

REDUCTION OF EPILEPTIC SEIZURES THROUGH EEG BIOFEEDBACK TRAINING

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Biofeedback training of the sensorimotor rhythm (SMR) was carried out in three male and three female adolescent epileptics and in two normal controls. The patients represented a cross-section of epilepsies including grand mal, myoclonic, afocal and psychomotor types. Three of the cases were mentally retarded. 12–14 Hz (SMR) activity was detected by a combination of sharp analog filtering and digital processing. The patients were provided with feedback whenever they produced 0.5 sec of 12–14 Hz activity of a specified amplitude. Additional feedback was provided for epileptiform activity slow waves or movement. Furthermore, feedback for SMR production was inhibited by digital logic circuitry when movement, slow waves or spikes were present. Seizure reduction was obtained in five of the six epileptics. Several patients showed increased percentage of SMR when feedback was provided and varying degrees of normalization in their EEG as demonstrated by fast Fourier, crossed power spectral density and coherence analyses.

1. Introduction

During the past decade it has been demonstrated for both humans and animals that it is possible to bring specific frequency bands of the EEG spectrum under behavioral control through operant conditioning (Kamiya, 1962, 1969; Lynch and Paskowitz, 1971; Beatty, 1973). While differential control of human EEG has been demonstrated for several frequency bands, specifically the alpha (8–13 Hz) and theta (4–7 Hz) rhythm, the origin of that control (Lynch and Paskowitz, 1971; Beatty, 1973), and the utility of such control still remains in question. Recent work, however, has shown that altering activity in one frequency range (12–14 Hz) can play an important role in the control of epileptic seizures (Sterman and Friar, 1972). Activity in the 12–14 Hz bandwidth localized over the sensorimotor cortex in both cats and humans has been termed the sensorimotor rhythm (SMR) by Sterman and Wywricka (1967). This activity has been shown to be associated with inhibition of voluntary movement (Roth, Sterman and Clemente, 1967). The sensorimotor rhythm has been operantly

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conditioned by providing food or positive brain stimulation as a reward (Sterman and Wywicka, 1967). Their SMR-trained cats assumed a stereotyped and motionless posture. Non-movement by itself, however, was insufficient to produce increased SMR (Sterman and Wywicka, 1967). Furthermore, these cats showed an overall reduction in time spent sleeping and a decreased number of motor disturbances during sleep and decreased somatic reflexes. More important, Sterman, Lo Presti and Fairchild (1969) demonstrated that SMR-trained cats were resistant to drug-induced convulsions caused by mono-methylhydrazine.

Based upon this work an initial and successful effort to operantly condition SMR in a 23-year-old epileptic female was reported by Sterman and Friar (1972). This patient had grand mal seizures at a rate of two per month. After one month of training a decline in seizure rate was already noted, and one year later the seizure rate was reduced to approximately one seizure every three months. In trying to develop a model to explain this effect, Howe and Sterman (1972) and Harper and Sterman (1972) found in cats that while SMR was occurring cortically there was inhibition of firing of single units in the ventro-basal complex of the thalamus. Based on these findings, a reasonable interpretation is that the occurrence of SMR represents a type of cortical idling associated with inhibition of motor activity; hence increasing this idling through operant conditioning of the SMR could block overt epileptic activity. More recently Sterman, McDonald and Stone (1972) have reported a significant reduction in both abnormal EEG activity as well as in clinical epileptic manifestations in four additional patients. These patients were all refractory epileptics that were not adequately controlled by anticonvulsant drug therapy.

In another laboratory Finley (1975) has also been successful in reducing the frequency of seizures in a severe epileptic using a similar procedure to Sterman's. Both Finley's and Sterman's procedures rely on the use of very highly tuned active band-pass filters. An additional circuit blocks feedback reinforcement while slow waves or epileptogenic spikes are present in the EEG. An alternative methodology involves the techniques of digital filtering. Kaplan (1973) using a PDP-12 computer and a variable-band pass filter attempted to train epileptics using this means of frequency selection. Her digital filter was very strict and only responded to 12–14 Hz activity and was not responsive to activity outside of this frequency range which can, through resonance or ringing, cause an active analog filter to respond. Kaplan was unable to reduce seizures in three epileptics using digital methods of processing the SMR, but was able to reduce seizures somewhat in two of the three subjects while training them at a lower frequency of 9 Hz. There is another rhythm known as the central mu or wicket rhythm described by Gastaut, Dongier and Courtois (1954) of 9 Hz which can be recorded over the Rolandic region and may also be associated with motor inhibition.

Presently it is not possible to conclude how central mu and SMR are related.

Nor have the reasons for the failure of digital filtering to provide conditioning of the SMR as opposed to analog filtering been resolved. Nevertheless, Finley was able to provide further evidence that SMR is probably a meaningful distinct phenomenon in humans by showing an acquisition curve in his patient for SMR over many training sessions. Furthermore, Sterman, et al. (1974) showed seizure rates increased upon withdrawal of training and Finley (1975) demonstrated that seizures increased if non-contingent or false feedback is provided to the patient, even for the very short period of only several training sessions.

Both Sterman's and Finley's research have primarily been restricted to epileptics with motor seizures. Virtually nothing is known about the effects of SMR conditioning on psychomotor epileptics. Also, methodological considerations seem to be particularly important in determining a successful outcome as far as reducing seizures is concerned. The purpose of the present study has been to carry out a more extensive examination of the phenomenon of the control of epileptic seizures through SMR training, using a very highly sophisticated hybrid analog and digital processing system which is capable of precisely demarcating 12–14 Hz activity in the raw electroencephalogram (EEG) reinforcing specific microvolt levels of this activity and at the same time not providing reinforcement when this activity is associated with slow waves, epileptiform spikes or gross movements.

In this study patients were not highly selected as in the previous studies, but were chosen for us by a consulting neurologist. The criteria for selection was that patients were severe epileptics, whose seizures were not well controlled by high levels of anticonvulsant medication, which in some cases approached toxicity. Some of the patients in this study showed varying degrees of mental retardation, from mild to very severe. A very strong emphasis has been placed on extremely accurate recording of seizure activity, day by day. Blood levels of anticonvulsant drugs were monitored periodically to determine the patient's compliance with the prescribed medications. The data presented here represent the results of the first three to four months of the SMR training. These patients are presently continuing their training until their seizure level has reached asymptote or until they remain seizure free for such extended periods of time that they can be 'weaned' from the biofeedback training procedures.

The effectiveness of the training procedures reported here is assessed in terms of:

- (1) The number of seizures as a function of time.
- (2) The percentage of 12–14 Hz activity which is the best estimate of SMR compared during training sessions where feedback is provided, as contrasted with baseline sessions where no feedback is provided.
- (3) Analysis of the clinical EEG before the initiation of training and after three to four months of training as analyzed by spectral analysis methodology

(fast Fourier transform, crossed power spectral density and coherence) to determine if there is any normalization of EEG or the appearance of activity in the 12–14 Hz frequency band.

(4) A final successful outcome for this study was a reduction in the levels of anticonvulsant medication. This was done for those patients whose blood levels of medication were excessive and could be reduced.

2. Method

2.1. Experimental design

The study included eight subjects: two normal controls (both female) and six epileptic patients (three male and three female adolescents) all receiving similar treatment. Each subject was given three 40 min training sessions per week. The initial session consisted of habituation during which there was no exteroceptive feedback nor instructions given. Passive recordings of the EEG were taken in order to establish a baseline for the emission of 12–14 Hz (SMR) periodic activity. The number of instances of SMR recorded per minute were printed on a teletype for statistical analysis. In all following sessions the first and last 5 min served as a habituation period to establish a daily baseline for the EEG. These baseline measures were always taken from the same hemisphere on any given day.

After the first 5 min of each training session, subjects were provided with two types of light cues which comprised the feedback. A solitary green light which the subject was instructed to keep on provided feedback based on EEG slow wave amplitude. Colored lamps provided EEG frequency feedback, contingent upon the green light being on. As the EEG response attained a present criterion based on frequency and amplitude requirements this serially advanced the illumination of the lamps and sounded a tone from an 80 dB sonalert. This criterion for receiving feedback was established for the subject prior to the session and controlled for by external logic. A two-decade digital display provided the subject with a running count of criteria met per minute and these were also recorded each minute on a teletype in a separate location. Termination of the 30 min contingency period was signaled by the extinction of all feedback and was followed by a second 5 min baseline.

2.2. Subject selection

The eight subjects were selected in two ways. The normal controls were selected from those undergraduate students volunteering for a brainwave-conditioning experiment. Selection of the six epileptic subjects began with the consulting neurologist presenting from his files a number of cases of refractory epilepsy. Each case was reviewed to establish that the individual was experiencing difficulty with seizure control. Those residing close enough to attend the three experimental sessions per week were then contacted by the neurologist. The proposed experimental treatment program was recommended to the parents

or guardians of the patients. An interview was then conducted by the investigator at the office of the neurologist. The experimental procedure, rationale and requirements were presented, with care being taken to answer all questions. Acceptance provided the individual with the advantage of the experimental treatment at no cost. In addition, the availability of and need for continued treatment after completion of this project was made known to the subject.

