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

A Preliminary Report[1]

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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validity and generality of the findings and will include the use of SMR biofeedback training after medication has been withdrawn.

[1]This 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, Sterman, & Clemente, 1967; Sterman & 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 (Sterman, LoPresti, & Fairchild, 1969), several investigators have reported marked decreases in seizure frequency following biofeedback training in various forms of human epilepsy (Sterman, 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; Sterman & Wyrwicka, 1967; Howe & Sterman, 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 denervated (Sterman 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	II	III	IV	V
	No Drug	Drug Only	Drug and SMR training I	Drug and SMR reversal training	Drug and SMR training II
EEG sessions	6	6	78	36	28
Behavioral sessions	15	15	45	15	15

[a] See 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 Sterman 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 μV;

(b) SMR events—each 12–14-Hz signal above 5 μV 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]

[3]Although 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.

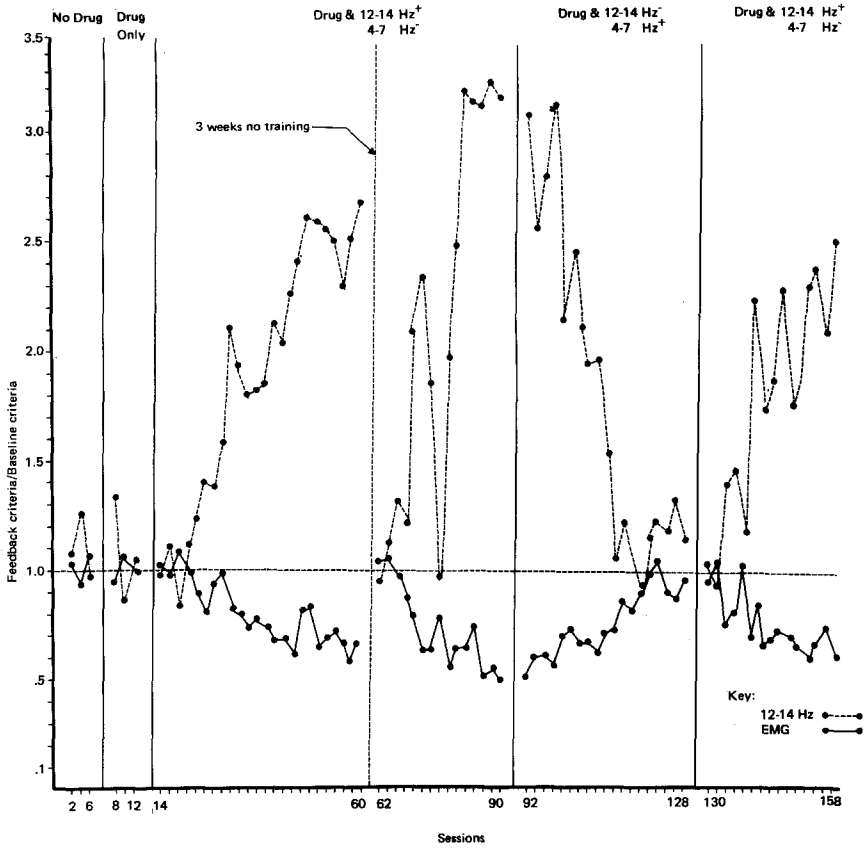

Fig. 1. SMR vs. EMG: Ratios of $\bar{X}$ criteria met per minute during the initial 15-minute feedback period to $\bar{X}$ criteria met per minute during the initial 5-minute baseline. Broken line between sessions 60 and 62 indicates 3-week period of no training.

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

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 criterions (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.

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

[5]This 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.

