

EEG Biofeedback: Physiological Behavior Modification

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STERMAN, M. B. *EEG biofeedback: Physiological behavior modification.* NEUROSCI. BIOBEHAV. REV. 5(3) 405-412, 1981.—The author reviews the use of operant conditioning to alter electroencephalogram (EEG) patterns. A discrete rhythmic EEG pattern directly related to modulation of motor patterns (sensorimotor rhythm, SMR) was brought under voluntary control in the cat. This technique was modified for use in epileptic human volunteers in order to reduce motor seizures. The use of a newer experimental design and its successful application in one subject is described.

EEG biofeedback Seizure reduction Epilepsy

SPONTANEOUS electrical patterns recorded from the human brain and known as the electroencephalogram, or EEG, have been considered in relation to concurrent behavior since their first appreciation by Berger [3]. While the correlation of EEG patterns with behavior has not provided a concise physiological parallel, it has allowed for general classification of "states of consciousness" and has given the neurologist an important tool for recognizing cerebral abnormalities. The EEG may be used today to accurately define the onset and various stages of sleep and the degree of cognitive orientation during wakefulness. Moreover, it is utilized to diagnose and localize cerebral damage and organic disorders of consciousness. Recent advances in concept and technology suggest that an even greater resolution of behavioral substrates may eventually be derived from the EEG.

To the extent that functional substrates of behavior are manifest in the EEG, the developing field of EEG biofeedback may represent the most direct method of behavior modification yet conceived. Indeed, it has been established that operant conditioning methods can reliably alter EEG patterns [11, 12, 22, 28] and correlated behaviors [2, 8, 19, 21]. These latter findings shall be the primary focus of this discussion.

Contrary to popular belief, biofeedback is not merely a means of teaching individuals to evoke pleasant subjective states which provide internal tranquility and combat psychological stress. To be sure, by its very nature the method of biofeedback requires that the subject assume personal responsibility for any beneficial effect to be had and provides the basis for a new level of self-awareness and relaxation. In this respect, there is usually a general response to biofeedback training which resembles some aspects of meditational and Yoga experiences. This can often be achieved efficiently in the biofeedback setting and can set the stage for other types of psychological intervention with a disturbed individual. While these indirect influences of the method are beneficial and provide a desirable alternative to drugs, they do not represent the most promising aspect of this technique,

which is the possibility of harnessing the well-documented plasticity of mammalian physiology. Response systems at all levels are a product of this plasticity and, accordingly, could eventually be vulnerable to modification by this approach.

Let us review some evidence which clearly demonstrates this intrinsic plasticity in nervous system function. As is often the case, this characteristic of regulation is most apparent in the modulation of extant pathology. Figure 1 shows a series of polygraphic tracings obtained from an epileptic during the transition from wakefulness through the various stages of sleep. This 18-year-old female patient suffered from frequent generalized seizures producing brief loss of consciousness, twitching and falling or akinetic attacks. While awake and alert (upper left) the EEG was essentially normal, showing sustained low voltage, fast activity. As soon as the patient became drowsy (upper right), a generalized burst of epileptiform discharge can be seen breaking into a slow-alpha dominated pattern. During Stages I and II of sleep abnormal discharge becomes more frequent and episodic (center). This pattern merges into one of almost continuous spike and wave paroxysmal activity during Stages III and IV (bottom left). Finally, accompanying the transition into the rapid-eye-movement or REM stage of sleep, abnormal discharge is essentially terminated (lower right). Thus, the same pathologic central nervous system can be seen to both permit, modulate and block abnormal activity as a function of various intrinsic organizational states. In fact, epilepsy, like many other abnormal conditions, including disorders of behavior, characteristically shows a variable course related to age, life events, biological periodicities and other illness. Such perversity may, indeed, be a clue we have yet to fully explore in treatment.

FEEDBACK OF THE SENSORIMOTOR RHYTHM

The conception of biofeedback as a potential behavioral approach to treatment of epilepsy began in our laboratory many years ago in relation to ongoing neurobehavioral

