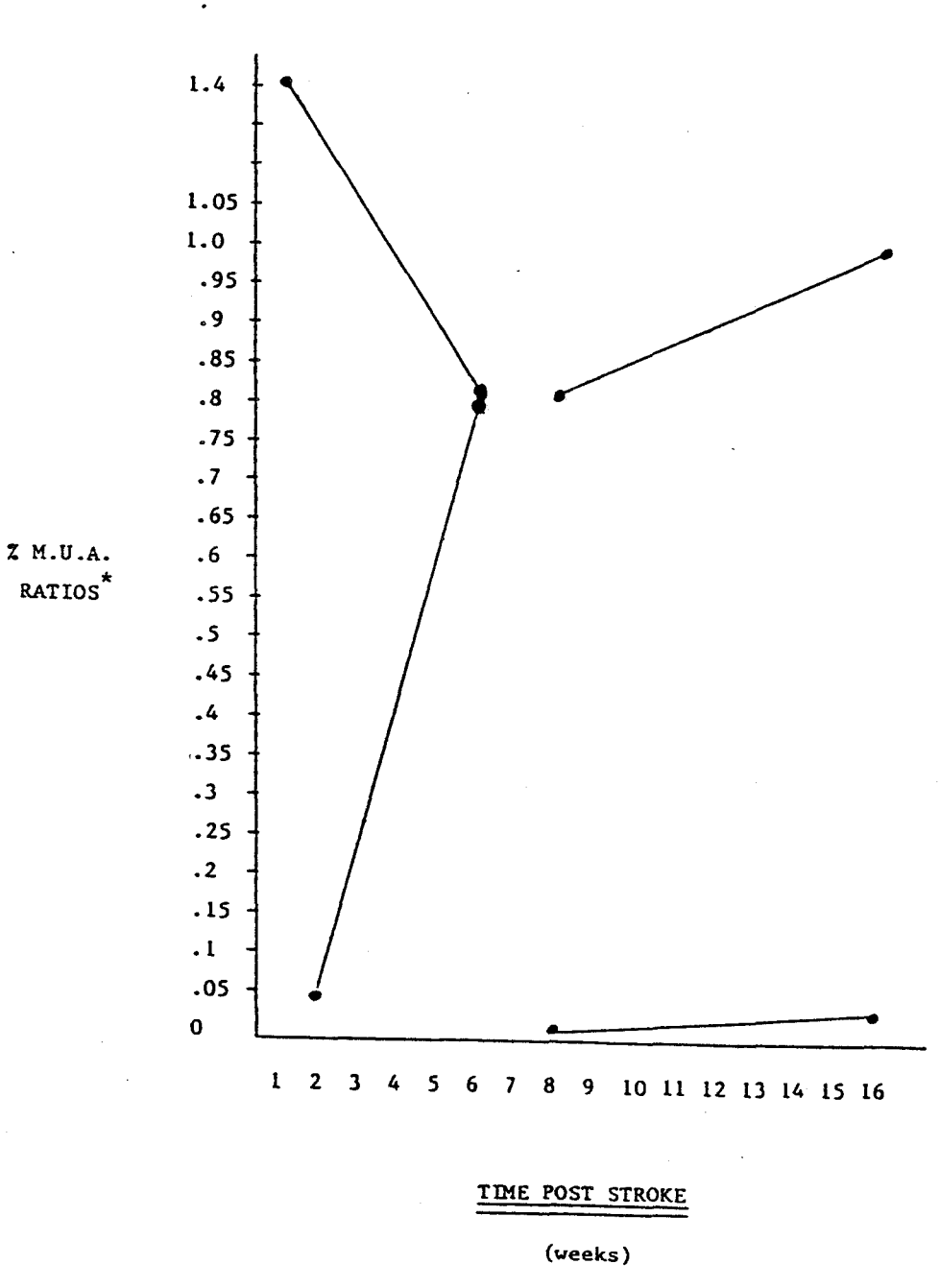
30-39
40-49
50-59
60-69
70-79
% M.U.A. RATIO
AGE

Figure 14: Percent Motor Unit Activation Ratios and Time Post Stroke in Subjects Tested More Than Once (N = 4).

Three subjects showed some improvement in degree of impairment with time. One subject who was severely confused showed a decrease in motor unit activation capabilities.

Figure 14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% M.U.A. RATIOS*
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
TIME POST STROKE
(weeks)

TABLE 11

Mean Motor Unit Counts, M-Wave Amplitudes, Maximum Voluntary Contraction and Percent Motor Unit Activation in Hemiparetic Subjects Under 60 Years of Age as a Function of Limb

Variable	N	Affected Limb	Unaffected Limb
Motor Unit Counts	9	73.8 ± 52	130 ± 61
E.D.B. M-wave amplitude (mV)	9	4.0 ± 2.3	5.7 ± 2.2
Maximum Voluntary Contraction (N.m)	9	7.1 ± 9.6	33.3 ± 17.0
% Motor Unit Activation	9	29.9 ± 30.4	92.4 ± 2.4

$\bar{x}$ ± SD

TABLE 12

Comparison of Peroneal Nerve Conduction Velocities Between the Total Sample of Hemiparetic Subjects and Subjects Under 60 Years of Age.*

Subjects		Mean Peroneal Nerve Conduction Velocities Affected Limb ms	Unaffected Limb ms
Total Sample N = 22	A/K	41 ± 17	53.5 ± 23
	B/K	43 ± 10	46.0 ± 11
Sub-Group* N = 9	A/K	43.4 ± 22	58.3 ± 30
	B/K	44.4 ± 13	46.6 ± 7

$\bar{x}$ ± SD

A/K - measurements taken above the knee.

