

CLINICAL NOTE

SUPPRESSION OF SEIZURES IN AN EPILEPTIC FOLLOWING SENSORIMOTOR EEG FEEDBACK TRAINING

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Localized recordings of electrical activity from functionally discrete regions of cerebral cortex in the cat have disclosed specific rhythmic patterns in the waking state which are uniformly associated with certain classes of behavior (Donohoffer and Lissak 1962; Roth *et al.* 1967). The observation of such localized slow wave rhythms related to specific behaviors provides a bio-electric label for these behaviors and the brain processes to which they are related. Of particular interest to us in this regard was a 12–14 c/sec rhythm appearing over sensorimotor cortex during the voluntary suppression of movement (Stermán and Wyrwicka 1967; Wyrwicka and Stermán 1968). Neurophysiological studies in the cat have indicated that this sensorimotor rhythm, or SMR as we have termed it (Roth *et al.* 1967), is generated by neurons within the ventrobasal thalamus, probably through alternate depolarization and recurrent inhibition within a thalamocortico-thalamic network (Andersson and Manson 1971; Howe and Stermán 1972). Extracellular single unit recordings from ventrobasal elements indeed do show recurrent bursting discharge and silence during cortical SMR activity in the cat (Harper and Stermán 1972). These findings have led us to conclude that thalamic and cortical inhibitory elements are activated during the production of this rhythm.

Behavioral studies have shown that the SMR is subject to operant conditioning through the application of biofeedback techniques (Stermán and Wyrwicka 1967; Wyrwicka and Stermán 1968; Stermán *et al.* 1969b). That is, by providing a food reward for the occurrence of this rhythm, cats learn to produce the rhythm in order to obtain food in much the same manner as the bar press is employed in other operant conditioning studies. Prolonged training of the SMR under these circumstances resulted in an enhancement of cortical EEG spindle-burst activity during sleep, accompanied by a reduction in motor activity and prolonged episodes of sustained sleep (Stermán *et al.* 1970). In other studies, such training produced evidence for resistance to drug-induced seizures (Stermán *et al.* 1969a). Convulsive doses of monomethylhydrazine resulted in typical pre-ictal symptoms in trained animals, but actual seizures were significantly delayed or absent in these animals as compared to controls.

These various observations in the cat led us to seek a human analog to the feline SMR. Utilizing techniques modified

for studies in man (Stermán and Friar, in preparation), we have established that (1) a localized rhythm resembling the SMR can be recorded from the rolandic area (sensorimotor cortex) with surface electrodes in man, and (2) this rhythm can be brought under operant control through biofeedback techniques as indicated by a significant increase in its rate and amplitude, differentiation from other cortical EEG rhythms and subjective labeling of a distinctive cognitive state.

Because of the evidence for enhancement of thalamic and cortical inhibitory elements, suppression of phasic motor activity and delay of drug-induced seizures in the cat, we were encouraged to examine the effects of SMR training in an epileptic human subject. The positive results of this preliminary investigation appear to warrant communication at this time.

Patient history

The subject studied was a 23-year-old white female with a history of convulsive disorder dating back to 1964. The onset, at age 16, was insidious and consisted of a nocturnal, generalized major motor seizure. There was a history of childhood febrile convulsions but no family history of epilepsy. Neurologic examinations, including angiogram and pneumoencephalogram, have consistently failed to demonstrate a localized lesion. The initial EEG examination was reported abnormal due to "generalized slowing" (intermixed 5–7 c/sec activity), but did not report any paroxysmal discharges. An EEG from 1966 showed generalized spike-wave activity increased by hyperventilation, and others taken in 1968 and 1971 showed some evidence of focal activity in the left fronto-parietal area.

The convulsion is preceded by a non-specific aura, and there have been no localizing features to any precipitating factors in the prodrome or in the post-ictal period. The ictus originally included tonic-clonic movements following loss of consciousness and falling; more recently, however, daytime incidents have consisted of wrinkling of the brow associated with left lateral deviation of the eyes, crossing of the right arm to the left knee, and falling to the left. The majority of incidents are currently nocturnal, occurring in the later portions of a night's sleep.

The frequency of occurrence of the seizures has varied from 2 to 4 per month at the onset to 1 per 3 months one year

later. During the 12 months immediately preceding the present investigation, seizures were experienced irregularly at an average rate of approximately 2 per month. The subject has had variable success with several combinations of Dilantin, Mysoline, Peganone, Diamox and Ritalin. The regimen during the past 12 months includes Dilantin and Mebarol, 200 mg each, daily.

Training procedure and methods

Techniques presently in use in our laboratory provide for the recording of rolandic (sensorimotor) and occipital cortex EEG activity, together with electrocardiogram (EKG), electromyogram (EMG) of the submental musculature and respiration, from patients seated comfortably in a reclining chair. EEG patterns were recorded from the scalp with needle electrodes. Placement of electrodes over these brain areas has been standardized by reference to the international 10-20 system. Optimal placement for recording the SMR in this subject was achieved by use of a longitudinal bipolar electrode pair, with the anterior lead intermediate between F₃ and C₃ sites and the posterior lead between C₃ and P₃. Recordings of the alpha rhythm were obtained with a bipolar electrode pair placed at P_z and O₁. EKG and EMG were recorded with paste-filled cup electrodes fixed to the skin with tape, and respiration was monitored by a pneumatic belt connected to a transducer and DC amplifier. These data were recorded on an 8-channel Sanborn Model 7700 polygraph. EEG signals were entered from the amplifiers of this polygraph into a 7-channel filter and logic system which was capable of a limited frequency analysis, automatic detection of specific frequencies and selection of precise signal-to-noise, duration and interval

criteria for activation of a feedback device. The feedback device consisted of a rectangular unit suspended from the ceiling several feet above and in front of the patient. This unit contained two rows of ten small lamps, each covered by transparent colored plexiglass. When appropriate EEG signals were detected at criterion amplitude and duration, the lamps in the top row were lighted in a left-to-right sequence, each advance being accompanied by the sounding of a single chime. With the eleventh successful EEG response, these lights were extinguished and the first light of the bottom row of lamps was activated together with a double chime. The next response initiated the lighting sequence in the top row again, and so on. This device provided for sets of 100 reinforcements with single and decade lamps and single and double chimes as the reward.

The patient was isolated in a comfortable, dimly lit room and instructions were delivered via standard tape recordings and an intercom unit from an adjacent room which housed the recording equipment and the experimenters. The patient was instructed to keep eyes open, clear the mind and think of past experiences or of nothing at all in an effort to achieve the desired mental state. When the appropriate EEG pattern was produced, the feedback device was operated. In such a manner, 200-300 rewards were obtained during training periods lasting 30 min to 1 h, depending on the point in training. Five min of pre- and post-training baseline data were collected also during each training session. Such sessions were provided once a week during the month of September 1971, and at least twice a week from that time to the present writing (see Table I).

TABLE I

Chronologic record from subject MF indicating occurrence of nocturnal grand mal seizures from September 1970, to end of current phase of study. During this entire period, subject has taken 200 mg Dilantin and 200 mg Mebarol daily. Seizures are indicated by "S". Sensory motor rhythm feedback training was initiated on 24 August, 1971, and subsequent training sessions are indicated by "t". One seizure has occurred since 31 August, 1971.

