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Hines, Anne Ewing, Ph.D.

Case Western Reserve University, 1994

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FUNCTIONAL ELECTRICAL STIMULATION FOR HAND OPENING IN
SPASTIC HEMIPLEGIA

by
ANNE EWING HINES

Submitted in partial fulfillment of the requirements
for the degree of Doctor of Philosophy

Thesis Advisor: Dr. Patrick E. Crago

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May, 1994

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GRADUATE STUDIES

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*We also certify that written approval has been obtained for any proprietary material contained therein.

FUNCTIONAL ELECTRICAL STIMULATION FOR HAND OPENING IN SPASTIC HEMIPLEGIA

Abstract

by

ANNE EWING HINES

Spasticity is a motor control disorder common after stroke, and can cause the fingers to involuntarily flex and form a fist. Functional electrical stimulation can provide hand opening in hemiplegics with spastic finger flexor muscles through stimulation of the extensor digitorum communis (EDC) muscle and the ulnar nerve. The ability of electrical stimulation to extend these patient's fingers will help them to acquire objects, which will assist them in the performance of bimanual tasks.

This study also sought to investigate the effects of finger extensor stimulation on the spasticity in finger flexor muscles. Electromyographic (EMG) activity was used to assess the presence of spasticity. Since EMG was recorded in muscles adjacent to stimulated muscles, it was contaminated by stimulus artifacts. An offline digital substitution procedure was developed to remove the stimulus artifacts from the flexor EMGs. Extensor stimulation did not reduce the activity in spastic finger flexor muscles, as hypothesized initially, and as might occur through a reciprocal inhibitory mechanism. In fact, flexor activity generally increased during extensor stimulation, presumably by generation of stretch reflexes. Tests conducted in able bodied subjects also found that extensor stimulation can increase the activity in finger flexor muscles; decreases in activity occurred in only a few trials of a few subjects.

Although extensor stimulation did not reduce the activity in the spastic finger flexor

muscles of hemiplegics, the extension force was sufficient to fully extend the fingers. Even in the presence of strong flexor spasticity, hand opening was achieved.

The influence of voluntary effort on finger extension achieved by stimulation was also studied. Finger extension was generally improved when the subject was relaxed during stimulation, and did not attempt to assist extension with voluntary effort. It was also found that finger extension was greatly diminished in some subjects after they had just voluntarily flexed their fingers. This has implications for the performance of functional tasks, in which subjects could use voluntary flexion to grasp an object and then use stimulation to release the object. Closed loop control may be needed to adjust stimulation levels to achieve finger extension following voluntary flexion.

ACKNOWLEDGEMENTS

I would like to extend my sincere appreciation to my committee members, Dr. Pat Crago, Dr. Hunter Peckham, Dr. John Leigh, and Dr. Cecil Thomas, whose guidance was invaluable. I would especially like to thank my advisor, Dr. Pat Crago, who has guided me through six and a half years of graduate studies, and has imparted so much of his knowledge to me.

Dr. Carl Billian collaborated on this project, and spent many hours recruiting and testing patients and implanting the electrodes. I would like to thank him for the time he spent working with me. Thanks are also extended to Chou-Ching Lin who helped during the lengthy experiments.

My gratitude is extended to Dr. Wendy Wilson who helped me find research subjects, and to all the staff of 5B at MetroHealth Medical Center who taught me about the rehabilitation of stroke patients. Dr. Michael Keith in the Department of Orthopaedics was also instrumental in finding research subjects.

Thanks are also extended to Gregg Chapman for his technical contributions to this project, and to Nancy Caris, for fabricating the percutaneous electrodes. I would like to acknowledge Hasan Fakhri and Hussein Abdallah who wrote the original version of the data collection software. Dr. Clayton Van Doren and members of the Department of Epidemiology provided valuable statistical advice.

I would also like to thank the staff and students of the Rehabilitation Engineering Center for their friendship and encouragement throughout this project.

Finally, I extend my appreciation to all the subjects who participated in this study, including the preliminary phase which is not reported in this thesis. Without the

participation of these subjects, none of this would have been possible.

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CHAPTER 1

INTRODUCTION

This chapter contains background information to acquaint the reader with the motor control disorder spasticity, and other physical impairments common in hemiplegia. Methods used to quantify spasticity will be reviewed, as well as various techniques in use for reducing spasticity, with special emphasis on the use of electrical stimulation. Previous work to provide hand function in hemiplegics using functional electrical stimulation will also be reviewed. The last section provides a preview of the remainder of the thesis.

LITERATURE REVIEW

SPASTICITY AND THE UPPER MOTOR NEURON SYNDROME

A stroke, which is a focal neurologic deficit of sudden onset due to a disturbance in the blood supply to the brain (Dombovy and Bach-y-Rita, 1988), can severely disable an individual depending on its location and the extent of tissue damaged. A lesion to the central nervous system which affects descending motor pathways will produce the upper motor neuron syndrome. This syndrome is characterized by the negative symptoms of weakness, loss of dexterity, and fatigability, and the positive symptoms of abnormal posture, exaggerated proprioceptive reflexes (tendon jerks), increased resistance to slower passive stretch, and exaggerated cutaneous reflexes (Landau, 1980; Young and Wiegner, 1987; Burke, 1988). Negative symptoms represent performance deficits, whereas positive symptoms represent exaggerated disturbances of normal function or reactions to specific external stimuli (Landau, 1980). Positive symptoms are thought to result from release of relatively intact motor subsystems from precise rostral control, while most negative symptoms result from disruption of commands from higher motor centers (Young, 1990).

The motor control disorder spasticity may also occur following damage to motor pathways in patients suffering from stroke, spinal cord injury, head injury, multiple sclerosis, and cerebral palsy. A frequently cited definition of spasticity was posed by Lance (1980b), who defined spasticity as

“a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome.”

Two broader definitions of spasticity were offered by Young (1990) who added either

positive symptoms, or both positive and negative symptoms of the upper motor neuron syndrome to the original definition of Lance (1980b).

Following a stroke, patients may develop a characteristic posture. In the upper extremity, the hemiplegic posture consists of flexion of the fingers, wrist, and elbow, forearm pronation, and internal rotation of the upper arm. In the lower extremity, the posture consists of extension and adduction of the hip, extension of the knee, and plantar flexion of the ankle (Twitchell, 1951). This abnormal posture results from an inability to relax the spastic muscles; resting EMG is typically present in the flexor muscles in the upper extremity, and the extensor muscles in the lower extremity of hemiplegics (Young and Wiegner, 1987). Dimitrijevic and Nathan (1967) also noted motor unit activity in the muscles of spinal cord injured patients with spasticity, who appeared to be relaxed clinically (i.e. they had resting motor unit activity). This resting EMG in the spastic muscles, and paretic weakness in their antagonists, contributes to an imbalance of strength in antagonistic muscles across joints, which can lead to permanent contractures (Young and Wiegner, 1987).

In Chapter 3 of this document, the resting motor unit activity seen in spastic muscles is defined as being a symptom of spasticity. In contrast, Young and Wiegner (1987) and Burke (1988) refer to this resting EMG as dystonia, and not spasticity. This illustrates the wide range of definitions for spasticity present in the literature.

The increased resistance to slow passive stretch noted in the upper motor neuron syndrome might be due to changes in the stretch reflex. When a muscle is stretched, it reflexively contracts. If there is an increase in the strength of this contraction (an increase in stretch reflex gain), or the muscle contracts in response to a smaller displacement (decrease in stretch reflex threshold), this would account for the increased

resistance to passive stretch. However, there is disagreement in the literature regarding what change in the stretch reflex contributes to hypertonicity of spastic muscles. Some researchers have proposed that there is an increase in gain of the stretch reflex in spasticity (Rack et al., 1984; Thilmann et al., 1991a). However, Lee et al. (1987) determined that in voluntarily activated muscle, the reflex component of muscle stiffness is not different in the spastic and contralateral limbs of the same subject. Next Powers et al. (1988) determined that in both passive and voluntarily activated muscles, there is a decrease in the threshold of the stretch reflex in the spastic limb. Thus the increase in muscle tone seen in patients with spasticity is a consequence of a reduction in stretch reflex threshold, not an increase in gain of the stretch reflex as postulated by other researchers, although Thilmann et al. (1991a) are still in disagreement. Further insight into the symptoms of spasticity was provided by a study by Powers et al. (1989) which showed that the velocity dependence of reflex output in active spastic muscles was comparable to that seen in normal muscles. Thus, velocity-dependence is not abnormal in the active spastic state, in contrast to the definition of spasticity posed by Lance (1980b).

Some spastic subjects have difficulty making rapid movements, which may also be caused by exaggerated stretch reflexes. In a case study reported by Mizrahi and Angel (1979), stretch reflexes in the biceps during rapid elbow extension actually halted elbow extension briefly. Sahrmann and Norton (1977) also studied elbow extension and flexion in spastic subjects. However, they concluded that the slow elbow extension they observed was not due to reflex activation of the biceps, but resulted from inappropriate activation of biceps; specifically, delayed cessation of agonist contraction following elbow flexion. Difficulty in relaxing muscles was also noted by

Twitchell (1951). It may be that antagonist stretch reflexes impede movement in some subjects more so than others.

The loss of dexterity seen in patients with the upper motor neuron syndrome could also be due to unwanted cocontraction of muscles in addition to heightened stretch reflexes. Cocontraction of antagonists during voluntary movement has been well documented in hemiplegic patients (Sahrmann and Norton, 1977; Hammond et al., 1988; Keenan et al., 1989; Carey, 1990), and has been noted to slow the speed of movement (Sahrmann and Norton, 1977). Cocontraction arises from difficulties in controlling the temporal relationship between agonists and antagonists (i.e. in activating and deactivating muscular activity) (Bourbonnais et al., 1992). Hammond et al. (1988) observed both impaired agonist recruitment and antagonist inhibition in hemiplegic subjects during isometric wrist flexion and extension, but did not study voluntary movements. Although cocontraction does seem to slow movements and even impede movements in some subjects, these results are not consistent across studies. Carey (1990) stated he did not believe cocontraction of a finger flexor muscle was the major cause of the finger extension impairment he noted in the hemiplegic subjects who participated in his study.

A contributing factor to the inability of hemiplegic patients to make coordinated movements may be inappropriate activity of synergistic muscles acting at a joint (noted by Bourbonnais et al., 1989). They discovered that the directional selectivity and preferred active range of motion of elbow muscle activation was different in hemiplegic subjects' involved extremities as compared to their unimpaired extremities. However, the EMG patterns noted during isometric contractions at the paretic elbow joint did not conform with established synergistic patterns. Bourbonnais et al. (1992) hypothesized

that deficits in coordinated voluntary movements were due to impaired regulation of spinal neurons, which receive input from both supraspinal centers and peripheral afferents. Lesions of descending pathways probably affect regulation of spinal interneurons.

Coordination difficulties are not the only symptoms of spasticity and the upper motor neuron syndrome that can be attributed to impaired regulation of spinal neurons. Hyperexcitability of the stretch reflex is believed to result from impairments of inhibition at the spinal cord level. Current evidence points to reductions in presynaptic inhibition of Ia terminals in the spinal cord and reductions in reciprocal Ia inhibition as being responsible for exaggerated stretch reflexes in the spastic state (Young and Wiegner, 1987; Burke, 1988). They propose that since descending supraspinal regulation is impaired in patients with an upper motor neuron syndrome, Ia afferent inhibition is also impaired, resulting in the hyperreflexia observed in this patient population.

In addition to the disturbances of spinal reflex regulation in spastic subjects previously noted, a further contribution to their disability has recently been recognized. Evidence suggests that changes in muscle mechanical properties occur in spastic muscles, and contribute significantly to the observed increase in resistance to passive stretch (Dietz and Berger, 1983). Thilmann et al. (1991b) hypothesized that another contributing factor to the increase in stiffness in ankle dorsiflexion they observed could be due to loss of compliance in the Achilles tendon following stroke.

It should be clear that many factors contribute to the impairments seen in individuals with an upper motor neuron lesion. Partial or total paralysis occurs in muscles due to damage of corticospinal pathways. Disruption of descending pathways responsible for

inhibition in the spinal cord lead to hyperactive reflexes (both proprioceptive and cutaneous). Changes in muscle mechanical properties cause an increased resistance to passive stretch. And finally, movements are often uncoordinated due to cocontraction of antagonists and synergists. The motor control disorder spasticity, which incorporates many or all of these features of the upper motor neuron syndrome, is very disabling to patients. For this reason, much effort has been focused on quantifying this disorder, and seeking methods for decreasing spasticity.

METHODS FOR QUANTIFYING SPASTICITY

Methods for quantifying spasticity can be grouped into three areas: clinical, neurophysiological, and biomechanical (Stefanovska et al., 1991). Clinical testing measures are highly subjective, and include (but are not limited to) assigning scores for tendon jerk briskness or resistance to passive stretch. The Ashworth scale (Ashworth, 1964) has been used to grade the resistance to passive stretch in relaxed muscles as an indicator of the severity of spasticity (Alfieri, 1982; Delwaide and Oliver, 1988). The limb is moved throughout its range of motion and a score from one to five is assigned; one for no increase in tone and five for the limb being rigid in flexion or extension (see Methods, Chapter 3 for the complete scale). Unfortunately, clinical testing lacks precision and standardization, and is not appropriate for studying mechanisms involved in spasticity or documenting small variations in spasticity (Stefanovska et al., 1991).

Neurophysiological testing includes testing of tendon jerks, H-reflexes, and F-responses. It has the advantage of well standardized methodology, but the disadvantage of weak correlation between an individual test and clinical assessment of spasticity (Stefanovska et al., 1991). The H-reflex is the electrical analog of the tendon

jerk reflex; it is monosynaptic and created by activation of Ia afferent fibers by low intensity stimulation. The ratio of $H_{\max}/M_{\max}$ has been used to assess the excitability of the motoneuron pool ($H_{\max}$ = maximum amplitude of the H reflex; $M_{\max}$ = maximum amplitude of the direct motor response) (Delwaide, 1985), and has been used to assess the effects of electrical stimulation on spasticity (Levin and Hui-Chan, 1992). Increased $H_{\max}/M_{\max}$ ratios have been reported in hemiplegics and spinal cord injured patients with spasticity (Katz and Rymer, 1989). H-reflexes have also been used to assess reciprocal Ia inhibition (Boorman et al., 1991; Artieda et al., 1991), and Ib inhibition in patients with spasticity (Delwaide and Oliver, 1988).

Another measure of motor neuron excitability is the F-response. It is elicited through supramaximal stimulation of a motor nerve, which antidromically activates motor neurons creating a late but inconstant low amplitude response. The amplitude of the F-response indicates the degree of excitation of the motoneuron pool (Delwaide, 1985), and an increased amplitude and persistence has been reported in spastic patients (Katz and Rymer, 1989). Central to the definition of spasticity is hyperexcitability of the stretch reflex; both H-reflexes and F-responses, which characterize motor neuron excitability, help quantify this aspect of spasticity.

Neurophysiological tests have also been used to elucidate mechanisms involved in spasticity. Some widely used tests include tonic vibration reflexes, used to document the activity of excitatory Ia interneurons, and microneurography, to study the activity of the gamma system (Delwaide, 1985). Understanding the mechanisms involved in spasticity is critical in developing therapeutic interventions.

The third group of tests used to quantify spasticity are the biomechanical tests. Biomechanical testing is widely used to quantify spasticity and can provide objective

assessment of impairment. Unfortunately, there is a lack of standardization of parameters, including initial joint angles and range of testing motion (Stefanovska et al., 1991).

One widely used biomechanical test is the pendulum test (e.g. Bajd and Vodovnik, 1984; Vodovnik et al., 1987a; Robinson et al., 1988a). It is typically used to assess spasticity in muscles which extend or flex the knee, but has also been applied in the upper extremity (Stefanovska et al., 1991). When testing the knee extensor muscles, the knee is fully extended and the lower limb is dropped (generating stretch reflexes) and allowed to swing. In a normal subject, the knee swings six or seven times (Bajd and Vodovnik, 1984). In spastic subjects, the spasticity stops the swings of the lower leg and pushes it back towards the initial position (Bajd and Bowman, 1982), reducing the number of oscillations from normal unless clonus is elicited (Bajd and Vodovnik, 1984). To quantify other alterations in the oscillation pattern, a goniometer measures joint position; up to six parameters including swinging time, an amplitude ratio, and a normalized relaxation index are calculated to characterize the entire time course of limb oscillation (Bajd and Vodovnik, 1984). These parameters can then be compared to those of a normal subject (as an example, the relaxation index for a normal subject is greater than one; a value less than one would quantify various levels of spasticity). Parameters can also be tracked over time to evaluate the effectiveness of treatment measures (Robinson et al., 1988a,b).

In pendulum testing, EMG of joint antagonists can also be measured to assess their response to the oscillation. However, Bajd and Vodovnik (1984) did not believe it provided additional information on the subject's spasticity beyond that obtained from the goniograms.

Lin and Rymer (1991) developed a nonlinear model of the gravity-induced oscillation of the lower leg during the pendulum drop test. The model accounted for amplitude dependent variations in stiffness and damping, asymmetries in the swing, and abnormal reflex activation, and was able to simulate the motions accurately. The clinical utility of the pendulum drop test was not evaluated in this study.

Other biomechanical tests seek to quantify stiffness or resistive torque of a spastic limb. These parameters can describe differences in limb mechanical properties, as compared to normal, and could also be used to assess the efficacy of treatment measures. In measuring resistive torque properties, the subject is relaxed and a joint is perturbed, often with a train of sinusoids (Rebersek et al., 1986; Stefanovska et al., 1991). It has been documented that resistive torque is much larger in spastic hemiplegic patients versus normals at high frequencies of oscillation (Rebersek et al., 1986). Gottlieb et al. (1978) measured decreased joint compliance (increased stiffness) of relaxed spastic muscles while rotating the ankle joint with sinusoidally modulated torque.

In order to estimate joint stiffness, which is a measure of stretch reflex gain (Katz and Rymer, 1989), ramp and hold movements at varying velocities can be applied to a joint and stiffness estimated from the ratio of measured torque to position. Lee et al. (1987) and Powers et al. (1988) have shown that reflex stiffness, extracted from total joint stiffness, is not different in spastic limbs as compared to normals. They characterize spasticity by the threshold which elicits a stretch reflex, measured in terms of the angle at which torque increases from baseline, and have noted a decrease in stretch reflex threshold of spastic hemiplegic subjects (Powers et al., 1988). When a reduction in stretch reflex threshold is noted, the motoneurons are probably in a state of

sustained depolarization. Changes in reflex threshold can be used to distinguish between spastic-hypertonic and normal reflexes (Katz and Rymer, 1989).

EMG is frequently used to augment biomechanical measurements (Rebersek et al., 1986; Powers et al., 1988; Stefanovska et al., 1989). Some investigators have chosen to characterize spasticity in terms of reflex responses to sudden muscle stretch, and used EMG to quantify the response. Cody et al. (1987) and Dietz et al. (1991) have documented increased short latency reflexes and decreased long latency reflexes in forearm muscles of spastic subjects versus those measured in normals. Other researchers have used EMG as their sole measure of spasticity (Keenan et al., 1989). EMG is particularly useful for observing the state of spastic muscles at rest.

It is evident that there are numerous methods available for quantifying characteristics of spasticity. What is important to remember is that all aspects of spasticity cannot be quantified by a single test, and multiple tests should be used to provide as complete a description as possible of a given patient's spasticity (Stefanovska et al., 1991).

METHODS FOR REDUCING SPASTICITY

Traditional methods for decreasing spasticity include surgery and medications. Surgical intervention is usually reserved for severe cases of spasticity. Its goal is to disrupt the stretch reflex arc by making lesions, thus reducing its hyperactivity. In general, surgical intervention will only decrease positive symptoms of the upper motor neuron syndrome, and will not promote recovery from negative symptoms (Young and Wiegner, 1987). Surgical options include cutting dorsal or ventral roots, performing complete or selective dorsal rhizotomies, performing a selective neurotomy, and surgical release of appropriate tendons (Young and Wiegner, 1987; Perry, 1980).

Cutting roots completely destroys movement and/ or sensation; selective rhizotomies have the ability to destroy only smaller diameter fibers, thus sparing the large ones (Young and Wiegner, 1987). Dimitrijevic and Sherwood (1980) discuss chemical rhizotomies using a phenol solution. The advantage of this procedure is that it is reversible, the effects lasting on the order of months. However, even some surgical procedures are not permanent since regeneration can occur (Dimitrijevic and Sherwood, 1980). In general, these surgical procedures can be quite effective in reducing spasticity. However, there is often little voluntary function present once the spasticity has been removed (Young and Wiegner, 1987).

Many pharmacologic interventions for spasticity have been introduced over the last few decades. In recent years, only five drugs have been shown to decrease spasticity objectively, and include dantrolene, diazepam, tizanidine, baclofen, and idrocilamide (Young and Wiegner, 1987). Dantrolene acts to decrease spasticity by reducing the release of calcium into the sarcoplasm, thus limiting muscular contraction. It tends to provide generalized weakness as a result. Diazepam and tizanidine increase presynaptic inhibition, while baclofen and idrocilamide interfere with the release of excitatory neurotransmitters (baclofen is a GABA agonist and reduces spasticity by imitating the effect of inhibitory spinal interneurons on motor activity (Muller et al., 1987)). Baclofen, tizanidine and idrocilamide are primarily used with spinal cord injured patients (Young and Wiegner, 1987). A double-blind cross-over trial with placebo was used to test the effectiveness of orally administered baclofen. Substantial reductions in spasticity were noted in spinal cord injured patients, but in none of the patients with cerebral spasticity (Lance, 1980a). Thus, in patients with hemiplegia, the most commonly used medications are diazepam and idrocilamide. Unfortunately, their

ability to reduce spasticity is far less dramatic than the effects produced by the drugs used in patients with spinal spasticity.

