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Treatment of a Depressive Disorder Patient with EEG-Driven Photic Stimulation

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This study examined the effects of electroencephalographic- (EEG-) driven photic stimulation on a case of depressive disorder, as measured by a psychometric test of mood states, EEG parameters, and several autonomic indices. The EEG-driven photic stimulation enhances the alpha rhythm of brain waves using photic signals, the brightness of which is modulated by a subject's own alpha rhythm. The patient was a 37-year-old businessman, who was treated for depression with medication during the 13 months prior to his first visit to our hospital. He underwent two sets of inpatient treatment sessions, comprising first 16 and then 18 treatment sessions. The treatments brought about the following changes: an improvement in general mood state, alpha rhythm increase, cardiac parasympathetic suppression, and increased skin conductance level. In addition, significant correlations between alpha rhythm increase and cardiac parasympathetic suppression or cardiac sympathetic predominance were observed with each inpatient treatment. Significant correlations between alpha rhythm increase, cardiac parasympathetic suppression, or cardiac sympathetic predominance and the improvement of general mood state were also observed. Thus, from these observations, it was concluded that the alpha enhancement induced by EEG-driven photic stimulation produced

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an improvement in the patient's depressive symptomatology connected with cardiac parasympathetic suppression and sympathetic predominance.

Descriptor Key Words: EEG-driven photic stimulation; alpha rhythm; depression; mood state; autonomic nervous system.

Based on the long-believed hypothesis that apprehension, anxiety, arousal, or tension is related to alpha suppression (Berger, 1969; Bond, James, & Lader, 1974), alpha biofeedback training has been applied to subjects suffering from anxiety, and some successful results have been obtained (Hardt & Kamiya, 1978; Hare, Timmons, Roberts, & Burman, 1982; Sasaki et al., 1988).

Photic driving is another method of enhancing alpha and other brain rhythms, and Yasushi, Saito, and Chijiiwa (1992) devised an "EEG photic feedback" system for clinical application. This system modulates the frequency and amplitude of sine-wave-modulated-light based on the subject's on-going alpha rhythm, which causes effective alpha enhancement while maintaining natural frequency and amplitude variations. As a result of the application of photic stimulation to various neurotic, depressive, and psychosomatic disorders, Chijiiwa et al. (1992) reported that it was especially effective for patients with depressive disorders. Kumano et al. (1993) presented a case report in which a patient with depressive neurosis was treated by photic stimulation and recovered from his illness. However, the mechanism of effectiveness has not yet been sufficiently elucidated.

The aim of the present case study was to clarify the mechanism of effectiveness of photic stimulation with depressive disorder through precise investigation of EEG parameters, several autonomic indices, a psychometric measure of mood state, and their interrelations. Because there was a possible confusion of this method and biofeedback due to prior use of the term "photic feedback," we use the alternative term "EEG-driven photic stimulation" in this study.

METHOD

Subject

The patient was a 37-year-old businessman whose complaints included "attacks" of anxiety and depression, low energy, general malaise, dizziness, pains in the neck, shoulder, and back, and insomnia. He developed a general malaise, shoulder stiffness, shortness of breath, and

paresthesia of legs and arms 3 months after his marriage and 13 months before his first visit to our hospital. He visited a psychosomatic clinic, where he was diagnosed as being in a depressed state, and was treated using antidepressive medication and psychotherapy. He took 5 weeks leave from work 5 months later, and most of his symptoms abated. However, 4 months after his return to work he relapsed, reporting increased symptoms of insomnia, dizziness, trembling, light-headedness, low energy, and difficulty performing work. He took sick leave again and was referred to our hospital for the EEG-driven photic stimulation treatment. According to *The Maudsley Personality Inventory* (MPI; MPI study group, 1969), he was introverted and very neurotic (Extroversion = 16, Neuroticism = 46, Lie = 1). He scored 39 on *The Beck Depression Inventory* (Freeman, 1989), which was compatible with being in a severe depressive state. He met *DSM-III-R* criteria for the diagnosis of "dysthymia" except for criterion A, and was therefore diagnosed as having a "mood disorder not otherwise specified."