B/K - measurements taken at neck of fibula.

While mean velocities for the affected and unaffected limb taken below the knee followed the same pattern, the analysis of below the knee velocities revealed no significant effects for limb, sex or time post stroke ($P > .10$, Table 12). For both the above knee and below knee velocities, there were no differences between initial and final testing in subjects that were tested more than once.

The above pattern of results for peroneal nerve conduction velocities was consistent for subjects under and over 60 years of age (Table 12).

D. TERMINAL LATENCIES

In the total sample, no difference was observed between limbs for terminal latencies and values were within the normal range for the peroneal nerve supplying both the EDB and tibialis anterior muscles. Mean values for EDB were 5.1 $\pm$ 2.0 ms affected limb vs 4.8 $\pm$ 2.0 ms unaffected limb (Table 13) and those for tibialis anterior were 3.5 $\pm$ 9 ms vs. 3.4 $\pm$ 0.8 ms (hemiparetic vs. non hemiparetic limb).

Peroneal nerve terminal latencies were comparable for subjects under and over 60 years of age (Table 14). Mean values for the affected limb were 5.6 $\pm$ 2.7 ms and 4.2 $\pm$ 6 ms for the unaffected limb.

TABLE 13

Mean Motor Unit Counts, EDB M-Wave Amplitudes and Latencies as a Function of Limb for the Total Sample

Variable	N	Affected Limb	Unaffected Limb
Motor Unit Counts (EDB)	21	98.3 ± 83	104.8 ± 58
EDB M-Wave amplitude (mV)	21	4.2 ± 2.4	5.1 ± 3.0
EDB M-Wave latencies (ms)	21	5.1 ± 2.0	4.8 ± 2.0

$\bar{x}$ ± SD

TABLE 14

Mean Motor Unit Counts, M-wave Amplitudes and Terminal Latencies as a Function of Limb for Subjects Under 60 Years of Age

Variable	Total Sample (N = 21)		SubGroup (N = 9)	
	Affected Limb	Unaffected Limb	Affected Limb	Unaffected Limb
Motor Unit Counts (EDB)	98.3 ± 83	104.8 ± 58	73.8 ± 52	130.0 ± 61
M-Wave amplitudes (EDB) mV	4.2 ± 2.4	5.1 ± 3.0	4.0 ± 2.3	5.7 ± 2.2*
Peroneal Nerve Terminal Latencies (EDB) ms	5.1 ± 2.0	4.8 ± 2.0	5.6 ± 2.7	4.2 ± .67

$\bar{x}$ ± SD

E. MOTOR UNIT COUNTS

The number of motor units within the peroneal innervated extensor digitorum brevis muscle was estimated. The mean values for the control limb were not significantly higher than the affected limb (Table 13). No effect was seen for time poststroke and sex of the subjects and no significant differences were found between trials for subjects tested more than once.

When the data for subjects under 60 years of age were analyzed (ANOVA), motor unit counts for the EDB muscle of the affected limb tended to be lower than those of the unaffected limb (73.8 ± 52 and 130 ± 61, $P = .06$). The results for these subjects, when compared with the total sample, showed that the subgroup had lower mean motor unit counts in the affected limb than the total sample and higher mean motor unit counts than the total sample, in the unaffected limb (Table 14). When the subgroup of subjects under 60 years of age in this study was compared with the subjects under 60 years of age examined by McComas et al. (1973), it is apparent that differences between the results of this study and that of McComas and colleaques are greater for the unaffected ($P < 0.05$) than the affected limb (n.s) (Table 15).

The differences noted between the two studies may be due to the fact that the subjects of this thesis study who

TABLE 15

Mean Motor Unit Counts, and M-Wave Amplitudes, For Subjects Under 60 Years of Age, McComas et al. (1973) and Values for Healthy Subjects.

Variable	Subgroup (N = 9)		McComas et al (1973) (N = 23)		Healthy* Subjects
	Affected Limb	Unaffected Limb	Affected Limb	Unaffected Limb	Normal
Motor Unit Counts (EDB)	73.8 ± 52	130 ± 61	93.7 ± 8.4	217 ± 79	210 ± 65
M-Wave Amplitudes EDB (mV)	4.0 ± 2.3	5.7 ± 2.2	4.42 ± 2.52	6.44 ± 2.44	3-5

*Goodgold J., Eberstein A. (1983) Electro Diagnosis of Neuromuscular Disease. 3rd Ed. Baltimore: Williams and Williams. 79-81

were under 60 years of age were more severely disabled than the subjects under 60 years in the McComas study. Further, motor unit counts in the unaffected limb of the McComas study were actually higher than normal values while this thesis study seems to indicate that the "normal" limb may also have been affected by the stroke. M-wave amplitudes in the thesis study are consistent with motor unit count values for both the total sample and the subgroup. The question of possible involvement of the affected limb would be resolved by analysis of age comparable healthy subjects.

