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THE ROLE OF CNS AROUSAL LEVELS IN THE MANAGEMENT OF HYPERKINESIS: METHYLPHENIDATE AND EEG BIOFEEDBACK TRAINING

A Dissertation

Presented for the

Doctor of Philosophy

Degree

The University of Tennessee, Knoxville

Margaret Nichols Shouse

August 1976

1288364

ACKNOWLEDGEMENTS

The author gratefully acknowledges the assistance of Jasper Brener, who contributed substantially to the development of the original research proposal and its design; the members of my doctoral committee, Joel F. Lubar, Chairman, William Calhoun, Robert G. Wahler, and Michael Vey Nordquist, for their steadfast supervision of the project; Dr. Hammond Pride and Dr. Robert Proffitt for referring the hyperkinetic subjects; the teachers of hyperkinetic subjects for nominating normal control children and for cooperating with my classroom observer teams; Ms. Rene and ORTEC Corporation (Oak Ridge, Tennessee) for their help in developing the research apparatus; John Maltry and Bret Boring for technical assistance in the laboratory; Jack Dryden, Steven Shoaf, and the several undergraduate assistants who collected the behavioral observations; and Ms. Dianna Wolfenbarger who has typed this manuscript.

ABSTRACT

Research over the past five years suggests that central nervous system (CNS) arousal level moderates both the severity of the hyperkinetic disorder in children and the degree of therapeutic success anticipated following stimulant drug therapy. It was hypothesized that low and high arousal groups of hyperkinetic children should be differentiated from normal controls on a variety of physiological arousal measures. Relative to high arousal subjects, low arousal subjects were expected to display the most severe pre-treatment behavioral profiles and to benefit most from stimulant drug therapy. Both groups were expected to benefit from biofeedback training of the sensorimotor rhythm (SMR), a 12-14 Hz EEG rhythm recorded over Rolandic cortex. Considerable research associates conditioned increases in this rhythm with voluntary motor inhibition and with the normalization of various EEG and autonomic indices of arousal.

Subjects were twelve hyperkinetic and twelve normal children matched for age, sex, and IQ. CNS arousal indices included the amplitude of auditory evoked cortical responses, the incidence of SMR, the amount of slow wave activity in the background electroencephalogram (EEG), and basal skin resistance (GSR). Indices of somatomotor activity included measures of muscular tone (Electromyogram or EMG), the Stewart teacher questionnaire,

and behavioral assessments of short attention span and overactivity in the classroom. The experiment consisted of six phases: I. NO DRUG; II. DRUG ONLY; III. DRUG AND SMR training I; IV. DRUG AND SMR reveral training; V. DRUG AND SMR training II; VI. NO DRUG AND SMR training. All measures were obtained for each subject during pre-training phases I and II and at the end of training phase VI. SMR, background EEG, EMG, and classroom behaviors were systematically recorded in training subjects throughout the study. GSR and evoked cortical responses were also obtained at the end of the first training period (III).

High arousal subjects could not be differentiated from the remainder of the hyperkinetic population in pre-training phases I and II. Subjects participating in the biofeedback training sequence were four low arousal hyperkinetic subjects who differed from normal control subjects and the remaining hyperkinetic subjects (hyperkinetic control subjects) in the predicted direction on all measures in the NO DRUG condition (I) and whose physiological and behavioral profiles during the DRUG ONLY phase (II) reflected significant but incomplete remission of symptoms. Constant drug regimens (Ritalin) were maintained during phases II-V and were gradually withdrawn (5mg/week) during phase VI.

The results of several months of SMR training tend to corroborate and extend previous findings. Substantial increases in SMR occurred with progressive training in three

of four subjects. Enhanced motor inhibition in the laboratory and classroom accompanied SMR training and was diminished following successful SMR reversal training. The effectiveness of the biofeedback technique is supported by the fact that combining medication and SMR training resulted in substantial improvement above and beyond the effects of drugs alone and that these effects were maintained following withdrawal of the drug. Anticipated changes in evoked cortical responses, GSR, and background EEG during training (III) and at the end of the experiment (VI) suggested increases in CNS arousal level. Three findings minimize the role of extraneous influences such as maturation on treatment outcomes: 1) the reductions in positive treatment effects following SMR counterconditioning; 2) the significant post-treatment (VI) improvement from DRUG ONLY (II) in low arousal subjects relative to changes over that period in hyperkinetic and normal control subjects; and 3) the selectivity of changes exhibited in effectively trained subjects. With the exception of GSR, teacher reported symptoms, and behavioral measures of distractability, subjects demonstrating acquisition of training could not be distinguished behaviorally or physiologically from normal controls at the end of the experiment.

Individual differences in training subjects were discussed in terms of pre-treatment physiological and behavioral profiles. The subject who failed to demonstrate

acquisition differed from the others in being excessively distractible. Otherwise, the similarity between effectively trained subjects is encouraging. Generally, subjects exhibiting the most severe pre-treatment arousal and behavioral profiles responded most favorably to both treatments. The lowest pre-treatment incidence of SMR and highest levels of motor excitability most effectively predicted subsequent response to treatment. These findings reaffirm the relationship between SMR and motor inhibition and suggest that the arousal changes observed may be a secondary outcome of motor control. Nevertheless, a much larger population of hyperkinetic subjects is required before any substantive conclusion is possible about the technique's utility or its basis in central mechanism of arousal or motor control.

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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

Introductory Statement

In the past five years, evidence has accumulated to suggest that hyperkinetic children with low CNS arousal levels display more severe overactivity than other children diagnosed with the disorder and that the administration of stimulant drugs effectively reduces hyperactivity in these children by enhancing their physiological arousal level. Establishing CNS arousal level as a moderating influence in the disorder may therefore account for the paradoxical calming effects associated with stimulant drugs administration in some hyperkinetic children and may permit more reliable predictions about these drugs' successful clinical application to the disorder. However, even if stimulant drugs were demonstratably more effective in hyperkinetic children with reduced arousal level, the role of arousal in this outcome would remain uncertain since these drugs are known to concurrently affect both arousal and motor symptoms (e.g., Millichap, 1968). A more conclusive assessment of CNS arousal functions in hyperkinesis is proposed here by conditioning increases in the somato-motor rhythm (SMR), an EEG rhythm associated with changes in CNS arousal measures as well as with enhanced peripheral

motor inhibition. A positive treatment outcome with SMR biofeedback training would provide a set of therapeutic procedures independent of the drug issue and perhaps of independent value when the use of drugs is contraindicated.

The goals of the project are:

1) to divide hyperkinetic subjects on the basis of CNS arousal level;

2) to demonstrate more severe pre-treatment over-activity and better treatment effects with stimulant drug (Ritalin) medication in subjects with reduced CNS arousal level than in subjects with elevated CNS arousal level;

3) to show greater treatment effects in low and high arousal hyperkinetic subjects with drugs and SMR training than can be obtained with medication alone;

4) to demonstrate that positive treatment effects can be maintained with SMR training after medication is withdrawn.

The remainder of the chapter focuses on three topics. First, some background information on hyperkinesis is provided to emphasis a number of current controversies about the disorder's symptoms, treatment, and etiology. Second, the empirical basis for the CNS arousal hypothesis is reviewed, and several insubstantial features are identified. Finally, a resolution of these insubstantial features is proposed through the investigation conducted here.