The subjects' responsibility was that of attending the three weekly training sessions and maintaining seizure records. Each subject and his or her family agreed to participate in recording the occurrence of seizures. Emphasis was placed on the necessity of accurate seizure count.

Parental approval of the subjects' acceptance into the experiment was followed by a multi-electrode EEG administered by a clinical electroencephalographer. Selected electrical activity of the EEG was recorded with an FM magnetic tape recorder and the subject was scheduled for the first session at the laboratory.

2.3. Epileptics' case descriptions

A summary of drugs, seizures and history for all epileptics is presented in table 1.

2.3.1. Case E1. Case E1 is a 19-year-old female. She is married and the mother of a 26-month-old son. The child appears normal and has given no indication of having a seizure-related disorder. Neither of the subject's parents nor her two older and one younger male siblings have any seizure-related disorder.

The onset of symptoms began approximately five years ago. This was precipitated by a fall with a mild head injury although no direct relationship was established. Initially the subject experienced olfactory and gustatory hallucinations. Later these hallucinations did not occur, but complex and generalized seizures had developed. A bilateral carotid and left vertebral arteriogram were found to be normal. The skull and paranasal sinuses were also normal. The EEG has a 9–10 Hz background alpha rhythm with low voltage spike and slow wave complexes. Neurological opinion was that the EEG shows unequivocal evidence of epileptogenic activity, maximal in the temporal regions but not clearly focal. Final diagnosis was that of a temporal lobe seizure disorder with a suspected organ structural lesion of the temporal lobe.

Over the five-year period prior to the onset of training this subject was treated with Dilantin, Phenobarbital and Valium. Seizures continued, however, and she reported an increase in seizures prior to the onset of training.

2.3.2. Case E2. Case E2 is a 16-year-old male of low-normal intelligence with no seizure history in the family. The onset of symptoms leading to a diagnosis of seizure disorder was at six years of age. Shortly thereafter the subject was in *status epilepticus* for a period of several weeks. While having been seen and

Table 1.
Subject history.

Subject	E1	E2	E3	E4	E5	E6
Sex	female	male	female	female	male	male
Age	19	16	19	14	12	16
Diagnosis and EEG	temporal lobe seizure disorder, suspected structural lesion; 9–10 Hz background alpha with bilateral paroxysms left with low voltage spike and slow wave; complex and generalized seizures	generalized; II, generalized; alpha with bilateral paroxysms left frontal origin; grand mal, petit mal and psychomotor seizures all in one	dysrhythmia grade III, generalized; 8–10 Hz background alpha; arrhythmic slow spike wave bursts; brain damage, severe retardation and psychomotor epilepsy	dysrhythmia grade III, focal right, frontal; 8–9 Hz background alpha with multiple spikes in the 250 mV range for several seconds; centrencephalic-type seizures	dysrhythmia grade III, focal right, frontal and generalized; 8–9 Hz background alpha with generalized poly spike and spike wave abnormality; family seizure history; mild retardation; grand mal seizure	dysrhythmia grade III, focal right, frontal and generalized; 8–9 Hz background alpha with no evidence of focal abnormality; mild retardation; tonic and myoclonic motor seizures
Daily medication: training onset	Dilantin 300 mg	Dilantin 360 mg Zarontin 1000 mg	Dilantin 200 mg Meberol 5.5 grains Tegretol 500 mg Ritalin 50 mg same as above	Phenobarbitol 1.5 grains	Dilantin 400 mg Escobarb 1 grain Mysoline 875 mg Tegretol 400 mg same as above	Dilantin 400 mg Mysoline 1000 mg Dilantin 400 mg Escobarb 1 grain Mysoline 750 mg
Daily medication: end of study	none	Dilantin 200 mg Zarontin 800 mg	same as above	same as above	12	12
Disorder duration in years	4	11	12	11	12	11
Training duration in weeks and sessions	20 weeks 54 sessions	16 weeks 47 sessions	15 weeks 42 sessions	11 weeks 33 sessions	15 weeks 39 sessions	14 weeks 32 sessions
Number and type of seizure prior to biofeedback training	15–20/month; complex and generalized; grand mal	8–9/month; grand mal, petit mal and psychomotor all in one	6–21/month; psychomotor, severe	65–85/month; psychomotor, severe	1–6/month; grand mal	11–21/month; tonic and myoclonic motor, as many as 10/day
Number and type of seizure after training	9–13/month; no grand mal	6/month; no grand mal	1–8/month; psychomotor, mild	12–14/month prior to 1–6/month; grand camp, 78/week after	1–6/month; grand mal	9–12/month; tonic and myoclonic motor

treated at various places the latest evaluation was conducted at the Columbia University Medical facility. While neurological examinations were negative, the EEG was not. The EEG has a background 8–9 Hz alpha with abnormal clinical and subclinical seizure phenomena associated with bilateral paroxysms, apparently having a left frontal origin. This led to a diagnosis of dysrhythmia grade II, generalized as defined by Gibbs, Gibbs and Lennox (1937). Clinically, grand mal, psychomotor and petit mal seizures are reported as 'all rolled into one'.

Over the 10 years prior to enrollment in this training program various drug regimes were tried. This individual was still reported to be having considerable trouble with seizures. While no records were kept prior to enrollment in the training program, 25 seizures were recorded during the first month of training.

2.3.3. Case E3. Case E3 is a 19-year-old severely mentally retarded female. Retardation is believed to be resultant of a birth trauma. The parents and an older male and younger female sibling are normal. There is no evidence of mental retardation or seizure disorder in the family of either parent.

The onset of symptoms leading to a diagnosis of a seizure disorder was at the age of about seven. The EEG has an 8–10 Hz background alpha activity. There is unequivocal evidence of generalized epileptogenic activity in the form of irregular arrhythmic slow and spike-wave bursts. These occur at approximately two per second for 3–4 sec duration. A diagnosis of dysrhythmia grade III, generalized, was given.

During the last three years a number of drug regimes have been administered with limited success. The subject was still reported to be having a lot of seizures. While no seizure records were available prior to enrollment in the training program, 15 seizures were recorded within the first eight days. During the first full month of seizure recording 46 were counted.

2.3.4. Case E4. Case E4 is a 14-year-old female. She is the youngest of four siblings (two males and two females). None of the three older siblings have any seizure-related disorder and are presently college students. Neither of the parents nor their respective families have any history of seizure disorder.

The onset of symptoms is difficult to determine. At a very early age a head movement was diagnosed as a 'tic' by the pediatrician. It was predicted that this would disappear with the onset of puberty. At the age of 14, the 'tic' was still evident at which time the neurological examination was conducted. The EEG had an 8–9 Hz background alpha activity with multiple spikes. This spike activity was in the 250 mV range and lasted for several seconds. The EEG was grossly abnormal with distinct unequivocal evidence of epileptogenic activity in the bifrontal region suggestive of centrocephalic-type seizure discharge. A diagnosis of dysrhythmia grade III, bitemporal and frontal, was given. Clinic-

ally this seizure appeared as a series of rapid successive myoclonic head jerks. This usually was to the right and occurred three or four times in succession.

This subject was treated with 1.5 grain of Phenobarbital daily for about a year with limited success. While no seizure records were kept, the family reported that a lot of seizures were still occurring. During the clinical EEG session just prior to training seven seizures occurred within 1 min. For the first 11 days after enrolling in the training program 134 seizures were recorded.

2.3.5. Case E5. Case E5 is a 12-year-old mildly mentally retarded male. Physical development appears normal although he has numerous allergies to many foods. The only sibling is a younger brother who is mentally and physically normal. Neither parent has had any seizure-related disorder. The father's sister, however, had a seizure disorder when young which subsequently stopped at a later age. A first cousin of the father died of a seizure disorder.

The onset of symptoms leading to a diagnosis of a seizure disorder was at 10 months of age. The EEG has an 8–9 Hz background alpha with an intermittent appearance of generalized poly-spike and spike-wave abnormality. A phase reversal was evident in the right frontal region during a period of spike-wave abnormality. The neurologist's opinion was that there was evidence of epileptogenic activity occurring primarily in a generalized fashion, but on one occasion arising from the right frontal region. This led to diagnosis of dysrhythmia grade III focal right, frontal and generalized. Clinically a diagnosis of grand mal seizure was given.

Over the 10-year period prior to the onset of training various drug regimes were administered. Blood anticonvulsant levels were within the normal therapeutic range. While on the present anticonvulsant regime grand mal seizures occurred between seven and 12 times per month from January through May. The rate of seizure incidence was increasing over time prior to the onset of training.

2.3.6. Case E6. Patient E6 is a 16-year-old male with learning disabilities. He has been enrolled in a special education curriculum and functions at about the 6th grade level. A speech disorder is attributed to an abnormal thickness of the tongue. The four older and one younger male siblings are apparently normal and exhibit no seizure disorder. Neither parent has had any seizure disorder nor has anyone in their respective families. The father had recently been institutionalized for a short period of time for mental dysfunction.