F. ISOMETRIC TWITCH CHARACTERISTICS

No significant differences were found between the limbs for any of the isometric twitch characteristics measured from the resting tibialis anterior muscle for either the total sample or the sub-group (Table 16). Values are all in the normal range for age comparable healthy adults (Vandervoort and McComas 1986). Significant differences in twitch torque were found between males and females, corresponding to differences in MVC force production. Males had twitch torque values of 2.9 $\pm$ 1.6 N.m. compared with females' values of 1.3 $\pm$ 0.6 N.m. ($P. < 0.05$). No interaction was seen between limb, sex and time post stroke. Twitch torque was not potentiated between trials.

TABLE 16

Isometric Twitch Characteristics for the Resting Tibialis Anterior Muscle For the Total Sample of Hemiplegic Patients and the Subgroup of Subjects Under 60 Years of Age

Variable	TOTAL SAMPLE (N = 21)		SUBGROUP (N = 9)	
	Affected Limb	Unaffected Limb	Affected Limb	Unaffected Limb
Twitch Torque (N.m)	2.3 ± 1.6	2.4 ± 1.5	2.2 ± 1.9	2.8 ± 1.7
Contraction Time (ms)	108 ± 33	106 ± 35	90 ± 42	94 ± 20
1/2 Relaxation Time (ms)	119.3 ± 41	114 ± 32	109 ± 59	111 ± 37

G. M-WAVE AMPLITUDES

Mean M-wave amplitudes for the extensor digitorum brevis muscle of the total sample, were not significantly different between limbs (4.2 $\pm$ 2.4 mV. affected limb vs 5.10 $\pm$ 3.0 mV unaffected limb) and were within the normal range for age comparable healthy subjects (McComas et al. 1973). These results are consistent with motor unit count values which also show no difference between limbs.

The M-wave amplitudes recorded from EDB for the total sample corresponded with those of the subgroup and those of McComas et al. 1973) (Table 14) In subjects under 60 years of age there was a significant difference ($P < 0.05$) in M-wave amplitudes between limbs for the EDB muscles (Table 14). These results were consistent with motor unit count results for the subgroup which approached significance.

Measures of excitable muscle mass for the tibialis anterior muscle in the total sample showed no difference between limbs (7.7 $\pm$ 2.0 mV. and 8.1 $\pm$ 2.0 mV, affected vs unaffected limb). These findings were inconsistent with maximum voluntary contraction values which indicated marked depression of MVC in the affected limb.

V. DISCUSSION

A) FORCE PRODUCTION

All acute and chronic subjects with stroke syndrome produced less force with the dorsiflexors of the affected limb than with those of the unaffected limb. Mean values for the total sample showed that force production of the affected limb was 38% of that produced by the unaffected limb. In the subgroup sample of subjects under 60 years of age, the affected limb force production was 21% of that generated by the unaffected limb. Mean percent motor unit activation results substantiated MVC results. Mean percent motor unit activation of the affected limb of the total sample was 58% of that determined for the unaffected limb. In subjects under 60 years of age, the hemiparetic limb had 32% of the activation of the non hemiparetic limb. A decrease in activation of 42% and 68% reduced force production by 62% and 79% respectively in the hemiparetic limb. A linear relationship cannot be assumed between muscle force production and motor unit activation because force of muscle contraction depends also on the muscle's contractile ability. Because twitch torque was the same bilaterally, it can be assumed that the force production deficit is related to the ability to activate motoneurons. Subjects were not separated into groups based on whether the muscle was flaccid/atrophic or spastic and it is impossible to determine the effects of motor unit activity on the force

production-motor unit activation seen in this study. If spatial summation of surviving cortical neurons is deficient due to the UMNL and surviving cortical neurons are compensating by firing more rapidly in order to raise the excitability of the AHC to fire, then decreased force production-motor unit activation may be due to the possibility that increased temporal summation is never enough (Landau 1980). Even if threshold were reached intermittently, such inconsistency would not only impede fine adjustment of motor control but would also be reflected in diminished maximum force output. If Landau's postulation is extended, it may be that not only is spatial and temporal summation of cortical neurons deficient but perhaps the anterior horn cell is also dysfunctional. Lance (1980) suggests that this indeed may be the case, and that muscle spasticity may be due to dysfunctional anterior horn cells which respond excessively to normal afferent input from muscle sensory receptors and conversely, flaccidity could be due to a decreased response of the AHC. Further, motor units are not homogeneous in size and it is conceivable that a few fast twitch units could produce more force than the same number of slow twitch units which supplied fewer muscle fibres in the same muscle. Decreases in force production might be related to which AHC in the motoneuron pool were dysfunctional as well as what proportion of the motoneuron pool was affected. In vascular hemiplegia, slowing of all

motor units has been shown as well as the appearance of a slow fatigable motor unit (Young and Mayer 1982). In addition, evidence in the literature (Ter Haar Romeny et al. 1984) shows that the medial aspect of biceps brachialis muscle is responsible for most of the force produced by that muscle in flexion. It becomes clear that future assessment of force production and motor unit activation in stroke must attempt to delineate the effects of muscle flaccidity, spasticity and specific anatomic characteristics (Ter Haar Romeny et al. 1984) of individual muscles.