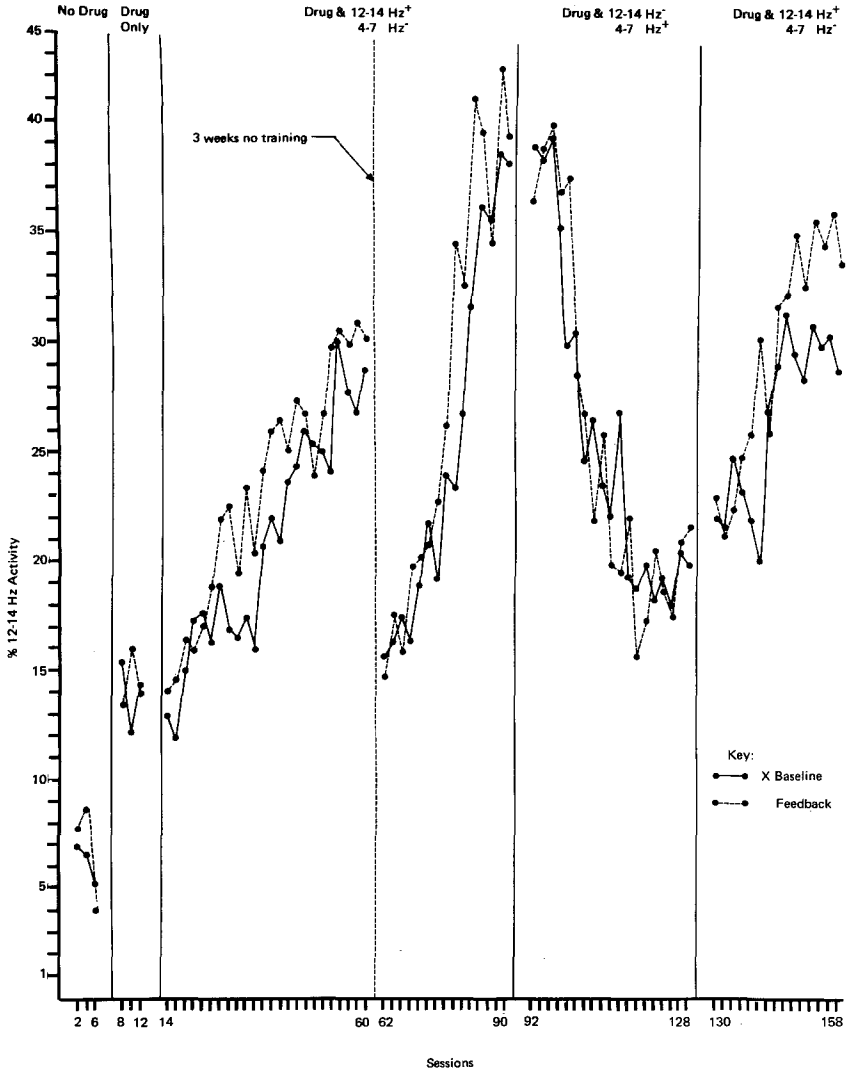

Fig. 2. Mean percent of seconds of 12–14-Hz activity per minute during initial 15-minute feedback period and 10-minute baseline period. Broken line between sessions 60 and 62 indicates 3-week period of no training.

Table II. Description of Behavior Categories Evaluated in the Classroom

Behavior categories and notations	Description
Undirected activities	
Self-stimulation (S)	*Scored* 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)	*Scored* 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. *OP* activities are usually regarded as a form of "absent-minded" fidgeting with objects
Self-talk (T)	*Scored* for any instance of intelligible verbalizations not directed to another person
Sustained noninteraction (NI)	*Scored* when the subject does not interact with any objects or people for a *full* interval. Object interactions which qualify as OP can be scored in conjunction with *NI*, as can *S*, *I*, and compliance and opposition when these behaviors do not accompany interactions
Cooperation and opposition	
Compliance (C)	*Scored* for any instance of obedience to any instruction
Opposition (O)	*Scored* for noncompliance to rules or instructions imposed by adults
Schoolwork	
Sustained school work (SS)	*Scored* 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 *SA*, not *SS*
Sustained attention (SA)	*Scored* 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)	*Scored* 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)	*Scored* 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 (*A*) is carried *uninterrupted* into subsequent intervals, it is then scored as social interaction. When another person initiates an interaction with the subject it is scored *SI* in the first and all following intervals. An ongoing interaction involving the subject is continuously scored *SI* 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)	*Scored* for any instance in which the subject is out of his seat. All other categories may be scored in conjunction with this one