As an alternative to surgical and pharmacological interventions, numerous researchers have used electrical stimulation techniques in an attempt to reduce spasticity. In the case of hemiplegics, pharmacologic interventions are far from successful. In all patient populations, surgical interventions are highly invasive. Electrical stimulation is a noninvasive alternative for the reduction of spasticity and has thus received much attention.

As early as 1952, Levine et al. stimulated the antagonists of spastic muscles in both the upper and lower extremity in many patient populations (spinal cord injury, stroke, head injury, and multiple sclerosis) and reported relaxation of the spastic muscle. They attributed this relaxation to the reciprocal innervation of the neuromuscular system. However, the degree of relaxation achieved was not quantified by this group.

Other studies have focused on using electrical stimulation to decrease spasticity in specific patient populations (e.g. spinal cord injury or stroke), and frequently concentrated on a specific joint, such as the knee. Vodovnik et al. (1984a) conducted a study in spinal cord injured patients with spasticity at the knee joint. Cyclical stimulation of quadriceps and contralateral hamstrings was applied for 30 minutes on three consecutive days (5 sec on, then 5 sec off while stimulation was applied to the opposite leg muscles). Qualitative assessment of the goniogram obtained during pendulum drop tests allowed the authors to conclude that electrical stimulation reduced spasticity in only half the spinal cord injured patients. When present, they noted a persistence of relaxation for up to 24 hours post-stimulation.

In an effort to identify the influence of stimulation parameters on the reduction of

spasticity, Vodovnik et al. (1987a) stimulated the quadriceps (the spastic muscle) of spinal cord injured patients, varying both the pulse width and frequency of stimulation, and used the pendulum test and EMG activity to quantify the degree of spasticity present pre-and post-stimulation. Electrical stimulation was applied cyclically (4 sec on, 4 sec off). They were able to achieve a reduction in spasticity as evidenced by a reduction in EMG, and an increase in the relaxation index computed in the pendulum drop test, with the best results achieved at a stimulation frequency of 100 Hz. However, they were not able to find a universal set of stimulation parameters effective in reducing spasticity for all subjects, and noted that the optimal stimulation parameters for a given subject varied from day to day due to the fact that the initial spasticity of a subject varies from day to day.

Robinson and coworkers (1988a,b) studied both the short term and the long term effects of cyclical electrical stimulation of the quadriceps in spinal cord injured subjects. In studying short term effects of the stimulation, subjects received 20 minutes of stimulation (2.5 sec on, 2.5 sec off) while the knee was held fixed; the authors felt this stimulation fatigued the spastic quadriceps muscle. Spasticity was quantified by the normalized relaxation index obtained during pendulum drop tests, and was found to be significantly decreased post-stimulation. Subjects with the highest levels of spasticity and the strongest contractions had the largest reductions post-stimulation. However, no carryover effect was noted 24 hours after the stimulation ended.

When studying the effects of four to eight weeks of stimulation applied two times per day, six days per week, these researchers found that most subjects had an increase in spasticity after four weeks of stimulation. After eight weeks of stimulation, fewer subjects had increases in spasticity (although many subjects stopped participating after

four weeks, decreasing the sample size). These researchers caution the use of electrical stimulation over long periods of time in order to decrease spasticity

Electrical stimulation has also been used to decrease spasticity in hemiplegic patients. Alfieri (1982) stimulated the antagonist to the spastic muscle in both the upper and lower extremities of hemiplegic patients and achieved relaxation in the spastic muscle. The stimulation parameters were set so that stretch reflexes were not elicited in the spastic muscle. The Ashworth scale was used as a qualitative measure of the degree of spasticity present in the muscle, and using this scale a reduction in spasticity following electrical stimulation was documented. However, spasticity reappeared on average one hour after the stimulation ended, depending on the movement efforts of the patient.

Other studies have used more quantitative measures to assess the efficacy of electrical stimulation in reducing hemiplegic spasticity. A study by Vodovnik and colleagues (1984b) assessed knee joint spasticity in hemiplegics following 30 minutes of cyclic stimulation of the hamstrings (5 sec on, 5 sec off). A second 30 minute stimulation paradigm of alternating stimulation to the quadriceps and hamstrings (5 sec stimulation periods) was also evaluated. Spasticity was quantified by calculating the normalized relaxation index obtained from the pendulum drop test. None of the subjects showed an increase in spasticity, but had either a decrease or no change in spasticity post-stimulation. An increase in selective knee flexion was noted in half the subjects. The investigators were not able to draw conclusions as to which stimulation protocol was the most effective in reducing spasticity.

In hemiplegic patients with spastic ankle joints, Vodovnik et al. (1987b) reported that spasticity characterized by a small permanent EMG in both the agonist and the antagonist, which was fairly independent of the velocity of the imposed stretch, was

decreased when cyclically stimulating both the spastic muscle and its antagonist at the ankle. The resistance to passive sinusoidal movements was also reduced. However, spasticity characterized by bursts of EMG activity only at high stretch velocities was increased in both the agonist and the antagonist following the same type of stimulation.

Stefanovska et al. (1989) conducted a study assessing the efficacy of chronic peroneal nerve stimulation on the reduction of hemiplegic spasticity at the ankle joint. In the previous studies of hemiplegic spasticity, stimulation was only applied acutely, but in this case it was applied for at least six months. Spasticity was quantified in terms of resistive torque as a function of passive stretching velocity, and was separated into the two categories described above. After six months of stimulation of the peroneal nerve, a decrease in resistive torque was documented in all subjects, indicating a decrease in the type of spasticity characterized by a small permanent EMG (independent of stretch velocity) in both the triceps surae and tibialis anterior muscles. The type of spasticity characterized by bursts of EMG activity at high stretch velocities was seen to increase in the tibialis anterior. An increase in voluntary strength was noted in both the plantar flexors and the dorsi flexors, the tibialis anterior showing the largest improvement. Note that specific muscles were not stimulated in this study, but rather whole nerve stimulation was performed. This group suggests that electrical stimulation should be used continuously for the beneficial effects to continue.

Recently, Levin and Hui-Chan (1992) studied the effects of 15 daily 60 minute applications of transcutaneous electrical nerve stimulation (TENS) to the common peroneal nerve in hemiplegic patients. They documented a decrease in spasticity based on clinical tests, and also noted a large improvement in ankle dorsiflexion forces. After three weeks of stimulation, there was a reduction in the magnitude of the spastic plantar

flexor stretch reflexes, as well as a decrease in EMG cocontraction ratios.

Researchers have also investigated the effects of stimulating dermatomes in order to decrease spasticity. In 1985, a study by Bajd and colleagues found dermatome stimulation to be effective in decreasing spinal spasticity at the knee joint in some subjects, but no carryover effect of the stimulation was noted. The stimulation was applied to the dermatome of the spastic muscle. Vodovnik et al. (1987b) also described stimulation of the dermatome associated with the spastic muscle as an effective mechanism for decreasing knee spasticity in the spinal cord injured patient, as documented by the pendulum drop test. However, the 1987 study found that dermatome stimulation wasn't effective for hemiplegic knee spasticity (the same dermatomes were stimulated in both patient populations). In contrast, Dewald et al. (1993) stimulated the antagonist to the spastic muscle in hemiplegics for 20 minutes below motor threshold, and documented reductions in torque of both elbow flexors and extensors during slow ramp perturbations of the elbow. These effects remained significant for 30 minutes post-stimulation. The observed reductions in spasticity were determined to be due to changes in stretch reflex threshold (versus gain).

The studies described have obtained a wide range of results regarding the ability of electrical stimulation to decrease spasticity when a whole nerve or branches of a motor nerve are stimulated, or when dermatomal or cutaneous levels of stimulation are applied. In addition, carryover effects of the stimulation are reported by some researchers and not by others. These discrepancies may be partially explained by the methods used to quantify spasticity. Some of the studies in which spasticity reduction following stimulation was noted (Levine et al., 1952; Alfieri, 1982) used only qualitative or clinical measures of spasticity which are highly subjective, and may not

have accurately assessed the influence of stimulation on spasticity.

In other studies, a wide range of stimulation paradigms were used in an effort to decrease spasticity. Variations in stimulation parameters, muscles stimulated, and stimulation durations likely account for much of the variation in results. These studies also included patients with spasticity of varying origin (complete and incomplete spinal cord injury; stroke). Even in the same patient population (e.g. stroke), subjects may have very different degrees of spasticity, which could be due to a variety of mechanisms. Thus, it is not surprising that the effects of electrical stimulation on spasticity have not had consistent results.

ELECTRICAL STIMULATION TO PROVIDE HAND FUNCTION IN HEMIPLEGIA

At present, little work has been done using functional electrical stimulation (FES) to provide hand function in hemiplegics. In 1973, Rebersek and Vodovnik reported the use of a simple FES system for hemiplegics who had preserved voluntary finger and thumb flexion, but needed assistance for extension. They did not mention that any of these subjects had spasticity. The authors describe the system as a one channel device with one surface electrode placed over the extensor digitorum communis (EDC), and another surface electrode placed over the abductor pollicis longus and the extensor pollicis longus muscles, thus providing extension of both the fingers and the thumb. Stimulation levels were proportionally controlled with movement of the shoulder on the subject's unimpaired side.

Eleven patients were evaluated with this device. Training consisted of learning to use the proportional command control source: patients were told to set and hold as many hand positions as possible, and the number of different positions achieved and

the quality of hand opening were documented. Further evaluations consisted of exercises simulating daily work. Patients had to place plastic pegs in and out of a board, and put graduated plastic baskets into each other and then remove them, which was reported to be a difficult task. The times required to complete these tasks were also recorded.

With repeated use, patients improved in their ability to proportionally control the device, and the extent of hand opening also increased with time. Some subjects could not manipulate the pegs or baskets without the device, but were able to perform the tasks with the electrical stimulation. Voluntary control of wrist and finger extension and thumb abduction either improved or remained the same with the use of the electrical stimulation, which provided therapeutic exercise.

In 1975, Merletti and colleagues performed a feasibility study of a multichannel electrophysiologic orthosis for hemiplegics. The device consisted of two channels of surface stimulation. Hand opening was provided by stimulating two wrist extensor muscles and the EDC, while elbow extension was provided by stimulating the triceps. The device was also proportionally controlled by movement of the nonimpaired shoulder.

Eight hemiplegic patients participated in the initial stage of the study. Three patients were rejected for further study because of strong biceps spasm (two subjects) and weak triceps (one subject). It was not stated if any of the subjects had spasticity present in their hand. The five remaining patients were evaluated over a three month period. Over this period of time, three subjects had an increase in voluntary elbow extension, while none had improvements in voluntary control of hand opening.

The performance of this orthosis was evaluated by having subjects grasp and release

objects in different positions on a flat surface. The evaluations were performed without any stimulation, with stimulation of only the wrist and finger extensors, or with two channels of stimulation. In this latter case, shoulder elevation-depression controlled elbow extension while protraction-retraction controlled hand opening. Three different control strategies were evaluated, but the results of each strategy were not reported.

None of the patients could perform the tasks with no stimulation or with only stimulation applied to the hand, except one patient who after one month of training could perform the tasks with only hand stimulation. The authors reported that a high degree of mental concentration was required to perform the task accurately and smoothly. Often spasms, tremor, and erratic shoulder movements occurred which decreased subject performance.

These are the only studies reported in the literature in which FES is used to provide hand function in hemiplegic patients. Although Merletti et al. did not specifically state that their subjects had spasticity, they did report tremors and spasms, which degraded performance of tasks. The presence of spasticity in many hemiplegic subjects, which can affect the ability of electrical stimulation to provide hand function, may be one factor contributing to the lack of research in this area.

PREVIEW OF REMAINDER OF THESIS

This thesis explores the ability of electrical stimulation to provide hand opening in patients with spastic hemiplegia. It was theorized that electrical stimulation of finger extensor muscles would decrease the spasticity in the finger flexor muscles while simultaneously providing finger extension. Spasticity was evaluated on the basis of intramuscular EMG recordings in two finger flexor muscles. Because I was recording EMGs from muscles close to the stimulation sites, the EMG recordings were contaminated with stimulus artifacts. Chapter 2 describes the signal processing techniques used to provide artifact free EMG recordings of the finger flexor muscles.

Chapter 3 describes the methodology and results obtained in two different studies. In the first study, reciprocal inhibition of finger flexor muscles during stimulation of a finger extensor muscle was evaluated in able bodied subjects. The second study investigated the ability of FES to provide finger extension in spastic hemiplegic patients, and documented the influence of the stimulation on the subject's spasticity. The final portion of this chapter contains suggestions for future research which will aid in the design of a neuroprosthesis to provide hand function in stroke patients with spasticity.

CHAPTER 2

INTRODUCTION

Chapter 2 contains the manuscript for a paper submitted to the *Journal of Neuroscience Methods*. It describes the methodology used to record intramuscular EMGs, and the processing tools developed to remove stimulus artifacts from the EMG in the muscles adjacent to the stimulated muscles. It also describes all the signal processing performed on the EMGs collected in this study.

Stimulus Artifact Removal in EMG from Muscles Adjacent to Stimulated Muscles

ABSTRACT

A digital technique for removing stimulus artifacts from EMG recordings in muscles which lie close to a stimulated muscle has been devised. The technique substitutes an estimate of the rectified EMG during the stimulus artifact into the rectified EMG during the period in which the artifact occurs, prior to lowpass filtering of the signal. Unlike other artifact elimination techniques, this method will not change the mean value of the EMG, regardless of artifact width, and provides a more accurate estimate of the EMG during the artifact interval than sample and hold techniques. Also discussed are other digital processing steps used to extract the low frequency information contained in rectified EMG signals.

INTRODUCTION

A major difficulty in recording the electrical activity (electromyogram, EMG) of muscles which are being stimulated or are near a stimulated muscle is the unwanted detection of stimulus artifacts by the EMG recording electrodes. Stimulus artifacts can be very large in magnitude and may last many milliseconds; these artifacts mask the underlying electrical activity in voluntarily contracting muscle and the electrically evoked M-waves, and much effort has been expended to reduce the duration of these artifacts and to delete them from EMG and neural recordings.

The duration of the stimulus artifact is dependent on many factors, including but not limited to amplifier recovery time from saturation (Walker and Kimura, 1978; McGill et al., 1982; Harding, 1991), highpass filtering stages in the recording amplifier

(Andersen and Buchthal, 1970; Freeman, 1971; Babb et al., 1978; Walker and Kimura, 1978; McGill et al., 1982; Merletti et al., 1992; Minzly et al., 1993), imbalance between recording electrode impedances which converts a common mode signal to a differential signal which the amplifier will not reject (McGill et al., 1982), and the type of stimulator output stage (Knaflitz and Merletti, 1988; McGill et al., 1982; Merletti et al., 1992). Note that although many authors advocate the use of a particular type of stimulator output stage, there is no published data to support their choice. For a summary of recording amplifier characteristics which can help minimize stimulus artifacts, see Merletti et al. (1992).

Methods to delete stimulus artifacts from EMG recordings fall into two major categories: hardware and software approaches. Hardware approaches have focused on providing circuitry which essentially blanks the artifact from the recorded signal using a sample and hold circuit (Freeman, 1971; Roby and Lettich, 1975; Babb et al., 1978; Shannon, 1979; Scott et al., 1980; Popovic and Schwirtlich, 1989; Benson et al., 1990), or which minimizes the duration of the artifact (Andersen and Buchthal, 1970; Walker and Kimura, 1978; Spencer, 1981; Knaflitz and Merletti, 1988; Millard et al., 1992; and Minzly et al., 1993). Artifact blanking was also employed by some authors in this latter group, either with a sample and hold circuit or by circuitry which held the output at an average past value (baseline) for the duration of the artifact (Knaflitz and Merletti, 1988). Software approaches have focused on digitally subtracting a template of the stimulus artifact from the recorded signal (McGill et al., 1982; Kiss and Shizgal, 1989; Blogg and Reid, 1990; Harding, 1991).

The approach taken in this paper is to use a software method for eliminating stimulus artifacts from EMG recordings. Unlike previous software methods where a template of

the artifact is subtracted from the EMG recording, we have used a digital substitution procedure, where an estimate of the rectified EMG during the stimulus artifact is substituted into the rectified EMG during the period in which the artifact occurs, prior to lowpass filtering of the signal. Rectification and low pass filtering extract the low frequency information pertaining to contraction magnitude. This paper also discusses digital filters used to extract the low frequencies in rectified EMG signals.

RATIONALE FOR SELECTED METHODOLOGY

We wanted to develop a technique for stimulus artifact removal that did not change the mean value of the rectified signal (which can occur when blanking the signal to a baseline value), and which provided an accurate estimate of the signal during the stimulus artifact period. In addition, we wanted to ensure that the stimulus artifact was removed down to the microvolt level, which is important when attempting to detect small changes in EMG levels. We chose not to use the method of subtracting a template of the artifact because our initial research revealed that the artifact waveform is not repeatable at the microvolt level, and thus subtraction of a template would be prone to errors. Another difficulty with the technique of subtracting a template is that large stimulus artifacts can cause the amplifier to operate in a nonlinear region, thus distorting the EMG recorded. Subtraction of an artifact template may remove the artifact, but the EMG remaining may be distorted.

Our research is investigating the possible reflex effects of stimulation of forearm extensor muscles. Since reflex effects might be manifested as very small changes in EMG in other muscles, time averages of rectified EMGs were used to assess their presence. Because the EMG changes may be very small, we desired a detection

sensitivity on the order of tens of microvolts. A small change in EMG could be masked if stimulus artifacts were present, so we decided that residual stimulus artifacts remaining after processing should be less than 10 μ V. This criterion is much more stringent than most cited in the literature.

STIMULATING AND RECORDING PARADIGM

Electrical stimulation was applied to a finger extensor muscle (extensor digitorum communis, EDC) and intramuscular EMG recordings were made in the EDC and two finger flexor muscles (flexor digitorum superficialis, FDS, and flexor digitorum profundus, FDP). Subjects were either able bodied humans or persons with hemiplegia secondary to hemispheric infarct or hemorrhage. Hemiplegic subjects also received simultaneous stimulation of the ulnar nerve which innervates intrinsic finger extensor muscles.

The stimulators used were constant current battery powered stimulators which produced the biphasic waveform shown in Figure 2.1. The waveform consists of a rectangular cathodic phase, followed by an interphase delay of 20 μ sec and a capacitively coupled anodic phase. Capacitive coupling guarantees true charge balancing, which is necessary to avoid tissue damage which could occur with chronic stimulation if charge balancing were not performed (Mortimer et al., 1980). In order to minimize stimulus artifacts the anodic recharge phase was limited to a maximum of 3.2 msec. These stimulators had an overhead voltage of 90 V.

The first stage of the recording amplifiers consisted of a differential preamplifier mounted on the subject's arm. This preamplifier had a gain of 200 and a highpass filter with a cutoff frequency of 10 Hz (one pole). It was mounted on the subject's arm to

limit 60 Hz noise pickup. The single ended output of the preamplifier was passed into the noninverting input of a commercial amplifier ¹ (inverting and reference inputs tied together), and the signal was lowpass filtered at 2 KHz (three poles). The commercial amplifier had an additional one pole highpass filter at 10 Hz. The overall system gain was 1400 and had a common mode rejection ratio of 58 - 72 dB at 1 KHz, depending on the channel.

Figure 2.1 Biphasic constant current capacitively coupled stimulus waveform. The interphase delay is 20 μsec in length.

The EMG recording electrodes were constructed from pairs of Polyimide-insulated stainless steel wires ² which had 2 mm deinsulated tips. The wires were bonded

¹ Model MMRS-1, BAK Electronics, Inc., Germantown, MD 20874

² .0018 inch stainless steel 316 LVM annealed wire, TML (Polyimide) insulation, California Fine Wire Company, Grover City, CA 93433

together ³ with a 1 mm spacing between the deinsulated areas. The entire electrode was bent to form a 1 cm long barb, and was placed in a hypodermic needle for insertion into the muscle. Following insertion the barb helped to anchor the electrode after the needle was withdrawn.

A carbon rubber surface reference electrode (4.1 cm by 8.8 cm) was used, with its location varied in order to minimize 60 Hz noise. A common placement was on the volar forearm lateral to the FDS recording electrode.

Because forearm muscles are so small, no attempt was made to place the recording electrodes in a particular position within the muscle. In order to avoid EMG pickup from muscles other than the target muscle (crosstalk), low level stimulation (amplitude = 20 mA, pulse width < 50 μ sec) was applied to each recording electrode in succession. If the target muscle contracted forcefully during stimulation prior to any activation of a neighboring muscle (i.e. there was a large separation in thresholds), then the recording electrode was left in place. If there was little separation in thresholds the recording electrode was replaced. Electrode impedances measured between the anode and one side of the recording electrode ranged from 42 K Ω to 170 K Ω (measurement made at 30 Hz with a commercial meter ⁴).

The type of stimulating electrode used varied depending on the type of subject (hemiplegic or able bodied). Hemiplegic subjects were stimulated simultaneously at two sites with surface electrodes (1.25 inches in diameter). The electrodes were placed in pairs along the length of the EDC muscle body and also along the course of the ulnar

³ SILASTIC BRAND medical adhesive silicone type A

⁴ Model EZM5, Grass Instrument Co., Quincy, MA 02169

nerve (Hines et al., 1993). Typical separation between the edges of the anode and cathode was 1 cm (anode proximal to cathode). The EDC stimulating electrodes were always proximal to the EDC recording electrode, usually by 1 cm (from recording electrode to edge of cathode). Able bodied subjects received intramuscular stimulation of only the EDC through a percutaneous coiled wire electrode. A carbon rubber surface electrode placed on the radial side of the forearm just distal to the elbow served as an anode.