Acute males in the present study had a greater deficit of voluntary strength and motor unit activation in the affected limb than the females. Clinical assessment (Brünnstrom) indicated that males were more severely disabled and older than females. In contrast, among the chronic patients, females suffered a greater deficit than the males.

When comparing results of MVC production and percent motor unit activation between the total sample and the sub-group, there was not a statistically significant difference for age. Neuromuscular dysfunction expressed as a ratio of MVC of the hemiparetic limb/MVC produced by the non-hemiplegic limb also showed no significant correlation for age. The values produced by older subjects seemed better

than those of younger subjects, but this result may be due to degree of disability and the extent of the lesion which was more severe in younger subjects. In addition, younger subjects had 10% greater activation on the unaffected side than did older patients. While normal motoneuron loss begins in the seventh decade (McComas 1977), Vandervoort and McComas (1986) report that the elderly were able to fully activate the motor units they had left. The loss of motor unit activation in the unaffected limb seen in this study may be due to previous non clinically detectable strokes in the older population.

Degree of impairment showed no correlation with time post-stroke (acute or chronic stage). However, the time classification of the study did not parallel traditional classification systems (Licht 1965). In the work presented, the acute stage was from onset to one year post insult. Any "real" change in dysfunction may occur within the time frame of the acute stage and would require longitudinal investigations involving a much larger sample size. This point is further substantiated by the observation that no significant correlations were observed between stage of motor recovery (Brünnstrom 1971) of the foot and leg and impairment (MVC ratios) indicating that motor function improved with time.

B) MOTOR UNIT ACTIVATION

Motor unit activation is viewed not only in relationship to the muscle's ability to produce force, but also as an indicator of motoneuron integrity. Of the total sample, subjects achieved only 58% of the motor unit activation of the non hemiparetic limb with the hemiparetic limb, while in the subgroups, subjects showed that they were able to activate 33% of the motor units of the unaffected limb, with the affected limb. These results indicate that a prime source of neuromuscular dysfunction in stroke is the inability to activate motoneurons. Because mean percent motor unit activation was not significantly correlated with time post stroke, this study offers no support to theories of motor neuron excitability (MNE) which suggests that MNE increased as subjects became more chronic (Garcia-Mullin and Mayer 1972). The findings of this thesis experiment do support the work of Sica, McComas and Upton (1971) who demonstrated impaired relex potentiation in the affected limb in stroke syndrome. The advantage of this present work is that motor unit activation could be quantified.

The observation that MVC and mean percent motor unit activation results are compatible coupled with the fact that the interpolated twitch technique from which the two measures are obtained is an evaluation of the ability of patients to activate motoneurons, shows that some healthy

motoneurons are not recruited voluntarily in stroke syndrome. Together these two observations add support to an UMN-LMN hypothesis, in which the UMN exerts a direct effect on the LMN, because it may be argued that if the motoneurons are healthy then failure to activate them must be due to a defective drive system from the U.M.N. However, an UMN-LMN hypothesis viewed from the perspective of neurotrophic effects cannot be supported by the results of this thesis work. It may be that stroke syndrome produces some abnormality in the soma and dendrites of motoneurons that makes them difficult to activate voluntarily. H-reflex studies (Garcia-Mulin and Mayer 1972) lend support to the above argument because these studies have shown that motoneuron excitability is enhanced with time particularly in chronic spastic cases. These reflex studies do not differentiate between effects produced in the axon and soma. These experiments were not designed to determine whether a correlation exists between MNE determined reflexly and the patients ability to drive motoneurons. The thesis work presented was able only to assess the motor units from the axon to the muscle and the most probable reason for impaired motor unit activation found in this experimental work appears to be due to a defective drive from the UMN.

C. MOTOR NERVE CONDUCTION VELOCITIES

The results of peroneal motor NCV are inconclusive. Peroneal motor NCV were 23% slower in the hemiparetic limb than in the non hemiparetic limb when measurements were taken above the knee and 6% slower when measured below the knee. Because a three-way ANOVA applied to the data above the knee, just failed to indicate significance, it may be argued that some LMN deficit in the peripheral nerve may be present in stroke. However, this argument is weakened because some patients were diabetic, and had previous histories of transient ischemic attacks (T.I.A.'s). Further, the study limitations were that limb temperature could not be controlled through out the experiment and decreased NCV has been shown to occur with a decrease in temperature (LeQuesne 1971). Also, the techniques used measured only the fastest conducting nerve fibres while change may occur in the slow fibres (Namba et al. 1973). Differences in NCV between limbs were not significant when measures were taken below the knee and those taken above the knee are questionable because of the short distances measured between stimulation points. On the other hand, other test measures (see motor unit counts) show that some subjects may have had involvement of the non hemiplegic limb which was not visible clinically and as a result, it cannot be concluded that there are no LMN deficits in the peripheral nerve in stroke syndrome. Future study should

include an age comparable control group and account for the above discussed variables.