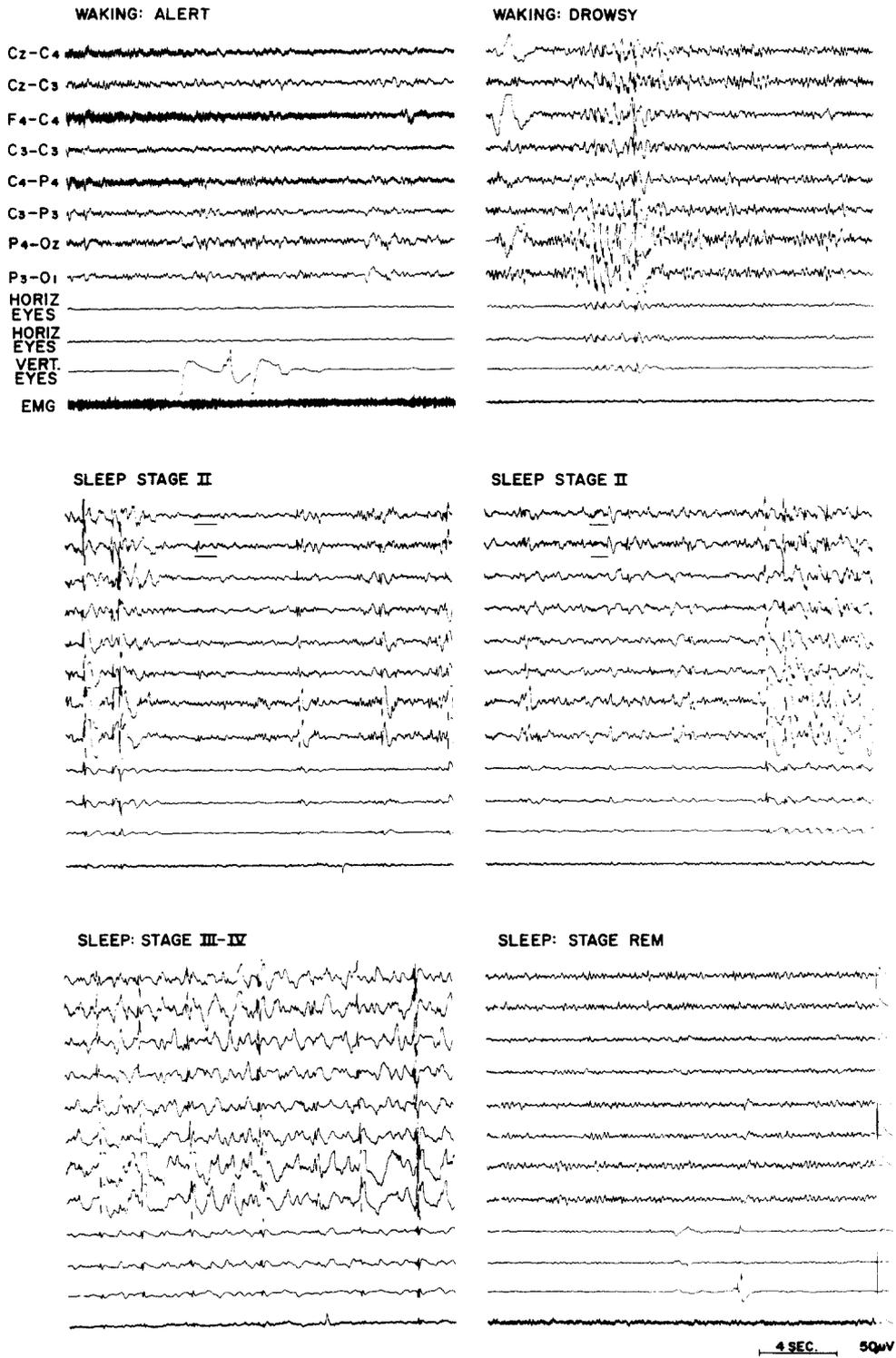


FIG. 1. Changes in the incidence and pattern of epileptiform EEG discharge in relation to stages of wakefulness and sleep in an epileptic patient. Polygraphic record samples show bilateral EEG traces as well as eye movements and chin muscle EMG. Underlined EEG traces in Stage II sleep emphasize poorly developed spindle burst activity in this patient.

