

Changes in EEG Power Spectra During Biofeedback of Slow Cortical Potentials in Epilepsy

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The goal of the study was to explore parallel changes in EEG spectral frequencies during biofeedback of slow cortical potentials (SCPs) in epilepsy patients. Thirty-four patients with intractable focal epilepsy participated in 35 sessions of SCP self-regulation training. The spectral analysis was carried out for the EEG recorded at the same electrode site (Cz) that was used for SCP feedback. The most prominent effect was the increase in the θ power (6.0–7.9 Hz) and the relative power decrement in all other frequency bands (particularly δ 1, α 2, and β 2) in transfer trials (i.e., where patients controlled their SCPs without continuous feedback) compared with feedback trials. In the second half of the training course (i.e., sessions 21–35) larger power values in the δ , θ , and α bands were found when patients were required to produce positive versus negative SCP shifts. Both across-subject and across-session (within-subject) correlations between spectral EEG parameters, on the one hand, and SCP data, on the other hand, were low and inconsistent, contrary to high and stable correlations between different spectral variables. This fact, as well as the lack of considerable task-dependent effects during the first part of training, indicates that learned SCP shifts did not directly lead to the specific dynamics of the EEG power spectra. Rather, these dynamics were related to nonspecific changes in patients' brain state.

KEY WORDS: EEG biofeedback; epilepsy; slow cortical potentials; spectral analysis.

INTRODUCTION

Two biofeedback approaches have been developed for intractable epilepsy, one of them based on rhythmical components of the EEG and the other on slow cortical potentials (SCPs). The proponents of the former approach (Lubar, 1984; Lubar *et al.*, 1981; Sterman, 1986) train their patients to enhance the sensorimotor rhythm (SMR) and/or to suppress slow rhythmic activities (e.g., in the theta band). In SCP training (Birbaumer *et al.*, 1991; Kotchoubey *et al.*, 1996; Rockstroh *et al.*, 1993), patients learn to control slow (i.e., lasting from several hundreds of milliseconds to several seconds) shifts of their cortical potentials; they are instructed either to increase or to decrease a negative SCP shift

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appearing between the onset and the offset of a trial. In the former approach, it remains unclear how far the SMR increase, rather than the decrease in slow rhythmic activity, underlies the observed changes in seizure frequency. In the latter approach, recent data (Kotchoubey *et al.*, 1996, 1999) indicate that it is the ability to suppress cortical negativity, rather than the ability to differentiate between the two tasks, that is related to the following clinical improvement.

No research has been conducted to date to compare directly the rate of clinical changes (e.g., seizure reduction or neuropsychological improvement) attained with these two kinds of biofeedback therapy, probably because of the high costs of such a study. A comparison of published research is difficult due to numerous differences between studies with respect to such factors as sample size, criteria for selection of patients, number, duration, and frequency of training sessions, and duration of the follow-up period during which the clinical dynamics were recorded. An inspection of the corresponding reports indicates that, on the average, the success rates of SCP and SMR biofeedback appear to be similar, with an improvement being observed in about two-thirds of the patients (Kotchoubey *et al.*, 1997, 1999; Lantz & Sterman, 1988; Lubar, 1984; Rockstroh *et al.*, 1993; Sterman, 1984).

These considerations may raise a question, whether the two approaches really affect different cortical mechanisms, i.e., whether the self-controlled EEG components in these two techniques really differ. It might be, for instance, that while learning to control the SMR, patients, in fact, modify their SCPs, which can also be recorded over the sensorimotor cortex and may correlate with rhythmical activity. Or during SCP self-regulation, patients might suppress their slow spectral EEG components (delta and/or theta). The biofeedback literature contains plenty of examples where subjects operantly control secondary functions, x_2, x_3, x_4, \dots , while learning to regulate x_1 (e.g., Lacroix, 1986; Plotkin, 1976, 1977).

To disentangle these relationships, one has to record other EEG components in addition to the target function. In SMR training, the use of AC EEG amplifiers with a relatively short time constant makes simultaneous SCP recording impossible, since SCPs require either AC amplifiers with a long time constant (at least 8 sec) or DC amplifiers (i.e., the time constant is infinite large). In contrast, relatively fast EEG oscillations can be recorded with a long or a short time constant, deliberately. Thus it is simpler to record EEG frequencies during SCP training than vice versa. In the present study, we did not examine what effect training in θ reduction and/or SMR increase might have on SCP. Rather, we were recording EEG rhythms in the range from 0.3 to 30 Hz in patients who learned to regulate their SCPs over extended periods.

Birbaumer *et al.* (1990, 1999) have shown that negative SCPs are generated in the upper cortical layers after excitatory thalamocortical, intracortical, or interhemispheric inflow to the dendritic trees. The slow synchronous depolarization underlying negative SCPs uses cholinergic and glutamatergic synapses and mobilizes the cell in the deeper layers for firing. The physiological basis of positive SCPs is less clear. In brain areas with extensive folding and on the orbitofrontal, temporal, and occipital pole, the positivities are particularly difficult to interpret due to variable dipole direction. However, if the dipoles under the electrode are vertically oriented, in most cases a positivity indicates a reduction of excitability (mediated by GABA-ergic systems) in the upper cortical layers, with a concomitant inhibition of behavioral output. Based on these physiological considerations, one should expect increased frequencies and desynchronization during learned cortical negativity and increased slow synchronized rhythmic patterns during self-produced positivities.

METHODS

Epilepsy Patients

Thirty-four patients (19 females) with drug-resistant focal epilepsy (10 right temporal, 9 left temporal, 15 with unlocalized or multifocal seizures) participated in the study. None of the patients had primarily generalized seizures with a sudden and complete loss of consciousness (i.e., grand mal or petit mal); however, many seizures were secondarily generalized, that is, a seizure began with focal symptoms (such as cramps in an arm or leg) followed by generalized convulsions and loss of consciousness. The mean age of the patients was 34.3 years (SD = 8.44 years), the mean seizure history was 23 years (SD = 10.6 years). The mean seizure frequency prior to training was 3.46 per week (SD = 6.31 per week; median = 1.21 per week). Patients with psychogenic seizures, psychotic symptoms, or progredient neurological diseases as well as those with an IQ below 80 (WAIS-R) were excluded. All patients were medicated with one or two antiepileptic drugs, with the medication regime remaining constant for at least 5 months prior to the beginning of treatment and up to 12 months after its termination.

Feedback Schedule

The training course consisted of two phases, the first including 20 daily sessions and the second 15 sessions. The two phases were separated by an 8-week interval. Each session entailed 144 trials. In *feedback trials*, lasting for 8 sec, the actual SCP amplitude was referred to the 1-sec pretrial baseline and presented in the form of a rocket-like object on a computer screen. Simultaneously a letter A or B signaled the type of task (i.e., producing positivity versus negativity) that had to be performed during that trial. These letters served as discriminative stimuli. In *transfer trials*, only the letter A or B was presented for 8 sec, and patients had to perform the corresponding task without feedback. The ratio between positivity and negativity trials was 50/50 during the first phase and 70/30 during the second phase (i.e., more trials with required positivity was presented), whereas the ratio between feedback and transfer trials varied between 70/30 and 30/70 according to the patient's performance. The first 30 trials in each session were always feedback trials.