Current Status of the Hyperkinetic Disorder: Symptoms, Etiology, and Treatment

The lack of agreement on the disorder's identifying features is reflected in estimates of its prevalance, which currently range from 5% to 22% of all elementary school children (e.g., Minskoff, 1973). Nevertheless, a uniform set of criteria may be derived from those characteristics most frequently agreed on by researchers. These include: 1) consensus of parents, teachers, and pediatricians on the coincidence of the two fundamental symptoms, undirected, ceaseless motor activity and abbreviated attention span (Diagnostic and Statistical Manual of Mental Disorders, 1968); 2) the gender of the hyperkinetic population, which is predominately male (e.g., Ounsted, 1955); 3) the course of the syndrome, which typically spans the six-to-twelve age range (Menkes, et al. 1967); 4) and the absence of specific sensorimotor deficits and other functional or physical handicaps (e.g., mental retardation), through which the syndrome's manisfestations may be simulated. Adopting such criteria not only appears justified on the basis of the available normative data (e.g., Ounsted, 1955), but should also yield incidence estimates of hyperkinesis in a more conservative and manageable range. Nevertheless, such prerequisites are infrequently employed in a systematic fashion, and the resulting confusion has especially

applied to organic orientations towards the disorder (Werry and Sprague, 1970).

Etiology

The disorder's etiology has been ascribed to malfunctions ranging in nature from pre- and/or postnatal afflictions to abnormal interpersonal relationships (Greenberg et al., 1970). Despite the diversity of opinion, an organic substrate for hyperkinesis has been customarily assumed, and chemotherapy has been the preferred treatment. Consequently, the remaining discussion focuses on the physiological basis for the disorder.

Historically, attention to hyperkinesis as a brain damage syndrome emanates from the frequency with which overactivity characterized postencephalitic cases in children following the St. Louis epidemic in the 1920's (Brain and Walton, 1969). The disorder's traditional association with brain damage is exemplified by frequent allusions in the medical literature to Minimal Brain Dysfunction (MBD) and hyperkinesis as interchangeable labels for the syndrome. This practice may be attributed to the following findings relating hyperkinetic subjects to the normal population: a 52% coincidence of a history of the disorder between children raised with real parents and siblings raised in foster settings in contrast to a 15% coincidence of the disorder between

hyperkinetic children and "foster" siblings with whom they were raised; a higher coincidence between hyperkinetic behavioral manifestations and a specific history of generalized brain damage, particularly encephalitis (Hohman, 1922); a higher probability for hyperkinesis to be diagnosed in males, suggesting sex-linked holandric transmission; a higher coincidence between a history of pre- and perinatal birth complications and subsequent diagnoses as hyperkinetic (Stewart et al., 1966); a 65% accuracy of differential MBD diagnosis on the Reitan battery relative to normal children and children with severe cases of brain damage (Reitan and Boll, 1973). Such evidence strongly suggests that brain damage and hyperkinetic symptoms are highly correlated and possibly of a genetic nature.

Nevertheless, it remains that until 1975 an epidemic encephalitis lethargica had not occurred in this country since 1930 (Brain and Walton, 1969). During the same time frame, the number of children diagnosed with MBD has not subsided proportionately. Also, even though a significantly higher percentage of hyperkinetic subjects display specific neurological diseases and soft neurological signs than do normal ones (e.g., Satterfield, 1973), the coincidence of hyperkinesis and other disorders such as encephalitis (Bond and Smith, 1935) and epilepsy (Ounsted, 1955), or pre- and perinatal incidents such as accidental poisoning during early childhood (Stewart, 1966) have rarely accounted

for a very large proportion of the children diagnosed as hyperkinetic. In addition, the empirical basis on which hereditary estimates are based is extremely meager. No studies have been conducted on twins and fewer than 25 families participated in the comparative study cited above on hyperkinetic children raised either in foster homes of by their natural parents (e.g., Omenn, 1973; Wender, 1971; Stewart, 1970; Eisenberg, 1972). Finally, although Reitan and Boll (1973) have successfully identified hyperkinetic symptoms with moderate but significant deficits on Reitan's battery, they failed to include a control group of children displaying a history of disruptive behavior problems typically dissociated with neurological origins. Knights and Tymchuk (1969) were unable to differentiate two such groups on the Halstead Categories Test which is a highly reliable diagnostic sub-test of the Reitan examination. Until some clarification of this data is provided, only an insubstantial case can be made for brain damage as a necessary or even likely concomittant of hyperkinesis.

Therapy

The weak basis for brain damage as a causal factor in hyperkinesis further obscures the puzzling outcomes reported with chemotherapy. The fact that sedatives exacerbate symptoms in hyperkinetic subjects apparently

contradicts the somnolent effects otherwise obtained with these drugs. In contrast, the unprecedented therapeutic success of stimulant drugs such as methylphenidate and the amphetamines is antithetical to the presumed excitatory nature of the disorder (e.g. Millichap, 1968). On the other hand, the paradoxical stimulant drug effect does not appear to be a completely generalized phenomenon in the disorder since moderate therapeutic benefits with tranquilizing agents have been reported in hyperkinetic children for whom stimulant drugs were either not administered or unsuccessfully applied (e.g. Millichap, 1973).

Such evidence on the disorder's nature, symptoms, and treatment hardly yields a uniform profile of the syndrome. Although few would speculate that all diagnosis cases of hyperkinesis are properly admissable under the same rubric, one recently identified moderator variable, CNS arousal level, may elucidate many of the controversies.

CNS Arousal Level As An Integrative Mechanism in Hyperkinesis: A Critical Evaluation

Supporting Evidence

The relevance of different behavioral and physiological arousal levels in the disorder is supported by the following: Rate-dependency findings with animals subjects; electrophysiological research with hyperkinetic children

prior to and following stimulant drug therapy; findings relating EEG slow wave activity to CNS pathology; and findings relating to CNS arousal measures as a group to mental disorders in general.

Rate-dependency findings. The "paradoxical" outcome of stimulant drug treatment has been somewhat clarified by animal investigations of drug effects on barpressing as a function of baserate. According to Stretch and Dalrymple's (1968) review, for example, amphetamine and methylphenidate have systematically increased initially low operant levels while the performance of subjects having comparatively higher operant levels was depressed. These data may provide a behavioral analogue for stimulant drug effects on hyperkinesis; nevertheless, their therapeutic value has remained a physiological anomaly until recent investigations by Satterfield and his associates, who inadvertently elaborated the scope of the animal studies to permit at least a partial reconciliation of the physiological and behavioral data in humans.

Studies differentiating hyperkinetic subject as a function of CNS arousal level. Satterfield (Satterfield et al., 1972) has suggested two sub-groups of hyperkinetic subjects, one having reduced CNS arousal and the other displaying heightened CNS

arousal levels. Excitatory behavioral manifestations in low arousal subjects are presumed to reflect the over-compensatory behavior of an otherwise sluggish organism. In high arousal subjects, on the other hand, excessive motor activity is presumably commensurate with the excitable state of the nervous system.