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31
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Nov	t			t					t		t	t			t				t				t		t					t	
Dec	S	t						t	t					t	t						t	t					t		t		

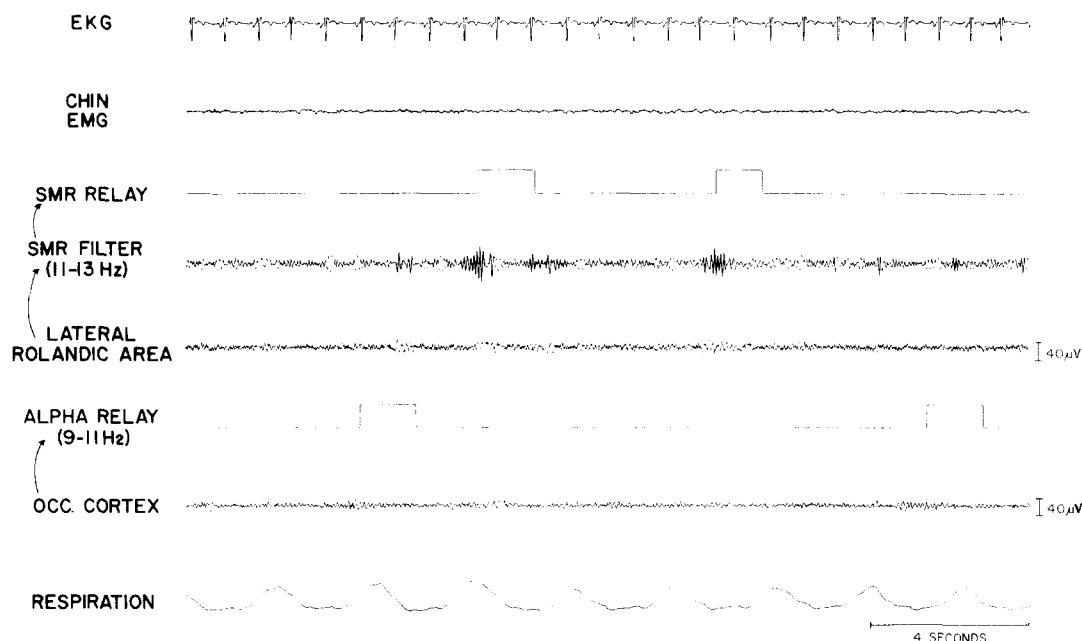


Fig. 1. Sample tracings of rolandic and occipital cortex EEG, showing detection of the sensorimotor and alpha rhythms by a system of tuned frequency filters and logic. Polygraphic EEG sensitivities were attenuated below 1 and above 40 c/sec. The signal-to-noise ratio and duration of the two rhythms required to activate feedback were subject to adjustment by the investigators. Other physiological measures obtained simultaneously included EKG, chin EMG and respiration.

RESULTS

As has been our experience with other subjects, this patient initially demonstrated a moderately low level of SMR activity spontaneously during the initial pre-feedback baseline recording. Because of her generalized EEG slowing mentioned earlier, it was decided to reward large amplitude trains in the 11–13 c/sec frequency range. This represented a slight adjustment from the 12–14 c/sec frequency utilized with other subjects. A typical tracing, showing filter response and relay detection of the SMR in this patient, is presented in Fig. 1, together with the relay detection of occipital alpha rhythm activity (9–11 c/sec) during the same period. Polygraph and logic system sensitivities were comparable for both signals. The peak SMR frequency rewarded was 12 c/sec, with 50% attenuation at 11 and 13 c/sec and a very sharp drop-off beyond these limits.

During the first several sessions of SMR feedback training, no evidence of learning could be detected in measurements of SMR and alpha production over time (Fig. 2). By the fourth session, the production of SMR had increased slightly in the pre-training baseline, and both rhythms showed a gradual increase in occurrence during training. Both post-training baselines were increased over initial levels. This generalized increase in the production of both rhythms was replaced by a selective enhancement of the SMR and depression of alpha during training by the 12th session. Pre-training levels of alpha were high but consistent, and production during feedback training was clearly reduced (Fig. 3). Pre-training levels of SMR were greatly increased over earlier sessions, and the

striking enhancement of this rhythm with feedback was sustained in the post-training period.

During the 12 month period between September 1970, and August 1971, the patient had averaged 1.92 grand mal seizures per month (Table I). In the 6 month period immediately preceding training, this rate dropped slightly to a mean of 1.67 per month. These seizures frequently occurred during sleep and were detected by blood stains on the bedwear the next day, and a sore mouth, tongue and jaw. Training was initiated on August 24, 1971, and continued, as described above, to the time of this communication. Six days after the first training session the patient experienced two seizures. Concurrent with evidence for acquisition of the SMR response by the third session, there were no further seizures for a period of 3 months. Additionally, the patient showed some interesting changes in personality during the course of training. Having previously been a quiet and unobtrusive individual, she progressively became more outgoing, showing increased personal confidence and an enhanced interest in her appearance. She also spontaneously reported experiencing a shorter latency to sleep onset, a more restful sleep, as indicated by a reduction of her normal physical re-orientation in bed throughout a night, and a more rapid awakening in the morning. None of the latter changes could be documented objectively, but they were particularly interesting in terms of the similar, quantified findings obtained with SMR in the cat.

The patient voluntarily ceased her previous practice of supplementing anticonvulsant medication upon experiencing an aura, without seizure, and reported a reduction in these

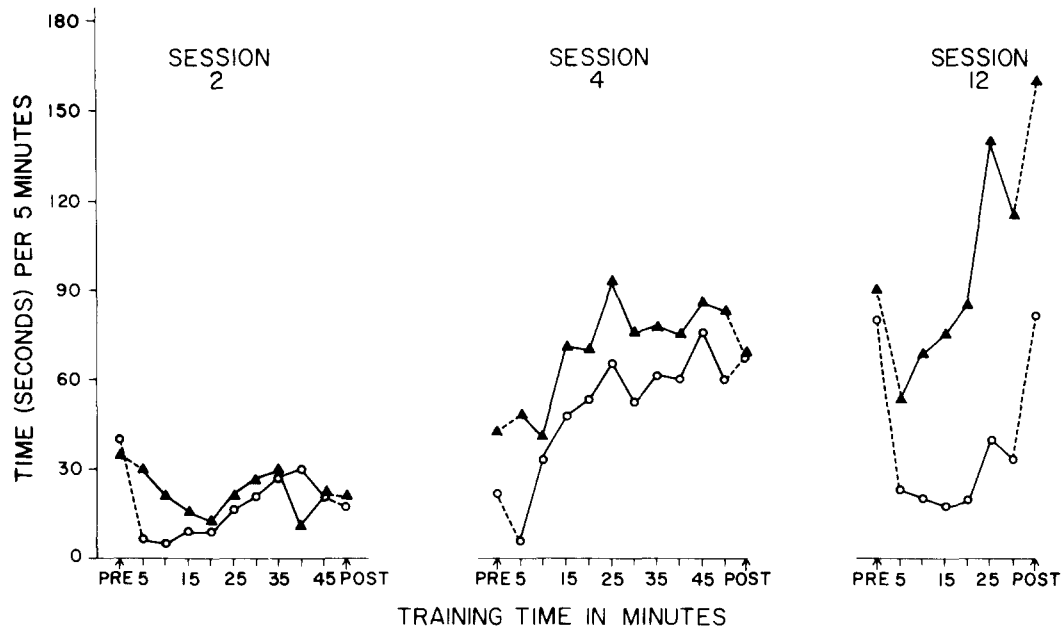


Fig. 2. Plot of SMR (solid triangles) and alpha rhythm (open circles) production during SMR training and pre-post baseline recordings at three points in the training sequence. Output is expressed in terms of the total occurrence of these rhythms, at criterion amplitude and duration, in pre- and post-training 5 min samples and in sequential 5 min epochs during training. Transitions between training and pre-post measures are indicated by broken line.

occurrences. Because of a skin condition, she had been taking 500 mg daily of oral Tetracycline for a period of approximately 6 months. She was taken off this drug on November 19, 1971. On December 1, the patient experienced a mild nocturnal seizure. She reported "knowing it was going to happen that night when I went to bed". It is impossible to determine whether or not any relationship existed between the termination of the antibiotic and the occurrence of this seizure. The patient had been complaining of boredom during training in the preceding weeks and had begun to show signs of drowsiness in concurrent sessions. The day after her first seizure experience in 3 months she requested a small monetary reward for each response and, with our approval, proceeded to perform at the highest rate of SMR production she had ever demonstrated. She has continued in high rate performance since that time and has had no additional seizure experiences.