Recording

The EEG was recorded at the vertex (Cz) referred to the two mastoid electrodes linked over a 15-k Ω shunt, using a high-frequency cutoff filter of 40 Hz and a time constant of 10 sec. The vertical EOG was recorded using two electrodes above and below the left eye. The data were analyzed in 9-sec epochs beginning 1 sec prior to the presentation of the discriminative stimulus (and the rocket, in feedback trials) and lasting until all visual stimuli disappeared. The EEG was on-line EOG corrected (for the correction algorithm see Kotchoubey *et al.*, 1996) and fed back as rocket movements, with each position of the rocket on the screen corresponding to the averaged EEG amplitude over a 500-msec epoch. These epochs slid with a 100-msec shift. Thus during an 8-sec trial, patients observed 80 subsequent positions of the rocket, which created an illusion of a quasi-continuous movement.

Data Analysis

Although the EEG was on-line artifact corrected, the noncorrected data were recorded. It was then additionally off-line EOG corrected using the algorithm of Gratton *et al.* (1983). Further, trials containing zero-line segments or amplitude values higher than $150 \mu\text{V}$ were removed. Fast Fourier transformation was carried out over the 8-sec feedback interval using a Hamming-type window, which resulted in squared amplitude values (i.e., μV^2). These data were square-root transformed, thus resulting in simple amplitude values (i.e., μV) and then averaged in the frequency domain according to Task (positivity versus negativity) and Trial Type (feedback versus transfer).⁴ For SCP analysis, the EEG amplitudes were averaged in the time domain as well. An example of the averages in both the time and the frequency domains is shown in Fig. 1.

The resulting average EEG power spectra were subdivided into the δ (0.3- to 3.9-Hz), θ (4.0- to 7.9-Hz), α (8- to 13-Hz), and β (13.1- to 30-Hz) bands. The maximal power and its location on the frequency scale were calculated for each band. In addition, mean power values were obtained for narrower frequency ranges: $\delta 1$ (0.3–2.0 Hz), $\delta 2$ (2.1–3.9 Hz), $\theta 1$ (4.0–5.9 Hz), $\theta 2$ (6.0–7.9 Hz), $\alpha 1$ (8.0–10.0 Hz), $\alpha 2$ (10.1–13.0 Hz), $\beta 1$ (13.1–18.0 Hz), and $\beta 2$ (18.1–30.0 Hz). All power values were taken in relation to the maximum power value in the particular spectrum, as shown in Fig. 1. The parameters of these frequency bands were subjected to a repeated-measures ANOVA, separately for the first and second training phase, with factors Task (2 levels: positivity versus negativity), Trial Type (2 levels: feedback versus transfer), and Session (20 or 15 levels, for the first and the second phase, respectively). The degrees of freedom for the last factor were corrected for nonsphericity using the Greenhouse–Geisser ϵ .

A correlational analysis included both within-subject (across session) and between-subject product–moment correlations. The former indicated whether parallel changes in different EEG parameters occurred in the course of SCP training, and the latter indicated whether patients with larger values in some EEG variable (e.g., negative SCP in transfer trials) also tended to have larger (or smaller) values in some other variable (e.g., $\delta 1$ power in feedback trials). The correlations were averaged using Fisher's logarithmic function.

RESULTS

SCP

The SCP data have been reported elsewhere (Kotchoubey *et al.*, 1996, 1997, 1998) and are summarized in Table I. In both phases of training, patients were able to produce the required directional SCP shift (highly significant effects of the factor Task), and better performance was achieved with than without feedback (highly significant Task \times Trial Type interactions). During the first training phase, the patients' SCP amplitudes became slightly more positive across sessions, as indicated by a marginally significant effect of the factor Session.

⁴We tested different kinds of spectral data analysis in addition to that described in the main text. Thus a log transformation of the spectral data was tested apart from the square-root transformation. Further, analyses of variance were run for both raw data and several types of normalization. The tendencies found with different techniques were very similar, but they were best pronounced when square-root transformed and normalized-to-maximum data were entered into the analysis. For this reason, only these results are reported.

Table I. Summary of the Significant ANOVA Effects on SCP During the First and Second Training Phase

Phase	Effect	df	F
1st	Task	1,33	10.75***
	Task × Trial Type	1,33	10.93***
	Session	19,627	1.76*
2nd	Task	1,33	12.60****
	Task × Trial Type	1,33	13.45****

Note. * $p < .10$; *** $p < .01$; **** $p < .001$.

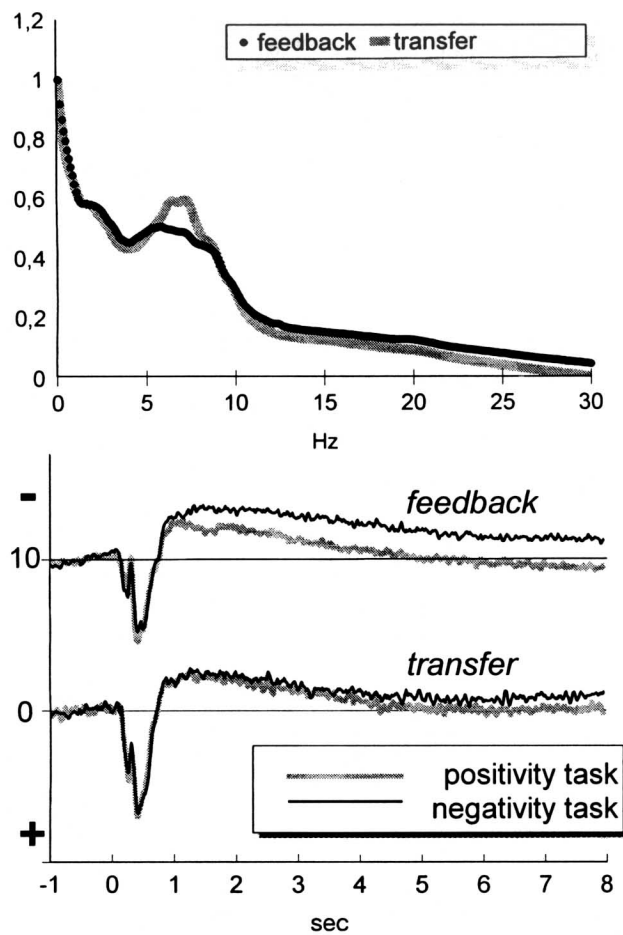


Fig. 1. The EEG averaged across the first training phase (20 sessions) in the frequency domain (top) and time domain (bottom). Top: One can see a clear difference between feedback trials and transfer trials. The power values are presented in relation to the maximal power taken as 1. Bottom: The effect of Task (positivity versus negativity) can be seen, which was larger in feedback trials than in transfer trials (Task × Trial Type interaction; see text). The amplitudes are presented as microvolts; the negativity is up.