In a study comparing hyperkinetic control subjects with the two arousal sub-classes of hyperkinetic children, data supporting a low CNS arousal hypothesis in some hyperkinetic subjects included the following: the high incidence of synchronized slow wave activity in the waking EEG, suggesting low arousal since alertness is typified by faster, low amplitude waves (e.g., Penfield and Jasper, 1954); the relatively depressed amplitude and incidence of tonic GSR, indicating reduced sympathetic and reticular arousal (Duffy, 1962); and the enhanced amplitude of auditory evoked cortical responses, reflecting relaxation and reduced alertness (Guerrero-Figuero and Health, 1964; Milner, 1970) and possibly abbreviated attention span (Satterfield, 1965). Subjects deviating from controls in the low arousal direction showed highest baserate levels of somatomotor excitation and benefitted most from methylphenidate (Ritalin) therapy. Their post-treatment profile, including CNS arousal measures and teacher ratings of behavior pathology, most closely approximated normality in that both moderate changes towards increased arousal and substantial decreases in behavior pathology (71%) resulted.

The second sub-group, on the other hand, exhibited the highest baserate level of CNS arousal, as indexed by low amplitude, high frequency peaks in the background EEG, elevated GSR conductance, and high amplitude evoked cortical responses; showed the least behavioral disturbance; and responded less well, if not unfavorably, to methylphenidate therapy. Post-treatment data indicated either unchanged or exacerbated behavioral disturbance combined with exceedingly reduced CNS arousal. In fact, the physiological outcome was so extreme in the originally high arousal hyperkinetics that their post-treatment profile most closely resembled the initially low CNS arousal group.

These data corroborate Stretch and Dalrymple's (1968) findings with animal subjects by demonstrating that stimulant drug treatment in humans may affect skeletal activity either by decreasing high baserate levels or by sustaining and possibly increasing relatively low baserates. Furthermore, analogous drug effects on GSR and EEG baserates implicate levels of CNS arousal as a physiological mediator for differing drug effects on hyperkinetic behavior.

Additional data lend credence to this conceptualization, although in a more piecemeal fashion. In an earlier study by Satterfield and Dawson (1971), low and high arousal hyperkinetic subjects were also distinguished on the basis of GSR recordings. Due to the paucity of

high CNS arousal subjects identified in this study, a separate data analysis on that group was not undertaken; however, in low CNS arousal subjects, depressed tonic GSRs and specific GSRs to novel stimuli during baseline were increased following methylphenidate administration to the experimental group but were sustained following placebo administration to a matched hyperkinetic control group. A compelling adjunct to the data is provided by a parenthetical observation in this study in which two low arousal subjects asked to remain still tended to fall asleep when they complied with the experimenter's instructions.

The arousal conceptualization has most recently been couched in a developmental context, in which the syndrome's clinical manifestations presumably result from a delay in CNS maturation that may or may not be associated with a documented history of brain damage (Satterfield et al., 1973; Wender, 1973). This so-called maturational lag hypothesis is not undermined by the opposing physiological characteristics displayed by the two arousal classes since this dissociation is not incompatible with longitudinal studies of the electroencephalogram. Antecedent conditions for both developmental trends are illustrated by increasing amplitudes in auditory evoked responses and background EEGs during the first years of life (e.g., Orlich et al., 1972), after which progressive amplitude reductions in both indices characterize the remainder of childhood. Although there is more evidence of arousal differences in the currently existing hyperkinetic population than of

their developmental origin per se, Connors (Connors, 1973) has reported that the high amplitude evoked cortical responses typical of hyperkinetic children in the low arousal class are significantly more resistant to the reductions associated with normal maturation. In support of a developmental basis for the reduced EEG amplitudes evidenced by high arousal children, Satterfield (Satterfield et al., 1973) noted qualitative EEG characteristics such as irregularities in wave forms that are more reminiscent of a poorly developed EEG than of its deterioration.

EEG slowing and CNS pathology. Satterfield's data are consistent with the general view that excessive synchronized EEG slowing represents inhibition of cortical activity. The subsequent release of subcortical structures from cortical control presumably accounts for a variety of abnormal symptoms, such as the convulsive motor activity typical of grand mal seizures (Gastaut and Fischer-Williams, 1959). In this context, Stevens and colleagues (Stevens et al., 1968) previously noted that the degree of somatomotor excitation varies directly with the amount of slow wave activity in the background EEG's of hyperkinetic children. A subsequent study by Satterfield (Satterfield et al., 1973; Satterfield, 1973) confirmed the utility of EEG slowing in predicting responsiveness to methylphenidate therapy, especially when this symptom is accompanied by neurological signs of

motor disturbance (e.g., reflex asymmetries, synkinesis, incoordination in the upper extremities, etc.). It is also interesting to note that the evoked cortical potentials obtained in this study (refer to both 1973 reports) were again consistent with amplitude characteristics of the background EEG data since this is characteristic of the evoked cortical response-background EEG relationship (Handbook, 1972) and since Conners (Conners, 1973) has reported two studies substantiating claims that evoked cortical response amplitudes significantly differentiate hyperkinetic subjects from normal control subjects.

CNS arousal measures and mental disorders in general. Finally, regarding GSR and the EEG measures as a class of arousal indices, all of these data coincide with findings that arousal measures, especially taken in concert, consistently differentiate individuals along an abnormal-normal continuum. Generally speaking, subjects diagnosed with mental disorders of all kinds display greater lability in arousal than do normal ones, although the specificity with which distinctions between syndromes may be made on the basis of arousal profiles is questionable (Duffy, 1962). Satterfield has marshalled considerable support for the reliability of these measures in discriminating both sub-classes of hyperkinetic subjects from hyperkinetic control subjects (Satterfield and Dawson, 1971; Satterfield et al., 1972; Satterfield et al., 1973; Satterfield, 1973)

and hyperkinetic from normal subjects (Satterfield and Dawson, 1971; Satterfield et al., 1973).

Insubstantial Features

In spite of these suggestive results, problematic methodological and interpretive aspects qualify complete acceptance of Satterfield's account. Methodological weaknesses are reflected in the inconclusive evidence thus far available to support: the temporal stability of arousal measures; a conclusive drug-induced change in the actual behavior of hyperkinetic children; and the situational generality of the findings. In addition to the fact that the findings have not as yet been fully replicated, physiological measures were obtained on only two occasions in all studies. In the most comprehensive study (Satterfield, et al., 1972), the single baseline session occurred three weeks prior to the second, post-treatment session. The fact that each individual measure is highly variable over time and across experimental settings is readily apparent in the literature, particularly in studies using children as subjects and/or employing typically redundant laboratory procedures (Duffy, 1962). The problem is underscored in Satterfield's 1972 study in which the placebo group of hyperkinetics demonstrated greater overall change from baseline than did either the originally high or low arousal hyperkinetics treated with methylphenidate. Of course, the data is further complicated

by the selective use of a stimulant drug which presumably affects the habituation process by altering arousal.