Another important finding is that terminal motor nerve latencies in the total sample and the subgroup were also longer in the affected limb than in the non-affected limb, but did not show statistical significance. The above observations were in agreement with the work of McComas et al. (1973) who found significant increases in terminal latencies in the affected limb suggestive of a dying back process of the peripheral nerve. In comparing the present results with those of McComas et al. (1971) it must be noted that only the sub group was comparable and the sample size was very small (n = 9). McComas et al. (1973), unlike the present study, did not examine subjects who were in the very early days post stroke. The results of the NCV studies of this thesis neither confirm nor disprove an UMN-LMN hypothesis.

D). MOTOR UNIT COUNTS

Motor unit counts were not significantly different between limbs for the total sample. In the sub group of subjects under 60 years of age, counts on the affected side were about 1/2 the value of the unaffected side. This observation failed to show significance with a confidence level of $P. < 0.05$, $F = 5.05$. The critical F value for a confidence level of $P. < 0.05$ is $F = 5.59$ and implies that

significance might occur with a larger sample size. Motor unit counts were also low (as compared with age comparable normal values) for the unaffected limb of both groups indicating the possibility of bilateral cerebral hemisphere involvement which had not produced clinical signs.

Results of motor unit estimations of this thesis do not conclusively support a theory of transynaptic motoneuron degeneration proposed by McComas et al. (1973) who found reduced motor unit counts in the affected limb of acute stroke subjects under 60 years of age, but do indicate a trend which requires further study. No support for the theory was evident for subjects over the age of 60 years.

E. M-WAVE POTENTIAL AMPLITUDES

The mean evoked potential amplitude (M-wave) of the peroneal nerve supplying the EDB muscle was significantly lower only in the hemiparetic limb of the sub group. The tibialis anterior muscle showed no difference between limbs for both the total sample and the subgroup and values were within ranges established for healthy subjects (Vandervoort 1986). Causes of decreased M-wave amplitudes for EDB in the subgroup are unclear. Depressed M-wave potential amplitudes from EDB in the affected leg of hemiplegic patients under 60 years of age are compatible with decreased motor unit counts in the same limb. Because some of the subjects in the sub group were as long as four years post stroke, it might be

suggested that some motoneurons in stroke syndrome became permanently dysfunctional. Against this argument is the fact that data analysis showed no effect for time post stroke. M-wave amplitudes of the total sample showed no difference between the limbs and this observation coupled with the finding of almost significantly different decreased motor unit counts in the affected limb leads to the speculation that collateral sprouting of denervated muscle fibres may have already occurred at the time of testing. This supposition would be stronger if twitch torque of EDB were found to be within normal ranges. The seemingly different results found in tibialis anterior may reflect a faster rate of recovery in this muscle because tibialis anterior is part of the flexor synergy and receives considerable reflex input from the spinal cord which might hasten the process of collateral sprouting to denervated muscle fibres. Brown, Holland, and Hopkins (1981) report the action of stimulation on the rate and extent of nerve sprouting in partly denervated muscle in which stimulation of the spinal cord and sciatic nerve roots increased the rate of nodal sprouting in the soleus and gastrocnemius muscles of rats. Normal twitch torque and M-wave amplitudes values seen in tibialis anterior support a speculation of re-innervation or no loss of innervation initially, which should be confirmed by estimation of motor unit counts in the tibialis anterior muscle.

F. CONTRACTILE PROPERTIES

Twitch torque, contraction time and 1/2 relaxation time showed no significant difference between limbs for either group and substantiated the work of Ismail and Ranatunga (1981) but is not in accordance with McComas et al. (1973) who found decreased twitch torque and prolonged contraction times in the affected limb of their subjects. The latter study also reported no difference between the limbs for 1/2 relaxation time. In the present study, difficulty in measuring 1/2 relaxation time may have been due to reflex activity triggered by the applied electrical impulse used to produce the twitch. It may also have occurred because of sponataneous reflex activity which might be occurring in stroke syndrome. Although mean values for the above measures were consistently decreased on the hemiparetic side, standard deviations were very great. It is concluded that skeletal muscle fibre integrity is maintained in stroke syndrome. It must be pointed out that the whole muscle study presented combined subjects with spastic and flaccid muscles and was not designed to detect possible compensatory muscle fibre type changes which might occur in stroke syndrome.