The onset of symptoms leading to the diagnosis of seizure disorder began at the age of five years. While there is an abnormal left maxillary sinus suggesting an inflammatory disease, no other abnormal signs are reported. The EEG is normal with a 7–8 Hz background alpha with no evidence of focal abnormalities. A diagnosis of temporal lobe epilepsy was given. Seizures are clinically expressed as tonic and myoclonic motor system activity. The subject is aware of

a large percentage of his seizures. Prior to and during the onset of training the mother of the patient reported that seizures were occurring during a period of about 2 hr after arising from the night's sleep. Various attempts to get out of a moving car on the way to school were reported. Seizures followed by deep sleep have been reported by teachers during school hours.

Over the 11-year period prior to the onset of training various drug regimes were administered. While no long-term seizure history was available prior to the onset of training, there were 29 seizures recorded in the last nine days of May. During the month of June 79 seizures were reported. Verbal reports of a high rate of seizures, as many as 10 per day, led to the neurologist's suggestion of including this subject.

2.3.7. Controls. Control C1 is a 21-year-old white female college senior. No seizure disorder was reported for this subject nor her siblings or parents. A pretraining clinical EEG was obtained. No abnormalities were evident and this subject was diagnosed as normal relative to this study.

Control C2 is a 22-year-old white female college senior. No seizure disorder was reported for this subject nor her siblings or parents. A pretraining clinical EEG was obtained. No abnormalities were evident and this subject was diagnosed as normal relative to this study.

2.4. Apparatus and procedures

Bipolar EEG electrodes (Grass type E5-SH) were placed bilaterally over both left and right Rolandic cortical areas, one at a point 10% off vertex and the second at a point 30% off the vertex with reference to the ear. The maximal acceptable impedance as determined by a Grass instrument impedance meter was 10 k Ω . The subject was then connected via a Grass Instrument Coronet (PEBN25) to a laboratory constructed, hemisphere switching relay device. This device was controlled by the master program control. (A block diagram of the equipment and circuitry used in this study is shown in fig. 1). This insured that the contingent or trained hemisphere was always amplified by the same amplifier while the non-contingent hemisphere was always amplified by the other amplifier. The patients and controls were trained in an electrically shielded sound-attenuated room under moderate levels of illumination. The bilateral EEG was amplified by a matched pair of Grass Instrument P-511E preamplifiers driven by a Grass Instrument PRS107A power supply. All FM magnetic tape recordings were taken at this point in the circuit prior to any on-line analysis. The output from the amplifier associated with the non-contingent hemisphere was then displayed directly on channel 5 of a Grass Instrument model 3D EEG machine modified for seven channels. The output from the amplifier associated with the contingent hemisphere served as the input for two circuits providing simultaneous discrimination of the EEG and was also displayed on EEG channel 1. The first circuit consisted of a 4–7 Hz,

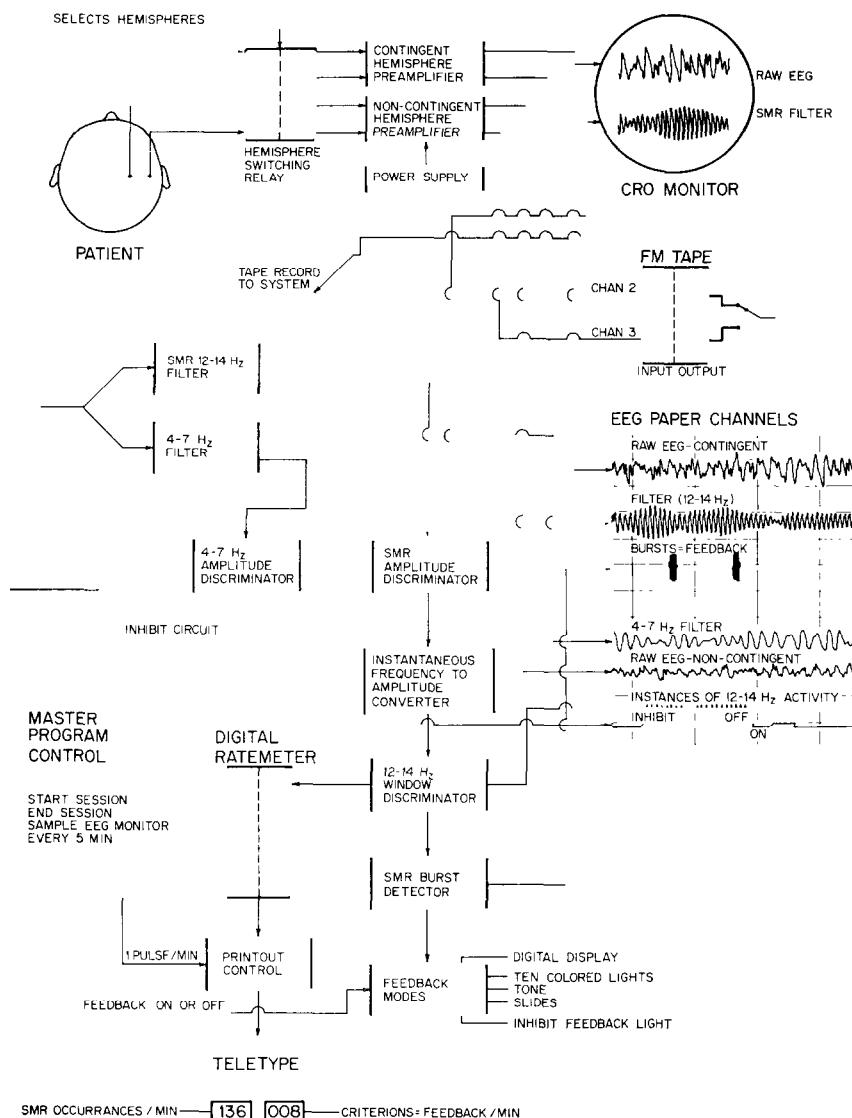


Fig. 1. Block diagram of the logic circuitry and electrode configuration used for 12-14 Hz (SMR) conditioning of epileptics with inhibiting circuitry for slow waves and spikes.

four-pole, twin T-type, 24 dB per octave tuned band-pass filter (White Instrument Co., Austin, Texas). This filter provided 'slow wave' frequency detection which was displayed on EEG channel 4. The filter output was connected to an amplitude discriminator with a variable cutoff. If the slow wave amplitude

was below a specifiable level a digital logic pulse was delivered to an AND gate in the second circuit. Feedback presentation was dependent upon meeting the requirements of this digital logic circuit.

A second circuit for processing the 12–14 Hz SMR activity began with an eight-pole, 80 dB per octave tuned band-pass filter (White Instrument Co.). This filter provided the initial SMR frequency detection and its output was displayed on EEG channel 2. A first analysis performed on the output of this filter consisted of an amplitude discrimination. For this function, if the filter output amplitude was above a preset level a digital frequency analysis was performed. This was carried out by the instantaneous frequency to amplitude converter (ORTEC 4672 instantaneous time-frequency meter). The output of this device passed through a 12–14 Hz adjustable window discriminator (ORTEC 730L single channel analyzer). The function of the instantaneous frequency amplitude converter was to convert each frequency to a voltage for each cycle of the SMR filter activity above the present amplitude cutoff. The function of the 12–14 Hz window discriminator was to pick out precisely 12-, 13- and 14-cycle activity in the SMR filter output. This then constituted the digital band-pass analysis which was superimposed on the original analog band-pass analysis. Now each cycle of SMR activity above a certain specified voltage (for example 5 μ V) was represented by digital logic pulses. These logic pulses represent instances of SMR and were displayed on EEG channel 6. These logic pulses were also delivered to the AND gate in conjunction with the first circuit which processed slow wave activity.

EEG channel 7 recorded the responses of this inhibit circuit. If the digital logic requirements of the AND gate were met a logic pulse was simultaneously delivered to additional circuits. The first of these circuits is shown as a direct connection with an ORTEC 432 digital ratemeter (see fig. 1). This recorded occurrences of SMR, accumulated them and printed them out on a teletype at the end of each minute. The second circuit was connected to a criterion or burst detector. The burst detector consisted of integrated circuit digital logic which included a digital counter and a timer. The counter could be present to any desired number between 1 and 99. The timer could be set for any time up to 9.9 sec in 100 msec increments. The burst detector was programmed to start timing as soon as it ‘saw’ a logic pulse from the AND gate. At the end of the preset sampling time the clock and the counter instantaneously reset whether a criterion was met or not. Criteria for this study required that the subject produce six wave forms of 12–14 Hz activity in 0.5 sec in order to receive feedback based on frequency. If a criterion was not met then another cycle began. Also, when a criterion was met a second cycle began and a logic pulse was simultaneously delivered to the feedback module and the ORTEC digital ratemeter for printout on the teletype. Criteria or bursts were also displayed on EEG channel 3.

