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EEG-based communication: analysis of concurrent EMG activity

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Abstract

Objective: Recent studies indicate that people can learn to control the amplitude of mu or beta rhythms in the EEG recorded from the scalp over sensorimotor cortex and can use that control to move a cursor to targets on the computer screen. While subjects do not move during performance, it is possible that inapparent or unconscious muscle contractions contribute to the changes in the mu and beta rhythm activity responsible for cursor movement. We evaluated this possibility.

Methods: EMG was recorded from 10 distal limb muscle groups while five trained subjects used mu or beta rhythms to move a cursor to targets at the bottom or top edge of a computer screen.

Results: EMG activity was very low during performance, averaging 4.0 ± 4.4% (SD) of maximum voluntary contraction. Most important, the correlation, measured as r^2, between target position and EMG activity averaged only 0.01 ± 0.02, much lower than the correlation between target position and the EEG activity that controlled cursor movement, which averaged 0.39 ± 0.18.

Conclusions: These results strongly support the conclusion that EEG-based cursor control does no depend on concurrent muscle activity. EEG-based communication and control might provide a new augmentative communication option for those with severe motor disabilities.

Keywords: Electroencephalography; Electromyography; Conditioning; Sensorimotor cortex; mu rhythm; Augmentative communication; Rehabilitation

1. Introduction

Mu rhythms are 8–12 Hz EEG rhythms recorded over sensorimotor cortex in awake individuals (Gastaut, 1952; Kuhlman, 1978; Pfurtscheller and Aranibar, 1979; Kozelka and Pedley, 1990; Arroyo et al., 1993; Niedermeyer and Lopes da Silva, 1993). They are most prominent when the person is relaxed, and usually attenuate prior to and during movement. They can be distinguished from one another by their precise topographical foci, frequencies, and/or relationships to movement. Mu rhythms are accompanied by 20–25 Hz beta rhythms, which also focus over sensorimotor cortex and are also correlated with movement (Pfurtscheller, 1981; Kozelka and Pedley, 1990; Pfurtscheller et al., 1994, 1996, 1997; Stancak et al., 1997).

Studies over the past 10 years have shown that people can learn to control mu rhythm and/or beta rhythm amplitude and use that control to move a cursor on a computer screen in one or two dimensions (Wolpaw et al., 1991; Wolpaw and McFarland, 1994; McFarland et al., 1993). This control develops over a period of training and is exercised by subjects who are sitting quietly in a comfortable chair, without visible movement. Furthermore, people with severe motor disabilities, including almost total paralysis, can develop mu rhythm control (e.g. Miner et al., 1996; McFarland et al., 1998). Thus, it appears that humans can learn to control mu and beta rhythm activity independent of concurrent muscle activity. This inference is consistent with animal data indicating that EEG control does not require peripheral mediation (Black et al., 1970). It suggests that EEG control might provide a valuable new augmentative communication channel for those who are paralyzed or have other severe motor disabilities (Vaughan et al., 1996).

At the same time, it is conceivable that individuals make imperceptible, perhaps largely unconscious, muscular contractions during performance, and that this contributes to their mu or beta rhythm control (for example, by producing

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somatosensory input that attenuates the rhythms). If this were true, it would suggest that mu or beta rhythm control is secondary to muscle control, and does not constitute an independent CNS output channel. The comprehensive scalp recording that is a standard part of our studies indicated that neck, forehead, extraocular, and jaw muscles were not mediating EEG-based communication either directly or by their effects on mu and beta rhythms (e.g. McFarland et al., 1997a), but the possible role of extracranial muscles remained unexplored.

The present study set out to address this issue by recording EMG from major limb muscle groups of trained subjects as they used EEG activity to control cursor movement to targets presented on a computer screen. We evaluated the correlation between EMG and target location and compared it with the correlation between EEG and target location. We chose distal muscles because they have the largest cortical representations (Porter and Lemon (1993) for review). While weak correlations between EMG and target location were occasionally detected, the results indicate that mu or beta rhythm control does not depend on concurrent EMG activity, and thus that these EEG rhythms might provide a new communication channel of value to those with severe motor disabilities. A portion of the results have been reported in abstract form (Vaughan et al., 1997).

