

# Surface Electromyography of Facilitated versus Free Arm Activation after Stroke

by

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Submitted to the Department of Electrical Engineering and Computer  
Science

in partial fulfillment of the requirements for the degree of

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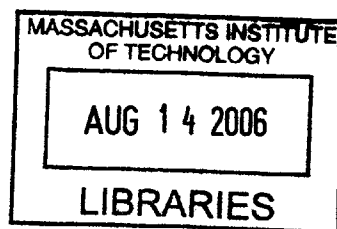
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BARKER

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## Abstract

The aim of this study is to determine if the amount of volitional muscle activation in a stroke affected arm is altered by facilitation. An experiment was designed in which surface EMG was recorded from the biceps brachii and triceps brachii of seven acute stroke patients, during three types of activities. Recordings are made during unassisted attempts at flexion and extension, weight bearing attempts through the arm, and therapist resistive facilitation. The results show that weight bearing and facilitation result in greater muscle activation, even in patients with fully paretic arms. Possible applications of this effect are discussed, particularly with regard to robotic therapy.

Thesis Supervisor: Woodie Flowers

Title: Pappalardo Professor of Mechanical Engineering

## Acknowledgments

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I would also like to acknowledge the patients who participated in this study. The contribution of their time made this thesis possible, and the contribution of their life stories made it fun and memorable. Every one was enthusiastic and cooperative, and I wish them the best of luck in their recovery.

I would like to thank the members of the MIT Active Joint Brace Lab, Kailas Narendran, John McBean, and Kate Zebrose, for all their help in the design and development of this project. I hope it will be useful.

Both of my advisors, Woodie Flowers and Dennis Freeman, provided guidance and insight, and helped me understand the nature of academic inquiry.

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# Chapter 1

## Introduction

This thesis describes an experiment in which motor impaired stroke patients were able to produce measurable, volitional muscular contractions in their paralyzed arm, under three different sets of conditions. Patients were measured attempting unassisted contraction, during a weight bearing exercise, and during therapist facilitation. The measurements show that with therapist facilitation, even paralyzed patients can selectively activate muscles with much greater intensity than without. It compares the results between patients and between activities, and analyzes the measurements made in an attempt to understand why the patients can produce muscular contractions, but cannot move. Finally, it discusses the usefulness of such results and the differences between the measurements, both from a therapeutic perspective and an engineering perspective.

### 1.1 Motivation

Stroke rehabilitation is a difficult process, experienced by over 4.5 million Americans, according to the CDC [38]. That number will increase in the coming years, as the national age demographic shifts upward. Acute rehabilitation is therapist-time intensive, and as the therapist to patient ratio shrinks, alternative approaches such as robotic therapy will become more valuable.

Current motor rehabilitation approaches include physical and occupational ther-

apy to improve strength and control and also to teach coping strategies for a patient's impairment. Improvement tends to plateau at approximately 6 months post-stroke, and about 50-60% of patients continue to have motor impairment after that point [32]. Recovery of function happens gradually, and while the external markers of progress are well known, the neurological mechanisms by which recovery happens are not well understood.

As doctors and therapists become more aware of which neurological activation patterns are good indicators for recovery, and as they become more aware of the activation caused by different therapies, they can begin to design treatment to optimize activation and learning for each patient. This study aims to determine the muscular activation patterns of one activity, namely facilitation. It further proposes some studies to assess the neural activation during facilitation.

Equally as important as the therapy design is the ability of the patient to see that his/her effort is succeeding at activating a muscle. Many patients have sensory deficits as well as motor control deficits, and even patients who have accurate position sensation can have inaccurate tension detection. Furthermore, many patients simply do not generate enough muscle contraction to be felt, or don't see any movement, and therefore assume that no signal is getting through.

Initial experiences with chronic hemiplegic stroke patients indicated that although they could not move the arm voluntarily, they could control their arm muscles to produce substantial tonic resistance, or isometric force, but only in response to facilitation. They were unaware of this fact, but very interested and excited by it. It indicates that stroke patients have some voluntary control, and given the opportunity to see some output, can be motivated and learn to control it further.

The amplification of the EMG signal due to facilitation also has important implications for using the signal in robotic control applications. It may additionally imply that there are ways of assisting the patient to amplify these signals independently. The following study examines changes to the EMG signal, based on mechanical facilitation provided by an occupational therapist.



# Chapter 2

## Biological Background

The results of this thesis show that some hemiparetic stroke patients, even those with no voluntary motion, have the ability to control strong muscular contractions in their paretic arm. In order to better understand what the results mean, a brief review of muscular function, motor control, and the effects of stroke are presented.

### 2.1 The Basis of Motor Control

Motor control lies in both the brain and the spinal cord. The spinal cord initiates reflexes, and contains sub-routines for coordinated muscle movements. The brain controls volitional movement, and commands the actions of the spinal cord. It controls execution of the sub-routines. For example, to walk, the brain must initiate the command to walk, but the spinal cord handles many of the low level logistics such as balance and the sequence of motions. Figure 2-1 shows the basic organization of motor control in the central nervous system.

#### 2.1.1 The Brain and Motor Control

Upper motor neurons are commonly injured by stroke, because they constitute such a large proportion of the cortex. Additionally, motor pathways wind throughout the brain, the cerebral cortex and the spinal cord. Injury to any of these areas can result

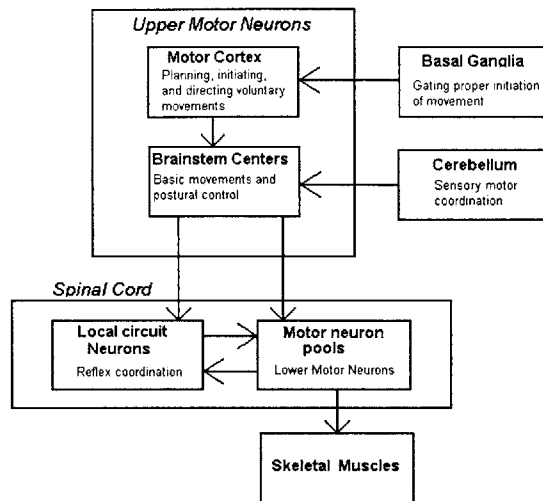


Figure 2-1: Central nervous system organization of motor control.[30, 8, 9]

in motor deficits.

The decision to move is made in the cerebral cortex, and motion is then initiated by the primary motor cortex (M1). The motor cortex commands actions of the brain stem and some areas of the spinal cord, which controls execution of low level motions. Additional regulation is performed by the cerebellum, which helps to coordinate muscular actions. The cerebellum also reports back to the motor cortex with the final position. More complete descriptions of brain motor control can be found in [8, 30].

One poorly understood neural region shown in Figure 2-1 is the basal ganglia. It is involved in the initiation of movements, but has no direct control of the lower motor neurons. Instead it acts by regulating activity of the cortex. Disorders of the basal ganglia include Parkinson's disease and Huntington's disease.

The basal ganglia are noted here because of a similarity seen in this study between stroke patients and Parkinson's patients. It has been well documented that Parkinson's patients are capable of many motor activities, such as walking, but that they have difficulty initiating motion. This lack of movement is known as *hypokinesia*. This may be similar to the phenomenon seen here in stroke patients, the ability to generate strong muscular contraction, but not to initiate it independently.