The fact that so many extraneous variables can produce either spuriously high or low correlations between physiological measures of arousal indicates both that multiple indices should be recorded simultaneously and that various measures of each index should be scrutinized (Duffy, 1962). Although Satterfield largely complied with both recommendations in the 1972 study, one shortcoming is especially blatant. The number of EEG movement artifacts and scores on teacher questionnaires were the sole sources of information about somatomotor activity. The significance of omitting more direct data is related in the fact that the degree of activation displayed by individual arousal indices has been associated with both general and specific somatic and nonsomatic factors. GSR, for example, has been correlated with general motor activity only when "motivational properties" such as instruction-induced CNS arousal changes have been present (e.g., Roberts and Young, 1971). On the other hand, variable activity level changes in individual peripheral segments (e.g., clinching the fist) have accompanied GSR shifts irrespective of their motivational aspects (Culp and Edelberg, 1966). Consequently, to ascertain the meaning of GSR -- or any other form of arousal data on hyperkinesis -- requires rather specific and direct information about somatomotor activity as well as about the motivational characteristics of the setting.

Finally, data reflecting generalization of effects to other settings were confined to questionnaire ratings. Since perceived behavioral improvements may diverge from objectively collected data (Wahler, unpublished data), and since the questionnaire data has been incompletely reported in all of Satterfield's published work, widespread effects cannot be presumed or even adequately assessed.

These methodological drawbacks necessarily undermine confidence in the proposed arousal mechanism. Two additional factors further hinder a clear-cut interpretation of the data. First, since the overcompensatory activity hypothesized in low arousal hyperkinetics presumably represents a way of augmenting sensory input and therefore arousal, one set of validation problems focuses on the credibility of a sensory input theory of arousal. Second, since stimulant drugs are known to affect both arousal and motor systems, a conclusive assessment of the role of arousal in the disorder requires the use of an alternative experimental manipulation, independent of the drug issue.

Credibility of a sensory input theory of arousal. The behavioral data on this issue are plausible enough although typically confounded by many significant uncontrolled variables. Generally, unaccustomed decreases in

sensory stimulation result in efforts to enhance input, and either extreme sensory deprivation or overload result in a variety of abnormal characteristics (e.g., Bexton, Heron, and Scott, 1954; Calhoun, 1949), some of which are consonant with hyperkinetic symptoms (e.g., hyperactivity, hyperaggression, diminished ability to concentrate).

In the physiological literature, on the other hand, a long term controversy exists over the effect of sensory stimulation on neuroanatomically defined pathways involved in arousal. The principal structures implicated are the ascending reticular activating system (RAS) and the hypothalamus, which are sometimes referred to in this context as the reticulo-hypothathalamic system (Gellhorn, 1954; Brain and Walton, 1969). To summarize the current status of the issue, a necessary relationship between the degree of sensory input to the lemniscal or primary somatosensory tracts and the amount of electrical activity in the bulbar RAS remains unverified. This basic lack of continuity in the data on reticulo-cortical pathways for arousal suggests that a sensory input theory is entirely too simplistic, and the conclusion is sustained by increasing experimental support both for the primary role of intrinsic rhythms in RAS operation (e.g., Batini, et al., 1959) as well as for the relative import of the hypothalamus in regulating behavioral arousal. For example, even though the two areas are highly inter-

connected anatomically, lesioning the posterior hypothalamus consistently produces a behaviorally quiescent subject, an outcome which does not always follow an absence of peripheral or reticular activation. Further, either peripheral or reticular stimulation yield a desynchronized EEG irrespective of behavioral signs of arousal (Feldman and Waller, 1962).

Despite the dubious standing of a purely sensory input theory for normal arousal, it is only necessary here to establish that some aspect of sensory input, however interwoven with intrinsic RAS rhythms or with alternative CNS arousal pathways, can have a demonstrable and meaningful effect on arousal levels and subsequently on behavior. Construed within that framework, Lindsley (Lindsley et al., 1950) selectively transected or ablated either the lemniscal pathways or the mesencephalic RAS in cats and found that sustained CNS arousal between auditory and/or somatosensory stimulus presentations is obliterated only when the RAS is damaged. Apparently, then, direct somatosensory information stimulates rather than maintains arousal, although the possibility may not be excluded that sensory collaterals to the RAS normally possess some indirect maintenance functions. An appropriate criticism of this study is that the lesions sometimes invaded the region of the posterior hypothalamus; however, the only differentiating factor was the length of coma so that the results should apply to both anatomical regions.

The fact that elevating sensory input is an inefficient method of maintaining arousal, especially in the absence of a completely intact RAS or posterior diencephalon, is quite consistent with the low arousal conceptualization of hyperkineses; viz., an organism attempting to sustain the temporary arousal effects accompanying sensory changes would require frequent and brief responses to novel stimuli. Such a conclusion also logically follows from Sharpless and Jasper's investigation (1954) establishing that the thalamus and RAS are mutually responsible for orienting responses and rapid habituation. Under such circumstances, enhanced general activity levels and diminished attention span would be highly adaptive qualities. Even if this interpretation renders the arousal hypothesis more tenable, another serious and interpretative problem remains for Satterfield's studies.

The need for experimental manipulation other than medication. Conclusive support for the arousal hypothesis requires corroborative data from independent manipulations of arousal level and/or somatomotor activity. Unfortunately, both stimulant drugs typically used in the treatment of hyperkinesis have pronounced effects on both arousal and motor systems. Since the two systems are interrelated, it is unlikely that any manipulations significantly

affecting one would negligibly affect the other. Nevertheless, supportive evidence with experimental manipulations other than drugs would be convincing.

The Proposed Study

In further testing the arousal hypothesis of hyperkinesis, the study proposed here is designed to remedy many of the previously described methodological and interpretive problems. The following discussion focuses on the treatment of these problems and on some preliminary predictions.

Resolving Problems from Earlier Investigations

Differences between measures. A more comprehensive and accurate profile of each subject's general arousal level may be provided by including more direct physiological and behavioral indices of motor activity. In addition to the variables utilized in Satterfield's 1972 study (background EEG, evoked cortical responses, GSR and teacher questionnaire), the following measures are employed; EMG, a physiological index of muscular tension; SMR, a 12-14 cps EEG frequency band, indicating the degree of voluntary motor inhibition (e.g. Howe and Sterman, 1972); a standard parent questionnaire; and a global behavioral assessment. Various standardized behavioral measures of sustained attention,

social interaction, and undirected activity are included in the behavioral evaluation (see Wahler et al., 1975) to supplement laboratory measures and questionnaire items whose ability to differentiate hyperkinetics from normal controls has been previously established (e.g., Stewart et al., 1970).

Differences within individual measures. The stability of differences between groups on individual behavioral and physiological measures should exceed intra-subject variance if the arousal hypothesis is valid. A vast amount of literature attests to the short-term fluctuations in each measure. For example, most arousal measures initially exhibit an excitatory characteristic that diminishes with repetition. Nevertheless, habituation should uniformly influence all groups; any real inter-group discrepancies should therefore be maintained. A greater number of baseline sessions should adequately assess this aspect.

Generalization. The relevance of long term temporal generality is assured by the extended nature of this study and the pronounced maturational changes affecting children in the age range of the subject population. Thus, intra-subject comparisons before, during, and at the end of treatment are supplemented with baseline measures obtained

during comparable time periods from untreated hyperkinetic and normal control subjects. Efforts to establish cross-situational generality are limited to determining the relationship between physiological activity levels in the laboratory and behavioral levels in the classroom setting.

Therapy. To circumvent uncertainty about the mechanism through which drugs operate on the nervous system, therapeutic aspects of the arousal hypothesis may be alternatively assessed by modifying through biofeedback one of the arousal and/or motor indices measured during baseline and by observing any correlated physiological and behavioral changes.

