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Abstract:

(This reprinted article originally appeared in Pavlovian Journal of Biological Science, 1977, Vol 12 [3], 147-185. The following abstract of the original article appeared in record 1978-25642-001.) Currently, considerable research is being directed toward developing methodologies for controlling internal processes. An applied branch of the basic field of psychophysiology, known as biofeedback, has developed to fulfill clinical needs related to such control. Current scientific and popular literature abounds with numerous examples of how biofeedback is being used. For example, germinal studies by J. Kamiya (1962), and later work by J. J. Lynch and D. A. Paskewitz (1971), J. Beatty (1973), as well as many others, have shown that the EEG alpha rhythm (8-13 Hz) recorded from occipital regions of the human brain can be behaviorally manipulated when feedback or reward is provided for changing the density of this activity. Other researchers have provided evidence that theta activity (4-7 Hz) and the beta activity (greater than 14 Hz) can also be controlled by humans, and analogs of this activity have been conditioned in animals as well (E. E. Green et al, 1971). . . . (PsycINFO Database Record (c) 2008 APA, all rights reserved)

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ELECTROENCEPHALOGRAPHIC BIOFEEDBACK METHODOLOGY AND THE MANAGEMENT OF EPILEPSY

EDITORIAL COMMENT: The historical contribution that follows was published in Volume 12, Number 3, July-September 1977 issue of The Pavlovian Journal of Biological Science (pages 147-185). It uses a therapeutic technique, electroencephalographic biofeedback, that was very popular twenty years ago but had not been used often to treat epilepsy. Several new, mainly pharmacologic, treatments for epilepsy have emerged during the last twenty years, but intractable seizures are still encountered despite all current therapeutic efforts. Dr. Lubar's carefully developed complex technique yielded encouraging results and may justify further experimentation with biofeedback in seizure disorders.

Abstract

--Currently considerable research is being directed toward developing methodologies for controlling internal processes. An applied branch of the basic field of psychophysiology, known as biofeedback, has developed to fulfill clinical needs related to such control. Current scientific and popular literature abounds with numerous examples of how biofeedback is being used. For example, germinal studies by Kamiya (1962), and later work by Lynch and Paskewitz (1971),

Beatty (1973), as well as many others have shown that the EEG alpha rhythm (8 to 13 Hz) recorded from occipital regions of the human brain can be behaviorally manipulated when feedback or reward is provided for changing the density of this activity. Other researchers have provided evidence that theta activity (4 to 7 Hz) and the beta activity (greater than 14 Hz) can also be controlled by humans and analogs of this activity have been conditioned in animals as well (Green, Green and Walters, 1971). In addition to the work that has been carried out with the EEG, researchers such as Engle and Bleecker (1973) have indicated that it might be possible to control cardiac arrhythmias through biofeedback. Studies by Elder et al. (1973) have provided some hope that blood pressure in humans might also be conditioned. Also, considerable effort has been directed to the control of responses from single muscles with particular applied emphasis in neuromuscular rehabilitation, control of muscle tension for tension headaches and the management of migraine headaches through vasomotor conditioning (Brudny et al., 1974; Basmajian, 1963, 1971; Sargent et al., 1973).

THE BEHAVIORAL CONTROL of human EEG has been particularly interesting because it has been implied that perhaps some rhythms are associated with rather distinct mental states. For example, Walsh (1974) has correlated alpha with "neutral to pleasant" emotions and with undirected free flowing thoughts, defocused visual inattention, and relaxation. The theta rhythm is associated with even lower states of arousal, free association, perhaps day dreaming. The Greens at the Menninger Foundation believe that this rhythm is also important in the creative process. Delta rhythms are correlated with deep sleep and pathologically with coma, while at the opposite end of the frequency spectrum beta activity is correlated with problem solving, high levels of anxiety, and high levels of arousal. Because of these suggested correlations between different emotional and cognitive states EEG feedback has been used with varying degrees of success as an adjunct to psychotherapy and for achieving deep levels of relaxation.

Schwartz (1975) has very effectively argued that many different autonomic and electrophysiological responses which are highly correlated are involved in any particular altered state. So, for example, deep relaxation is associated with either alpha or theta activity but also low levels of forearm and frontalis EMG increased peripheral skin temperature, decreased respiration, perhaps decreased blood pressure. Basically this state is that which Gellhorn (1968) has called the "state of parasympathetic dominance."

It can be seen that the commonly studied brain rhythms, delta, theta, alpha, and beta are associated with rather broad classes of behavior. One particular type of activity however may be much more specific and have more clinical import. It has been recently shown that behavioral control of specific activity of 12 Hz to 15 Hz recorded from the scalp electrodes overlying the sensorimotor or Rolandic cortex might be of considerable significance for the management of human epileptic seizures (Serman and Friar, 1972; Serman, McDonald and Stone, 1974; Finley, Smith and Etherton, 1975; Seifert and Lubar, 1975; Lubar, 1975; and Lubar and Bahler, 1976). Unlike other brain rhythms that have been studied using biofeedback an electrophysiological and anatomical theory can be proposed to explain why this 12 Hz to 15 Hz activity, presently called the sensorimotor rhythm (SMR) might be effective for the management of epilepsy. Currently in the laboratories that are working in this area, more than forty patients have received intensive sensorimotor (SMR) biofeedback training. Many of these patients have achieved considerable control of their seizures as a result of these techniques. The remainder of this chapter will be

devoted to the question of why training of Rolandic rhythms might be effective for seizure reduction in terms of models of epilepsy. The different types of Rolandic rhythms will be considered in conjunction with the methodologies for detecting these types of EEG activity. Results will be presented for the work carried out in our laboratory for the past three years dealing with seizure control in twelve patients. The future of EEG feedback for the control of seizures will be discussed and the implications of this type of research.

Rationale for Using Biofeedback to Control Seizures

Epilepsy appears in many forms but basically it is a disorder of the nervous system characterized by paroxysmal phasic discharges of pools of neurons. Sometimes these discharges occur in portions of the brain concerned with motor functions in which case the seizures are manifested as Tonic-clonic (grand mal) convulsions or myoclonic (muscle twitching) seizures. Other seizures are associated with a loss of motor tonus and the patient falls (akinetic seizures). Still other seizures are associated with changes in sensory functions as well as changes in emotionality. These seizure types encompass the petit mal and psychomotor types. Some seizures are triggered by repetitive visual (photic seizures) or auditory stimuli (musicogenic seizures). According to Jovanovic (1974), all forms of epilepsy have in common that the threshold for paroxysmal discharges regardless of whether they are cortical, subcortical, sensory, motor, or centrencephalic has been lowered.

The decrease in seizure threshold can be the result of traumatic injury, genetics, anoxia associated with birth injury, chemical poisoning of the nervous system, tumors, and a variety of other cerebral insults. Jovanovic's model of epilepsy proposes that there are different CNS levels where natural barriers to paroxysmal or uncontrolled discharges exist. He proposes a cortical barrier, a barrier at the thalamic level, in the upper brain stem, the lower brain stem, and the spinal cord. In psychomotor epilepsy the temporal lobe and especially the hippocampal formation is also a possible barrier site. If all barriers are inoperative the patient experiences continual seizures known as status epilepticus. In the case of grand mal seizures status, if not broken, can be fatal. For petit mal and psychomotor seizures status is characterized by a prolonged state of confusion, disorientation, and, in some cases, unconsciousness. If one or two of the barriers are disrupted the patient might exhibit seizure activity in the EEG consisting of slow waves, and sometimes occasional spikes. However, he or she may not be aware of anything unusual in terms of disorientation, dizziness, confusion, emotional changes, or sensorimotor effects. If additional barriers are disrupted, the slow wave discharges eventually give rise to spikes and polyspike and wave complexes which are associated with overt seizures of the many types described above.