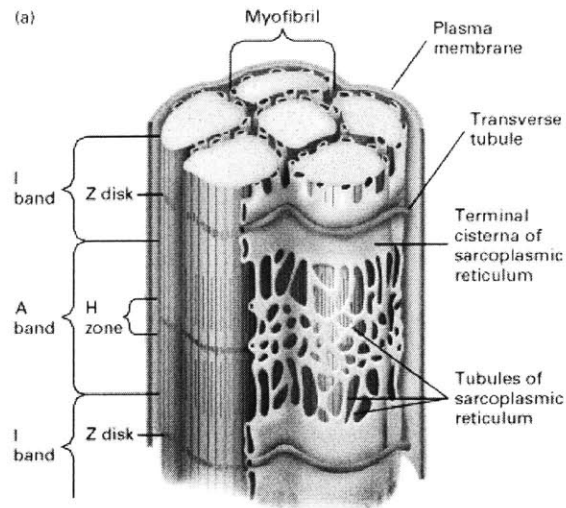


Figure 2-2: A short section of muscle fiber. From [30].

### 2.1.2 Mechanisms of Muscle Activation

A brief look at the physiology of muscle will aid in understanding the mechanism of electromyography. An illustration of a muscle fiber is shown in Figure 2-2.

Skeletal muscles are composed of fibers, which are long multinucleated cells containing *myofibrils*, the individual contractile structures in muscle. The myofibrils are surrounded by the *sarcoplasmic reticulum*, an extensive network of channels which sequester and store  $\text{Ca}^+$  ions. The whole fiber is encased in an electrically excitable cell membrane called the *sarcolemma*. The sarcolemma is continuous with a network of tubules throughout the fiber called the *transverse tubules* which are electrically excitable channels of extracellular fluid within the cell, much like inside-out axons. Like axons, the sarcolemma and transverse tubules contain voltage gated sodium channels, and active sodium/potassium pumps to maintain the membrane polarization.

Muscular contraction is directly controlled by alpha motor neurons, each of which has its body in the spinal cord and an axon which innervates it's muscle fibers. A *motor unit* consists of an alpha motor neuron and all the muscle fibers that it innervates. A muscle contains multiple motor units, which are the quanta of contraction.

A motor unit may contain from 3 to 1000 muscle fibers, and usually the alpha motor neuron axon size is proportional to the number of fibers. For reference, research suggests that the number of muscle fibers per motor unit in the biceps brachii of an infant is around 700 [7]. Similarly, a muscle can contain any number of motor units. Each motor unit contains fibers distributed throughout the muscle, leading to smoothness and homogeneity of contraction even if very few motor units are activated. All of the motor units which make up a muscle are called a *motor neuron pool*.

Each action potential from an alpha motor neuron acts as an impulse stimulus to the muscle fibers, and triggers a depolarization of the sarcolemma at the *motor end plate*, which causes an action potential to sweep down the sarcolemma in both directions from the motor end plate. The action potential is propagated by the influx of sodium ions, and the subsequent current along the cell membrane. This current causes a voltage to develop, which then opens the sodium channels to depolarize the next segment of membrane, just as in a nerve cell. A more complete analysis and model of action potential propagation is given in [15]. The depolarization of the transverse tubules along with the sarcolemma causes the adjacent sarcoplasmic reticulum to release  $\text{Ca}^+$  into the myofibrils.  $\text{Ca}^+$  then binds troponin and causes the myofibril to contract. For a more in-depth explanation of myofibril contraction and structure, please refer to [8, 9, 30].

When a motor pool receives a signal to contract, either from the upper motor neurons or from a reflex arc, strength of the contraction is graded in two ways. One is the number of motor units recruited, and the other is the frequency of action potentials. In response to a stimulus, smaller alpha neurons depolarize more easily due to their larger surface area to volume ratio. This results in the *size principle* which says that smaller motor units are recruited first and fire more often, and larger motor units are recruited later, when more force is needed. This explains why motor control is finer when dealing with small loads.

There are additional distinctions between large motor units and small motor units (sometimes referred to as *fast fatiguing* and *fatigue resistant*, respectively). Fast fatiguing motor units have large muscle fibers, large motor neuron axons, and lower lev-

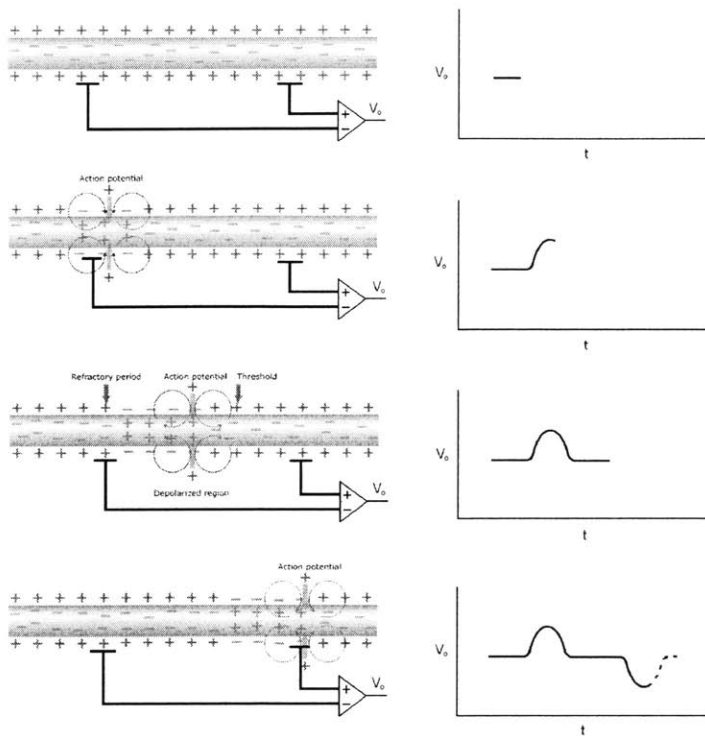


Figure 2-3: Diagram of how differential electrodes on the surface of a muscle fiber measure an action potential as it propagates. The action potential recording from this setup is biphasic.

els of mitochondria. This results in faster nerve conduction velocity, higher threshold for excitation, slower motor neuron firing rate, stronger and faster contraction, and faster fatigue due to primarily glycolytic metabolism. Conversely, fatigue resistant motor units have smaller muscle fibers, smaller neuron axons, and more mitochondria for oxidative metabolism. These motor units have the opposite properties, they have slower nerve conduction velocity, a lower threshold, higher firing rate, slower and weaker contraction, and slower fatigue due to oxidative metabolism. All of these properties can, in part, be attributed to size and mitochondrial concentration.

## 2.2 The Basis of Electromyography

Electromyography is based on the linear propagation of action potentials along a muscle fiber, as described in Section 2.1.2. Most commonly, the electrodes are attached to a differential amplifier, which measures the difference in potential at two locations along the length of the fiber. Figure 2-3 demonstrates how differential electrodes detect an action potential. Electromyography can be performed using electrodes on the surface of the skin (surface EMG or sEMG), or using needle electrodes inserted into the muscle. Needle electrodes detect action potentials in one or a few fibers, whereas surface electrodes measure the summation of all action potentials in the muscle fibers within their recording area.

Detection is based on the relative difference in surface voltages as the action potential moves. Notice that the placement of electrodes is very important. If the electrodes are too close together, then no action potential will be recorded, as the voltage at both electrodes will change simultaneously. However, greater distance between the electrodes also causes more filtering to occur. If a series of action potentials has a wavelength equal to the distance between electrodes (if the inter-pulse distance is the same as the inter-electrode distance) then no differential will be recorded. This is true for all integer multiples of the wavelength. As a result, the further apart the electrodes are, the lower the fundamental filtering frequency will be.

Additionally, it is important that the electrodes not be placed directly over the motor end plate. While it is somewhat natural to think that placing the electrodes over the belly of the muscle will result in the best measurement, this is actually not true. If the electrodes occur on opposite sides of the motor end plate, then as the action potential moves away from the plate in both directions, it may pass both electrodes at nearly the same time. In this case there is little or no differential measurement between the electrodes, and no voltage is recorded.

Further explanations and analysis of EMG can be found in [7, 38].