DISCUSSION

The functional and topographic characteristics of the sensorimotor rhythm in the cat led us previously to compare it with the rolandic *arceau* or "wicket" rhythm described in man by Gastaut *et al.* (1954), and to conclude that both represent, functionally, EEG correlates of voluntary phasic motor suppression (Howe and Sterman 1972). Extension of our studies to man have, indeed, confirmed the presence of a 12-14 c/sec rhythm in the vicinity of the central sulcus and have indicated that it is blocked by phasic motor activity, not disrupted by sensory stimuli in the absence of movement, and related subjectively to a distinct cognitive state (Sterman and Friar, in preparation). This rhythm is subject to operant con-

ditioning also in man and, in the case reported here, continued EEG feedback training of this nature was associated with a suppression of grand mal seizures.

The basis for this effect cannot be specified at this time. Epileptic conditions are subject to a diversity of physiological and psychological influences, and the potential for spontaneous organic change or so-called "secondary gain" should not be overlooked. The patient, nevertheless, demonstrated a rather abrupt change in an active seizure pattern, corresponding to the acquisition, or learning, of this EEG slow wave response. It is possible, therefore, that functional changes associated with the voluntary enhancement of the SMR were related to the observed effect.

It is known that the interruption or withdrawal of sensory input to neurons and neural systems produces changes in synaptic morphology and alterations in the functional capacities of an entire system. If presynaptic axons to a neuron are severed, a micro-environmental upheaval occurs. Necrotizing presynaptic terminals are engulfed by microglia, and synaptic sites that are affixed to spines are incorporated within the dendrite or by the microglia (Colonnier 1964). The sensitive balance of postsynaptic potentials swings from electrical silence to hypersensitivity (Burns 1958) and, if the input is primary to the neuron, transneuronal degeneration occurs (Hess 1958). Withdrawal of input to a system through sensory deprivation in young animals leads not only to alteration in synaptic morphology but also to clear reductions in sensory cortical volume (Globus 1971).

We are less certain about the influence of enhanced utilization of neurons and neuronal circuits upon their morphology and function, but there are numerous findings which

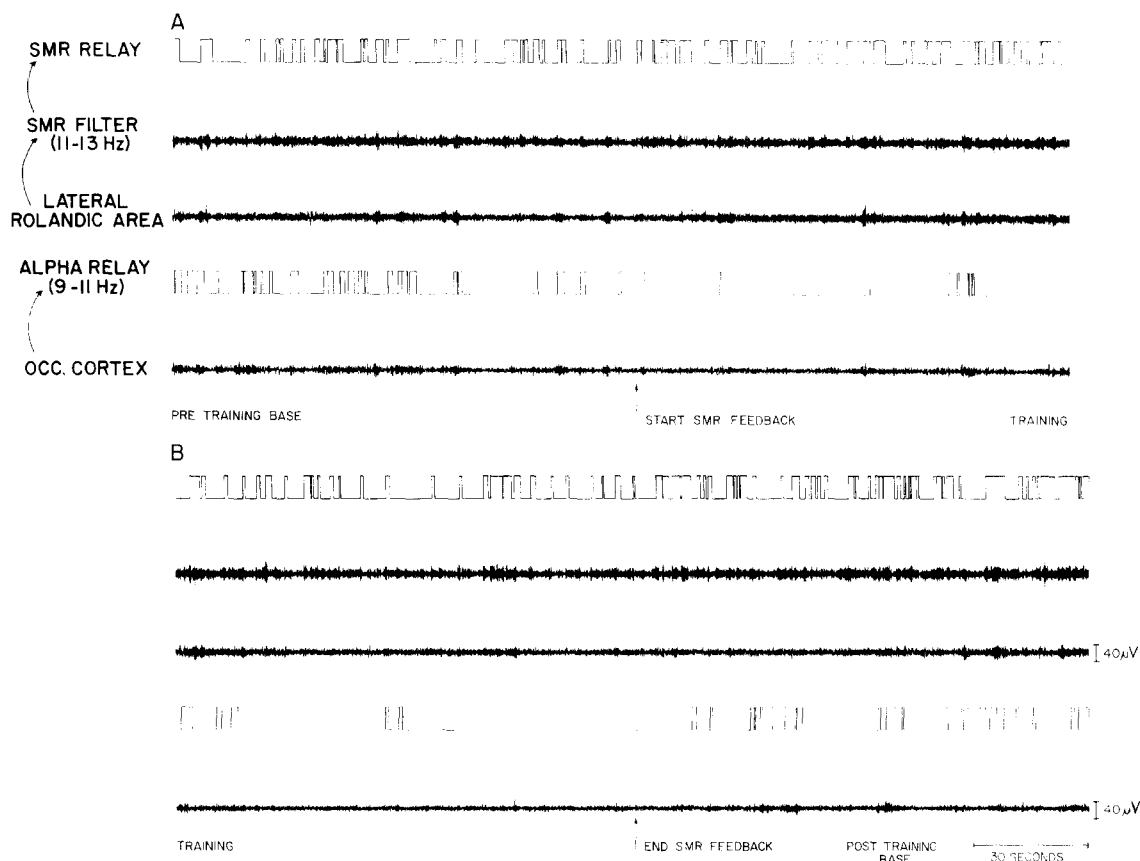


Fig. 3. Polygraphic data obtained at slow paper speed during transitions from pre-training baseline to the initiation of SMR feedback (A) and from the end of feedback training to the initiation of post-training measurement (B). Note the marked reduction of alpha rhythm activity and general desynchronization of the occipital EEG during SMR feedback occurring in conjunction with a high level of SMR production and a generally synchronized rolandic cortex EEG. SMR production tended to remain elevated or increased even further during post-training baseline measurements.

favor the conclusion that alterations do occur which lead to a functional enhancement of the mechanisms in which these elements participate. The prolonged post-tetanic potentiation recorded after high frequency stimulation of spinal cord synapses led Eccles and McIntyre (1953) to propose the intriguing possibility that such stimulation results in a swelling of afferent terminals, a process which might also occur at higher levels of organization in relation to learning. More recently, Bliss *et al.* (1968) found that constant stimulation of short-latency neuronal pathways in an isolated slab of cat cortex resulted in persistent alterations of synaptic conductivities in these pathways. Directly related to the present considerations are the findings of Rutledge *et al.* (1967). They reported that daily stimulation of undercut segments of marginal cortex in the cat prevented the development of neuronal supersensitivity in these areas, as indicated by the absence of electrically induced after-discharges characteristically observed in unstimulated animals. Repeated activation of the thalamo-cortical network found to mediate the SMR in cats (Howe and Serman 1972), associated as it is with the synchronous discharge of inhibitory neuronal pools (Andersen and

Andersson 1968; Harper and Serman 1972), could provide a basis for such functional alterations. In turn, alterations of this nature would not be incompatible with the suppression of seizure activity reported here.

Numerous attempts have been made in the past to utilize operant conditioning in the treatment of epilepsy (Efron 1957; Stevens 1960; Guerrero-Figueroa *et al.* 1963; Stevens *et al.* 1967). These efforts relied upon behavioral concepts, associating distractive or aversive stimuli with the patient's aura or epileptic paroxysms, with limited success. In contrast, the present attempt developed from a neurophysiological framework, and based its predictions upon the possibility of achieving an altered neuronal integration through repeated activation of specific neural circuits with EEG feedback training.

SUMMARY

Previous studies of a 12-14 c/sec slow wave rhythm localized to sensorimotor cortex in the cat indicated its functional relationship to thalamo-cortical inhibitory discharge, sup-

pression of phasic motor behavior and suppression of drug-induced convulsions. Investigations in man showed the presence of a similar rhythm in rolandic cortex. Biofeedback techniques for the operant conditioning of this rhythm developed in studies of the cat provided a basis for similar EEG feedback training in man. The functional characteristics mentioned above suggested that this training could be of some benefit in the treatment of epilepsy. This communication reports preliminary findings from such a study in a 23-year-old female subject with moderately controlled major motor seizures of frontoparietal origin. Biofeedback training of this sensorimotor rhythm resulted in a striking enhancement of the rhythm's occurrence, differentiation from simultaneously recorded alpha rhythm activity, and a marked suppression of seizures. Changes in sleep patterns and personality were noted also.