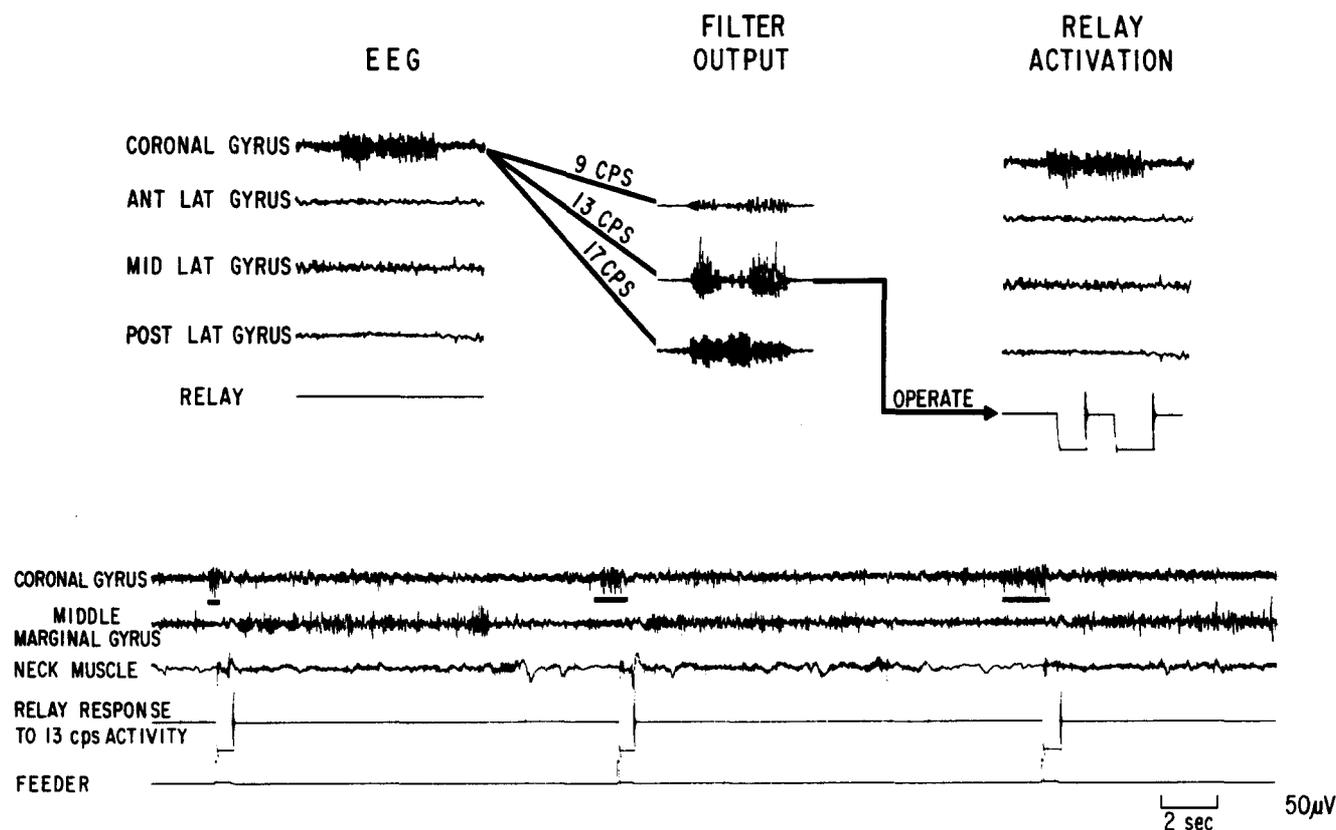


FIG. 2. An example of sensorimotor rhythm (SMR) activity recorded from an alert, motionless cat is shown at top together with a simplified description of signal detection procedures. The rhythmic EEG activity is directed to a set of tuned filters, the response of which indicates primary activity at 13 Hz (actually 12–14 Hz band). This filter output is then used to operate a relay which automatically detects the rhythmic SMR activity in subsequent replay of recorded signal. Traces at bottom show sequence of SMR responses from sensorimotor (coronal) cortex in a feedback trained animal, together with slower EEG rhythmic activity from marginal cortex related to food consumption, neck muscle activity, relay and feeder operation. This record was recorded from right to left.

studies in the cat. Animals prepared surgically with indwelling cortical electrodes were trained to obtain food contingent upon a lever press. A negative discriminant stimulus was introduced, in the form of a light or tone, which signaled that a lever press would significantly delay the opportunity to obtain food. As the cats learned to suppress responding in the presence of this stimulus, a unique pattern of rhythmic activity began to appear in the EEG over the sensorimotor cortical area (Fig. 2). Careful examination of this relationship disclosed a striking correlation between the voluntary suppression of movement and this EEG pattern, which we subsequently termed the sensorimotor rhythm or SMR.

Because of the prominence of the SMR within the generally desynchronized sensorimotor EEG of the cat, we were able to consider operant conditioning of this response in order to explore further its physiological and behavioral correlates. A signal detection system was developed which could be triggered by the 12–16 cps rhythmic activity and activate a feeding device in the animal's chamber. The cats learned to perform this EEG instrumental response rapidly, with even greater ease than lever pressing [30]. Two groups of animals were provided with differential operant conditioning, one to enhance and the other to suppress the SMR in order to receive food. Animals trained to produce the SMR did so by becoming motionless, whereas those trained to

suppress it remained active. The motionlessness associated with trained SMR performance provided an excellent opportunity for assessment of concurrent neurophysiological data. It was found that phasic and tonic motor discharge was inhibited specifically in relation to production of the SMR and that reflex excitability was suppressed [1,6]. Furthermore, animals trained to produce the SMR showed specific changes in neuronal activity recorded with microelectrodes from structures in the motor pathways [9].

Behavioral studies were carried out also. In particular, sleep was found to be altered as a result of SMR training, such that movements were decreased, sustained epochs were prolonged, and the total amount of sleep occurring in a given 24-hour period was reduced [15,19]. Perhaps most significant was the observation that animals provided with SMR training showed resistance to drug induced seizures, a fact accidentally discovered within the context of a different experiment [18] and later confirmed in specific studies [23].