No clear conclusion can be made on the cause of muscle atrophy in stroke syndrome based on the findings of this study. Normal twitch torque and M-wave amplitudes for tibialis anterior muscle for both the total sample and sub-

group provide indirect evidence to suggest that muscle atrophy was not a significant feature in the patient sample. The fact that MVC values did not decrease on repeated testing (in one session), an expectation of a disuse model in which fatigue would play a part, suggests that muscle atrophy in vascular hemiplegia may be due to other causes. Voluntary disuse can not be dismissed as a cause of muscle atrophy in vascular hemiplegia because it may be that the normal contractile properties of the tibialis anterior muscle seen in this work are due, in fact, to spinal reflex activity and that the integrity of the whole motor unit (as demonstrated by normal M-wave amplitudes) may be maintained also by reflex activation. If so, a disuse model would not apply to assessment of tibialis anterior muscle. Evaluation of another muscle group, perhaps the intrinsic muscles of the hand or foot which are not directly involved in synergistic patterning or normally subject to muscle atrophy as is the case for EDB (Roselle and Stevens 1973) would provide useful information about the cause of muscle atrophy in stroke syndrome.

Although data were not significantly different between limbs for motor unit counts for EDB muscle and terminal latencies for both EDB and tibialis anterior muscles, values were consistently lower on the affected side. These observations would also support the contention that further study on the cause of muscle atrophy in vascular hemiplegia is needed.

VI. SUMMARY

This investigation explored the effects of cerebrovascular disease on the evoked contractile properties, and the excitable muscle mass of skeletal muscle to determine whether there was a secondary lower motoneuron lesion in stroke syndrome. Estimates of motor unit counts and peripheral nerve conduction velocities, the interpolated twitch and force of the maximum voluntary contraction of the tibialis anterior muscle were also measured. In addition, the degree of motor unit dysfunction between limbs expressed as a function of motor unit activation and maximal voluntary contraction was measured. A summary of the main effects of a stroke on skeletal muscle follows.

1) Electrophysiological estimates of contraction time, one half relaxation time, twitch torque and M-wave amplitudes suggests that the integrity of the skeletal muscle (Tibialis anterior) is preserved after a stroke and muscle atrophy was not a significant feature in the patient sample.

2) Motoneurons may be "sick" or dysfunctional as indicated by decreased (but not significantly different) motor unit counts, longer terminal latencies of the axon and slowing of nerve conduction velocities. On the other hand, the fact that twitch torque and M-wave amplitudes in tibialis anterior show no difference between limbs and are within the

range of normal argue against a sick motoneuron hypothesis.

3) No evidence was found to support a hypothesis of transynaptic motoneuron degeneration even though motor unit counts just failed to reach significance between limbs in subjects under 60 years of age.

4) Assessment of motor unit dysfunction between limbs expressed as a ratio of maximum voluntary contraction of the affected limb/unaffected limb indicates that chronic cases over 1 year post stroke were functionally better than the acute cases. This finding is interpreted cautiously because of the small sample size and warrants further investigation.

The significance of this experimental work to clinical treatment is that a major neuromuscular problem in stroke syndrome seems to be an inability to fully activate motor units. Both maximum voluntary force and percentage of motor unit activation in tibialis anterior muscle in stroke patients has been quantified. Because skeletal muscle integrity is maintained, physiotherapeutic techniques which view the neuromuscular dysfunction in stroke as being "central" in nature rather than "peripheral" seem to be appropriate. Future study is required to determine the effects of spasticity and flaccidity on skeletal muscle function in vascular hemipleigia. Longitudinal studies are needed to carefully assess whether improved neuromuscular function occurs in hemiplegic patients who are greater than 1 year post stroke. These studies are needed to better

delineate the process of neuromuscular dysfunction in stroke patients so that current physiotherapeutic interventions may be objectively assessed and appropriately modified.

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APPENDIX A

Figure 1

Modified Brunnstrom Assessment of Motor Recovery of the Leg and Foot*

Leg	Foot
Stage 1	
☐ not yet stage 2	☐ not yet stage 2
Stage 2 - (patient in crook lying)	
☐ resistance to passive hip or knee flexion	☐ resistance to passive dorsiflexion or extension of great toe facilitated plantar flexion
☐ facilitated flexion facilitated extension	
Stage 3 - (patient in crook lying)	
☐ abduction: adduction to neutral hip flexion to 90° full extension	☐ dorsiflexion > 1/2 range extension of gr. toe > 1/2 range
	☐ (supine): plantarflexion > 1/2 range
Stage 4 - (patient standing)	
☐ hip flexion to 90° then extension synergy bridging hips to 0° equal weight bearing	☐ (sit): eversion > 1/2 range ☐ inversion ☐ legs crossed: plantar-flexion ☐ then dorsiflexion
- (patient sitting)	
☐ knee flexion beyond 90° heel on floor.	
Stage 5 - (patient in crook lying)	
☐ extension synergy then flexion synergy	☐ legs crossed: toe extension with plantarflexion
- (patient in sitting)	
☐ raise thigh from bed	☐ heel on floor: toe flexion with dorsiflexion
- (patient in standng)	
☐ on strong leg with support: hip extension, knee flexion, foot off floor.	☐ heel on floor: eversion

Stage 6 - (patient sits)

☐ lift foot from floor 3 times/5 seconds.

☐ heel on floor: tap foot 5 times/5 seconds.