2. Methods

Subjects were five adults (one woman and four men, ages 59, 32, 33, 44, and 46 years). Four had no disabilities, while one had an abnormal gait due to lower limb ankyloses secondary to hemophilia. All gave informed consent for the study, which had been reviewed and approved by the New York State Department of Health Institutional Review Board. After an initial evaluation defined the frequencies and scalp locations of each subject's spontaneous mu and beta rhythm activity during motor imagery and actual movement, he or she learned EEG-based cursor control in 10–30-min sessions (2–3/week) and then participated for an additional 17–132 sessions. These sessions were devoted to a variety of studies of EEG-based communication (e.g. the present study; McFarland et al., 1998; Miner et al., 1998). Over the course of each subject's participation, offline data evaluations and concurrent improvements in system hardware and software led to adjustments in the electrode locations, frequency bands, and spatial filter used by the online algorithm that controlled cursor movement. The next section summarizes the online methodology used for the present study. A detailed description of system configuration and operation is available elsewhere (McFarland et al., 1997a).

2.1. EEG-based cursor control

The subject sat in a reclining chair facing a video screen and was asked to remain motionless during performance. Scalp electrodes recorded 64 channels of EEG (Sharbrough et al., 1991), each referred to an electrode on the right ear (amplification 20 000; bandpass 1–60 Hz). A subset of channels were digitized at 196 Hz and used to control cursor movement online as described below. In addition, all 64 channels were digitized at 128 Hz and stored for later analysis.

In this study, the subjects used mu and/or beta rhythms in several EEG channels over sensorimotor cortex to control one-dimensional (i.e. vertical) cursor movement to a target at the top or bottom edge of the video screen. Selection of the electrode location(s), frequency band(s), and spatial filter used in each subject was initially guided by evaluation of the subject's spontaneous mu and beta rhythms, and then adjusted as training proceeded in order to maximize the translation of the subject's EEG control into cursor control (McFarland et al., 1997a,b). Each session consisted of eight runs of 3 min each, separated by 1 min breaks. A run consisted of a series of trials. Each trial began with a 1 s period during which the screen was blank. Then, a target appeared at the top or bottom edge of the screen. One second later, the cursor appeared in the center of the screen and began to move vertically 10 times/s controlled by the subjects' EEG as described below. The cursor had 188 possible vertical positions. The subject's goal was to move the cursor to the target. The trial ended when the cursor reached the top or bottom edge. When it touched the correct edge, the target flashed for 1 s as a reward and the computer recorded a hit. When it touched the other edge, the target disappeared, the cursor remained fixed on the screen for 1 s, and the computer recorded a miss. In either case, the next trial then began with 1 s of blank screen. Equal numbers of top and bottom targets appeared in an order randomized in blocks of 8, and a miss did not cause the target to be repeated. Thus, accuracy expected in the absence of any EEG control was 50%. After initial training, the subjects routinely achieved session accuracies of 80–99%.

Cursor movement was controlled as follows. Several EEG channels over sensorimotor cortex were re-referenced either to a common average reference or to a Laplacian reference (McFarland et al., 1997b). The Laplacian reference used four next-nearest-neighbor electrodes (e.g. for C_3, these were F_3, C_z, P_3, and T_7). Every 100 ms, the most recent 200 ms segment from each channel was analyzed by an autoregressive algorithm (Jansen et al., 1981; Marple, 1987), and the amplitude (i.e. the square root of power) in a 3-Hz wide frequency band in the mu or beta rhythm range was calculated. Amplitude in the selected band from the selected channels provided the EEG control signal that was used as the independent variable for a linear equation that controlled cursor movement. For three subjects, the EEG control signal was mu or beta rhythm amplitude over the hand area of left sensorimotor cortex or the sum of the amplitudes over the hand areas of right and left sensorimotor cortices (e.g. locations C_3 and C_4). For the other two

subjects, the EEG control signal was the amplitude in the mu or beta band over the foot area (e.g. C_z) minus the amplitude over one or both hand areas. As described in detail elsewhere (McFarland et al., 1997a; Ramoser et al., 1997), the intercept and gain in the equation were defined on the basis of the subject's past performance so that net cursor movement over many trials was zero, and so that the average duration of cursor movement was several seconds.