Able bodied subjects (intramuscular stimulation) received stimulation currents fixed at 20 mA, and pulse widths were varied as multiples of motor threshold (thresholds ranged from 8 - 110 μ sec). Hemiplegic subjects (surface stimulation) required larger currents. The amplitude was first fixed at 20 mA and pulse widths were increased up to 210 μ sec. If a suitable motor response had not been achieved with these parameters, the pulse width was fixed near 200 μ sec and the amplitude was increased up to 75 mA. All subjects were stimulated at a frequency that provided a fused contraction of the EDC (between 15 and 25 Hz).

All EMGs, and a trigger signal indicating the occurrence of stimulus pulses, were sampled by a computer at 3012 or 3048 Hz. The computer also controlled the stimulator outputs. Able bodied subjects were asked to maintain a 10% maximal voluntary contraction during each of the trials. Hemiplegic subjects were asked to perform a variety of tasks, and generally had resting motor unit activity present in their forearm flexor muscles even if they were asked to relax (Hines et al., 1993).

ARTIFACT DELETION TECHNIQUE

The length of the stimulus artifact on the FDS and FDP EMG channels was

determined in separate tests prior to the trials assessing reflex effects of extensor stimulation. The subject was asked to relax so that no motor unit activity was present in the flexor muscles. Lack of spontaneous EMG was confirmed visually on an oscilloscope. For the hemiplegic patients we sometimes had to wait minutes in order for motor unit activity to cease in the flexor muscles. A brief train of approximately 100 stimulus pulses was delivered through the stimulating electrodes at a rate of 20 Hz, producing an interpulse interval (IPI) of 50 msec. The current amplitude and pulse width were set at the maximum values to be used during the subsequent trials. Since we were recording in muscles which were not being stimulated, no M-waves were recorded, only stimulus artifact.

The recorded (raw) signal between stimulus pulses was ensemble averaged for each flexor channel (referred to as IPI ensemble averaging). Ensemble averaging decreases the residual random noise on the recordings so that the artifact can be accurately detected at the microvolt level.

By inspecting many artifact recordings it was clear that there was no artifact present in the last 10 msec of the interpulse interval, even at the microvolt level (since the voltage remained relatively constant). The mean value of the signal over the last 10 msec of the ensemble average was computed to obtain the baseline value of the amplifier output. Then the time from stimulus onset to the time at which the ensemble averaged artifact decreased to and stayed within 10 μ V (regardless of sign) of the baseline was determined, and was defined as the artifact interval (Figure 2.2). This process was repeated one or two more times, and the longest recorded artifact interval for either channel was used in the subsequent data processing steps. The selection of a 10 μ V criterion for defining the end of an artifact was a compromise between

minimizing the amount of residual artifact present in the signal post-processing, and keeping the artifact as short as possible to minimize the amount of EMG that must be deleted in the following steps.

All processing of the artifact was done offline in software following each of the subsequent trials. The first step in data processing was to calculate the offset of each EMG channel by averaging the first one second of data. Then the time that each

Figure 2.2 IPI ensemble averaged stimulus artifact waveform. The artifact interval duration is 6.97 msec. In this example, stimulation was applied only to the ulnar nerve (pulse width = 150 μ sec, amplitude = 20 mA), and EMG was recorded in the FDP muscle.

stimulus pulse occurred was determined using the sampled trigger signal. Starting at the onset of each stimulus pulse, and for the remainder of the artifact interval, the EMG was set equal to the offset for that data channel. Next each EMG channel had its offset subtracted and was rectified (Figure 2.3a). In order to provide an estimate for the EMG

Figure 2.3 EMG processing steps used to remove stimulus artifacts and to extract the low frequency content of the signal.

during the artifact interval, 75 samples of EMG (approximately 25 msec) before and after the artifact interval were averaged and substituted into the artifact interval (Figure 2.3b). The tradeoffs involved in selecting a time over which to average are that longer times will provide more smoothing of the EMG, and shorter times will produce a less accurate estimate of the true EMG magnitude. All EMG channels were then digitally lowpass filtered at approximately 100 Hz (Figure 2.3c) with a causal infinite impulse response (IIR) filter. The last data processing steps included decimation of the data by a factor of four (to reduce data storage requirements) and scaling of the EMG to units of μV .

The selection of a lowpass filter is very important to prevent aliasing of sampled signals. Many authors use moving average filters to eliminate the high frequency content of rectified EMG and to estimate the magnitude of the low frequency envelope (Gottlieb and Agarwal, 1970; Kreifeldt, 1971; Hogan and Mann, 1980 a,b). However, our investigation showed that a moving averager does not provide good attenuation above the cutoff frequency (3 dB point) and as an alternative we selected an IIR filter. This filter was constructed using the yulewalk algorithm in MATLAB ⁵, a recursive filter design using a least-squares method. Figure 2.4a displays the frequency response of a digital moving averager with a 3 dB point of 105 Hz, and Figure 2.4b displays the frequency response of the filter used in this study (3 dB point = 123 Hz). The power remaining in an EMG signal after filtering is shown for both filters in Figure 2.4c, d (dashed lines indicate signal power prior to any processing). The power remaining in EMG that has had its baseline subtracted, and has been rectified and filtered is shown in Figure 2.4 e, f (this is equivalent to the processing done to our data). Dashed lines here

⁵ The MathWorks, South Natick, MA 01760

Figure 2.4 The frequency response of a digital moving averager (a) and a digital IIR filter (b). Power remaining in filtered EMG (100% maximal voluntary contraction, solid line); in (c) the moving averager was used, in (d) the IIR filter was used. Dashed lines indicate the power of the unfiltered signal. In (e) and (f) the signal had its baseline subtracted and was rectified prior to filtering with the moving averager (e) and the IIR filter (f). The dashed lines indicate the power of the signal after baseline subtraction and rectification. Note that the moving averager does not attenuate high frequencies as well as the IIR filter.

indicate signal power after baseline subtraction and rectification, prior to filtering. For the IIR filter, 95% of the power in the rectified and filtered signal lies below 170 Hz, while for the moving averager 95% of the power lies below 316 Hz. The attenuation with the IIR filter is generally 20 - 40 dB greater than the attenuation with the moving averager in the frequency range above 300 Hz. Thus the data filtered with the moving averager cannot be decimated to the same degree as the data filtered with the IIR filter if aliasing is to be prevented (decimation is equivalent to lowering the sampling frequency of the signal).

SIMULATION STUDY

A simulation study was performed to compare the effects of various techniques for removing stimulus artifacts from signals. The test signals used in the simulation were either Gaussian noise, or Gaussian noise multiplied by a 2 Hz sine wave which provided amplitude modulation (EMG is frequently modeled as amplitude modulated Gaussian noise (Hogan and Mann, 1980a)). A stimulus train (series of truncated exponentials) was added to each test signal. The "artifact" widths were 10 msec in duration, and the stimulus frequency was 20 Hz. All signals were then rectified.

The three artifact removal techniques studied included the substitution procedure described in this paper, blanking the signal to baseline during the artifact interval, and the sample and hold technique. Both test signals were processed using the three artifact removal techniques. The mean of the rectified Gaussian signal with added stimulus train (after artifact deletion was performed) was computed (all three methods) and compared to the mean of this same signal without the added stimulus train (artifact-free signal). Unpaired t-tests ($N = 20$) were performed to assess the effects of the artifact

deletion techniques on signal mean.

RESULTS

Recorded artifact widths varied greatly both across subjects, and for different electrodes in the same subject. Recorded artifact widths varied from 1 msec up to 17 msec. On occasion longer artifact widths were recorded; however, these were not acceptable and suitable measures were taken to decrease the artifact width (such as varying the position of the EMG reference electrode, and in a few cases decreasing the stimulus amplitude or pulse width). Artifact width measurements were made on 25 electrodes during 13 different experiments. Of these 25 electrodes, three had artifact widths which could not be decreased below 17 msec with stimulus parameters set at values providing a suitable motor response. When the stimulus frequency is 20 Hz, 17 msec corresponds to one third of the interpulse interval. We did not want to delete more than one third of this interval. In these three cases, residual artifact over 10 μ V remained in the EMG recordings.

Figure 2.5a shows unprocessed (raw) EMG recorded from the FDS muscle of a hemiplegic subject during simultaneous stimulation of the EDC and the ulnar nerve. Tick marks at the bottom of the figure indicate the occurrence of the stimulus pulses. Figure 2.5b shows the processed EMG both with (solid line) and without (dashed line) artifact deletion. In both cases the EMG has its offset subtracted, is rectified, lowpass filtered, and decimated. It is obvious that without the digital artifact deletion the recorded EMG would appear to be much larger than it truly is due to contamination by the artifact.

Figure 2.6 displays EMG that has been fully processed to remove stimulus artifacts

Figure 2.5 Unprocessed EMG containing stimulus artifacts (a). The occurrence of stimulus pulses is indicated by the tick marks at the bottom of the figure. Fully processed EMG is shown in (b) both with artifact deletion (solid line) and without artifact deletion (dashed line).

and has then been IPI ensemble averaged in order to reduce any noise (116 IPIs were averaged). The EMG was collected when no motor units were firing, and thus the only recorded signal is stimulus artifact. The figure indicates that the residual artifact is below the 10 μV criterion. All data processed in this manner had residual stimulus artifacts ranging in magnitude from 3 - 6 μV .

Figure 2.6 Fully processed EMG that has been IPI ensemble averaged to show residual stimulus artifact (14 msec of artifact deletion was used). Stimulation was applied to the EDC muscle (pulse width = 40 μsec , amplitude = 20 mA), and EMG was recorded in the FDP muscle. Residual stimulus artifact is well below the 10 μV criterion.

The results of two different artifact deletion techniques are shown in Figure 2.7. Figure 2.7a,b displays the effect of the sample and hold technique on Gaussian noise (a) and amplitude modulated Gaussian noise (b). Figure 2.7c,d displays the effect of the substitution procedure on the same signals. Note that the signal estimate during the artifact interval is much worse in many instances when the sample and hold technique is

Figure 2.7 Examples of the effect of using the sample and hold technique (a) and (b), and the substitution procedure (c) and (d) to remove stimulus artifacts from a Gaussian noise signal. Stimulus artifacts are 10 msec in length.

used (e.g. the first, second, and fifth artifact intervals in Figure 2.7a). The substitution procedure provides a more accurate estimate of the true signal magnitude during each of the artifact intervals.

Unpaired t-tests indicate that there is no statistical difference in the means of the artifact-free signal versus the signals processed with the substitution procedure or the sample and hold technique. However, the mean of the signal processed by blanking to baseline during the artifact interval is statistically decreased versus the mean of the artifact-free signal ($p = .0001$). The mean of the signal processed by blanking to baseline will be decreased from the mean of the artifact-free signal according to the following formula:

$$\text{Mean}_{\text{blanked signal}} = \text{Mean}_{\text{artifact-free signal}} * (\text{IPI} - \text{artifact width}) / \text{IPI}$$

DISCUSSION

The artifacts recorded in our experiments were frequently over 10 msec in length. Long artifacts increase the information loss in the signal since a greater portion of the signal must be blanked, and effort must be expended to minimize the length of the artifact. Our long artifacts are likely due to a combination of factors, including the 10 Hz highpass filtering in the recording amplifiers, mismatched electrode impedances which convert a common mode signal into a differential signal which is not rejected by the amplifier (common mode conversion, McGill et al., 1982), amplifier recovery time from saturation, and the 3.2 msec long recharge current in our stimulators.

Some authors (Freeman, 1971; Babb et al., 1978; Walker and Kimura, 1978; Blogg and Reid, 1990; Millard et al., 1992; Minzly et al., 1993) have chosen to use DC coupled amplifiers in order to avoid highpass filtering of the stimulus artifact, which

can possibly cause amplifier saturation, and which adds an exponential tail to the artifact. However, a DC coupled signal can be easily contaminated by motion artifacts. Walker and Kimura (1978) solved this issue in their DC coupled amplifier by using a feedback loop which provided highpass filtering (the feedforward path was entirely DC coupled). During the stimulus artifact this feedback loop was opened; following the overloading input the loop was closed which quickly returned the amplifier output to baseline.

Mismatched electrode impedances contribute to stimulus artifacts, so care should be taken to construct recording electrodes with closely matched impedances. This will require careful removal of the insulation to ensure that equal areas of wire are exposed on both sides of the electrode. In vitro testing of electrode impedances in a saline bath prior to sterilization and implantation can also be done in order to identify electrodes with a large impedance imbalance.

A contributing factor to our long artifact widths was the 3.2 msec long recharge current in our stimulators. These stimulators were built for chronic stimulation purposes, and capacitive coupling was used to guarantee true charge balancing to avoid tissue damage (true charge balancing is more difficult to achieve in a rectangular biphasic waveform). However, a study was recently performed which indicates that tissue damage does not occur with an imbalanced rectangular biphasic waveform if the net DC current density is less than $35 \mu\text{A}/\text{mm}^2$ (Scheiner and Mortimer, 1990). Thus an imbalanced rectangular biphasic waveform could be used, and would eliminate the long recharge currents created by capacitive coupling of the anodic phase.

One reason that artifact lengths varied across experiments is that subjects were stimulated at intensities relating to their motor response, and thus most subjects were

not stimulated at the same pulse widths and current levels. In addition, new recording electrodes were used in each experiment, and electrode impedances were found to vary considerably. It is thus likely that the degree of electrode impedance imbalance varied across experiments, varying the amount of common mode conversion. This also helps account for differing artifact lengths of the FDP and FDS electrodes in the same subject. Another contributing factor for the differences between these two electrodes is that since these electrodes were located in different geometries relative to the stimulating electrodes, they were exposed to different currents which affected the potential difference recorded by the electrode pairs (McGill et al., 1982).

Our preliminary investigations indicated that the artifact shape and duration were not constant at the microvolt level. When this is the case, subtracting an artifact template will not provide adequate artifact removal, and we therefore chose a substitution procedure as an alternative.

The digital substitution procedure we have used to eliminate stimulus artifacts from EMG recordings has many advantages over the technique of sample and hold, or blanking the signal to baseline during the artifact interval (both of which can be implemented either in hardware or software). A problem with simply blanking the signal to baseline during the artifact interval is that it will decrease the mean value of the rectified signal. Our method substitutes an estimate of the true EMG level (versus the baseline) into the artifact interval, and will not change the mean value, regardless of the length of the artifact interval.

The sample and hold methodology can be implemented by performing a sample and hold of either the raw or the rectified EMG signal. These methods do not change the mean value of the signal. However, they provide a poor estimate of the magnitude of

the signal during the blanking interval, as demonstrated in Figure 2.7. Our method provides a far better estimate of the EMG during the blanking interval. If it is necessary to implement the artifact deletion system in real time, then it is not possible to substitute into the artifact interval an average including values post-artifact. A possible real time system configuration would be to lowpass filter the rectified EMG during periods of no stimulation. When a stimulation pulse occurs, the EMG input would be disconnected and the output of the lowpass filter would be fed into its input, thus providing an input signal representing the recent level of the EMG. Once the artifact interval has passed, the rectified EMG signal would again be connected to the input of the lowpass filter. This technique should be superior to a sample and hold since the filter input during stimulus pulses would be a recent average value of the EMG, versus a single sample which is held constant for the duration of the artifact.

The technique we have developed for stimulus artifact removal is only appropriate for removing artifacts from muscles which are not being stimulated since the technique relies on being able to estimate the artifact length when no M-waves are present. The presence of M-waves makes it impossible to estimate the artifact length at the microvolt level since the M-waves and artifact overlap in time. However, there are many instances when EMG recordings are made in nonstimulated muscles. One such example is when the EMG of a muscle under voluntary control is used to provide a command control signal for a prosthesis employing electrical stimulation. The stimulation may either activate paralyzed muscles (Handa et al., 1990), or provide electrocutaneous sensory feedback in an artificial arm prosthesis (Shannon, 1979; Scott et al., 1980). EMG recordings from the muscle providing control signals may be contaminated by stimulus artifacts from neighboring stimulated muscles or the

electrocutaneous stimulation. The real time artifact deletion method described above could be utilized to provide a command control signal free of stimulus artifacts.

ACKNOWLEDGEMENTS

This research was supported by National Institute on Disabilities and Rehabilitation Research Grant H133E80020. We would like to thank Drs. Michael Keith and Wendy Wilson for referring subjects to the study, and Dr. Chou-Ching Lin for his help in conducting the experiments.

CHAPTER 3

INTRODUCTION

Spasticity is a motor control disorder common in persons suffering from cerebral palsy, spinal cord injury, multiple sclerosis, and stroke. In all these disabilities, there is a disruption of descending motor pathways, and spasticity frequently develops post injury. Spasticity has many symptoms, a primary one being an involuntary increase in muscle tone, causing the affected muscles to contract. Thus a person with spasticity may have muscles that cannot be completely relaxed. In some persons the muscles can be relaxed, but may be activated inappropriately; both the agonist and antagonist muscles may be activated simultaneously making it difficult for the person to make desired movements.

Following a stroke, a person may be partially or totally paralyzed on one side of the body (hemiplegic). In the upper extremity many of the flexor muscles develop spasticity, causing the fingers to involuntarily tighten into a fist. The spasticity can be very strong. Even if the stroke patient has some residual voluntary control over the finger extensor muscles, they may be too weak to overcome the high tone in the flexor muscles. As a consequence, the hand cannot be opened voluntarily in order to acquire an object, and the person's ability to perform activities of daily living is severely impaired.

Techniques have been developed to activate paralyzed muscles using electrical stimulation, and neuroprostheses have been produced which provide hand function to quadriplegic patients (Peckham et al., 1983). Stroke patients are another potential user

population for neuroprostheses that restore hand function. The goal of this thesis is to begin to investigate how to apply this technology to stroke patients, specifically those who have spasticity. Electrical stimulation may have the added benefit of reducing spasticity in addition to activating paralyzed muscles.

Over the years there has been anecdotal evidence in the literature that stimulating the antagonist muscle may inhibit the spastic muscle's activity. As early as 1952, Levine et al. stimulated the antagonists of spastic muscles in hemiplegics and reported relaxation of the spastic muscle. They attributed this relaxation to the reciprocal innervation of the neuromuscular system. However, the degree of relaxation achieved was not quantified by this group. More recently, Alfieri (1982) also stimulated the antagonist to the spastic muscle in hemiplegics and achieved relaxation in the spastic muscle. A clinical scale was used as a qualitative measure of the degree of spasticity present in the muscle. Alfieri postulated that reciprocal inhibition could be one mechanism responsible for the observed reduction of spasticity.

Based on the anecdotal evidence in the literature that reciprocal inhibition may be exploited in order to reduce spasticity, I decided to test this theory first in able bodied individuals. Experiments were conducted to determine if stimulation of a finger extensor muscle could inhibit the activity in two finger flexor muscles.

The next set of experiments was conducted with hemiplegic patients who had spasticity present in their hand. I investigated the use of electrical stimulation of an extrinsic finger extensor muscle and the ulnar nerve (which activates intrinsic finger muscles to extend the distal finger joints) to provide functional opening of the fingers. Stimulation of finger extensor muscles will directly provide some degree of finger extension. I theorized that extensor stimulation might have the added benefit of

relaxing the spastic finger flexor muscles, which would further aid hand opening. The goals of these experiments were 1) to provide functional opening of the fingers in this patient population, 2) to observe the interaction of electrical stimulation with the subject's spasticity, and 3) to study the influence of voluntary effort on hand opening. Additional tests were performed to document the subject's ability to voluntarily flex and extend his or her fingers.

The long term goal of our research is to provide the hemiplegic patient with grasp and release capabilities in the impaired hand. This hand could then be used as a helper hand in the performance of bimanual tasks, such as unscrewing the lid from a jar, or stirring the contents of a pot. The research presented here on the use of electrical stimulation to provide finger extension is a first step towards achieving the goal of providing spastic hemiplegic patients with a helper hand. The use of both hands will further their independence, and will assist them in the performance of many activities of daily living.

METHODS

Subjects

Seven able bodied subjects participated in the experiments testing if reciprocal inhibition of finger flexor muscles could be achieved by stimulating a finger extensor muscle. Six hemiplegic subjects participated in the experiments investigating if spasticity could be reduced and functional hand opening achieved with electrical stimulation. These subjects all had hemispheric strokes resulting from an infarct or hemorrhage, and had sufficient cognition to give informed consent to participate in the study and to perform the tasks required in the experiment. Each hemiplegic subject had spasticity in their finger flexor muscles and was at least one month post stroke (most subjects were at least one year post stroke) (Table 3.1). None of the subjects were taking medications which would be anticipated to affect their spasticity. The selection criteria for participating in this study are listed in Table 3.2.

Clinical Testing of Hemiplegic Subjects

Clinical testing was performed to verify that subjects met the selection criteria to participate in the study, and to provide a clinical measure of their impairment. The following tests were performed:

Qualitative Assessment of Spasticity: The Ashworth scale (Ashworth, 1964) was used to grade spasticity in most upper extremity joints of the subject's impaired side (Appendix A). In some subjects the unimpaired side was also tested. The joint being tested was moved throughout its range of motion at both slow and fast velocities, and a

HEMIPLEGIC SUBJECTS						
Initials	Sex	Impairment	Age	Years Post-stroke	Handedness Prior to Stroke	Ashworth Score
JM	M	R HP	57	8	left	2-3
TC	F	R HP	20	1.5	right	3-4
CR	M	R HP	28	8.5	left (ambidextrous)	2
LMM	F	L HP	37	9.5	right	3-4
HM	M	L HP	71	5.5	right	2-3
EB	M	L HP	68	6 weeks	right	3

Table 3.1 Hemiplegic subjects participating in the study. Ashworth scores represent the range of scores for the subject's fingers obtained in the pre-experiment evaluation.

spasticity score was assigned based on the criteria listed in Table 3.3. When testing the fingers and thumb, the subject's wrist was held in neutral flexion-extension by the examiner. The fingers and thumb were only tested in extension. In this study, a score was given for joint movement in a specific direction (e.g. flexion); scores were not assigned for specific muscles. In order to participate in the study, subjects needed a score of two or greater for finger extension (either MP or IP joints). Spasticity testing was done prior to the start of the experiment (before all other clinical testing), and at its conclusion.