- (patient stands)

☐ on strong leg with support: trace a pattern; forward, side, back, return

☐ legs crossed: foot circumduction

☐ abduction beyond neutral with knee extension

☐ knee straight, heel off floor: eversion

Stage 7

Normal ☐ # Stage ☐ Normal ☐ # Stage ☐

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* The patient must be able to complete 2 out of 3 tasks in a particular stage.

APPENDIX B

Table B1. Individual Data for Maximum Voluntary Contraction and Percent Motor Unit Activation and Limb.

Subject #	Initial	Sex	Age (year)	M.V.C. (N.M.) affected limb	M.V.C. (N.M.) unaffected limb	% M.U.A. affected limb	% M.U.A. unaffected limb
1.	J.D.	M	68	0.5	2.0	11	32
2.	D.W.	M	55	0.0	48.0	0	97
3.	D.D.	M	57	3.0	48.0	0	100
4.	C.E.	M	63	1.6	16.0	60	75
5.	M.K.	F	73	0.0	10.0	0	45
6.	G.O.	M	43	0.8	64.0	0	93
7.	V.H.	F	52	9.0	20.0	33	100
8.	J.H.	M	72	19.0	36.0	24	100
9.	G.H.	M	71	0.0	19.0	11	80
10.	K.O.	F	42	25.0	25.0	63	100
11.	J.B.	F	40	12.0	25.0	67	100
12.	C.K.	M	62	44.0	38.0	79	100
13.	V.C.	F	66	12.0	12.0	100	100
14.	A.W.	M	48	0.4	44.0	0	100
15.	K.M.	M	54	5.0	42.0	25	100
16.	V.M.	F	66	0.4	24.0	*	100
17.	D.H.	F	36	0.0	17.0	*	100
18.	W.P.	M	62	5.0	14.5	16	100
19.	G.M.	M	71	28.0	36.0	87	100
20.	H.M.	M	73	6.0	28.0	100	100
21.	C.D.	M	71	26.0	26.0	100	100
22.	G.R.	F	69	20.0	18.0	75	100

* indicates interpolated twitch was > than resting twitch.

Table B2. Individual Data for Resting Dorsiflexor Twitches for the Affected and Unaffected Limbs. Variables are Compound Muscle Action Potential (M-Wave), Contraction Time (CT), One-Half Relaxation Time (1/2 RT) and Twitch Torque (T.T.).

Subject #	Int.	Sex	Age (yr)	Affected Limb M-Wave (Mv)	CT (MS)	Hz RT (MS)	T.T. (N.m.)	Unaffected Limb M-Wave (Mv)	CT (MS)	1/2 R.T. (MS)	T.T. (N.m.)
1.	J.D.	M	68	8.0	92	188	3.0	8.8	100	98	2.7
2.	D.W.	M	55	2.8	N/A	N/A	N/A	11.6	100	95	3.0
3.	D.D.	M	57	6.8	80	100	1.3	6.6	80	120	3.4
4.	C.E.	M	63	9.2	95	100	2.8	8.0	85	110	1.6
5.	M.K.	F	73	9.6	245	90	1.0	7.0	135	170	2.0
6.	G.O.	M	43	5.8	95	100	6.5	7.6	80	120	7.0
7.	V.H.	F	52	8.8	95	160	1.7	8.6	70	57	0.7
8.	J.H.	M	72	7.2	120	130	5.0	7.2	95	150	2.8
9.	G.H.	M	71	8.4	90	90	2.4	8.8	90	110	3.8
10.	K.O.	F	42	8.4	105	210	2.0	8.4	130	190	2.6
11.	J.B.	F	40	9.0	145	130	1.0	9.6	115	100	1.2
12.	C.K.	M	62	8.0	103	107	3.4	7.2	120	105	3.0
13.	V.C.	F	66	8.0	105	160	1.8	9.2	95	165	1.6
14.	A.W.	M	48	9.6	105	110	3.5	6.4	100	100	3.0
15.	K.M.	M	54	4.6	125	70	4.0	6.4	100	80	4.2
16.	V.M.	F	66	6.8	125	160	1.2	9.6	100	150	1.4
17.	D.H.	F	36	8.4	60	85	0.8	6.8	75	120	1.8
18.	W.P.	M	62	8.8	105	90	3.5	8.0	-	-	0.6
19.	G.M.	M	71	6.4	105	145	2.2	7.2	105	130	2.6
20.	H.M.	M	73	8.8	100	70	1.0	7.2	90	110	1.4
21.	C.D.	M	71	7.2	85	60	2.4	6.4	115	95	2.4
22.	G.R.	F	69	7.2	115	100	1.7	8.4	115	100	0.6

Table B3. Individual Data for Twitch Torque (T.T.) and Maximum Voluntary Contraction (MVC) for Three Trials for the Affected Limb.