The feedback module consisted of integrated circuit digital logic. Control

settings enabled presentation of multiple combinations of: (1) a digital display, showing an accumulating record of bursts per minute; (2) a row of 10 colored lights; (3) a tone; (4) a slide projector; and (5) a large green light, representing inhibition of slow wave activity.

The ORTEC 432 digital ratemeter delivered the accumulating occurrences of SMR and the accumulating criteria of SMR resulting in feedback to an ORTEC printout control. The printout control received a one pulse per minute logic pulse from the master program control which in turn operated a teletype, where a hard copy of this information was recorded.

The master program control consisted of integrated circuit digital logic, and once manually started automatically programmed each 40 min session. A session consisted of 5 min base-rate measurement, a 15 min contingency period on the same hemisphere where the base rate was taken, a 15 min contingency period on the opposite hemisphere, and a 5 min post-contingency base rate again on the initial hemisphere. The order of the hemispheres was alternated for each session to reduce any order effect.

The master program control also controlled the operation of the EEG machine. The first and every successive fifth minute were sampled to conserve paper. However, the analyses, processing and recording of all EEG data was continuous throughout the entire 40 min session regardless of whether or not the paper drive was in operation. A manual override switch enabled sampling to be continuous. In addition, logic pulses activated indicators on the front panel of the master program control to show: (1) the onset and offset of feedback; (2) the hemisphere being conditioned; (3) the fact that the program control was operative; (4) that the EEG machine manual override was on; and (5) whether the feedback manual override was in operation. At the end of every session a logic pulse initiated by the program control sounded a tone indicating session termination.

3. Results

3.1. Case E1

Prior to the onset of biofeedback training E1 was taking 300 mg of Dilantin per day and experiencing 15 to 20 seizures per month on average (table 1). Midway through the investigation it was learned that prior to the beginning of the biofeedback training and after the pretraining clinical EEG was conducted this patient stopped taking the anticonvulsant medication. This was done despite the fact that during the pre-enrollment interview emphasis was placed upon taking the prescribed dosage of the anticonvulsant drug for research purposes. When it was discovered that she was not taking the medication the supervising neurologist was consulted. At that time the neurologist decided to not return case E1 to anticonvulsant therapy until such time that it be deemed necessary.

Biofeedback training for this subject was conducted for a total of 20 weeks and for 54 sessions (table 1). The only exception to this was a 12-day period

during the fifth and sixth weeks when it was necessary to leave the area for a death in the family. As can be seen in fig. 2 an increase in seizure rate was recorded for the two-week period following this interruption in training. After 20 weeks of biofeedback training, however, seizure reduction was developing. The average seizure rate, as seen in table 1, at this time was 9–13 seizures per month compared to 15–20 seizures per month at the onset of biofeedback training.

Seizure rate was correlated with sessions ($r = -0.43$) to determine if a progressive trend toward seizure reduction existed. This correlation was significant ($p < 0.05$ one-tailed). Case E1 also reported that she had learned to block seizures. No grand mal seizures occurred during the last two months of training.

Percentage of 12–14 Hz bandwidth activity was plotted and found to be greater for the feedback period than for the base-rate period, as seen in fig. 3 (case E1). The rate of change, however, does not appear to demonstrate an increment for the feedback period. Since the percentage of 12–14 Hz bandwidth activity was determined from periodic FM tape-recorded samples, caution must be exercised in interpretation of these results. Large variations were observed from session to session and no attempt was made to record only those training sessions representative of good performance. Thus, the 12–14 Hz bandwidth change may have occurred relative to the total EEG but not be represented by this measure. In fig. 4, showing the fast Fourier analysis with plotted compressed isometric routines, a change in the relative contribution of the 12–14 Hz bandwidth of the EEG over sessions can be seen. By session 45 the predominant activity was within the 12–14 Hz bandwidth and remained there on session 54. During sessions 1, 15 and 30 the predominant activity was in the 2.5–7.5 Hz bandwidth. Fig. 5 shows the predominance of 10–15 μ V bursts of 12–14 Hz in the raw EEG of this patient which appeared in the later training sessions. We propose that this activity represents the SMR.

A second fast Fourier analysis was conducted for the same FM tape-recorded sessions to determine the correlation across frequency of the two hemispheres and their corresponding combined power spectral density. Hemispheric relationships are represented by coherence and interhemispheric cross-power spectral density represented as CPSD in fig. 6. Samples of the coherence measures for this case for the base-rate and feedback periods over sessions are provided. While these data are complex and difficult to assess several observations can be made. First, for session 45 the coherence for both the base-rate and feedback periods is greater for the 12–14 Hz bandwidth than for the slow wave epileptogenic related bandwidth. Second, the CPSD on session 45 is relatively greater during feedback than during the base-rate period for the same 12–14 Hz bandwidth.

Representative samples of the FM tape-recorded pre- and post-clinical EEG are presented in fig. 7. Fast Fourier analysis was conducted and resultant

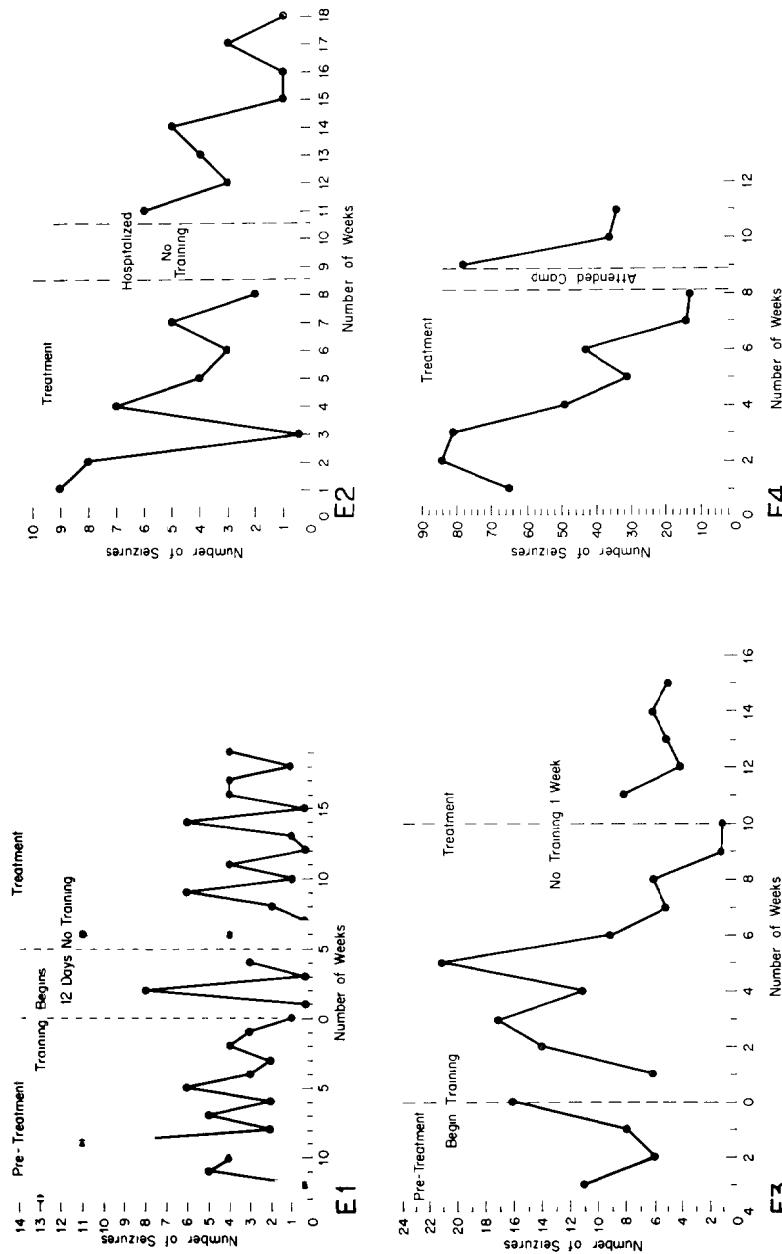


Fig. 2. Graphical display of the number of seizures over the course of the pretreatment and treatment phases of this study, for all epileptics E1-E6.

