International Journal of Psychophysiology, 6 (1988) 185–194
Elsevier

PSP 00200

EEG Biofeedback and relaxation training in the control of epileptic seizures

Carmen A. Tozzo, Lloyd F. Elfner and Jack G. May, Jr.

Department of Psychology, Florida State University, Tallahassee, FL 32306 (U.S.A.)

(Accepted 9 February 1988)

Key words: Electroencephalogram; Epilepsy; Relaxation; Biofeedback

Research utilizing sensorimotor rhythm (SMR) biofeedback with epileptics suggests that it is useful in decreasing seizures. Subjects were 6 young adults with a diagnosis of epilepsy of at least two years who had been unable to control their seizures with different regimens of anticonvulsant medications. Subjects ranged from severely mentally handicapped to above average functioning. Seizure type, frequency, and duration were recorded by subjects and caretakers. Measures of operant learning were percent time in SMR. The experiment utilized a single subject multiple baseline design which consisted of 6 phases: baseline one, relaxation training; baseline two, biofeedback training one; baseline three, biofeedback treatment two and follow-up. The results of this study are in agreement with other studies using SMR biofeedback. All subjects were able to significantly increase percent time in SMR. Five out of the 6 subjects demonstrated decreases in seizure frequency during the treatment phase. Two of the 6 subjects benefited from relaxation training. Four subjects demonstrated significant negative correlations between percent SMR and seizure rates. Consistent with other studies utilizing multiple baseline designs, a majority of the subjects did not follow the design of the study.

INTRODUCTION

Sterman et al. (1969) demonstrated that animals who had been reinforced to produce sensorimotor rhythm (SMR) decreased seizure latency or showed seizure absence. Encouraged by these findings, Sterman and Friar (1972) attempted a study with a 23-year-old epileptic who displayed about two seizures a month. After 12 sessions, there was a significant increase in SMR activity and a shorter latency to sleep onset. After 6 months, seizures decreased to an average of two seizures every 3 months.

One year later, this treatment was reported to be successful with a 7-year-old epileptic displaying both grand mal and petit mal seizure patterns (Sterman, 1973; Sterman et al., 1974). Dilantin and phenobarbitol had not controlled seizures since the child maintained an average of 4 epileptic episodes per week. Treatment consisted of reinforcing production of 12–14 Hz activity with illumination of 10 colored lights in ascending order and the onset of scenes from a slide projector. After two months of three 20–40 min sessions per week, there were significant decreases in grand and petit mal seizure compared with baseline. While the average seizure rate during the study was one to two seizures per week, the subject remained seizure-free for a 5-month period. When treatment was discontinued for 9 weeks, there was a significant increase in the amount of reported seizures. Although the authors did not use adequate control periods, the subject was followed for one year and maintained a decreased seizure rate throughout with periodic booster sessions.

Correspondence: L.F. Elfner, Department of Psychology, Florida State University, Tallahassee, FL 32306, U.S.A.

A second researcher two utilized SMR feedback with epilepsy after Sterman's initial results was Lubar (Seifert and Lubar, 1975). Three male and 3 female adolescents whose seizures were not controlled by near-toxic levels of anticonvulsants were trained to produce 12–14 Hz activity. After 3 months of treatment, 5 of the 6 subjects demonstrated significant decreases in reported seizure activity, although Fourier analysis of EEG activity did not indicate changes in percent SMR.

Sterman and his colleagues (Sterman and McDonald, 1978) first attempted a controlled experiment using 8 epileptics with a history of poorly controlled seizures. The basic design was a double ABA crossover. Each subject was first reinforced for increasing the incidence of one frequency band and decreasing the frequency of a second frequency band. After 3 months, the reinforcement schedule was reversed, that is, subjects were rewarded for increasing the incidence of the second frequency band and decreasing the incidence of the first frequency band. After another 3 months, the original reinforcement schedule was reinstated. Three frequency bands (6–9 Hz, 12–15 Hz, and 18–23 Hz) were used in different combinations with 8 subjects. Three subjects demonstrated significant decreases in seizure rates only when they were reinforced for the production of 12–15 Hz (SMR) activity in the absence of 6–9 Hz activity. Another 3 subjects exhibited a decrease in seizure rates after initial training to increase high frequency rhythms, and maintained a decrease in seizure rates, despite reversal in the reinforcement schedule. The experimenters hypothesized that SMR biofeedback training can produce an EEG normalization effect in some subjects which is resistant to epileptiform activity.