Predictions about Initial Baselines Differences

If the arousal hypothesis is valid, then certain intercorrelations between measures are expected to emerge during initial baseline. These should differentiate not only subclasses of hyperkinetic subjects as a function of arousal, but should also systematically differentiate hyperkinetic arousal groups from normal control subjects.

Normal control vs. hyperkinetic subjects. Table 1 includes predictions about baseline differences between normal children and hyperkinetic subjects as a function of CNS arousal level. In conjunction with Satterfield's studies (Satterfield and Dawson, 1971; Satterfield et al., 1972), high and

TABLE 1

EXPECTED BASELINE DIFFERENCES IN AROUSAL AND SOMATIC INDEXES AS A FUNCTION OF SUBJECTS' AROUSAL LEVEL

EXPECTED BASELINE DIFFERENCES	NORMAL CONTROL SUBJECTS	LOW AROUSAL HYPERKINETIC SUBJECTS	HIGH AROUSAL HYPERKINETIC SUBJECTS
AROUSAL INDICES	1. intermediate evoked cortical response amplitudes	1. highest evoked cortical response amplitudes	1. lowest evoked cortical response amplitudes
	2. intermediate EEG slowing	2. most EEG slowing	2. least EEG slowing
	3. intermediate GSR conductance	3. lowest GSR conductance	3. highest GSR conductance
	4. highest *SMR incidence	4. lowest SMR incidence	4. intermediate SMR incidence
	5. lowest tonic EMG activity	5. highest tonic EMG activity	5. intermediate EMG activity
SOMATIC INDICES	6. lowest incidence of hyperkinetic symptoms on behavioral and questionnaire profiles	6. highest incidence of hyperkinetic symptoms on behavioral and questionnaire profiles	6. intermediate incidence of hyperkinetic symptoms on behavioral and qyestionnaire profiles

*Due to its relationship with both somatosensory activity in the thalamus and extreme slow or fast wave activity in the background EEG, SMR may be construed here as an arousal index; however, its strongest (i.e., most consistently demonstrated) behavioral correlate is active motor suppression.

low CNS arousal groups of hyperkinetic subjects should differ from normal control subjects who are expected to display intermediate levels of arousal. Considering the severity of motor impersistence as a function of arousal level, on the other hand, low arousal hyperkinetic subjects should occupy the extreme position relative to normals, whereas high arousal hyperkinetic subjects should exhibit an intermediate degree of motor excitability (e.g., Stevens et al., 1968).

Low arousal vs. high arousal hyperkinetic subjects. Table 2 (top panel only) includes predictions about low and high arousal hyperkinetic subjects during the NO DRUG baseline condition. Like Satterfield's low arousal subjects, low arousal subjects in this study are expected to display the following: enhanced cortical evoked response amplitudes; predominantly high amplitude, low frequency background EEGs; and depressed GSR activity. In contrast, high arousal hyperkinetics should exhibit reverse amplitude and frequency characteristics (i.e., low amplitude cortical evoked response amplitudes, primarily desynchronized, high frequency background EEGs, and elevated GSR activity).

Due to the paucity of somesthetic input or processing hypothesized for low arousal subjects, one related over-compensatory activity may be above normal frequencies of self-stimulation. Additional activities directed

TABLE 2

EXPECTED BASELINE, METHOD OF TREATMENT, AND POST-TREATMENT DIFFERENCES IN AROUSAL AND SOMATIC INDICES AS A FUNCTION OF HYPERKINETIC SUBJECTS' AROUSAL LEVEL

	LOW AROUSAL HYPERKINETIC SUBJECTS	HIGH AROUSAL HYPERKINETIC SUBJECTS
EXPECTED BASELINE DIFFERENCES	1. higher evoked cortical potential amplitudes. 2. greater EEG slowing 3. lower GSR conductance 4. lower SMR incidence 5. lower tonic EMG activity 6. more severe symptoms of hyperkinesis on behavioral and questionnaire profiles	1. lower evoked cortical potential amplitudes. 2. less EEG slowing 3. higher GSR conductance 4. higher SMR incidence 5. higher tonic EMG activity 6. less severe symptoms of hyperkinesis on behavioral and questionnaire profiles
PROPOSED TREATMENT	Conditioned Increases in SMR	** Conditioned Increases in SMR
EXPECTED POST-TREATMENT DIFFERENCES	Normalization of CNS arousal measures and remission of hyperkinetic symptoms on behavioral and questionnaire profiles in treated subjects relative to their pretreatment baseline and to undertreated hyperkinetic and normal controls	**Normalization of CNS arousal measures and remission of hyperkinetic symptoms on behavioral and questionnaire profiles in treated subjects relative to their pretreatment baselines and to untreated hyperkinetic and normal controls

**These phases of the experiment were not conducted since a high arousal group could not be differentiated from the sample population.

towards counter-balancing CNS arousal deficits may include the classic excitatory symptoms of hyperkinesis, viz: 1) short attention span, which may be revealed behaviorally by a low incidence of sustained attention; and 2) enhanced skeletal activity, which may be displayed in elevated behavioral ratings of general movement as well as physiological ratings of tonic EMG activity. Although high CNS arousal is also commensurate with some increase in motor activity, the overcompensatory characteristic is not relevant; therefore, heightened baseline arousal symptoms should be accompanied by signs of somatic excitation that are similar though less excessive than those of low arousal hyperkinetics (i.e., moderately high EMG, self-stimulation, and general movement records and low frequency sustained attention).

Expected SMR levels are less easily assessed due to complex interactions with both somatomotor and arousal indices. According to correlative studies of the background EEG (Chase and Harper, 1971; Sterman and Friar, 1972), SMR is equally scarce during periods of extremely high frequency desynchronized activity (characteristic of high arousal) and of EEG slowing (characteristic of low arousal). Such a finding suggests no special differences other than that SMR activity should be uniformly less prominent in the two hyperkinetic arousal groups than in normal controls.

Additional data, on the other hand, is less equivocal. For example, SMR activity reportedly varies directly as a function of both active or conditioned motor inhibition and subcortical activity in the ventral posterior lateral nucleus (VPL) of the thalamus (Howe and Sterman, 1972). Since VPL is a primary relay nucleus for somesthetic sensations from all parts of the body, this outcome indicates that SMR is sensitive to the degree of somesthetic input and coincides with the sensory input notion of arousal levels in the two groups. In conjunction with the moderate degree of somatomotor impersistence and somesthetic self-stimulation already anticipated as correlates of high arousal hyperkinesis, these two findings suggest that it may also be reasonable to expect intermediate levels of SMR in this group relative to normal controls and low arousal hyperkinetic subjects. In contrast, lowest SMR levels should be shown by low arousal subjects due to their excessive motor activity levels and to the limited effectiveness of augmenting self-stimulation in heightening arousal.

Rationale for SMR Biofeedback as a Treatment Procedure

Table 2 (bottom panel only) also includes the proposed alternative treatments to drugs, SMR biofeedback training, and the expected result at the end of training.