The finding that this discrete rhythmic EEG pattern could be brought under voluntary control in the cat, and was related directly to modulation of motor patterns, encouraged us to extend our study of this phenomenon to man. Specifically, we were interested in the possible therapeutic benefits of this approach to epilepsy. The transition from cat to human research proved both fascinating and difficult. We

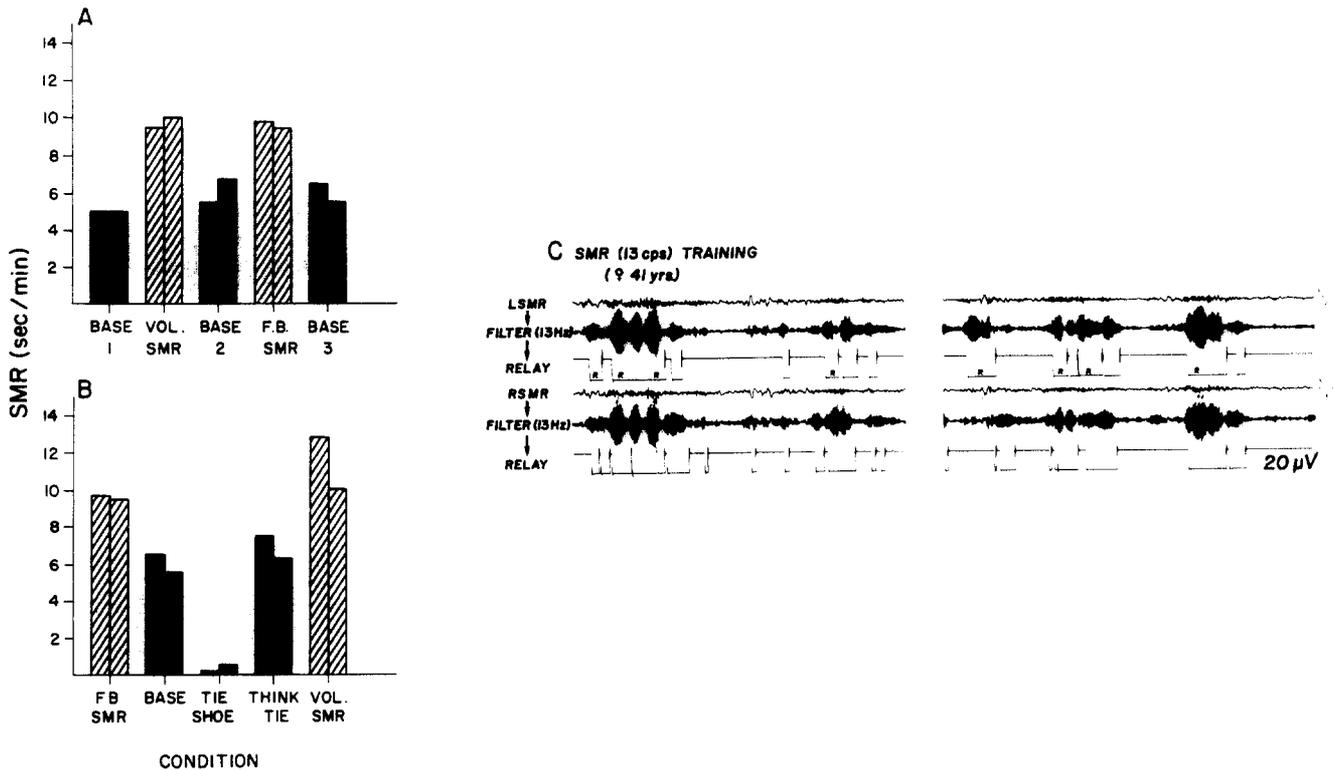


FIG. 3. Trained SMR response characteristics in man are depicted here in relation to various conditions (A and B) and in terms of actual EEG trace and filter-relay detection (C). In A incidence of SMR is expressed from left and right hemispheres (two vertical bars) under baseline conditions (base 1, 2 and 3), during learned voluntary production (vol. SMR), and during actual feedback training (F.B. SMR). In B data are shown also when subject is asked to tie and untie a shoe (tie shoe) and to merely think about this behavior (think tie). All values are based upon two minute recorded epochs subjected to band-pass analysis. Note increased production of SMR activity during both voluntary and feedback related performance and marked suppression during motor activity related to tying of shoe. Data at C show sample EEG traces from a well trained subject together with filter and relay response to SMR pattern. R indicates reward (light and tone).

found ourselves utilizing a behavioral procedure which was being explored in several areas under the general rubric of biofeedback. We had arrived there, however, by virtue of animal studies which suggested that this technique might be utilized to manipulate neural circuitry.

Our initial task was to identify an EEG rhythm homologous to the feline SMR and to develop an effective feedback reward system for use in studies of man. Recordings from the human sensorimotor cortex (central or rolandic area) disclosed low voltage rhythmic patterns ranging in frequency from 8–16 cps. Utilizing lights, tones and other rewards as feedback, we were able to provide specific reinforcement for the 12–16 cps. (SMR) component of sensorimotor cortex EEG activity (Fig. 3). Subjects reported the experience to be pleasant, associating it with a central state of focused attention, usually involving a previously experienced motor behavior. However, after several weeks of training, verbal responses were difficult to elicit; subjects merely stated that they were able to perform the required task without being able to label the associated subjective state.