The question now arises of what keeps the non-epileptic from experiencing breakdowns of these CNS barriers for seizure activity. The answer probably lies in the large number of inhibitory control systems which exist in the brain. Many of these systems are comprised of pathways that are mediated by inhibitory neurotransmitters, such as gamma amino butyric acid (GABA) which through IPSP's maintain enough inhibition to prevent seizure discharges. Some of this inhibitory activity is expressed in terms of rather specific sinusoidal brain rhythms which are associated with inhibition of movement, quiet states or specific stages of sleep.

These "idling" cortical rhythms can be seen in many different species. In 1963, M. Brazier described 13 Hz to 14 Hz activity that can be recorded from the sensorimotor cortex in cats. Another rhythm prescribed by Gastaut in 1952 is called the "somato-motor rhythm" (rhythm en arceau) or mu rhythm. Gastaut suggested that this represented inhibition of sensory and motor representations at the level of the Rolandic cortex (Gastaut et al., 1964, 1965). According to Andersen and Andersson (1968) who have developed an elegant model of the EEG all sinusoidal cortical rhythms represent a type of "cortical idling" associated mentally or physically with inactivity. This is why the alpha rhythm which is dominant in the human is generally correlated with relaxed or defocused mental states. Theta rhythm (4 Hz to 7 Hz) is perhaps even more so. In contrast when the brain is actively involved in data processing and problem solving or with high levels of emotionally desynchronized low amplitude beta activity (> 14 Hz) is most commonly seen.

There has been some disagreement and confusion in the literature as to how many inhibitory rhythms exist that can be recorded from the sensorimotor cortex or overlying scalp. The mu rhythm sometimes called the wicket rhythm because of its appearance when the EEG is run at certain paper speeds is of 9 Hz and can be recorded specifically from the Rolandic cortex and overlying scalp. Mu has the interesting property that it is usually blocked by clenching the contralateral hand which overcomes this type of cortical inhibition, if in fact this is what mu represents. In cats Roth et al. (1967), Wyrwicka and Sterman (1968), and Sterman et al. (1969) described a sensorimotor rhythm (SMR) of 12 Hz to 16 Hz which can be recorded in very specific locations around the cruciate sulcus.

This SMR activity has been operantly conditioned by these investigators and its acquisition is correlated with behavioral immobility and a marked increased resistance to monomethylhydrazine induced epileptiform seizures. Neurophysiologically, SMR is correlated with inhibitory activity in subcortical motor structures and pathways (Harper and Sterman, 1972). For example, a correlate of SMR activity occurs simultaneously in the ventroposterolateral thalamic nucleus (VPL). Both cortical SMR and VPL activity are disrupted simultaneously during phasic motor activity (Howe and Sterman, 1972). During SMR bursts unit discharges from the red nucleus which receives inhibitory inputs from the cerebellum as well as units in VPL are partially suppressed.

Anatomically, it has been well established by Eccles et al. (1967) that the entire outflow of the cerebellum which is comprised of the axons of Purkinje cells is inhibitory. A primary role of the cerebellum is through its inhibition of the modulation of centrifugal pathways from the motor cortex and the extrapyramidal nuclei. Prominent pathways from the cerebellum are the dentatorubral and dentatothalamic tracts. These fibers exit from the dentate nucleus of the cerebellum and project via the brachium conjunctivum to the magnocellular portion of the red nucleus in the rostral midbrain. From there rubrothalamic fibers are relayed to the ventral lateral and ventral anterior nuclei of the thalamus and thence to the somatosensory and motor cortex.

Although it is not yet clear perhaps the SMR, at least in the cat and perhaps in other species, is generated by these pathways. One compelling piece of evidence to support this hypothesis comes from the work of Dr. Irving S. Cooper at St. Barnabas Hospital. Cooper has been able to control seizures in a certain proportion of intractable epileptics following implantation of stimulating

electrodes in the cerebellar cortex. Electrical impulses are delivered continuously over a period of several months by a portable electrostimulator implanted in the cerebellar cortex. Perhaps the reason this works for some patients is because it sets up SMR or other types of inhibitory activity in the dentatorubrothalamic pathway which is perhaps deficient in the severe epileptic as a result of the disease process.

Recent findings that SMR conditioning in humans can help to control severe seizures make considerable sense in light of Cooper's results. Perhaps in these patients traces of SMR which occur as an attempt to naturally block seizures is being detected and reinforced by the feedback paradigm. By increasing the amount of this activity, at least according to the Jovanovic model of epilepsy, seizure thresholds are increased until eventually the control which had been lost is again exerted.

One of the current areas of confusion, however, has to do with the definition of the sensorimotor (SMR) rhythm. All of the electrophysiological subcortical recording discussed so far has been carried out in nonhumans, i.e., in cats and other primates. Sterman has made the assumption that sensorimotor rhythm of somewhere between 12 Hz and 15 Hz (+/- Hz) also exists in the human and can be recorded from the scalp overlying the Rolandic cortex. He argues that this rhythm is distinctly different from the mu rhythm of 9 Hz. At the present time there are no definitive studies of the effect of mu rhythm conditioning in humans. If it were found that mu rhythm could be conditioned and that enhancing this specific 9 Hz activity would result in seizure reductions equal to or greater than that produced with SMR conditioning then the whole question of whether distinctly different mechanisms are involved in the generation of these two rhythms would be of paramount importance. If mu conditioning had no particular effect of seizure reduction it would indicate that this type of Rolandic activity is not involved in seizure inhibition or threshold changes.

One of the reasons, however, at the present time that it is not possible to unequivocally condition the mu rhythm is because of alpha activity. Alpha rhythm is usually associated with the occipital lobe and occurs most commonly when the eyes are closed. But in some individuals alpha spreads, and it can be picked up from central leads and sometimes can even be recorded from the frontal area. In epileptics the alpha rhythm is usually slower than that of nonepileptics. Whereas normal alpha lies in the 8 Hz to 13 Hz range or EEG band, in epileptics alpha is often seen between 7 Hz and 10 or 11 Hz. The reasons for this are not clear. It might be due to the disease process which produces general slowing of many brain rhythms as evidenced by the slow waves that represent seizure activity. It may also be due to the action of the various anti-convulsant medications. Thus, much of the alpha in epileptics center around 9 Hz, which is exactly the frequency of the mu rhythm.

We do not know whether mu rhythm, SMR, beta, etc., are also proportionally slower in epileptics. But if the assumption is made for the time being that mu rhythm remains around 9 Hz and the alpha is also centered about 9 Hz then both types of activity though generated by different neuronal mechanisms can be picked up at the same time from the same electrode. It can be seen how difficult it would be to provide feedback reinforcement for mu that is not contaminated by alpha.

There are ways in which this problem might be solved and will be dealt with later in the methodology section. Also one investigator, Rouse (1975, Meetings of the Biofeedback Research Society, Monterey, California) has reported seizure reduction following alpha conditioning in epileptics. This could have been due to coincidentally reinforcing mu rhythm which might have been beneficial. Also this result could have occurred if Rouse's filter and detection system was not precise enough to eliminate activity in the low range of the SMR frequency band, in which case conditioning part of the SMR activity might have caused the seizure reduction effects. At the present time then there is a considerable need to sharpen our techniques and determine specifically which rhythms are involved in seizure control and which are irrelevant. As we will see this is a most challenging and difficult task.