## 2.3 Biological Effects of Stroke

Stroke is defined as a "clinical syndrome characterized by rapidly developing clinical symptoms and or signs of focal and at times global loss of cerebral function, with symptoms lasting longer than 24 hours or leading to death, with no apparent cause other than vascular". Approximately 85 percent of all strokes are ischemic strokes, where blood flow to a portion of the brain is occluded resulting in neuronal death. The remaining strokes are hemorrhagic, resulting from a ruptured blood vessel in the brain. As discussed earlier in Section 2.1.1 there are many parts of the brain which contribute to motor function, and which can cause motor deficits if injured. Neurons require tremendous nutrition to remain active and alive, and any interruption to the brain's blood flow results in almost immediate injury. Ten seconds without cerebral blood flow will result in unconsciousness. After stroke, approximately 80 percent of sufferers have motor symptoms, usually confined to one side (the *contralesional side*—the side opposite the stroke lesion). Symptoms typically include some combination of weakness, ataxia, "heaviness", and/or "clumsiness" on the affected side [40, 30].

Predicting the eventual extent of motor recovery is extremely inexact<sup>1</sup>. Our understanding of motor recovery has been largely observational, because only recently have researchers been able to directly study brain function in humans, and therefore learn more about the biological mechanisms behind recovery. For a comprehensive review of these studies and their findings, see [11, 32]. Overall, altered excitability in primary motor cortex on the lesioned side has been observed in the weeks immediately following stroke, along with the hypotonia indicative of spinal shock. Physiologically, initial recovery can be attributable to the reduction of edema and the restoration of circulation to the affected regions.

Later, patients often develop hypertonia, attributed to amplified reflex activity in the spinal cord. The cerebral cortex is thought to exert an inhibitory influence on spinal reflexes, which can be negated by its injury. Depending on the size of the lesion, the patient may experience a return of function on the affected side. It is not

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<sup>1</sup>Researchers disagree whether age, side of stroke, and initial recovery are accurate predictors of eventual recovery. Some of the varied findings can be found in [32, 12, 6, 20, 31].

exactly known how recovery occurs. Some tissues may have recovered. Additionally, some areas may be remapped to control the lost functions. This can include areas directly surrounding the lesion, as well as the corresponding areas in the opposite hemisphere [11, 33].

In the arm, occupational therapists and doctors at Spaulding Rehabilitation Hospital all agreed that stroke patients typically regain bicep function before tricep function, and that many stroke patients never regain tricep function. Bicep recovery is typically much stronger than tricep recovery. Bicep spasticity is also common among chronic stroke patients, making it difficult to train functional arm movements. Tricep function in the acute phase is considered a good indicator for eventual recovery.



# Chapter 3

## Current Medical Therapy

This study shows that therapists may find EMG useful in the evaluation of patients and in the design of therapy. EMG can be a more sensitive indicator of muscle activation than force output. It is already used for this in several rehabilitation technologies, and can find further applications in force feedback rehabilitation robots. As doctors and therapists learn more about the neural, muscular, and neurological activation which results from different activities, they can begin to build therapy regimens with that information in mind. Therapy can be designed to create the activation patterns associated with stroke recovery.

### 3.1 Current Therapy for Stroke

Standard therapy for stroke patients usually consists of 30-60 minutes per day of occupational or physical therapy, decreasing over the initial 6 months, and discontinued after that point. However, this can vary widely depending on the institution, the patient's means, and the patient's impairment. Some patients receive "intensive" motor rehabilitation, which means that they have more time per day in therapy. Some studies have shown that this modestly improves functional outcomes [32, 35]. A variety of approaches have been used and studied experimentally in this intensive rehabilitation, the studies are discussed in Section 3.2.

Normal therapy is provided by occupational and physical therapists, and typi-

cally falls into two categories, general *neurofacilitation techniques*, and *task-specific techniques*. Therapists begin with general neurofacilitation, to get return of muscle function, and then balance and gross movement. They subsequently move on to more specific training.

In neurofacilitation techniques, the therapist attempts to retrain motor control. The goals are to inhibit abnormal movement and spasticity, and encourage normal movement through developmental approaches. It is very important that patients not develop abnormal compensatory movement patterns early on. Therapists focus initially on isometric control, including weight bearing and joint position sensation. As one therapist said, “We try to bombard the sensory system with information”. Later, the therapist moves on to eccentric control, allowing muscle relaxation to control movement. Finally, the therapist will focus on the concentric aspect of control, control of contractions (from interviewing therapists and [32]).

Task-specific training includes retraining the normal movements necessary to accomplish independent living, such as transfers, walking, and eating. It also includes finer movement control. In addition, patients may eventually be taught some compensatory methods for tasks which they are still unable to execute normally.

## **3.2 Recent Studies on the Effects of Stroke Therapy**

In the last decade, the development of functional brain imaging techniques (high resolution brain imaging techniques that can be performed while the patient is able to move around) has allowed researchers to begin understanding the neurological progression leading to recovery from stroke. Current research aims to determine which factors are good indicators of eventual recovery, which activities can help lead to recovery, and what functional and neurological recovery in the brain actually entails.

Thus far, the outcomes of such research has not significantly affected the therapy that most stroke patients actually receive. Very few studies have looked at the ef-

fects on the brain of particular therapeutic approaches or activities. However, a few intensive rehabilitation techniques have been studied using these techniques.

*Robotic-assisted therapy* uses a specially designed robot, which instructs the patient to execute therapeutic exercises and sometimes assists by helping the patient to move [14, 16, 24]. Robot-assisted therapy is of particular interest for this study, because many robots cannot detect near zero force output. However, a more sensitive measurement of muscle activation could make robots more responsive to patients. They could also be trained to facilitate muscles, in much the same way that a therapist does. *Bilateral therapy* consists of performing standard rehabilitation exercises with both hands simultaneously [27, 23]. In *Constraint induced therapy*, a patient who has deficiency, learned disuse, or neglect on one side wears a sling or other restraint on the healthy arm, forcing use of the impaired arm [36, 21].

Additionally, a few studies have looked at changes in the motor evoked potentials (MEP)<sup>1</sup> generated by transcranial magnetic stimulation (TMS) during various neurofacilitation approaches [17, 18, 26]. The purpose of these TMS studies is to find which actions lead to the successful transmission of neurological events from the brain to the muscles. In TMS studies, the neurological event is created externally by a magnet, not by the patient.

With a better understanding of what kinds of activation patterns indicate and lead to better recovery, therapists and doctors can begin to design therapies which target those regions of the brain. In order for them to make well-informed designs though, therapists will also need to know what activation occurs during various activities, so they can assess its effectiveness as a therapy. Of the previously mentioned therapies, only the TMS studies have been used on patients with severe paresis. The effectiveness of therapy is difficult to assess if there is no motion of the affected limb. By using a more sensitive measure of muscular activation, such as EMG, the therapist can help to better determine the efficacy of an exercise, even when no motion is present. Additionally, with the possibility of robotics which assist and facilitate voluntary motion, patients with more severe impairments may be able to take advantage of

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<sup>1</sup>An MEP is the same as an EMG signal.

therapies like constraint induced therapy, and prevent the learned disuse and atrophy which accompany paresis.

### 3.3 Current Medical Uses of Electromyography

Electromyography has been used to observe muscular phenomena since it was invented in the 1850's. Surface electrodes have made it easier to observe muscular activity, allowing EMG to be more widely utilized. EMG is currently used in a number of medically related disciplines. It is used in ergonomics to determine muscular activation. In fact, the definitive text on electromyography by Basmajian and DeLuca [7] contains an impressively complete set of ergonomic studies which elucidate the muscular mechanisms behind many body systems and their motions. Current studies of muscle activation look at changes in the EMG due to muscle tension, position, and fatigue, in an attempt to further understand how various factors affect muscle contractions [29]. EMG is also used by neurologists to diagnose and study neuropathies, it can measure nerve activation and nerve conduction velocities.