THERAPEUTIC BENEFITS IN THE CASE OF EPILEPSY

Because most epileptics are adequately controlled by anticonvulsant medications and the use of biofeedback as a

therapy for this disorder represents a new and rather novel approach, the patients which we have worked with to date have all suffered from severe, uncontrolled seizure conditions. They represent a segment of epileptics who do not respond satisfactorily to anticonvulsant drugs. Accordingly, seizure rates were usually frequent in these patients and EEG abnormalities abundant. Twenty epileptic patients, ranging in age from 6 to 46 years, have been provided with SMR biofeedback training in our program to date. The first four were studied for 6–24 months in our initial experiment [21]. This study was concerned with a simple question: Does EEG biofeedback, involving reward for central cortical 12–15 Hz activity, reduce seizures in epileptics? Positive results were obtained with all of these patients, despite the fact that a wide variety of epileptic conditions were represented. The best results, however, were demonstrated in cases with primary motor seizures (i.e., grand mal and atonic seizures) as opposed to cognitive disorders. These findings were encouraging and consistent with our observations in the cat. However, because of the lack of appropriate control conditions it was impossible to specify which aspect of the biofeedback procedure was responsible. Critics pointed out that epileptics often show transient improvement with any change in treatment and the all powerful "placebo effect" was invoked once again.

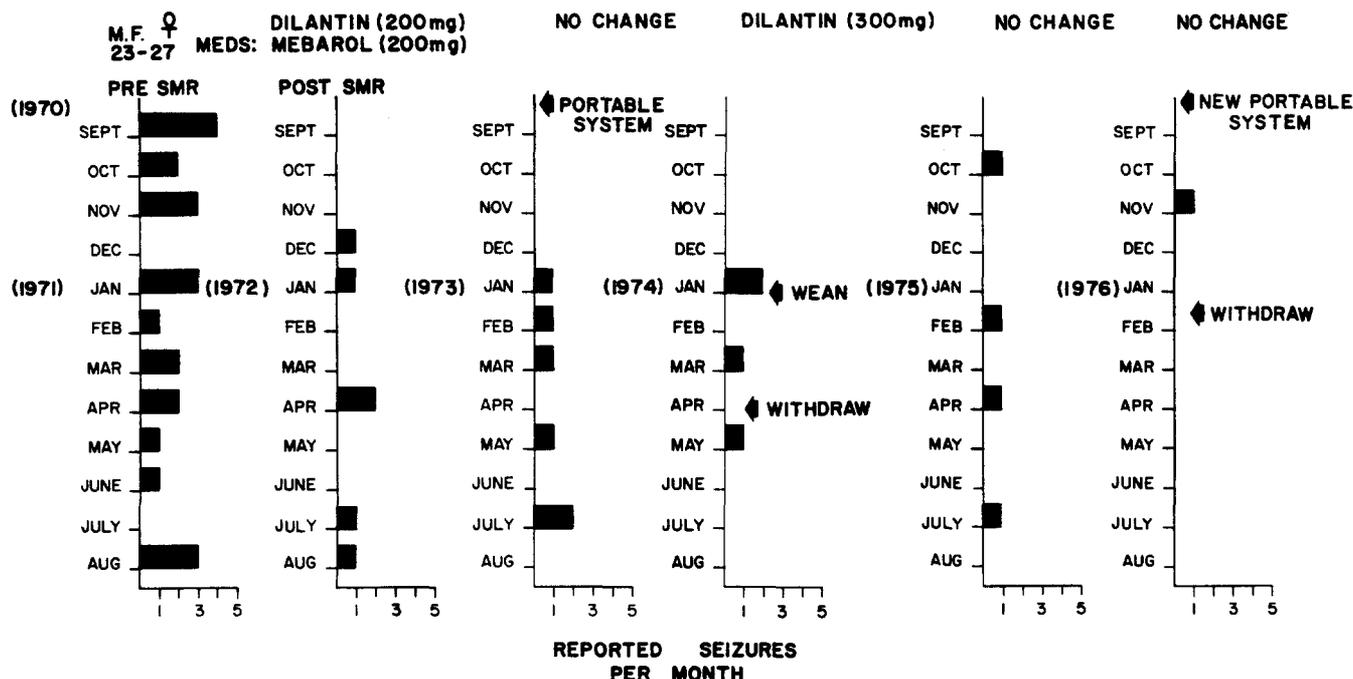


FIG. 4. Reported seizure incidence (generalized major motor) in patient M.F. from September 1970 (prior to SMR training) to August 1976 (after three years of training). Anticonvulsant medications were not changed until September of 1973, when the phenytoin dosage was increased slightly, as indicated. Patient was trained in the laboratory during first year, then with early model of portable home unit during subsequent 18-month period, and finally with advanced model during the fall of 1975. Patient was withdrawn from training for a 17-month period in 1974-1975, as indicated, and again in February 1976 (from Serman [21]).

This criticism was rather annoying, since we had only sought to explore the feedback approach in order to justify the more elaborate investigation desired. Considering the cost in time for both patient and investigator and the sophisticated equipment required this preliminary study was felt to be necessary, if not final. Yet, this criticism once again points out the potent modulating effect which behavioral manipulations can have on disease. What is the placebo effect? Surely it is not magic or divine intervention. It is merely another expression of the intrinsic plasticity of physiology mentioned earlier; evidence that what the brain has torn asunder, the brain can indeed put back together. This plasticity is mediated, no doubt, by the highest levels of CNS function, by "expectations" or "set" or even simply by "attention" on the part of the patient. If only we could harness this powerful medicine with formal behavioral methods (in contrast to its informal exploitation by the medical profession).