RESUME

SUPPRESSION DES CRISES CHEZ UN EPILEPTIQUE APRES APPRENTISSAGE FEEDBACK EEG SENSORI-MOTEUR

Des études antérieures d'un rythme lent de 12 à 14 c/sec localisé au cortex sensori-moteur du chat, ont indiqué sa relation fonctionnelle à la décharge inhibitrice thalamo-corticale, la suppression du comportement moteur phasique et la suppression des convulsions chimiquement induites. Des investigations chez l'homme ont montré la présence d'un rythme similaire au niveau du cortex rolandique. Les techniques de bio-feedback pour conditionnement opérant de ce rythme, mises au point dans les études sur le chat, ont fourni une base pour un entraînement feedback EEG similaire chez l'homme. Les caractéristiques fonctionnelles mentionnées ci-dessus ont suggéré que cet apprentissage pouvait entraîner un certain bénéfice dans le traitement de l'épilepsie. Cette communication rapporte les résultats préliminaires d'une telle étude chez une femme âgée de 23 ans, présentant des crises motrices majeures, généralisées, relativement contrôlées. L'apprentissage bio-feedback de ce rythme sensori-moteur provoque une augmentation frappante de sa survenue, une différenciation de l'activité alpha enregistrée simultanément, et une suppression nette des crises. Des modifications des patterns de sommeil et de la personnalité ont été également notées.

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REFERENCES

- ANDERSEN, P. and ANDERSSON, S. A. *Physiological basis of the alpha rhythm*. Appleton-Century Crofts, New York, **1968**, 335 p.
- ANDERSSON, S. A. and MANSON, J. R. Rhythmic activity in the thalamus of the unanesthetized decorticate cat. *Electroenceph. clin. Neurophysiol.*, **1971**, 31: 21-34.
- BLISS, T. V. P., BURNS, B. D. and UTTLEY, A. M. Factors affecting the conductivity of pathways in the cerebral cortex. *J. Physiol. (Lond.)*, **1968**, 195: 339-367.
- BURNS, B. D. *The mammalian cerebral cortex*. Arnold, London, **1958**, 119 p.
- COLONNIER, M. Experimental degeneration in the cerebral cortex. *J. Anat. (Lond.)*, **1964**, 98: 47-53.
- DONHOFFER, H. and LISSAK, K. EEG changes associated with the elaboration of conditioned reflexes. *Acta physiol. Acad. Sci. hung.*, **1962**, 21: 249-255.
- ECCLES, J. E. and MCINTYRE, A. K. The effects of disuse and of activity on mammalian spinal reflexes. *J. Physiol. (Lond.)*, **1953**, 121: 492-516.
- EFRON, R. The conditioned inhibition of uncinate fits. *Brain*, **1957**, 80: 251-262.
- GASTAUT, H., DONGIER, M. and COURTOIS, G. On the significance of "wicket" rhythms (rythmes en arceau) in psychosomatic medicine. *Electroenceph. clin. Neurophysiol.*, **1954**, 6: 687.
- GLOBUS, A. Neuronal ontogeny: its use in tracing connectivity. In M. B. STERMAN *et al.* (Eds.), *Brain development and behavior*. Academic Press, New York, **1971**, 13: 253-263.
- GUERRERO-FIGUEROA, R., BARROS, A. and DE BALBIAN VERSTER, F. Some inhibitory effects of attentive factors on experimental epilepsy. *Epilepsia (Amst.)*, **1963**, 4: 225-240.
- HARPER, R. M. and STERMAN, M. B. Subcortical unit activity during a conditioned 12-14 Hz sensorimotor EEG rhythm in the cat. *Fed. Proc.*, **1972**, 31.
- HESS, A. Optic centers and pathways after eye removal in fetal guinea pigs. *J. comp. Neurol.*, **1958**, 109: 91-115.
- HOWE, R. C. and STERMAN, M. B. Cortical-subcortical EEG correlates of suppressed motor behavior during sleep and waking in the cat. *Electroenceph. clin. Neurophysiol.*, **1972**, 32: 681-695.
- ROTH, S. R., STERMAN, M. B. and CLEMENTE, C. D. Comparison of EEG correlates of reinforcement, internal inhibition and sleep. *Electroenceph. clin. Neurophysiol.*, **1967**, 23: 509-520.
- RUTLEDGE, L. T., RANCK JR., J. B. and DUNCAN, J. A. Prevention of supersensitivity in partially isolated cerebral cortex. *Electroenceph. clin. Neurophysiol.*, **1967**, 23: 256-262.
- STERMAN, M. B. and WYRWICKA, W. EEG correlates of sleep: evidence for separate forebrain substrates. *Brain Res.*, **1967**, 6: 143-163.
- STERMAN, M. B., HOWE, R. C. and MACDONALD, L. R. Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. *Science*, **1970**, 167: 1146-1148.
- STERMAN, M. B., LOPRESTI, R. W. and FAIRCHILD, M. D. *Electroencephalographic and behavioral studies of monomethylhydrazine toxicity in the cat*. Technical Report AMRL-TR-69-3. Aerospace Medical Research Laboratory, Aerospace Medical Division, Air Force Systems Command, Wright-Patterson Air Force Base, Ohio, **1969a**.
- STERMAN, M. B., WYRWICKA, W. and ROTH, S. R. Electrophysiological correlates and neural substrates of alimentary behavior in the cat. *Ann. N.Y. Acad. Sci.*, **1969b**, 157: 723-739.

- STEVENS, J. R. Electroencephalographic studies of conditioned cerebral responses in epileptic subjects. *Electroenceph. clin. Neurophysiol.*, **1960**, 12: 431-444.
- STEVENS, J. R., MILSTEIN, V. M. and DODDS, S. A. Endogenous spike discharges as conditioned stimuli in man. *Electroenceph. clin. Neurophysiol.*, **1967**, 23: 57-66.
- WYRWICKA, W. and STERMAN, M. B. Instrumental conditioning of sensorimotor cortex EEG spindles in the waking cat. *Physiol. Behav.*, **1968**, 3: 703-707.

Reference: STERMAN, M. B. and FRIAR, L. Suppression of seizures in an epileptic following sensorimotor EEG feedback training. *Electroenceph. clin. Neurophysiol.*, **1972**, 33: 89-95.

EEG and Behavioral Changes in a Hyperkinetic Child Concurrent with Training of the Sensorimotor Rhythm (SMR) A Preliminary Report¹

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Reduced seizure incidence coupled with voluntary motor inhibition accompanied conditioned increases in the sensorimotor rhythm (SMR), a 12–14 Hz rhythm appearing over rolandic cortex. Although SMR biofeedback training has been successfully applied to various forms of epilepsy in humans, its potential use in decreasing hyperactivity has been limited to a few cases in which a seizure history was also a significant feature. The present study represents a first attempt to explore the technique's applicability to the problem of hyperkinesis independent of the epilepsy issue. The results of several months of EEG biofeedback training in a hyperkinetic child tend to corroborate and extend previous findings. Feedback presentations for SMR were contingent on the production of 12–14-Hz activity in the absence of 4–7-Hz slow-wave activity. A substantial increase in SMR occurred with progressive SMR training and was associated with enhanced motor inhibition, as gauged by laboratory measures of muscular tone (chin EMG) and by a global behavioral assessment in the classroom. Opposite trends in motor inhibition occurred when the training procedure was reversed and feedback presentations were contingent on the production of 4–7 Hz in the absence of 12–14-Hz activity. Although the preliminary nature of these results is stressed, the subject population has recently been increased to establish the validity and generality of the findings and will include the use of SMR biofeedback training after medication has been withdrawn.

¹This research was a segment of the junior author's dissertation research.

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Considerable research during the past five years suggests that there is a generalized inhibitory effect associated with the sensorimotor rhythm (SMR) of 12–14 Hz recorded over sensorimotor cortex. Although autonomic, behavioral, and muscular inhibition are noted correlates of the rhythm (Donhoffer & Lissak, 1962; Roth, Serman, & Clemente, 1967; Serman & Wyrwicka, 1967; Chase & Harper, 1971), the potent anticonvulsant effects associated with conditioned increases in SMR have received the most attention thus far. Since the original demonstration in which SMR training enhanced resistance to convulsant drugs in cats (Serman, LoPresti, & Fairchild, 1969), several investigators have reported marked decreases in seizure frequency following biofeedback training in various forms of human epilepsy (Serman, Macdonald, & Stone, 1974; Finley, Smith, Etherton, 1975; Seifert & Lubar, 1975; Lubar & Bahler, 1976).