In addition to its diagnostic and academic uses, the EMG can be used in therapy and rehabilitation, as a control signal for assistive devices, therapeutic devices, and as an input for biofeedback.

#### 3.3.1 EMG Controlled Orthoses and Prostheses

Surface EMG is currently used to control a number of prostheses, such as the Utah Arm, the WIME hand, the Boston arm, and the Ergoarm<sup>2</sup>. All of these prostheses use EMG signals from muscles in the remaining arm to control the prosthesis. The control mechanism is learned by the patient, it is not based on the EMG resulting from attempts at natural motion. Rather, the EMG is located to get optimal signal strength from muscles that the patient can isolate and control easily.

There are no widely used EMG controlled orthoses, partially because the market is much smaller and the task more difficult. To make an orthosis that feels natural, using

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<sup>2</sup>For more information about these prostheses, see the product literature at [5, 1, 2, 4]

EMG, is harder- most powered orthoses use force feedback to regulate movement. However, for stroke patients who cannot generate force, this method is ineffective. The MIT Active Joint Brace is an experimental powered orthosis that uses EMG signals and force feedback together, and could potentially benefit from improved EMG detection in stroke patients.

### **3.3.2 EMG Controlled Functional Electrical Stimulation**

A recently developed area of EMG applications is the use of indwelling EMG electrodes to control functional electrical stimulation (FES), such as in the BION2. FES works by applying an electrical stimulus to a muscle, causing the muscle to contract. FES can be applied either via implanted electrodes or via external electrodes applied to the surface of the skin. EMG controlled FES can give functionality in a number of different circumstances. In spinal cord injury patients, the FES electrode can deliver muscular stimulus larger than the injured or severed nerves are capable of carrying. In this cases, the EMG signal is not picked up from the target muscle, but instead from other muscles trained as controls, or directly from nerves above the point of injury. The goals of such a system are to promote muscular control and strength in a muscle that would otherwise be impossible to activate. These devices are typically permanent implants, and have not been widely applied, due to the risks associated with their implantation.

FES and EMG are also used together in the biofeedback system Neuromove [3], which monitors EMG, outputs the EMG visually, and "rewards" the patient with a muscular contraction for each successful EMG signal. Neuromove is a rehabilitation aid, and not a functional aid. In clinical trials this method has proved somewhat effective at restoring arm function [3].

### **3.3.3 Biofeedback**

Biofeedback takes EMG signals and makes them visible or audible to the patient. The goal is to help the patient gain greater control of his/her muscles volitionally, by

allowing him to see or hear what the muscle is doing with every thought or attempt. For stroke patients, the most common use of EMG biofeedback is to try and eliminate involuntary spasticity by practicing relaxation.

# Chapter 4

## Experiment Design

As discussed earlier, the goal of this experiment is to develop a quantitative measure of a patient's ability to control the muscular output of his or her arm. A comparison can then be made between two different types of arm movement attempts, those made independently, and attempts in response to facilitation.

### 4.1 Experiment Protocol

Consultation with therapists indicated that patients often had trouble pushing an object (such as a door or a block) and might not achieve the desired muscle activation if asked to push against a scale or other inanimate object. One occupational therapist said, "Activating a muscle really requires facilitation, you have to feel what the patient is responding to." To achieve this, the experiment is built around an actual patient/therapist interaction.

Each experimental session was essentially a truncated therapy session with the therapist and the patient, and with the addition of an sEMG recording and a video recording. The sEMG would be recorded continuously for the entire duration of the session.

Each patient was informed of the experiment, and then signed a consent form. He/She then answered some questions regarding the current condition of each arm, while a cuff was placed over the upper paretic arm, with two electrodes each on the

triceps and the biceps brachii. The therapist for each session was Paul Petrone, Head of Occupational Therapy (OT) on the inpatient stroke ward at Spaulding Rehabilitation Hospital in Boston, MA. Paul would then take the patient through six specific exercises, at least three times each. He would also move their arm, while they were told to relax (passive motion) to determine what involuntary reactions the patient had to facilitation. This would then be repeated on the opposite, healthy arm.

The exercises were chosen to isolate the biceps and the triceps in voluntary motion attempts by the patient. The exercise protocol was:

**Unfacilitated Biceps** From a relaxed position, arm down, attempt to flex your arm ("touch your nose")

**Unfacilitated Triceps** With arm held horizontal (by therapist if necessary), attempt to extend your arm

**Unfacilitated** With hand on the mat or therapist's chair, attempt to push down into the mat (as if lifting body for a transfer)

**Facilitated** With hand on mat, now attempt to push down, but with the therapist supporting the patient's elbow

**Facilitated Passive** Rest for 2 minutes, while the occupational therapist takes the arm through passive motions

**Facilitated Triceps** Push against resistance from the therapist ("Don't let me touch your nose")

**Facilitated Biceps** Pull against resistance from the therapist ("Don't let me straighten your arm")

Each of these was executed three times. Figure 4-1 shows a subject executing bicep flexion, both free and facilitated. During all of these exercises, the patient and the therapist also had the opportunity to look at a real-time display showing the raw EMG data and the rectified EMG amplitude for each muscle. If the patient





Figure 4-1: The researcher demonstrates free bicep flexion and facilitated bicep flexion, as done by the subjects, with the therapist. The computer is providing biofeedback from the cuff on the subject's arm. Images taken at Spaulding Rehabilitation Hospital.

had hemi-neglect, the display was placed on the healthy side, such that he/she could observe it. Figure 4-2 shows a screenshot of the display.

Surface EMG (sEMG) voltage recordings are, as described in Section 2.2, a direct result of the physiological mechanism of muscular contraction. EMG is not a direct measure of force output, but in healthy patients it is highly correlated <sup>1</sup>.

## 4.2 Subject Selection

Subjects were seven acute stroke inpatients (the stroke had occurred less than 4 weeks prior to testing) at Spaulding Rehabilitation Hospital. All patients had some impairment of movement on the contralesional side. The level of impairment varied greatly, from no volitional movement to almost full range of motion, but with decreased strength and control.

Acute patients were chosen for several reasons. They have no long-term tone effects. They do not have atrophy on the paretic side yet. They all have similar

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<sup>1</sup>The correlation between EMG and force output has been independently determined by a number of researchers, who disagree whether the relationship is linear or of higher order [7]

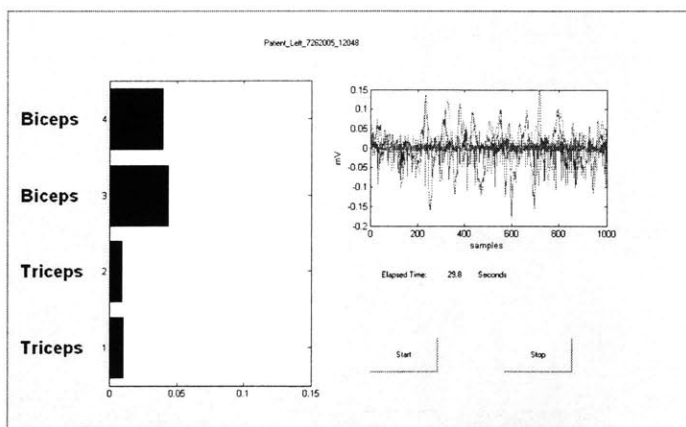


Figure 4-2: A screenshot of the graphical interface each patient was able to see during the therapy session. Bars on the left indicate RMS of the EMG signal, and the plot on the right is the raw EMG.

post-stroke timelines, they have all had similar frequency and intensity of therapy, and approximately the same amount of time has elapsed post-stroke<sup>2</sup>.