The patient underwent inpatient treatment composed of 2 baseline and 16 treatment sessions lasting 21 days, and recovered from his depressive state. However, he relapsed 2 months later, shortly after returning to work. He reported difficulty concentrating, a general malaise, anxiety, depressive moods, and headaches. He was readmitted to our hospital 6 months after his initial discharge and underwent treatment consisting of 2 baseline and 18 treatment sessions lasting 19 days, and eventually recovered. He continued receiving psychotherapy and medication for some time after his discharge. The medication had been used before referral to our hospital, and consisted of tricyclic and tetracyclic antidepressants, sulpiride, and benzodiazepines. This medication was not modified immediately before or during each inpatient treatment period.

Apparatus

The EEG-driven photic stimulation system used was developed by Yasushi et al. (1992), and its block diagram is presented in Figure 1. The participant's EEG was filtered to pass alpha, and then this sinusoidal processed signal was used to drive a photic stimulator at the subject's own alpha frequency. This would be very much like positive-feedback in the engineering sense of an analog, positive feedback amplifier, and might be expected to enhance alpha as in any positive feedback loop. Occipital brain waves were monitored from electrode O_z of the 10-20 EEG system, with A_1 as a reference electrode and the forehead electrode as the ground electrode. The bandpass filter consisted of a switched capacitor filter with a Q of ten.

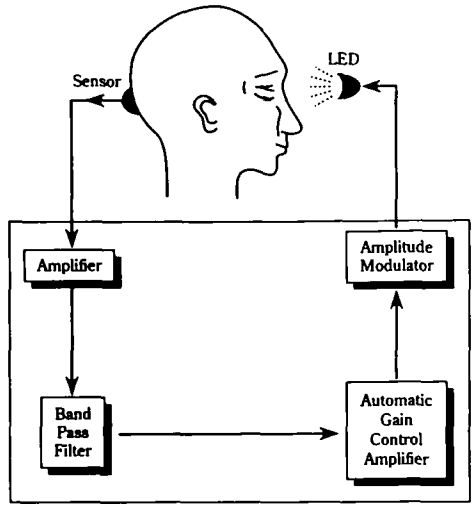

Fig. 1. The block diagram of EEG-driven photic stimulation. LED, light-emitting diodes.

Thus, for example, an output signal at 9.5 or 10.5 Hz was attenuated by 3 db when the center frequency of the filter was 10 Hz.

The input brain wave signal converted from analog to digital can be regarded as a single sine wave, varying in amplitude and frequency. The instantaneous frequency and amplitude were determined every 20 milliseconds before passing through the bandpass filter, by the complex demodulation method (Walter, 1968), which is less susceptible to the nonstationary nature of EEG data than Fast Fourier Transformation (FFT) (Papp & Ktonas, 1977). The amplitude was averaged over 80 milliseconds to be used in the automatic gain control (AGC) circuit in which input brain wave signals were divided by this average amplitude. The time constant of the AGC circuit was approximately .3 second to pass amplitude variation components with a period of one to several seconds, called waxing and waning, and suppress slow and large amplitude variation components (.1 hertz or less). The AGC circuit was necessary to meet the ringing problem and was also effective in uncovering alpha components when alpha waves were particularly small. The output, converted from digital to analog, was offset by a constant voltage, and the resultant signal applied directly to a voltage-cur-

rent converter to drive the red-light-emitting diodes. Sixteen photodiodes provided light stimulus of 3 to 10 lux and were located 5 to 10 centimeters in front of the participant's closed eyes. The participant looked toward the light without focusing on any particular point.

Besides the utilization in the AGC circuit, the instantaneous amplitude and frequency determined by the complex demodulation method were sampled every second, and the resultant data used for calculating the center frequency of the bandpass filter. The data sampled every second were also simultaneously recorded by a personal computer (EPSON PC386NOTE-A, Tokyo, Japan) for the purpose of off-line analysis.

The system control microcomputer controlled the EEG-driven photic stimulation system. The microcomputer had two modes, a rest mode and a stimulation mode. The functions of these modes were as follows: (1) In the rest mode, the microcomputer sampled the EEG frequency and amplitude when stimulation was not applied. The mean of the instantaneous frequencies weighted by the square of corresponding instantaneous amplitude ($FRQM^2$), in the last 3 minutes of the rest mode, was used to determine the center frequency in the stimulation mode. (2) In the stimulation mode, the center frequency was fixed, and EEG-driven photic stimulation was performed.