At the same time, evidence has accumulated which suggests an equally strong relationship between SMR and somatomotor inhibition. The rhythm's most characteristic behavioral correlate is immobility (Donhoffer & Lissak, 1962; Roth et al., 1967; Serman & Wyrwicka, 1967; Howe & Serman, 1972; Chase & Harper, 1971), and a reduction in muscular tension accompanying trained SMR increases in cats has also been documented by Chase and Harper (1971). Further evidence of this inhibitory effect is an abundance of SMR appearing in the EEG spectra of paraplegics and quadraplegics, in whom lower-motor neurons have been partially denerated (Serman et al., 1974).

Given the potential therapeutic value of SMR training in at least one disorder and the evidence indicating a functional relationship between SMR and motor inhibition, it seems appropriate to consider the possible application of this biofeedback technique to the hyperkinetic syndrome, in which overactivity is a central feature. Recent data from our laboratory suggest that some improvement in motor control occurred following SMR training

Table I. Number of EEG and Behavioral Recording Sessions Conducted during Five Consecutive Experimental Phases

	Baseline sequence		EEG biofeedback training sequence ^a		
	I No Drug	II Drug Only	III Drug and SMR training I	IV Drug and SMR reversal training	V Drug and SMR training II
EEG sessions	6	6	78	36	28
Behavioral sessions	15	15	45	15	15

^aSee text for explanation of feedback contingencies.

in a 14-year-old subject who was both epileptic and overactive (Lubar & Bahler, 1976). However, it was difficult to assess whether this outcome reflects training factors, maturation, or a secondary effect from seizure control.

The purpose of the present study is to explore the potential application of SMR training to hyperkinesis in the absence of a seizure history. The data reviewed here represent a progress report following several months of SMR training in a hyperkinetic child. Changes in motor inhibition are indexed by muscular tension in the laboratory and by behavioral observations in the classroom.

METHOD

Subject Selection Criteria and Description

In order to participate in all phases of the experiment (see Table I), the hyperkinetic subject had to meet a dual set of criteria. First, a uniform set of diagnostic criteria were required for admission to the baseline sequence. A child was considered an acceptable representative of the hyperkinetic population if he was (a) male; (b) within the age range of 6–12 years; (c) diagnosed as hyperkinetic by a pediatrician who considered the case severe enough to warrant medication; (d) regularly taking Ritalin; (e) diagnosed as hyperkinetic according to the Stewart teacher questionnaire, requiring definite indication of at least 6 symptoms, including overactivity and short attention span; (f) without specific sensory defects or any other functional or physical illness (e.g., mental retardation, epilepsy, etc.) that might contribute to or otherwise be confounded with the target syndrome.

Second, participation in the SMR training sequence was dependent upon demonstrable improvement in the classroom on selected behavioral categories and in laboratory performance (EEG only) following Ritalin administration. Improvement is not intended to denote complete remission of symptoms; rather, a substantial enough change is required to establish the reliability of the measurements and the degree to which they may be expected to concur prior to SMR training. Significant improvement in behaviors included an overall reduction in undirected activity accompanied by an increase in school-oriented activity (see Results section). Significant changes in EEG measures included a reduction of slow-wave activity between 4 Hz and 7 Hz and an increase in 12–14-Hz production. Changes in other physiological measures were evaluated prior to the training sequence when subjects were on and off drugs. With the exception of EMG, these findings will be reported at a later date.

The subject described in this report is one of 12 hyperkinetic subjects participating in baseline studies and is one of 4 hyperkinetics selected from the larger sample for biofeedback training. This subject's data have been extracted from an ongoing group design study because he has been in training for a significantly longer period of time than the others. The remainder of the report is confined to information applicable to this individual.

The subject, a male Caucasian of normal intelligence (Otis Lennon, form KE2, IQ = 104), was 11 years, 8 months old at the beginning of the biofeedback training period in October, 1974. Although his parents reported difficulty in managing his overactivity since he began walking at 15 months, he was first professionally diagnosed by a school psychologist when he entered elementary school in September, 1969 (6 years, 9 months). Two other local professionals, a child psychiatrist and a pediatrician, subsequently concurred in the diagnosis. The parents had been reluctant to employ drug therapy but finally conceded after unsuccessfully engaging in a series of alternative therapeutic endeavors. Ritalin (10 mg/school day) was prescribed in March, 1974 for a trial period. Although the usual procedure is to increase dosage until an optimal level for school performance is achieved, the pediatrician agreed to maintain this regimen after referring the subject for this study. Exceptions were made at the experimenters' request.

Experimental Design

Table I outlines the sequence of experimental conditions (Phases I–V) and the number of sessions per phase during which dependent changes in laboratory and classroom performance are monitored. Initial baselines under No Drug (I) and Drug Only (II) conditions were conducted in the absence of EEG biofeedback. Reference to these pertinent data permitted assessments of the training procedure's therapeutic effects whether the drug regimen is sustained or withdrawn. The same dosage levels were maintained during all phases (II–V) employing chemotherapy.

The feedback contingency implemented in Phase III rewards both the production of 12–14-Hz EEG activity and the inhibition of 4–7-Hz activity. Acquisition of this task (12–14 Hz + ; 4–7–) is followed by a contingency reversal (12–14 Hz–; 4–7 Hz +) in Phase IV. If pretraining performance levels under the Drug Only condition (II) are resumed, the feedback manipulation used in training is assumed to produce whatever changes are evidenced during that time. The original contingency (12–14 Hz + ; 4–7 Hz–) is reinstated during phase V. In a final phase (VI), which is currently in progress, the same EEG contingency (12–14 + ; 4–7 Hz–) is maintained after drug is withdrawn.

Apparatus and Procedures in Classroom Observations

Thirteen behavioral categories were selected as indices of overactivity and short attention span, the two fundamental characteristics of the syndrome (Stewart, Pitts, Craig, & Dierak, 1966). These categories were extracted from a larger sample described in detail by Wahler, who developed the category system (Wahler, House, & Stanbaugh, 1975). As described in the Results section, the categories have been adapted intact with two exceptions. Sustained attention was limited here to circumstances specifically related to school work; also, the out-of-seat category represented a novel category and was not scored separately in Wahler's system.

Ten-second observation intervals alternated with five-second recording intervals during each 30-minute session. The subject was observed for one week prior to the actual data collection in order to establish a reasonable level of reliability between independent observers and to adapt the class to their presence. Trial observations occurred during a 3-week adaptation period preceding phase I (No Drug) when the subject was withdrawn from medication. Experimental phases followed each other immediately with the exception of a 2-month interval between phases II and III.

Table I presents the total number of observation sessions in each phase. Daily sessions of 15 consecutive school days occurred at the end of each phase. The same observation sequence was repeated after every 2-month period in phase III. The subject was observed during one of two classes immediately following medication. Since medication was scheduled before morning and noon meals, approximately 45 minutes elapsed prior to the observation. Observations were conducted at the same time period under the No Drug condition.

Laboratory Apparatus and Procedures

Laboratory procedures and the basic apparatus for signal detection, analysis, and feedback have been extensively described in previous publications (Seifert & Lubar, 1975; Lubar & Bahler, 1976; Shouse & Lubar, 1976). Distinctive features of the procedures used in this study are summarized below.

EEG recordings were obtained from bipolar electrodes situated bilaterally at 10% and 30% of the distance between vertex and the preauricular point. These were the same coordinates used by Serman and Friar (1972) with an epileptic. EMG electrodes were placed 20 cm along the midline of the chin on the inferior and superior surfaces.