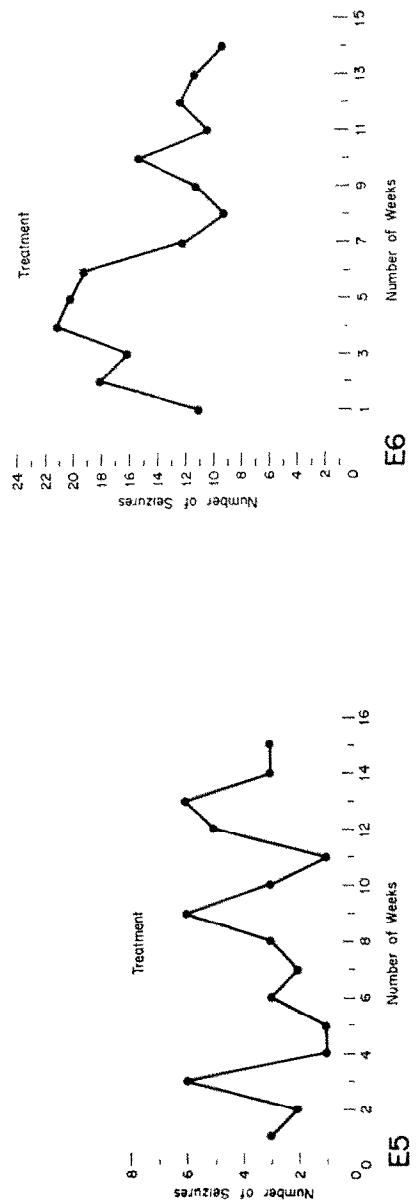


Fig. 2 (continued).

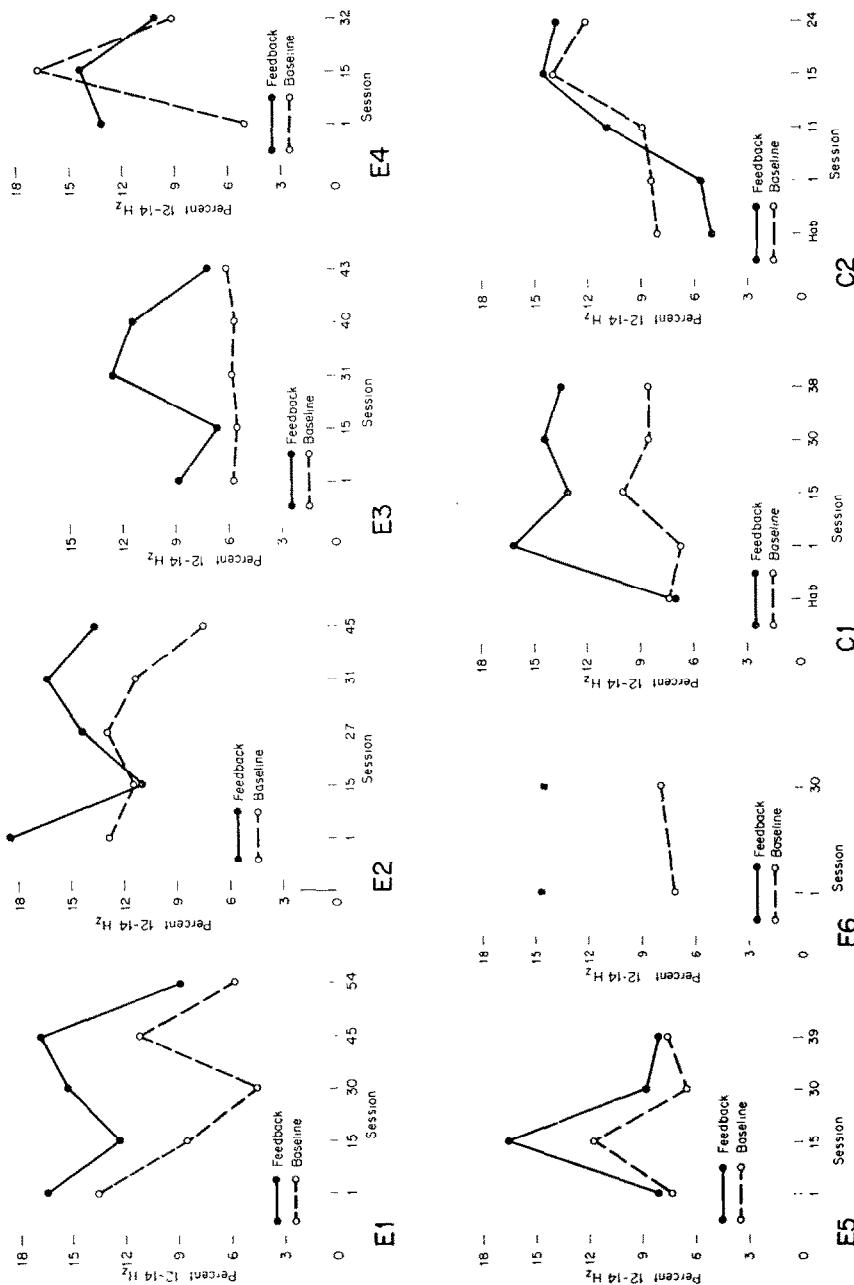


Fig. 3. Percentage of 12-14 Hz (SMR) activity for all patients and controls. These data were obtained from FM tape-recorded EEGs during the indicated sessions.

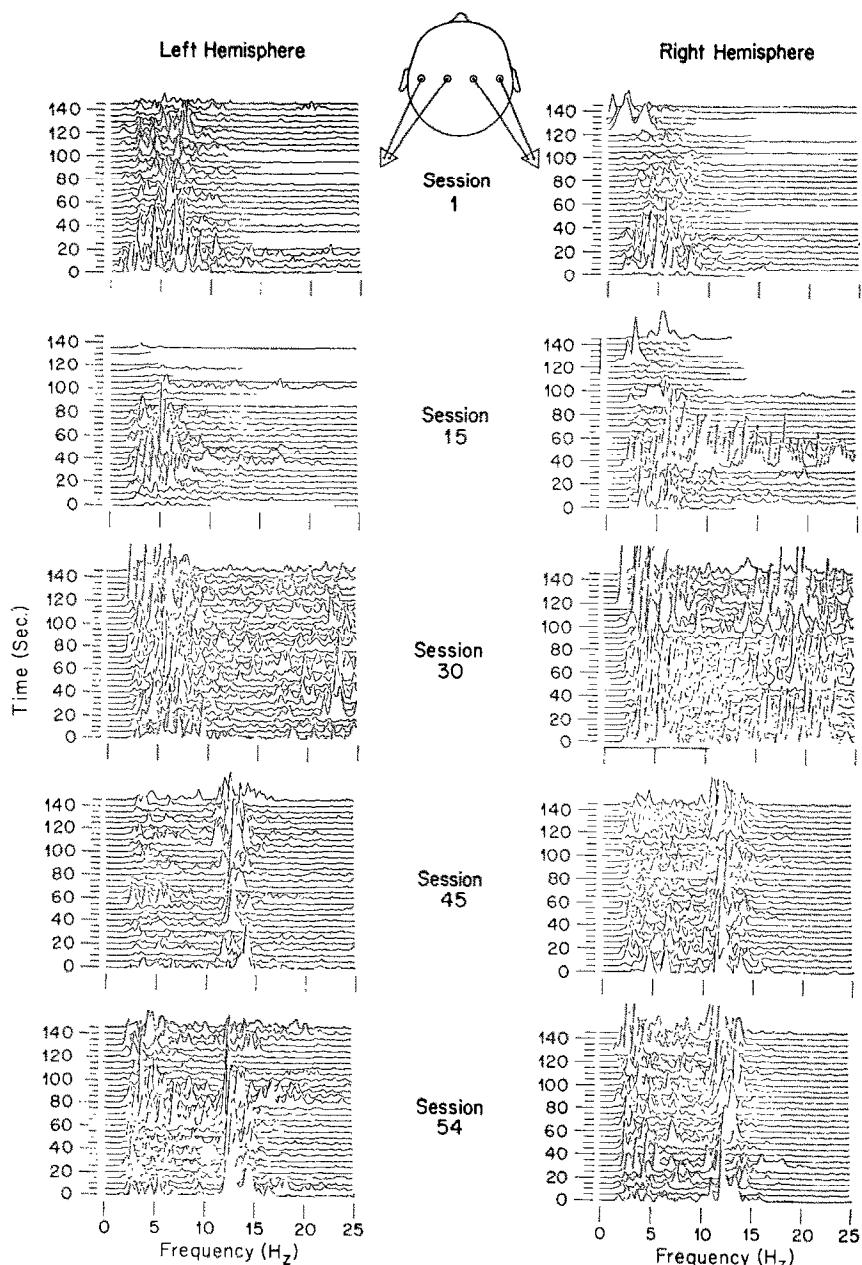


Fig. 4. Continuous fast Fourier spectral analysis with compressed isometric plots for sequential 5 sec epochs of EEG. These measures represent the EEG when feedback was available for 12-14 Hz activity; case E1.

