

# Training to Improve Volitional Muscle Activity in Clinically Paralyzed Muscles for Neuroprosthesis Control

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**Abstract**— Neuroprostheses are devices that use electrical stimulation to activate paralyzed muscles in a coordinated manner to restore functional movements. These systems utilize a voluntarily-generated command signal for control of function. Current command signals include electromyographic (EMG) activity from muscles above the injury level that remain under volitional control. In individuals with cervical level spinal cord injury (SCI), these signal sources are limited in number. Our recent research suggests that volitional muscle activity from *below* the injury level in individuals with motor complete spinal cord injury may be a viable source of command information. The signals from these muscles are small, and therefore the goal of this study is to determine if training using visual feedback can improve the quality of these muscle signals. Results to date indicate that training with visual feedback can increase both the magnitude and consistency of EMG signals in clinically paralyzed muscles.

## I. INTRODUCTION

SPINAL cord injury (SCI) resulting in paralysis in muscles below the injury level can be extremely debilitating. Specifically, injury at the cervical level impairs hand function in addition lower limb mobility and genitourinary function, limiting an individual's ability to perform tasks of daily living and leaving them dependent on others for care. Neuroprostheses are assistive devices that use electrical stimulation to elicit functional contractions in paralyzed muscles. These devices have been used to successfully restore hand grasp, bladder function, standing, trunk control, or walking after SCI [1, 2]. Current implantable systems allow for restoration of a single function. A more advanced device under development in our lab is designed to restore multiple functions in a single individual [3]. Successful implementation of this system, including control of each function, is expected to significantly improve functional capabilities after cervical SCI.

Manuscript received April 15, 2011. This work was supported in part by the State of Ohio Biomedical Research and Commercialization Program under Grant BRCP 06-22, the National Institute of Health under Grants T-32-EB04314 and U01 NS-069517, and VA Pre-Doctoral Fellowship.

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Assistive devices, such as neuroprostheses, require an input or "command signal" to control the restored function. This command signal must be under voluntary control by the user. Current command signals include myoelectric activity from volitional muscles above the injury level [4]. Unfortunately, for individuals with a cervical level SCI, the volitional muscles available for neuroprosthetic control are limited. Further, as technology advances towards the restoration of multiple functions in a single individual, there is an increased need to consider additional options for command signals.

We have recently discovered that muscles *below* the injury level may be a viable command signal for a neuroprosthesis, even in individuals with motor complete (ASIA A or B) SCI [5]. A feasibility study was completed with twelve chronic SCI subjects, all classified with cervical level motor complete SCI, meaning that they had no visible or palpable movement in major muscles below the level of injury. We found that all twelve (100%) had at least some significant, recordable activity present in lower leg muscles in response to attempted voluntary movements. Typically, no visible movement was seen at all; toes flickered in three of the 12 participants. This ability to detect very small electromyographic (EMG) signals from muscles below the injury level is consistent with anatomical studies that suggest that spinal cord injuries are rarely anatomically complete [6]. Results from the feasibility study identified at least one muscle below the knee in each of the participants that met our criteria for possible use as a command signal for a neuroprosthesis.

The goal of the work presented here is to determine whether or not we can enhance the strength and reliability of these small volitional EMG signals from below the level of injury. While prior studies looked at using biofeedback in muscles at or just below the level of injury in the arm and hand [7, 8], this is the first study to look at muscles considered clinically paralyzed, located well below the injury level in the lower leg. A majority of muscles identified in our feasibility study demonstrated very low amplitude EMG signals, consistent with recruitment of a small number of motor units. No training and minimal visual feedback was provided in the feasibility study. While a small subset of the muscles identified in the study were robust enough for use as a command signal, the majority were inconsistently activated. We expect that it is difficult for individuals with SCI to volitionally contract these muscles because of the absence of visual movement and proprioception. Thus, we performed training studies using

visual feedback of EMG to determine if subjects can learn to improve signal quality and consistency.