The following physiological responses were monitored during each 40-minute session: (a) 4–7-Hz events—each 4–7-Hz signal above 12.5 μ V;

(b) SMR events—each 12–14-Hz signal above 5 μ V occurring in the absence of 4–7-Hz events; (c) 6 SMR events occurring within a .5-second interval (burst); (d) EMG criteria—a predetermined number of EMG signals. EMG signals between 30 Hz and 300 Hz were passed through a Schmitt trigger with an adjustable threshold. Signals exceeding present threshold levels triggered logic pulses; they were fed into a digital integrator of our own design. The integrator automatically reset when predetermined criteria levels were reached. Response frequency measures for EMG criteria and both SMR indices were printed out on a teletype at one-minute intervals.

Baseline laboratory sessions under No Drug and Drug Only conditions were conducted 45 minutes after medication was scheduled on each weekend day during the course of the behavioral observation sequence. Training sessions occurred three times per week, during which scheduling difficulties precluded maintaining the 45-minute interval relative to drug intake.

As in previous studies, training sessions were subdivided into two 5-minute baseline periods and two 15-minute feedback periods, during which feedback presentations were confined to 4–7-Hz events and SMR bursts. The burst criterion used in this study corresponds to Sterman's definition of SMR (Sterman et al., 1974). Separate modes of feedback were uniformly employed for the production (correct light, tone, or money) and inhibition (incorrect light) of the target responses. The nature of the response to be produced or inhibited at any given time was dictated by the sequence of experimental conditions.

RESULTS

Laboratory Findings

Figure 1 presents evidence supporting the concept that contingent EEG feedback produced orderly changes in SMR production relative to daily and pretraining baselines and that corresponding changes in EMG occurred in the opposite direction as training progressed. SMR and EMG values during training sequence were averaged over 2-session blocks and represent the following ratios:³

³Although a 15-minute training period occurred for each hemisphere during each session, we have limited the daily feedback values to that hemisphere for which baseline data were also available. In addition, the final 5-minute baseline period is omitted because the number of SMR criterions met then is unusually high and presumably represent a carry-over effect from the preceding feedback period. A similar phenomenon occurs regularly in our laboratory with both epileptic and hyperkinetic subjects and has also been noted in some subjects studied in other laboratories (e.g., Sterman & Friar, 1972; Sterman et al., 1974). EMG values are restricted to the same time intervals.

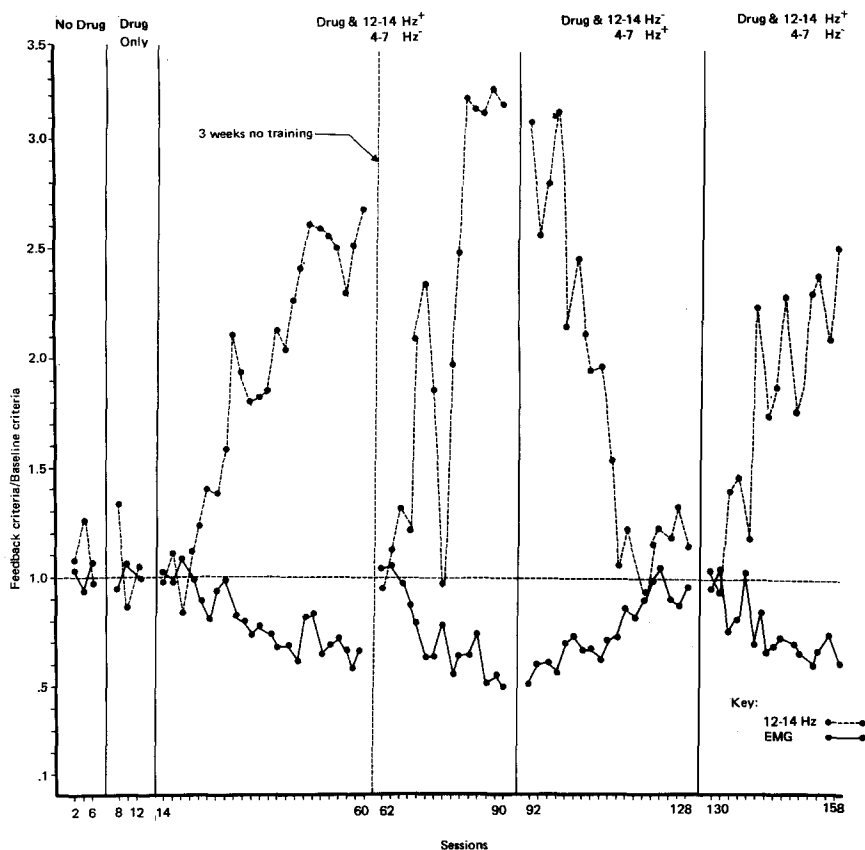


Fig. 1. SMR vs. EMG: Ratios of \bar{X} criteria met per minute during the initial 15-minute feedback period to \bar{X} criteria met per minute during the initial 5-minute baseline. Broken line between sessions 60 and 62 indicates 3-week period of no training.

$$\frac{\bar{X} \text{ criteria per minute during initial 15-minute feedback period}}{\bar{X} \text{ criteria per minute during initial 5-minute baseline period}}$$

Comparable values are provided for pretraining baseline sessions when no feedback is presented.

Relative to pretraining baselines, negligible training effects occur during early SMR training sessions and are successively replaced by substantial increases in SMR ratios. Pretraining performance levels are temporarily resumed following both a 3-week lapse in SMR training and the contingency reversal phase. Original training effects are recovered when the SMR contingency is reinstated. Finally, the inverse relationship between SMR and EMG is supported by moderate but statistically insignificant out-

comes in the brief pretraining phases (I: $r = -.31$, $F = .42$, $df = 4$, $p > .1$; II: $r = -.33$, $F = .49$, $df = 4$, $p > .1$)⁴ and by statistically significant ones during each training phase (III: $r = -.48$, $F = 11.50$, $df = 37$, $p < .01$; IV: $r = -.63$, $F = 10$, $df = 16$, $p < .01$; V: $r = -.65$, $F = 8.40$, $df = 12$, $p < .05$).

Figure 2 includes additional data supporting the conclusion that SMR production increased with biofeedback training. SMR values for the 10-minute baseline period and the initial 15-minute feedback period represent average percentages of seconds of 12–14-Hz activity and are derived from the following formula:⁵

$$\frac{\bar{X} \text{ instances 12-14-Hz activity/minute}}{13} \times \frac{100}{60}$$

Even though feedback was not presented for individual SMR instances, trends in this measure correspond to those for feedback criteria (SMR bursts) in Figure 1. The percent of 12–14-Hz activity generated during the No Drug phase increased during the Drug Only phase. A similar change in the absolute frequency of SMR bursts is concealed by the ratio transformation used in Figure 1.

Relative to pretraining sessions, training effects in Figure 2 were absent in the first few training sessions, after which feedback values regularly exceeded those for the baseline period. However, the difference between the two periods did not progressively increase with training time and was not statistically significant across either of the two SMR training phases (III: $F = .69$, $df = 37$, $p > .1$; V: $F = .91$, $df = 12$, $p > .1$). However, note that the greatest separation between baseline and feedback percentages for SMR occurred at the end of phase V between sessions 148 and 158. Nevertheless, the fact that the initial percentage of SMR activity was virtually tripled in both feedback and baseline periods by the end of the SMR training phases tends to suggest that a substantial baseline shift has masked learning effects rather than that learning has not taken place. This view is further strengthened by the overall decline in 12–14-Hz activity by the end of the contingency reversal phase. Furthermore, a demonstrable shift in baseline SMR appears to be essential if valid extrapolations from the laboratory to general experience are to be made.

⁴Correlations were computed across daily sessions in each pretraining phase.

⁵This conversion was partly justified on the basis of our detection system, which requires a signal duration from one-twelfth to one-fourteenth second for any wave to qualify as a discrete SMR instance. Assuming the center frequency of 13 Hz, each instance represents one-thirteenth second of 12–14-Hz activity.