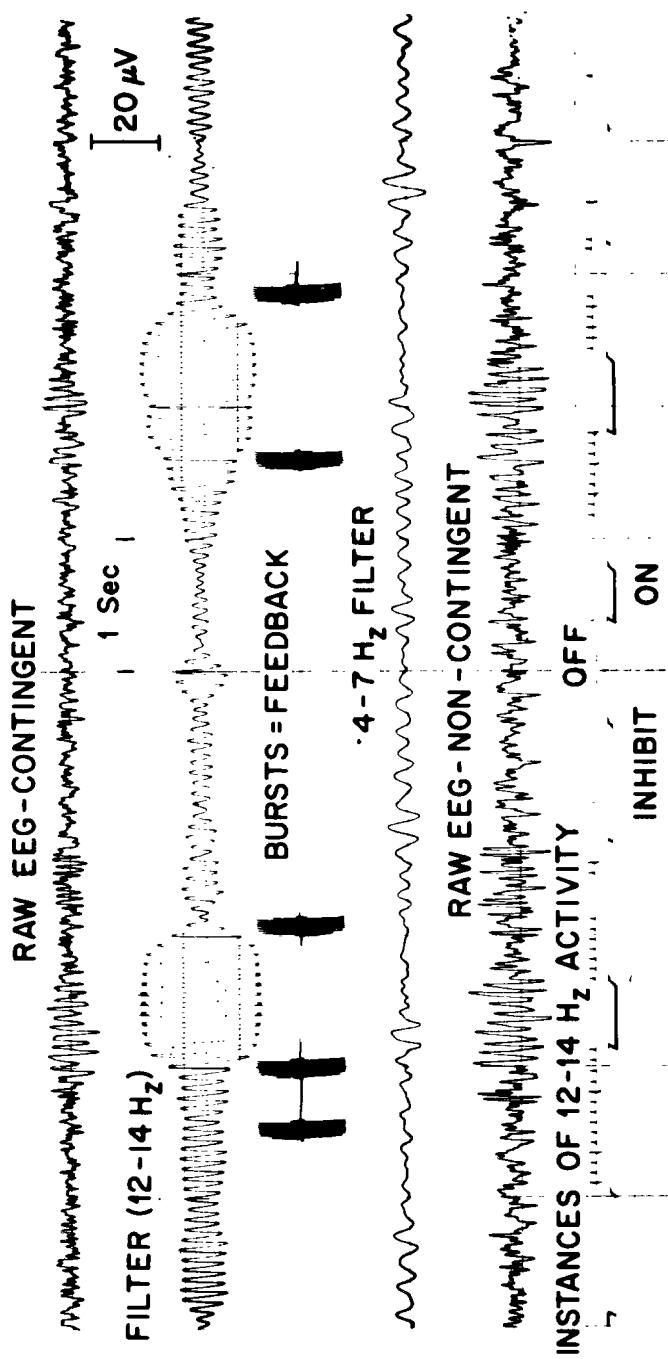


Fig. 5. Sample EEG with filter outputs, inhibit circuit responses and feedback indicated for case E1. Note bursts of sinusoidal 12-14 Hz activity occurring bilaterally in the raw EEG and associated feedback. Time and amplitude calibrations are indicated.

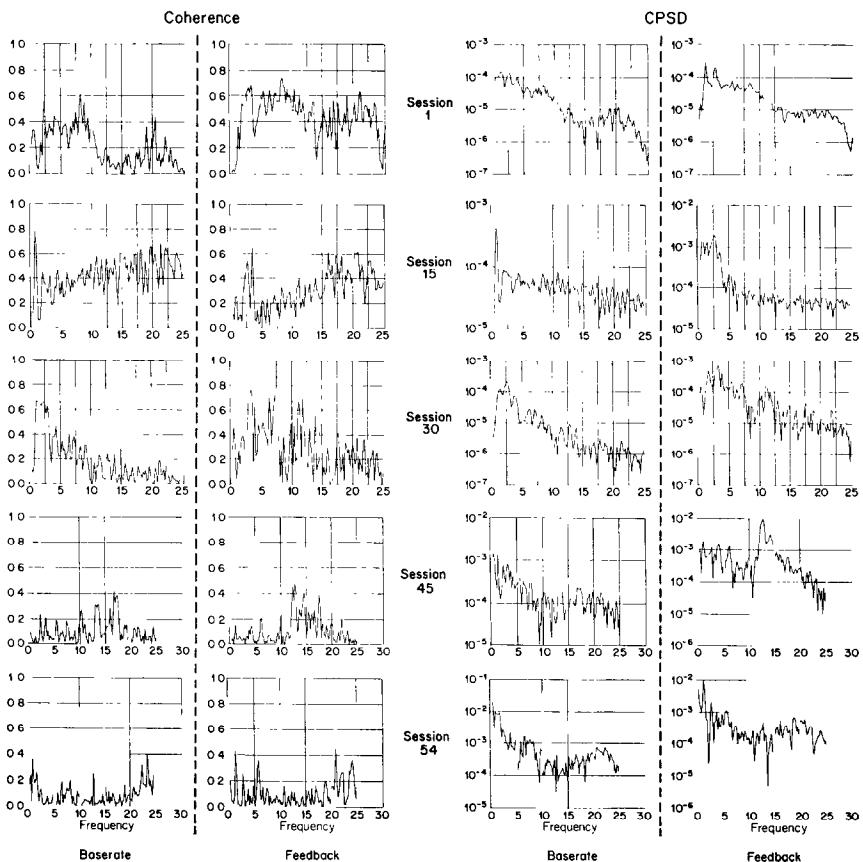


Fig. 6. Continuous fast Fourier analysis for coherence and cross-power spectral density (CPSD) for base-rate and feedback conditions for specific training sessions; case E1. Note relatively high coherences in session 45 and the high CPSD for this band during feedback for this session.

compressed isometric routines were plotted. The 10% and 30% from vertex bilateral electrode positions representing the placements for biofeedback training purposes indicate an overall reduction in the slow wave, high voltage, epileptogenic bandwidth, while the 12–14 Hz bandwidth, as well as higher frequencies, has increased for the same electrode placements.

The bipolar bilateral electrode placements C3–T3 and C4–T4 (anterior triangles configuration) represent a most obvious change. The pre-EEG record is dominated by the slow wave activity and the post-EEG record has a high amplitude representation of the 12–14 Hz bandwidth.

The O1–T3 and O2–T4 bilateral electrode placements (posterior triangles configuration) illustrate the electrical activity of the occipital pole. In the

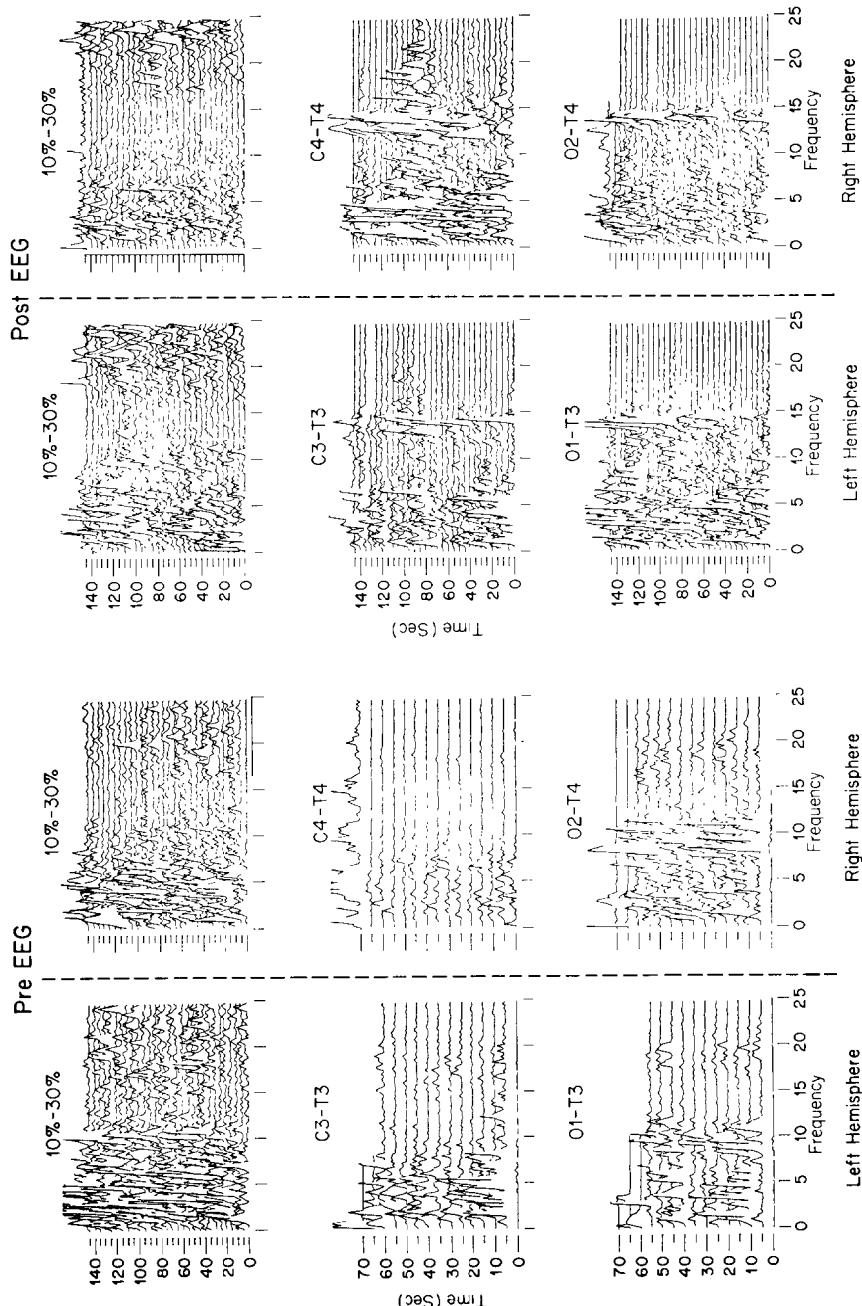


Fig. 7. Continuous fast Fourier analysis with compressed isometric plots for repeated 5 sec epochs; case E1. The pretraining and post-training clinical EEG is represented. Letter designations are according to the international 10-20 system of electrode configuration. Note occurrence of significant activity in the 12-14 Hz band in the post-training EEG for C_3-T_3 , C_4-T_4 and $O_1-T_3-O_2-T_4$ positions.

pre-EEG the predominant frequencies are within the 8–11 Hz bandwidth classically associated with what is called ‘alpha rhythm’. The post-EEG measure illustrates the occurrence of less alpha and clearly more activity in the 12–14 Hz bandwidth. The right hemisphere has less slow wave activity than the left over the same period.