Finley (1976, 1977) similarly introduced noncontingent feedback and found that as percent SMR activity decreased, seizure severity (but not frequency) significantly increased.

A third research group (Lubar et al., 1981) utilized a blind multiple baseline design with 5 subjects. Three of the 5 subjects showed significant changes in overall seizure frequency with contingent SMR biofeedback. Two subjects demonstrated significant changes in one type of seizure frequency with SMR biofeedback. Whitsett et al. (1982) used a similar experimental design, an ABA crossover, but added double-blind controls. Eight epileptics having refractory seizures, ranging in age from 11 to 50 years, participated in the study. During an initial baseline period, all subjects received non-contingent feedback. Group 1 subjects ($n = 3$) then received reinforcement for suppression of 3–8 Hz (epileptiform) activity. Group 2 ($n = 2$) received reinforcement for production of 12–15 Hz (SMR) activity. Group 3 ($n = 3$) subjects were reinforced for suppression of 3–8 Hz activity plus production of 11–19 Hz activity. During the reversal phase, all groups were reinforced for production of 3–8 Hz activity. During the final phase, all subjects returned to their original reinforcement schedule. Five of the 8 subjects reported decreased seizure frequency during the first and second contingent SMR phases and 5 subjects demonstrated setbacks when epileptiform activity was reinforced. It should be noted that Lubar et al.'s (1981) experiment used more subjects who were mentally handicapped.

Although results of some experimental studies using SMR biofeedback with epileptics appear promising, there are studies showing equivocal results. Kaplan (1975) employed 12–14 Hz activity biofeedback in two human epileptics and was unable to change either clinical seizure activity or EEG spectral activity. However, Kaplan did show that biofeedback training of 6–12 Hz activity was effective in reducing seizure activity in two of three subjects in a second study. However, the subjects above did not demonstrate change in EEG activity and Kaplan suggested that the major factor involved in the seizure rate change was the relaxation produced by the biofeedback procedure. Gastaut (1975) who discovered the SMR activity in the Rolandic area of the cortex also expressed concern over the interpretation of the usage of SMR biofeedback in control epilepsy, since the experimental studies have not provided clear statistical evidence of the relationship between biofeedback training in SMR activity and the incidence of eleptiform activity. One problem with the studies is failure to utilize more powerful reinforcers with some subjects. Children do not always find blinking lights reinforcing. Mentally handicapped subjects need more appropriate rein-

forcers with seizure frequency prevention. Lubar et al. (1981) reported that a thirteen-year-old mentally handicapped subject was unable to completely achieve criteria levels for target EEG frequencies. One reason for this may be the complicated nature of simultaneously increasing a 12–14 Hz frequency band and decreasing a 3–8 Hz frequency band in order to receive reinforcement. Another reason is that more powerful rewards, such as an automatically powered electric train or cartoons may be necessary to produce appropriate results with this population.

Individuals exhibiting chronic seizures, despite consistent use of anticonvulsant medication may be handicapped in their ability to learn new tasks or complete work in a classroom or vocational settings. An alternative treatment which appears to hold promise is biofeedback of sensorimotor rhythms. Studies utilizing SMR biofeedback with epileptics appear to indicate that it decreases the frequency of seizures. Better controlled studies are needed to establish the utility and cost-effectiveness of this treatment.

The purpose of this study was to provide evidence for the efficacy of SMR biofeedback in epileptics. Subjects ranged from severely mentally handicapped to above average intellectual functioning, providing a good representation of social and skills functioning. A single subject multiple baseline was used. It was anticipated that seizure rates would significantly decrease by the end of the study, when compared to baseline. Secondly, it was anticipated that subjects, regardless of cognitive functioning could learn to change EEG frequency through operant conditioning. Finally seizure rates were anticipated to be inversely related to percent SMR activity. Formal hypothesis are not appropriate for this individual subject design. Rather, the functional relationships will be presented for the reader to assess the efficacy of SMR biofeedback treatment for seizures in both mentally handicapped and average intellectually functioning subjects.

MATERIALS AND METHODS

Subjects

The subjects for this study were 6 young adults diagnosed as having an epileptic disorder. Criteria for selection were failure to control seizures with standard anticonvulsant medication for a period of at least one year. One subject was severely mentally handicapped, one subject was mildly mentally handicapped, one was functioning in the borderline range, two were within the average range of intelligence and one was functioning in the above average range of intelligence (see Table I).