A microprocessor contained in the EEG-driven photic stimulation system also measured skin conductance level (SCL) and plethysmogram (PLE). SCL was detected with two 1.44 cm^2 gold electrodes attached to volar surfaces of medial phalanges of the subject's left index and middle fingers. PLE was detected by the back-scattered method with a light-emitting diode, and the phototransducer was attached to the volar surface of the distal phalanx of the participant's left index finger (for the first set of treatment sessions) or to his right earlobe (for the second set of treatment sessions). The microprocessor sampled the SCL and PLE signals at a frequency of 100 Hz, and the value of SCL was recorded once per second by the same personal computer used for EEG recording. The value of PLE was differentiated and the temporal positions of peaks detected. Electrocardiographic (ECG) R-R interval (RRI) was then estimated as the interval between two consecutive peaks of PLE and stored in a buffer until the next peak was detected and the new RRI determined. RRI was recorded five times per second in order to calculate the power spectral densities of the RRI variability.

$$^2\mathrm{FRQM} = \sum_{n=1}^{180} (\mathrm{FRQ}(n) \times [\mathrm{AMP}(n)]^2) / \sum_{n=1}^{180} [\mathrm{AMP}(n)]^2$$

where FRQ(n) is the instantaneous frequency and AMP(n) is the instantaneous amplitude.

Procedure

Treatment Schedule

Baseline and treatment sessions were performed in an air-conditioned (24-27°C for the first set; 15-22°C for the second set), sound-attenuated, and dimly lit room at 10 a.m., 2 p.m., or both on weekdays. After the electrodes were attached, the participant reclined on a comfortable couch at about 30 degrees to the horizontal. Baseline sessions consisted of 9 minutes of rest, and treatment sessions consisted of 9 minutes of rest and a 15-minute photic stimulation period.

Psychological Measures

For psychological assessment, *The Mood Inventory* (MOOD; Sakano et al., 1994) was administered at the beginning of each session starting at 10 a.m. in the first admission and soon after waking up for every hospital day including weekends in the second admission. The 40-item MOOD measures five mood states, including tension and excitement, lack of refreshing mood, fatigue, depressed mood, and anxious mood, each of which is scored from 8 to 32 points. The higher the score, the worse the respective mood state. Because there were noticeably large intercorrelations among five subscales of the MOOD (first admission: Spearman correlation coefficients (r_s) = .729–.958; second admission: r_s = .782–.946), we decided to use the total score in this study.

Physiological Measures

For physiological assessment, EEG amplitude (AMP), the standard deviation of EEG frequency weighted by the square of AMP (FRQSD[3]), SCL, the high-frequency components of the power spectral densities of RRI variability (HF), and the ratio between the low-frequency and high-frequency components (LF:HF ratio) were utilized. AMP, EEG frequency, SCL, and RRI were recorded during the last 6 minutes of the rest period and throughout the 15-minute photic stimulation period.

FRQSD was calculated every two minutes after the treatment. FRQSD decreased as the variability of frequency decreased, the amplitude

$$^{3}\mathrm{FRQSD} = \left(\sum_{n=1}^{120} ([\mathrm{FRQ}(n) - \mathrm{FRQM}]^2 \times [\mathrm{AMP}(n)]^2 / \sum_{n=1}^{120} [\mathrm{AMP}(n)]^2 \right)^{1/2}.$$

close to FRQM increased, or both, and thus its decrease indicated not only an increase in alpha amplitude but also convergence of alpha frequency to a certain frequency level. When EEG-driven photic stimulation effectively enhanced alpha rhythm, the amplitude close to the center frequency of the bandpass filter (FRQM in the rest mode) increased more because of the steep and narrow characteristics of the filter, and FRQSD may subsequently decrease. The correlations between FRQSD and AMP were highly significant (first admission: $r_s = -.820$, $p = .0001$; second admission: $r_s = -.830$, $p = .0001$) in this study. FRQSD might be the better indicator of alpha enhancement than AMP because the instantaneous frequency and amplitude are determined before passing through the bandpass filter, and thus, AMP is the nonweighted mean of the amplitude of not only alpha rhythm but also other brain rhythms. Kamei, Yasushi, Kumano, Suematsu, and Masumura (1993) indicated that five out of six participants showed a decrease of FRQSD (not an increase of AMP) as a result of EEG-driven photic stimulation intervention. Thus, we used FRQSD as well as AMP in the present study.

HF and LF:HF ratio were calculated by FFT every two minutes after the treatment. Power spectral components at .04-.15 Hz of RRI variability were defined as low-frequency components (LF), while those at .16-.50 Hz were defined as HF. The magnitude of LF and HF were presented in effective mean amplitude (square of power). HF and LF:HF ratio were used for estimating cardiac parasympathetic activity (Hayano et al., 1991) and cardiac sympatho-vagal balance (Pagani et al., 1986), respectively.

