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RESEARCH NOTE

Effects of Cerebellar Stimulation on Abnormal Proprioceptive Reflexes in Spastic Primates

TIMOTHY J. EBNER, JERROLD L. VITEK, ANDREW B. SCHWARTZ, AND JAMES R. BLOEDEL[1]

Departments of Neurosurgery and Physiology, University of Minnesota, Minneapolis, Minnesota 55455

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The effects of cerebellar stimulation on the passively evoked electromyogram (EMG) responses of the biceps and triceps in spastic primates were studied. High-frequency stimulation decreased the abnormal cocontraction present during passive flexion and extension of the forearm. Furthermore, cerebellar stimulation resulted in a reciprocal pattern of EMG activity in the biceps and triceps, not present in control periods.

Since the introduction of cerebellar stimulation as a treatment for spasticity in certain movement disorders (3), various degrees of success and failure have been reported in clinical trials (4, 5, 7, 9). This variability indicates that experimental investigations are required to determine the precise action of this procedure on specific segmental reflexes and features of voluntary movement. However, studies examining the physiologic effects of cerebellar stimulation in animal models of spasticity are limited. In one brief report Hemmy and colleagues (6) reported that stimulation of the cerebellar surface in unilaterally decorticate primates decreased the force necessary to passively flex or extend the forearms. The present study was designed to systematically evaluate the effects of cerebellar surface stimulation on the tonic and reflexly evoked electromyogram (EMG) activity of the biceps and triceps muscles in spastic primates during passive forelimb movement.

Abbreviation: EMG—electromyogram.

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In this study two different models of supratentorial spasticity were used in two different species of primates (*Macaca mulatta* and *Cercopithecus aethiops*). In two animals (one of each species) a staged bilateral decortication was carried out under halothane anesthesia. In the first stage, after the appropriate craniotomy, the animal underwent a unilateral decortication on the left side by combined coagulation and extirpation of the premotor, supplementary motor, primary motor, and sensory cortices on one side. After a recovery period of 2 to 3 months a second decortication was made on the contralateral side, producing a lesion similar to the one in the opposite hemisphere. Another macaque underwent a left unilateral decortication involving the same cortical regions as in the other two monkeys. In the bilaterally decorticate animals a marked extensor hypertonus and increased resistance to passive stretch were observed in all four limbs after completion of the second stage of the decortication. In contrast, the unilaterally decorticate animal gradually developed only modest spasticity in the right extremities after a period of 3 months. All animals were implanted with stimulating electrodes on the anterior lobe of the cerebellum. Each electrode consisted of three platinum disks (7.6 mm^2/disk) embedded in a silastic strip. One electrode was implanted over the pars intermedia and medial cerebellar hemispheres on each side of the cerebellum. The electrodes were positioned primarily over the anterior lobe; however, the most posterior electrode disk was placed over the anterior part of the posterior lobe. The electrode leads were led to an external connector which was bolted and glued to the animal's skull.

After recovery from the surgical procedures, the biceps and triceps EMG activity in the right arm was recorded with either surface or intramuscular wire EMG electrodes during passive displacement of this forearm. The displacement consisted of a three step ramp-and-hold movement. The limb was first flexed 30 to 60° rapidly at the elbow. The arm remained flexed for 2 s, and then it was rapidly extended to the initial position. Displacement was monitored by a precision potentiometer and was used as feedback to control the torque motor. Torque evoked during the movement was monitored by a reaction torque sensor. The EMG activity from the biceps and triceps was amplified, rectified, and integrated (τ = 10 ms) by conventional electrophysiologic technique. The EMG data, displacement, and torque measurements were placed on magnetic tape for future data reduction by a PDP 11/40 computer system which averaged the EMG responses and the torque measurements for 80 consecutive trials.

Cerebellar stimulation utilized 0.2-ms pulses delivered simultaneously to each side of the anterior lobe. Although different frequencies of cerebellar stimulation (10 to 300 Hz) and different charge densities (1.5 to 10 $\mu C/cm^2$ electrode surface area) have been studied, this report will discuss those

combinations of stimulus frequencies and charge densities which were most effective in altering the EMG activity and the torque in each animal. In this study, brief periods of cerebellar stimulation (10 to 30 min) were used. Control periods in which EMG activity and torque were monitored during forearm displacement always preceded and followed periods of cerebellar stimulation.

An example of the EMG and torque responses obtained during control and stimulation periods for one of the bilaterally decorticate animals is illustrated in Fig. 1. In the pre- and post-stimulation control periods the reflex responses of the triceps and biceps were abnormal. During the stretch of the triceps, the reflexly evoked activity in the triceps and biceps increased, despite the fact that the latter was shortened. During the stretch of the biceps, there was a burst of EMG activity in the triceps despite the fact that its length decreased. This coactivation of antagonist muscles during stretch has been observed in cerebral palsy (8) and in parkinsonian rigidity (1, 11).

Cerebellar stimulation at high frequencies had marked effects on the organization of these reflexes and the torque necessary to produce the displacement of the limb in all these animals. As shown in Fig. 1, cerebellar stimulation decreased both the phasic and the tonic components of the stretch reflex in the triceps. The torque required to generate the flexion of the forearm was decreased 50% at these stimulus parameters. When stimulation was discontinued, the phasic and tonic components of the triceps response and the torque returned to prestimulus control values. Similar changes were evoked by cerebellar stimulation in the unilaterally decorticate animal.