## II. METHODS

### A. Research Subjects

To date, two participants with cervical level SCI have been enrolled in the study. Both subjects are at least five years post injury. The first subject has a Brown-Sequard injury at the C3 level. Her injury completely damaged half of the spinal cord and due to the location of the injury, she is motor complete (but sensory incomplete) on the right side of her body and sensory complete (but motor incomplete) on the left. Because our focus is on paralyzed muscles below the level of injury, we only examined muscles on her right side. The second participant has an injury classified as ASIA B (motor complete, sensory incomplete) at the C3 level. All subjects gave their informed consent and all testing procedures were approved by the local institutional review board.

### B. Instrumentation

In a given training session, at least two muscles below the knee in a single leg were instrumented with surface electrodes for EMG signal recording. Amplifiers located inches from the electrodes provided an initial gain of 100 and a high pass filter at 10Hz to minimize movement artifact. Programmable EMG amplifiers low pass filtered the signal at 1 kHz and added an additional gain of 99. Signals were sampled at 2.5 kHz and displayed on a monitor for the participant in real time using Matlab and XPC target (The MathWorks, Inc., Natick, MA). Full bandwidth EMG was also recorded for post processing offline.

### C. Pre/Post Training Evaluation

At the start of each training session, each muscle was evaluated as a discrete command signal *without* the presence of visual feedback. The subject was instructed to attempt a movement specific to the muscle of interest in response to an audio cue. Two trials were performed for each muscle. In a given trial, the subject heard computerized tones signaling them to attempt a movement three different times for a total of six movement attempts per muscle. Baseline or rest activity was recorded before and after the movement attempt trials. Post-training, these trials were repeated with the visual feedback *removed*.

Analysis was performed offline to determine if significant EMG activity was present during volitional movement attempts. For each muscle, a threshold scheme was used to classify the data as either "movement" or "rest" as threshold levels incrementally increased. Classification of the data to determine the discrete state of muscle activity allowed for calculation of the true positive rate (TPR) and false positive rate (FPR) at different threshold levels. The TPR was then plotted vs. the FPR for each muscle to create a receiver operating characteristic (ROC) curve and the area under the curve (AUC) was calculated pre- and post-training.

Additionally, characteristics of the muscle signal itself were calculated. Maximum amplitude levels during movement attempts were recorded and the percent change in amplitude levels was calculated. Also, the full-bandwidth EMG signal was rectified, averaged and integrated to evaluate the robustness of the volitional muscle activity.

### D. Training Protocol

Initially, each subject trained each muscle using a program designed to improve the amplitude of the muscle signal. Full-bandwidth signals were rectified, averaged and then displayed in real time to the participant as a scrolling signal on a computerized chart. A single muscle was displayed at a time. A baseline value was first calculated and displayed as an initial target level on the same chart. During 10s trials, the subject was instructed to activate the muscle of interest in order to raise the scrolling signal above the current target level. At the end of each trial, the target was increased to the level of the previous trial's maximum value. Two types of trials were performed. In one type, the subject was instructed to go above and below the target level as many times as possible. In another type, the subject was instructed to sustain a muscle contraction as long as possible above the target. Each of the two muscles was trained using