New studies were initiated in our laboratory which sought to determine a specific basis for the therapeutic effects observed. At the same time numerous other laboratories began to explore this approach to treatment and, with the initial data established, often with appropriate control procedures. Significant seizure reductions following SMR feedback training were reported by Finley *et al.* [8] and by Lubar and Bahler [14]. Kuhlman [13] chose to reward a more dominant central cortical frequency band (9-14 Hz) and utilized an initial period on non-contingent reward. Consistent seizure reduction was observed in three of five patients *only after contingent reward was initiated*. Similar methods and results were described by Wyler *et al.* [29], who provided reward for

higher frequencies in the central cortical EEG (14-26 Hz). To round out the list of reported studies, Quay and Grant [17], utilizing careful control conditions noted significant seizure reductions related exclusively to either 8-11 Hz or 12-15 Hz central EEG feedback reward. Additional positive findings from our own expanded studies have been reported in detail [24,27].

QUANTIFICATION OF THE EEG RESPONSE

We have found that a serious approach to this kind of research requires sophisticated design and equipment. Patients must be well documented prior to entry in experiments and should be followed for many years. For example, we are still monitoring our very first patient after more than five years (see summary in Fig. 4). The most difficult problems relate to quantitative evaluation of the operant response and objective analysis of clinical outcome. The EEG is a very unique response measure. As mentioned at the outset of this discussion, much progress has been made in understanding both the physiological substrates and behavioral significance of this signal. However, studies of the EEG suffer from the lack of a powerful quantification methodology. Brief samples of manually measured alpha (8-11 Hz) and theta (4-7 Hz) activity can be obtained because of relative stability and significant amplitude. It is disappointing, however, that meaningful functional correlations are yet to be established with such biofeedback studies of these "popular" rhythms [5,10].

A variety of techniques were utilized to evaluate acquisition of the SMR response in our laboratory. The functional