A difference between the positivity and the negativity task attained a significance level only in feedback trials ($p < .001$), and not in transfer trials ($p < .20$). During the second training phase, no effect of Session was obtained. Despite the significant Task \times Trial Type interaction, the difference between the two tasks was found to be significant in both feedback and transfer trials ($p < .001$ and $p < .01$, respectively).

Seizures

While we were writing this paper, 28 of the 34 patients already had a follow-up of at least 6 months. A technique of sequence analysis (Künkel, 1979) was used to estimate the significance of individual changes in seizure frequency during this time period. Nine patients demonstrated a significant decrease in the seizure rate (with an error probability of $\alpha = .05$), by 72%, on the average. A further 11 patients also showed a decreasing tendency (by 19%, on the average), which, however, did not reach the .05 significance level. A significant lack of change in seizure frequency (with an error probability of $\beta = .10$) was found in the remaining eight patients. None of the patients showed an increase in the seizure rate. Most changes started after the end of training.

Spectral Data: General

Table II represents the descriptive data on EEG spectral parameters and their correlations with the target function, i.e., the SCP amplitude. As shown in Table II, these correlations were generally low and inconsistent. Specifically, 97.6% of between-subject correlations and 91.1% of within-subject (across-session) correlations were nonsignificant. Chi-square tests were then run to test the hypothesis that correlations in the four frequency bands were randomly distributed, with $\rho = 0$. For the α band only, a highly significant $\chi^2 = 94.3$ ($df = 24$, $p < .001$) indicated that the randomness hypothesis had to be rejected.

Table II. Mean Values of EEG Spectral Parameters and Their Correlations with SCPs

Band	Variable	Mean (SE)	Mean correlation (r) with SCP amplitude (range)	
			Within subject	Between subjects
δ	Power ($\delta 1$)	0.926 (0.0027)	.102 (.439, -.172)	-.031 (.298, -.227)
	Power ($\delta 2$)	0.566 (0.0020)	-.000 (.426, -.412)	-.041 (.045, -.159)
	Peak frequency	0.803 (0.0079)	.023 (.404, -.309)	-.031 (.034, -.097)
	Minimum frequency	3.393 (0.0094)	-.080 (.342, -.379)	-.001 (.078, -.059)
θ	Power ($\theta 1$)	0.452 (0.0014)	-.050 (.314, -.388)	-.021 (.020, -.152)
	Power ($\theta 2$)	0.673 (0.0026)	-.075 (.240, -.426)	.074 (.582, -.085)
	Peak frequency	5.950 (0.0107)	-.066 (.266, -.492)	.012 (.069, -.055)
	Minimum frequency	6.357 (0.0166)	.174 (.482, -.242)	.047 (.090, .000)
α	Power ($\alpha 1$)	0.514 (0.0014)	-.170 (.298, -.558)	-.017 (.060, -.107)
	Power ($\alpha 2$)	0.285 (0.0008)	-.172 (.327, -.529)	-.033 (.037, -.130)
	Peak frequency	8.824 (0.0075)	.040 (.524, -.253)	.003 (.084, -.143)
	Minimum frequency	12.308 (0.0105)	-.058 (.286, -.419)	.007 (.071, -.046)
β	Power ($\beta 1$)	0.225 (0.0008)	-.025 (.306, -.362)	-.009 (.052, -.079)
	Power ($\beta 2$)	0.135 (0.0006)	-.111 (.209, -.398)	-.018 (.071, -.084)
	Peak frequency	15.382 (0.0366)	-.153 (.156, -.516)	-.006 (.043, -.061)
	Minimum frequency	28.625 (0.0272)	.099 (.456, -.365)	-.005 (.054, -.064)

This was due to a tendency for negative SCP shifts to demonstrate moderate but consistent correlations (mostly between $-.15$ and $-.3$) with lower α power.

EEG Oscillations: First Training Phase

ANOVA results for the first training phase are summarized in Table III. During this phase, the power in both the $\delta 1$, and the $\delta 2$ range was higher in feedback trials than in transfer trials (main effects of Trial Type; see Table III). The same was true for the $\theta 1$ power. In contrast, $\theta 2$ was significantly higher in transfer trials than in feedback trials. Additionally, the $\delta 1$ power was higher with the positivity than the negativity task (main effect of Task), particularly in feedback trials (marginally significant Task \times Trial Type interaction). The $\delta 1$ power correlated positively with the $\theta 1$ power (r from $.39$ to $.64$), and the $\delta 2$ power also correlated with the $\theta 2$ power (r from $.22$ to $.46$). However, peak power values of the δ and θ bands were inversely correlated across subjects (r from $-.52$ to $-.88$) as well as across sessions (mean $r = -.57$). Thus on the one hand, mean power values of low-frequency oscillations were directly related to each other, presumably reflecting an overall tendency

Table III. Significant ANOVA Effects on EEG Spectral Data (For Details, See the Appendix)

Index	Effect	df	F
1st training phase			
$\delta 1$ power	Task	1,33	5.25**
	Trial Type	1,33	8.44****
$\delta 2$ power	Session	19,627	2.18**
	Trial Type	1,33	16.76****
δ peak frequency	Trial Type	1,33	4.44**
$\theta 1$ power	Trial Type	1,33	7.57****
	Session	19,627	2.78****
$\theta 2$ power	Trial Type	1,33	7.45**
	Session	19,627	2.70**
$\alpha 2$ power	Trial Type	1,33	10.92****
$\beta 2$ power	Trial Type	1,33	11.47****
2nd training phase			
$\delta 1$ power	Trial Type	1,33	8.93****
$\delta 2$ power	Task	1,33	54.85****
	Trial Type (TT)	1,33	10.43****
	TT \times Session	14,462	2.29**
δ peak frequency	Task	1,33	3.53*
$\theta 1$ power	Trial Type	1,33	6.57**
$\theta 2$ power	Trial Type	1,33	7.53****
	Task	1,33	75.66****
	TT \times Session	14,462	2.02**
$\alpha 2$ power	Task	1,33	23.47****
α peak frequency	Task	1,33	6.95**
$\beta 2$ power	Task	1,33	9.59****
	Trial Type	1,33	15.67****

Note. * $p < .10$; ** $p < .05$; *** $p < .01$; **** $p < .001$.