3.2. Case E2

Approximately three weeks after initiation of biofeedback training this patient required an anticonvulsant drug reduction due to toxicity. This was not accompanied by hospitalization or loss of training time. Fig. 2, case E2, represents the rate of seizures by weeks over the biofeedback training period. Training at the average rate of three sessions per week was followed by a gradual reduction in seizures until the beginning of the ninth week. At this time the patient was hospitalized for anticonvulsant drug toxicity. Hospitalization lasted for more than two weeks before returning to biofeedback training. Seizure records could not be reliably maintained during this period.

Upon returning to biofeedback training at a new reduced anticonvulsant drug level the three sessions per week schedule was reinstated. Seizure rate, as represented in fig. 2, had increased above the level attained just prior to hospitalization. The following five weeks of training were accompanied by further marked seizure reduction, with no grand mal despite reduced medication. The correlation of seizures over sessions was equal to -0.543 ($p < 0.05$, two-tailed).

The periodically sampled FM tape recordings of the biofeedback training were analyzed for the percentage of 12–14 Hz bandwidth activity. Fig. 3, case E2, does not indicate an increase in the percentage of 12–14 Hz activity. This may, however, be confounded by the two drug reductions. While the feedback period usually had a greater percentage of 12–14 Hz activity this was reversed in session 15. No recorded differences for this session can account for this, but it was observed occasionally for several other patients as well. When asked what they were doing differently most often it was reported that they were trying some new strategy to make the feedback lights move.

The fast Fourier analysis showed no obvious increases in the 12–14 Hz bandwidth over sessions. There was, however, a general reduction of activity in the low-frequency bandwidths associated with the slow wave epileptogenic activity.

A reduction in slow wave activity was also observed from the pre- to the post-clinical EEG recorded at the 10% and 30% points from vertex where training was undertaken. No evident increases within the 12–14 Hz bandwidth were observed in the pre- to post-clinical EEG. However, there was an increase in the 8–11 Hz bandwidth in the post-clinical EEG for the bipolar bilateral electrode placements T5–O1 and T6–O2. The slow wave, high voltage epileptogenic activity was still present at these loci. The P3–O1 and P4–O2 bipolar

bilateral electrode recordings for the pre- and post-clinical EEG showed an increase in the 8–11 Hz alpha and a decrease for the high voltage epileptogenic slow waves. Pre- and post-clinical EEG comparisons for bipolar bilateral electrodes C3-T3 and C4-T4 were similar to previous recording sites. A decrease in slow wave, high voltage epileptogenic activity was found. Overall then, the EEG for this case tended toward normalization.

3.3. Case E3

Biofeedback training for this subject consisted of 42 sessions over a 15-week period. The sessions were conducted three times per week on the average, except for a one-week period when case E3 attended camp. As seen in fig. 2, an increase in seizures followed the week off from training. The overall correlation of seizure reduction over sessions was equal to -0.481 ($p < 0.05$, two-tailed). Comparison of seizure rates in table 1 indicates a reduction of from 6–21 to 1–8 seizures per week at the end of this investigation.

Examination of fig. 3 shows a general increase in the 12–14 Hz bandwidth activity over sessions during feedback with a nearly constant rate during base-rate periods. An exception to this was day 43. As previously mentioned for other subjects, a great deal of variability existed from session to session, therefore, significance need not be assigned to this decrease.

The FM tape recordings from the periodically sampled EEG training sessions were analyzed by the fast Fourier analysis method. An increase in higher frequency activity and decrease in amplitude was apparent over sessions sampled. By session 43 the record appeared much more normalized. During session 31 a relatively high coherence developed for the 12–14 Hz band.

The FM tape-recorded pre- and post-clinical EEGs were also analyzed by the fast Fourier analysis method with compressed isometric plotting routine. The 10% and 30% from vertex bilateral electrode positions representative of the electrode placements used for biofeedback training purposes indicated no major change in this patient. The post-clinical EEG record for the left hemisphere had a greater degree of higher frequency activity, but no clear-cut evidence for 12–14 Hz bandwidth change was apparent. Slow wave, high voltage epileptogenic activity was diminished somewhat for the right hemisphere during the post-EEG recording.

The P3-O1 and P4-O2 bipolar bilateral electrode placements (sagittal configuration) were more illustrative of a change in the higher frequencies from pre- to post-EEG recording. Here there was electrical activity in the post-EEG record corresponding to the 12–14 Hz bandwidth.

3.4. Case E4

As seen in table 1, initially case E4's seizures were not controlled and were occurring initially at the rate of 65–86 per week. Training for case E4 took place on the average of three times per week for 33 sessions over a 15-week period.

Seizure records were lost by her family for the final three weeks of training. Fig. 2 is illustrative of the rapid reduction in seizure rate experienced by this patient. Prior to the one-week period without training (camp) seizures had been reduced from as high as 84 to as low as 11. Immediately following camp the seizure rate was recorded at 78 per week. Seizure rate had dropped to 36 and 34 during the following two weeks.

In addition to the week at camp this patient's training was further interfered with by a fall from a top bunk bed requiring 30 stitches to the left side of her face. Following that she was involved as a passenger in a major automobile accident. Despite these non-training-related mishaps the correlation for seizure rate and sessions was equal to -0.521 ($p < 0.05$, one-tailed).

Figure 3, representing the percentage of 12–14 Hz bandwidth activity over training sessions, does not indicate a clear picture. Session 1 is similar to other records taken for other subjects with the feedback period having a higher percentage of 12–14 Hz activity than the base-rate period. Session 15 is the reverse of this, however, and while not indicated here the patient performed in this manner for several sessions. As noted for a previous subject, this inverse relationship was often associated with post-session verbal reports of the subject trying a new tactic to advance the feedback lights. Session 32 indicates a lower percentage of 12–14 Hz activity than in the two previously reported sessions, but the feedback period is slightly higher than the base-rate period.

The 10% and 30% from vertex bipolar bilateral electrode placements used for biofeedback training did not indicate improvement over sessions. The slow wave, high voltage epileptogenic activity was the predominant rhythm. Neither coherence nor CPSD provide any clear trend over sessions. But in session 3 there was a slight increase for coherence at the 12–14 Hz bandwidth. There was also a decrease in coherence during the feedback period for slow wave activity on the same session. The only major change over sessions appears to be a decrease in coherence and CPSD for activity within the 10–12 Hz bandwidth. In later sessions occasional bursts of 12–14 Hz activity could be seen in the raw EEG.

As in other cases the pre- and post-clinical EEG FM tape recordings were analyzed with the fast Fourier program. The 10% and 30% from vertex bipolar bilateral electrode placements indicated a bilateral reduction in the slow wave, high voltage bandwidths. The bilaterally temporal to vertex placement, T3–CZ and T4–CZ, did not indicate a major change in activity for the pre- and post-EEG. The bilateral bipolar electrode placements T5–O1 and T6–O2 appeared to be very similar with regard to the representation of frequencies. There was a reduction in amplitude for the 10–11 Hz bandwidth in the post-clinical EEG record with consequent relative higher amplitude, slow wave activity. This should not be construed as an increase in slow wave amplitude since the plot is relative to the highest amplitude detected. Thus the decrease in 10–11 Hz would indicate an increase for all other frequencies. Finally, the post-clinical EEG

record for bipolar bilateral electrode placements C3-T3 and C4-T4 indicated a decrease in slow wave amplitude. In this case, then, only slight normalization could be seen in the EEG despite a marked reduction in seizures.

3.5. Case E5

At the onset of biofeedback training this patient, a 12-year-old male, was having one to six seizures per week which were predominantly grand mal. Fig. 2 is illustrative of the lack of success with this subject, the only one for which the treatment has not yet been of value. Table 2 substantiates that this was the only subject to not have some degree of negative correlation for seizures over sessions ($r = +0.233$, not significant). As expected fig. 3 does not indicate any increase in the percentage of 12–14 Hz bandwidth activity, except for session 15.