Methodologies for Detecting and Training Rolandic Activity

Detection of EEG Activity

At the present time there are two basic methods for detecting EEG activity which can be paired with appropriate feedback. One is the method of zero crossing, sometimes referred to as a digital analysis, the other is an analog analysis based on highly tuned active bandpass filters. Neither methodology is completely satisfactory. Let us consider first the digital or zero crossing method. The assumption is made that the EEG wave forms are symmetrical about zero voltage line. The period of each wave which is the inverse of the frequency is measured. Specifically this is by circuitry which measures the time interval between positive going voltages that cross zero line and represent a single cycle of activity. These periods can then be converted into amplitudes by period to amplitude converters such as the ORTEC Corporation 4672 Instantaneous Time Frequency Meter. The output of this or similar device can then be passed through a window discriminator such as the ORTEC 730L Single Channel analyzer. The latter is set for the desired band pass, which in the case of our SMR studies is 12 Hz to 15 Hz. The instantaneous period to amplitude converter converts each period into a voltage for each cycle of EEG activity; the window discriminator is set precisely to respond only to activity in the desired band pass. This method has the advantage that if all the assumptions are met only 12 Hz to 15 Hz will be reinforced once the output of this circuitry is keyed to various types of feedback. It can be set so precisely that activity of 11.99 cycles or 15.01 cycles cannot be reinforced.

However, the disadvantages of this type of detection system probably outweighs its advantages. For one thing EEG activity is not symmetrical about a zero line. Very often fast activity will ride on slower waves. In the case of SMR much of the 12 Hz to 15 Hz activity rides on top of a carrier wave of between four and eight cycles. A zero crossing analysis, of the type described will only respond to the slow waves which cross the zero line and hence miss a large proportion of the total 12 Hz to 15 Hz activity. This will eventuate in very low levels of SMR detection and partial reinforcement. It is very unlikely that patients would be able to benefit from this type of frequency analysis.

One solution to the problem is to differentiate the raw EEG. Then every absolute and relative maximum and minimum in the raw EEG will become a zero point in the differentiated signal. The differentiated signal can then be passed through a period of amplitude converter and window discriminator. This will increase considerably the amount of detected 12 Hz to 15 Hz activity and

will result in a higher level of reinforcement. At the present time a system employing differentiation and digital analysis has not been reported on for SMR work, so it is not possible to assess how effective it would be.

Here again, even with differentiation of the EEG, there is an important disadvantage. If fast activity is riding on slow waves the differentiator will favor the higher frequencies. This is shown diagrammatically in Figure 1.

One reason why this may be a serious drawback is because the severely epileptic EEG often contains traces of 12 Hz to 15 Hz activity which do ride on slow epileptogenic activity of between 4 Hz and 8 Hz. We have found that it is important to provide the patient with information about this slow wave activity either in the form of additional feedback or through inhibit circuits to be described later. Differentiation obliterates and ignores the slow waves and when fast activity is being reinforced, there are some serious implications. What for example would this mean if, from the psychological viewpoint, the slow activity is more discriminable as an "altered state" than the fast activity? Differentiation would provide partial reinforcement for both the slow waves as well as for the faster Rolandic rhythms. The danger is that the patient might learn to increase epileptogenic activity as well as SMR. This in turn might negate the effect of sensorimotor rhythm training, and could result in increased seizure activity. Clearly then a purely digital analysis even with differentiation is not the optimal way to carry out epilepsy management using biofeedback.

Analog Methods

Analog methods depend upon passing the raw EEG through a highly tuned active bandpass filter. At the present time there are several filter designs, Butterworth, Chebyshev and Elliptic. Filters have the advantage that it does not make any difference whether the input is symmetrical about a zero line or consists of faster frequencies riding on slower frequencies. The ideal filter would respond only to waves within the bandpass. However, this ideal is only approximated by present technology.

Suppose we consider a bandpass filter of 12 Hz to 14 Hz with a center frequency of 13 Hz. Such a filter will also respond to harmonic frequencies of 7.5 Hz and 26 Hz. Good filter design necessitates that responses at harmonics be very attenuated--ideally by more than 40 db as compared with the response at the center frequency. The rolloff of bandpass filters should be a minimum of 24 db within the first octave; but ideally a design which allows for greater than 40 db is better. The ones that we employ have a rolloff of 80 db within the first octave and respond to harmonics at minus 40 db compared with the center frequency. This means that in order for a first harmonic to excite the filter as much as the center frequency it would have to be several thousand times greater in amplitude which is not the case with EEG signals.

Another complication is that active bandpass filters do ring when transients with a rapid rise time are passed through them. Such transients can be caused by epileptogenic spikes, gross movements, and artifacts produced by poor electrode contact with the patient. The amount of filter ringing, unfortunately depends upon how sharp the filters are made. A filter with 100 db rolloff in the first octave and of a narrow bandpass will be much more susceptible to ringing than

a filter of 24 db per octave rolloff and a broader bandpass. The ringing depends upon a quantity known as the "Q" of the filter which is equal to the center frequency divided by the bandpass. The narrower the bandpass and the higher the center frequency the more the filter will ring.

The significance of filter ringing is that one cycle of SMR activity might give rise to anywhere from 4 to 10 cycles of filter activity. If the criterion for feedback is a sustained filter output for a selected period of time this could mean that a single instance of SMR activity in the raw signal might be sufficient to provide feedback. It would therefore be erroneous to say that the feedback was based on raw EEG activity of a preset minimal duration when in fact the feedback is actually based on a filter which rings for a period which exceeds this chosen criterion. At the present time there is no filter in existence or perhaps even theoretically possible of the active bandpass variety which will put out one cycle of filter activity for one cycle of EEG activity within its bandpass. Filters of the elliptic design are superior to the other types in this regard. It is these filters that we have used in our work as contrasted to the Chebyshev and Butterworth types that have been used by others employing SMR methodology. The elliptic filter that we employ consists of ganged low pass and high pass filters each of 7 poles. This provides 14 poles of discrimination. Our elliptic filter is flat within the bandpass and falls off very rapidly to a floor of 80 db. The response of this filter to mixed frequencies is shown in Figure 2.

Feedback is keyed to our analog detection system by allowing the feedback to remain on in the form of a tone or light as long as the filter output is above a preset amplitude threshold.

Hybrid Analog and Digital Systems

In order to circumvent the disadvantages of both systems described previously we have employed a hybrid system. The raw EEG is split several times and passed through different and detection circuits. We'll describe first the system that is used for SMR analysis. The main signal is first passed through a clipping circuit. The purpose of the clipping circuit is to reduce filter ringing produced by large transients, large slow waves, and gross body movements.

The design of such a clipper circuit is crucially important. ([n1](#)) The clipper must not reduce high amplitude waves to wave forms that have been truncated or squared. Square waves also considerably increase filter ringing. Our clipping circuit reduces large amplitude waves to small waves with rounded wave forms and in contrast to earlier clipping circuit designs it reduces filter ringing considerably. The use of such a clipper is totally legitimate since we are interested in SMR activity which lies between 1 microvolt and 6 microvolts and rarely exceeds 10 microvolts and do not want the detection of this activity contaminated by large amplitude activity which will produce filter ringing. After clipping the raw EEG it is passed through the elliptic filter and then a zero crossing analysis is performed on the output of the filter. This is completely satisfactory because the output of the filter is symmetrical around the zero line. This digital analysis now imposes on the filter curve very sharp cut-off skirts and allows for extremely precise detection within the 12 Hz to 15 Hz bandpass.