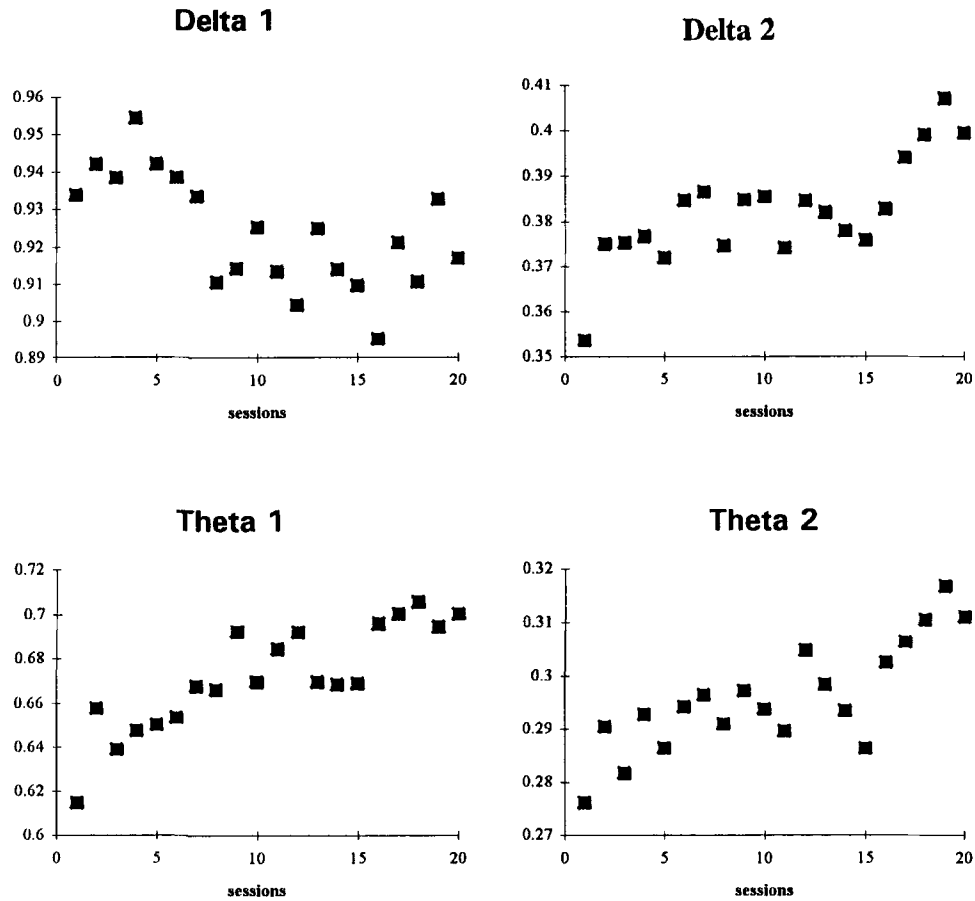


Fig. 2. Significant changes in the mean power of $\delta 1$, $\delta 2$, $\theta 1$, and $\theta 2$ as a function of session number during the first training phase.

of some patients to have more θ and δ waves than others. On the other hand, there was a trend of the peak of those slow oscillations to be either in the θ or in the δ domain, which might explain the negative correlations.

Further, with increasing $\delta 2$, the peak frequency of the θ band tended to decrease (with moderate r , from $-.19$ to $-.27$), thus approaching the δ band. With increasing $\theta 1$, conversely, the frequency of the δ peak increased (r from $.31$ to $.66$), thus moving toward θ .

As shown in Fig. 2, the power of both $\theta 1$ and $\theta 2$ increased consistently across sessions, regardless of the task and type of trial. A similar (although nonsignificant) trend was obtained for $\delta 2$. In contrast, the $\delta 1$ power significantly decreased with training. The within-subject (across-session) correlations indicated that these changes were interrelated, with the $\theta 1$ and $\theta 2$ power being significantly and inversely related to the $\delta 1$ power (mean $r = -.56$, $p < .001$).

A higher power in the $\alpha 2$ and $\beta 2$ ranges was found in trials in which patients received continuous SCP feedback, compared with transfer trials (main effects of Trial Type; see Table III). The spectral power of the whole α and β bands was strongly correlated, with between-subject correlations varying from $.74$ to $.80$ and the mean within-subject

correlation being $r = .67$ ($p < .01$). Moreover, power values of α correlated with those of θ (r from .66 to .76) and δ (r from .60 to .71).

EEG Oscillations: Second Training Phase

During the second phase, most differences related to the Trial Type (i.e., feedback versus transfer) factor were basically the same as in the first phase, i.e., the power in the δ band, as well as $\alpha 2$ and $\beta 2$, were larger in trials with feedback, while the $\theta 2$ power was larger in trials without feedback (see Table III and the Appendix). The peak θ frequency was higher with feedback than without feedback, repeating the effect observed in the first phase. In addition, as shown in Table III, highly significant *task-dependent* differences in the power spectrum appeared, which were not observed in the first phase. Thus the higher power in the $\delta 1$, $\delta 2$ and $\theta 1$ bands was found with required positivity than with required negativity. Further, the frequency of the δ peak was significantly shifted from 0.74 Hz in the negativity task to 0.86 Hz in the positivity task. As was already found in the first phase, the parameters of δ and θ were interrelated, with the mean within-subject correlation between the δ and the θ power being .70 ($p < .001$), and the mean between-subject correlation being .44 ($p < .01$). Also, the EEG power in $\delta 2$ and $\theta 1$ decreased across sessions particularly in feedback trials, thereby yielding significant Trial Type \times Session interactions.

Concerning faster EEG rhythms, the EEG power in the ranges of $\alpha 2$ and $\beta 2$ was larger in feedback trials than in transfer trials, again replicating the finding of the first phase. Furthermore, these power values were larger with required positivity than with required negativity (main effects of Task; Table III). In the positivity task, the peak frequency of the α band was lower (i.e., closer to the θ band: 8.83 Hz, versus 8.92 Hz in the negativity task), whereas the minimum frequency was significantly ($p < .01$) higher (i.e., closer to the β band) than in the negativity task (12.37 versus 12.24 Hz).

As in the first phase, power values of α were strongly related with those of β (between-subject r from .75 to .80; mean within-subject $r = .67$, $p < .01$), θ (between-subject r from .68 to .73; mean within-subject $r = .66$) and δ (between-subject r from .59 to .64; mean within-subject $r = .61$).

EOG Activity

The activity in the vertical EOG channel was FFT-analyzed and underwent a similar ANOVA with factors Task, Trial Type, and Session. As in the Cz channel, the activity in the range between 0.3 and 6 Hz (i.e., $\delta 1$, $\delta 2$, and $\theta 1$) was slightly higher in feedback trials than in transfer trials, but the opposite was true for $\theta 2$. However, none of these differences attained even a .10 level of significance. Thus the effects obtained in the Cz channel cannot be attributed to eye movement artifacts.

DISCUSSION

The main objective of the present study was to test the hypothesis that self-control training of slow cortical potentials (SCPs) can lead to consistent changes in the EEG power spectrum related to the target SCP function. Specifically, negativity should correlate with increased desynchronization, and positivity with the appearance of slow synchronized activity. If this hypothesis were correct, one might further suggest that the therapeutic effects of SCP

training (see Kotchoubey *et al.*, 1999; Rockstroh *et al.*, 1993) are mediated by changes in EEG oscillating activity.