All subjects had sufficient attention to remain focused on the therapy for the entire duration of the session. They all had sufficient motivation to attempt each exercise.

#### 4.2.1 Patient Controls

As discussed in Section 2.2, Surface EMG amplitude and frequency measurements are significantly affected by subcutaneous composition, skin type and moisture, muscular density, and location [38]. Because these factors vary substantially from patient to patient, comparisons between patients are difficult. For this study most comparisons are between values for a single patient, using a patient’s unaffected arm as a skin-type matched control. Several studies have shown that activation of primary motor cortex by unaffected arm movements does not depend on the stroke-affected side of the brain, thereby making it a reasonable “healthy control” for comparison [25].

<sup>2</sup>Additionally, their therapy is the most likely to be affected by changes, because it is frequent and held at the hospital. Given our interest in potentially assisting recovery through resistive inpatient therapy, they are the most relevant population to study.

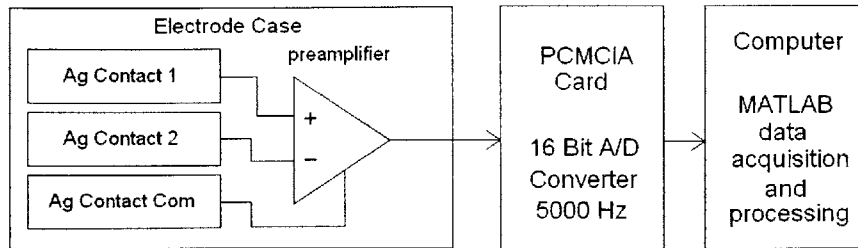


Figure 4-3: A block diagram of the hardware used in recording the EMG for this study. Block 3 is illustrated in more detail in Figure 5-1.

### 4.3 Design and Construction of the Experiment Setup

Surface EMG is a technique that has been in use since the 1960's, and thus is fairly well understood. The basic physiological mechanism behind sEMG was explained in Section 2.2, but here are presented some of the issues involved in recording and interpreting the sEMG signal. A system diagram of the hardware used in this experiment is shown in Figure 4-3. The software processing done in the third block is shown in detail in Figure 5-1.

#### 4.3.1 Choices Regarding Signal Recording and Analysis

For this study, the biceps brachii and the triceps brachii were recorded, the two primary muscles for gross movement of the elbow.<sup>3</sup> Electrodes recorded a medial and a lateral signal for each, in order to obtain the best location. According to Basmajian [7] both the long and the short head of the the biceps are activated during flexion and isometric contraction, as are the medial and lateral heads of the triceps during contraction, so activation should be recorded for virtually all motions attempted. Electrodes were placed between the insertion tendon and motor end plate, and fastened tightly with a high pressure strap for the most robust signal recording [7].

<sup>3</sup>The brachialis and the brachioradialis are also gross elbow flexors, and the anconeus an extensor, but they are not close enough to the skin to record using surface electrodes.

## 4.4 Data Collection and Analysis

The EMG was recorded using silver contact, double differential electrodes. The signal was recorded using a Measurement Computing PCMCIA Analog-to-Digital converter in conjunction with the Data Acquisition Toolbox in MATLAB. The analog signal was sampled at 5000 Hz and converted into 16 bit digital values. The recording was continuous for the duration of exercises on each arm, allowing for between-exercise analysis, and making very accurate baseline determinations possible.

Data was analyzed in MATLAB. The video record was observed in Windows Media Player. Event times were recorded, and then correlated with the EMG record in MATLAB.

EMG filtering was done in MATLAB using 5-30 second segments of the record, depending on the activity length. Frequency content was analyzed by performing a 25000 point (5 second) Fast Fourier Transform (FFT) on the data.

Basmajian recommends using the Root Mean Square (RMS) of the EMG as the primary signal for comparison, because it contains the most physiological information about the firing of the muscle [7]. The RMS of muscle signal  $m$  at time  $t$  over  $2n$  samples is computed as:

$$RMS[m(t)] = \left[ \frac{1}{2n} \sum_{k=t-n}^{t+n} m^2(k) \right]^{\frac{1}{2}} \quad (4.1)$$

Basmajian further recommends that the RMS be computed over a time window of 100-200 ms. For this experiment, the sampling rate was 5000 Hz, and the window chosen was 200 ms, so  $n = 500$ .

A binary threshold can then be set to detect when the patient has contracted a muscle, or a graded RMS value can be used to determine the desired force output of the muscle.

Specifics of analysis are included in Chapter 5 for ease of reference.

# Chapter 5

## Results

### 5.1 Observations of Patient Force Output

Patients were grouped according to their free motion abilities into three categories: little or no motion, moderate motion, and full motion. Out of nine patients, six had little or no unassisted motion of the arm on the paretic limb. One patient could execute bicep flexion, but could not extend using the triceps, and was classed as having moderate motion. Two patients had almost full range of motion, but reduced control.

After each facilitated action, the therapist was asked if he could feel resistance, and whether it was weak (barely discernable) or strong. During facilitated flexion, all nine patients produced strong resistive force.

During facilitated extension, seven out of nine patients produced strong resistive force, and one patient produced weak force. In one patient, the therapist was unable to engage the triceps. Additionally, in one patient with full motion, the therapist thought that the paretic triceps felt stronger than both the paretic bicep and the healthy triceps.

## 5.2 EMG Results

Seven patients had full EMG records from their recording sessions. Patients with an incomplete record were not analyzed, because they could not be normalized or filtered properly.

As discussed in Section 4.2.1, surface EMG measurements are affected by a number of factors including subcutaneous composition, individual skin characteristics, moisture, and the tightness of the contact. As a result, in the following analysis of the EMG each patient is compared to himself, but not directly compared to any other patient. Common normalization practices are also discussed in section 5.2.1.

### 5.2.1 Magnitude of the EMG Signal

For this study, the potential generated by some patients was very small compared to the noise floor, so an additional bandpass filter was used to improve the quality of the signal. Figure 5-1 shows the filters that were applied to the signal, and the resulting changes in the signal output. The signal was first bandpass filtered through an eighth order Butterworth filter with a passband from 60 Hz to 500 Hz. This attenuated the movement artifact, which had previously dominated the signal during many actions, by at least 40 dB. Movement artifacts occur because patient movements cause the electrodes to move across the skin. It also attenuated high frequency noise and muscle jitter. The signal in Figure 5-1 shows a patient attempting motion at  $t = 876$  and  $t = 881$ . The patient succeeds in mild pronation of the affected arm, but no change in elbow angle. A movement artifact can be seen originally at  $t = 870$  due to repositioning of the patient's arm. That artifact is eliminated in the output of the filter. After the RMS is computed the data can be low pass filtered to extract unique peaks for each contraction.