2.2. Concurrent EMG recording

In each subject, surface EMG was recorded over 10 distal muscle groups during performance. These muscle groups were right and left: hand anterior forearm muscles (AF), posterior forearm muscles (PF), thenar eminence muscles (TE), anterior calf muscles (AC), and posterior calf muscles (PC). As noted above, these groups were chosen because they have large cortical representations. EMG from each muscle group was recorded (amplification 20 000; bandpass 10–3000) with a pair of 1-cm Grass gold-cup electrodes placed 2.5 cm apart over the muscle group along the axis of the limb at the midpoint of the limb segment (i.e. over flexor digitorum superficialis for the AF group, extensor digitorum communis for the PF group, abductor pollicis brevis for the TE group, anterior tibialis for the AC group, and gastrocnemius and soleus for the PC group), and digitized at 1024 Hz. Each subject was studied for one or two sessions, and in each session each muscle group was studied for two runs of 25–30 trials each. In addition, at the session's end, EMG from each muscle group was recorded (amplification 1000) and digitized during an isometric maximum voluntary contraction (MVC) for three 4 s periods separated by 4 s breaks.

2.3. Data analysis

The digitized EMG was converted to absolute value (i.e. equivalent to full-wave rectification). For each muscle group, average EMG absolute value during each cursor-control trial was calculated and expressed in percent of the single highest 4 s MVC value. From these results, we calculated for each muscle group the average EMG amplitudes for top target trials and for bottom target trials in percent of MVC.

We evaluated the relationships between target position (i.e. top or bottom) and the EEC measure that controlled cursor movement (e.g. mu rhythm amplitude over the hand area of left sensorimotor cortex; Fig. 1), and between target position and the EMG from each of the muscle groups. These relationships were assessed by r^2, the coefficient of determination, which is the proportion of the total variance of the two distributions (i.e. the distribution of EEG or EMG values for top targets and the distribution for bottom targets) that is accounted for by the difference in their means (Wonnacott and Wonnacott, 1977). For each muscle group of each subject, we also performed an analysis of covariance

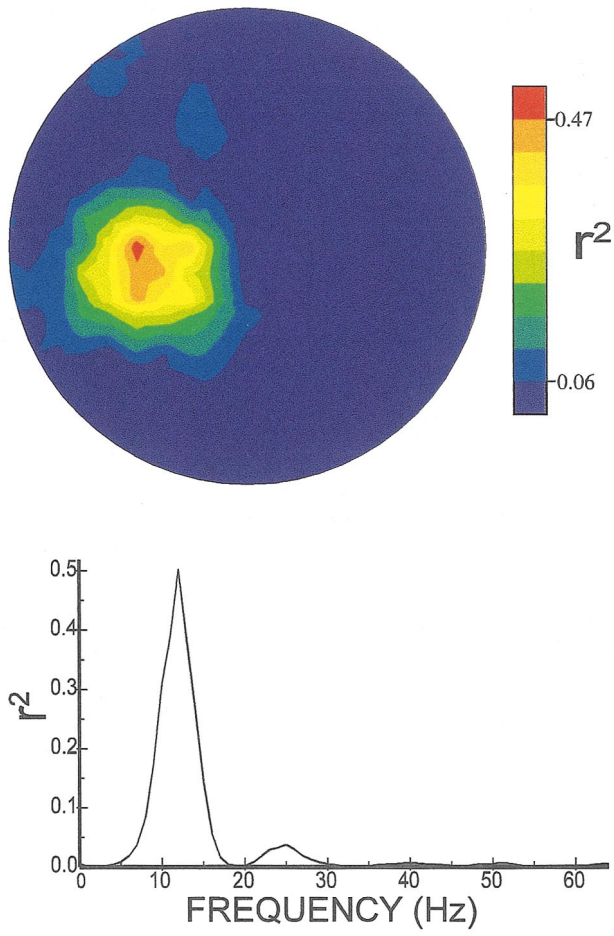

Fig. 1. Topographical and spectral specificity of EEG control in one subject. Top: Scalp topography of r^2 for the mu rhythm frequency band that control cursor movement online. (The nose is at the top.) Control is focused over the left sensorimotor cortex. Bottom: r^2 spectrum for electrode C_3 (i.e. over the midpoint of the left central sulcus), which controlled cursor movement online. Control is focused in the mu rhythm frequency band.

to determine what portion of the EEG r^2 value was unique to the EEG, that is, could not be accounted for by the EMG.