Apparatus

All EEG recordings were made using silver electrodes placed over international EEG recording sites C_3–T_3 or C_4–T_4. The sites were 10% and 30% toward the vertex, with reference to the ear contralateral to handedness. A ground electrode was placed on the earlobe. Standard Grass EC2 EEG cream was used to place electrodes at each site. Electrode sites were prepared with alcohol and Omni prep (Weaver and Co.). Electrode placement was checked at the beginning of each session by a Grass Model EZM3 Electrode impedance meter. The impedance was maintained below 5 kΩ.

The electrodes were connected to an Autogen 120 Encephalograph Analyzer interfaced with an SMR Inhibit Instrument. The raw EEG signal was split several times through 3 circuits. The first circuit allowed for detection of 12–15 Hz activity which lies between 1 and 6 μV. A second circuit detected 4–8 Hz frequency band which is characteristic of epileptiform activity. When the amplitude of this bandpass exceeded 30 mV, it blocked SMR biofeedback. This inhibit circuit made it impossible for a subject to receive reinforcement if he was producing slow wave epileptiform frequencies. A third circuit detected muscle activity associated with tension and gross body movements, and also inhibited SMR feedback when muscle activity was above a preset threshold.

Design

A multiple baseline single subject design was utilized. Subjects were contacted 3 weeks before

TABLE I

Subject, diagnosis, and seizure frequency

Subjects	*1*	*2*	*3*	*4*	*5*	*6*
Sex	M	F	M	M	F	M
I.Q.	average	mildly H.M.	high average	borderline	average	severely M.H.
Age	27	28	27	29	19	18
Diagnosis and seizure type	Lennox-Gastaut atonic, absence	Lennox-Gastaut, atonic	atonic	Lennox-Gastaut atonic, absence	atonic, clinic-tonic	Lennox-Gastaut, absence
Duration	20 years	19 years	20 years	22 years	2 years	16 years
Medication	Dilantin (250 mg qid) Tegretol (200 mg qid) Valproic Acid Midion Ritalin	Depakane (250 mg qid) Secontin (250 mg qid) Mebarol (250 mg qid) Dimax (250 mg qid)	Tegretol (200 mg qid) Depakane (250 mg qid)	Tegretol (200 mg qid) Depakote (250 mg qid) Tranxene (7.5 mg qid)	Tegretol (200 mg qid) Phenobarbitol (50 mg qid)	Depakane (250 mg qid) Phenobarbitol (50 mg qid)
Seizure frequency						
Before treatment	11 absences and 3 atonic/week	1 atonic/week	2.5 atonic/week	1 absence and 5 atonic/week	3 atonic and 4 clonic-tonic/week	50 abscence/week
With relaxation training	10 absences and 3 atonic/week	1 atonic/week	0.5 atonic/week	1 absence and 1 atonic/week	3 atonic and 4 clonic-tonic/week	–
During treatment	14 absences and 1 atonic/week	0.5 atonic/week	0.5 atonic/week	1 absence and 0.5 atonic/week	0.5 atonic/week	10 absences/week
Follow-up	5 absences and 1 atonic/week	1 atonic/week	1.5 atonic/week	1 absence and 2 atonic/week	0.25 atonic/week	8 absences/week

the training period and the electrodes from the biofeedback unit were attached, but no feedback was given. A 5-week training period ensued which consisted of two 30-min sessions of contingent reinforcement of SMR. A two-week period followed during which subjects were not given any feedback. During the final phase of the study, subjects were given contingent auditory biofeedback training for 3 weeks.

Procedure

After subjects were identified by neurologists, caretakers were contacted and a release form was obtained. A structured interview (Mostovsky, personal communication) was conducted with a parent or caretaker. Subjects and caretakers were instructed on keeping daily records of seizures and medications taken. Subjects were given an initial cognitive battery to assess intellectual functioning. During the first several sessions, subjects were escorted to a well-lit room in either a sheltered workshop or a hospital and taught relaxation techniques. Several subjects who expressed an interest were given relaxation tapes and instructed to practice twice a day. During the following two weeks, subjects were given relaxation instructions and hooked up to the feedback monitor, without receiving reinforcement. Frequency, amplitude, and percent SMR readings were obtained at 3-min intervals. The treatment phase which followed consisted of auditory and visual biofeedback. If subjects produced 12–18 Hz activity, without epileptiform activity, a pleasant tone was heard. If subjects produced epileptiform activity, a red light and unpleasant tone was heard. A third tone (white noise) signalled muscle activity. Subjects were told to keep the red light off and to produce the pleasant sound. They were encouraged to use different techniques in doing this. Each session included a 3-min baseline with no feedback and four 5-min trials with 2-min rest periods in between. Finally, a 3-min baseline with no feedback ended the session.