Although several significant changes in EEG power spectrum were found in the present data, the hypothesis about consistent relationships between SCP self-regulation and subsequent secondary dynamics of EEG rhythms received no consistent support. First, the SCP amplitude and polarity varied according to the task requirements from the very beginning (i.e., the SCP changed significantly in the required direction), while the main effect of Trial Type was not significant. At the same time, EEG spectral variables depended mostly on whether or not the feedback signal was presented, whereas task-dependent effects appeared only in the second phase of training, i.e., after 20 sessions. Second, linear changes in θ and δ power across sessions did not parallel SCP changes. Third, neither across-subject nor across-session correlations between the SCP amplitudes and the spectral data showed a pattern that would indicate consistent relationships between the two classes of EEG parameters. Spectral parameters with a frequency close to the SCP (i.e., δ) did not demonstrate higher correlations than variables in the remote frequency bands (i.e., β). Thus, even when similar effects in terms of group means were observed, they did not show that a change in one variable (e.g., the SCP amplitude) was accompanied by a corresponding change in another variable (e.g., the mean δ power). It may be argued that changes in EEG spectrum might have been recorded at other sites (e.g., in the occipital area for the α rhythm), but it seems unlikely that higher correlations would be found between the SCP and the EEG frequencies recorded in different locations than at an identical recording site (i.e., vertex, in the present study).

It is therefore implausible that the effects of SCP training may be conceived as mediated by concomitant changes in faster EEG rhythms. This conclusion is in line with neurophysiological data indicating that cortical oscillations with frequencies above and below 1 Hz (i.e., SCP and δ waves, respectively) are generated in sleeping animals by two principally different mechanisms (Steriade *et al.*, 1993), though it remains unclear how far these data can be generalized to the waking state. Interestingly enough, slow waves were characterized by very consistent relationships among the firing patterns in reticular thalamic, thalamocortical, and pyramidal neurons, whereas all rhythmic oscillations in the ranges of 1 to 4 and 7 to 14 Hz and fast rhythms (15 to 40 Hz) displayed continuous variation in frequency and in phase relation between cells in both sleep and wakefulness (Contreras & Steriade, 1995, 1997; Steriade *et al.*, 1996). This intrinsic variability and temporal changeability of faster EEG rhythms, especially those characterizing active wakefulness (Steriade *et al.*, 1996), compared with a stable pattern of cellular excitations and inhibitions during SCP shifts, may be a cause of the inconsistent correlations between the two domains of EEG parameters found in the present study.

Transfer trials were characterized by increased power of θ activity and decreased power in all other spectral bands. On these trials patients were confronted with an extremely difficult task, as they had to control their brain processes using exclusively their internal cues, without external feedback. It may be speculated that the autocentric attention (Schachtel, 1959), directed onto changes inside the body, is related to the increment in the θ power and that this ability to concentrate on one's own brain events increased across sessions, as indicated by an increment in the θ power and a parallel decrement in the δ power. This speculation finds support in the data demonstrating an increase in θ activity in conditions related to inner concentration, such as meditation (Delmonte, 1984; Herbert & Lehmann, 1977), autogenic training (Jacobs & Lubar, 1989), and other relaxation procedures (e.g., Ikemi,

1988). This does not imply that patients during SCP training really practice techniques similar to meditation or relaxation, since no indication of such techniques or relaxation experience was found in the self-report data (Roberts *et al.*, 1989). However, these concentration techniques and SCP self-regulation without external feedback, though very different in many other respects, both are states of inward attention. Furthermore, the observed θ increment may be related to, or contain a component of, the frontal θ that manifests itself in conditions of focused attention, particularly during working memory tasks (Gevins, Smith, McEvoy, & Yu, 1997; Lang, Lang, Diekmann, & Kornhuber, 1987; Laukka, Jarvilehto, Alexandrov, & Lindquist, 1995; Wei, Zhao, Yan, Duan, & Li, 1997). The frontal theta activity may constitute the cortical expression of septo-hippocampal theta which correlates with cortical activation during skill acquisition.

The inhibitory change related to cortical positivity may also have affected oscillatory EEG activity. Though theoretically, depending on the depth and the location of electrical dipoles, superficial positive EEG shifts can manifest both excitatory and inhibitory processes. Numerous behavioral changes during positive slow waves (Bauer, 1984; Birbaumer *et al.*, 1992; Lutzenberger *et al.*, 1982, 1993) clearly indicate that these shifts are related to cortical inhibition. Already in the first training phase a slightly higher power in the δ band was observed with required positivity than with required negativity. In the second phase, the condition of required positivity was characterized by a power increase in all three “slow” bands, namely, δ , θ , and α . Maximal peak frequencies in this condition were shifted toward lower frequencies; this tendency was significant for δ and α and close to significance for θ . All these changes may indicate an increment of inhibitory activity in the cortical area underlying Cz. This statement might be questioned on the basis of the finding of a greater β_2 activity with required positivity than with required negativity, since the oscillations in the range of 18–30 Hz are usually regarded as “desynchronization.” However, the complexity of the patients’ state during the positivity condition should not be underestimated. The difficulty of this task, combined with the random change of task demands during a session, requires the maintenance of a state of high tonic vigilance. Simultaneously, phasic local inhibition should be achieved in the central brain regions. Recent data clearly indicate a compound nature of the scalp-recorded β that consists, first, of overlapping asynchronous α waves of different frequencies and, second, of highly synchronized fast oscillations in the β and γ range (Mulholland, 1995; Steriade *et al.*, 1993). These oscillations can, moreover, rapidly fluctuate in their frequency within an epoch as short as 8 sec (i.e., the duration of one trial in the present study). Therefore the conventional spectral analysis does not permit unequivocal interpretation of the observed increase in the β_2 power as increasing activation, since “one never knows by looking at a broad spectrum whether it is generated by an oscillation with continuously changing frequencies, by an oscillation with stable frequencies at multiple peaks, or by a combination of the two” (Steriade *et al.*, 1996, p. 415).

To summarize, the EEG power spectra of patients with drug-resistant epilepsy were found to change significantly during learning of SCP self-regulation. These changes, however, cannot be regarded as an immediate consequence of SCP dynamics. They may be hypothesized to depend, first, on cortical inhibitory processes related to generation of slow positive shifts and, second, on strategies of autocentric attention employed in those trials when no external feedback was presented and patients had to concentrate on their internal state. Further studies using a large electrode montage are necessary to test these preliminary explanations.