3. Results

Target accuracies for the five subjects for the sessions in which EMG was recorded were 80–97%. (In several subjects, performance appeared to be slightly degraded by the novelty and obtrusiveness of the 10 pairs of EMG electrodes.) Fig. 1 illustrates with data from Subject A the topographic and spectral specificity of the EEG control displayed by the subjects. In this person, cursor movement was controlled by mu rhythm amplitude at electrode C_3 (i.e. over the midpoint of the left central sulcus). The r^2 topography for the mu rhythm frequency band shows that control was sharply focused at C_3, and the r^2 spectrum for C_3 show that control was also sharply focused in the mu rhythm band.

As expected from the high levels of performance achieved by the subjects, the r^2 values for the EEG control signal that determined cursor movement (e.g. for Subject A in Fig. 1, this measure was mu rhythm amplitude at C_3) were also high. For the sessions of EMG recording, the EEG r^2 values for the 5 subjects, A-E, were 0.50, 0.21, 0.63, 0.36, and 0.24, respectively.

Fig. 2 displays the EMG results. It shows, for each muscle group of each subject, the EMG amplitudes (in percent of MVC) for top and bottom targets and their corresponding r^2 values. It also shows for each subject the target accuracy and the EEG r^2 value. EMG levels during performance were low, averaging 4.0 ± 4.4% (SD) of MVC for top targets and bottom targets. Most important, the EMG r^2 values, i.e. the measure of the correlation between EMG level and target position, were very low, averaging 0.01 ± 0.02. They contrasted sharply with the EEG r^2 values which averaged 0.39 ± 0.18.

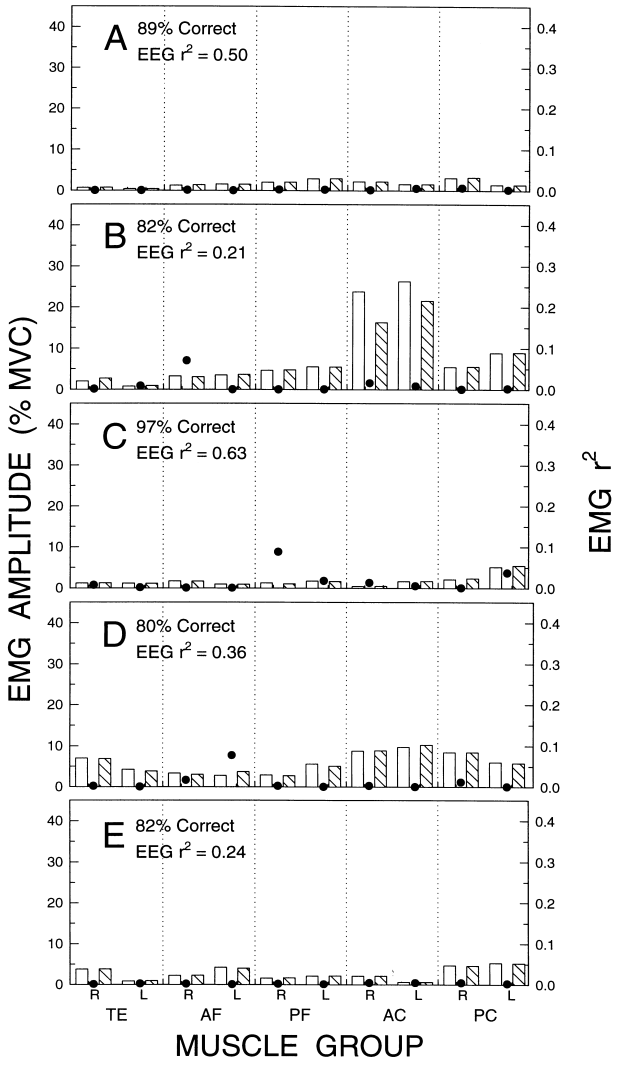

Fig. 2. EMG amplitude (in percent of maximum voluntary contraction (MVC)) for top targets (hatched bar) and bottom targets (open bar) and their corresponding r^2 value (filled circle) for right (R) and left (L) thenar eminence (TE), anterior forearm (AF), posterior forearm (PF), anterior calf (AC), and posterior calf (PC) muscle groups of each subject. Also shown for each subject are the performance accuracy (i.e. percent of targets hit) and the r^2 value of the EEG control signal for the sessions in which EMG was recorded. EMG activity is low, and minimally if at all correlated with target position. In contrast, the EEG control signal that determined cursor movement is clearly correlated with target position.