Data Reduction and Analysis

Each of AMP, FRQSD, HF, LF:HF ratio, and SCL was averaged over the 6-minute rest period and over the first 6 minutes of the photic stimulation period, and plotted against the session number. Because the participant often fell asleep in the late phase of the photic stimulation period, the data of the first half were used in this study. The total score of the MOOD was also plotted against the session number. Whether or not the time series of the physiological parameters in the rest period and that of the MOOD contained any trends were analyzed by the C statistic with a significant level at .05 (one-tailed). The C statistic is a simple method of time-series analysis that can be used on small data sets to evaluate the effects of treatment interventions (Tryon, 1982).

Next, Spearman correlations between the value of 6 minutes rest of AMP or FRQSD and that of HF, LF/HF, or SCL were calculated in each inpatient treatment to investigate the relationship between alpha activity

and autonomic nervous system activity. Spearman correlations between the MOOD and the value of 6 minutes rest of AMP, FRQSD, HF, LF/HF, or SCL were also calculated in each admission to investigate the relationship between alpha activity or autonomic nervous system activity and the general mood state.

Correlational analysis and graphical presentation were done by use of the Statistical Analysis System (SAS Institute Japan, Tokyo, Japan) CORR and GPLOT procedures, and the C statistic was done by use of Microsoft Excel (Microsoft Corporation, Tokyo, Japan).

RESULTS

Intrasession Changes and Intersession Trends

Intrasession changes of FRQSD, AMP, HF, LF/HF, and SCL are presented in Figure 2, with solid and open circles denoting the values at rest and those during stimulation respectively. Sessions 1 through 18 corresponded to the first admission, and sessions 20 through 39 corresponded to the second admission. Out of 33 sessions available for data analyses, FRQSD increased in 26 and AMP decreased in 24 sessions. HF increased in 23 sessions, whereas LF/HF decreased in 20 sessions, and SCL decreased in 21 sessions.

Intersession changes of FRQSD, AMP, HF, LF/HF, SCL, and the MOOD are also shown in Figure 2. The C statistic revealed the following trends of these parameters: FRQSD decreased in both admissions (first admission: $C = .4046$, $n = 18$, $p < .05$; second admission: $C = .4951$, $n = 19$, $p < .01$), AMP increased in the second admission ($C = .4760$, $n = 19$, $p < .05$), HF decreased in the second admission ($C = .3680$, $n = 19$, $p < .05$), SCL increased in the second admission ($C = .7788$, $n = 19$, $p < .01$), and the MOOD score decreased in both admissions (first admission: $C = .8831$, $n = 10$, $p < .01$; second admission: $C = .8333$, $n = 11$, $p < .01$). Therefore, there were reciprocal tendencies between short-term and long-term changes for the physiological variables.

Correlations Between AMP or FRQSD and HF, LF/HF, or SCL over Each Inpatient Treatment

There was a significant negative correlation over sessions between AMP and HF ($r_s = -.567$, $p = .014$), a positive correlation between FRQSD and HF ($r_s = .556$, $p = .017$), and a trend for a negative correlation be-