APPENDIX

Table A1. Full ANOVA Results for the δ Band

Phase	Index	Effect	df	F
First	$\delta 1$ power	Task	1,33	5.25**
		Trial Type (TT)	1,33	8.44***
		Session	19,627	.27 [§]
		Task \times TT	1,33	3.49*
		Task \times Session	19,627	.80 [§]
		TT \times Session	19,627	.44 [§]
		Task \times TT \times Session	19,627	.97 [§]
	$\delta 2$ power	Task	1,33	2.06 [§]
		Trial Type	1,33	16.76****
		Session	19,627	2.18**
		Task \times TT	1,33	.01 [§]
		Task \times Session	19,627	.51 [§]
		TT \times Session	19,627	.05 [§]
		Task \times TT \times Session	19,627	.58 [§]
	Peak frequency	Task	1,33	3.31*
		Trial Type	1,33	4.44**
		Session	19,627	.19*
		Task \times TT	1,33	.42 [§]
		Task \times Session	19,627	.85 [§]
		TT \times Session	19,627	.98 [§]
		Task \times TT \times Session	19,627	.22 [§]
	Minimum frequency	Task	1,33	.45 [§]
		Trial Type	1,33	2.68 [§]
		Session	19,627	.81 [§]
Task \times TT		1,33	.94 [§]	
Task \times Session		19,627	.08 [§]	
TT \times Session		19,627	.01 [§]	
Task \times TT \times Session		19,627	.21 [§]	
Second	$\delta 1$ power	Task	1,33	3.29*
		Trial Type (TT)	1,33	8.93***
		Session	19,627	.44 [§]
		Task \times TT	1,33	2.37 [§]
		Task \times Session	19,627	.40 [§]
		TT \times Session	19,627	.43 [§]
		Task \times TT \times Session	19,627	.01 [§]
	$\delta 2$ power	Task	1,33	54.85****
		Trial Type	1,33	10.43****
		Session	19,627	.46 [§]
		Task \times TT	1,33	.01 [§]
		Task \times Session	14,462	.27 [§]
		TT \times Session	14,462	2.29** [§]
		Task \times TT \times Session	14,462	.32 [§]
	Peak frequency	Task	1,33	3.53*
		Trial Type	1,33	.25 [§]
		Session	14,462	.01 [§]
		Task \times TT	1,33	.43 [§]
		Task \times Session	14,462	.95 [§]
		TT \times Session	14,462	.45 [§]
		Task \times TT \times Session	14,462	.90 [§]
	Minimum frequency	Task	1,33	.43 [§]
		Trial Type	1,33	.71 [§]
		Session	14,462	.08 [§]
Task \times TT		1,33	1.47 [§]	
Task \times Session		14,462	.34 [§]	
TT \times Session		14,462	.95 [§]	
Task \times TT \times Session		14,462	.07 [§]	

Note. [§]Nonsignificant ($p \geq .10$); * $p < .10$; ** $p < .05$; *** $p < .01$; **** $p < .001$. Small values are rounded up to .01.

Table AII. Full ANOVA Results for the θ Band

Phase	Index	Effect	df	F
First	$\theta 1$ power	Task	1,33	1.21 [§]
		Trial Type (TT)	1,33	7.57***
		Session	19,627	2.78***
		Task \times TT	1,33	.06 [§]
		Task \times Session	19,627	.96 [§]
		TT \times Session	19,627	.90 [§]
		Task \times TT \times Session	19,627	.91 [§]
	$\theta 2$ power	Task	1,33	.01 [§]
		Trial Type	1,33	7.45**
		Session	19,627	2.70***
		Task \times TT	1,33	3.17*
		Task \times Session	19,627	.51 [§]
		TT \times Session	19,627	.11 [§]
		Task \times TT \times Session	19,627	.45 [§]
	Peak frequency	Task	1,33	.85 [§]
		Trial Type	1,33	.09 [§]
		Session	19,627	1.00 [§]
		Task \times TT	1,33	.18 [§]
		Task \times Session	19,627	.01 [§]
		TT \times Session	19,627	.21 [§]
		Task \times TT \times Session	19,627	.23 [§]
	Minimum frequency	Task	1,33	.94 [§]
		Trial Type	1,33	.32 [§]
		Session	19,627	.98 [§]
Task \times TT		1,33	.84 [§]	
Task \times Session		19,627	.19 [§]	
TT \times Session		19,627	.03 [§]	
Task \times TT \times Session		19,627	.56 [§]	
Second	$\theta 1$ power	Task	1,33	.99 [§]
		Trial Type (TT)	1,33	6.57**
		Session	14,462	.52 [§]
		Task \times TT	1,33	1.74 [§]
		Task \times Session	14,462	.66 [§]
		TT \times Session	14,462	.18 [§]
		Task \times TT \times Session	14,462	.39 [§]
	$\theta 2$ power	Task	1,33	75.66****
		Trial Type	1,33	7.53***
		Session	14,462	.01 [§]
		Task \times TT	1,33	.10 [§]
		Task \times Session	14,462	.60 [§]
		TT \times Session	14,462	2.02**
		Task \times TT \times Session	14,462	.70 [§]
	Peak frequency	Task	1,33	30 [§]
		Trial Type	1,33	3.21**
		Session	14,462	.49 [§]
		Task \times TT	1,33	.01 [§]
		Task \times Session	14,462	.18 [§]
		TT \times Session	14,462	.46 [§]
		Task \times TT \times Session	14,462	.01 [§]
	Minimum frequency	Task	1,33	.24 [§]
		Trial Type	1,33	.71 [§]
		Session	14,462	.78 [§]
Task \times TT		1,33	1.18 [§]	
Task \times Session		14,462	.74 [§]	
TT \times Session		14,462	.90 [§]	
Task \times TT \times Session		14,462	.76 [§]	

Note. [§]Nonsignificant ($p \geq .10$); * $p < .10$; ** $p < .05$; *** $p < .01$; **** $p < .001$. Small values are rounded up to .01.

Table AIII. Full ANOVA Results for the α Band

Phase	Index	Effect	df	F
First	$\alpha 1$ power	Task	1,33	.22 [§]
		Trial Type (TT)	1,33	.37 [§]
		Session	19,627	.12 [§]
		Task \times TT	1,33	2.56 [§]
		Task \times Session	19,627	.60 [§]
		TT \times Session	19,627	.55 [§]
		Task \times TT \times Session	19,627	.01 [§]
		$\alpha 2$ power	Task	1,33
	Trial Type		1,33	10.92 ^{***}
	Session		19,627	.84 [§]
	Task \times TT		1,33	.01 [§]
	Task \times Session		19,627	.19 [§]
	TT \times Session		19,627	.23 [§]
	Task \times TT \times Session		19,627	.17 [§]
	Peak frequency		Task	1,33
		Trial Type	1,33	.35 [§]
		Session	19,627	.67 [§]
		Task \times TT	1,33	.29 [§]
		Task \times Session	19,627	.01 [§]
		TT \times Session	19,627	.50 [§]
		Task \times TT \times Session	19,627	.51 [§]
		Minimum frequency	Task	1,33
	Trial Type		1,33	.82 [§]
	Session		19,627	.03 [§]
	Task \times TT		1,33	.03 [§]
	Task \times Session		19,627	.72 [§]
	TT \times Session		19,627	.22 [§]
	Task \times TT \times Session		19,627	.43 [§]
Second	$\alpha 1$ power		Task	1,33
		Trial Type (TT)	1,33	1.45 [§]
		Session	14,462	.16 [§]
		Task \times TT	1,33	1.76 [§]
		Task \times Session	14,462	.17 [§]
		TT \times Session	14,462	.47 [§]
		Task \times TT \times Session	14,462	.01 [§]
		$\alpha 2$ power	Task	1,33
	Trial Type		1,33	10.65 ^{****}
	Session		14,462	.67 [§]
	Task \times TT		1,33	.57 [§]
	Task \times Session		14,462	.15 [§]
	TT \times Session		14,462	.01 [§]
	Task \times TT \times Session		14,462	.42 [§]
	Peak frequency		Task	1,33
		Trial Type	1,33	.69 [§]
		Session	14,462	.01 [§]
		Task \times TT	1,33	.56 [§]
		Task \times Session	14,462	.23 [§]
		TT \times Session	14,462	.79 [§]
		Task \times TT \times Session	14,462	.16 [§]
		Minimum frequency	Task	1,33
	Trial Type		1,33	.18 [§]
	Session		14,462	.86 [§]
	Task \times TT		1,33	.52 [§]
	Task \times Session		14,462	.08 [§]
	TT \times Session		14,462	.33 [§]
	Task \times TT \times Session		14,462	.01 [§]