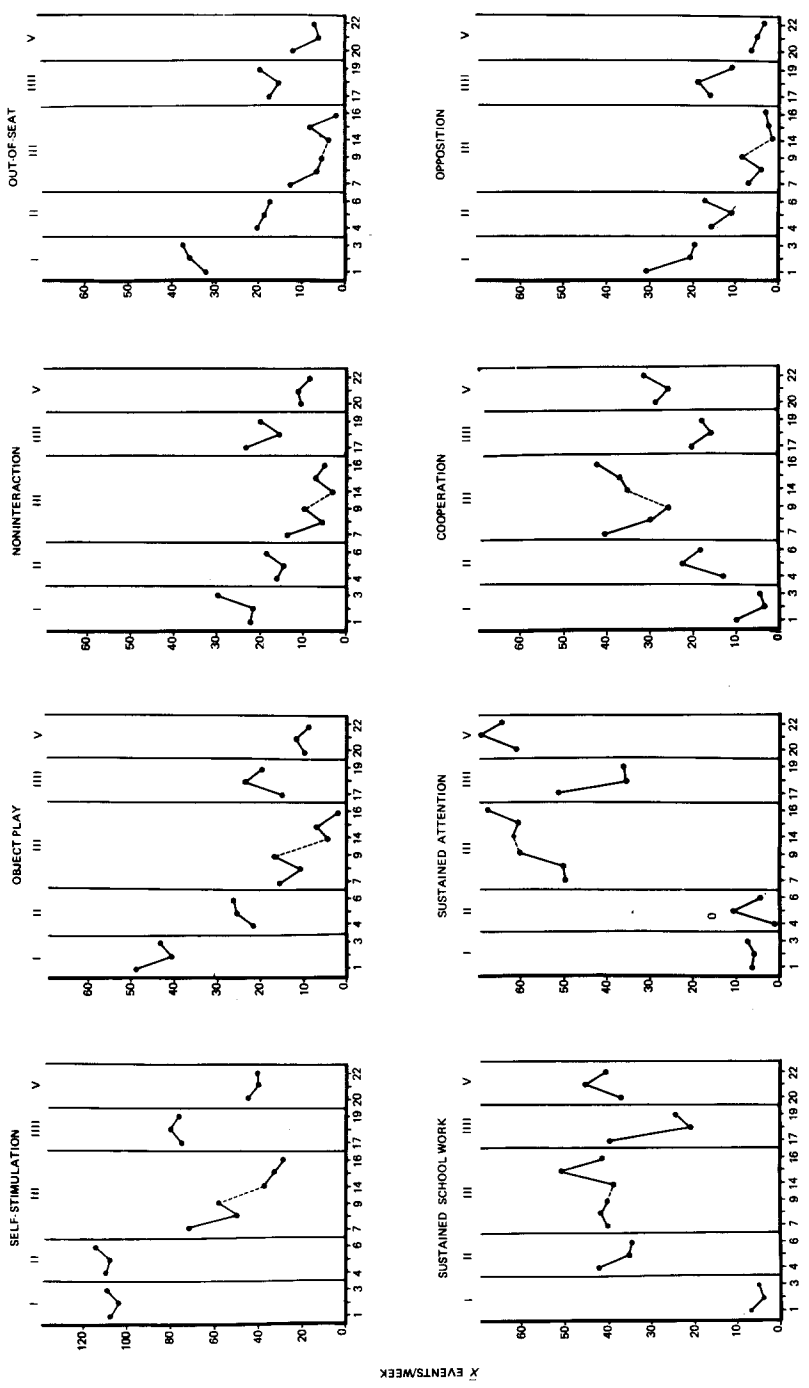

Fig. 3. Comparison of mean activity levels per week under five conditions. Key: I, No Drug; II, Drug Only; III, Drug and 12–14 Hz+, 4–7 Hz–; IIII, Drug and 12–14 Hz–, 4–7 Hz+; V, Drug and 12–14 Hz+, 4–7 Hz–.

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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