Use of Electromyographic Feedback to Increase Inhibitory Control of Spastic Muscles

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> Electromyographic (EMG) feedback was used to train a patient to inhibit spastic muscles voluntarily. The EMG activity was recorded with surface electrodes placed over the belly of the medial gastrocnemius muscle and displayed on a meter. The patient trained himself at home for thirty minutes daily for two months. Following training, improved muscle control could be seen even without the use of the EMG feedback device.

Kecent studies using biofeedback training have shown that patients can learn to control a variety of cardiovascular and neuromuscular abnormalities.¹⁻⁹ Briefly, such training involves recording the activity of a particu-

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lar response system, e.g., electromyographic activity (EMG), transducing the physiological activity into a simplified visual or auditory signal, and feeding the modified information back to the subject. With appropriate motivation and instructions, subjects can use this feedback along with that from normal proprioceptive, kinesthetic, and visual sensory input to gain an often remarkable degree of voluntary control over many physiological responses previously considered involuntary, reflexive, or unconscious.

Weiss and Engel have trained cardiac patients to control the rate of occurrence of premature ventricular contractions.¹ Benson and colleagues have trained essentially hypertensive patients to produce clinically significant decreases in systolic blood pressure.²

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With regard to somatic muscle activity, Budzynski and others have reported a reduction in tension headaches following training in relaxation of the deep muscles of the body.³ Ballard and coworkers trained a woman patient to inhibit the severe spasms of the eyelids and accessory musculature that are symptomatic of essential blepharospasm.⁴

It is of particular interest that, in several of the above studies, voluntary control persisted beyond the termination of training; i.e., patients were able to exert control over cardiovascular or somatic muscular responses without the assistance of the biofeedback apparatus.

Several recent studies have demonstrated that biofeedback training may be of considerable use in neuromuscular retraining. Block and colleagues have reported success in treating pathological and congenital nystagmus, oscillopsia, ataxia, and chronic hand spasticity in cases where traditional physical and occupational therapy methods have not been successful.^{5,6} Other investigators have used EMG feedback successfully in increasing the function of muscles previously considered paralyzed.^{7–9}

PATIENT M. B.

The patient was a twenty-two-year-old white male who was a college graduate. He was cooperative and motivated to experiment with this new technique, though skeptical as to its potential effectiveness. More than nine years before biofeedback training, he had sustained a traumatic right-sided head injury which resulted in left hemiparesis with spastic overlay. The patient stated that, following his injury, he had undergone long periods of physical therapy, but severe residual muscle function loss remained.

Initial evaluation revealed normal passive range of motion, good position sense, and good body scheme orientation. Touch and texture sensation were normal.

Muscle function tests revealed hypertonicity of both left upper limb flexor synergists and left lower limb extensor synergists. Proximal muscles were less affected by spasticity than distal muscles. Good muscle strength was shown in left shoulder motions and left elbow flexion; elbow extension was only fair. Testing of forearm, wrist, and finger motion was impossible because of intense overflow in finger-wrist flexor muscles. The extensor muscles did not function effectively.

In the left lower limb, the hip had good function but with minimal flexor synergy overlay on abduction. The knee could be flexed actively to 90 degrees, but lacked 20 degrees of full active extension. Ankle motions were minimal because of gastrocnemius-soleus hypertonicity. Anterior tibialis function was largely negated by gastrocnemius action. Foot evertors were functioning with a trace grade.

The patient had a typical spastic hemiparesis gait. The left foot inverted and dorsiflexed at toe-off, the hip circumducted, and knee flexion was minimal. At heel strike, the foot often contacted the floor on the lateral surface and barely allowed weight bearing on the sole; however, use of a short leg brace was unnecessary. The left arm was held in adduction with about 30 degrees of elbow flexion. The hand was supinated and flexed.

Before training, the active range of motion of the left ankle was taken with the hip and knee flexed to 90 degrees. Dorsiflexion was zero degrees with 10 degrees inversion; plantar flexion was 35 degrees. No isolated active inversion or eversion was seen.

The EMG feedback was provided by a Model BFT 113 Feedback Encephalograph* operated in the EMG mode. This device responds to signals from 20 to 100 hertz. A meter, whose amplitude of deflection was logarithmically related to EMG frequency, provided continuous visual feedback of muscle activity.

During the initial treatment session, both the nature of his disorder and the operation of the feedback device were explained to the patient. Surface electrodes were placed over the belly of the left medial gastrocnemius and the patient observed the response on the meter as he contracted and relaxed the muscle repeatedly (Figure). The patient was told that the range of meter deflection observed even during relaxation was related to the cause of his inability to dorsiflex the left ankle normally. He was instructed to try to learn what the muscle "feels" like when contracted or relaxed.

Following this exercise, the patient was

^{*} Bio-Feedback Technology Inc., Garden Grove, CA 92643.



Figure. Surface electrodes of EMG feedback device in place. Note meter which provides continuous visual feedback to patient.

instructed to attempt dorsiflexion of the left ankle while simultaneously inhibiting (i.e, relaxing) the gastrocnemius. During the initial session, this motion was impossible to execute because of constant gastrocnemius contraction. The patient, however, understood what was expected of him.

Following the initial evaluation-treatment session, the patient was allowed to take the feedback device home. He was instructed to practice twice a day for thirty minutes each session. The first ten minutes of each session were devoted to repetitive contraction and relaxation of the left gastrocnemius. During relaxation, the patient was instructed to "try to get the meter to register as near zero as possible"; i.e., to try to inhibit gastrocnemius contraction actively.

The remaining twenty minutes of each training session were devoted to practicing repeated dorsiflexion of the left ankle with the hip and knee at 90 degrees. The patient was instructed to "raise your toes and the front of your foot off the ground as much as you can while keeping the meter reading as low as possible."

At the beginning of training, the threshold control of the feedback device was set so that a considerable amount of EMG activity was required to cause the meter to deflect. As training progressed and the patient was satisfied that his skill in inhibiting gastrocnemius activity was improving, he was instructed to decrease the threshold of the feedback device periodically. Thus, in order to keep the meter reading at the same low level, the patient had to demonstrate increasing inhibition of gastrocnemius activity.

Following two months of training, the active range of motion of the left ankle was again taken with the hip and knee flexed to 90 degrees. Dorsiflexion with neutral supination and pronation was 15 degrees. No isolated inversion or eversion was observed, but muscle action of both was stronger than previously observed. The patient's gait had also improved and he was able to place the foot flat on the floor from heel strike into mid-stance.

COMMENTS

EMG feedback was used in this case because the patient's injury did not affect lower motor neuron paths; therefore, the possibility of increasing motor function was good. Though several earlier studies have used EMG feedback to increase the function of muscles previously believed to be paralyzed, this is the first case reported in which feedback was successfully used to increase inhibitory control of an antagonistic muscle directly. The ease of operation of the feedback equipment, the fact that patients can operate the equipment themselves, the speed and effectiveness of the treatment, and the fact that the improved motor function persisted beyond the termination of biofeedback training indicate the possibilities of EMG feedback in neuromuscular retraining.

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