Three case studies demonstrate possible uses for surface electromyography training in the rehabilitation of the arm and hand. Benefits were obtained in all 3 cases. Training protocols were customized to the needs of the clients.

Introduction
Surface electromyography (SEMG) training has a long history in physical therapy but is seldom used in most rehabilitation clinics today. The absence of biofeedback reflects many issues—lack of training, poor research support, reimbursement concerns, etc. In particular, SEMG training for deficits in upper extremity functioning is rarely available. Jeannette Tries (1989) wrote an excellent paper on this topic and noted that there are several factors that make upper extremity biofeedback problematic. In contrast with the relatively fixed patterns of movement required for normal lower extremity functioning, upper extremity functioning requires a diverse range of movements involving the simultaneous coordination of multiple muscles from the trunk to the hand. Hand function is incredibly complex with functions ranging from precision tasks, such as threading a needle, to power tasks, like lifting heavy objects. The complexity of hand control is reflected in the sensorimotor homunculus, which shows a marked increase in the cortical areas devoted to hand control.

Anatomical considerations also contribute to the difficulties in providing SEMG biofeedback for arm and hand dysfunction. The muscles of the leg tend to be larger and many of them are readily accessible with surface electrodes. In contrast, the forearm contains many smaller muscles that overlap with each other (see Figure 1). This makes it more difficult to isolate specific muscles with surface electrodes as overlying muscles contribute to cross-talk. In addition to the extrinsic muscles of the forearm, there are also 3 sets of smaller intrinsic muscles in the hand that are essential for normal functioning.

With regard to electrode sites, I have found that with judicious placements, one can obtain reasonably clean recordings from many important hand muscles. When working with flexor functions, it is desirable to distinguish between wrist flexion and finger flexion. While the flexor wad (the origin of many of the flexor muscles just below the elbow) near the elbow contains at least 6 muscles, finger flexors can be isolated from the wrist flexors by using a distal placement on the ventral surface of the forearm, 2–3 cm from the wrist crease. Figure 2 shows the simultaneous activity from 4 triode electrodes spanning the ventral forearm from the wrist to the elbow. While finger flexor activity is recorded throughout the forearm, the most distal site produces a record that is relatively free from wrist flexor activity.

All of my SEMG biofeedback is conducted with the goal of improving patterns of muscular recruitment in order to enhance functional abilities. I rely on a model of motor control comprised of 4 functions: initiation, coordination, maintenance of recruitment, and termination of motor activity. I select sites and devise training protocols based on kinesiological principles and my clinical experience. While a few standard protocols have been formulated, to a large degree, SEMG biofeedback in physical medicine and rehabilitation continues to be an art as much as a science.

In this paper, I will present 3 case studies that demonstrate how SEMG was used to help increase upper extremity function following a physical injury. All of the biofeedback training in these cases was conducted with a modular J&J I330 (J&J Engineering, Poulsbo, WA) system connected to a Pentium 1 computer. I typically use a wide bandwidth (25–1000 Hz) and a high sensitivity setting in which the maximum readings are just above 150 µV. I use 10-second trials most often, usually sampling 4–5 times per second, and I display amplitude activity in line graphs of various colors with minimal smoothing. I also add auditory feedback for most clients, presenting a 220 Hz tone as a “reward” signal whenever all training requirements are met. I usually use triode disposable sensors from Multi-BioSensor (El Paso, TX), but when those are not suitable, I use closely spaced pre-gelled electrodes.
Case Study 1

Mr. H was a young man who managed a convenience store in Miami. The store was robbed, and he was shot near the web space of his left hand between the thumb and index finger. He was receiving both physical and occupational therapy but was struggling to regain control of his thumb, and his hand was stiff and clumsy.

In the initial evaluation, I used closely spaced electrodes over the flexor pollicis longus (FPL) and the extensor pollicis longus (EPL), the muscles primarily responsible for bending and straightening the tip of the thumb. The FPL lies lateral to the flexor tendons of the fingers; it is readily palpated and can be recorded without difficulty. The EPL tendon forms one side of the “snuff box” on the dorsum of the hand, and the muscle runs obliquely across the forearm. Its activity is also readily recorded with diagonally oriented dorsal electrodes, but it is not possible to isolate the EPL from the long finger extensors with surface electrodes. The initial results showed modest agonist recruitment in both thumb flexion and extension (see Figure 3). There was excessive corecruitment of the opposing muscles in both movements, with the ratios of agonist to antagonist activity ranging from 1:1 to 3:2.

For training, I used 3 sets of electrodes, with an additional placement parallel to and just medial to the FPL site. These electrodes were intended to record from the distal end of the finger flexors, although I anticipated that there would be a significant cross-talk between the 2 flexor channels. I added the finger flexor site to determine if increased FPL activity might be reflecting overflow of general finger flexion. Training consisted of several 10-second trials of sustained thumb extension followed by training for sustained thumb flexion. I finished each session with alternation training, working to improve the reciprocal recruitment pattern of the EPL and FPL as the client tried to repeatedly extend and then flex the thumb.