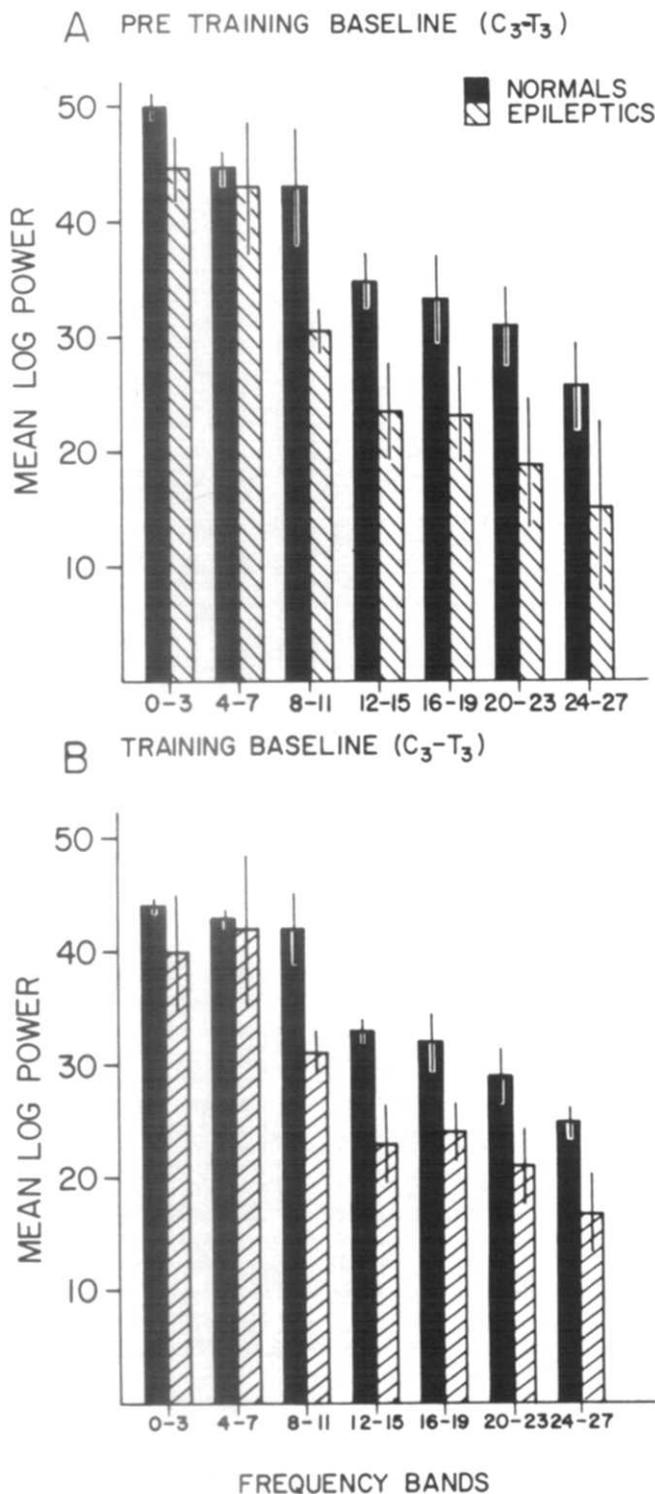


FIG. 5. Distribution of spectral density in seven 4 Hz frequency bands is compared here between epileptic and non-epileptic subject groups ($n=6$) from central cortical (C_3-T_3) data obtained during baseline and initial training sessions. Pre-training baseline activity was recorded during three separate 30 minute sessions in the training laboratory. Training baseline data were obtained from the first three 30 minute training sessions in the same laboratory. Note that mean log power in all of the higher frequency bands was deficient in epileptics as compared to non-epileptics.

parameters of the EEG which are most clearly understood today include frequency (cycles per second—when this remains stable over time a train of similar waveforms develops which is called a rhythm), voltage (or amplitude) and topography (anatomical distribution over the cerebral hemispheres). We have utilized both band-pass analysis and power-spectral analysis in order to investigate long-term reward contingent changes in the EEG. It should be stressed that the SMR differs from the more familiar alpha rhythm in terms of frequency (12–16 cps. as opposed to 8–13 cps.), topography (central cortex rather than occipital) and subjective correlates (focused vs floating mental state). Moreover, the human SMR is characteristically much lower in voltage and difficult to detect in the unfiltered EEG. Yet with precise detection methods, the most effective utilizing a tuned active filter and amplitude cutoff circuit (see [26]), and appropriate contingent reward, this rhythm can be reliably increased in approximately 80% of subjects provided with training.

Response to training can be seen in terms of increment in rewards obtained, but was best quantified with power-spectral analysis in more recent experiments. This computer approach to quantification provides an overall evaluation of the relative distribution of frequency and amplitude in a particular EEG sample. Recently developed programs provide also for the plotting of many successive EEG samples, quantification of the activity in multiple frequency bands, and comparison of these distributions among individuals and over time in the same individual [4, 7, 16]. With this methodology, baseline EEG characteristics have been compared in normal subjects, epileptics, and other clinical groups, including insomniacs and spinal injury patients [20,25]. The severe epileptics we have studied showed a significant reduction of higher frequency activity (8–27 Hz) during wakefulness when compared to non-epileptics (Fig. 5). Evaluation of sleep data indicated a specific deficiency in the 8–15 Hz range, corresponding to the so-called "spindle burst" of the sleep EEG. Many patients also showed abnormal low frequency activity when compared to non-epileptics.

PHYSIOLOGICAL BEHAVIOR MODIFICATION

The new experimental design we have utilized involved four phases of investigation carried out over a period of 12 months. These phases included: (1) a baseline data collection period, (2) initial laboratory training, (3) home practice, and (4) follow-up. The baseline period consisted of multiple laboratory EEG recordings during both wakefulness and sleep. Initial training in the laboratory was then provided to familiarize the patient with equipment and training procedures. Following this period the patient was issued a portable EEG training device adjusted to reward the presence of one central cortical (C_3-T_3) frequency band in the absence of a second frequency band. After three months of daily home practice, with recorded laboratory performance at two week intervals, baseline measures were again obtained and the reward contingencies for the two frequencies reversed. The patient was not advised of this reversal. The same home training and laboratory sampling procedure was followed for three more months with reversed reward contingencies and then a third set of baseline recordings was obtained and rewards reset to the original contingencies. This approach constituted an ABA design, with the patient as his/her own control.

This design and the outcome of power-spectral analysis are both illustrated in the summary of data from one of our