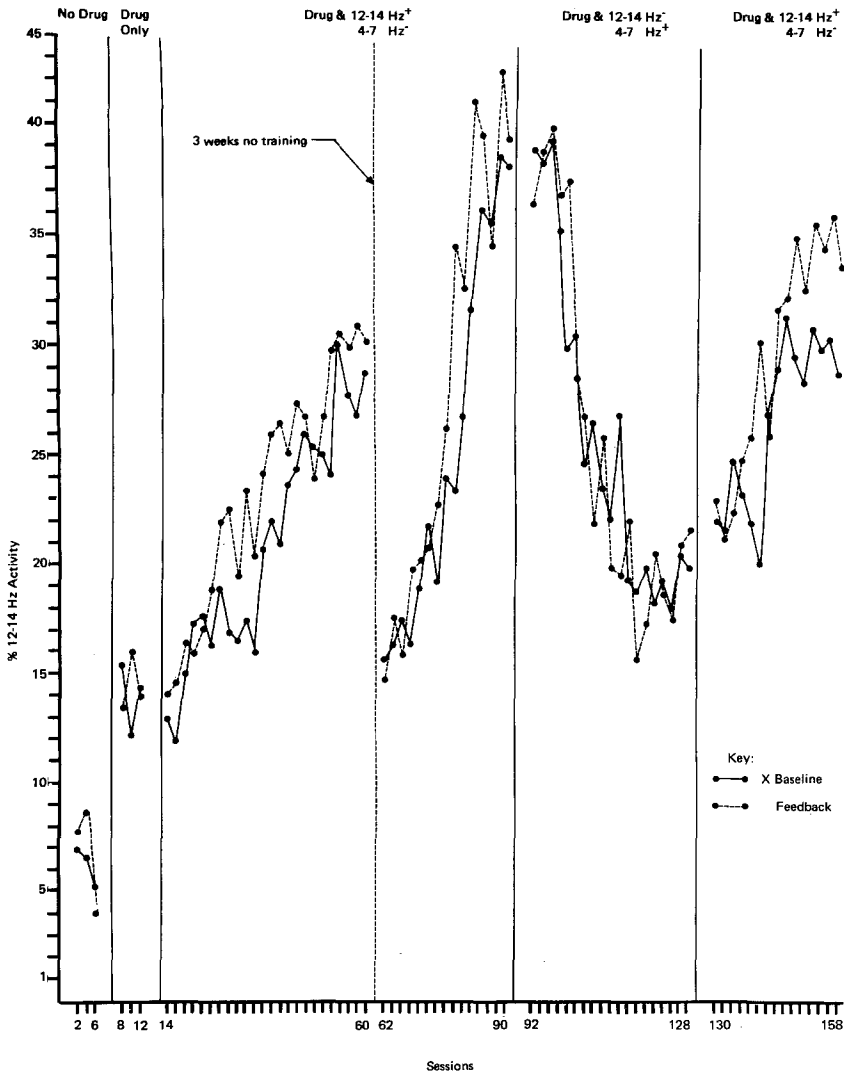


Fig. 2. Mean percent of seconds of 12-14-Hz activity per minute during initial 15-minute feedback period and 10-minute baseline period. Broken line between sessions 60 and 62 indicates 3-week period of no training.

Table II. Description of Behavior Categories Evaluated in the Classroom

Behavior categories and notations	Description
Undirected activities	
Self-stimulation (S)	<i>Scored</i> for any instance of the subject's manipulation of his own body, or for any instance of nonpurposive, nondirected motor or vocal actions
Object play (OP)	<i>Scored</i> for any instance of simple repetitive manipulation of an object, in which the manipulation of an object is not part of a more complex activity. <i>OP</i> activities are usually regarded as a form of "absent-minded" fidgeting with objects
Self-talk (T)	<i>Scored</i> for any instance of intelligible verbalizations not directed to another person
Sustained noninteraction (NI)	<i>Scored</i> when the subject does not interact with any objects or people for a <i>full</i> interval. Object interactions which qualify as <i>OP</i> can be scored in conjunction with <i>NI</i> , as can <i>S</i> , <i>I</i> , and compliance and opposition when these behaviors do not accompany interactions
Cooperation and opposition	
Compliance (C)	<i>Scored</i> for any instance of obedience to any instruction
Opposition (O)	<i>Scored</i> for noncompliance to rules or instructions imposed by adults
Schoolwork	
Sustained school work (SS)	<i>Scored</i> for a full interval of uninterrupted school related activity such as reading or writing. Attending to a teacher's explanations or work on the blackboard is scored <i>SA</i> , not <i>SS</i>
Sustained attention (SA)	<i>Scored</i> for a full interval of uninterrupted attending to or staring at people or activities. The object of attention must be specifically related to school work
Social activity	
Social approach (Ac) (Aa)	<i>Scored</i> for any instance of deliberate physical contact, directed verbalizations, or other nonverbal methods used by the subject to attract another's attention or to initiate an interaction. The subscript indicates approach to an adult (Aa) or a child (Ac)
Social interaction (SIc) (SIa)	<i>Scored</i> for any instance of the subject's interaction with other people. Interaction in this case implies that the behaviors of the subject and the other person are directed to each other, and thus are interdependent. If an interaction initiated by the subject (<i>A</i>) is carried <i>uninterrupted</i> into subsequent intervals, it is then scored as social interaction. When another person initiates an interaction with the subject it is scored <i>SI</i> in the first and all following intervals. An ongoing interaction involving the subject is continuously scored <i>SI</i> until it is obvious that the subject has terminated his participation. The subject identifies the other person as an adult (SIa) or a child (SIc)
Other categories	
Out-of-seat (OS)	<i>Scored</i> for any instance in which the subject is out of his seat. All other categories may be scored in conjunction with this one