The training emphasized improved isolation of the antagonistic muscles in both extension and flexion. To accomplish this goal, I used a combination of amplitude and proportional feedback. I created a virtual channel that was a proportion—the amplitude of channel 1 divided by the sums of the amplitudes of channels 1 and 2. This virtual signal ranges from zero to 1 and is equal to .5 whenever the amplitudes are equal. The proportional channel was not displayed but was used to control additional auditory feedback. Initially, I kept the amplitude requirement low and used a shaping procedure to improve isolation in each movement. The visual display presented EPL and FPL amplitudes only, and auditory feedback was used to signal success. The reward tone was silenced when the amplitude or proportional threshold was not met. As agonist isolation improved, I gradually increased the amplitude threshold, requiring more effort but still maintaining a good separation of the opposing muscles.

This training was very effective, and Mr. H. made major gains during 8 sessions of biofeedback over 4 weeks. He showed improvement in both recruitment patterns (see
Figure 4) and functional gains. Isolation increased to more than 3:1 in both movements, and amplitudes nearly doubled. Functionally, there were improvements in grip strength, lateral pinch, and pegboard speed and reports of greater ease of use in routine daily activities (dressing, feeding, and grooming). It was interesting to note that even with the close proximity of the finger flexor and FPL electrodes we were able to show distinctly different patterns of recruitment between the power grip (requiring activation of both thumb and finger flexors) and thumb flexion (requiring activation of only the thumb flexors).

**Case Study 2**

Ms. P was a 20-year-old woman who had been in a major motor vehicle accident and sustained a significant head injury. She had some mild cognitive deficits as well as ataxia, or problems with coordination. I started biofeedback training with her about 10 months after her accident. By that time she had made major strides in her rehabilitation efforts and was walking on her own. However, she continued to have problems in hand control and, as a result, she was unable to feed or dress herself without assistance.

In my initial evaluation, I used a 2-channel protocol and recorded from the wrist flexors and wrist extensors. I was pleased to see full normal amplitudes in both extension and flexion with only minor problems in sustaining appropriate activity. In alternating movements, the signals were well isolated, with no signs of spastic corecruitment. I then gave her a plastic spoon and recorded the SEMG activity while she simulated feeding herself. She tended to extend the wrist strongly while grasping the spoon, and there was a marked tremor as she approached her mouth. The SEMG tracings showed increased variability, particularly from the extensor site, and extensor activity was much higher than is typically seen in this task.

Initially, I was at a loss regarding how I might train this client. Amplitude training clearly was not called for, and there was no reason to train for improved isolation. I realized that if the major problem was excessive variability, then training to improve the stability of the EMG tracing might be beneficial.

I devised a stabilization protocol that is graphically presented in Figure 4. It included 2 additional copies of her wrist extensor EMG signal that were used to provide auditory feedback and were not presented visually. One copy set an upper limit for feedback, and the second copy set a lower limit. A visual target between the 2 limits was presented, and she was instructed to maintain the moving signal as close to the target as possible. Whenever the actual signal moved outside of the range defined by the upper and lower limits, a fairly loud pure tone was presented. Maintaining the signal between the limits kept the system quiet. In Figure 5, the shaded area is the “quiet zone,” and the solid line in the middle of the shaded area is the target level.

Targets were set at 20 or 30 µV on most sessions with a training window that was gradually restricted over sessions. Within each session the training targets were adjusted so that the noise was presented 10%–20% of the time. As training progressed, we added tasks that were more functional in nature to the situation. We trained for reduced variability while holding objects, moving them about and bringing them towards the mouth.

Over the course of 20 training sessions there was a marked improvement in terms of EMG stability. Standard deviations of extensor amplitudes in a given task diminished, and hand position was more stable with the wrist in neutral. However,
there were limitations in the effectiveness of the training. When bringing objects toward her mouth, we saw increased variability as her hand got closer to her face. It is my belief that as her hand approached her face, the motor control systems attempted to control hand position more precisely, and the increased demand caused the system to break down to a degree.

My work with this client revealed many interesting observations on motor control after a traumatic brain injury. The marked extension I noticed in the evaluation was Ms. P’s way of coping with the tremor—locking the wrist in the fully extended position allowed a reasonable grip and reduced the intensity of the tremor. I believe that biofeedback training needs to be done with functional tasks because these present different demands on the sensorimotor systems. Despite the relative crudity of my biofeedback training approach with this client, she was pleased with the outcome. She was able to bathe herself and brush her hair more effectively. She was also able to put away clothing in her dresser and place shirts on a hanger and put them in a closet. In terms of feeding, she was able to use a fork fairly effectively and could hold a cup and drink with a straw. She was unable to use a spoon with liquids such as soup.