<p>Required Inclusions</p> <p>Small (Lacunar) or large vessel <u>hemispheric</u> stroke</p> <p>At least one month post-stroke</p> <p>Spasticity present in finger flexor muscles</p>
<p>Allowable Inclusions</p> <p>Patient only using medications which do not affect tone (Baclofen and Dantrium are examples of medications affecting tone)</p> <p>Language deficits if cognitive deficits are not severe</p> <p>Left or right sided lesion</p>
<p>Exclusions</p> <p>Previous forearm nerve or brachial plexus injury</p> <p>Severe cognitive deficits, inability to cooperate</p> <p>Significant multiple or bilateral strokes</p> <p>Dysesthesia or hyperesthesia</p> <p>Significant fixed contractures</p> <p> Patient must have the following passive range of motion:</p> <p> MP joints: 0 - 70 degrees</p> <p> Wrist: 45 degrees (flexion/extension)</p> <p> Elbow: 140 degrees extension</p>

Table 3.2 Selection criteria for hemiplegic patients. Patients must meet the required inclusion criteria to participate in the study, and are excluded if any of the exclusion criteria are met. They may participate in the study if they meet any of the allowable inclusions, but these are not required.

Sensory Evaluation: The sensations of light touch and pinprick were tested on the subject's impaired upper extremity in the positions listed in Appendix A; the subject was asked to compare the sensation to the sensation elicited by the same stimulus on the unimpaired extremity. Sensation was graded according to the following scale:

The Ashworth Scale Passive movements of joints are made. The resistance encountered to passive movement is scored as follows:	
Score	Characteristic
1	No increase in tone
2	Slight increase in tone, giving a “catch” when the affected part(s) is moved in flexion or extension
3	More marked increase in tone but affected part(s) easily flexed
4	Considerable increase in tone; passive movement difficult
5	Affected part(s) rigid in flexion or extension

Table 3.3 The Ashworth scale.

0 = absent; 1 = impaired; 2 = normal; 3 = hyperesthesia (enhanced). The sensation could also be identified as dysesthesia (nonnoxious stimulus is painful) or paresthesia (unrelated sensation). Subjects with substantial hyperesthesia or dysesthesia were excluded from the study.

Reflex Testing: Tendon jerk reflexes were tested bilaterally on the finger flexors, biceps, triceps, brachioradialis, and pectoralis, and were graded on the following scale (Bates, 1983) (finger flexor and pectoralis were graded as present or absent):

0 = no response

1 = somewhat diminished; low normal

2 = average; normal

3 = brisker than average; possibly but not necessarily indicative of disease

4 = very brisk, hyperactive; often indicative of disease; often associated with clonus

Any spread of these reflexes to other muscles was noted (reflex spread occurs when tapping on a tendon excites muscles other than the muscle being stretched). The presence or absence of the Hoffman reflex was assessed (stimulus = brief flexion of terminal phalanx of the long finger, response = flexion of fingers and thumb), and the presence of wrist clonus in response to a quick extension of the wrist was noted. Spread of reflexes, and the presence of the Hoffman reflex and wrist clonus indicate that the subject's reflexes are hyperactive (Mayo Clinic Department of Neurology, 1981). Reflex testing was always performed immediately prior to the experiment by Dr. Carl Billian, a neurologist.

Upper Extremity Active Range of Motion (AROM): AROM was tested with gravity eliminated on the joints listed in Appendix A (impaired extremity). Scoring was based on the following scale: 0 = none, 1 = less than normal, 2 = full range of motion.

Voluntary Muscle Strength: Voluntary muscle strength was assessed in both flexion and extension in the fingers, thumb, and wrist of the impaired extremity, using the MRC scale:

0 = absent (total paralysis)

1 = trace (palpable or visible contraction)

2 = poor (active movement with gravity eliminated)

3 = fair (active movement against gravity)

4 = good (active movement against resistance)

5 = normal

General Study Design

During the experiment electromyographic (EMG) signals were recorded in the extensor digitorum communis (EDC), the flexor digitorum superficialis (FDS), and the flexor digitorum profundus (FDP) muscles while the subjects performed specified tasks. The EDC extends the metacarpophalangeal (MP) joints of the fingers. The FDS flexes the MP joints and the proximal interphalangeal (IP) joints. The FDP flexes the MP and both the proximal and distal IP joints of the fingers. In most trials the subjects received electrical stimulation to the EDC, or the EDC and ulnar nerve simultaneously. A trigger signal was sampled which indicated the occurrence of stimulus pulses.

EMG Recordings and Processing

EMG recordings were made with intramuscular bipolar straight wire electrodes and a surface reference electrode. Signals were amplified and bandpass filtered (10 Hz - 2 KHz) prior to sampling at 3048 Hz. Offline processing included subtraction of offsets, rectification, and removal of stimulus artifacts, followed by digital lowpass filtering at 100 Hz. Chapter 2 describes these techniques in further detail.

Electrical Stimulation

Able bodied subjects only received stimulation to the EDC (Chapter 2). Hemiplegic subjects received simultaneous stimulation of the EDC and the ulnar nerve. Preliminary

experiments with six hemiplegic subjects indicated that in order to achieve functional opening of the fingers (extension of both MP and IP joints), both the EDC and the ulnar nerve must be stimulated. Stimulation of the ulnar nerve at the wrist produces extension of the finger IP joints, and flexion of the MP joints. By adjusting the stimulation applied to the EDC (which extends the MP joints), it is possible to achieve net extension at the MP joints. Pairs of surface electrodes (1.25 inches in diameter) were placed over the muscle belly of the EDC and the ulnar nerve at the wrist. Typical separation between the edges of the anode and cathode was 1 cm (anode proximal to cathode). An EMPI Focus stimulator (overhead voltage of 120 V) was used to determine the optimal electrode locations. Since these stimulators could not be triggered, laboratory stimulators were used during the experiment (these stimulators had a lower overhead voltage, which limited stimulus amplitudes).

All able bodied subjects were stimulated at a constant amplitude of 20 mA, and pulse widths were varied in multiples of the motor threshold pulse width (Th). For the hemiplegic subjects, the current was initially set to 20 mA. The pulse widths were then increased until a suitable motor response was obtained. For the EDC this was extension of the MP joints, and for the ulnar nerve it was extension of the IP joints. EDC stimulation was increased if needed in order to achieve net MP extension during simultaneous ulnar nerve stimulation. If a suitable motor response was not obtained before the maximum pulse width was reached (210 μ sec), the pulse width was fixed near 200 μ sec and the amplitude was increased until the desired motor response was achieved (the highest amplitude used was 50 mA). All subjects (both able bodied and hemiplegic) were stimulated at a frequency that provided a fused contraction of the EDC, usually between 15 and 25 Hz.

Joint Angle Measurement

During some of the experiments with able bodied subjects, and all of the experiments with hemiplegic subjects, a Penny and Giles® joint angle sensor was used to measure extension of the MP joint of one finger. The sensor was calibrated so that zero degrees MP flexion/ extension corresponded to zero degrees, 90 degrees of MP flexion corresponded to -90 degrees, and MP hyperextension produced a positive angle. The sensor was placed on a finger that was extended well by the stimulation, and was also activated by low level stimulation through all the recording electrodes (indicating that the recording electrodes were recording activity from muscle fibers acting on that finger). All trials were videotaped to provide a qualitative measure of extension of all other finger joints.

Grasp Pressure Measurement and Subject Positioning

Subjects were seated with their impaired arm (hemiplegics) or dominant arm (able bodied subjects) resting in an open forearm cast with their arm in neutral pronation - supination. In the hemiplegic experiments, the subjects grasped a rubber cylinder connected to a pressure transducer which measured the pressure generated by the subject's hand grasp. Able bodied subjects gripped a wooden post. A strap was placed over the wrist to constrain the forearm during the stimulation. The EMG, trigger, MP joint angle, and grasp pressure signals were all sampled by a Macintosh IIfx computer at either 3012 or 3048 Hz. The computer also controlled the stimulation.

Experimental Protocol - Able Bodied Subjects

The goal of these experiments was to determine if reciprocal inhibition of finger

flexor muscles could be documented while stimulating a finger extensor muscle. In order to provide background EMG activity in the finger flexor muscles, the subject maintained a 10% maximal voluntary contraction (MVC) in his or her FDS while gripping the wooden post. The averaged EMG (time constant = 2 sec) from this muscle was displayed on an oscilloscope screen, and by keeping this visual feedback signal at a designated position on the screen, the subject was able to maintain a constant EMG magnitude contraction. The fact that subjects were able to do this without visual feedback suggests the subjects were generating a constant effort contraction.

Each trial consisted of 11 periods 12 seconds in length; periods alternated between electrical stimulation and no stimulation. Data was collected for three seconds before and after the transitions (stimulation to no stimulation, and no stimulation to stimulation, six seconds total). The subject was instructed "do not react to the stimulation" (Appendix B). Two seconds prior to the stimulation the visual feedback was removed from the display, and was not returned until two seconds after the stimulation ended (Figure 3.1). Visual feedback was removed because I did not want the subject to compensate for any change of the EMG which might result from the stimulation (for the first three subjects tested, visual feedback was removed only during the stimulation).

A total of 14 trials were performed, each separated by a four minute rest period. Two trials at the same stimulation level were conducted in succession which provided a total of ten transitions from no stimulation to stimulation, and ten transitions from stimulation to no stimulation at each pulse width. The stimulation levels used were 0, .98, 1.0, 1.5, 2.0, 3.0, and 5.0 x Th. Some of the subjects were not stimulated at .98 x Th because their muscle had already reached threshold at the lowest pulse width our

Figure 3.1 Timing diagram for experiments with able bodied subjects.

stimulators could generate ($8 \mu\text{sec}$). For those subjects we treated $8 \mu\text{sec}$ as the motor threshold. The sham trials with no stimulation ($0 \times \text{Th}$) were conducted to assess if the subjects could maintain a constant effort contraction in the absence of stimulation.

Able bodied subjects also participated in one trial assessing their ability to voluntarily extend and then flex their fingers. The trial consisted of 11 periods 12 seconds in length. Subjects were initially relaxed; a lighted display then instructed them to alternately extend and then flex their fingers. Subjects were instructed to make a medium strength fist when asked to flex. No instructions were given on how quickly to make the movements. Again, data was only collected for three seconds before and after the transitions from flexion to extension or extension to flexion.

Data Analysis (Able Bodied Subject Data)

1. EMG Magnitude Comparison, Pre- Versus Post-Transition:

Time averages of the flexor EMGs were computed pre- and post-transition over 250 msec and 500 msec intervals. The hypothesis was that there would be a decrease in the flexor EMGs during extensor stimulation if reciprocal inhibition was prominent. A paired t-test ($N=10$) was performed to determine if statistically significant differences existed in the EMG levels pre- versus post-stimulation. Trials with visible stimulus artifacts were excluded from the analysis (subject MH: trials at 3 x and 5 x Th).

2. Interpulse Interval Ensemble Averages:

Interpulse interval (IPI) ensemble averaging was also performed on the EMG signals. Ensemble averages were computed over the entire trial by taking the EMG data between each stimulus pulse (interpulse interval) and lining them up in time beginning at the stimulus pulse. This created M intervals of N data points. The EMG at each point in time (data points 1 through N) was then averaged across all M intervals of data. As an illustration, the value at point N in the ensemble average is the average of M data points at point N in each interval. The ensemble average depicts the time course of the average EMG response between each stimulus pulse. Ensemble averaging was done for the EMG from each muscle.

3. Transition Ensemble Averages:

At each stimulation level, the EMG from each of the ten transitions from stimulation to no stimulation (and vice versa) were averaged together, starting one second before the transition and ending one second after the transition. These ensemble averages were used to detect voluntary intervention by the subject.

Experimental Protocol - Hemiplegic Subjects

Subjects participated in a minimum of ten trials, each trial consisting of nine alternating periods 15 seconds in length, with alternating conditions and instructions to the subject. In some subjects a tenth period was added in which the subject was instructed to relax. Each trial was followed by a rest period of approximately six minutes in order to avoid muscle fatigue. A lighted display placed in front of the subject instructed the subject to **relax**, **flex** the fingers, or **extend** the fingers during the trials. The experimenter simultaneously gave the instruction verbally to the subject (Appendix B). Each trial began with a period in which the subject was instructed to relax.

Spastic stroke patients frequently have preserved finger flexion but may lack finger extension. A neuroprosthesis for patients with these voluntary motor capabilities could be implemented in three ways: 1) the neuroprosthesis provides finger extension (patient relaxed), and the patient voluntarily flexes her fingers; 2) the neuroprosthesis provides finger extension while the subject assists voluntarily, and the patient voluntarily flexes her fingers; or 3) the neuroprosthesis provides both finger extension and finger flexion while the patient is relaxed. Trials assessing the ability of FES to extend the fingers (below) are meant to reproduce the first two of these scenarios. The third scenario is not tested in this study. All trials, including those assessing the subject's voluntary motor capabilities, are described below:

Trials Assessing Subject's Voluntary Capabilities:

- 1) **Voluntary flexion alternating with relaxation.** Documents the subject's ability to flex the fingers voluntarily and to relax the muscles after flexion.
- 2) **Voluntary extension alternating with relaxation.** Documents the subject's

ability to extend the fingers voluntarily and to relax the muscles after extension.

3) **Voluntary extension alternating with voluntary flexion.** Documents the subject's ability to make alternating flexion and extension movements (which would be used, for example, in a functional task such as acquiring and then releasing an object).

Trials Assessing Ability of FES to Extend the Fingers Under Three Conditions:

4,5) **Extensor stimulation alone alternating with relaxation** (subject remains relaxed throughout the trial). Repeated once. Documents the effects of electrical stimulation on spasticity and hand opening while the subject remains relaxed.

6,7) **Extensor stimulation with simultaneous voluntary extension alternating with relaxation.** Repeated once. Documents the effects of electrical stimulation on spasticity and hand opening while the subject attempts to voluntarily assist finger extension.

8) **Extensor stimulation alone (subject remains relaxed) alternating with voluntary flexion.** Documents the effects of electrical stimulation on spasticity and hand opening while the subject remains relaxed but has just voluntarily flexed, which could occur in the performance of a functional task.

9) **Extensor stimulation with simultaneous voluntary extension alternating with voluntary flexion.** Documents the effects of electrical stimulation on spasticity and hand opening while the subject attempts to voluntarily assist finger extension but has just voluntarily flexed, which could occur in the performance of a functional task.

Repeated Trial Assessing Changes Over Time:

10) **Extensor stimulation alone alternating with relaxation** (subject remains relaxed throughout the trial).

The protocol used in these experiments is detailed in Appendix C.

Data Analysis (Hemiplegic Data)

1. Visual Examination of Patterns of Muscle Activity:

Plots of the EMGs, grasp pressure, and MP extension signals were constructed for each trial. These were examined to determine the patterns of muscle activity in each

stimulation paradigm, and how this influenced MP joint angle and grasp pressure. For plotting purposes only, the data was lowpass filtered at 10 Hz and decimated by a factor of 10 prior to plotting. Note that data was not collected during the middle one second of each period (data was transferred from RAM to the hard disk during this time). Most graphs only show data from two seconds prior to and following the transition from relaxation to voluntary effort or stimulation, and vice versa.

2. Calculation of Reaction Time During Voluntary Movements:

Hemiplegic subjects appeared to take much longer in making voluntary movements as compared to able bodied subjects. In order to quantify these differences, subject reaction times were calculated during the trials in which the subject was initially relaxed and was then asked to voluntarily flex (or extend) his or her fingers (trials 1 and 2). There were four transitions from relaxation to extension, and four transitions from relaxation to flexion for each hemiplegic subject.

The time taken by subjects to activate all three muscles (FDS, FDP, EDC) after being given the command to “flex” or “extend” was calculated. Reaction time was defined as the shortest of these three times. If muscles were activated inappropriately, the reaction time in response to the command “extend” might be the time taken to activate a flexor muscle.

The following algorithm was used to compute the time to activate a single muscle: A 200 msec time average of the muscle’s EMG (baseline) was computed approximately 55 msec prior to the command to “flex” or “extend” (7.055 seconds into a period). Next, time averages of the EMG within a sliding 100 msec window were computed. When the value within this 100 msec window exceeded a detection threshold, the time

corresponding to the center of this 100 msec window was defined as the muscle activation time (Figure 3.2). If the baseline value was less than $1.0 \mu\text{V}$, the detection threshold was $3 \times \text{baseline}$, otherwise the detection threshold was $2 \times \text{baseline}$. The larger detection threshold was used when EMG amplitudes were low in order to avoid erroneous detection of muscle activation. In some recordings of subject LMM, the background noise level was large and the EMG amplitude was small, and a detection threshold of $2 \times \text{baseline}$ could not detect muscle activation. In these instances, the detection threshold was set at $1.3 \times \text{baseline}$. In some muscles no reaction time was found.

The window widths (200 and 100 msec) and the detection thresholds (2, 3, $1.3 \times \text{baseline}$) were selected based on processing only a subset of the trials (roughly half the trials from one hemiplegic subject, and a few trials from two others). In these trials the reaction time could be identified clearly by visual inspection. These parameters gave the best indication of reaction times over this subset of trials. In some instances the reaction times were earlier or later than those identified visually; however, these values provided the best results on average.

Able bodied subjects performed one trial of alternating voluntary extension and voluntary flexion. The reaction time for each subject to make the transition from relaxation to extension was calculated, and a mean reaction time for six of the able bodied subjects was computed. Able bodied subjects had very abrupt EMG changes when making the transition from relaxation to extension, and detection thresholds were adjusted according to the following rules in order to detect muscle activation well (these criteria were selected based on processing data from a few of these subjects).

$$0.0 < \text{baseline} \leq .10 \qquad \text{detection threshold} = 10 \times \text{baseline}$$

Figure 3.2 Detection of muscle activation time. The top plot shows the EMG in the FDP muscle (file tcboth.2, period 8). The bottom plot shows the output of the 100 msec sliding window. In this example the baseline is $12.5395 \mu V$. The muscle activation time is defined as the time when the window average is greater than $2 \times \text{baseline}$ ($25.08 \mu V$). Note that the muscle activation time occurs just prior to when the firing rate increases.

.10 < baseline $\leq$.50 detection threshold = 5*baseline

.50 < baseline detection threshold = 2*baseline

As with the hemiplegic subjects, when the baseline is small, large detection thresholds are needed to detect true increases in EMG levels. Otherwise, random variations may be detected as muscle activation.

A one-sample sign test was performed comparing the reaction time for the hemiplegic subjects to make the transition from relaxation to extension (4 observations per subject) versus the mean reaction time for the able bodied subjects. A Mann-Whitney test was performed to assess if an individual hemiplegic subject had different reaction times when making the transition from relaxation to extension versus the transition from relaxation to flexion.

3. Assessment of IP and MP Extension:

Videotapes recorded during the experiments were viewed in order to qualitatively document the IP extension of all fingers during extensor stimulation, and how this extension was affected by voluntary effort. Since MP joint angle was only measured on one finger, the videotape was also analyzed to determine which fingers were extended at the MP joint by the extensor stimulation.

4. Assessment of Influence of Simultaneous Voluntary Extension on MP Extension During Stimulation:

Most hemiplegic subjects had decreased MP extension when they tried to voluntarily assist finger extension. In order to quantify this, the MP joint angle (position) prior to the start of each stimulation period (P_{before}) was calculated by taking the time average of

the position recording over 0.5 seconds prior to the start of stimulation. The MP angle at the end of each period of stimulation (P_{end}) was calculated by taking the time average over the last 0.5 seconds of stimulation. Analysis of covariance (ANCOVA) was performed within each subject to assess if the factor **treatment** (extensor stimulation alone or extensor stimulation with simultaneous voluntary extension), or the factor **block** (one or two) affected the dependent variable P_{end} , the amount of MP extension achieved at the end of each period of stimulation. The factor block represents the block of four stimulation periods in each trial of a given treatment. There were two blocks per treatment. The covariate in the analysis was P_{before} . The MP joint angle prior to the start of each stimulation period was not fixed in these experiments; adding the covariate to the analysis controlled for the variability in P_{before} when assessing if either of the factors affected the dependent variable.

When calculating the ANCOVA, the three-way interaction term, followed by all two-way interaction terms as a group, were removed from the analysis since they were not significant ($p > .05$). The covariate P_{before} was also removed from the analysis ($p > .05$) if either factor (**block** or **treatment**) was not significant at this point (it only needed to be removed for subject TC). Because of limited degrees of freedom, it was necessary to remove terms when appropriate so that statistically significant results could be achieved.