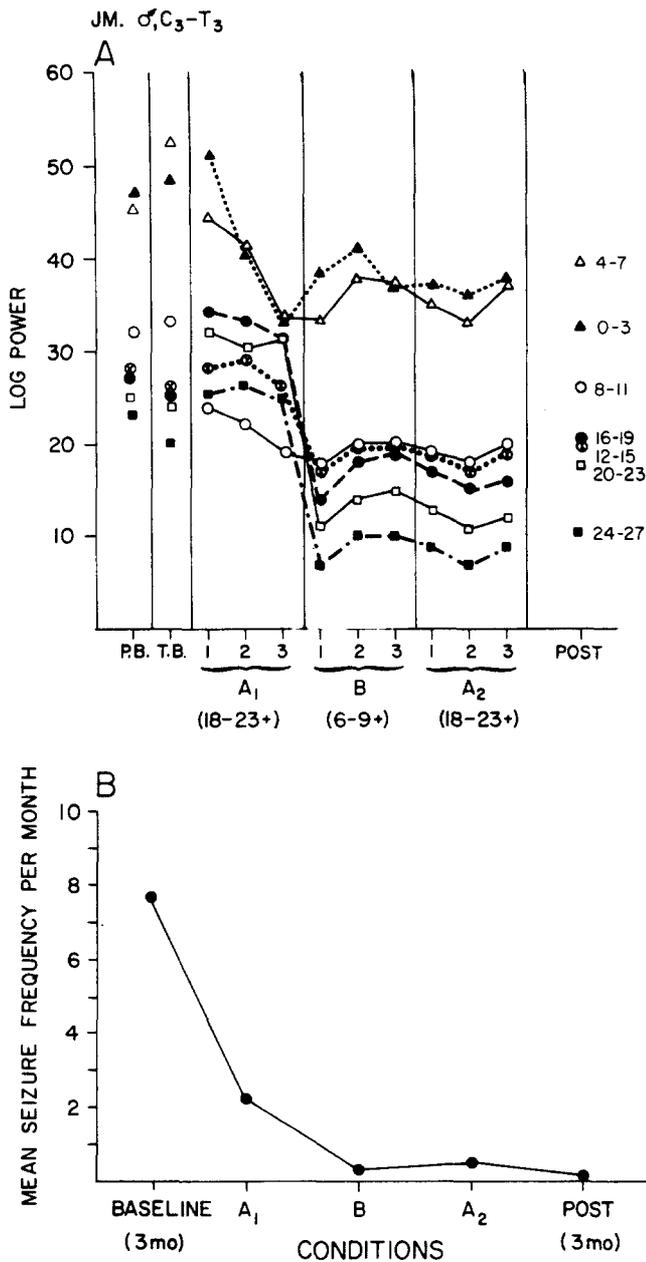


FIG. 6. Distribution of spectral density is shown here in an epileptic subject (J.M.) over all phases of our current double crossover experimental design for evaluation of EEG feedback effects, together with concurrent incidence of reported major motor seizures. See details in text.

patients shown in Fig. 6. Pre-training baseline measures (P.B.) are tabulated in the first column to the left and reflect the mean log-power in each of seven central cortical EEG frequency bands derived from three 30 minute baseline recordings. The next column shows similar mean log-power data for the first three 30 minute training sessions (training baseline, or T.B.). The subject then began the nine month ABA training design, working at home and coming to the laboratory at two-week intervals for recorded training sessions. The power-spectral values calculated at monthly

intervals during each phase of this design are displayed in successive columns with the corresponding frequency contingencies indicated at the bottom. Finally, a mean log-power value is shown for each frequency band from three 30 minute post-training sessions carried out 6-14 weeks after the training phase was completed (Post). A parallel tabulation of reported seizure incidence is shown below these EEG data.

Careful inspection of these complex data indicates a significant shift in the distribution of power in the EEG during the first two phases of training. In condition A₁, when high frequencies were rewarded and lower frequencies suppressed, the spectral density distribution shifted appropriately. With the initiation of condition B, where these contingencies were reversed, high frequencies dropped precipitously while lower frequencies tended to increase. Thus, the EEG showed a frequency response to the feedback training paradigm. Other data indicate that this response was topographically specific as well, since high frequencies responded most in frontal and central cortex and low frequency changes were essentially limited to central and occipital cortex.

Inspection of concurrent seizure incidence data shows that a significant decrease in reported seizures began during condition A₁, an observation consistent with previous findings [21]. To our surprise, however, seizures continued to decrease after the first reversal of EEG frequency contingencies. It was clear from these data that low frequency attenuation was sustained throughout the B and A₂ conditions, suggesting that this EEG dimension was associated with seizure reduction. Analysis of sleep EEG data provided further insight. Indeed, low frequency abnormal EEG discharge during sleep was essentially abolished after the condition A₁ training period, and higher frequency patterns (i.e., sleep spindles) were enhanced. However, these changes did not reverse with the B condition, as the waking EEG data indicated. We concluded from this analysis that the EEG was "normalized" during the A₁ condition and remained improved from that point on, seizure reduction being the primary reinforcer. It appears that in order to satisfy the training requirements in condition B, the patient merely attenuated high frequency amplitudes, without altering their incidence. This strategy was resistant to change, even after the original reward contingencies were re-established in the A₂ condition. Perhaps this was related to the fact that seizure activity had essentially ceased.

These data represent the beginning of a comprehensive, quantitative evaluation of EEG patterns associated with epilepsy in general and with feedback training in particular. They indicate that the electrical activity of the brain can be measured objectively so as to document and classify pathology. Moreover, EEG feedback training can modify both the electrical and clinical manifestations of that pathology. The EEG response dimension is complex, however, and must be understood and followed comprehensively, much like the behavioral domain in studies of behavior modification. That it is subject to manipulation, or plastic, however, seems certain.

The results of EEG biofeedback studies in epileptics reviewed here are most promising in this regard. It should be pointed out, however, that some types of seizure disorder do not respond well to this approach. Moreover, certain patients are unable to benefit from such a "voluntary" therapy. In these instances we believe that motivational factors related to established dependencies, life styles and secondary

gains interfere with successful exploitation of this method. The patient can be viewed as a totality of learned response

patterns, and our control of reinforcers must always be limited.

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