Case Study 3

Mr. K was a 33-year-old police officer who was shot in the neck and sustained a left brachial plexus injury. The brachial plexus is similar to a complex highway interchange. Nerve roots from the third cervical vertebra through the first thoracic vertebra exit from the spinal cord and the fibers divide, reunite, and intertwine in the neck and shoulder. All of the sensory and motor information for the arm and scapular region passes through the brachial plexus. Damage to this area can range from a mild “stinger” resulting in transient weakness and sensory disturbances to a transection or avulsion injury that permanently and completely paralyzes the arm and shoulder.

Mr. K. noted that he initially lost all use of his left arm and hand, but that movement and sensation gradually returned in the months after the shooting. I first saw him about 10 months after he was shot. He had full range of motion at the shoulder and elbow, although strength and endurance were diminished. His wrist was stiff, and there was significant wrist flexor activity during passive extension of the wrist. Mr. K. also reported increased pain and discomfort as time had passed since the incident.

Because Mr. K. was from a foreign country, I was only able to see him on 4 separate occasions over the course of 1 year. The first set of sessions was for 5 consecutive days, followed almost a month later by 2 more days of training. After 3 more months, I saw him for 4 days followed by 2 more days some 8 months later.

The initial SEMG results showed normal peak levels of activity from the biceps and triceps during elbow movements, but Mr. K. had difficulty sustaining maximal levels. Mr. K. was able to increase mean biceps EMG for a 10-second trial from 55 µV to 102 µV over the first 3 days of biofeedback training. In extension, triceps recruitment was more effective when the arm was extended downwards. In forward elbow extension, there was deceased triceps recruitment, even when the arm was supported. We worked on increasing triceps activity in forward extension by initially using a downward elbow extension and then requiring continued triceps recruitment as the shoulder was flexed with assistance. This training overlapped with the shoulder flexion training and generated a doubling...
of triceps activity to almost 120 µV in the third block of training sessions.

Proximally, the most significant finding was a marked reduction in anterior deltoid activity during shoulder flexion. We also noted that biceps activity was very high, approaching the levels obtained in elbow flexion. The long head of the biceps crosses the shoulder and can assist in shoulder flexion; it is not unusual to see moderate biceps activity in this movement. However, in this case, the reliance on the biceps was making it difficult to increase anterior deltoid recruitment.

To deal with this problem, I created a proportional channel from the anterior deltoid and biceps signals. I used a similar training strategy from the first case study, initially requiring a low level of anterior deltoid activity with minimal recruitment of the biceps. Once this was established, I started increasing the amplitude requirement for the anterior deltoid. At the same time, I changed the proportional threshold to permit more biceps activity, while still maintaining a modest inhibitory emphasis. The training was quite effective, resulting in a doubling of anterior deltoid activity from initial levels (see Figure 5). I made sure to emphasize that the strength in the shoulder would take months to return as the muscles recovered from the extended period of disuse atrophy.

Distally, I saw near normal recruitment patterns in sustained finger extension and in the power grip. The main difficulty was with the wrist flexors—they were sluggish and inconsistent in wrist flexion and showed increased activity in wrist extension. I was able to obtain increased wrist flexor recruitment when flexing the wrist against resistance (an isometric flexion), but there was little carryover to a concentric movement (a basic motion in which the muscle shortens as it contracts). In the final 2 sessions, we worked on improving the reciprocal pattern of activation of these muscles during alternating between wrist extension and flexion. I worked with the wrist in neutral rotation (thumb facing up) and the forearm supported. I was able to get improved isolation at slower speeds (2–3 movements per 10 seconds), but whenever Mr. K. tried to move more rapidly, he would increase flexor corecruitment as he started to extend the wrist.

Concluding Remarks

In presenting these 3 cases, it was my goal to demonstrate that SEMG training can be an important modality in the restoration of function of the impaired arm and hand. This is a challenging clinical task. It requires a diverse range of technical skills, as well as patience, sensitivity, and creativity. It can also be a frustrating experience due to the ignorance of referral sources, insurance companies, and patients.

The most fundamental reason for the lack of biofeedback in rehabilitation is poor research support. I am not aware of any large-scale, randomized, controlled studies demonstrating the efficacy of SEMG training in restoring motor function. In terms of hand function, 2 studies using small groups of participants have demonstrated benefits for biofeedback (Stein, Brucker, & Ayyar, 1990; Armagan, Tascioglu, & Oner, 2003). Given the current emphasis on evidence-based practice guidelines, the disregard for biofeedback is not unfounded.

I am not sure what can be done about this situation. Large-scale research is expensive, and sources of funding for biofeedback studies are not well endowed. In addition, research studies typically use standardized protocols, and these may not be well suited to assess the effectiveness of SEMG training in rehabilitation. In the interim, we need to publicize our successes and strive to refine and improve our techniques.

References


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