This method of filtering gave significantly improved estimates of when patients made movement attempts. More importantly, it virtually eliminated noise that would previously have been interpreted as patient attempts. One drawback to filtering is that it attenuates a wide range of signal bandwidth where there is muscular activity,

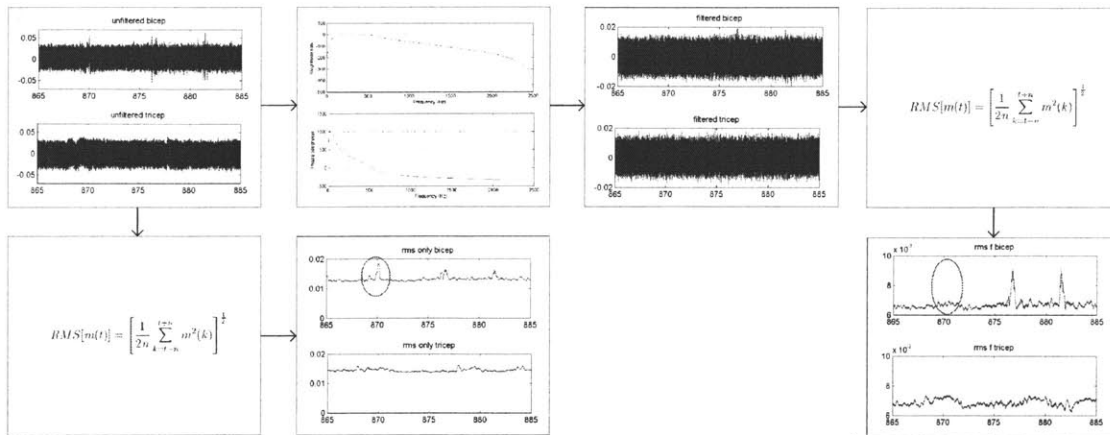


Figure 5-1: A schematic diagram showing the filters used in analysis of the EMG data. Filters are shown applied to a sample from a patient with almost no angular motion. Patient attempted bicep flexion at  $t = 876$  and  $t = 882$ , but succeeded only in pronating the wrist. Ovals show where the bandpass filter removes the undesirable electrode movement artifact at  $t = 870$ .

particularly below 60 Hz. This results in lower peaks. Nonetheless, for eliminating false positives, it is necessary.

Typically, to compare EMG between patients, all RMS magnitudes would then be normalized to a percentage of the Maximum Voluntary Contraction (MVC). One finding of this study is that the MVC is a very poorly defined parameter in stroke.

Throughout the literature, defining the MVC is not a well standardized procedure [7, 38]. Most researchers ask subjects to push (or pull) against a resistive force, usually immobile, to measure the maximum isometric contractile force that the subject can voluntarily generate.

In stroke, the MVC is an ill defined parameter because it varies within a patient. The word *voluntary* presumes that the subject has full neurological control, not always the case in stroke. A paralyzed patient may not be able to move her arm against gravity at all. Thus, that attempt is one kind of maximal voluntary contraction; she is voluntarily trying to lift her arm but all her strength this way is insufficient. Asking her to lift a box or a weight produces a similar result.

Using a different activity, such as pushing down on the seat of her chair, a different

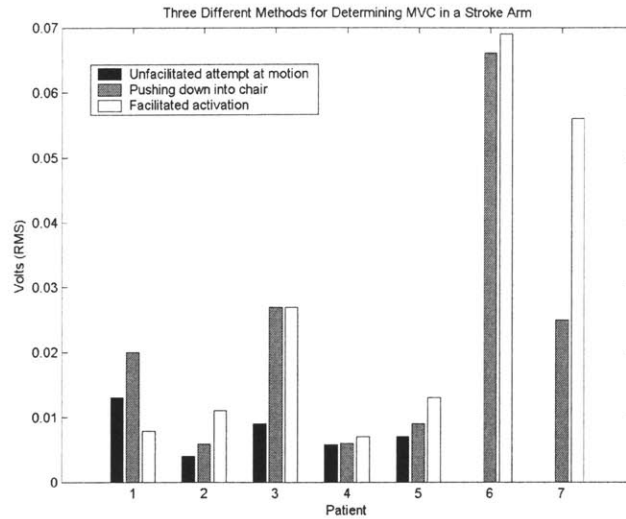


Figure 5-2: Three different methods for determining MVC in a stroke impaired arm. In most patients, they result in different values. Patients 6 and 7 are capable of voluntary motion, therefore unfacilitated motion for them is not an MVC.

level of muscle activation is often possible. Finally, almost all the patients could make an even more forceful contraction if pushing against the therapist's hand, with the therapist attempting to engage the muscle or *facilitating* the contraction. In all three of these cases, the patient was asked to push as hard as possible, thereby making each an MVC for the same muscle. Yet the three activities generate different levels of activation. Figure 5-2 shows the maximum activation for each patient, on the paretic side, under each of the conditions. Patients 6 and 7 exhibited weakness but not paralysis, they were able to move this muscle voluntarily, therefore free motion was not a maximum contraction for them.

These measurements show that stroke patients have a varying ability to activate a muscle, depending on the activity. In addition, to be sure that the contraction was voluntary, and not the result of a brain-independent reflex, the EMG amplitude during similar, passive motions was recorded. These were dominated by electrode movement artifacts, and showed that the above results are not from a reflex.

Figures 5-3 and 5-4 show the EMG measured activation levels of patient triceps and biceps, respectively. Observe that in all patients, activation was greater when



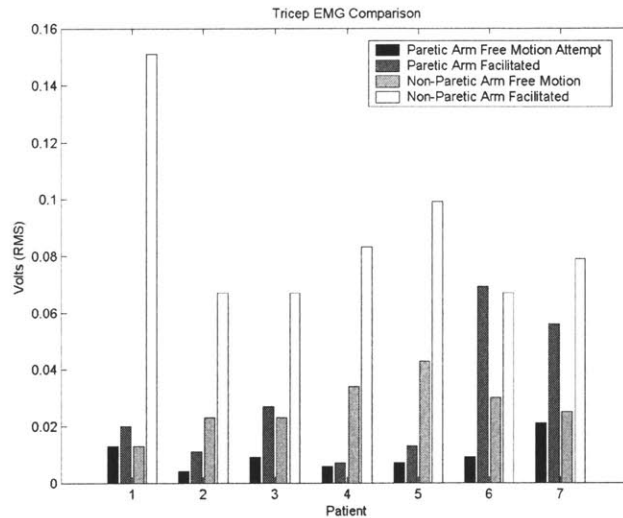


Figure 5-3: Triceps EMG magnitude during different activities. Only patients 6 and 7 are capable of moving their arm using the triceps, notice their magnitude of facilitated activation on the affected side.

facilitated than when not facilitated, for both muscles.

Further examination of the data shows that amplitude of the EMG for facilitated tricep activation on the paretic side is well correlated with the ability to move the arm using triceps. Only patients 6 and 7 have functional triceps. Figure 5-3 shows the tricep EMG activation measured in each patient. Based only on force judgements, the therapist stated that patient 6 had stronger triceps on the affected side, which can also be seen in the figure, showing a good correlation between EMG measurements and force output.

No clear correlation with movement ability existed in this data set for bicep movement. Patients 5,6 and 7 all possessed voluntary bicep movement. Figure 5-4 shows bicep EMG measured for each patient. It is possible that the high levels of bicep activation in some non-moving patients indicates a near future recovery of bicep control, since bicep recovery is typically earlier and more widespread than triceps recovery. However, another study would be required to determine if this is true.

Figure 5-4 contains another comparison on the right, between the healthy and the paretic arm. Movements of the healthy arm can indicate how much EMG activity

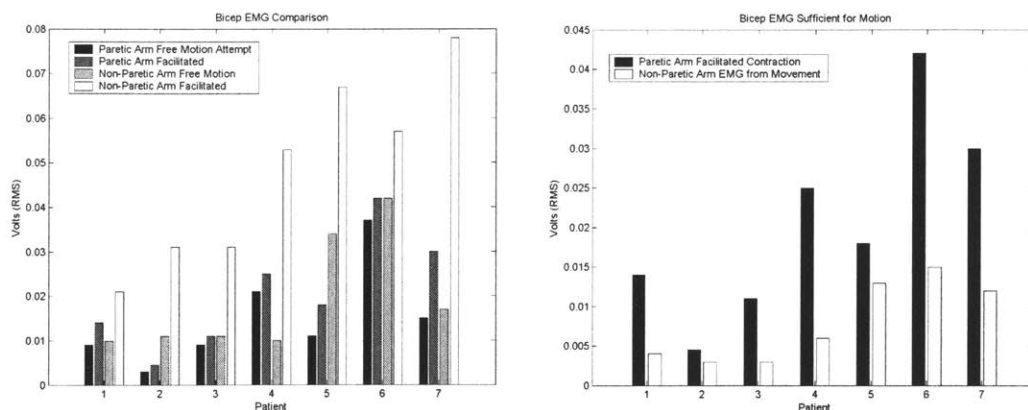


Figure 5-4: *Left*: Biceps EMG magnitude during different activities. No unique correlation can be seen between ability to move (patients 5, 6, and 7) and the measured EMG. Correlations with force output were not measured. *Right*: Comparison between EMG measured during facilitation on the paretic side, and during movement on the non-paretic side. Note that every paralyzed arm could produce an EMG with a magnitude greater than the EMG corresponding to movement.

corresponds to motion of the arm. That is, how much muscle activation is necessary for that particular patient in order for the arm to move. During bicep facilitation, all patients had an EMG of magnitude larger than the EMG necessary for motion. Comparison of bicep EMG in the right-hand plot of Figure 5-4 shows that in every patient, the EMG of the paretic arm during facilitation is greater in amplitude than an EMG of the healthy arm when moving without resistance. The healthy arm EMG values in this plot are the minimum EMG values that accompanied motions, not mean values.