Feedback is keyed to individual instances of filter activity between 12 Hz to 15 Hz or sustained bursts that exceed 0.5 second. Although it is true that an 0.5 second burst of activity is based on the filter response this sustained activity has to be above a present amplitude level. This is the

best way we know of trying to approximate the ideal of one wave of raw EEG activity within the bandpass producing one wave of filter activity.

Now consider some of the problems that still remain to be solved. What happens if the Rolandic or SMR activity is riding on epileptogenic slow waves? In order to separate one from the other a second detection circuit is used. As mentioned previously the raw signal is split and part of it is sent to the SMR detection system. A second portion of this raw signal is passed through a 4 Hz to 8 Hz 24 db/octave bandpass filter. If the amplitude of this filter output exceeds a preset criterion, usually corresponding to approximately 30 microvolts in the raw signal, this output via digital logic circuits blocks feedback for SMR. Such a system is called an inhibit circuit. The operation of the various inhibit circuits used in our system is shown in Figure 3.

As a result of this inhibit circuit it is impossible for the patient to receive SMR feedback if he or she is simultaneously producing slow waves, epileptogenic spikes, or gross body movements. The slow wave activity is keyed to a second feedback stimulus in the form of a large green light which the patient is told must remain on in order to receive SMR feedback. Hence, the patient knows when abnormal EEG activity or other artifacts are blocking the feedback.

A third detection system has been recently added which has resulted in an increase in the rate of SMR acquisition and a further increase of the precision of the detection circuitry. This is an inhibit circuit based on EMG activity recorded from the EEG electrodes. The raw signal is split a third time and passed through a 30 Hz to 300 Hz filter and amplitude discriminator. EMG activity produced by high levels of anxiety or movement activates this circuit and blocks SMR feedback. This circuit was instituted based on the neurophysiological model that SMR is associated with decreased phasic motor activity. After monitoring EMG activity in several hyperkinetic children it was found that there was a strong negative correlation ($r = -.87$) between EMG amplitude and SMR density (Lubar and Shouse, 1976).

It is now suggested that relaxation training might be a good way to prime a patient for producing SMR. However, rather than doing this we have developed the EMG inhibit circuit so that the patient was required to be relaxed in order to receive SMR feedback. The use of two inhibit circuits now makes it possible to eliminate artifacts produced by movement, slow waves, epileptogenic activity, spikes, and high levels of gross scalp EMG (Figure 3).

The only remaining problems that have to be dealt with are the question of theta rhythms, alpha rhythms, and beta rhythms. As mentioned earlier in most epileptics, alpha activity is slower--between 7 Hz and 10 Hz. Because the inhibit circuit based on four to eight cycles activity is broadly tuned, it will also respond to 9 and 10 Hz activity. Especially since alpha activity is generally of much higher amplitude than SMR, alpha activates the slow wave detection circuit and it is very rare that alpha activity is able to eventuate in SMR feedback. We also have evidence to indicate that as SMR is acquired centrally, occipital alpha decreases.

Kaplan has suggested that beta activity may be effective for SMR conditioning. An EEG spectrum consisting of high frequency low voltage activity is in a sense a "mirror image" of the epileptogenic EEG consisting of lower frequencies and higher voltages. Often after a patient has had one or several seizures there is a decrease in amplitude and an increase in frequency

indicating normalization has temporarily taken place. It is therefore a legitimate question whether conditioning of beta activity might not also be of benefit to epileptics. This has not been adequately answered at the present time, but is being investigated by Sterman's laboratory. It is important to point out if broad band beta detecting filters are used and if we define beta activity as 16 Hz and above then these filters might respond to 14 to 15 cycle Hz activity. This overlaps the SMR range, and unless specific inhibitory circuits are used for beta as well as for slow waves, alpha and other artifacts it is not possible to determine whether SMR and SMR alone is the only frequency that is beneficial for controlling seizures. The possibility that beta activity might also be beneficial is still an open question.

Other Detection Methods

The discussion up to this point has clarified the advantages and disadvantages of the analog, zero crossing, and hybrid analysis systems. Is there any way to design a fool-proof bandpass system for SMR detection? A more perfect solution to the problem could be based on Fourier analysis of the EEG. Fourier or spectral analysis has been used by both Sterman's group and ours to determine whether training eventuates in increases in SMR band activity and a simultaneous decrease in slow wave or epileptogenic activity. Both Sterman's research and ours have provided a number of illustrative cases in which this has occurred. Figure 4 shows a clear example of this. Over sessions for this patient a peak formed at 13 Hz as the SMR became a dominant frequency. There also was some decrease in the amount of slow wave activity. In some patients slow wave activity does not diminish significantly but SMR activity does increase. In some patients it is not possible to obtain clear evidence of positive spectral changes yet seizure reductions still occur. The reason for this is not clear.

An ideal detection system could be based on Fourier analysis but would involve a large computer or at least very elaborate electronics. Such a system would measure the relative power of the activity in the SMR band and would provide reinforcement on a second by second analysis for such band power each time it exceeds a preset criterion. Such a circuit could also provide inhibitory feedback for spectral power between 0 Hz and 3 Hz representing body movements when it exceeded a preset criterion. Another circuit could be keyed to the 4 to 8 Hz power spectrum representing epileptogenic activity. Additional inhibitory circuits could even be keyed to the alpha band. This type of analysis has the advantage that it is completely independent of filters and problems of filter ringing. It has a disadvantage that it requires time for the EEG activity to be analyzed in order to be statistically valid. A one second epoch may or may not be enough time for such an analysis. Feedback keyed to epochs of greater than five seconds would suffer from the problem of partial reinforcement or worse, reinforcement which is not really contingent on the activity. Such an analysis system has not been reported on by any investigator at the present time and is proposed as a step for future endeavor. Such a proposed method would help to clarify considerably exactly which frequency band is most important for the control of seizures.

Studies of Epileptic Patients

We have worked with a total of twelve patients for a period ranging up to thirty months. Additional patients have recently entered our program. Our patients were chosen in conjunction

with a consulting neurologist on the basis that their seizures were severe, uncontrolled by high levels of anticonvulsant medication, and were very frequent. Furthermore, and even more challenging, some of our patients have additional incapacitating problems including retardation, schizophrenia, and strong dependencies developed with parents or family members as a result of their extreme levels of disability.

Our patient population represents a cross section of the epilepsies, including grand mal, akinetic, myoclonic, psychomotor, and mixed seizure types, both generalized and focal. We specifically did not include any pure petit mal patients because these seizures are so frequent that they're impossible to record accurately. We also do not have any patients with photoconvulsive seizures or auditory induced seizures. Unlike other investigators, a large proportion of our population consists of psychomotor epileptics which represents a major group of epileptics and perhaps therefore are most important clinically to work with.