SMR has been selected as the target index for modification through biofeedback because of its therapeutic practicability and its potential value in clarifying arousal functions in the disorder. In addition to the fact that the rhythm is conditionable (e.g., Sterman and Friar, 1972), the utility of SMR modification in the treatment of hyperkinesis is supplemented by its demonstrable therapeutic value in at least one disorder, epilepsy (Sterman and Friar, 1972; Sterman, McDonald and Stone, 1975; Finley, 1975; Seibert and Lubar, 1975; Lubar and Bahler, 1976), and by the rhythm's functional relationship with two significant hyperkinetic characteristics. Trained increases in SMR have been accompanied by relevant changes in several somatomotor measures, including decreased frequency of eye movements, decreased amplitudes of tonic EMG recordings from the neck (Chase and Harper, 1971), and increased conditioned motor suppression (e.g., Roth et al., 1967; Sterman and Wywricka, 1967; Chase and Harper, 1971; Howe and Sterman, 1972). Augmenting this index has also been correlated with changes in general arousal measures, such as decreased respiration and heart rate (Chase and Harper, 1971) and normalization of abnormal EEG spectrum profiles (Sterman and Friar, 1972). For these reasons, SMR biofeedback training should result in reduced motor activity in both groups, increased physiological arousal in the low arousal group, and decreased physiological arousal in the high arousal hyperkinetic subjects.

If contingent increases in SMR can be demonstrated, there are three conditions under which the arousal hypothesis may be evaluated. The exclusive display of training effects either in arousal level or in motor activity are results that would undermine the relevance of arousal as a central factor in the disorder. On the other hand, if contingent modifications in both variables occur, Satterfield's hypothesis would be strengthened. In any event, a favorable outcome would provide a set of therapeutic procedures independent of the drug issue and perhaps of independent value when the use of drugs is contraindicated.

CHAPTER II

METHODOLOGY

Subject Selection

Hyperkinetic Subjects

In order to participate in all phases of the experiment (see Table 3), hyperkinetic subjects (total sample N=12) 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: 1) male; 2) within the age range of 6-12 years; 3) diagnosed as hyperkinetic by a pediatrician who considered the case severe enough to warrant medication; 4) regularly taking methylphenidate (Ritalin); 5) diagnosed as hyperkinetic according to the Stewart teacher questionnaire, requiring definite indication of at least six symptoms, including overactivity and short attention span; 6) without specific sensory deficits 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 (training N=4) was dependent upon demonstrable improvement in the classroom in selected behavioral categories, and in laboratory performance (EEG only) following medication. Improvement is not intended to denote complete remission

TABLE 3

NUMBER OF LABORATORY AND CLASSROOM OBSERVATION SESSIONS CONDUCTED DURING SIX EXPERIMENTAL PHASES

		Experimental Phases					
		BASELINE SEQUENCE			BIOFEEDBACK SEQUENCE		
	EXP. SUBJECTS	I NO DRUG	II DRUG ONLY	III DRUG & 12-14 Hz+ 4-7 Hz FEEDBACK	IV DRUG & 12-14 Hz- 4-7 Hz+ FEEDBACK	V DRUG & 12-14+ 4-7 Hz- FEECBACK	VI NO DRUGS & 12-14 Hz+ 4-7 Hz- FEEDBACK
LABORATORY SESSIONS	1	6	6	78	38	30	24
	2	6	6	60	30	24	24
	3	6	6	74	-	-	-
	4	6	6	68	26	24	26
BEHAVIORAL SESSIONS	1	15	15	45	15	15	15
	2	15	15	30	15	15	15
	3	15	15	30	-	-	-
	4	15	15	30	15	15	15
LABORATORY SESSIONS	HYPER-KINETIC (5-12) & NORMAL (1-12) CONTROL SUBJECTS	6	6	-	-	-	6
BEHAVIORAL SESSIONS		15	15	-	-	-	15

of symptoms; rather, a sufficient 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 changes in EEG measures included an overall reduction in undirected activity accompanied by an increase in school oriented activity (see Chapter III). Significant changes in EEG measures included a reduction of slow wave activity between 4-7 Hz and an increase in 12-14 Hz production. The remaining eight subjects who did not participate in the training sequence served as hyperkinetic control subjects.

Stewart Parent Questionnaire were administered prior to baseline sessions, and changes in all other behavioral and physiological measures were assessed when each subject was on and off drug. However, these findings were not relevant to the selection procedure.

Normal Control Subjects

Twelve normal control subjects were selected from a group of children nominated by the teachers of each hyperkinetic subject. At least two children from each class were nominated, and the final selection was based on the relevant matching characteristics (age, sex, IQ). In addition, the absence of criteria 3, 4 and 5 for hyperkinetic subjects was required for each normal control.

Experimental Design

Table 3 outlines the sequence of experimental conditions for hyperkinetic subjects and the number of sessions per phase during which dependent changes in laboratory and classroom performance were monitored. Initial baseline sessions (hyperkinetic N=12) under NO DRUG (I) and DRUG ONLY (II) conditions were conducted in the absence of EEG biofeedback. Reference to these pre-training data permit 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 (training N=4) rewarded the production of 12-14 Hz EEG activity and the inhibition of 4-7 Hz activity. A contingent increase in SMR production (12-14 Hz +; 4-7 Hz-) was followed by a contingency reversal (12-14 Hz; 4-7 Hz+) in Phase IV. If pre-training 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-) was reinstated during Phase V. In the final training phase (VI), the same EEG contingency was maintained after medication was withdrawn.

At the end of the training sequence, baseline measures under NO DRUG (VI) were reassessed in all hyperkinetic

subjects (N=12). Baseline measures were also obtained from all 12 normal children before and at the end of the training period. The number and timing of these sessions in hyperkinetic and normal control subjects relative to those for training subjects is indicated in the lower portion of Table 3.

Classroom Procedures and Apparatus

Thirteen behavioral categories were selected as indices of overactivity and short attention span, the two fundamental characteristics of the syndrome (Stewart, Pitts, Craig and Dierak, 1966). Twelve of these categories and six environmental events were extracted from a larger sample described in detail by Wahler, who developed the category system (Wahler, House and Stanbaugh, 1975). As described in Table A-1, Appendix A, the categories have been adapted intact with two exceptions. Sustained attention is limited here to circumstances specifically related to school work; also, the out-of-seat category represents a novel category and is not scored separately in Wahler's system.

Observers were equipped with battery-operated tape recorders, pre-recorded tapes indicating when to observe and when to record, and a double earphone jack to permit reliability checks. Ten-second observation intervals alternated with five-second recording intervals during

each 30-minute session. Before entering the classroom, each observer memorized the category system and practiced the observation sequence during 10, 1-hour training sessions. These were conducted at The University of Tennessee with three pre-recorded videotapes of children's behavior. Each subject was observed in the classroom for one week prior to the actual data collection in order to establish a reasonable level of reliability ($\geq$ .70) between independent observers and to adapt the class to their presence. Trial observations occurred during a three-week adaptation period preceding Phase I (NO DRUG) when the hyperkinetic subjects were withdrawn from medication.

Experimental phases followed each other immediately with the exception of a two-month interval between phases II and III. Table 3 presents the total number of observation sessions conducted for each subject during each experimental phase. Daily sessions on 15 consecutive school days occurred at the end of each phase. The same observation sequence was repeated after every two-month period in phase III (training subjects only). Hyperkinetic subjects were observed during one of two classes immediately following medication (Phases II-V). Since medication was scheduled before morning and noon meals, approximately 45 minutes elapsed prior to the observation. Sessions were conducted at the same time period under the NO DRUG condition on hyperkinetic and normal control subjects.