5. Assessment of Influence of Voluntary Flexion on Subsequent MP Extension During Stimulation:

Voluntary finger flexion can limit subsequent MP extension. In order to assess the influence of alternating voluntary flexion, a one factor ANCOVA was performed within

each subject to determine if the factor **treatment** (either extensor stimulation alone or extensor stimulation alone alternating with voluntary flexion) affected the dependent variable P_{end} . The covariate in the analysis was P_{before} . Another one factor ANCOVA was performed with **treatment** being either extensor stimulation with simultaneous voluntary extension or extensor stimulation with simultaneous voluntary extension alternating with voluntary flexion. The purpose of the ANCOVA was to determine how voluntary flexion affected subsequent MP extension. Thus only the second, third and fourth stimulation periods were compared in the analysis since relaxation preceded the first stimulation period. Only one trial was performed of each of the paradigms with voluntary flexion. Thus only one trial of each of the paradigms with alternating relaxation were used in the ANCOVAs. The trial which was selected was the one which occurred closest in time to the appropriate alternating voluntary flexion trial. Trials 10 and 8 and trials 7 and 9 were paired. Interaction terms and the covariate were removed from the analyses according to the conditions previously stated.

6. Assessment of EMG Magnitudes Pre- Versus Post-Stimulation:

The increase in flexor EMGs during stimulation often persists after the stimulation ends. It was hypothesized that the flexor EMGs were significantly larger after stimulation ended versus levels prior to the start of stimulation. To verify this, time averages of FDS and FDP EMGs were computed over a one second interval immediately preceding the start of stimulation, and over two other one second intervals after the stimulation had ended for all trials of extensor stimulation alone. One interval started 100 msec after the stop of stimulation, and the other interval started five seconds

after the stop of stimulation. Position (MP joint angle) averages were computed over the same time intervals. Paired t-tests were performed to compare flexor EMG levels pre- versus post-stimulation.

Three separate tests were conducted to assess the correlation between the change in flexor EMG and the change in MP joint angle from pre- to post-stimulation. A linear regression was performed and the correlation coefficient computed. Two nonparametric tests (Spearman's rho and Kendall's tau (which compute rank correlation coefficients)) were also performed since the normalcy of the data was not known. If p values for the correlation coefficients computed in two of three tests were significant, a significant correlation between EMG change and position change was assumed.

7. Documentation of the Warmup Effect:

The rate of finger extension was not always constant depending on the stimulation period within a block (one through four). Finger extension appeared to occur more slowly in some periods, and this slow extension was named the "warmup effect." In order to quantify the warmup effect, the position waveform (MP joint angle) from each extensor stimulation alone trial was filtered at 10 Hz, and the largest change in position during each period of stimulation was calculated (the initial position was taken as the position value at the time of occurrence of the first stimulus pulse). The time required for the finger to extend to 50% of this total position change was defined as the half rise time (Figure 3.3).

Figure 3.3 Calculation of the half rise time (time to reach 50% of the largest change in position). Shown is one portion of the position recording from trial `tcbth.5` (portions of three stimulation periods are shown). The discontinuity near the maximum position is present because of a one second gap in data collection.

8. Statistical Tests Used in this Study:

Nonparametric tests were used to analyze subject reaction times since it was not known if reaction times were normally distributed. Paired t-tests were used on the data consisting of EMG time averages. Since each EMG average was computed from over 150 samples, the means (averages) are normally distributed by the central limit theorem (Daniel, 1987). The requirement of a near normal distribution for using a t-test was thus met. ANCOVA has no nonparametric equivalent, so no alternative test was available for use. The correlation between the change in flexor EMG and change in MP joint angle was assessed by calculating both parametric (regression) and nonparametric (Spearman and Kendall) correlation coefficients, since normalcy of the position data was not known.

RESULTS

Experiments with Able Bodied Subjects

Tests on able bodied subjects were performed to determine the effects of finger extensor (EDC) stimulation on the EMGs of finger flexor muscles (the FDS and FDP). If stimulation of the EDC causes reciprocal inhibition of the finger flexor muscles as hypothesized, then the flexor EMGs should decrease during stimulation. The following results pertain to the FDS muscle, from which the majority of the data was collected. A separate section describes the results obtained from the FDP muscle.

1. Trials with No Stimulation (Sham Trials):

Sham trials assessed the ability of a subject to maintain a constant effort contraction in the absence of stimulation while visual feedback was removed. Five subjects performed these trials, and all were able to maintain a constant effort contraction in the FDS muscle (paired t-tests comparing EMG time averages computed before and after a fixed period in time indicate no significant difference in values). Figure 3.4 shows a transition ensemble from one subject during a sham trial; the EMG magnitude remains relatively constant.

2. Transition Ensembles:

Transition ensembles were used to detect voluntary intervention (i.e. the subject did not maintain a constant effort contraction). Figure 3.5 shows the transition ensemble from one subject during a trial with stimulation at $2 \times Th$. Transition ensembles that showed a long (150 - 200 msec) latency change in EMG, such as this one, were taken

Figure 3.4. Transition ensemble during a sham trial (no stimulation), subject SP. The EMG is from the FDS muscle (files spsham.0 and spsham.2 were ensemble averaged together). The transition occurred at 1.0 seconds (i.e. at the tip of the arrow). Visual feedback was removed during this entire period of time.

Figure 3.5 Transition ensemble indicating subject voluntary intervention during stimulation (subject PC, files pcedc.5 and pcedc.6). Stimulation pulse widths were $2 \times Th$. The transition from no stimulation to stimulation occurred at 1.0 seconds.

to indicate possible voluntary intervention, since it was assumed that a spinal reflex would have a short latency, i.e. 20 msec for disynaptic reciprocal inhibition (based on 60 m/sec conduction velocity and a distance from the extensor to the spinal cord of 60 cm). All trials in which voluntary intervention was suspected were excluded from EMG magnitude comparisons (subject PC: trials at 2, 3, and 5 x Th; subject LM: trials at 5 x Th). Transition ensembles were computed for all subjects except MH and ML (neither of these subjects had repeated trials at each stimulation level, therefore there were not enough stimulation periods to ensemble average).

3. EMG Magnitude Comparison, Pre- Versus Post-Transition:

Flexor EMG magnitude comparisons (pre- versus post-transition) were performed across both 250 and 500 msec time periods to assess the influence of EDC stimulation on finger flexor EMGs. Prominent changes were not evident in the FDS EMG at the transitions from stimulation to no stimulation and vice versa (Table 3.4). The changes were inconsistent both within and across subjects. Increases, decreases, and no change in EMG magnitudes were all seen. The values in Table 3.4 are pooled for both types of transition (stimulation to no stimulation and vice versa), and for all stimulation levels. Seven subjects were studied; when results of the two time averages are also pooled, a statistically significant decrease in FDS EMG during stimulation (t-test, $p < .05$) was noted in subjects MH and SP in up to 30% of the transitions. Subjects ML and PC showed both increases and decreases in up to 25% of the transitions. Subjects WG, AS, and LM had only increases in up to 62.5% of the transitions. If percentages are averaged across subjects, increases were seen in 20.8% of the transitions, and decreases were seen in 12.6% of the transitions.

Subject	EMG change	FDS		FDP	
		250 msec	500 msec	250 msec	500 msec
MH	increase	0/8	0/8	--	--
	decrease	2/8	2/8	--	--
ML	increase	1/12	1/12	--	--
	decrease	0/12	1/12	--	--
PC	increase	0/4	1/4	--	--
	decrease	1/4	1/4	--	--
WG	increase	2/10	3/10	0/10	0/10
	decrease	0/10	0/10	0/10	3/10
SP	increase	0/10	0/10	--	--
	decrease	0/10	3/10	--	--
AS	increase	4/10	2/10	--	--
	decrease	0/10	0/10	--	--
LM	increase	5/8	5/8	0/4	0/4
	decrease	0/8	0/8	0/4	0/4

Table 3.4 Results of EMG magnitude comparisons for able bodied subjects. Increase (or decrease) refers to an increase (or decrease) in EMG magnitude during extensor stimulation compared to the magnitude during no stimulation. Ratios represent the number of transitions with an increase or decrease (all stimulation levels and both types of transition are pooled together).

If the results are grouped together for all subjects but separated over the two time intervals, there were the same number of increases detected with both intervals, but decreases in FDS EMG during stimulation were detected twice as frequently with the

500 versus the 250 msec interval. When the results are separated by the type of transition, there were approximately the same number of increases and decreases in EMGs for both types of transition (250 msec interval only). When results for all subjects are separated by stimulation level, EMG increases were noted at all stimulation levels except .98 x Th (only used with two subjects). EMG decreases were only seen at the three lowest stimulation levels (.98, 1.0, and 1.5 x Th).

4. Interpulse Interval Ensemble Averages:

Inspection of the IPI ensemble averages reveals a broad peak beginning at approximately 30 msec and reaching maximum amplitude at 50-60 msec in the muscles of some subjects (Figure 3.6). It is hypothesized that this peak in EMG is a phasic response to muscle stretch, as will be explained in the discussion section. Some subjects were stimulated at 25 Hz, making the IPI only 40 msec in length. For these subjects it was not possible to determine the presence or absence of this peak. In subjects in which a peak was observed, its amplitude usually increased as the stimulus pulse width increased.

5. Effect of EDC Stimulation on the FDP EMG:

The effect of EDC stimulation on the FDP muscle was also assessed. In two subjects I was able to record successfully from the FDP muscle (a variety of problems were encountered in the other five subjects including stimulus artifacts in the FDP recordings (MH), not being able to implant the electrode (ML, SP), subject not maintaining a constant effort contraction (PC), or no voluntary EMG present in the FDP during stimulation (AS)). Subject LM had a few transitions in which there was no

Figure 3.6. IPI ensemble average indicates the presence of a broad peak (subject AS, file asdc.7.new2)

voluntary FDP activity, and these were excluded from the analysis.

Of the two subjects with successful FDP recordings, one subject had a statistically significant decrease in FDP EMG during stimulation (t-test, $p < .05$) in 30% of the transitions (500 msec interval only); for the other subject there was no change in FDP EMG during stimulation.

Experiments with Hemiplegic Subjects

A) Voluntary Motor Control Capabilities

Hemiplegic patients with spasticity frequently exhibit the characteristics of slowness of movement (Twitchell, 1951; Sahrmann and Norton, 1977), inappropriate muscle activation and cocontraction of antagonists (Dimitrijevic and Nathan, 1967; Mizrahi and Angel, 1979; Hammond et al., 1988; Bourbonnais et al., 1989), resting motor unit

activity (Dimitrijevic and Nathan, 1967; Young and Wiegner, 1987), and inability to extend their fingers (Carey, 1990). This section documents the voluntary motor capabilities of the hemiplegic subjects who participated in this study. The findings presented here are not new, but help explain each subject's motor responses to the various extensor stimulation paradigms used in this study.

1. Clinical Measures of Subject Impairment:

The results of the clinical testing of hemiplegic subjects are listed in Appendix D. All subjects had spasticity present in many upper extremity joints (Ashworth scores greater than one), and all subjects had hyperactive reflexes (score of three or four). In general, spasticity (Ashworth) scores changed little pre- to post-stimulation.

2. Resting EMG:

Figure 3.7 shows EMG recordings in a spastic subject who was instructed to relax. This particular subject had resting motor unit activity in both finger flexor and extensor muscles. Figure 3.8 shows EMG recordings in a second subject who was yawning, which is known to cause an increase in muscle tone in some spastic subjects. There was a large increase in flexor EMG which persisted for several seconds. There was also a brief increase in EDC activity during the yawn.

3. Voluntary Control:

The following figures show one portion of a longer trial. In each trial, the subject was instructed to flex and/or extend his or her fingers four times.

Voluntary Finger Flexion: All six subjects voluntarily activated their finger flexor

Figure 3.7 EMG recordings in finger flexor and extensor muscles; subject was instructed to relax (subject HM: file hmboth.6, period 1).

Figure 3.8 Increase in finger flexor and extensor EMGs when subject yawned (subject EB: file ebboth.7, period 8).

muscles when given the command to flex. Finger flexor activation caused flexion of the MP joint (indicated by a decrease in the position trace, Figure 3.9) and an increase in grasp pressure. All subjects activated both the FDS and FDP muscles during finger flexion except subject TC, who activated only the FDP muscle (Figure 3.10). Some subjects had cocontraction of the EDC during voluntary flexion (always seen in able bodied subjects, Figure 3.12), but subjects TC, LMM, and HM had little or no EDC activation.

Subjects TC, CR, LMM, and EB also had forearm pronation during finger flexion (mass activation). All subjects had delays in producing finger flexion, and most had difficulty relaxing their finger flexors when told to relax. Subject HM inappropriately activated his EDC when attempting to relax. Another example of voluntary finger flexion is shown in Appendix E.

Voluntary Finger Extension: Only one of six subjects had voluntary finger extension (subject HM), but he could not consistently extend his fingers. The remaining subjects inappropriately activated their finger flexor muscles while trying to extend, which caused finger flexion and an increase in grasp pressure. Based on calculation of reaction times (next section), in 19 of 20 transitions from relaxation to finger extension, hemiplegic subjects activated the FDS or FDP muscle first. This contrasts with able bodied subjects: five of six activated their EDC first when making the transition from relaxation to finger extension.

Figure 3.11 shows a typical example of a hemiplegic subject making the transition from relaxation to finger extension: the subject had slight activation of the EDC, but FDP activation was much larger and net finger flexion resulted. The FDS had only sporadic activity at the start of extension. The subject took approximately four seconds

Figure 3.9 Typical example of voluntary finger flexion in a hemiplegic patient. The command to flex was given at 7 seconds, the command to relax occurred at 22 seconds. Both the EDC and FDP EMGs increased slowly; large activation occurred three seconds later at which time the FDS was also activated. This subject had little ability to relax the flexor muscles after voluntary flexion, but was able to relax the extensor muscle (subject CR: file crboth.1, periods 4 and 5).

Figure 3.10 Voluntary finger flexion in another hemiplegic patient. The command to flex was given at 7 seconds, the command to relax occurred at 22 seconds. This subject had virtually no EDC cocontraction during flexion, and the FDS remained silent throughout this entire trial. The subject had strong activation of the FDP when instructed to flex, but relaxed this muscle slowly (subject TC: file tcboth.1, periods 2 and 3).

to activate any muscles, and was not able to relax the FDP muscle following finger extension. This pattern of muscle activation was common among hemiplegic subjects. In addition, subjects TC, CR, and LMM had forearm pronation during voluntary finger extension.

For comparison, Figure 3.12 shows EMGs from an able bodied subject performing a trial of alternating voluntary extension and voluntary flexion. The subject was initially relaxed, then made the transition from relaxation to finger extension with a delay of less than 0.5 seconds. The flexor muscles were virtually silent during finger extension, while the EDC was highly active. The upward deflection in the position trace indicates MP joint extension.

One hemiplegic subject had voluntary finger extension capabilities. Figure 3.13 shows data from subject HM making the transition from relaxation to finger extension. Although both finger flexor muscles were activated, this subject also activated the EDC, and slight finger extension was achieved. However, this subject was not always able to extend his fingers on command. The pressure increase during finger extension may indicate that either his thumb or other fingers were flexing and not extending, meaning he doesn't have voluntary control over all his digits. Appendix E contains further examples of trials of voluntary finger extension.

Voluntary Finger Extension Alternating with Voluntary Finger Flexion: This sequence of movements would be used in order to first acquire an object, then grasp it, and finally release the object. Only subject HM was able to successfully perform these movements. A typical example from the other subjects is shown in Figure 3.14. The subject could flex her fingers after trying to extend them (pressure increases and position decreases), but when trying to extend her fingers again, the flexor EMGs

Figure 3.11 Typical example of voluntary finger extension in a hemiplegic subject. The command to extend was given at 7 seconds, the command to relax occurred at 22 seconds. Typical example of inappropriate muscle activation when the subject attempted to extend his fingers (subject EB: file ebboth.3, periods 4 and 5).

Figure 3.12 Able bodied subject voluntarily extending his fingers. The command to extend was given at 3 seconds (a command to flex was given at 15 seconds following the command to extend). Note the abrupt onset of extensor EMG and the silence in the finger flexor muscles (subject: AS, file asvol.1, periods 2 and 3). There was a six second gap in data collection.

Figure 3.13 Voluntary finger extension in a hemiplegic subject who could extend his fingers. The command to extend was given at 7 seconds, the command to relax occurred at 22 seconds. Although the subject could extend his fingers, there was an activation delay of approximately three seconds (subject HM: file hmboth.5, periods 8 and 9).

Figure 3.14 Typical example of alternating voluntary finger extension and finger flexion in a hemiplegic subject. The subject was initially attempting to extend her fingers. The command to flex was given at 7 seconds, and the command to resume extension occurred at 22 seconds. Note that the subject could not make the transition from finger flexion back to extension; the fingers flexed slightly (subject LMM: file lmmboth.3, periods 3 and 4).

remained fairly constant, there was only a minor increase in EDC activity, and no finger extension occurred.

For comparison, Figure 3.15 shows the results of a similar trial performed by an able bodied subject. During the initial extension the flexor EMGs were silent, the extensor EMG was large, and the MP joint was fully extended (zero degrees). When told to flex, the extensor EMG had a brief off period during which the flexor EMGs increased; the EDC then became active but at a low amplitude (cocontraction). The fingers flexed approximately 70 degrees. When asked to extend, the EDC EMG amplitude increased rapidly, and the flexors became silent quickly. The MP joint was again fully extended.

Reaction Time During Voluntary Movements: Data for able bodied subjects are limited to their reaction times in making the transition from relaxation to finger extension. The mean reaction time for able bodied subjects was 0.534 seconds (Table 3.5). Five of six subjects activated their EDC muscle first; the sixth subject activated her FDS muscle first, followed by the EDC muscle in 54 msec.

The mean reaction time for hemiplegic subjects making the transition from relaxation to finger extension was 2.707 seconds (range = 2.314 - 3.311 seconds) (Table 3.6), approximately five times longer than the reaction time for able bodied subjects making the same movements. Most subjects activated the FDP muscle first. A few subjects activated the FDS first in some transitions. One subject (LMM) activated the EDC muscle first in one transition. Subject JM did not activate any muscles in the four transitions within the trial. A one-sample sign test indicates that hemiplegic reaction times in making the transition from relaxation to finger extension were significantly longer than the mean reaction time for the same movement in able bodied subjects ($p <$

Figure 3.15 Alternating voluntary finger extension and finger flexion, able bodied subject. The subject was initially extending his fingers. The command to flex was given at 3 seconds, and the command to resume extension occurred at 15 seconds. There was a six second gap in the data collection (subject AS: file asvol.1, periods 3 and 4).

Subject	Muscle Activated First	Reaction Time (seconds)
ML	EDC	1.034
PC	EDC	.662
WG	EDC	.563
SP	EDC	.231
AS	EDC	.335
LM	FDS	.379
mean reaction time		.534

Table 3.5 Reaction times for able bodied subjects making the transition from relaxation to finger extension (one transition per subject).

Subject	Times Muscle Is Activated First			Mean Reaction Time (seconds)
	EDC	FDS	FDP	
TC	0	0	4	2.314
CR	0	0	4	2.444
LMM	1	1	2	3.311
HM	0	2	2	2.873
EB	0	0	4	2.592
JM	-	-	-	-
mean reaction time				2.707

Table 3.6 Reaction times for hemiplegic subjects making the transition from relaxation to finger extension (four transitions per subject).

.0001).

The mean reaction time for hemiplegic subjects making the transition from relaxation to finger flexion was 1.804 seconds (range = 1.297 - 2.310 seconds) (Table 3.7). All subjects activated a finger flexor muscle first except for subject JM, who activated the EDC first in two of four transitions. A Mann-Whitney test indicates that four of five hemiplegic subjects did not have significantly different reaction times in making the transition from relaxation to finger extension versus relaxation to finger flexion. This is not surprising since the muscle activation patterns were similar for finger flexion and extension in the majority of patients. The fifth subject (CR) had significantly longer reaction times for extension versus flexion ($p < .03$). Subject JM did not activate any muscles when asked to extend his fingers, thus reaction times were not compared for this subject.

Subject	Times Muscle Is Activated First			Mean Reaction Time (seconds)
	EDC	FDS	FDP	
TC	0	0	4	1.297
CR	1	0	3	1.350
LMM	0	1	3	1.939
HM	0	3	1	2.310
EB	0	1	3	2.250
JM	2	2	0	1.675
mean reaction time				1.804

Table 3.7 Reaction times for hemiplegic subjects making the transition from relaxation to finger flexion (four transitions per subject).

B) Effects of Extensor Stimulation

The goal of this research was to provide functional opening of the fingers in patients with spastic hemiplegia. In order to achieve this goal, stimulation was applied to both the EDC and the ulnar nerve. Because the patients had some voluntary control over finger movement, the effects of voluntary finger extension during stimulation and voluntary finger flexion prior to stimulation were also investigated to see if they would aid hand opening.

1. Finger Extension Achieved by Simultaneous Stimulation of the EDC and Ulnar Nerve:

IP extension was achieved in all six subjects, although the IP joints were not fully extended in subject CR. This subject found the ulnar nerve stimulation to be painful, and stimulation levels were decreased to tolerable levels (which did not provide full IP extension). Four of six subjects achieved simultaneous extension of both IP and MP joints. In most subjects only three fingers were extended at the MP joint (the index finger often had little or no MP extension).