Our sample ranges in age from twelve to thirty and consists of seven males and five females. Throughout the course of their treatment very strict adherence to certain regulations was required. First, the patient, their parents, and teachers had to report every seizure that occurred during waking hours and as many nocturnal seizures as feasible. In some of the patients it was a further requirement that the seizures be rated on a numerical scale according to their intensity and/or duration. If several different seizure types occurred in the same patient each type needed to be categorized. Periodically blood tests are performed to make sure that the patients comply with their anticonvulsant medication requirements. The patients were told to take their medications at exactly the same time each day and not to change the medication under any circumstances with the exception of planned reductions that were instituted in those cases that were successful in reducing their seizures. This rule was violated in rare instances, and these violations were noted in the patients' records.

We explained to each person before they began their training that the treatment was experimental, that no guarantee could be made as to its effectiveness, and that it would be provided without cost to them. They would however, be required to come in for three sessions per week, and not to miss any training sessions unless there was an emergency, illness, or for vacations which were planned in advance. We told the parents and the patients that they would remain in treatment until either: (A) it was clear that it was not effective, which would take a minimum of six months to determine, and (B) if the treatments were effective, they would remain until their seizure level had reached an asymptote and then after consultation with the referring neurologist medication reductions would be considered. We explained that our goal was to try to reduce medications, and seizure levels until a final asymptote occurred, at which time the patient would be slowly weaned from the treatment regimen. The weaning would consist of having each epileptic come first for three sessions a week, then twice a week, once a week, once every two weeks and, finally, monthly. In some cases treatment was terminated entirely. We also requested permission to carry out a follow-up study based on seizure records for as long as necessary after termination of the treatment even if it required several years.

Training Procedures

The training sessions were conducted initially three times a week and each lasted for forty minutes. Prior to training, periodically during training and after termination of training a clinical EEG was taken. During this clinical EEG numerous recordings were obtained on FM tape for spectral analysis.

Before the first training session patients were taken to the laboratory and shown the equipment that would be used. Baseline measures were made of their EEG at that time. During an initial habituation session patients were just told to relax in a reclining chair for the entire forty minute period with their eyes open. No feedback was provided. We recorded with bipolar bilateral electrodes over C3-T3 and C4-T4 according to the international system and referred the signal to one of the ear lobes.

Following the habituation session training sessions were initiated. During each training session the first five minutes served as a habituation period in order to obtain a baseline for the daily EEG measurements. Baseline measurements were taken from the same hemisphere on a particular day, but the order of the hemisphere trained first alternated with each succeeding session.

Feedback indicating the presence of 12 Hz to 15 Hz activity representing SMR consisted of ten colored lamps that lit sequentially whenever activity in this EEG band attained a preset criterion based on duration and amplitude considerations. A tone produced by an 80 db sonalert could also be used for additional feedback indicating criteria had been met. For some of the younger patients the lighting of each tenth lamp in the sequence resulted in a slide being automatically projected onto a screen for seven seconds. While the slide was projecting additional tone feedback indicated that more criteria were being met. After the slide had been terminated a new slide could only be introduced after ten more successive criteria had been achieved. Sometimes the patients found it desirable to know how close they came to reaching a particular criterion. Since the criteria were based on amplitude and duration for example 6 microvolts of SMR activity being sustained for greater than 0.5 seconds it was desirable to provide additional feedback for SMR activity that did not meet this criterion. To do this each individual instance of SMR detected by the digital analysis that was superimposed on the filter output was keyed to a second tone. The patient could hear a series of pulsating tones of one frequency indicating individual instances of SMR filter activity and a second tone or light which told them when they had achieved the specific 0.5 second criterion. This recent addition, to the system produced a marked increase in the rate at which the task was learned.

Slow waves, epileptiform spikes, or gross movements were signaled to the patient by the second type of feedback described previously. This is a large pair of green lights which turned off whenever these activities occurred. The patient was told to keep the light on and that SMR would only occur when the light was on: Recently a red warning light has been added to the system to tell patients when their EMG was sufficiently great to throw the second inhibit circuit described previously.

Each training session consisted of the following sequence of events. Five minutes of baseline recording without feedback, fifteen minutes of contingent feedback presented for frequency, duration and amplitude criteria from the same hemisphere as the initial baseline, fifteen minutes

of training on the contralateral hemisphere and then a five minute final baseline reading obtained from the same hemisphere from which the initial baseline was taken. On a given day then the training sequence might be a right side baseline, right side feedback, left side feedback, right side baseline. This was followed in the next session by the sequence left baseline, left feedback, right feedback, left baseline. Since the number of training sessions employed were very large all data were averaged over several sessions and the number of left starting sessions and right starting sessions were virtually equal.

Effects of SMR Conditioning on Seizures

Significant seizure reductions were obtained in most of our patients. The single exception was a male aged twelve who was in training for a period of seven months. This, our youngest patient, experienced a mixed seizure type consisting of grand mal and myoclonic seizures. The case was complicated by mild mental retardation, severe scoliosis of the back which required orthopedic treatment, and hyperplasia of the gums, due to high levels of dilantin. Another factor was that this child had to be brought a total of 250 miles per week from a distant town in order to receive his treatment.

All of the other eleven cases showed moderate to marked degrees of improvement during training. This improvement has been maintained in cases being weaned from treatment so far. The other patients also showed varying positive degrees of change with regard to their EEG (Figures 5 and 6). Figure 5 shows an EEG from a fourteen-year-old female (K.S.) and Figure 6 EEG's from cases G.B. (female, twenty-nine years) and G.M. (male, nineteen years). The most noticeable changes in these records are the decreased slow wave and polyspike complexes in K.S. who also experienced a myoclonic seizure during the recording. Note in the records for all three cases that following several months of training there is a decrease in slow wave amplitudes and an increase in EEG frequency.

Seizure records for several cases are presented in Figures 7 and 8. These cases have been described in detail in our previous reports (Seifert and Lubar, 1975; Lubar, 1975; Lubar and Bahler, 1976). It can be seen that in the case of two of our male adolescent patients both of whom parenthetically had schizophrenic symptoms we were able to obtain seizure free periods. These are M.T. and C.D. In both of these cases we were assured by both parents and teachers that in fact these seizure free periods were real. M.T. voluntarily left treatment because he moved to another town, and has been doing very well for the past year while out of treatment having relatively few seizures. Case C.D.'s treatment was terminated for the present so that more attention could be devoted to his psychiatric problems. This patient is now in a total care facility but experiencing very few seizures of either the grand mal or psychomotor type.

One of our cases, a female of age twenty-nine (G.B.) has not shown any decrease in the number of seizures, but a marked decrease in their severity (Figure 8). Initially, her seizures lasted from one to five minutes, and consisted of psychomotor absences and black-outs in which she lost complete "contact with the world." Now, she's not sure that she is having seizures at all. Although she has the same number of occurrences each month as previously, they consist of periods of from one to several seconds of dizziness and partial disorientation, during which there is a partial loss of consciousness but she is still aware of her environment. An attempt to show

this change more emphatically was undertaken late in the treatment phase. After 400 days of data collection, it was decided to try graphing a seizure index for this patient. The seizure index equalled the intensity of each seizure multiplied by a rating number 1, 2, or 3. Seizures rated 3 lasted for one minute or longer, seizures rated 2 lasted from five seconds to one minute, and seizures rated 1 lasted less than five seconds or a loss of consciousness did not occur. It can be seen that while G.B. is being weaned from the treatment, most of her seizures are very mild.