Subjects were contacted 4 and 8 weeks after the last treatment session for final feedback sessions. If seizure rates were returning to baseline levels, additional feedback sessions were offered.

RESULTS

Six patients aged 18–29 were included in this study during a one-year period. Each subject's data will be presented individually, since each subject was his/her own control. Dependent *t*-tests were conducted to test for seizure differences between pretreatment, baseline, training and post-treatment means. Dependent *t*-tests were also conducted to test for differences in percent time in SMR between baseline and training means. A χ^2-square analysis was performed between training and non-training periods. Correlation coefficients (Pearson product-moment) were computed to determine the relationship between EEG data and seizure frequency. All subjects demonstrated significant changes ($P < 0.05$) in percent time SMR with biofeedback. Five of 6 subjects demonstrated significant decreases in seizure rates between pre- and post-treatment phases.

Subject 1

Subject 1 (see closed bars in Fig. 1) was a 27-year-old male of average intelligence. He had a diagnosis of Lennox-Gastaut syndrome since the age of seven. At the beginning of this study, he was experiencing an average of three atonic and two generalized non-convulsive (absence) seizures per day. He wore a helmet to prevent injury and had not been to work for several months. Subject

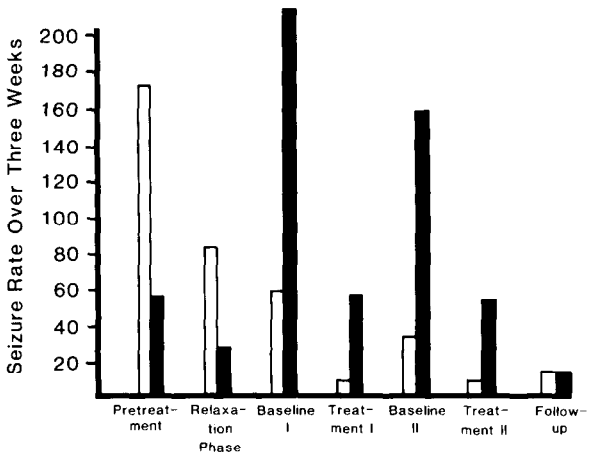

Fig. 1. Seizure rate over five phases. Dark bars, Subject 1; open bars, Subject 6.

1 was on a long list of medications including dilantin, tegretol, valproic acid, midion, ritaline and mebaral. EEG data indicated slow moderate atrophy and left focal activity.

Subject 1 was cooperative, but was anxious about the biofeedback session. He was given a relaxation tape two weeks prior to the beginning of the treatment phase. There was no change in seizure frequency as a result of relation training.

There were significant differences between seizure frequency at baseline and during the first feedback period ($t = 4.79$, $P = 0.001$, df = 9). Subject 1 had an increase in seizures during the second feedback period, but demonstrated significant decreases in seizure frequency at follow-up with booster sessions ($t = 14.4$, $P < 0.001$, df = 90). (See Fig. 1).

The total number of seizures for conditioning periods were tested against non-conditioning periods; there were no significant differences ($\chi^2 = 36.05$, $P = 0.21$, df = 30). Overall, the correlation between seizure frequency and percent time in SMR was not significant ($r = 0.28$, $P = 0.11$). This suggests that seizure frequency, which initially decreased with biofeedback, did not correlate or coincide with changes in percent SMR.

After the end of the treatment, this subject was able to stop using a helmet and returned to work. He continued to receive booster sessions for 3 months after treatment ended on a biweekly basis.

Subject 2

Subject 2 (see open bars in Fig. 2) was a 28-year-old white female with a diagnosis of Lennox-Gastaut syndrome since the age of 9 years. She was functioning in the mild mentally handicapped range of intelligence. Before treatment, subject 2 had about 10 atonic seizures per month, despite high doses of anticonvulsant medication: depakane (250 mg), secontin (250 mg), dilantin (400 mg), mebarol (250 mg) and dimax (250 mg). Seizures tended to cluster around menstruation.