Subject #	Ini.	Sex	Age (yr)	Trial #1 T.T. (N.m)	Trial #1 M.V.C. (N.m)	Trial #2 T.T. (N.m)	Trial #2 M.V.C. (N.m)	Trial #3 T.T. (N.m)	Trial #3 M.V.C. (N.m)
1.	J.D.*	M	68	2.8	0.5	-	-	-	-
2.	D.W.*	M	55	-	-	-	-	-	-
3.	D.D.*	M	57	2.0	1.0	1.4	3.0	-	-
4.	C.E.*	M	63	8.0	6.3	5.0	1.6	-	-
5.	M.K.*	F	73	1.5	0.0	-	-	-	-
6.	G.O.	M	43	6.0	0.8	-	-	-	-
7.	V.H.	F	52	1.5	9.0	-	-	-	-
8.	J.H.	M	72	2.5	19.0	-	-	-	-
9.	G.H.	M	71	2.5	0.0	1.5	0.0	2.3	0.0
10.	K.O.	F	42	2.0	25.0	3.0	24.0	3.5	23.0
11.	J.B.	F	40	1.8	12.0	1.8	14.0	2.0	15.0
12.	C.K.	M	62	4.8	44.0	5.0	20.0	5.6	20.0
13.	V.C.	F	66	1.8	6.0	4.0	7.0	1.1	9.0
14.	A.W.	M	48	3.0	0.4	3.2	0.4	-	-
15.	K.M.	M	54	3.8	5.0	4.0	1.5	4.0	3.0
16.	V.M.	F	66	1.8	0.4	1.8	3.2	1.8	0.8
17.	D.H.	F	36	0.5	0.0	3.0	0.4	3.0	0.4
18.	W.P.	M	62	1.8	5.0	1.8	2.0	2.0	-
19.	G.M.	M	71	2.4	28.0	3.0	28.0	3.2	28.0
20.	H.M.	M	73	0.8	6.0	1.6	6.0	2.1	6.0
21.	C.D.	M	71	2.4	26.0	3.2	24.0	-	-
22.	G.R.	F	69	1.6	20.0	2.4	18.0	2.8	16.0

* indicates subjects who were unable to perform more than once.

Table B4. Individual Data For Subjects Tested More Than Once. Variables Are Motor Unit Counts (MUC), M-Wave Amplitudes, Twitch Torque (T.T.), Maximum Voluntary Contraction (MVC) and Percent Motor Unit Activation for the Affected Limb.

Subject #	Ini.	Age (yr)	Sex	Initial Test * M.U.C.	M-Wave (Mv)	T.T. (N.m)	M.V.C. (N.m)	Final Test * * M.U.C.	M-Wave (Mv)	T.T. (N.m)	M.V.C. (N.m)
1.	J.D.	68	M	23.0	6.8	-	-	105.0	4.0	3.0	0.5
2.	D.W.	55	M								
3.	D.D.	57	M	28.0	1.6	3.5	1.0	-	1.6	1.3	3.0
4.	C.E.	63	M	383.0	9.2	2.8	6.3	144.0	5.2	0.9	1.6
5.	M.K.	73	F	13.0	0.8	-	-	2.0	0.8	1.8	0.0
9.	G.H.	71	M	105.0	4.4	2.4	0.0	30.0	2.2	2.5	0.0
13.	V.C.	66	F	143.0	6.0	1.8	6.0	200.0	5.6	0.8	12.0

* Time post stroke of subjects for the initial test ranged from 9 days to 3 months.

* * Time post stroke of subjects for the final test ranged from 2 months to 4 months.

Appendix C

F Ratios Reported In The Text

Maximum Voluntary Contraction (Total Sample)

	df	ss	F
Limb	1	2492.8	16.37
Error	18	2740.7	
Limb x Sex	1	709.8	4.66
Error	18	2740.7	
Limb x Time	1	537.6	3.53
Error	18	2740.7	

Maximum Voluntary Contraction (Sub group)

	df	ss	F
Limb	1	3830.56	14.29
Error	7	1875.84	

Maximum Voluntary Contraction (Total Sample)

	df	ss	F
Time	1	.97	8.05
Error	17	2.03	

Mean Percent Motor Activation (Total Sample)

	df	ss	F
Limb	1	10573.03	15.05
Error	16	11240.69	
Time	1	7521.04	6.46
Error	16	18632.69	

Percent Motor Unit Activation (Total Sample)

	df	ss	F
Limb	1	17087.06	33.02
Error	16	8280.23	
Time	1	3149.00	6.55
Error	16	7689.23	
Sex x Time	1	4029.02	8.38
Error	16	7689.23	
Sex x Time x Limb	1	2782.40	5.38
Error	16	8280.23	

Percent Motor Unit Activation (Sub groups)

	df	ss	F
Limb	1	21207.56	69.89
Error	7	2124.06	
Time	1	2269.54	6.94
	7	2288.56	
Limb x Time	1	1795.15	5.92
	7	2124.06	

Motor Nerve Conduction Velocities (Above Knee Total Sample)

	df	ss	F
Limb	1	1345.10	4.10
Error	17	5582.17	

Motor Nerve Conduction Velocities (Below Knee Total Sample)

	df	ss	F
Limb	1	48.06	.384
Error	17	2127.73	

Motor Unit Counts (Sub groups)

	df	ss	F
Limb	1	15708.01	5.05
	7	21783.1	