## Tibialis Anterior: Pre/Post Training

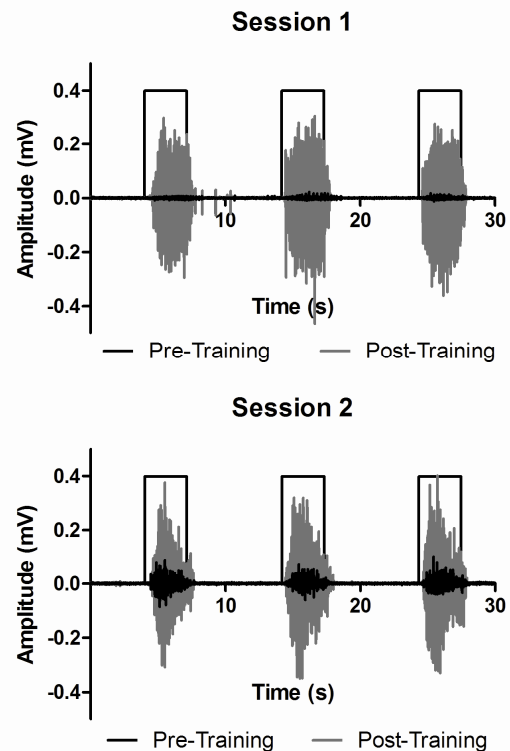


Fig. 1. Full-bandwidth EMG signals from Subject 1's tibialis anterior muscle before and after training for two different training sessions. Training sessions were approximately one month apart. Note surface electrodes were used for recording so slight differences in amplitude may be due to electrode placement. The subject did not have visual feedback of the signal during these trials.

the program until the EMG signal amplitude reached a plateau.

Next, a second program was used to train the subject to activate two muscles independently. The subject controlled the position of a cursor within a large box on the screen using the two muscle signals. The first muscle corresponded to the x-coordinate of the cursor and second to the y-coordinate. Axes of the box were scaled using the maximum values calculated during the amplitude training trials. Targets were displayed within the range of the cursor in either the bottom right corner (muscle 1 only), top left corner (muscle 2 only), or top right corner (co-contraction). Trials of 10s each were recorded in which the subject was instructed to try and reach a specific target.

Offline analysis was performed to determine the maximum amplitude value for each muscle signal during the training trials. Additionally, the number of times the subject was able to go above the target value and the maximum hold time above the target were calculated. For the independence program, the number of times the subject was able to reach the target of interest was determined as well as the number of times the subject hit one of the other targets in a given trial by mistake (false positives).

### III. RESULTS

Figure 1 shows the changes in full bandwidth EMG signal for the tibialis anterior (TA) muscle of subject one after two training sessions. The top plot shows the results of the initial training session. The dark black line along the x-axis is the signal before training. The square pulse represents the times when the subject heard the audio cue to move. Note the significantly larger amplitude of the signal post-training. At the start of the second training session, the amplitude of the TA muscle was lower than it was at the end of training session one, but still much larger than it was at the start of training session one (bottom plot). During training session two, the subject was able to use the visual feedback to learn to increase the signal approximately back to the amplitude it reached at the end of training session one. Of the five muscles tested to date, this one showed the most improvement from training.

TABLE 1

Subject #	Visit #	Muscle <sup>a</sup>	PRE-TRAINING		DURING TRAINING	POST-TRAINING	
			Max(mV)	INT <sup>b</sup>	Max(mV)	Max(mV)	INT
1	1	FDB	.411	8.455	.447	.449	7.822
		TA	.034	.366	.513	.465	12.773
	2	Soleus	.075	.747	.081	.050	1.127
		TA	.102	1.908	.561	.434	10.677
2	1	FDB	.142	.870	.117	.089	.918
		PL	.006	.020	.034	.013	.080

<sup>a</sup>FDB = flexor digitorum brevis, TA = tibialis anterior, PL = peroneus longus

<sup>b</sup>INT = integrated area under the curve

Table 1: Maximum amplitude values for all subjects and muscles studied during training and pre- and post-training evaluation. Results for muscles that showed significant improvements post training are bolded. Note that the subjects did not have any visual feedback during pre- and post-training. Visual feedback of muscle activity was shown during training.