Subject 2 did not demonstrate any significant decrease in seizures during the initial treatment phase ($t = 1.5$, $P = 0.17$, df = 9), but did exhibit a significant decrease in seizures during the second treatment phase ($t = 3.0$, $P = 0.01$, df = 9). She was able to increase her percent time in SMR,

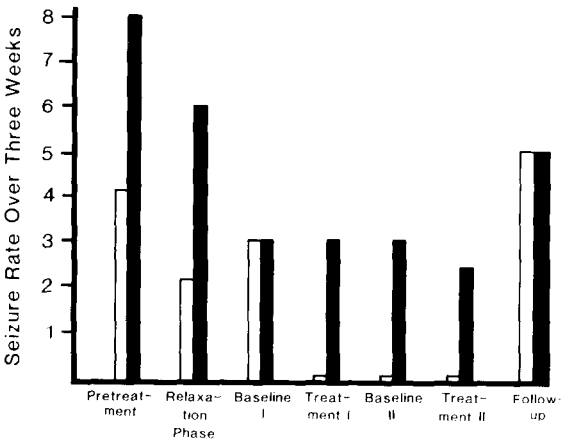

Fig. 2. Seizure rate over five phases. Open bars, Subject 2; dark bars, Subject 3.

both during the first treatment phase ($t = 26.2$, $P < 0.001$, df = 7) (see Fig. 2). If the total number of seizures for biofeedback phases are tested against total number of seizures for non-biofeedback periods, there are no significant differences ($\chi^2 = 3.01$, $P = 0.22$, df = 2). However, percent time in SMR was inversely correlated with seizure rat ($r = 0.37$, $P = 0.05$).

Subject 2 took longer to gain control of SMR. Two months after treatment ended, seizure rates had returned to baseline levels ($t = 1.15$, $P = 0.28$, df = 9).

Subject 3

Subject 3 (see dark bars in Fig. 2) was a 27-year-old white male of above average intelligence with a diagnosed seizure disorder involving atonic episodes since the age of seven.

Subject 3 was living independently, but was impaired in the work setting because of continued seizures (about 9 atonic seizures a month). His medications included tegretol (200 mg) and depakane (250 mg). He was given a relaxation tape, which he was instructed to use twice daily. There were significant decreases in seizures after relaxation training ($t = 2.3$, $P = 0.04$, df = 9), which were maintained throughout the study. Subject 3 was able to learn to control percent SMR, utilize biofeedback very quickly, and demonstrated significant increases in percent SMR ($t = 2.62$, $P = 0.02$,

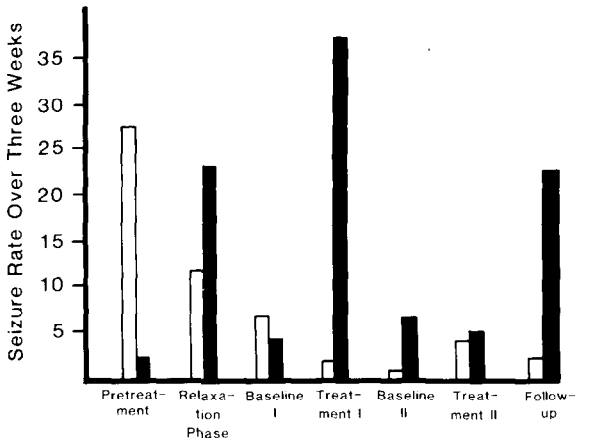

Fig. 3. Seizure rate over five phases. Dark bars, Subject 4; open bars, Subject 5.

df = 14). Further changes in seizure frequency did not occur, however, with biofeedback (see Fig. 3). This may be because this subject was having an average of two seizures per month after the relaxation phase. There was no correlation between percent time in SMR and seizure frequency ($r = 0.14$, $P = 0.25$).

Two months after treatment ended, the subjects' frequency had returned to baseline levels ($t = 1.15$, $P = 0.28$, df = 9).

Subject 4

Subject 4 (see dark bars in Fig. 3) was a 29-year-old white male functioning in the borderline range of intelligence with a diagnosis of Lennox-Gastaut syndrome since the age of seven. At the time of the initial intake, subject 4 was experiencing approximately 3 atonic and 2 generalized non-convulsive episodes per week. Subject 4 lived in a group home and was frequently absent from the work setting because of his seizures. He wore a helmet most of the time. Subject 4 was taking tegretol (200 mg), depakote (250 mg) and tranxene (7.5 mg). He had experience with EEG biofeedback 8 years previously, which was successful in decreasing his seizure frequency.

Subject 4 was given deep muscle relaxation training and appeared to benefit from using a relaxation tape, as evidenced by significant decreases in seizure frequency ($t = 4.3$, $P = 0.002$, df = 9). Seizure frequency remained stable during the initial treatment phase ($t = 1.5$, $P = 0.14$, df = 9), increased during the second baseline phase ($t = 3.75$, $P = 0.005$, df = 9), and decreased significantly from baseline during the second treatment phase ($t = 3.0$, $P = 0.002$, df = 9).