This case illustrates the Jovanovic model discussed previously. What we think is happening is that as the training has proceeded more levels of inhibition have been built up until what were at first clinical seizures are now becoming subclinical seizures. Her EEG still shows two marked dominant rhythms, a slow wave pattern of 7.5 Hz and beta activity which initially centered around 23 Hz, but now has been reduced to 17 Hz which is close to the SMR range. Occasionally SMR activity is seen in her raw EEG and there are days in which the amount of slow wave activity is decreased considerably. It is our anticipation that this patient might be eventually capable of reducing her seizure activity to a subclinical level. Whether it will be possible to fully normalize the EEG with enough training we do not know.

In many of the cases illustrated drug reductions were possible. These patients had originally been maintained on very high levels of medication, because this was the only way their seizures could be controlled. Now, after varying degrees of training, drug reductions have been undertaken which did not result in increases and in some cases further decreases in seizure levels.

One interesting case in point is patient D.K. This patient had suffered from grand mal seizures and initially had one to two of these per month plus fifteen to twenty psychomotor seizures. After eight and one half months the psychomotor seizures were gone and she experienced less than one grand mal seizure per month. The grand mal seizures were also reduced in severity. At that point it was decided to change her medication of 300 mg dilantin per day to 200 mg of Tegretol. As shown in her seizure graph the seizures fell immediately to nearly zero and have remained there for almost one year. We feel that although this is a complex case to interpret, we may have "primed" this patient for a new medication which has allowed her to achieve nearly full control of all of her seizures

Some of our patients experienced varying degrees of mental retardation. The most striking case was that of S.R., an akinetic case. Each time a seizure occurred she would suddenly fall forward without any protective motor reflexes and hit her head. This resulted in considerable facial disfigurement as well as possible additional brain damage. At the time we began working with this patient her I.Q. was only about 40. She could read a few words, write her name, and recognize number symbols. We have been working with this patient since May of 1974 and during that time have been successful in reducing the number of her seizures by nearly 50 percent, as well as their severity sufficiently, so that now at times she is able to catch herself when she begins to fall forward. She was weaned from treatment during the summer of 1975 for nearly three months during which time her seizure level remained decreased. But she is now coming in for booster sessions every two to three weeks. It is felt that in this particular case it:: may never be possible to wean her totally from treatment because of the cognitive difficulties in being able to deal with the complexity of the biofeedback task. But it may be possible with

continued wide-spaced treatments to maintain this patient at a lower seizure level than she had been initially.

Other cases, as shown from their graphs, have been weaned from treatment. Some of them are coming in once every two weeks, some of them once a month, and others are still being treated once weekly. In most cases the weaning has been highly successful provided it is carded out slowly.

Other Measures of Acquisition

Besides seizures other measures are taken to show the degree of learning that had taken place. One of these measures which is illustrated for some of the cases is of the ratio of the number of SMR criteria achieved with feedback as compared with the number of SMR criteria met in the initial baseline period. A ratio of greater than 1.0 would indicate that the patient is able to do better when feedback is provided than without feedback. In most cases as shown in Figure 9 positive results were obtained in this measure.

Spectral and Bandpass Analyses

Periodically data were analyzed using the Fast Fourier Transform with compressed isometric routines of Bickford and Fleming (1970). Illustrative examples are shown for several patients in Figures 10 and 11. FM tape recordings were taken at different times during the training phase and also during the pre-training and post-training clinical EEG. Ideally, spectral analyses should be done every day, but we will not have an on-line computer facility until summer 1977. In many of our patients we were able to demonstrate spectral changes that were correlated with seizure reduction. There are two possible ways in which success of the treatment can be illustrated. One is a decrease in the amount of slow wave activity between three and 9 cycles. Most of the activity in this range represents the epileptogenic activity for which inhibit circuits are provided. Activity lower than three cycles represent gross body and eye movements and is usually handled by the EMG or slow wave inhibit circuits. Activity between 12 and 15 or 16 cycles most likely represents the sensorimotor rhythm which is being reinforced.

As seen in the figures, there was a decrease over training sessions in the amount of activity in the low frequency portion of the EEG spectrum. A spectral peak developed in some of our patients at 13 cycles representing SMR activity. In the case of E6's (Subject G.M.) activity, SMR peaks only developed in the right side, at least up to the present time, indicating an asymmetry in learning. E1 (Subject D.K.) experienced this activity bilaterally (see Figure 4) and relative traces of this activity can be seen in G.C.'s record.

Pre- versus post-operative clinical EEG spectral data is shown in Figure 12 for case D.K. Here, there are several significant points. One is that initially there was no SMR activity at the active record-site C3-T3, C4-T4, nor was there any significant 12 Hz to 15 Hz activity in occipital and temporal regions represented by O1-T3 and O2-T4. However, the alpha rhythm clearly stands out in this record at 9 or 10 cycles. The other recording points shown represent a location that was used by Serman in his initial studies of SMR conditioning. This consisted of 2 points, one 10 percent from the vertex and the other 30 percent from the vertex toward the ipsilateral ear for

each hemisphere. These recording points are closer to the vertex than C3-T3 and C4-T4. After this patient had been trained for fifty-four sessions a second clinical EEG was taken. Notice that at C3-T3, C4-T4 a clear peak develops at 13 Hz. Similarly a 3 Hz peak can be seen at O1-T3 and O2-T4, but the alpha peak has vanished. It is evidence of this type also corroborated by Sterman that tends to indicate that SMR and alpha are quite different entities and that after SMR peaks have developed, alpha often diminishes both in central and occipital-temporal loci. In the 10 percent, 30 percent position the only significant change is a small decrease in the amount of slow wave activity bilaterally, but no peak has developed in the SMR region. Generally then, spectral analyses have been useful in corroborating objectively the finding that there has been an acquisition of 12 Hz to 14 or 15 Hz activity over the regions trained and a concomitant reduction in epileptiform activity.

Another way of analyzing data is through bandpass analysis. Here the output of the active filter can be converted into a percent figure which represents the percent time that the SMR activity was above a specific microvolt level. This measure is illustrated for Case G.C. in Figure 13.

The microvolt level chosen was 6. In other words all 12 Hz to 15 Hz activity of 6 microvolts or greater is converted to a percent figure and plotted. The acquisition is asymmetrical in terms of cerebral hemisphere, but a rapid acquisition of activity in this frequency band did occur. This measure is not without its problems. First of all, it is a measure of filter activity rather than a measure taken from the raw EEG. Second, it is a measure which has to be kept constant each day and, therefore, requires that parameters for conditioning not be varied. In our case this problem was overcome by providing a separate circuit for this analysis which could be maintained at a fixed microvolt level. This arrangement would still allow us to vary the amplitude criteria for SMR reinforcement on a day to day basis. We still feel that this is far from a satisfactory measure and are currently working on a different type of bandpass analysis which is free from the problems of filters and inhibit circuits.

Present Status of Our Projects

Currently five of our original group of patients are in various degrees of weaning from training with the eventual hope that they can be maintained with reduced seizures and levels of medications and very occasional training (booster) sessions. Four other patients have been terminated from the training program entirely. These patients are D.K., M.T., C.D., and R.H. Of these R.H., as mentioned earlier, was the only patient that did not benefit from the treatment and lived a long distance from Knoxville. D.K. is virtually seizure free on Tegretol. M.T. is having very few seizures compared to previous levels, and the same is true of C.D.