Cerebellar stimulation also modified the cocontraction observed in these animals. The phasic and tonic cocontraction of the biceps during stretch of the triceps so apparent in control periods was suppressed by cerebellar stimulation (Fig. 1). Similarly, the cocontraction of the triceps during the stretch of the biceps was also reversed by the cerebellar stimulation. It should be emphasized that stimulation actually resulted in the appearance of a reciprocal pattern of EMG activity in the flexor and extensor in which the passive stretch of the muscle evoked a reflex activation of the agonist and a reflex reduction in the EMG activity in the antagonist. This pattern of reflex behavior is similar to that expected in a normal animal (2, 10). The abnormal cocontraction of the biceps and triceps returned when stimulation was discontinued.

In addition to suppressing the cocontraction in the flexor and extensor muscles, cerebellar stimulation augmented the phasic and tonic response to stretch of the biceps. Notice that during the pre- and poststimulus control periods extension of the arm did not evoke a reflex response in this muscle.

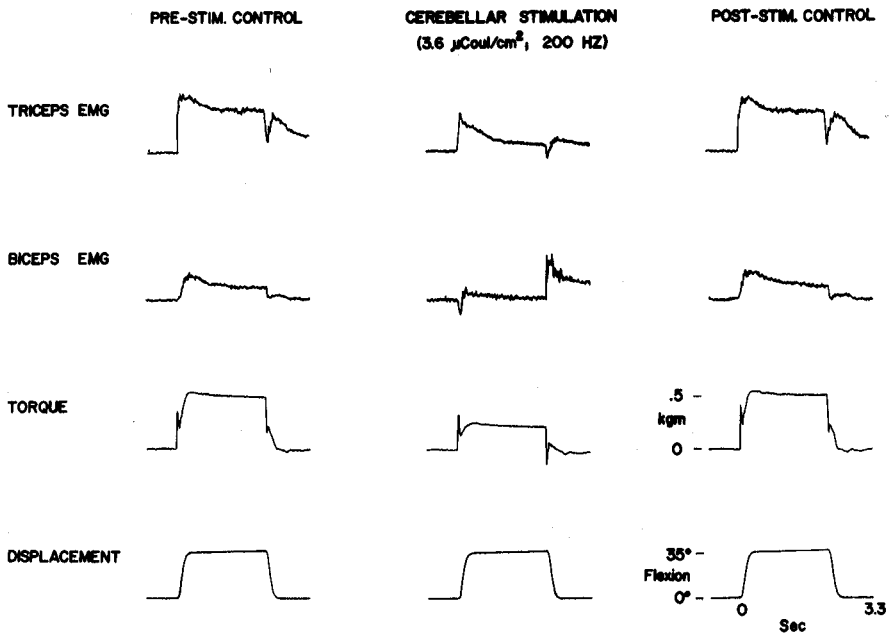

FIG. 1. Effects of cerebellar stimulation on the averaged, rectified, integrated EMG activity of the triceps and biceps ($N = 80$, $\tau = 10$ ms) during ramp and hold flexion of the forearm. Both the phasic and the tonic components of the triceps response were decreased by anterior lobe stimulation. The torque required to generate the displacement was decreased 50% by cerebellar stimulation. The response of the biceps during *stretch* was increased. Notice that the abnormal activation of the biceps during *shortening* was decreased by cerebellar stimulation. The EMG traces are all displayed at equal gains for control and stimulation periods.

As noted above, the accentuated reflex response to stretch of the triceps in these spastic animals was reduced during cerebellar stimulation.

The four principal effects of cerebellar stimulation presented, (i) reduction of the phasic and tonic reflex response of the triceps during passive flexion, (ii) decreased cocontraction, (iii) increased reflex response of the biceps during extension, and (iv) decreased torque required to displace the limb, were observed in all three animals. These findings demonstrate that cerebellar stimulation can reduce the phasic and tonic components of the stretch reflex in an extensor muscle and reduce the amount of torque required to passively displace the forearm. Extremely important is the observation that cerebellar stimulation can alter the abnormal *pattern* of the stretch reflex in a spastic animal, changing it from a pattern of marked cocontraction to the reciprocal pattern seen under normal conditions. Furthermore, cerebellar stimulation improved the

combinations of stimulus frequencies and charge densities which were most effective in altering the EMG activity and the torque in each animal. In this study, brief periods of cerebellar stimulation (10 to 30 min) were used. Control periods in which EMG activity and torque were monitored during forearm displacement always preceded and followed periods of cerebellar stimulation.

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response of the biceps to passive extension of the forearm and decreased the exaggerated stretch reflex in the triceps.

Although these findings were obtained in primate models of spasticity, they suggest that the possible therapeutic effects of cerebellar stimulation may not necessarily be confined to only a general reduction in muscle tone and proprioceptive reflex activity. Rather this technique may decrease or even reverse abnormal reflex patterns in patients with movement disorders. The cocontraction exhibited by the spastic monkeys used in these experiments has been suggested to contribute to the functional deficit in patients with cerebral palsy (8). Because cerebellar stimulation can clearly alter this abnormal reflex pattern, the decreased cocontraction seen in these experiments may be of clinical benefit if it can be systematically reproduced in patients.

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