Figure 3.16 displays MP joint angle (position) plots for extensor stimulation alone trials for all six subjects. Subjects CR, EB, and JM (Figures 3.16 (a), (b), and (c), respectively) achieved nearly full extension or slight hyperextension of the MP joint (as did subject TC based on visual observation). Subjects EB (b), LMM (d), and most notably TC (g), had initial MP movement in the flexion direction followed by extension in at least some of the stimulation periods. It appears that the finger intrinsic muscles innervated by the ulnar nerve have faster contraction times compared to the EDC muscle in these subjects. The intrinsics produce initial MP flexion before the EDC can

Figure 3.16 Position plots during trials of extensor stimulation alone (subjects were relaxed throughout the entire trial). Zero degrees MP flexion/ extension corresponds to zero degrees and 90 degrees MP flexion corresponds to -90 degrees. Plots are from the second trial of extensor stimulation alone except for subject LMM ((d) - the first trial is shown since even poorer extension was obtained in the second trial). (a) subject CR: file crboth.5, (b) subject EB: file ebboth.8, (c) subject JM: file jmboth.6, (d) subject LMM: file lmmboth.6 (stimulation periods were 20 seconds in length), (e) subject HM: file hmboth.9 (angle sensor on MP joint), (f) subject HM: file hmboth.16 (angle sensor on PIP joint), (g) subject TC: file tcboth.5. The offset of the joint angle sensor was not measured for subject TC, thus her position values can only provide information on relative changes in position. Observations made during the trials indicated that she achieved very good MP extension. Jumps in the position signal (c) were due to noise. Discontinuities in the middle of a stimulation period (e.g. the first stimulation period in (f)) were due to a one second gap in data collection.

counteract this and provide net MP extension.

Subject LMM (d) had only a few degrees of MP extension; it was even less in the second trial of extensor stimulation alone. The joint angle sensor was placed on the long finger (the only finger with EMG recordings from all three muscles), but videotapes indicate that the MP extension of the ring and small fingers was large in trials of extensor stimulation alone. Finger extension achieved with an EMPI Focus stimulator prior to the actual experiment (not quantified) appeared to be large enough to allow her to grasp an object such as a glass (all fingers extended well).

Subject HM had net MP flexion during extensor stimulation (e). EDC stimulation alone provided good extension at the MP joint. However, the ulnar nerve stimulation provided very strong MP flexion, and could not be counterbalanced by the EDC stimulation when the two sites were stimulated simultaneously. Thus extensor stimulation produced net MP flexion in this subject. A trial was conducted with the joint angle sensor placed on his PIP joint. Figure 3.16 (f) indicates that the PIP joint achieved nearly full extension during extensor stimulation.

Note how most subject's fingers closed rapidly when the stimulation turned off. In contrast is subject TC, whose finger closed slowly when the stimulation stopped (g). Visual observation indicated that subject JM's IP joints remained very extended after the stimulation ended. For a few subjects in some stimulation periods (e.g. stimulation period one, Figure 3.16 (g)), it appears that further finger extension may have occurred if the stimulation period were longer than 15 seconds.

A third trial of extensor stimulation alone was conducted at the very end of the experiment to determine if there were changes in finger extension over time. For most subjects the stimulation provided good extension over many periods of stimulation. All subjects except EB had only a slight reduction in MP extension during this last trial as compared to the two earlier trials, suggesting that the EDC muscle had barely fatigued after all the trials with stimulation. Subject EB experienced a profound decrease in extension after the first two trials of extensor stimulation alone. Following these trials he had either slight MP extension or flexion during extensor stimulation.

2. The Warmup Effect:

The warmup effect refers to the slow rate of finger extension encountered in some periods of stimulation within a trial (see Figure 3.16 (b)). Half rise times for each stimulation period within a trial are shown in Figure 3.17, and were calculated for each trial of extensor stimulation alone (three per subject). Half rise times were not calculated for subjects LMM and HM (MP joint only) since little or no MP extension was achieved, but half rise times were calculated for the single trial documenting PIP extension (subject HM). Subject EB experienced EDC muscle fatigue during the experiment, and his third trial is not included in the data.

Figure 3.17 Half rise times for trials of extensor stimulation alone. The times indicate the time required for the finger to extend to 50% of the total position change. (a) subject TC, (b) subject CR, (c) subject HM, (d) subject EB, (f) subject JM.

The longest half rise times usually occurred in the first stimulation period within the trial. In some trials there were no apparent differences in the half rise times for the different periods of stimulation (e.g. Figure 3.17 (e), trial JM_6 (jmboth.6)).

3. Effects of Simultaneous Voluntary Extension on MP Extension During Stimulation:

It was hypothesized that simultaneous voluntary extension would actually impede rather than enhance the amount of finger extension produced by the stimulation since most of the subjects flexed their fingers when attempting to extend them. Figure 3.18 (a) displays the position waveform from a trial of extensor stimulation alone (subject relaxed), while Figure 3.18 (b) displays the position waveform from a trial when the subject was trying to voluntarily extend his fingers during the stimulation. When the subject was relaxed, stimulation extended the MP joint to nearly full extension (zero degrees). For this subject, the amount of finger extension was severely decreased during simultaneous voluntary effort; only a few degrees of MP extension were achieved.

Analysis of covariance was used to test the hypothesis that simultaneous voluntary extension would decrease the finger extension provided by the extensor stimulation. Subject EB was excluded due to EDC muscle fatigue. With the interaction terms (and the covariate for subject TC) removed from the analysis, four of five subjects had a significant decrease in MP extension during voluntary effort ($p < .03$), in accord with the hypothesis. Subject HM had a significant increase in MP extension during voluntary effort ($p < .01$). This subject was the only subject with voluntary finger extension. However, when measuring PIP extension, this subject had a significant decrease in PIP extension when voluntarily assisting the stimulation ($p = .0001$). The

Figure 3.18 Example of the effect of simultaneous voluntary extension on MP extension. In (a) the subject was relaxed during stimulation (subject CR: file crboth.5). In (b) the subject was trying to extend during the stimulation (subject CR: file crboth.6).

angle prior to the start of stimulation (the covariate, P_{before}) did not significantly affect the amount of MP extension achieved at the end of stimulation in all subjects ($p > .05$). The block number (the first or second of the two trials) also did not significantly affect MP extension (if fatigue had occurred between the first two trials of extensor stimulation alone, it is anticipated that block would have had a significant effect on P_{end}).

Analysis of videotapes indicate that for subjects LMM and TC, the influence of voluntary effort was different for different fingers. For example, subject TC had more flexion of the ring finger versus the long finger during simultaneous voluntary extension (the angle sensor was on the ring finger).

4. Effects of Voluntary Flexion on Subsequent MP Extension During Stimulation:

These trials were performed to determine how well the MP joint extends if the subject has just voluntarily flexed his or her fingers, which would occur when grasping an object. Based on visual observations during preliminary experiments, it was hypothesized that alternating voluntary flexion would impede the ability of electrical stimulation to extend the fingers after the subject has just flexed them. Figure 3.19 (a) displays the position waveform from a trial of extensor stimulation alone (subject relaxed throughout), while Figure 3.19 (b) displays the position waveform from a trial of extensor stimulation alone (subject relaxed) alternating with voluntary flexion. For this particular subject, the MP joint extended well in all periods of stimulation with the subject relaxed (Figure 3.19 (a)). With alternating voluntary flexion, the MP joint always extended to some degree during stimulation, but not as well as in the trial of extensor stimulation alone. The hand became progressively more flexed with each

Figure 3.19 Example of the effect of voluntary flexion on subsequent MP extension during stimulation (subject relaxed during stimulation). For this subject, alternating voluntary flexion hindered MP extension. In (a) the subject was relaxed during stimulation (subject CR: file crboth.11). In (b) the subject made alternating voluntary flexion movements (subject CR: file crboth.8).

subsequent period of voluntary flexion. Note that the MP joint did not extend as far in the first stimulation period of Figure 3.19 (b) (prior to any voluntary flexion) as it did in Figure 3.19 (a). It is not known what caused the difference in the amount of MP extension achieved; it would be expected to be approximately the same in the first stimulation period of both paradigms.

Analysis of covariance was performed to test the hypothesis that alternating voluntary flexion would decrease finger extension, and indicates that three of five subjects (CR, LMM, JM) had significantly less extension after voluntary flexion compared to similar trials in which the subjects were relaxed between stimulation periods ($p < .03$). Another subject (HM) had significantly more finger extension after voluntary flexion ($p < .01$). For these subjects, voluntary flexion had a significant effect on the amount of finger extension achieved. For subject HM (see Appendix F), the angle prior to the start of stimulation (the covariate, P_{before}) also had a significant effect on P_{end} . The covariate P_{before} was not significant ($p > .05$) in the other subjects. For the fifth subject TC, alternating voluntary flexion did not have a significant effect on the amount of finger extension achieved.

Having the subject assist the stimulation by simultaneous voluntary extension did not counteract the effects of alternating voluntary flexion. Figure 3.20 (a) displays the position waveform from a trial of extensor stimulation with simultaneous voluntary extension, while Figure 3.20 (b) displays the position waveform from a similar trial with alternating voluntary flexion. For this particular subject, the MP joint became more flexed in the trial with alternating voluntary flexion, compared to the trial with alternating relaxation.

Analysis of covariance was again used to test the hypothesis that alternating

Figure 3.20 Example of the effect of voluntary flexion on subsequent MP extension during extensor stimulation with simultaneous voluntary extension. For this subject, alternating voluntary flexion hindered MP extension. In (a) the subject was relaxed following stimulation (subject CR: file crboth.7). In (b) the subject made alternating voluntary flexion movements (subject CR: file crboth.9).

voluntary flexion would decrease finger extension, and indicates that three of five subjects (CR, HM, JM) had significantly less extension after voluntary flexion compared to similar trials in which the subjects were relaxed following stimulation ($p < .03$), while subject LMM (see Appendix F) had significantly more finger extension after voluntary flexion ($p < .05$). For these subjects voluntary flexion had a significant effect on the final position P_{end} . For the fifth subject TC, alternating voluntary flexion did not have a significant effect on the amount of finger extension achieved.

5. Effect of Extensor Stimulation on the EMG in Flexor Muscles:

It was originally hypothesized that simultaneous stimulation of the EDC and the ulnar nerve would reduce the resting motor unit activity in the spastic finger flexor muscles, both during the stimulation and after it ended. In contrast, stimulation frequently increased the flexor EMGs, and this increase sometimes persisted for many seconds.

In all the following figures, the increase in EDC EMG was due to M-waves and artifacts caused by the EDC stimulation. An example of what occurred in flexor EMGs during extensor stimulation alone (subject relaxed throughout the trial) is shown in Figure 3.21. This figure shows one portion of a longer trial containing four stimulation periods. In this subject, there was a large increase in FDP EMG soon after the start of stimulation; this increase was present for the duration of the stimulation, and persisted at a lower amplitude after the stimulation ended. Apart from an initial burst, there was only a minor increase in the FDS EMG during stimulation. Note the upward deflection of the position waveform, indicating that the MP joint was extending and thus the flexors were being stretched. There was also an increase in the subject's grasp pressure.

Figure 3.21 Example of flexor EMGs during extensor stimulation alone (subject TC: file tcboth.4, periods 8 and 9). The stimulation turns on at 7 seconds and turns off at 22 seconds.

All subjects showed an increase in the EMG of at least one flexor muscle during extensor stimulation, but not in all periods of stimulation. There was frequently a burst in the EMG of one of the flexors at the start of stimulation. One subject (EB) had clonic movement in his fingers coinciding with the onset of stimulation (see Fig. 3.23). These very strong repetitive stretch reflexes were initiated by the initial stretch to the finger flexor muscles. No other subject had finger clonus in response to the extensor stimulation. Appendix G contains figures which demonstrate the large variability of flexor EMGs in response to extensor stimulation. Some subjects also had forearm pronation during the stimulation.

Figure 3.22 shows EMGs during extensor stimulation alone on an expanded time scale. Stimulus pulses are superimposed on the extensor EMG recording. The motor unit firings in the FDP EMG were not time locked to the stimulus pulses. One large amplitude motor unit started firing when the stimulation began, and was joined by at least one more large amplitude motor unit after 8.5 seconds. In the FDS there was a single burst of very large magnitude. Another example is shown in Appendix G.

The increased flexor EMG was observed to outlast the period of stimulation. In order to quantify the observed increase in finger flexor EMGs post-stimulation, time averages of flexor EMGs were computed just prior to the start of stimulation and just after the stimulation ended. Paired t-tests indicate that three of six subjects had a significant increase in FDS EMG post-stimulation ($p < .02$) (subjects TC, CR, and JM). Two of these subjects had a significant increase in FDP EMG post-stimulation ($p < .002$) (subjects TC and JM). A tonic stretch reflex might be expected to produce a maintained increase in the flexor EMG if the muscle was at a longer length following the stimulation. However, only one subject (TC) showed a significant increase in MP

Figure 3.22 Example of flexor EMGs during extensor stimulation alone, expanded time scale (subject CR: file crboth.5, period 2). The stimulus pulses are plotted on top of the EMG. The first stimulus pulse indicates the onset of stimulation.

extension that correlated with an increased flexor EMG (FDP) post-stimulation (all three measures of correlation were significant ($p < .05$)). Another subject, HM, had a flexor EMG increase (not significant) correlated with a *decrease* in MP joint angle. No subject had a significant decrease in flexor EMG post-stimulation.

The EMG differences decreased with time post-stimulation. Time averages of flexor EMGs were also computed five seconds after the stimulation ended. Paired t-tests indicate that there was no significant difference in the EMG levels pre- versus post-stimulation (all six subjects).

6. Videotape Analysis: Activation of Thumb Adductor:

Large or moderate pressure increases during extensor stimulation were noted in all trials for subjects TC, LMM, and HM. These pressure increases were not due to finger flexion, but rather were due to thumb adductor activation and/ or forearm pronation. Videotapes indicate activation of the thumb adductor via the ulnar nerve stimulation in subjects TC and HM (the thumb adductor was not visible in the videotape for subject LMM, but strong forearm pronation during stimulation was evident). Subject TC also had strong forearm pronation and subject EB had minor forearm pronation during stimulation. Two subjects who had either no change in grasp pressure, or only small fluctuations during stimulation, did not appear to have any thumb adductor activation.

7. Variable EDC M-Waves During Extensor Stimulation:

Figure 3.21 displays typical EDC M-waves recorded during stimulation; their amplitude remains constant for the duration of the stimulation. However, EDC M-waves did not always remain constant during stimulation as might be expected. Figure

Figure 3.23 Variable EDC M-waves during stimulation (subject EB: file ebbboth.7, periods 2 and 3). The amplitude of the M-waves gradually increased during the stimulation and the MP joint extended slowly (the warmup effect; this was the first stimulation period in the trial). This subject also had large increases in both FDS and FDP EMG at the onset of stimulation. Finger clonus was evident when the stimulation first turned on (visual observation during the experiment). The FDS had only sporadic activity for the duration of the stimulation, and was quiet when the stimulation ended. The FDP had sustained activity during the stimulation, which persisted at a slightly lower amplitude when the stimulation stopped. There was little change in the subject's grasp pressure during stimulation.

3.23 displays a pattern of EDC M-waves seen frequently in subjects EB and LMM, and seen in only two periods in subject HM. This gradual rise in M-wave amplitude was not always associated with gradual finger extension as it is in this figure. Figure 3.24 displays an M-wave pattern (called a tent top) seen mostly in subject JM, but also seen in one stimulation period in subject LMM. It was also seen in data for a subject that is not reported here (subject CC).

The data in Figure 3.24 (a) was taken from a period prior to any voluntary finger flexion. The data shown in Figure 3.24 (b) was taken from the same trial, just after the subject had voluntarily flexed. Note how the amplitude of the M-waves was severely depressed following voluntary flexion, and how the MP joint was virtually motionless (small changes in the position waveform are only noise). Note also that there was continuing EMG activity in the FDS and FDP muscles.

Figure 3.24 (Following page) EDC M-waves during stimulation prior to (a) and following (b) subject voluntary flexion; (a) was the first stimulation period and (b) was the second stimulation period in a trial of extensor stimulation alone alternating with voluntary flexion. In (a) the subject was initially relaxed, and in (b) the subject was initially flexing his fingers. The stimulation started at 7 seconds along with the command to relax, and the command to flex occurred at 22 seconds. In (a) the subject's finger hyperextended during the stimulation period. Note that the FDP had increased activity during stimulation while the FDS was silent. In (b) the EDC M-waves are a fraction of their original magnitude in (a), and the finger did not extend at all. Both finger flexor muscles have much larger activity than in (a). (Subject JM: file jmboth.9, periods 2 and 3 (a); periods 4 and 5 (b)).

DISCUSSION

The goals of this thesis were to study methods for using electrical stimulation to provide functional opening of the fingers in patients with spastic hemiplegia, and to determine the influence of subject voluntary effort on the ability of electrical stimulation to extend the fingers. This study also sought to determine the effects of the stimulation on the subject's spasticity. It was initially hypothesized that stimulation of finger extensor muscles would reciprocally inhibit the spastic finger flexor muscles. However, it was found that finger extensor stimulation frequently increased the activity in the finger flexor muscles, and did not decrease the activity. Although spasticity was not reduced, extensor stimulation was able to overpower the spastic finger flexor muscles, and hand opening was achieved in the majority of patients.

Functional Electrical Stimulation for Finger Extension in Spastic Hemiplegia

The first step in this study was to determine what sites must be stimulated in order to achieve finger extension in subjects with spastic hemiplegia. Since surface electrodes were chosen for stimulation, it was also necessary to evaluate if these electrodes would be suitable for long term use.

This study found that both the EDC and the ulnar nerve must be stimulated simultaneously in order to achieve functional opening of the fingers (extension of both MP and IP joints). Functional opening of the fingers was documented in four of six subjects during simultaneous stimulation of these sites. The stimulation was effective over the duration of the experiment except for one of the four subjects.

Although functional opening of the fingers was documented in only four of six

hemiplegic patients (all except HM and LMM), there is evidence to suggest that functional opening of the fingers could have been achieved in at least one other subject had stimulators with a higher overhead voltage been used. Subject LMM had functional opening of the fingers when an EMPI Focus stimulator was used prior to the actual experiment. This stimulator could deliver higher intensity stimuli than the stimulators used during the experiment. It is possible that some degree of MP extension could also have been attained with subject HM if more powerful stimulators had been used.

Difficulties were encountered using surface electrodes for stimulation. The primary disadvantage is that they have poor selectivity. A neuroprosthesis for long term use should use multiple intramuscular electrodes in order to selectively activate all branches of the EDC without activating any neighboring muscles. With surface electrodes, I frequently could not get extension of all four fingers without activating a wrist extensor muscle (in order to avoid activating wrist extensors I sacrificed obtaining index finger MP extension in many subjects). A surface stimulating electrode was chosen for the experiment because it usually activated three or four digits at stimulation levels that were tolerable to the subject. In contrast, intramuscular stimulating electrodes usually activated only one digit at comfortable stimulation levels. To avoid multiple intramuscular electrode implantations, I chose a single surface stimulating electrode for the experiment at the cost of achieving selective extension of all four digits.

Ulnar nerve stimulation provided finger IP extension. Unfortunately, it also had the undesirable effects of activating the thumb adductor in several subjects (which hinders the ability of subjects to acquire objects since the thumb is in the palm of the hand), was intolerable in one subject (CR) if stimulus amplitudes were high enough to provide

adequate IP extension, and may have inadvertently activated the pronator quadratus muscle in some subjects.

Pronator quadratus activation is suspected because forearm pronation during stimulation was observed in many subjects. It is hypothesized that the ulnar nerve electrodes spilled over to the pronator quadratus muscle, which is the main forearm pronator. Forearm pronation may also have contributed to the increase in grasp pressure noted in some subjects, since pronation caused the hand to press into the rubber cylinder the subject's hand was grasping. It is also a possibility that forearm pronation during stimulation may have been part of a flexion withdrawal response. The nervous system could perceive the stimulation to be noxious, and activate this response. However, flexion of the elbow, a component of the flexion withdrawal response (Trombly, 1989), was not noted during the stimulation, and it is more likely that forearm pronation was due to inadvertent activation of the pronator quadratus.

The painful sensations produced by ulnar nerve stimulation could make a neuroprosthesis unacceptable to some hemiplegic subjects (this nerve contains many cutaneous afferents). However, a study conducted by Baker et al. (1979) demonstrated that most hemiplegic subjects can accommodate to the sensations elicited by surface electrical stimulation over a period of three to seven days. While whole nerve stimulation was not performed in Baker's study, patients still found the stimulation to be initially uncomfortable, but were able to accommodate to the sensations.

An alternative approach to placing surface electrodes over the ulnar nerve at the wrist would be to implant intramuscular electrodes into the dorsal interossei muscles just proximal to the MP joints. These electrodes would still activate branches of the ulnar nerve, but the branch to the thumb adductor would not be present at this distal location,

and selective activation of the intrinsic finger extensor muscles would occur. Since fewer cutaneous nerves will be present at this distal location of the ulnar nerve, the stimulation may also be more comfortable to subjects. Placement of the electrodes near the finger MP joints would also alleviate any unwanted activation of the pronator quadratus.

Muscles can fatigue during electrical stimulation, and this limits the ability of a neuroprosthesis to provide function. However, fatigue resistance can be acquired with regular exercise with electrical stimulation (Lieber, 1986). Most of the study participants did not experience any difficulty with fatigue except subject EB, who experienced severe fatigue of his EDC after only two trials with stimulation. Immediately following a stroke, muscles become flaccid (Adams and Victor, 1989) and disuse atrophy can occur prior to the development of spasticity. This subject was only six weeks post-stroke, and disuse atrophy may still have been present, decreasing his muscles' fatigue resistance.

The speed of MP extension (determined by the rate of EDC contraction) will affect a subject's performance of functional tasks. The warmup effect, or slow extension of the MP joint, was most prominent in the first period of stimulation within a block. It is possible that a period of time is needed for muscle potentiation (Burke et al., 1976) to occur before the joint begins to move. When the patient first entered the laboratory, he or she was stimulated with a hand held stimulator in order to locate the EDC motor point for insertion of the EMG recording electrodes. It often took tens of seconds of stimulation before EDC stimulation caused the MP joint to extend. During the actual experiment, half rise times were much shorter (however, they were sometimes as long as ten seconds; this would still drastically limit the speed with which a subject could

acquire an object).