Recently we have turned our attention to several new patients. Since all of our original patients were severely epileptic many inquiries have asked what would happen if we were to work with a moderate epileptic who had real hopes of becoming seizure free? To this end we have started a patient who had approximately ten seizures of the psychomotor and myoclonic type each month. This patient is still not adequately controlled by medication but is not as severe as the others. This patient has only been in treatment for one month but during this first month has already shown a 70 percent seizure reduction. It's far too early to tell whether this is a placebo effect or if the potency of the treatment might be even greater for the moderately controlled epileptic than

the severe epileptic. Unlike our other patients this patient's EEG shows relatively few abnormalities.

We are in the process now of collecting three months of baseline data on two new patients. One is a severe epileptic who also has cerebral palsy, cataracts, and is retarded. We have undertaken this case as a challenge to see what, if anything, can be done. Several additional patients have been interviewed and preliminary seizure data is being collected at the present time. Currently, we are requiring two to three months of baseline seizure data before we begin training. But we are still taking all seizure types except the exclusively petit mal.

Unanswered Questions and Future Directions

Although we are very encouraged with our preliminary results during the past three years and even more encouraged since we have been able to corroborate the findings of Sterman's group and Finley's group, there are still some very important questions that must be answered before we can consider that this modality of treatment for epilepsy has clinical promise. First, there is the question of whether there is an altered state of consciousness produced by SMR conditioning. Many inquiries addressed to us both by patients as well as our research group is "how do you make SMR occur?" There doesn't seem to be any simple answer to this question. Unlike alpha rhythm, which might be linked with a relaxed, defocused state, or theta rhythm with a dreamy, sleepy state or beta rhythm with arousal and anxiety, SMR is not clear in this context. One idea that is consistently mentioned is that SMR is a focused state that seems to be related to motoric tasks requiring inhibition. We often suggest to our patients, "Pretend that you are riding a car around a track and that as you put the brakes on the car refuses to slow down, and that you're trying harder and harder to stop the car." Sterman has made a similar suggestion' "Imagine skiing down a hill and negotiating the turns." These are motor tasks requiring motor inhibition and for several of our patients these suggestions have helped to improve SMR acquisition.

One very interesting finding is that many of our patients and non-epileptic control that we have tested report SMR is associated with sexual fantasizing. We don't know whether this is consistent enough to be meaningful and perhaps sexual fantasies are also associated with some of the other brain rhythms. But it has been mentioned enough times that we are wondering whether we are dealing with a sensorimotor rhythm or a "sensual motor" rhythm. Perhaps SMR conditioning similar to autonomic conditioning is a type of activity which becomes automatic, but cannot be conceptualized any more than explaining how do we raise our arm, or how do we pick up a pencil? We just do it.

Another unanswered problem is "how should patients be weaned from the program?" Sterman, Macdonald, and Stone (1974), and Sterman and Friar (1972) showed that if patients are abruptly withdrawn from treatment there is a temporary maintenance of decreased seizures followed by an increase back toward initial levels. Clearly abrupt withdrawal from treatment is not satisfactory. We have also seen this in patients who have taken long vacations (note the record for case M.T., Figure 7). Our feeling is that patients have to be weaned very gradually with heavy emphasis placed on home practice. Such home practice includes their imagining they are in the feedback room when they feel a seizure coming on. The latter has been successful for most of our patients in blocking many of their seizures. At the present time, we move gradually from

three treatments a week to once a month. Hopefully, the patients may be removed from the feedback in this way.

Emotional factors play an extremely important part in determining seizures. Consider for example the case of K.S. shown in Figure 7. There was a rapid rise in seizures that stands out in this case for Christmas day alone. Probably, as the patient put it, it was the excitement of unwrapping Christmas presents that produced a lot of small seizures. We now know that if our patients are (A) sleep deprived, (B) physically ill, or (C) emotionally upset, then seizure rates will increase independent of anything that we do to bring them down. Although we do not make any attempt to regulate our patients' lives, we do point out these factors and we encourage them to try to keep themselves healthy, well rested, and out of emotional difficulties.

Another example of emotional factors as shown in the seizure graphs of Case G.C. is the portion of the graph marked "family conflict." As can be seen, the seizure level immediately responded to this stress.

Another question is whether mentally retarded patients are good candidates for biofeedback. We have not been afraid to deal with a number of retarded cases. This is because a significant proportion of very severe epileptics are retarded. If SMR conditioning is to become a meaningful modality of treatment the practitioner has to deal with whatever comes along. Our feeling now is that it is definitely worthwhile to work with retarded cases including both children and adults, but not to set one's expectations too high and to remember that these patients may have to be kept in treatment in some fashion as long as they live.

For retarded patients, it is also necessary to develop alternate modes of feedback that hold their attention. Several reinforcers that we have used successfully are coins keyed to a certain number of criteria being met. Perhaps analog feedback would be better than binary feedback and the electric train that has been so heavily publicized in Barbara Brown's laboratory would be an outstanding reinforcer for a retardate or child epileptic. A transformer operating such a device could be linked directly to the amplitude of the SMR filter.

One of the most significant problems in dealing with epileptics or any group of individuals in which there is either a physical or psychological disorder is the problem of secondary gain. Because of their seizures many of our patients have developed strong dependencies on parents and other family members. Some of our patients have told us that they either like some of their seizures or that their seizures were useful in getting them what they wanted. This problem could be ruinous for a study of the type that we have been conducting if it is not dealt with insightfully. It is important whenever possible to point out to the patient that there are more possibilities open to them in life if they can control their seizures than are presently available through secondary gain.

One of our cases, G.M., who has shown a sustained reduction in seizures of greater than 70 percent, came to realize this when it became clear that he could not obtain a driver's license although he was at the appropriate age unless his seizure level could be reduced. In a program which is treatment rather than research oriented, secondary gain can be best dealt with by integrating a psychotherapeutic approach with the SMR training.

Finally, there is the problem of how can sensorimotor rhythm training be made available to the many more epileptics. According to the Epilepsy Foundation of America there are four million epileptics in the United States at this time. Of these one million are not adequately controlled with anticonvulsant medication. Yet, at the present time, there are fewer than ten laboratories working with SMR training all using complex and cumbersome equipment. There is clearly a need for portable instrumentation that can be established in neurological, psychiatric, and psychological clinics, outpatient treatment centers, and other appropriate settings. At the present time it does not seem practical to develop a portable instrument that the patients can use entirely on their own. This is because the number of calibrations and settings are far too complex. Several instrument companies have become involved in the development of portable instrumentation during the last year and perhaps these needs will be fulfilled.

Summary

Our studies have shown significant epileptic seizure reductions based on the development of inhibitory activity in the nervous system through sensorimotor rhythm training. The technology involved is very complex and the procedures are time consuming and expensive. However, biofeedback does offer a viable alternative for the one million epileptics whose medication is not sufficient to adequately control their seizures. It is a viable alternative to some of the medications which are damaging to organ systems and possibly even life limiting. In contrast, new medications are being developed and should be developed as rapidly as possible, as it is far easier to deal with epilepsy pharmacologically than behaviorally. Recently, Tegretol (carbamazepine) was introduced into the armamentarium of neurologists and has been tremendously helpful in controlling certain cases of refractory psychomotor epilepsy. New medications such as Tranxene are currently being tested and other compounds are being studied in Europe that have not yet become available here.

Still, for many years to come there will probably be a significant number of epileptics that cannot be adequately controlled either pharmacologically or surgically. For them perhaps biofeedback training of the sensorimotor rhythm or other Rolandic activity is the only hope they will have to achieve relative control of their seizures. It is to these people that this research has primarily been dedicated.