Subject 4 was able to learn to significantly increase percent time in SMR ($t = 3.65$, $P = 0.003$, df = 12; $t = 4.69$, $P = 0.001$, df = 8) during both treatment phases.

If the total number of seizures for conditioning periods is tested against non-conditioning periods, there is a significant difference ($\chi^2 = 16.11$, $P = 0.04$, df = 8). There is also a significant negative correlation between percent SMR and seizure frequency ($r = 0.476$, $P = 0.01$).

Subject 5

Subject 5 (see open bars in Fig. 3) was a 19-year-old Hispanic female of average intelligence with a diagnosed seizure disorder of two years. At the time of the initial interview, the subject was having an average of one atonic and/or clonic/tonic seizure per day. EEG records indicated slow waves intermittent in both temporal regions and focal activity in the left temporal area. Subject 5 was on tegretol (200 mg), phenobarbital (60 mg) and nordil (45 mg). She was depressed and negativistic during the initial sessions, and refused to use a relaxation tape.

Subject 5 displayed a significant decrease in seizures during both treatment phases when compared with baseline ($t = 14.23$, $P < 0.01$, df = 9; $t = 21.6$, $P < 0.01$, df = 9). She was able to maintain this decrease in seizure frequency at an 8-week follow-up.

Subject 5 was able to significantly increase percent time in SMR over baseline during treatment phase one ($t = 2.47$, $P = 0.03$, df = 12), and treatment phase two ($t = 6.04$, $P < 0.01$, df = 8). If the total number of seizures for biofeedback periods are tested against total number of seizures for non-feedback sessions, there is a significant difference ($\chi^2 = 22.2$, $P = 0.004$, df = 8). A negative correlation between percent time in SMR and seizure frequency was demonstrated ($r = 0.46$, $P = 0.01$).

Subject 6

Subject 6 (see open bars in Fig. 1) was an 18-year-old severely mentally handicapped white male with a diagnosis of Lennox-Gastaut syndrome since the age of two. Subject 6 was experiencing at least 20 generalized non-convulsive (absence) episodes per day. He had very little adaptive behaviors and needed assistance in daily living skills. He was on depakane (250 mg) and phenobarbitol (50 mg). Visual and auditory biofeedback were initially coupled with edible reinforcement and verbal reinforcement.

Subject 6 demonstrated significant decreases in seizure frequency during both treatment phases when compared to baseline ($t = 7.1$, $P < 0.01$, df $= 9$; $t = 10.5$, $P < 0.01$, df $= 9$). Subject 6 was able to learn to increase percent time in SMR during both treatment phases ($t = 4.36$, $P = 0.001$, df $= 12$; $t = 3.63$, $P = 0.007$, df $= 8$). If the total number of seizures for biofeedback periods are tested against total number of seizures for non-biofeedback periods, there are significant differences ($\chi^2 = 27.17$, $P = 0.01$, df $= 8$). A significant negative correlation between percent time in SMR and seizure frequency was also evidenced ($r = 0.505$, $P = 0.01$).

Subject 6 was able to maintain a decrease in seizure frequency 8 weeks after treatment ended ($t = 12.2$, $P < 0.01$, df $= 9$). There was evidence of an increase in attention span and improvement in independent self-care at a 6-week follow-up.

DISCUSSION

At the end of the study, 5 of the 6 subjects demonstrated significant reductions in seizure rates. This is consistent with other studies which have demonstrated significant decreases in seizure frequency with SMR biofeedback in a majority of subjects (Sterman and Friar, 1972; Sterman, 1973; Sterman et al., 1974; Siefert and Lubar, 1974; Finley, 1977; Sterman and McDonald, 1978; Lubar et al., 1981; Whitsett et al., 1982).

All of the subjects demonstrated significant increases in percent SMR with training (0.01). In 4 subjects, there was a statistically significant relationship between increases in percent time in SMR and decreases in seizure frequency. All subjects showed gains (significant changes) from baseline to the initial treatment phase. Gains were maintained during the second baseline phase. Similarly, other researchers have reported that only a minority of subjects follow the research design (Sterman and McDonald, 1978; Lubar et al., 1981; Whitsett et al., 1982).

Two of the 5 subjects who were given relaxation training demonstrated significant changes in seizure frequency with training. This is consistent with case history reports that suggest that some epileptics benefit from relaxation training (Johnson and Meyer, 1974; Cabral and Scott, 1976). Also, this interpretation agrees with Kaplan's (1975) suggested explanation.