Note. [§]Nonsignificant ($p \geq .10$); ** $p < .05$; *** $p < .01$; **** $p < .001$. Small values are rounded up to .01.

Table AIV. Full ANOVA Results for the β Band

Phase	Index	Effect	df	F
First	$\beta 1$ power	Task	1,33	1.33 [§]
		Trial Type (TT)	1,33	.58 [§]
		Session	19,627	.58 [§]
		Task \times TT	1,33	.43 [§]
		Task \times Session	19,627	.28 [§]
		TT \times Session	19,627	.82 [§]
		Task \times TT \times Session	19,627	.27 [§]
		$\beta 2$ power	Task	1,33
	Trial Type		1,33	11.47***
	Session		19,627	.01 [§]
	Task \times TT		1,33	.40 [§]
	Task \times Session		19,627	.94 [§]
	TT \times Session		19,627	1.09 [§]
	Task \times TT \times Session		19,627	.40 [§]
	Peak frequency		Task	1,33
		Trial Type	1,33	.48 [§]
		Session	19,627	.31 [§]
		Task \times TT	1,33	.25 [§]
		Task \times Session	19,627	.01 [§]
		TT \times Session	19,627	.01 [§]
		Task \times TT \times Session	19,627	.02 [§]
		Minimum frequency	Task	1,33
	Trial Type		1,33	.75 [§]
	Session		19,627	.83 [§]
Task \times TT	1,33		1.63 [§]	
Task \times Session	19,627		.64 [§]	
TT \times Session	19,627		.52 [§]	
Task \times TT \times Session	19,627		.01 [§]	
Second	$\beta 1$ power		Task	1,33
		Trial Type (TT)	1,33	.24 [§]
		Session	14,462	.59 [§]
		Task \times TT	1,33	.79 [§]
		Task \times Session	14,462	.01 [§]
		TT \times Session	14,462	.01 [§]
		Task \times TT \times Session	14,462	.46 [§]
		$\beta 2$ power	Task	1,33
	Trial Type		1,33	15.67****
	Session		14,462	.05 [§]
	Task \times TT		1,33	.33 [§]
	Task \times Session		14,462	.56 [§]
	TT \times Session		14,462	.07 [§]
	Task \times TT \times Session		14,462	.50 [§]
	Peak frequency		Task	1,33
		Trial Type	1,33	1.06 [§]
		Session	14,462	.86 [§]
		Task \times TT	1,33	.01 [§]
		Task \times Session	14,462	.19 [§]
		TT \times Session	14,462	.05 [§]
		Task \times TT \times Session	14,462	.16 [§]
		Minimum frequency	Task	1,33
	Trial Type		1,33	.38 [§]
	Session		14,462	.85 [§]
Task \times TT	1,33		.66 [§]	
Task \times Session	14,462		.87 [§]	
TT \times Session	14,462		.01 [§]	
Task \times TT \times Session	14,462		.65 [§]	

Note. [§]Nonsignificant ($p \geq .10$); * $p < .10$; *** $p < .01$; **** $p < .001$. Small values are rounded up to .01.

Table A.V. Descriptive Statistics of the δ Band

Phase	Index	Task	Trial Type	Mean (SD)	Range
First	$\delta 1$ power	Negativity	Feedback	0.94 (0.12)	0.53–1.00
			Transfer	0.91 (0.16)	0.44–1.00
		Positivity	Feedback	0.96 (0.09)	0.51–1.00
			Transfer	0.91 (0.17)	0.44–1.00
	$\delta 2$ power	Negativity	Feedback	0.58 (0.08)	0.39–0.75
			Transfer	0.55 (0.10)	0.32–0.69
		Positivity	Feedback	0.59 (0.08)	0.40–0.73
			Transfer	0.55 (0.09)	0.32–0.68
	Peak frequency	Negativity	Feedback	0.89 (0.49)	0.49–2.30
			Transfer	0.81 (0.45)	0.49–2.43
		Positivity	Feedback	0.81 (0.40)	0.49–2.25
			Transfer	0.78 (0.40)	0.49–2.27
	Minimum frequency	Negativity	Feedback	3.42 (0.43)	1.81–3.97
			Transfer	3.44 (0.40)	2.14–3.91
Positivity		Feedback	3.43 (0.45)	2.12–3.93	
		Transfer	3.46 (0.41)	2.02–3.90	
Second	$\delta 1$ power	Negativity	Feedback	0.95 (0.11)	0.52–1.00
			Transfer	0.90 (0.16)	0.41–1.00
		Positivity	Feedback	0.97 (0.09)	0.51–1.00
			Transfer	0.91 (0.17)	0.36–1.00
	$\delta 2$ power	Negativity	Feedback	0.59 (0.08)	0.41–0.71
			Transfer	0.55 (0.09)	0.31–0.68
		Positivity	Feedback	0.60 (0.08)	0.39–0.74
			Transfer	0.57 (0.10)	0.28–0.71
	Peak frequency	Negativity	Feedback	0.88 (0.52)	0.49–2.95
			Transfer	0.84 (0.49)	0.49–2.87
		Positivity	Feedback	0.74 (0.38)	0.49–2.57
			Transfer	0.75 (0.47)	0.49–2.78
	Minimum frequency	Negativity	Feedback	3.29 (0.47)	1.81–3.87
			Transfer	3.34 (0.44)	1.57–3.85
Positivity		Feedback	3.33 (0.52)	1.74–3.96	
		Transfer	3.35 (0.51)	1.69–3.98	