The MP joint often extended much more quickly in later stimulation periods within a block as compared to the first period of stimulation (a carryover effect). This could be due to long lasting muscle potentiation. Another possibility is that the 15 seconds of maintained stretch during the first period of stimulation lengthened the intrafusal muscle fibers in the flexor muscles (Trombly, 1989), resulting in a decreased sensitivity to stretch in later periods of extensor stimulation (Hasan and Houk (1975) found decreased sensitivity in deafferented muscle spindles following maintained stretch).

Carey (1990) reported improved control of active finger extension movement at the MP joint of the index finger in spastic hemiplegics who had manual stretch applied to their extrinsic finger flexor muscles. He hypothesized that manual stretch may have improved control of finger extension by reducing the viscoelastic stiffness in periarticular connective tissue, thereby improving joint mobility and reducing the mechanical impediment that weak extensor muscles must move against. It is possible that manual stretch improved the rate of finger extension of the subjects in the study described here by a combination of two mechanisms: lengthening intrafusal muscle fibers, and improving joint mobility.

Sometimes there is no obvious difference in half rise times for each stimulation period within a block (i.e. CR_5 and JM_6 in Figure 3.17 (b) and (e), respectively). This usually occurs in the second trial of extensor stimulation alone, suggesting that the carryover effect lasts from the first trial of extensor stimulation alone to the second. This may mean that the carryover effect lasts long enough that the initial slow warmup may not be a problem functionally.

Effect of Voluntary Movements on Finger Extension

I wanted to determine if the effects of stimulation could be augmented with patient voluntary effort. The questions I wanted to answer were: 1) can finger extension be increased if the patients attempt to voluntarily extend their fingers during stimulation, and 2) will stimulation provide effective hand opening if patients use voluntary finger flexion to grasp an object.

Simultaneous Voluntary Finger Extension:

The influence of simultaneous voluntary extension on MP extension during extensor stimulation was assessed to determine if it would aid finger extension. In fact, four of five subjects had significantly decreased MP extension during simultaneous voluntary extension compared to a trial of extensor stimulation alone. This reduction in MP extension is a result of inappropriate muscle activation; when most of the hemiplegic subjects attempted to extend their fingers, they activated their finger flexor muscles which caused a decrease in MP extension. The one exception was subject HM; voluntary finger extension increased the MP extension provided by the extensor stimulation. However, he had a decrease in PIP extension. When this subject voluntarily extended his fingers, his EDC activation was sufficient to extend his MP joint. However, low levels of flexor coactivation flexed the PIP joint, and he did not have enough intrinsic activation to extend the PIP joint.

The result that simultaneous voluntary extension hinders finger extension indicates that subjects should remain relaxed during the extensor stimulation in order to achieve the best possible finger extension. Even in the case of subject HM, better PIP extension would be achieved if he didn't voluntarily extend his fingers during

stimulation.

Analysis of videotapes indicates that simultaneous voluntary extension affected some fingers more than others in subjects TC and LMM. However, all fingers must have good extension in order for the subject to acquire an object easily. Fingers with the least amount of extension will be the limiting factor in determining how easily the subject can get his or her hand around an object.

Alternating Voluntary Finger Flexion:

All of the hemiplegic subjects who participated in this study had the ability to flex their fingers, and had voluntary muscle strength scores for finger flexion ranging between 3 and 4 on clinical exam. In performing a functional task, hemiplegics with motor capabilities similar to those of our study participants could use stimulation to open their hand, then voluntarily flex their fingers to grasp the object. Following use of the object, the hemiplegic could then use electrical stimulation to open their hand in order to release the object.

However, in both paradigms with alternating voluntary flexion (extensor stimulation alone alternating with voluntary flexion, and extensor stimulation with simultaneous voluntary extension alternating with voluntary flexion), three of five subjects had significantly less MP extension following voluntary flexion versus the trial when stimulation was alternating with relaxation. Subjects CR and JM had significantly less MP extension in both of the paradigms with alternating voluntary flexion. These subjects could not relax their finger flexor muscles following voluntary finger flexion, thus their flexor EMGs remained large, and stimulation levels which previously extended the fingers could no longer provide full finger extension. In addition, subject

CR's fingers became progressively more flexed following each period of finger flexion. In fact, all the hemiplegic subjects in this study had difficulty relaxing their muscles after they had been voluntarily activated (also noted by Twitchell, 1951; Sahrmann and Norton, 1977). This inability to relax muscles is an indication that their muscle tone is abnormal, due to the presence of spasticity, and interferes with the ability of electrical stimulation to provide hand function.

For subject TC, MP extension was not affected by alternating voluntary flexion (it might be expected that some patients will not be affected by voluntary effort). In contrast, for subject LMM one trial with alternating voluntary flexion had significantly better MP extension than the corresponding trial with alternating relaxation. It wouldn't be expected that alternating voluntary flexion would enhance MP extension.

An explanation for the enhanced MP extension in the trial of extensor stimulation with simultaneous voluntary extension alternating with voluntary flexion is that subject LMM did not appear to voluntarily attempt to extend her fingers during two of three periods of stimulation (which has been documented to decrease her MP extension), thus her MP joint extended with stimulation. In the corresponding trial with alternating relaxation, the subject did attempt to voluntarily extend during stimulation which either flexed the MP joint, or allowed only a few degrees of extension. It appears that superior MP extension was achieved in the trial with alternating voluntary flexion because she did not try to extend her fingers during the stimulation.

Subject HM also had significantly better MP extension during one trial with alternating voluntary flexion versus the corresponding trial with alternating relaxation. What is particularly surprising is that MP extension was achieved at all (in all other trials, stimulation produced net MP flexion). In the alternating voluntary flexion trial in

which subject HM achieved significantly better MP extension, the covariate P_{before} had a significant effect on the dependent variable P_{end} (P_{before} was not significant in any of the other subjects). This suggests that the EDC electrode may have had position dependent recruitment, providing better MP extension when the MP joint started in a more flexed position. This would explain why his MP extension was improved in this trial.

In the analyses of covariance used to assess the effects of alternating voluntary flexion on MP extension, there were only five degrees of freedom, and thus it was difficult to achieve statistical significance. However, in both types of trials with alternating voluntary flexion, three of five subjects had statistically decreased MP extension during stimulation after having voluntarily flexed their fingers. The cause for the decrease in MP extension appears to be the inability of the hemiplegic subjects to relax their finger flexor muscles after having just voluntarily flexed; the increased flexor activity inhibits the ability of the extensor stimulation to extend the MP joint.

Alternative Approaches to Providing Finger Extension and Flexion

Voluntary finger extension and finger flexion can impede the ability of electrical stimulation to provide finger extension in patients with spastic hemiplegia. This study indicates that subjects should remain relaxed during stimulation in order to achieve the best MP extension; voluntary finger extension causes inappropriate activation of finger flexor muscles leading to decreased MP extension. The inability of subjects to relax their finger flexor muscles following voluntary flexion indicates that some subjects will need stimulation levels to be increased following voluntary effort. This could be achieved either with closed loop control (to be discussed in a later section), or by

allowing the patient to adjust the stimulation levels manually with their nonimpaired extremity.

An alternative approach would be to instruct the subject to remain relaxed. Finger flexion would be achieved by stimulating the finger flexor muscles to allow the subject to grasp an object. If the subject is able to remain relaxed during the flexor stimulation and while manipulating the object, then extensor stimulation might be able to open the hand with the same stimulation parameters used before the object was grasped.

Hemiplegic subjects had very long reaction times when making voluntary movements such as finger flexion and finger extension. Part of the reaction time of hemiplegic subjects can probably be attributed to attention deficits, although these were not obviously present in some subjects, who simply appeared to take a few seconds to activate their muscles. The use of electrical stimulation to provide both finger extension and finger flexion should increase the speed with which subjects can grasp and release objects, except in initial periods of stimulation due to the warmup effect. Some subjects will still have attention deficits, but the time delays related to gradual muscle activation will be eliminated.

Electrically activating both finger flexor and extensor muscles will avoid the forearm pronation (part of a flexor synergy) noted when subjects voluntarily flex or extend their fingers. Involuntary forearm pronation could make it more difficult for subjects to perform activities of daily living.

Effect of Extensor Stimulation on Spasticity

EMG was used as the only measure of spasticity in this study. EMG by itself cannot quantify all aspects of spasticity, such as the increase in resistance to passive stretch

observed in the spastic state (Powers et al., 1989). However, EMG does provide insight into what is occurring in the muscles of a spastic subject at rest. Figure 3.12 demonstrates that an able bodied subject is able to completely relax all his finger flexor and extensor muscles when instructed to relax; no resting EMG is present in these muscles. Many figures display resting EMG activity in spastic subjects while they were instructed to relax (e.g. Figure 3.7). There is often resting motor unit activity in one or more muscles; this activity cannot be consciously suppressed by these subjects, and indicates that spasticity is present. Resting EMG is most common in the finger flexor muscles, although subject HM frequently had this activity in his EDC muscle as well.

EMG is also a useful tool in observing the difficulty spastic subjects have in decreasing activation to a muscle once it has been activated (e.g. Figure 3.9). EMG is also necessary to observe the reflex effects of electrical stimulation on muscle activation. Knowledge of these reflex effects will aid in the design of a neuroprosthesis to provide hand function in the spastic stroke patient.

Prior to conducting this study it was hypothesized that stimulation of a finger extensor muscle would inhibit the activity in spastic finger flexor muscles. It was thought that if inhibition could be produced with the stimulation, then the decrease in spastic flexor activity would enhance the finger extension produced by stimulating the EDC and the ulnar nerve.

However, the experiments with spastic subjects indicate that stimulation parameters that are large enough to provide functional opening of the fingers also cause large and sometimes long lasting increases in finger flexor EMGs, contrary to the original hypothesis. This has not been demonstrated previously. It is believed that the

pronounced increase in finger flexor EMGs during extensor stimulation in the spastic subjects are due to stretch reflexes.

When the MP joint is extended by the extensor stimulation, both the FDS and FDP muscles are stretched since they both cross the MP joint. Ulnar nerve stimulation extends the IP joints which causes further stretch of the two finger flexor muscles (both cross the PIP joint, and the FDP also crosses the DIP joint). There is often a burst of flexor EMG at the start of stimulation; it is too quick to be a voluntary reaction, and is believed to be a phasic component of a stretch reflex. The flexor EMG increase that persists for the duration of the stimulation is a tonic response to the muscle stretch.

Stretch reflexes in response to mechanical perturbations which stretch muscles have been documented frequently in relaxed upper extremity flexor muscles in patients with spastic hemiplegia (Powers et al., 1888; Thilmann et al., 1991). In the study by Powers et al. (1988), stretch reflexes were never seen in the hemiplegic subject's nonimpaired arm when the subject was relaxed. In the study by Thilmann et al., (1991), stretch reflexes were not seen in the muscles of able bodied subjects when stretch velocities were below 175 degrees/ second. Similarly, Stefanovska et al. (1989) reported no reflex EMG when the ankle joint of able bodied subjects was rotated at up to 200 degrees/ second. It is therefore not anticipated that stimulation of the EDC and the ulnar nerve would elicit stretch reflexes in persons without a motor control disorder if the subject were completely relaxed (this stimulation would produce joint rotations significantly slower than 175 or 200 degrees/ second).

The presence of stretch reflexes in the hemiplegic subjects in this study indicates the presence of abnormal muscle tone, such as that observed in subjects with spasticity. Studies have been conducted which indicate that the threshold for eliciting the stretch

reflex is decreased in the spastic state (Lee et al., 1987; Powers et al., 1988). Thus, a stimulus which does not elicit stretch reflexes in able bodied subjects easily elicits them in the spastic hemiplegic subjects. In the experiments on able bodied subjects described in this study, the subjects were maintaining a 10% maximal voluntary contraction, and were therefore not relaxed. This excitation to the flexor motor neuron pool was sufficient to allow generation of stretch reflexes in some of those subjects.

I do not have direct evidence to support the hypothesis that these increases in flexor EMG are stretch reflexes. Further support for this hypothesis could be obtained by recording from Ia afferent fibers to determine if they fire when the extensors are stimulated. However, many afferents are probably stimulated in the paradigm described here, and other reflex pathways could cause the excitation to the finger flexor muscles, or contribute to the excitation produced by muscle stretch. Whatever the mechanism, it is obvious that extensor stimulation causes net excitation and not inhibition of the finger flexor muscles.

It is also possible that the increased activity in the finger flexor muscles could be related to the patient's mental state. Some patients found the electrical stimulation to be uncomfortable, and in some instances painful. This may have heightened their spasticity, and in turn increased the response of their finger flexor muscles during stimulation. However, one subject (JM) found the stimulation to be very comfortable, yet he still had pronounced flexor activity during stimulation. Thus although mental state may play a role in the ease with which stimulation generates stretch reflexes, it is not the only factor.

The variability in subject responses over time is probably due to the subject's spasticity not remaining constant. If the threshold for eliciting a stretch reflex increases

over the course of the experiment, then stimulation levels which produce strong flexor EMGs in an early trial (e.g. subject CR, Figure G1, FDS EMG) may cause only a few flexor motor unit firings in a later trial (e.g. subject CR, Figure G2, FDS EMG).

Of interest are the flexor EMG increases during stimulation of subject HM (see Figure G3). He has a large increase in both flexor EMGs during stimulation, despite the fact that his MP joint is flexed by the stimulation. This subject had very strong IP extension. It is not known if the FDS and FDP muscles experienced net shortening or lengthening when the IP joints extended and the MP joints flexed. If net shortening occurred, then stretch reflexes were not the mechanism responsible for the increase in flexor EMG.

A possible explanation for flexor EMG increases other than stretch reflexes is that the stimulation might be perceived by the nervous system as being noxious. A flexion withdrawal response might occur in response to this noxious stimulus, thereby activating the finger flexor muscles. However, a flexion withdrawal response would be expected to involve most flexor muscles in the upper extremity (Trombly, 1989), and this was not noted in the experiments.

In the experiments described here, the stimulation turned on abruptly, i.e. with stimulation parameters at their maximal values. An alternative approach would be to ramp the pulse width for a few seconds until the maximal pulse width was reached (amplitude fixed at maximum). If the stimulation level were slowly increased in this manner, it might be possible to avoid what is believed to be a phasic response to muscle stretch in the finger flexor muscles. By decreasing this response, better finger extension might be achieved. In addition, this stimulation paradigm would probably be more comfortable for the subject.

Many subjects had increased flexor EMGs that persisted after the stimulation stopped; by some mechanism the stimulation raised the excitability of the flexor motor neuron pool. An increase in flexor muscle length does not appear to be responsible for the increase in flexor EMG levels in most subjects. There was a correlation between an increase in MP extension (meaning the flexor muscles were more stretched) and an increase in EMG levels pre- to post-stimulation in only one muscle of one subject. There was not a significant correlation in any of the other subjects who experienced significant increases in flexor EMGs post-stimulation. Time averages of flexor EMGs indicate that the increase in EMG is short lasting; five seconds after the stimulation ends there is no increase compared to pre-stimulation levels.

Variable M-Waves During Extensor Stimulation

The significance of variable M-waves during stimulation is not known. In general, they were not correlated with the rate of finger extension (the warmup effect). Three subjects had increases in the amplitude of their EDC M-waves over the course of a period of stimulation. Two possible mechanisms for this phenomenon have been hypothesized. The first hypothesis is that length dependent recruitment was occurring. When the stimulation causes the EDC to contract, the distance between the surface stimulating electrode and the EDC motor point may change. If the contraction brings the motor point closer to the electrode, and the recruitment curve is in a high gain region, then larger recruitment of the EDC could occur causing the muscle to contract more forcefully. This in turn could bring the motor point closer to the electrode, and the cycle would continue until the stimulation stops. The net effect would be a progressive increase in the recruitment of the EDC, which might produce the gradual

increase in M-wave amplitude observed in three subjects.

A second hypothesis is that an H-reflex with time varying gain occurred during the stimulation. The H-reflex is an electrical analog of the stretch reflex. It is elicited with low intensity stimulation of a mixed nerve containing Ia afferent fibers. It has been determined that this reflex is monosynaptic (Delwaide, 1985). When the EDC muscle is stimulated, Ia afferents within the muscle may also be stimulated. The Ia afferents synapse in the spinal cord on alpha motor neurons of the EDC, causing it to contract reflexively. This would cause an increase in EDC EMG over that resulting from the M-waves. It is hypothesized that some subjects may experience pronounced increases in EMG from the H-reflex; in other subjects the increase may be minimal. If the strength of the H-reflex is time varying, and also creates large increases in EDC EMG, then the time varying increase in EMG amplitude seen in Figure 3.23 may result. A mechanism for time varying gain of the H-reflex is not known.

A tonic vibration reflex was also considered as a possible mechanism for generating the time varying EDC M-waves. In the case of subject EB, his contraction was not well fused, and muscle vibration probably occurred. Vibration can selectively activate Ia afferents, thus producing reflex contraction of the muscle being vibrated. However, the tonic vibration reflex is activated with vibration frequencies greater than 50 Hz, the optimal frequency in humans being between 100-200 Hz (Trombly, 1989). Thus, it is unlikely that a tonic vibration reflex occurred during extensor stimulation.

It is not known what caused the tent top pattern of EDC EMG of subject JM (see Figure 3.24a). It may be artifactual, but no method of generation has been hypothesized. It too is not correlated with finger extension. This is the same subject in which voluntary finger flexion greatly diminished subsequent EDC M-waves (Figure

3.24b). These diminished M-waves are correlated with minimal finger extension; this subject would not be able to acquire any object after voluntary flexion. It may be that voluntary finger flexion caused movement of the wrist which increased the distance between the stimulating electrodes and the EDC motor point, thus making subsequent stimulation ineffective due to position dependent recruitment.

Reciprocal Inhibition in Able Bodied Subjects

Tests in able bodied subjects also indicate that stimulation of the EDC has weak inhibitory effects on finger flexor EMGs, and in many cases produces excitation of finger flexor muscles. These tests relied upon the subject being able to maintain a constant effort contraction throughout the trial as evidenced by a lack of long latency EMG changes. Any observed changes in flexor EMG were then assumed to be a result of a short latency reflex, and not subject voluntary intervention.

Based on time averages of flexor EMGs pre- and post-transition, there is little evidence for strong and consistent inhibition of finger flexor muscles during stimulation of the EDC muscle. Note that small degrees of voluntary intervention might not be detected from the transition ensembles, and the possibility that voluntary intervention accounts for these changes in EMG cannot be completely ruled out.

IPI ensemble averages reveal a broad peak beginning at approximately 30 msec and reaching maximum amplitude at 50-60 msec in the flexor muscles of some subjects. It is believed to be a phasic response to stretch caused by stimulation induced contraction of the EDC, which stretches the flexor muscles. Maximum EDC force generation (which corresponds to maximum shortening of the flexors) occurs approximately 30 msec after a stimulus pulse (Monster and Chan, 1977). When stretch reflex

transmission time in the upper extremity (approximately 20 msec) is added, the phasic component of a stretch reflex would be anticipated to reach maximum amplitude at approximately 50 msec, which is close to when the observed peak occurs.

Both phasic stretch reflexes (which can be observed in IPI ensemble averages when they are synchronized to the stimulus pulses), and tonic stretch reflexes (a response to maintained stretch) are likely responsible for many of the observed increases in flexor EMGs during EDC stimulation. Decreases in flexor EMGs were only seen at lower stimulation levels. The subjects were grasping a wooden post during the stimulation, and these lower stimulation levels may not have produced the net stretch of the flexor muscles needed to elicit a stretch reflex. A reciprocal inhibitory mechanism may have produced the decrease in flexor EMGs seen at the lower stimulation levels.

Whether net inhibition or excitation occurs in the finger flexor muscles during stimulation of the EDC depends on the relative strengths of the excitation produced by stretching the flexors and any inhibition produced by a reciprocal inhibitory mechanism. It is apparent from these tests that stimulation of the EDC is unlikely to produce significant reductions in finger flexor EMGs.

Transient reciprocal inhibition has been demonstrated previously in able bodied subjects. A brief stimulus train applied to the peroneal nerve (which innervates the tibialis anterior) has been shown to inhibit a mechanically induced stretch reflex of the soleus muscle (the antagonist) (Apkarian and Naumann, 1991). However, a second stretch applied immediately following the first stretch was not inhibited, indicating that there were no long lasting effects of the stimulation. Reciprocal inhibition of the soleus muscle has also been demonstrated using H-reflexes; soleus H-reflexes are depressed when the antagonist's Ia afferent fibers are stimulated (Tanaka, 1983). However, this

inhibition lasts only 40 msec. In the study presented here, I was trying to produce a maintained inhibition of the spastic finger flexor muscles, which is necessary in order for the hand to be able to perform functional tasks. Extending the H-reflex results to tonic stimulation might not be appropriate.

CONCLUSIONS

This study investigated the use of electrical stimulation to provide hand opening in patients with spastic hemiplegia. It was shown that electrical stimulation of finger extensor muscles in both able bodied and hemiplegic subjects generally does not decrease finger flexor activity, as might occur by reciprocal inhibition. Instead, finger extensor stimulation frequently causes an increase in finger flexor activity, presumably by eliciting stretch reflexes. Thus, finger extensor stimulation which is powerful enough to provide full extension of the fingers is not an appropriate method for inhibiting the spastic flexor muscles in hemiplegic subjects.

Although finger flexor spasticity was not reduced by the electrical stimulation, functional opening of the fingers was achieved in the majority of hemiplegic subjects because the extension force produced by the stimulation was sufficient to overcome the flexion force. Stimulation of both the EDC and the ulnar nerve is necessary in order to extend both the MP and IP joints of the fingers. Finger extension should allow subjects to acquire objects, and enhance their ability to perform activities of daily living.