### 5.2.2 Frequency Composition of the EMG Signal

The RMS indicates how many motor units are engaged, and the frequency content of the EMG can indicate the firing rate of the motor units, as well as the rapidity of contraction onset. The motor unit firing rate can indicate which motor units are being used (fast or slow) and if motor units are becoming fatigued.

Frequency composition was determined by performing a 25000 point FFT in MATLAB on the 5 second window including the contraction. Most samples contained a

spike at 60 Hz, due to line noise. This was removed for calculations of mean frequency. The mean frequency  $[f]$  of signal  $m(t)$  with FFT  $M(f)$  is computed as

$$[f] = \frac{\sum_{f=0}^{500} f \cdot M(f)}{\sum_{f=0}^{500} M(f)} \quad (5.1)$$

Equation 5.1 calculates the average of all frequencies between 0 and 500 Hz. If a patient has very little or no EMG signal due, then the average is  $\approx 250Hz$ , due to the broadband noise. This is not an accurate representation of the frequency content of the contraction, so patients with negligible contraction are omitted in the following analysis.

The literature shows that as a contraction is maintained at a constant intensity, the frequency content of the EMG becomes lower [7, 34]. The exact causes are unknown. In addition, as the intensity of a contraction is increased, the frequency also becomes lower. This is likely due to the recruitment of larger, stronger, fatigue resistant motor units, which have a lower firing frequency, explained in Section 2.1.2.

For this study, patients executed a sub-maximal contraction (free) and a maximal contraction (facilitated) on each side. The frequency shift between maximal and sub-maximal contractions was calculated for both arms. The average difference in mean frequency on the healthy side was calculated to be 20.5 for the biceps and 22.1 for the triceps. The shift may be due in part to the method of calculation, equation 5.1. Due to the lower SNR during a minimal contraction, the frequency distribution of the noise has more of an impact on the mean frequency, moving it closer to 250 Hz.

Patients exhibit a similar frequency shift on the paretic side even though they do not necessarily produce motion with the free contraction attempt. The average difference was 17.4 Hz for the biceps and 18.9 Hz for the triceps<sup>1</sup>. An example of the frequency shift can be seen in both categories of patient in Figure 5-6.

Additionally, all the patients with little or no motion showed a lower mean frequency in paretic triceps versus their healthy triceps. The patients who could move

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<sup>1</sup>negligible contractions were omitted, therefore the biceps average is based on only 5 patients, and the triceps is based on only 3 patients

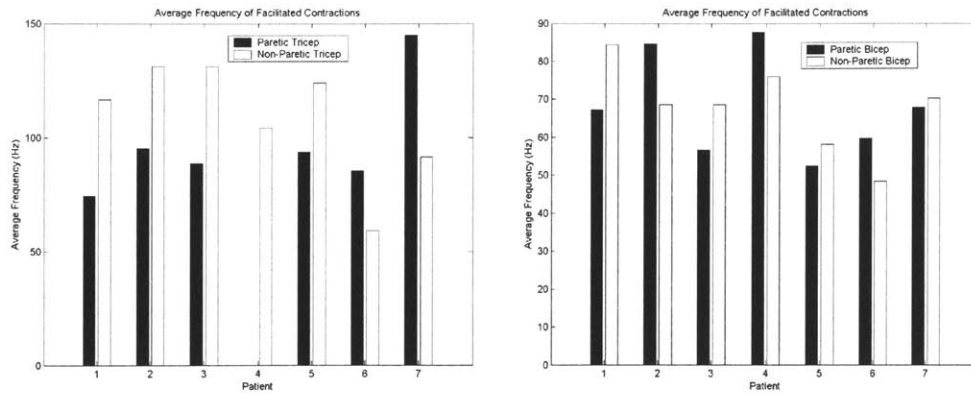


Figure 5-5: The mean frequencies of facilitated motion, compared between the paretic and non-parietic arms. Notice that for the triceps (left graph) the paretic mean frequency is significantly lower in all patients with no tricep function (patients 1 through 5).

their triceps did not have this. Also, the same downward shift applied to several patients for their biceps, but it was not universal. This is shown in Figure 5-5. A comparison of individual frequency spectra from two patients is shown in Figure 5-6 to illustrate these frequency differences. The patient on the left is unable to move either the biceps or the triceps, while the patient on the right can move both. Both patients show lower frequency content when motions are facilitated (opposed) than when motions are not facilitated. Notice that the patient without motion is missing the highest frequencies in the paretic arm. This is true under both facilitated and unfacilitated conditions.

One final observation is that the triceps tend to have a higher frequency EMG than do biceps. Figure 5-7 shows this relationship in healthy arms. This could result from the physiology of the muscles, it is possible the proportion of slow and fast motor units is different between the two muscles.

This could possibly be related to the difficulty many stroke patients have regaining tricep function, compared with bicep function. If stroke causes a patient to lose some of the higher frequency drives to motor neurons, and the triceps require higher frequencies to operate, then stroke patients would have more difficulty activating their triceps.

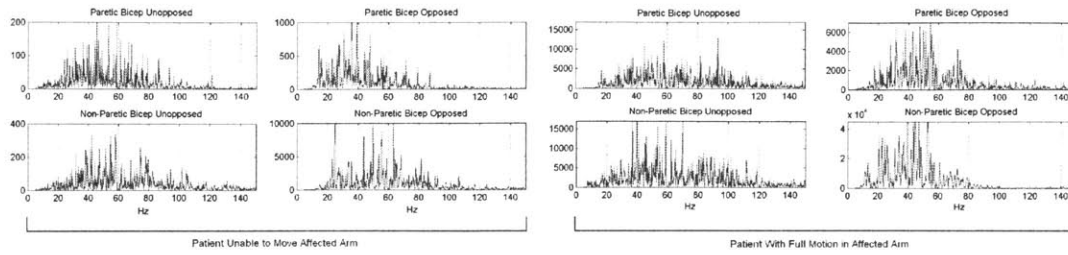


Figure 5-6: Sample frequency spectra from two patients. The 4 spectra on the left are from a patient who cannot move using either the triceps or the biceps. Notice that the frequencies in the paralyzed arm (top row) are lacking some of the high frequency components present in the healthy arm (bottom row). The 4 spectra on the right are from a patient who has almost full motion in both directions. Both arms exhibit more similar proportions of each frequency. Notice that for both patients, the frequency shifts down as intensity of contraction increases.