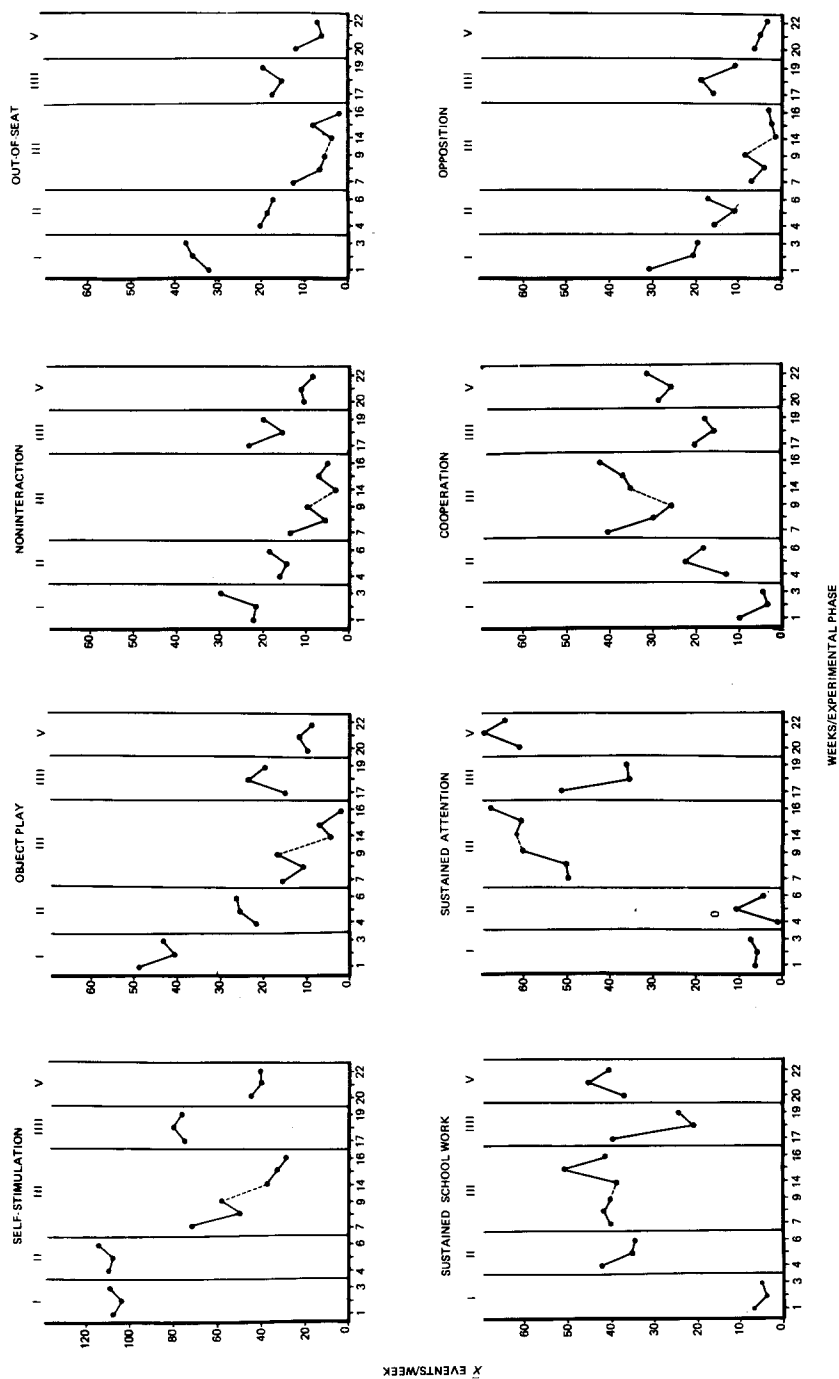


Fig. 3. Comparison of mean activity levels per week under five conditions. Key: I, No Drug; II, Drug Only; III, Drug and 12-14 Hz+, 4-7 Hz-; IIII, Drug and 12-14 Hz-, 4-7 Hz+; V, Drug and 12-14 Hz+, 4-7 Hz-.

Classroom Observations

The behavioral categories assessed in the classroom are described in Table II. Figure 3 presents the average number of daily events scored per week for selected categories under 5 conditions. Reliabilities were computed for 2 independent observers in half the sessions in each condition. Using the formula

$$\frac{\text{\#categories of agreement}}{\text{\#categories of agreement and disagreement}}$$

the average reliabilities per session were 83%, 88%, 92%, 90%, and 85% in the 5 conditions, respectively.

Combining medication and SMR training was intended to enhance the level of improvement already achieved with drugs alone. The 8 behaviors included in Figure 3 exhibit predicted changes during SMR training. Relative to the No Drug phase, improvement in 6 of those categories occurred during the Drug Only phase. The most dramatic effect occurred in the classroom oriented toward academic materials (sustained school work) to the relative exclusion of attending to lectures (sustained attention). The slight but unanticipated increase in self-stimulation in phase II may be accounted for by the rather exclusive increase in classwork with which it was usually related positively (Wahler, 1975). When Drug and SMR were combined, even further improvement occurred in those behaviors benefited by medication, and substantial change also was evidenced in the two that were not (i.e., self-stimulation and sustained attention).

The 5 behaviors omitted from Figure 3 failed to change in the predicted direction during SMR training. One undirected activity (self-talk) and all 4 social behaviors occurred at moderate or high frequencies during the No Drug phase. Uniform decreases in them following Drug Only were reversed during SMR training. No explanation is currently available for this trend in self-talk. However, the unanticipated increase in social behavior may have resulted from the subject's being reinstated into work groups with other children throughout all phases of biofeedback training. Under the first 2 conditions during the baseline sequence the child was isolated from the remainder of the class in order to restrict his social interactions.

DISCUSSION

Acquisition of the SMR task was indicated by contingent increases in SMR production during feedback periods relative to daily and pretraining baselines. Corresponding decreases in EMG tended to support the view that

SMR is correlated with motor inhibition. Also, baseline shifts in the percent of 12–14-Hz EEG activity over the training period strengthen the basis for our postulating a carry-over effect outside the laboratory setting.

Objective measures of overactivity and distractibility in the classroom further support this view. At least 8 of the total 13 behaviors changed in the predicted direction under the two treatment conditions. Generally, decreases in undirected activities, out-of-seat, and oppositional behaviors were accompanied by increased cooperation and school work. Relative to the No Drug condition, the improvement shown with Drug Only tends to assure the validity and reliability of these behavior categories in assessing the hyperkinetic disorder, since Ritalin is currently considered to be the most effective treatment (e.g., Millichap, 1968). The effectiveness of the biofeedback technique is supported by the fact that the combined effects of drug administration and SMR training result in substantial improvement above and beyond the effect of drug alone. Also, the reversal of positive changes following SMR counterconditioning minimizes the possible role of extraneous influences (e.g., maturation) on treatment outcomes.

We are now in the process of replicating these preliminary results for three additional hyperkinetic children. So far, it appears that SMR biofeedback is most beneficial for those children in whom the hyperkinetic symptoms are the most pronounced. Although our initial findings are encouraging, a more complete evaluation of the technique will be possible after medication has been withdrawn. Finally, a larger subject population is required before any substantive conclusions can be drawn about the clinical utility of this new methodology.

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REFERENCES

- Chase, M. H., & Harper, R. M. Somatomotor and visceromotor correlates of operantly conditioned 12–14 C/sec sensorimotor cortical activity. *Electroencephalography and Clinical Neurophysiology*, 1971, 31, 85–92.

- Donhoffer, H., & Lissak, K. EEG changes associated with the elaboration of conditioned reflexes. *Acta Physiologica Academy of Science, Hungary*, 1962, 21, 249-255.
- Finley, W. W., Smith, H. A., & Etherton, M. D. "Reduction of seizures and normalization of the EEG in a severe epileptic following sensorimotor biofeedback training: Preliminary study. *Biological Psychology*, 1975, 2, 195-209.
- Howe, R. C., & Sterman, M. B. "Cortical-subcortical EEG correlates of suppressed motor behavior during sleep and waking in the cat. *Electroencephalography and Clinical Neurophysiology*, 1972, 32, 681-695.
- Lubar, J. F., & Bahler, W. W. Behavioral management of epileptic seizures following EEG biofeedback training of the sensorimotor rhythm. *Biofeedback and Self-Regulation*, 1976, 1, 47-61.
- Millichap, J. G. Drugs in the management of hyperactive children. *Journal of American Medical Association*, 1968, 206, 1527-1530.
- Roth, S. R., Sterman, M. B., & Clemente, C. D. Comparison of EEG correlates of reinforcement, internal inhibition and sleep. *Electroencephalography and Clinical Neurophysiology*, 1967, 23, 509-520.
- Seifert, A. R., & Lubar, J. F. Reduction of epileptic seizures through EEG Biofeedback Training. *Biological Psychology*, 1975, 3, 157-184.
- Shouse, M. N., & Lubar, J. F. Management of the hyperkinetic syndrome in children concurrent with sensorimotor rhythm (SMR) biofeedback training. *Proceedings of the Biofeedback Research Society*, Colorado Springs, Colorado, 1976.
- Sterman, M. B., & Friar, L. Suppression of seizures in an epileptic following sensorimotor EEG feedback training. *Electroencephalography and Clinical Neurophysiology*, 1972, 33, 89-95.
- Sterman, M. B., LoPresti, R. W., & Fairchild, M. D. Electroencephalographic and behavioral studies of monomethylhydrazine toxicity in the cat. *Technical Report AMRL-TR-69-3*, Aerospace Medical Research Laboratories, Aerospace Medical Division Air Force Systems Command, Wright-Patterson Air Force Base, Ohio, 1969.
- Sterman, M. B., Macdonald, L. R., & Stone, R. K. Biofeedback training of the sensorimotor electroencephalographic rhythm in man: Effects on epilepsy. *Epilepsia*, 1974, 15, 395-416.
- Sterman, M. B., & Wyrwicka, W. EEG correlates of sleep: Evidence for separate forebrain substrates. *Brain Research*, 1967, 6, 143-163.
- Stewart, M. A., Pitts, F. N., Craig, A. G., & Dierak, W. The hyperactive child syndrome. *American Journal of Orthopsychiatry*, 1966, 36, 861-867.
- Wahler, R. G. Some structural aspects of deviant child behavior. *Journal of Applied Behavior Analysis*, 1975, 8, 27-42.
- Wahler, R. G., House, A. E., & Starbaugh, E. E. *Ecological assessment of child problem behavior*. Pergamon Press. In press, 1975.

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