Hemiplegic subjects should remain relaxed during extensor stimulation in order to achieve optimal MP extension. Simultaneous voluntary finger extension decreases MP extension in most subjects due to inappropriate activation of finger flexor muscles. It was also discovered that alternating voluntary finger flexion decreases subsequent MP extension in some hemiplegic subjects. This is due in part to an inability of the subjects to relax their finger flexor muscles; the flexor EMG remains large, and stimulation levels which extended the fingers previously are no longer sufficient to provide full finger extension. In these patients it may be necessary to provide closed loop control of

finger extension. An alternative approach would be to stimulate the finger flexor muscles to provide hand grasp. If the subject can remain relaxed during finger flexor stimulation and object manipulation, then a constant level of stimulation to the finger extensors may provide adequate finger extension so that the subject can release the object following use.

FUTURE WORK

A difficulty that must be overcome in developing a neuroprosthesis for spastic stroke patients is that voluntary effort can change the level of a patient's spasticity, thereby changing the amount of stimulation needed to provide subsequent finger extension. Future work should investigate the degree to which stimulation levels must be adjusted in order to achieve finger extension while the patient is performing functional tasks. Once it is known how much stimulation levels must be varied, then a strategy for changing stimulation levels must be developed.

One possible mechanism for adjusting stimulation levels would be for the patient to use his nonimpaired extremity to change the stimulus pulse width or amplitude. This would mean that this hand would not be able to assist in task performance. However, if stimulation levels need only be adjusted after the subject has finished grasping and using an object, then the subject may not need the nonimpaired hand at that moment, and it could be used to adjust stimulus parameters.

Another alternative would be to use closed loop control. Closed loop control has the potential to keep the amount of finger extension constant even if the patient's spasticity changes, muscles begin to fatigue, or muscle recruitment properties change due to length-tension properties of muscle. In a closed loop control system, stimulation parameters are varied automatically to compensate for changes in system properties with time (Crago et al., 1991). In order for finger extension to be controlled, a joint angle transducer(s) would need to be worn by the patient to provide the control system with information on joint angle.

Another issue that must be addressed before a neuroprosthesis can be used by spastic

stroke patients is the source for a command control site. On-off control is probably all that is needed in a system that only provides finger extension. Some mechanism must be used for the patient to turn the stimulation on to provide the extension, and to turn the stimulation off when the subject has his or her hand positioned around the object and is ready to grasp it.

A switch operated by the subject's nonimpaired extremity could initially turn the neuroprosthesis on to provide finger extension. If the subject has very strong finger flexion, it is possible that she could flex her fingers against the stimulation and still grasp the object once the hand is correctly positioned (assuming no closed loop control is being used). However, this would cause unneeded stimulation to the finger extensor muscles which would accelerate muscle fatigue. Alternatively, if the subject wears a joint angle sensor and voluntarily flexes her fingers, then when the joint flexes to a predetermined angle, the extensor stimulation could be turned off, and the subject would continue grasping the object under voluntary control. However, a mechanism is still needed to turn the stimulation back on so that the subject can release the object.

An alternative would be to use EMG from a muscle under voluntary control as a control source to turn the stimulation off when the subject needs to grasp the object, and to turn it on again when the object must be released. Since voluntary motor capabilities vary greatly across subjects, an appropriate muscle for EMG control would need to be selected for each individual patient.

A neuroprosthesis for stroke patients may need to provide thumb extension in addition to finger extension. As previously discussed, stimulation controlled finger flexion would be an alternative to having subjects voluntarily flex their fingers to grasp objects, and would add additional stimulation channels to a neuroprosthesis. In order

to provide selective stimulation of all branches of the EDC, and selective stimulation of the finger intrinsic extensor muscles, multiple intramuscular stimulation channels will also be needed. It is clear that a neuroprosthesis for spastic stroke patients will require many channels of stimulation and will be much more complicated than the one and two channel devices previously reported in the literature.

APPENDIX A

Forms Used in Clinical Testing of Hemiplegic Subjects

Qualitative Assessment of Spasticity - Ashworth Scale

Patient Name: _____

Date: _____ Impaired side = _____

Before Experiment

Time = _____

	<u>impaired side</u>	<u>unimpaired side</u>
Fingers:	MP: IP:	
Thumb:	CMC: MCP: IP:	
Wrist:	Flexion: Extension:	
Elbow:	Flexion: Extension:	
Forearm:	Pronation: Supination:	
Shoulder:	Abduction: Adduction:	

Qualitative Assessment of Spasticity - Ashworth Scale (con't)**After Experiment**

Time = _____

		<u>impaired side</u>	<u>unimpaired side</u>
Fingers:	MP: IP:		
Thumb:	CMC: MCP: IP:		
Wrist:	Flexion: Extension:		
Elbow:	Flexion: Extension:		
Forearm:	Pronation: Supination:		
Shoulder:	Abduction: Adduction:		

Sensory Evaluation

side being tested (tested against nonimpaired side) : _____

	<u>light touch</u>	<u>pinprick</u>
shoulder:	_____	_____
inside upper arm:	_____	_____
forearm: dorsal	_____	_____
volar	_____	_____
hand: dorsal	_____	_____
volar	_____	_____

Reflex Testing

	<u>Right side</u>	<u>Left Side</u>
Tendon jerk:		
finger flexors	_____	_____
biceps	_____	_____
triceps	_____	_____
brachioradialis	_____	_____
pectoralis	_____	_____
Hoffman:	_____	_____
Wrist clonus:	_____	_____
Spread of Reflexes:	_____	_____

Upper Extremity Active Range of Motion (AROM)

	<u>extension</u>	<u>flexion</u>		
fingers	_____	_____		
thumb	_____	_____		
wrist	_____	_____		
elbow	_____	_____		
forearm	supination: _____	shoulder	abduction: _____	
	pronation: _____		adduction: _____	

Voluntary Muscle Strength

	<u>extension</u>	<u>flexion</u>
finger	_____	_____
thumb	_____	_____
wrist	_____	_____

APPENDIX B

Instructions to Subjects

Instructions to Able Bodied Subjects

During the experiment I will ask you to flex your fingers while gripping a wooden post. The electrical activity of your finger flexor muscles will be displayed on the oscilloscope screen (the visual feedback). I want you to keep the oscilloscope display (a horizontal line) at a fixed position on the screen, indicated by a blue line.

Each trial will last for 2 - 2.5 minutes. During the trial you will receive alternating periods of electrical stimulation which will last for 12 seconds. There is a 12 second period without stimulation preceding each period of stimulation. There are a total of 11 periods. Just prior to the stimulation, and for the duration of the stimulation, the visual feedback on the screen will be removed. Try to maintain your contraction level constant during the entire trial; don't react to the stimulation. When the visual feedback returns to the oscilloscope after the stimulation, make sure the display is at the blue line.

There are a total of 14 trials, and you will be stimulated at seven different levels. Two trials in succession will be conducted at the same stimulation level, separated by a four minute rest period.

Instructions to Hemiplegic Subjects

During the experiment I will ask you to either flex, extend, or relax your fingers. Lights on this display box will tell you when to do this. I will also say the words "flex," "relax," "extend" when you are supposed to do this.

Each trial will last approximately 2.5 minutes. In some trials you will receive alternating periods of electrical stimulation which will last for 15 seconds. There is a 15 second period without stimulation before each period of stimulation. In other trials there will not be any stimulation; you will just voluntarily move your fingers.

There are a total of 10 trials, with a six minute rest period between trials.

APPENDIX C

Hemiplegic Experimental Protocol

- 1) Test spasticity (Ashworth scale) and reflexes
- 2) Place EDC and ulnar nerve surface electrodes (cathode & anode; use EMPI Focus stimulators to determine optimal electrode locations)
- 3) Implant recording electrodes (test electrode with arm in neutral)
- 4) Zero pressure offset
- 5) Connect electrodes to equipment and determine noise levels
- 6) Determine stim freq => fused contraction (≈ 20 Hz), and PW and amplitude that produce best finger extension
- 7) Determine EDC and ulnar nerve threshold PWs (software: **Anne's newzap**)
 - 1 Hz freq.
 - look at skin over EDC (look at fingers for ulnar nerve, possibly thumb)
- 8) Determine stimulus artifact width at these PWs (software: **FES-5**, parameter file: **teststart.param***)
 - FDS (Ch 0)
 - FDP (Ch 0) (will have to switch channels) **Tell subject to remain relaxed!**
- 9) Calibrate MP joint angle sensor
- 10) * Change **art_width** in software if necessary (**FES-5**)
 - * Stop saving _temp files
- 11) Set amplifier gains to 2K
- 12) Explain trials to subject
 - Read hemiplegic instructions
 - Demonstrate to subject what finger flexion and extension are
- 13) Trials (software = **FES-5**). File = **cva_short.param** or **cva10.param**
Enter correct stim freq!

Trial Type

Stimulation Menu

- 1) Voluntary flexion alone

b

2) Voluntary extension alone	c
3) Alternating voluntary extension & voluntary flexion	f
4) Extensor stimulation alone	a
5) Extensor stimulation alone	a
6) Simultaneous extensor stimulation & voluntary extension	d
7) Simultaneous extensor stimulation & voluntary extension	d
8) Alternating extensor stimulation & voluntary flexion	g
9) Alternating voluntary extension / extensor stimulation & voluntary flexion	k
10) Extensor stimulation alone	a

14) Retest spasticity (Ashworth scale). Do not remove subject's hand from the apparatus when testing the fingers and the thumb.

15) Videotape electrode locations

The most recent version of the **FES-5** software is in a folder labeled **2-15-93 software**. The parameter file `cva10.param` has an additional 10th period in which the subject is asked to relax. If the trial has alternating voluntary flexion, then the relax light only comes on for the second half of period 10 (the last seven seconds). This file was only used with subjects HM, EB, and JM.

*Stimulus artifact width detection can also be done using Labview software. It is located on the Quadra 900 in a folder labeled **artifact_detection** (it is also on diskette). This software collects data from both the FDS and FDP simultaneously.

APPENDIX D

Clinical Measures of Subject Impairment

The following tables include the results of clinical testing of hemiplegic subjects. Blank entries indicate that the testing was not performed. For subjects LMM, HM, and EB, a second examiner (AEH) made the Ashworth measurements following the experiment (“after” column). All other Ashworth measurements were made by Dr. Carl Billian.

Key of abbreviations:

p	paresthesia
+	present
-	absent
flex	flexion
ext	extension
pro	pronation
sup	supination
abd	abduction
add	adduction
MP	metacarpophalangeal joint
IP	interphalangeal joint
CMC	carpometacarpal joint
MCP	metacarpophalangeal joint (thumb)
BR	brachioradialis

Table D1. Clinical scores, subject TC.

Subject	Sensory light touch pinprick	Reflex involved side uninvolved side	AROM ext flex	Voluntary Muscle Strength ext flex	Ashworth before after
TC	shoulder 2	finger flexors	0	finger 1	finger MP 3 2
	inside upper arm 2	biceps 3	0	thumb 1	finger IP 4,3 3
	dorsal forearm 3	triceps 3	1	wrist 2	thumb CMC 4 1
	volar forearm 3	BR 3	2	elbow 2	thumb MCP 3 2
	dorsal hand 3	pectoralis +	forearm pro. sup. 1 0	thumb IP 3	thumb IP 3 2
	volar hand 3	Hoffman +	shoulder abd. add. 2 2	wrist flex. 2	wrist flex. 2 2
		wrist clonus -		wrist ext. 3	wrist ext. 3 3
		reflex spread +		elbow flex. 3	elbow flex. 3 2
				elbow ext. 3	elbow ext. 3 2
				forearm pro. 1	forearm pro. 1 2
				forearm sup. 2	forearm sup. 2 3
				shoulder abd. 2	shoulder abd. 2 2
				shoulder add. 1	shoulder add. 1 1

Table D2. Clinical scores, subject CR.

Subject	Sensory light touch pinprick	Reflex involved side uninvolved side	AROM ext flex	Voluntary Muscle Strength ext flex	Ashworth before after
CR	shoulder 1	finger flexors +	fingers 0	finger 0	finger MP 2 3
	inside upper arm 1	biceps 3	thumb 0	thumb 0	finger IP 2 3
	dorsal forearm 1	triceps 3	wrist 0	wrist 1	thumb CMC 2 2
	volar forearm 1	BR 3	elbow 2		thumb MCP 1 2
	dorsal hand 1	pectoralis +	forearm pro. 1		thumb IP 1 2
	volar hand 3 (p)	Hoffman +	forearm pro. sup. 1		wrist flex. 1 1
		wrist clonus +	shoulder abd. 1		wrist ext. 3 3
		reflex spread +			elbow flex. 3 2
					elbow ext. 3 2
					forearm pro. 2 2
					forearm sup. 3 3
					shoulder abd. 2 2
					shoulder add. 2 2

Table D3. Clinical scores, subject LMM.

Subject	Sensory light touch pinprick	Reflex involved side uninvolved side	AROM ext flex	Voluntary Muscle Strength ext flex	Ashworth before after
LMM	shoulder 1	finger flexors +	fingers 1	finger 1	finger MP 3 2
	inside upper arm 1	biceps 3	thumb 1	thumb 1	finger IP 4 3,2
	dorsal forearm 1	triceps 3	wrist 1	wrist 2	thumb CMC 3 1
	volar forearm 1	BR 4	elbow 1		thumb MCP 3 2
	dorsal hand 1	pectoralis +	forearm pro. 2		thumb IP 3 2
	volar hand 1	Hoffman +	shoulder abd. 1		wrist flex. 1
		wrist clonus +			wrist ext. 3
		reflex spread +			elbow flex. 4
					elbow ext. 3
					forearm pro. 1
					forearm sup. 3
					shoulder abd. 3
					shoulder add. 4

Table D4. Clinical scores, subject HM.

Subject	Sensory light touch pinprick	Reflex involved side uninvolved side	AROM ext flex	Voluntary Muscle Strength ext flex	Ashworth before after
HM	shoulder 1	finger flexors +	fingers 1	finger 3	finger MP 3
	inside upper arm 1	biceps 3	thumb 1	thumb 1	finger IP 3,2
	dorsal forearm 1	triceps 3	wrist 1	wrist 4	thumb CMC 4
	volar forearm 1	BR 3	elbow 2		thumb MCP 4
	dorsal hand 1	pectoralis +	forearm pro. 1		thumb IP 3
	volar hand 2	Hoffman +	shoulder abd. 1		wrist flex. 4
		wrist clonus -			wrist ext. 4
		reflex spread +			elbow flex. 5
					elbow ext. 4
					forearm pro. 2
					forearm sup. 3
					shoulder abd. 4
					shoulder add. 3

Subject	Sensory		Reflex		AROM		Voluntary Muscle Strength		Ashworth				
	light touch	pinprick	involved side	uninvolved side	ext	flex	ext	flex	before	after			
EB	shoulder	2	2	finger flexors	+	+	fingers 0	1	finger 0	4	finger MP	3	3
	inside upper arm	2	1	biceps	3	3	thumb 0	1	thumb 0	2	finger IP	3	3
	dorsal forearm	2	1	triceps	3	3	wrist 1	1	wrist 1	2	thumb CMC	1	1
	volar forearm	2	1	BR	3	2	elbow 1	1			thumb MCP	1	1
	dorsal hand	2	1	pectoralis	+	-	forearm pro. 1	sup. 1			thumb IP	1	1
	volar hand	2	1	Hoffman	+	-	shoulder abd. 2	add. 2			wrist flex.	2	2
				wrist clonus	+						wrist ext.	2	2
				reflex spread	+	+					elbow flex.	1	
											elbow ext.	2	
											forearm pro.	1	
											forearm sup.	2	
											shoulder abd.	1	
											shoulder add.	1	

Table D6. Clinical scores, subject JM.

Subject	Sensory		Reflex		AROM		Voluntary Muscle Strength		Ashworth	
	light touch	pinprick	involved side	uninvolved side	ext	flex	ext	flex	before	after
JM	shoulder 1	3	finger flexors	+	fingers 0	1	finger 0	3	finger MP	3 2
	inside upper arm 2	3	biceps	3	thumb 0	1	thumb 0	2	finger IP	2 2
	dorsal forearm 2	3	triceps	3	wrist 0	1	wrist 1	2	thumb CMC	2 1
	volar forearm 3 (p)	3	BR	2	elbow 1	0			thumb MCP	2 1
	dorsal hand 2	3	pectoralis	+	forearm pro. 1	sup. 0			thumb IP	1 1
	volar hand 2	3	Hoffman	+	shoulder abd. 1	add.			wrist flex.	2 1
			wrist clonus	-					wrist ext.	3 2
			reflex spread	+					elbow flex.	3 1
									elbow ext.	2 2
									forearm pro.	1 1
									forearm sup.	2 1
									shoulder abd.	2 1
									shoulder add.	1 1

APPENDIX E

Selected Examples of Hemiplegic Subject Voluntary Capabilities

Figure E1. Example of voluntary finger flexion. The command to flex was given at 7 seconds, the command to relax occurred at 22 seconds. This subject actually activated the EDC muscle first; activation of the two flexor muscles occurred approximately two seconds later, and net flexion occurred (downward deflection of the position trace). This subject was having difficulty relaxing all three muscles when the command to relax was given at 22 seconds (subject JM: file jmboth.2, periods 2 and 3).

Figure E2. Example of voluntary finger extension. The command to extend was given at 7 seconds, the command to relax occurred at 22 seconds. Note the scale on the vertical axis. This subject did not have activation of any muscles when trying to extend. All other subjects activated at least one or both flexor muscles (subject JM: file jmboth.3, periods 8 and 9).

Figure E3. Example of voluntary finger extension. The command to extend was given at 7 seconds, the command to relax occurred at 22 seconds. Note that the muscle activation patterns were very similar to those occurring in voluntary finger flexion for this same subject (Figure 3.10). This subject only briefly activated the EDC. The FDS remained silent but there was strong activation of the FDP. Most subjects activated both the FDS and FDP; this subject and subject LMM were exceptions, only activating the FDP muscle (subject TC: file tcbth.2, periods 8 and 9).

Figure E4. Example of alternating voluntary finger extension and finger flexion. The subject was initially attempting to extend his fingers. The command to flex was given at 7 seconds, and the command to resume extension occurred at 22 seconds. Both the EDC and the FDP were initially active. When the command to flex occurred, the flexor EMGs increased, the fingers flexed (downward deflection of position trace), and the grasp pressure increased. When asked to resume extension, there was an increase in both flexor and extensor EMGs; however, the increase in extensor EMG was sufficient to produce finger extension, and the grasp pressure decreased (subject HM: file hmboth.6, periods 3 and 4). This was the only subject who had any voluntary finger extension.

APPENDIX F

Selected Examples of the Effects of Voluntary Flexion on Subsequent MP Extension During Stimulation

Figure F1. Effect of voluntary flexion on subsequent MP extension during stimulation (subject relaxed). For this subject, alternating voluntary flexion actually increased subsequent MP extension. In (a) the subject was relaxed during stimulation (subject HM: file hmboth.15). This subject had net flexion during extensor stimulation. In (b) the subject made alternating voluntary flexion movements (subject HM: file hmboth.12). Note that following flexion and during stimulation there was slight MP extension.

Figure F2. Effect of voluntary flexion on subsequent MP extension during extensor stimulation with simultaneous voluntary extension. For this subject, alternating voluntary flexion increased subsequent MP extension. In (a) the subject was relaxed following stimulation (subject LMM: file lmmboth.10). In (b) the subject made alternating voluntary flexion movements (subject LMM: file lmmboth.12).

APPENDIX G

Examples of Flexor EMGs During Extensor Stimulation Alone

In all the following figures, the subject was relaxed throughout the trial. In each figure, the stimulation turned on at 7 seconds and turned off at 22 seconds. One portion of a longer trial consisting of four stimulation periods is shown. Also see Figure 3.23 in the text.

Figure G1. Response of flexor EMGs during extensor stimulation alone (subject CR: file crboth.4, periods 6 and 7). This subject had a small increase in FDS EMG during stimulation, but only a brief response in the FDP when the stimulation turned on, with a few spikes later in the period. The FDS remained active for the duration of the stimulation, but decreased to pre-stimulation levels following the cessation of stimulation. As the MP joint extended, there was a decrease in grasp pressure.

Figure G2. Response of flexor EMGs during extensor stimulation alone (subject CR: file crboth.11, periods 2 and 3). This is a later trial for the same subject shown in Figure G1. In this stimulation period, both the FDS and FDP EMGs were virtually silent, except for a tiny burst in the FDP at the onset of stimulation, and a few spikes later in the period. The small ripples in the FDS EMG are due to processing of tiny stimulation artifacts which remained in the signal post-processing. Note the very slow increase in MP joint angle, and the decrease in grasp pressure as the fingers extended.

Figure G3. Response of flexor EMGs during extensor stimulation alone (subject HM: file hmboth.8, periods 2 and 3). This subject had MP flexion in response to the extensor stimulation. Both the FDS and FDP EMGs had an increase in activity when the stimulation began; this increase persisted in both muscles for the duration of the stimulation, and their amplitude remained high even after the stimulation ended. This subject also had a large increase in grasp pressure during extensor stimulation. Subject HM is unusual in that he had EDC spasticity; note the increase in EDC activity following the cessation of stimulation.

Figure G4. Response of flexor EMGs during extensor stimulation alone, expanded time scale (subject TC: file tcboth.5, period 2). The superimposed stimulus pulses indicate when the stimulation occurred. Although the MP joint was initially flexed (it extended slowly), the FDP muscle had very large motor unit activity when the stimulation began; this activity was not time locked to the occurrence of the stimulus pulses. The FDS had only a brief increase when the stimulation started.

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