Laboratory Procedures and Apparatus

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 characteristic of baseline.

Training sessions were subdivided into two baseline periods and two feedback periods. A 5-minute baseline taken from one hemisphere was followed by 15 minutes of feedback. A second 15-minute feedback period was provided for the contralateral hemisphere after which a final 5-minute baseline was obtained from the original side. Feedback presentations were limited to two of the seven physiological indices monitored (4-7 Hz events and SMR bursts). Separate modes of feedback were uniformly employed for the production (correct light, tone, money) and inhibition (incorrect light) of the target responses. The nature of the response to be produced or exhibited at any given time was dictated by the sequence of experimental conditions (see Table 3).

The following physiological responses were monitored in the laboratory setting:

1) EEG indices:

    a) 4-7 Hz events - each 4-7 Hz signal above 12.5 uV

b) SMR events - each 12-14 Hz signal above 5 uV in the absence of 4-7 events

c) SMR bursts - six cycles of SMR within .5 seconds

d) background EEG

e) auditory evoked cortical responses

2) EMG criterions - each set of 50 EMG signals of predetermined amplitude between 30-300 Hz.

3) GSR - basal skin resistance in response to 18uA constant current read at 30-second intervals and converted to conductance units (umhos). Four of these indices (4-7 Hz events, SMR events, SMR bursts, EMG criterions) were monitored during each 40-minute laboratory session. The remaining measures were obtained during baseline sessions, and tape recordings of background EEG activity were periodically collected during training sessions.

The basic procedures and apparatus involved in EEG, EMG, and GSR signal detection, analysis, and feedback are discussed with reference to the block diagram in Figure A-1, Appendix A. Subjects were not grounded because laboratory sessions were conducted in a sound attentuated, electrically shielded chamber where two matched P5-11E pre-amplifiers and the Grass Instrument power supply were grounded to a single point. Electrode impedance was established with a Grass Instrument Impedence Meter at the onset of laboratory sessions. Fifteen K-ohms constituted the maximum acceptable impedence.

EEG Indices and Electronic Detection Circuitry

All EEG indices were bipolar recordings obtained from silver electrodes (Grass Instrument type E5-SH) situated bilaterally at 10% and 30% of the distance between vertex and the preauricular point. These are the same coordinates used by Sterman and Friar (1972) to record background EEG, SMR, and 4-7 Hz activity in epileptics. This electrode montage differs somewhat from that used by Satterfield (Satterfield et al., 1972) to measure evoked cortical potentials. In the latter case, monopolar recordings were obtained from bilateral recording electrodes situated at a constant vertical distance (.5 cm) from vertex and were referred to the respective earlobe.

The signal averaging apparatus was by design mutually exclusive from the other circuitry involved in further processing the raw EEG signal. Since either SMR and theta activity, or auditory evoked responses were measurable at one time, evoked responses were obtained prior to regularly scheduled laboratory sessions during which background EEG, SMR and theta were measured concurrently.

A storage oscilloscope, an ORTEC signal averager (#4623) and matching memory control (ORTEC #4620) were interfaced with the system at the output of the 2 pre-amplifiers for analyses of evoked responses. Additionally, a Grass stimulator (S-88) fed into the signal averager and an eight ohm speaker. The stimulator was programmed to deliver 70, 90db auditory clicks of 1-msec. duration at a

constant 5-second interstimulus interval. Following stimulus presentations, the averager was programmed to initiate a 250 msec. sweep of the background EEG. These were stored in the memory, averaged over each consecutive sweep, and displayed on the oscilloscope.

Periodic samples of background EEG activity (all baseline sessions and two per month during training) were recorded on an FM magnetic tape recorder (TEAC corporation), which was also interfaced with the system at the output of the two preamplifiers. Taped data were fed into a PDP-11 computer that has been pre-programmed to perform continuous fast Fourier analyses and to print out compressed isometric plots for sequential eight-second epochs of the EEG. Frequencies between 0 and 32 Hz were sampled at one Hz intervals during each epoch.

On line processing of SMR and theta activity was initiated by the double discriminator circuit, through which the amplified EEG output from scalp electrodes was split into two signals. One drove a 12-14 Hz, 8-pole tuned band pass filter (White Instrument Company) with greater than 80 db attenuation during the first octave. Its output was displayed on EEG paper channel 2. Figure A-2, Appendix A represents a power spectrum analysis of the filter's response. A PDP-11 computer analysis over a 4-minute epoch illustrates that the filter responds maximally to activity between 12 and 14 cycles with a roll-off factor of 50 thousand times at 10 and 17 Hz.

The second signal drove a 4-7 Hz filter (White Instrument Company) with 24 db per octave attenuation. Its output was displayed on EEG paper channel 4.

SMR detection was dependent upon meeting a dual criterion (12-14 Hz; greater than 5 uV) which was initially accomplished by the 12-14 Hz filter described above and by an amplitude discriminator. Criterion waves from these sources were then digitally analyzed by passing the signal through the two additional modules (ORTEC #4672 Instanteous Time-Frequency by Meter and Ortec 730-E single Channel Analyzer). The instantaneous frequency-to-amplitude converter transformed each frequency of the filter output into a voltage representing each cycle of 12-14 Hz activity exceeding the predetermined amplitude level. Subsequent evaluation by the Single Channel Analyzer, which is a 12-14 Hz adjustable window discriminator, confirmed the stipulated frequency criteria, after which digital logic pulses were printed on EEG channel 6. Simply stated, the analytical process required a signal duration ranging from 1/12th-1/14th second and possessing the minimum amplitude requirement for any wave to qualify as a discrete instance of SMR.

At the same time, however, the 4-7 Hz filter drove an inhibit circuit which also consists of an amplitude discriminator that blocked digital processing of SMR whenever a preset amplitude of slow wave activity was exceeded.

The activity of the inhibit circuit was pictorally displayed on EEG paper channel 7. In effect, then, only EEG activity meeting all three conditions triggered a digital logic pulse from the single Channel Analyzer indicating the incidence of SMR. Each instance of SMR as well as each SMR "burst" (the number of instances occurring within a preset time interval) was summed by an Ortec #434 Digital Ratemeter and printed out by a teletype at one-minute intervals. The burst criterion in this study was 6 instances of 12-14 Hz in .5 seconds and was displayed on EEG paper channel 3.

During training sessions, verbal and monetary feedback was presented to the subject regarding the number of SMR bursts attained relative to daily baseline output. The following feedback modes were presented regarding the number of SMR burst attained and the status of the theta inhibit circuit during feedback periods irrespective of baseline performance: A horizontal colored light display in which individual lights were consecutively illuminated following the achievement of individual burst criteria; a slide projected on a screen in front of the subject for each set of 10 SMR criterions achieved; a tone accompanying each SMR burst detected; a digital display representing a cumulative count of SMR bursts achiever per minute; a large green light illuminated to represent inactivation of the 4-7 Hz inhibit circuit.