It should be noted that decreases in seizure frequency were accompanied by decreases in seizure severity. In addition, one subject was able to learn to detect seizure onset and prevent seizure occurrence after treatment. Two subjects were able to return to work at the end of treatment. Overall, SMR biofeedback appears to be effective in decreasing seizure frequency in patients with intractable epilepsy. Some subjects appear to benefit from a combination of relaxation training and biofeedback. Some subjects need additional booster sessions to maintain treatment gains. Adults who are mentally handicapped, as well as those who are of average intellectual functioning learned to increase 12–14 Hz activity and this increase was associated with treatment benefits.

Several comments concerning the results appear warranted. Results are not likely to be due to placebo effects. Other researchers have ruled out the involvement of placebo by increased baseline periods of up to 6 months before SMR training (Wyler et al., 1976; Lubar et al., 1981). While subjects' EEGs were not monitored prior to the introduction of relaxation training in this study, 4 subjects did not exhibit any changes in seizure rates during the initial two weeks of intake and orientation. Two subjects demonstrated decreased seizure rates after the introduction of relaxation training.

Drug levels were not monitored in all subjects. Two subjects who were tested for changes in blood levels did not demonstrate significant changes in

blood serum anticonvulsant levels in the study. It is unlikely that results were due to changes in medication compliance, since 5 of the 6 subjects lived in group homes where medication was dispensed on a consistent schedule.

While positive results were found in the present study, caution must be used in generalizing data to the general population. Other studies have utilized other EEG frequencies and obtained equivalent results. Alpha enhancement (Finley et al., 1975; Cabral and Scott, 1976), feedback of focal spike EEG discharges (Upton and Longmire, 1975; Ellertson et al., 1976), and 18 Hz enhancement (Wyler et al., 1979) have all been reported to significantly change seizure occurrence in epileptics. A possible explanation for these results is what Sterman (Sterman and Friar, 1972; Sterman and McDonald, 1978) and Wyler (Wyler et al., 1976) have hypothesized to be a normalization of EEG activity with biofeedback. Changes in synaptic morphology may be caused by enhancement of SMR or other EEG rhythms. These changes appear to be resistant to reinforcement of spike activity. The thalamocortical network, which is activated with SMR enhancement in the cat (Howe and Sterman, 1973) may play a role in EEG normalization.

There appears to be a marked advantage to the use of SMR biofeedback in the control of intractable epilepsy. Between 20 and 25% of epileptics using anticonvulsant medication continue to have uncontrolled seizures. In addition, high anticonvulsant serum levels have been associated with decreased concentration, decreased short-term memory and increased latency of response in abstract problem solving (Thompson and Trimple, 1983). Other experimenters have reported that subjects who benefited from SMR biofeedback were able to maintain decreased seizure frequency with lowered medication dosages (Lubar and Bahler, 1976).

While SMR biofeedback appears warranted for epileptics who have uncontrolled seizures, many questions remain. Some studies have found beneficial results within 4 weeks of training (Kuhlman and Allison, 1976), while other researchers have proposed that longer treatment periods are needed (Lubar, 1977; Sterman, 1977). The active ingredients of EEG biofeedback are an ongoing question. Cott et al. (1979) demonstrated that reinforcement of 12–14 Hz activity is not necessary for decreases in seizure frequency to occur and that reinforcement for suppression of focal spike discharges is not sufficient, in most subjects, to produce decreases in seizure rates.

This research is promising in demonstrating the efficacy of biofeedback-assisted control of epileptic seizures. Future research needs to focus on the relative efficacy of feedback in different EEG frequencies. Future studies also need to address the issue of cost-effectiveness of shorter treatment phases and look at schedules needed to maintain these gains. It would be wise to follow the cautions proposed by Gastaut (1975) and to consider the total body of literature to interpret the significance of procedures employed in the behavioral investigations of eleptiform activity.

REFERENCES

Cabral, R.S. and Scott, D.F. (1976) EEG features associated with the occurrence of epilepsy after surgery for intracranion aneurysm and acoustic neuroma. *J. Neurol. Neurosurg. Psychiatry*, 40: 97–99.

Cott, A., Pavlovski, R.P. and Black, A.H. (1979) Reducing epileptic seizures through operant conditioning of central nervous system activity procedural variables. *Science*, 4375: 73–75.

Ellertson, B., Born, Kove, H. and Hallgrin, E. (1976) Clinical application of biofeedback training in epilepsy. *Scand. J. Behav. Ther.*, 5: 133–144.