No change was found in the fast Fourier analysis across sessions for either the 12–14 Hz bandwidth or the slow wave, high voltage epileptogenic activity. A fast Fourier analysis for interhemispheric coherence and interhemispheric cross-power spectral density across sessions confirms the previous lack of effect. Coherence and CPSD for base-rate and feedback conditions across sessions indicated the predominance of slow wave, high voltage epileptogenic activity.

Finally, pre- and post-clinical EEG analysis by fast Fourier analysis unequivocally illustrated no change. Bipolar bilateral electrode placements for 10% and 30% from vertex, T3-CZ and T4-CZ, T5-O1 and T6-O2, as well as P3-O1, all indicated a pre- and post-clinical EEG totally dominated by slow wave, high voltage epileptogenic activity.

3.6. Case E6

Figure 2 indicates a slowly declining seizure rate over sessions. At the end of the period serving for data collection for this study this patient was experiencing 9–12 seizures per week. Statistically the correlation of seizures over time was equal to -0.583 which is significant ($p < 0.05$ level, two-tailed).

The percentage change for 12–14 Hz bandwidth activity in fig. 3 showed no increment over time except for a marked difference between feedback and base-rate levels. Slow wave, high voltage epileptogenic activity decreased over sessions, however, with the greatest decrease occurring for the right hemisphere.

Interhemispheric measures of coherence and CPSD across sessions indicated an increase in coherence during base rate for 12–14 Hz bandwidth activity and a similar increase in coherence for the 12–14 Hz bandwidth during the feedback periods across sessions. During many training sessions 12–14 Hz bursts could be seen in this patient's raw EEG similar to those seen in case E4 and as shown in fig. 5 for case E1. Slow wave, high voltage epileptogenic activity decreased slightly over time. CPSD also decreased for the low frequencies.

The T3-CZ pre-clinical EEG versus the T3-CZ post-clinical EEG as well as the T4-CZ (temporal-vertex configuration) indicated a decrease in slow wave, high voltage activity for the post-clinical EEG record. A similar finding held for the pre-clinical EEG at T5-O1 contrasted with the post-clinical EEG at T5-O1 and T6-O2 (posterior temporal configuration). There was no obvious increase, however, of activity in the 12–14 Hz bandwidth.

3.7. Control C1

Percentage of change for the 12–14 Hz bandwidth, as represented in fig. 3, indicates an increase over the habituation session, but there is no increment for the following sessions. Base rate is consistently below feedback across sessions, however, indicating differential control.

The Fourier analysis showed that from sessions 1 to 15 there was a decrease in low frequency activity with an increment for the 9–11 Hz bandwidth. By session 30 there was an overall increase in power for the higher frequencies. Coherence across sessions increased for 12–14 Hz bandwidth activity and was particularly strong by session 30. There were no apparent differences between base-rate and feedback conditions for the measure of coherence. The contributing CPSD for the 12–14 Hz bandwidth also increased over recorded sessions. There was no apparent difference between the base-rate and feedback conditions for the CPSD measure.

The 10% and 30% from vertex bipolar bilateral electrode placements indicated a concentration of power at the 8–11 Hz bandwidth. This is true for the pre- and post-EEG measure but is slightly greater in the post-EEG condition. This was similar at the bipolar bilateral electrode sites T3-CZ pre-clinical EEG, T3-CZ and T4-CZ post-clinical EEG; C3-T3 pre-clinical EEG, C3-T3 and C4-T4 post-clinical EEG. This predominant 8–11 Hz activity represents the alpha band which is readily apparent in non-epileptics. In contrast for the epileptics predominant activity is found in the 5–9 Hz band which represents in part theta or slowed alpha. The latter may be in part due to the effect of the medications.

3.8. Control C2

Biofeedback training for this individual consisted of 24 sessions at an average of three sessions per week. Percentage of 12–14 Hz bandwidth activity can be seen as an increasing function over time in fig. 3. Base rate falls below the feedback period after the first session and remains slightly less for the remaining sessions.

The fast Fourier analysis showed no clear increases in the 12–14 Hz bandwidth; however, there was some higher amplitude representation of the higher frequencies for the right hemisphere during session 24.

No overall trend for coherence to increase for the 12–14 Hz bandwidth was

found across sessions. But a noticeable increase of coherence during the feedback period for the 12–14 Hz bandwidth did occur during session 15. Preclinical EEG data for this case could not be analyzed due to malfunction of the tape recorder.

4. Discussion

The results of this preliminary experimental treatment were most encouraging. Five of the six epileptics showed significant reductions in seizures during the first three to four months of their treatment. One notable exception, however, was case E5. According to the hypothesis presented previously that the occurrence of SMR is associated with inhibition of motor activity and unit activity in the ventrobasal thalamic nuclei, it was expected that this patient would be particularly amenable to treatment since his seizures were primarily of the grand mal type. There were, however, several factors which might have explained his poor performance during the first three and a half months of treatment. First, this subject was the youngest – 11 years old. Although this patient enjoyed his visits to the laboratory and was very gregarious in interacting with laboratory personnel, midway through the training procedure he reported that he was frightened of being alone in the sound-attenuated room in which the training was conducted, even though he could communicate by a two-way intercom with the experimenter. He also had considerable motivational problems in keeping his attention fixed on the task. By the time this study had been completed a slide projector was introduced which is now programmed so that after the 10 colored lights are lit in sequence a slide is projected for several seconds. It is hoped that with this – perhaps more interesting – type of feedback display progress will be made with this subject. It is also possible that for some patients other electrode positions than the 10–30 are better. It should be pointed out that Finley (1975) records unilaterally placing one electrode over the Rolandic region which is referenced to the contralateral ear lobe. This recording array would result in a larger amplitude overall signal and perhaps larger amplitude SMR, which might be more easily discriminated by the patient.

Another finding in this study was that only in a few cases was it possible to obtain convincing evidence for the formation of a peak in the 12–14 Hz band in the power spectrum, representing the frequency for which feedback has been provided. The reason for this is in part due to the nature of Fourier analysis. High amplitude, low frequency activity will dominate the frequency spectrum. The raw EEG of the epileptics reported here all showed a predominance of high amplitude, low frequency slow waves primarily in the 3–8 Hz range. SMR on the other hand has been reported by both Sterman's and Finley's groups to be a relatively minute signal, usually of less than 10 μ V. Even large bursts of 12–14 Hz illustrated in fig. 5 for case E1 reached only 12–15 μ V which was considerably smaller than the concurrent slow wave activity. It may very well be that 12–14 Hz activity did increase with training, but the most appropriate analysis

to prove this would be a pure band-pass analysis. For such analyses a frequency histogram is developed for the different frequency bands which in turn are selected by highly tuned, active band-pass filters. Our current research program is working toward this refinement. On the other hand the development of some coherences in the 12–14 Hz band do indicate that two cerebral hemispheres tend to respond together for the frequency band representing the SMR. However, high coherences also occur when there are slow waves present.

The current study indicated that for some patients there was some increase in the percentage of 12–14 Hz activity over recorded sessions. It must be emphasized that this measure was derived from analyses of FM tape-recorded sessions. Because there is much variability between sessions and no attempt was made to select particularly good sessions for recording, the data were highly variable on this measure. Nevertheless, in most cases we did find higher percentages of SMR during feedback periods than during baseline periods.

One question which can be asked of any new treatment is to what extent it represents the placebo effect. In this regard Shapiro (1960) pointed out that placebo effects occurring when treatments have been changed or new treatments are introduced are usually transient and eventually wear off. Although the effect of biofeedback training for these patients was undoubtedly novel initially, after two or three months the novelty has largely worn off, and to some extent coming to the laboratory from long distances three times a week has been reported by a few patients to be tiresome.

In view of the work of Sterman's and Finley's laboratories showing that biofeedback training of the sensorimotor rhythm has been effective in reduction of epileptic seizures we doubt whether a placebo effect accounts for our significant positive cases using this technique. Even more crucial to this point was Finley's report that his patient showed a decrease in the percentage of SMR and increased seizures after a few sessions were interposed in which false (non-contingent) feedback was provided without the patient's knowledge.

The greatest advantage of the SMR biofeedback approach to the management of epileptic seizures is that it offers hope for severely epileptic patients that their seizures might be controlled when anticonvulsant medication is not sufficient. This accounts for approximately 25% of epileptics. For many patients whose seizures are controlled by drugs the hope exists that drug therapy might be reduced, and perhaps even eliminated from some. This was already confirmed in the case of E1 whose drugs were not taken during the course of the treatment reported here. This is in itself important since anticonvulsant medication produces severe side effects in many patients that are reflected mentally, physically and emotionally. The other advantage of this new approach is that it places the emphasis upon *voluntary* control and on the patients managing their own seizures. Since epilepsy is associated with psychological dependency this gives them the opportunity to build self-esteem and confidence by being able to manage their own disability.

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