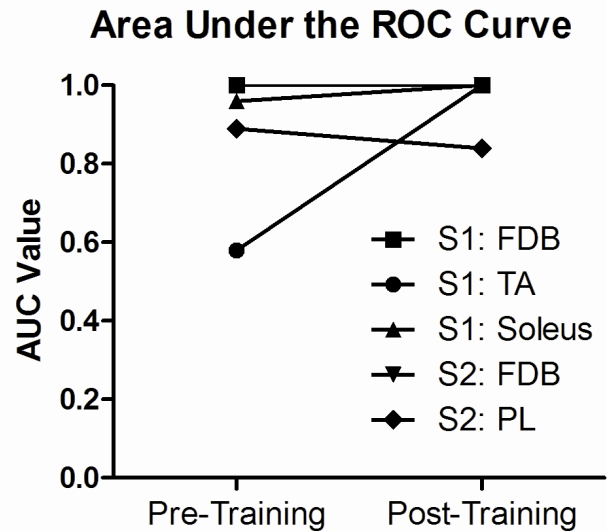


Fig. 2: Area under the ROC curve values are shown pre- and post-training for the five muscles studied. (Only Subject 1's TA was evaluated in two training sessions and this figure only shows the results from the first training session with the TA. During the second training session, post pre- and post-training AUC values were equal to 1.0.) Both subjects' FDB muscles also had AUC values equal to 1.0 pre- and post-training.

Maximum signal amplitudes for all muscles tested are shown in Table 1. Pre- and post-training trials were recorded without visual feedback of the muscle signal. Note that in four of the six cases, post-training amplitudes were substantially larger than pre-training maximum amplitudes. During training, the subjects did have visual feedback of the muscle of interest. In five cases, the signal was largest during training, when visual feedback was present. For both subjects, the muscle that initially had the largest magnitude did not improve as much as the other muscles with smaller initial maximums during the pre-training trials. For subject 1, the largest improvement was observed for the tibialis anterior muscle. For subject 2, the largest improvement was in the peroneus longus muscle. Both subjects had stronger FDB muscles right from the start.

Also included in Table 1 is the area under the rectified and averaged signal (INT) from the trials used for pre- and post-training evaluation. As evident from Figure 1, during each trial the subjects attempted a specific movement three times. A larger integration value indicates that the subject was able to sustain a contraction at either a higher level and/or for a longer period of time. Larger INT values post training were seen for every muscle except subject 1's FDB, which had a high integration value even before training.

For evaluation of each muscle as a discrete command signal pre- and post-training, ROC curves were created, which relate the true positive rate vs. the false positive rate at different threshold classification values. A single measure representative of the ability to classify the signal discretely is the area under the ROC curve (AUC), which ranges from 0.0 – 1.0. An AUC value equal to 1.0 indicates perfect classification, suggesting the muscle would be an excellent discrete command signal. AUC values were calculated for

each muscle pre- and post-training. These values are shown in Figure 2. The FDB muscle for each subject had AUC values equal to 1.0 before training. This was sustained after training. The TA and Soleus muscles for subject 1 increased to a value of 1.0 post-training. The peroneus longus for subject 2 was the only muscle that did not improve in AUC value post-training.

#### IV. DISCUSSION

Results indicate that training with visual feedback can improve the amplitude and robustness of volitional muscle signals below the level of injury in individuals with SCI. The majority of muscles studied improved both in maximum amplitude level and robustness (measured with the integrated, low pass filtered signal) after training with visual feedback.

Visual feedback seemed crucial to the improvement in muscle signal strength. Because this population has little if any visible movement below the injury level, visual feedback of the muscle activity is helpful to the subject in learning to activate the muscle. For subject 1, the only visible movement evident prior to training was slight movement of the toes. By the completion of the first training protocol, subject 1 obtained visible dorsiflexion of the ankle by contracting the TA muscle (less than full range). In the second subject we observed only a slight flicker in the toes. Typical movements that would involve recruitment of muscles of interest were suggested to both subjects; however, once they were presented with the visual EMG feedback, they were instructed to attempt any type of lower extremity movement that produced an increase in the EMG signal. Other training studies suggest that typical movements are not always ideal for activating muscles below the injury level [8]. As expected, almost all of the muscles showed higher maximum values during training, when visual feedback was present, rather than pre/post-training when it was absent. Subjects were still able to activate these muscles at higher amplitude levels even without the visual feedback, which is promising for use of these muscles as a command signal for a neuroprosthesis.