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Notes

(n1.) I wish to acknowledge and thank Dr. William Finley for providing the design for the clipper circuitry employed in our system,

DIAGRAM: FIG. 1. Raw EEG is shown in top trace, and the differential signal in bottom trace. Note the increased number of zero crossings in the differentiated signal and the decreased variability about the zero voltage line in the bottom trace produced by higher frequencies riding on low frequency activity in the raw signal.

GRAPH: FIG. 2. Frequency response of elliptic filter designed to detect sensorimotor rhythm of 12-15 Hz. Note that the response of the filter is virtually flat between 12-15 cycles and drops off by a factor of greater than 10 thousand between 15-17 Hz at the upper end and 10-12 Hz at the lower end of its frequency response. There are small side lobes at 7 Hz and 26 Hz corresponding to first harmonics. This tuning curve was obtained by passing mixed frequencies with a mean amplitude of 150 microvolts through the clipping circuit and filter. 150 microvolts was chosen since it represents a level which exceeds the majority of EEG activity. Even with this high level input the filter response is vary sharp and relatively free from ringing.

DIAGRAM: FIG. 3. In this figure the different EEG paper channels are shown. In particular the operation of the inhibit circuits for EMG activity and slow wave activity are illustrated. These inhibit circuits are linked to the filter outputs (left) and EMG outputs (right) shown in paper channel 5.

DIAGRAM: FIG. 4. Spectral analysis for patient E1 (D.K.) over 54 training sessions. Note the development of clear 13 Hz activity in sessions 45 and 54 along with a concomitant decrease in slow wave activity between 5 and 10 Hz in these latter records. This is in contrast to the increased slow wave activity in sessions 15 and 30 and the lack of a 13 Hz peak in the earlier analyses. These spectral pilots were obtained using the Fast Fourier Transform with compressed isometric routines as described in the text.

DIAGRAM: FIG. 5. Raw EEG and data processing channels for patient K.S. (14-year-old female). These EEG samples were obtained at different times three months apart. Note the marked normalization shown in traces B as compared with traces A. This normalization of the EEG is reflected in an increase in the frequency of feedback for SMR and a decrease in the amplitude of slow wave activity as well as an overall decrease in EEG amplitude. Record A shows a myoclonic seizure. These were frequent for this patient during the early phases of training. Both records were obtained under identical calibrations and were chosen as representative samples of the types of positive changes that we have seen in many of our patients over training sessions.

DIAGRAM: FIG. 6A. EEG changes for patients G.B. (29-year-old female) and G.M. (19-year-old male), as training progressed. Note the normalization in both of these cases similar to that shown in Figure 5. This normalization is characterized by decreased slow wave activity and decrease in overall amplitude of the EEG as well as an increase in the amount of feedback and a decrease in the activity in inhibitory circuits. Identical calibrations were used in all traces.

DIAGRAM: FIG. 6B. See For Figure 6A.

Seizure records for K.S., G.M., S.R., M.T., C.D., and G.C.:

GRAPH: FIG. 7A. K.S. Note the rapid drop in myoclonic seizures in the early phases of training. A peak occurred on Christmas day, associated with the excitement of the holiday. After 240 days the hard seizures differentiated into hard and medium seizures. After 340 days the patient was gradually weaned from treatment, resulting in a transient increase in seizure activity followed by a decrease.

GRAPH: FIG. 7B. G.M This patient has achieved very long-term asymptotic maintenance of a decreased seizure level, approximately 70% below his initial starting point. Note the low level of seizures was maintained even after weaning from treatment had been initiated.

GRAPH: FIG. 7C. S.R. This patient, severely retarded, had akinetic seizures. Overall, she experienced a seizure reduction of approximately 50% as compared with initial levels.

GRAPH: FIG. 7D. M.T. Note the rapid decrease in seizure-free periods. Treatment was abruptly terminated during a one-month vacation. Note here an increase in seizure activity followed by a total drop following reinstatement of treatment.

GRAPH: FIG. 7E. C.D. Mixed seizure type, grand mal and psychomotor. This patient experienced rapid reduction of seizures and drug reduction. This case became seizure free after 170 days of training. Weaning from treatment was variable, but ultimately a very low seizure level has been achieved.

GRAPH: FIG. 7F. G.C. Initial baseline for this patient is represented by the point at the far left of the graph. Following the administration of Tegretol a transient decrease in seizures occurred followed by an increase to previous levels. During treatment the patient's seizure record has been highly variable in part due to family problems. A low point had been reached after 170 days of treatment. Reduction in Phenurone medication resulted in a transient increase in seizures followed by a decrease. Overall, this patient's seizure reduction has been about 30%.

GRAPH: FIG. 8. Seizure records for patient G.B. As shown by the top graph, the only notable change was a reduction in one of the anticonvulsant medications (Mysoline). Although it appears that no progress has been made with this patient, there has been a noticeable decrease in the intensity and duration of the seizures as shown by the seizure index. This seizure index is equal to the number of seizures multiplied by the intensity as rated on the three-point scale described in the text This index was initiated on day 400.

GRAPH: FIG. 9. Ratio of the mean number of SMR criteria achieved when feedback was presented divided by the mean number of criteria which were achieved without feedback in the initial baseline period. Values greater than 1.0 indicate behavioral control of the feedback and that learning has taken place. Note that for most patients on most sessions, ratios of greater than 1.0 were achieved.

DIAGRAM: FIG. 10. Spectral analysis based on Fast Fourier transform with compressed isometric routines for patient G.C. (male psychomotor epileptic). Note the very slight decrease in slow wave activity in later sessions compared to that in session 14 for the left hemisphere and 40 for the right hemisphere. Although no clear-cut SMR band of activity has occurred in the SMR

frequency range, relative amounts of such activity appear in session 40 and bilaterally and some activity (between 14 and 17 Hz) is evident for the right hemisphere in session 70.

DIAGRAM: Fig. 11. Spectral analysis for patient (E6) G.M. (myoclonic seizures). This patient shows clear evidence of acquisition of sensorimotor rhythm in the right hemisphere in later sessions.

DIAGRAM: FIG. 12. Spectral analysis based on clinical EEG taken before and after training for patient E1 (D.K.). Training was for coordinates C3-T3 and C4-T4. Note that in both of these electrode sites and for the occipital-temporal locus clear peaks at 13 Hz developed after training. Interestingly, the initial alpha peak at 9 to 10 Hz in O1-T3 and O2-T4 has disappeared, indicating that alpha and SMR are different EEG entities and perhaps inversely related to each other. The 1030 position shown in the top trace is a more medial rolandic placement. Little change was shown in these top records except for a slight decrease in slow wave activity.

GRAPH: FIG. 13. Percent SMR over blocks of sessions for left and right hemispheres during baseline and feedback for psychomotor epileptic G.C. In the early block of sessions shown on the left portion of the diagram there is a noticeable difference between the amount of SMR during baseline and feedback. Generally, feedback resulted in higher levels. In the later blocks of sessions both baseline and feedback levels of SMR are increased to high levels. This is interpreted as beneficial, indicating carryover effects of training to the baseline sessions and also shows that high percent levels of this activity can be attained in the EEG spectrum. The amplitude level set for sensorimotor activity was 6 microvolts. All activity above 6 microvolts was analyzed during each session. For this particular measure sensorimotor rhythm activity was recorded with separate circuits that were not inhibited by slow waves.

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