Table A VI. Descriptive Statistics of the θ Band

Phase	Index	Task	Trial Type	Mean (SD)	Range
First	$\theta 1$ power	Negativity	Feedback	0.44 (0.11)	0.20–0.62
			Transfer	0.44 (0.11)	0.22–0.67
		Positivity	Feedback	0.44 (0.11)	0.24–0.61
			Transfer	0.44 (0.11)	0.25–0.67
	$\theta 2$ power	Negativity	Feedback	0.63 (0.19)	0.26–1.00
			Transfer	0.68 (0.22)	0.29–1.00
		Positivity	Feedback	0.63 (0.19)	0.35–1.00
			Transfer	0.68 (0.22)	0.33–1.00
	Peak frequency	Negativity	Feedback	5.87 (1.16)	4.19–7.95
			Transfer	6.03 (1.21)	4.37–7.95
		Positivity	Feedback	5.88 (1.17)	4.23–7.90
			Transfer	6.04 (1.20)	4.28–7.98
Minimum frequency	Negativity	Feedback	6.55 (1.26)	4.23–7.93	
		Transfer	6.22 (1.33)	4.39–7.93	
	Positivity	Feedback	6.56 (1.28)	4.29–7.95	
		Transfer	6.23 (1.31)	4.41–7.95	
Second	$\theta 1$ power	Negativity	Feedback	0.45 (0.12)	0.27–0.74
			Transfer	0.43 (0.12)	0.28–0.71
		Positivity	Feedback	0.46 (0.14)	0.21–0.75
			Transfer	0.46 (0.13)	0.27–0.74
	$\theta 2$ power	Negativity	Feedback	0.64 (0.19)	0.39–0.99
			Transfer	0.67 (0.22)	0.38–1.00
		Positivity	Feedback	0.64 (0.20)	0.33–1.00
			Transfer	0.68 (0.23)	0.37–1.00
	Peak frequency	Negativity	Feedback	5.80 (1.12)	4.15–7.60
			Transfer	5.97 (1.14)	4.32–7.91
		Positivity	Feedback	5.81 (1.08)	4.28–7.69
			Transfer	5.97 (0.22)	4.11–7.92
Minimum frequency	Negativity	Feedback	6.57 (1.19)	4.23–7.90	
		Transfer	6.25 (1.25)	4.33–7.92	
	Positivity	Feedback	6.54 (1.25)	4.21–7.97	
		Transfer	6.26 (1.32)	4.38–7.93	

Table AVII. Descriptive Statistics of the α Band

Phase	Index	Task	Trial Type	Mean (SD)	Range
First	$\alpha 1$ power	Negativity	Feedback	0.51 (0.25)	0.18–0.99
			Transfer	0.52 (0.22)	0.22–0.96
		Positivity	Feedback	0.52 (0.23)	0.22–0.94
			Transfer	0.50 (0.25)	0.24–0.96
	$\alpha 2$ power	Negativity	Feedback	0.29 (0.09)	0.13–0.50
			Transfer	0.28 (0.09)	0.16–0.51
		Positivity	Feedback	0.29 (0.10)	0.16–0.51
			Transfer	0.28 (0.09)	0.16–0.52
	Peak frequency	Negativity	Feedback	8.87 (0.67)	8.13–10.68
			Transfer	8.89 (0.62)	8.11–10.33
		Positivity	Feedback	8.86 (0.61)	8.15–10.50
			Transfer	8.90 (0.62)	8.11–10.34
Minimum frequency	Negativity	Feedback	12.35 (0.58)	9.75–12.91	
		Transfer	12.28 (0.39)	11.42–12.77	
	Positivity	Feedback	12.36 (0.48)	10.44–12.89	
		Transfer	12.30 (0.37)	11.62–12.86	
Second	$\alpha 1$ power	Negativity	Feedback	0.52 (0.24)	0.22–0.97
			Transfer	0.52 (0.22)	0.23–0.96
		Positivity	Feedback	0.51 (0.23)	0.22–0.95
			Transfer	0.52 (0.21)	0.22–0.93
	$\alpha 2$ power	Negativity	Feedback	0.29 (0.09)	0.15–0.50
			Transfer	0.28 (0.08)	0.16–0.49
		Positivity	Feedback	0.30 (0.10)	0.15–0.52
			Transfer	0.29 (0.08)	0.15–0.50
	Peak frequency	Negativity	Feedback	8.92 (0.65)	8.15–10.78
			Transfer	8.90 (0.63)	8.13–10.31
		Positivity	Feedback	8.83 (0.57)	8.13–10.47
			Transfer	8.86 (0.64)	8.13–10.61
Minimum frequency	Negativity	Feedback	12.27 (0.53)	9.97–12.79	
		Transfer	12.22 (0.37)	11.47–12.85	
	Positivity	Feedback	12.39 (0.52)	10.06–12.86	
		Transfer	12.34 (0.41)	11.01–12.83	

Table AVIII. Descriptive Statistics of the β Band

Phase	Index	Task	Trial Type	Mean (SD)	Range
First	β_1 power	Negativity	Feedback	0.22 (0.06)	0.14–0.37
			Transfer	0.22 (0.06)	0.14–0.34
		Positivity	Feedback	0.22 (0.06)	0.14–0.36
			Transfer	0.22 (0.06)	0.14–0.34
	β_2 power	Negativity	Feedback	0.14 (0.04)	0.09–0.24
			Transfer	0.13 (0.05)	0.07–0.25
		Positivity	Feedback	0.14 (0.04)	0.07–0.24
			Transfer	0.13 (0.04)	0.07–0.24
	Peak frequency	Negativity	Feedback	15.17 (1.93)	13.38–20.98
			Transfer	15.34 (1.81)	13.17–19.96
		Positivity	Feedback	15.16 (1.69)	13.31–19.32
			Transfer	15.44 (1.77)	13.32–20.23
Minimum frequency	Negativity	Feedback	28.93 (1.06)	24.87–29.85	
		Transfer	28.82 (1.19)	24.72–29.88	
	Positivity	Feedback	28.90 (0.98)	24.95–29.79	
		Transfer	28.80 (1.31)	23.69–29.77	
Second	β_1 power	Negativity	Feedback	0.24 (0.07)	0.13–0.38
			Transfer	0.23 (0.06)	0.12–0.38
		Positivity	Feedback	0.23 (0.07)	0.11–0.40
			Transfer	0.22 (0.06)	0.10–0.34
	β_2 power	Negativity	Feedback	0.14 (0.05)	0.06–0.25
			Transfer	0.13 (0.04)	0.06–0.22
		Positivity	Feedback	0.14 (0.05)	0.05–0.24
			Transfer	0.13 (0.04)	0.05–0.22
	Peak frequency	Negativity	Feedback	15.21 (1.85)	13.37–19.62
			Transfer	15.44 (1.90)	13.41–19.65
		Positivity	Feedback	15.24 (2.08)	13.19–22.12
			Transfer	15.35 (1.94)	13.28–20.03
	Minimum frequency	Negativity	Feedback	28.57 (1.32)	24.17–29.58
			Transfer	28.54 (1.30)	24.58–29.65
		Positivity	Feedback	28.57 (2.24)	19.10–29.80
			Transfer	28.74 (1.90)	21.34–29.77

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