To further evaluate the possible contribution of muscle activity to EEG control, we performed an analysis of covariance to determine what portion of the EEG r^2 value was unique to the EEG, that is, could not be accounted for by the EMG. For the 50 evaluations (i.e. 10 muscle groups in each of 5 subjects) 99 ± 3% (SD) of the EEG r^2 was unique to the EEG. Even in the three worst cases (i.e. Fig. 2, right PF of Subject C, left AF of Subject D, and right AF of Subject B, in which EMG r^2 values are 0.09, 0.08, and 0.07, respectively), 82%, 87%, and 90% of the EEG r^2 was unique to the EEG. This result supports the conclusion that distal limb muscles had little or no role in the EEG control signal that the subject produced in response to target location.

Finally, because recent studies of the relationships of mu and beta rhythms to actual motor activity indicate that changes in rhythm amplitude may follow muscle activity (Pfurtscheller and Aranibar, 1979; Pfurtscheller et al., 1996), we also evaluated EMG during the period immediately before cursor movement (i.e. the 1-s pause between the appearance of the target and the beginning of cursor movement). The results were comparable to those in Fig. 2. EMG activity averaged 5.6 ± 4.2% (SD) of MVC, and EMG r^2 averaged 0.03 ± 0.06 SD. Thus, neither EMG during cursor movement nor EMG before cursor movement could account for the subjects' EEG control.

4. Discussion

The data constitute a representative sample of the activity of distal limb muscle groups during EEG-based cursor control. They indicate that, during accurate cursor control by trained subjects, the activity in these muscle groups is very low and is minimally if at all correlated with target position. These muscle groups have the largest cortical representations of limb muscles (Porter and Lemon (1993) for review). More proximal limb muscles and trunk muscles have much smaller representations, so that it is unlikely that activity in them could account for the mu or beta rhythm control displayed by the subjects. It is also unlikely that proximal muscle activity would do so without comparable activity in distal muscles. While cranial and facial muscles have substantial cortical representations, their mediation of cursor control is largely ruled out by the 64 EEG channels themselves. These cover the scalp from forehead to inion and from preauricular point to preauricular point, and, as illustrated in McFarland et al. (1997a), would detect, even with a bandpass of 1–60 Hz, correlations between target position and EMG activity from posterior neck muscles, forehead muscles, extraocular muscles, or jaw muscles. Furthermore, the foci of mu and beta rhythm control are

typically located over the arm and/or leg areas of sensorimotor cortex (e.g. Fig. 1), rather than over the facial area, which is more lateral.

Thus, while it remains theoretically possible that muscle activity somewhere in the body is highly correlated with cursor control, the results strongly indicate that EEG-based cursor control does not depend on muscle activity, and thus is not mediated through the effects of muscle activity on mu and beta rhythms. In this lack of dependence on actual muscle activity, the EEG control exercised by trained subjects resembles the lesser impact that motor imagery has on mu and beta rhythms in untrained subjects (McFarland et al., 1997c). Indeed, subjects often report using various kinds of motor imagery to control cursor movement, particularly early in training.

While motor activity, measured here as EMG, does not appear to be responsible for EEG-based cursor control, it would not be surprising to find some correlations between mu and beta rhythm amplitudes and concurrent EMG. These rhythms reflect the state of sensorimotor cortex and that state affects activity in direct and indirect descending connections to motoneurons in the spinal cord. Depending on other influences on these connections and on the motoneurons, the changes in sensorimotor cortex that produce target-specific changes in the mu or beta rhythms recorded from the overlying scalp might also produce changes in EMG activity. EMG does not produce the EEG control described here, but it might reflect to some small degree the cortical processes that do produce this control.

The results are consistent with evidence to date that individuals with severe motor disabilities are able to learn EEG-based cursor control (e.g. Miner et al., 1996; McFarland et al., 1998). At the same time, it is likely that the ability to learn will depend on the nature of the disability. Disorders of spinal cord motoneurons and interneurons, or of spinal cord descending pathways, such as spinal cord injury, are likely to be compatible with EEG-based communication using mu or beta rhythms, while disorders of sensorimotor cortex, which affect the generators of mu and beta rhythm, may not be compatible.

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