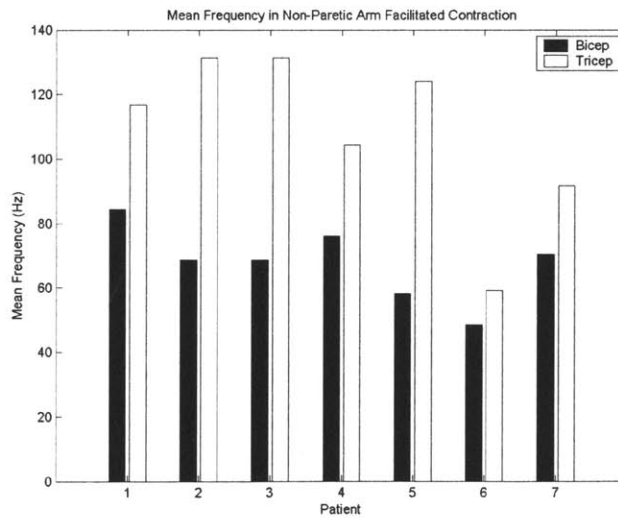


Figure 5-7: Mean bicep versus mean triceps EMG frequency. Measurements from the healthy arm are shown. Note that the triceps has a higher mean frequency than the biceps.

## **5.3 Biofeedback Effects**

From watching the patient-therapist sessions, it became apparent that having the EMG displayed had an effect on the participants. A few observations follow.

### **5.3.1 Patient Biofeedback Effects**

All of the patients who had sufficient attention to watch the screen while producing a contraction were surprised and motivated by the feedback from the screen. Most had become discouraged by their apparent inability to control their paretic arm, but this demonstrated to them that they do have some connection, something to build on. Patients seemed more interested in doing further repetitions and exercises when observing the output on the screen, and saw the potential for recovery. One patient kept doing exercises after the therapist was done, excited by the amount of activity she could initiate.

### **5.3.2 Therapist Use of Visible EMG Signals**

The therapist also found the EMG display useful. Several times he chose patients because he was curious how much activity they had, particularly in the triceps, when he could not feel any activation in the physical evaluation. He used the EMG display to determine when an exercise was successfully activating a muscle, even if the force produced was too small to discern. For example, he discovered that one exercise did not produce any muscle activation in any arm, and thereby learned that it may not be accomplishing what he thought. During one session, a patient was having trouble activating a muscle. After the protocol was finished, the therapist tried a wide variety of exercises, to see if he could get the muscle to respond. It also helped him to encourage the patient, he could tell when they were actually trying, instead of losing focus.

# Chapter 6

## Discussion and Future Work

### 6.1 Facilitation

The results clearly show that stroke patients can push harder when they are pushing back, that facilitation increases the amount of muscle activation in the stroke arm. The mechanism and the reason for this are unknown.

One hypothesis is that the brain requires some proprioceptive input to help “re-mind” areas that have been remapped. As the brain attempts to recover and compensate for the damaged regions, the surrounding areas are frequently reorganized to perform new tasks as well as the old, so that the lost function will eventually return. Areas that are being reassigned may have trouble initially responding to their new commands, since the new connections have not yet been ingrained. This need for feedback would not be surprising, referring back to section 2.1.1, the brain’s control of motion is never open-loop.

Research has shown that in the acute phase after stroke, brain activity is highly abnormal. In particular, studies have shown that the cortex and cerebellum on the side of the lesion have reduced activity immediately after stroke. Motor maps on that side are reduced, and the hemisphere opposite the lesion shows increased activity in corresponding areas. Later after stroke, recovery of motor function has been well correlated with a return of activation to the lesioned hemisphere, as well as with the extent of motor map size increase. The mechanisms that assist with these

reassignments are unknown. [32, 39, 11, 25]

Additional research using TMS in stroke patients has shown that various facilitation techniques including proprioceptive inputs, stimulation of related muscles, and patient attention on the related limb increases the MEP response to TMS [17, 18]. This indicates that input from the affected limbs may help “open up” the pathways to the newly assigned cortical areas.

To know if facilitation truly assists with rehabilitation, long term studies of therapeutic approaches must examine the final outcomes and rates of recovery. A different study, using fMRI while patients attempt facilitated and free movements could help to indicate if the facilitation is helping the neural centers make a connection. A long term study based on different therapy regimens could help determine if intensive facilitation assists in motor recovery.

If facilitation is a beneficial aspect of stroke rehabilitation therapy, then further work can help determine what causes the activation. Studying a therapy session using a load cell or other device capable of measuring force could help to clarify what a therapist is actually doing that results in activation. For example, does the therapist need to initially move the arm so the patient can identify it, and then activate it? Is it the position or the force that is essential? How much is needed? EMG studies coupled with force and position measurements can help to document this process. It is discussed further in Section 6.3.

Another possibility is that regulation of motor initiation by the basal ganglia is affected in stroke. This could explain why patients cannot begin motions. This can be studied further through fMRI, and also by studying whether patients are able to continue motions after they have started. For example, a study could see if the patient was able to move the arm, if the therapist gradually decreased resistance during facilitation.



## 6.2 EMG Feedback in Stroke

EMG based biofeedback (BFB) has been used for stroke patients since the 1970's, to help reduce spasticity, as well as to help strengthen muscles and teach movements [10]. It has some successful results, but according to the therapists interviewed, most therapists do not use it because it is expensive, very time intensive working with the patient, difficult to use, and they don't feel it works very well.

The results from this study indicate another way that EMG can be used for therapist mediated feedback, as well as biofeedback. Therapists complained that the audio feedback from traditional BFB systems was difficult to use and interpret. The visual feedback came from an oscilloscope, and was therefore too expensive for most clinics. However, the therapists found the simple, single number "magnitude" visual bar of this feedback system intuitive to understand, and useful, and they noted that patients seemed more interested in it. If a cheap and simple version were available, the therapist said he "would definitely use it, not on a day to day basis, but for evaluations." In addition, it could be used to design therapy routines specific for each of the patients, which resulted in the best activation for those patients.

## 6.3 Impact on Robotic Therapy

As mentioned earlier, facilitation clearly helps increase muscle activity. A long term study to determine if this kind of activation is beneficial to recovery would help doctors and therapists design therapy. The execution of such a study, and similar studies, might be more easily controlled by using a robot facilitator. Furthermore, if the studies show that facilitation significantly helps recovery, robotic facilitators could become common and very useful.

Development of an algorithm to successfully facilitate muscle activity is fundamental to this idea. Robots which could accomplish this exist. For example, the MIT-Manus is an actuator which could be reprogrammed with many different facilitation methods. However, the stroke patients with more impairment cannot move

their own arm at all, therefore they would have similar trouble pushing the facilitator arm of the MIT-Manus, despite its design for backdriveability. With the addition of EMG input, the MIT-Manus (and similar therapeutic robots) could be made more sensitive to slight muscle fasciculations. This could potentially make it more helpful for highly paretic patients.

Another application for facilitation could use the fact that for all patients, the EMG becomes amplified with resistance. This fact can be utilized when taking EMG measurements for control, such as in an active orthosis. It can help to verify whether measurements that appear to be very small fasciculations are attempts at movement, or just artifacts or noise.

Assistive robotics can also use these results in their design. The benefits of well controlled assistive robots are clear, the prevention of atrophy and learned disuse in the acute stage, increased functionality and independence. Such robots can be owned by rehabilitation centers, and used by patients in acute care. For example, the MIT Active Joint Brace, or a similar device, could be used in an acute care setting a number of different ways. It could act as an assistive device, amplifying patient attempts at motion. This function could potentially be improved by using slight resistance to perform patient checks. For example using a small negative motion check to force a larger EMG, as demonstrated by the results, could help the brace determine if motion was desired. On another setting, it could act as a facilitator, taking the patient through repetitions of a facilitated contraction, to help strengthen and build neural connections. In a third setting, worn during a therapy session in a passive mode, it can provide feedback to the therapist indicating patient activation and force output for different activities. It can be used to chart a patient's progress, and design future therapy.

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