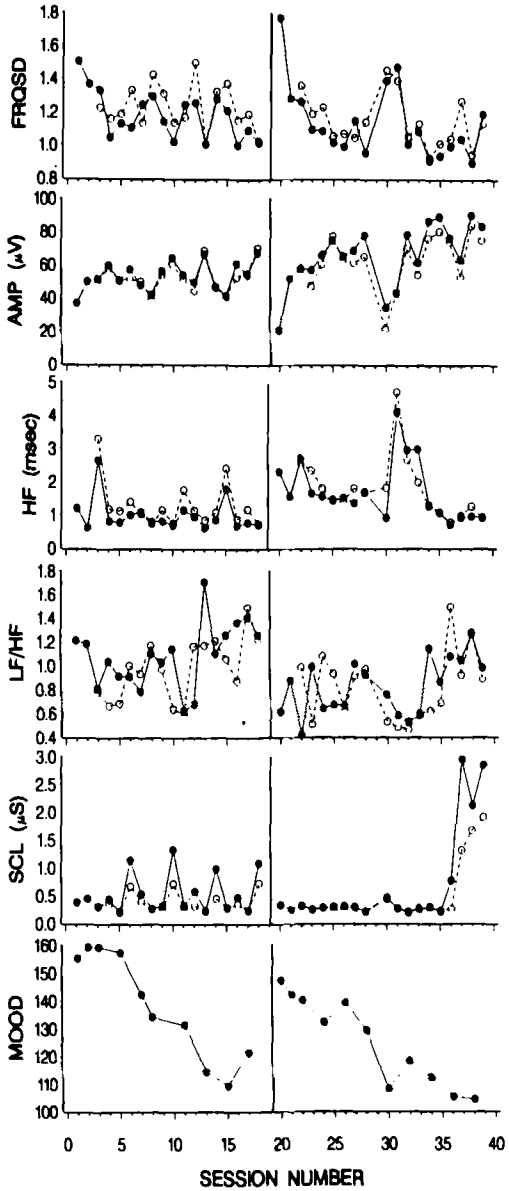

Fig. 2. Changes of the standard deviation of EEG frequency weighted by the square of the EEG amplitude (FRQSD), the EEG amplitude (AMP), the high-frequency components of the power spectral densities of ECG R-R interval variability (HF), the ratio between the low-frequency and high-frequency components (LF:HF ratio), the skin conductance level (SCL), and the total score of *The Mood Inventory* (MOOD). Solid circles denote the value at rest, and open circles denote the value during photic stimulation.

tween FRQSD and LF/HF (r_s = –.412, p = .090) in the first admission. There was a significant positive correlation between AMP and LF/HF (r_s = .467, p = .044) and a negative correlation between FRQSD and LF/HF (r_s = –.463, p = .046) in the second admission. On the other hand, there was no significant correlation between AMP or FRQSD and SCL during either admission. Therefore, if alpha rhythm increased, cardiac parasympathetic activity decreased and cardiac sympathetic predominance increased.

Correlations Between the MOOD and AMP, FRQSD, HF, LF/HF, or SCL over Each Inpatient Treatment

There was a significant positive correlation over sessions between FRQSD and the MOOD (r_s = .657, p = .039) in the first admission, a negative correlation between AMP and the MOOD (r_s = –.664, p = .026) and a trend for a positive correlation between FRQSD and the MOOD (r_s = .591, p = .056) in the second admission. There was a significant positive correlation between HF and the MOOD (r_s = .655, p = .029) and a negative correlation between LF/HF and the MOOD (r_s = –.645, p = .032) in the second admission. On the other hand, there was no significant correlation between SCL and the MOOD in either admission. Therefore, if alpha rhythm increased, cardiac parasympathetic activity decreased, or cardiac sympathetic predominance increased, the general mood state improved.

DISCUSSION

First, it should be mentioned that the control for medication and other nonspecific factors was not sufficient in this study. Because the medication had been used before referral to our hospital and was not modified immediately before or during each inpatient treatment, it can be claimed that the medication did not cause the effects exclusively. Also, because the patient's mood did not change during the baseline periods lasting 5 or 4 days, respectively, in each admission, it can be claimed that the inpatient treatment alone was not enough for the amelioration of his symptoms. However, the combined effects of EEG-driven photic stimulation, medication, and inpatient treatment were likely present. Thus, participants free of medication, inpatient treatment, or both should be included in future studies.

Because alpha rhythm increment, cardiac parasympathetic suppression, and cardiac sympathetic predominance correlated with the improvement of a general mood state over sessions, the intrasession changes of alpha and autonomic nervous system activities in the opposite direction may not represent the treatment effects of EEG-driven photic stimulation. If that is the case, those intrasession changes could be explained by one of the oldest EEG findings: Alpha activity decreases in amplitude and frequency as a participant becomes drowsy and approaches sleep (Adrian & Mathews, 1934; Malmo, 1959). Parasympathetic activation and sympathetic suppression would also follow this change of arousal.

While the generalized alpha increment of occipital lobes correlated with improved affect in the present study, Tomarken et al. (1992) suggested that relative left anterior activation (i.e., alpha suppression) at rest is linked to increased positive affect and decreased negative affect. On the other hand, Moss, Davidson, and Saron (1985) also found that Japanese participants exhibited less left hemisphere activation than Westerners. Therefore, it would be very interesting to know what kind of activation asymmetry Japanese depressive patients have and how it is changed by the EEG-driven photic stimulation treatment. It should also be clarified whether or not the results contradict the present conclusion. However, a replication of the present study with larger numbers of depressive patients will be needed first before advancing such studies.

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