Finley, W.W. (1976) Effects of sham feedback following successful SMR and training in an epileptic: follow-up study. *Biofeedback Self-Regul.*, 1: 227–235.

Finley, W.W. (1977) Operant conditioning of the EEG in two patients with epilepsy; methodologic and clinical consideration. *Pavlov. J. Biol. Sci.*, 12: 93–111.

Finley, W.W., Smith, H.A. and Etherton, M.D. (1975) Reduction of seizures and normalization or the EEG in a severe epileptic following sensorimotor biofeedback training: a preliminary study. *Biol. Psychol.*, 2: 189–203.

Gastaut, H. (1975) Comments on "Biofeedback in epileptics: equivocal relationship of reinforced EEG frequency in seizure reduction". *Epilepsia*, 16: 487–490.

Howe, R.C. and Sterman, M.B. (1973) Somatosensory evoked potentials during working behavior and sleep in the cat. *EEG Clin. Neurophysiol.*, 34: 605–618.

Kaplan, B.J. (1975) Biofeedback in epileptics: equivocal relationship of reinforced EEG frequency to seizure reduction. *Epilepsia*, 16: 477–485.

Kuhlman, W.N. and Allison, T (1976) EEG feedback training in the treatment of epilepsy: some questions and answers. *Pavlov. J. Biol. Sci.*, 12: 112–122.

Lubar, J.F. (1977) EEG biofeedback methodology and the management of epilepsy. *Pavlov. J. Biol. Sci.*, 12: 147–185.

Lubar, J.F. and Bahler, W.N. (1976) Behavioural management of epileptic seizures following EEG biofeedback training of SMR. *Biofeedback Self-Regul.*, 1: 77–104.

Lubar, J.F., Shabsin, H.S., Natelson, S.E., Holder, G.S., Whitsett, S.F., Pamplin, W.E. and Krulikowski, D.I. (1981) EEG operant conditioning in intractable epileptics. *Arch. Neurol.*, 38: 700–704.

Seifert, A.R. and Lubar, J.F. (1975) Reduction of epileptic seizures through EEG biofeedback training. *Biol. Psychol.*, 3: 157–184.

Sterman, M.B. (1977) EEG operant conditioning: experimental and clinical effects. *Pavlov. J. Biol. Sci.*, 12: 63–92.

Sterman, M.B. (1973) Neurologic and clinic studies of sensorimotor EEG biofeedback training: some effects on epilepsy. *Semin. Psychiatry*, 5: 507–524.

Sterman, M.B. and Friar, L. (1972) Suppression of seizures in an epileptic following sensorimotor EEG feedback training. *Electroencephalogr. Clin. Neurophysiol.*, 33: 89.

Sterman, M.B., LoPresti, R.W. and Fairchild, M. (1969) *Electroencephalographic and Behavioral Studies of Monomethylhydrazine Toxicity in the Cat.* Technical report AMRL-TR-69-3 Air Force Systems Command, Wright-Patterson Air Force Base, OH.

Sterman, M.B. and McDonald, L.R. (1978) Effects of central cortical EEG feedback on incidence of poorly controlled seizures. *Epilepsia*, 19: 207–222.

Sterman, M.B., McDonald, L.R. and Stone, R.K. (1974) Biofeedback training of the sensorimotor electroencephalographic rhythm in man: effects on epilepsy. *Epilepsia*, 15: 395.

Thompson, P.J. and Trimple, M.R. (1983) Anticonvulsant serum levels: relationship to impairments of cognitive functioning. *J. Neurol. Neurosurg. Psychiatry*, 46: 227–233.

Upton, A.R. and Longmire, D. (1975) The effects of feedback on focal epileptic discharge in man: a preliminary report. *Can. J. Neurol. Sci.* 2: 153–167.

Whitsett, S.F., Lubar, J.F., Holder, G.S., Pamplin, W.E. and Shabsin, H.S. (1982) A double-blind investigation of the relationship between seizure activity and the sleep EEG following EEG biofeedback training. *Biofeedback Self-Regul.*, 7: 193–209.

Wyler, A.R., Robbins, C.A. and Dodr, L. (1979) EEG operant conditioning for central of epilepsy. *Epilepsia*, 20 (3): 279–286.

Wyler, A.R., Lockard, J.S., Ward, A.A. and Finch, C.A. (1976) Conditioned EEG desynchronization and seizure occurrence in patients. *Electroencephalogr. Clin. Neurophysiol.*, 41: 501–512.