The mechanism causing improvement in the signal amplitude we observed in a few muscles in this study is unknown. Possible explanations for changes in signal strength include an increase in the firing rate of the residual motor units, an increase in the number of motor units being recruited, or a synchronization of the intact motor units [8]. The improvement may result from a combination of these possible mechanisms. A subject learning to activate the muscle by attempting a variety of movements suggests that they may be learning to activate more of the motor units that remain intact post-injury, but further research is required to fully explain the mechanism behind these results.

Not every muscle increased in amplitude post-training. For instance, the FDB muscle for subject 2 decreased in maximum amplitude level during training. One reason for this may be fatigue. Anecdotally, both subjects reported

feeling as if they had significantly exerted themselves throughout the training. They even compared it to exercising or weight lifting. Subjects were instructed to rest whenever necessary; however, the excitement at seeing signals on a computer screen of muscles previously considered paralyzed may have trumped the request to rest. If in fact only a few motor units remain intact and these are continually activated during training, fatigue is a reasonable explanation for slight decreases in amplitude.

This study reports on the first subjects involved in a training study to improve volitional muscle activity below the injury level in individuals with motor complete SCI. Initial results suggest that training can significantly improve the amplitude level and robustness of these small muscle signals. Increased activity below the injury level is critical for the use of clinically paralyzed muscles as a command signal for a neuroprosthesis. Additionally, these results have implications for other research areas in the field of SCI rehabilitation including activity dependent plasticity. Although these signals were not large enough to produce functional movements, their use as a command signal or as feedback in an activity dependent therapy may significantly impact recovery of function after SCI.

#### ACKNOWLEDGMENT

The authors would like to acknowledge the research participants that have made this study possible and the Clinical Research Unit at MetroHealth Medical Center in Cleveland, OH.

#### REFERENCES

- [1] P. H. Peckham, and J. S. Knutson, "Functional electrical stimulation for neuromuscular applications," *Annu Rev Biomed Eng*, vol. 7, pp. 327-60, 2005.
- [2] R. J. Triolo, L. Boggs, M. E. Miller *et al.*, "Implanted electrical stimulation of the trunk for seated postural stability and function after cervical spinal cord injury: a single case study," *Arch Phys Med Rehabil*, vol. 90, no. 2, pp. 340-7, Feb, 2009.
- [3] K. L. Kilgore, P. H. Peckham, T. J. Crish *et al.*, *Implantable networked neural system*, U.S.A., to Case Western Reserve University, U. S. P. Office, 2007.
- [4] K. L. Kilgore, H. A. Hoyen, A. M. Bryden *et al.*, "An implanted upper-extremity neuroprosthesis using myoelectric control," *J Hand Surg [Am]*, vol. 33, no. 4, pp. 539-50, Apr, 2008.
- [5] C. W. Moss, K. L. Kilgore, and P. H. Peckham, "A Novel Command Signal for Motor Neuroprosthetic Control," *Neurorehabilitation and Neural Repair*, vol. In Press, 2011.
- [6] B. A. Kakulas, "Neuropathology: the foundation for new treatments in spinal cord injury," *Spinal Cord*, vol. 42, no. 10, pp. 549-63, Oct, 2004.
- [7] B. S. Brucker, and N. V. Bulaeva, "Biofeedback effect on electromyography responses in patients with spinal cord injury," *Arch Phys Med Rehabil*, vol. 77, no. 2, pp. 133-7, Feb, 1996.
- [8] R. B. Stein, B. S. Brucker, and D. R. Ayyar, "Motor units in incomplete spinal cord injury: electrical activity, contractile properties and the effects of biofeedback," *J Neurol Neurosurg Psychiatry*, vol. 53, no. 10, pp. 880-5, Oct, 1990.