EMG

EMG recordings were obtained from two Beckman silver-silver chloride electrodes centered 3/4 inch from the midline of the chin on the inferior and superior surfaces. EMG signals between 30-300 Hz (via the second Grass amplifier) passed through a Schmitt trigger with an adjustable threshold. Signals exceeding preset threshold levels triggered logic pulses that were fed into a digital integrator. The integrator automatically reset when pre-determined criterion levels were reached. Frequency of responses measures for EMG criterions were printed out on a teletype at one-minute intervals and the raw signal appeared on EEG channel 5.

GSR

Monopolar GSR recordings were obtained from two Beckman silver-silver chloride electrodes with the recording electrode attached to the inferior surface of the middle finger and referred to the forearm. Changes in resistance to a constant current (18 uA) passed through the electrodes were monitored by a DC bridge (Wheatstone configuration) and amplified by a E & M Instrument (TMC model 138) to yield graded voltage outputs ranging from ± 4 volts around a basal or stabilized resistance registered in K-ohms on the external surface of the DC bridge. The amplified signal was then conditioned by a Harvard model 355 amplifier for linear output to a Harvard ink writer (model 480). Measures of basal resistance were obtained manually by

re-establishing basal resistance levels at 30-second intervals during baseline laboratory sessions.

CHAPTER III

PRE-TRAINING DATA: NO DRUG VS. DRUG ONLY

Preliminary Background Data

Matching characteristics for hyperkinetic and normal subjects are provided in Table 4. The data in Table 4 indicate that the two groups are relatively well-matched for age and IQ and that the subjects in each group are evenly distributed over the age and IQ ranges sampled. Two children from each group fall into each of the one-year intervals from 6-11. Similarly, 3 subjects from each group rank in the lowest (80-89) and highest (100-110) thirds of the IQ spectrum while 6 hyperkinetics and 6 normal controls scored in the intermediate range (90-99). The correspondence between groups on both factors is statistically significant (<u>age</u>: $\rho = .64$, $t = 3.16$, $df = 10$, $p. < .01$; IQ: $\rho = .57$ $t = 2.18$, $df = 10$, $p < .05$).

Additional background data on the age of sympton onset and current medication dosages is also provided in Table 4 for hyperkinetic subjects. Current dosage levels vary considerably between hyperkinetic subjects (range = 5-100mg/school day; $\bar{X} = 33.3$ mg.), even though optimal dosage levels for classwork had been established and stabilized at least six months prior to study in 10 subjects. With the exception of subjects 1 and 3, who were placed on Ritalin approximately 1.5 months before the baseline

TABLE 4

PRELIMINARY BACKGROUND DATA ON HYPERKINETIC SUBJECTS AND NORMAL CONTROL SUBJECTS

	HYPERKINETICS												
ITEM	1*	2*	3*	4*	5	6	7	8	9	10	11	12	$\bar{X}$
Age	11/8	10/5	10/10	10/5	7/10	8/11	9/0	8/3	6/9	11/0	7/0	6/10	8.8
IQ (standard score)	105	92	95	98	80	93	91	100	85	105	81	96	93.8
Ritalin (mg/day)	10	20	10	35	5	25	20	30	10	10	100	10	13.3
Age of Parents 1st concern (years)	2	7	5	4	4	3.5	6	1	6	7	3	3	4.2
Age of other's 1st concern (years)	6	7	5	4	4	6	6	1	7	8	4	7	5.23
	NORMAL CONTROLS												
Age (yr/mo)	10/0	10/1	7/4	8/11	9/1	6/7	10/2	8/7	9/5	11/5	7/3	6/2	8.83
IQ (standard score)	102	93	108	93	90	85	97	104	86	96	94	89	94.75

*Low arousal subjects

sequence began, all hyperkinetic subjects had been on medication for a minimum of one year and a maximum of three. Periodic adjustments in medication regimens were not uncommon especially over long time periods; however, the consulting pediatricians agreed to maintain all current dosage levels after referring the subjects for this study. Exceptions were made at the experimenter's request.

Excluding subjects 1 and 3 from the sample, there is a slight, but statistically insignificant, tendency for age and current Ritalin dosages to correlate positively (ρ = .45, t = 1.42, df = 8, $p \gtrsim .1$; 6-8 year olds: $\bar{X}$ dosage = 30 mg; 9-11 year olds; X dosage = 38.75 mg). This outcome is commensurate with a drug sensitization account since most subjects have been on continuous medication since at least the second grade. Also, the two subjects (S's 5 and 11) under medication for the longest duration also take the highest dosages prescribed (90 and 100 mg., respectively). IQ and medication levels are also correlated, with subjects in the highest, lowest, and intermediate portions of the IQ spectrum taking progressively higher dosages (ρ = .47, t = 1.51, df = 8, $p \gtrsim .1$). This statistically insignificant outcome is consistent with the majority of the normative data.

The age at which parents report exhibiting their first concern over a child's behavior ($\bar{X}$ age = 4.2 years) is equally divided between pre- and post-school years and is

uncorrelated with current Ritalin dosages ($\prec$5 yrs: $\bar{X}$ dosage = 34 mg; $\succ$ 5 yrs; $\bar{X}$ dosage = 33 mg). Adults other than the parents are reported to have first exhibited concern somewhat later ($\bar{X}$ age = 5.23) than the parents. The "other" adult is most often a teacher, and the occasion for mentioning concern corresponds in 75% of the cases with the child's initial exposure to school, typically either in kindergarten or first grade. The age at which either parents or other adults first noticed problem behaviors inadequately predicts subsequent Ritalin dosages (ρ parent x dosage = .02, df = 8, p $\prec$ .1; ρ "other adult" x dosage = .27, df = 8, p $\prec$ .1), although agreement between the two observations, as reflected in the mean age at which symptom onset is first noticed, significantly predicts the dosage variable (ρ: X age x current dosage = .57, t = 1.96, df = 8, p $\prec$ .05). Nevertheless, since interobserver agreement on early symptom onset tended to facilitate a child's early placement on medication, it is difficult to determine whether this outcome reflects symptom severity, sensitization to medication, or a combination of both.

Phase I (NO DRUG) Differences Between Hyperkinetic And Normal Subjects

Table 5 summarizes the overall differences between hyperkinetic and normal children on Phase I (NO DRUG) questionnaire, behavioral, and laboratory data. With one exception, t-tests for correlated observations between

TABLE 5

PHASE I (NO DRUG) DIFFERENCES BETWEEN HYPERKINETIC SUBJECTS AND NORMAL CONTROL SUBJECTS ON LABORATORY, QUESTIONNAIRE, AND BEHAVIOR INDICES

	HYPERKINETICS (N=12)	NORMAL CONTROLS (N=12)	
STEWART QUESTIONNAIRE DATA			
Mean No. parent-reported problems (n=30)	10.58	2.25	*t=8.34, df=11, p. < .05
Mean No. teacher-reported developmental problems (n=11)	4.29	.43	-
Mean No. parent-reported symptoms (n=47)	17.5	4.17	*t=5.58, df=11, p. < .05
Mean No. teacher-reported symptoms (n=37)	13.03	2.0	*t=7.27, df=11, p. < .05
BEHAVIORAL DATA			
Mean No. undesirable behaviors (n=6)	24.64	14.41	*t=4.15; df=14, p. < .05
Mean No. desirable behaviors (n=3)	18.98	50.83	*t=7.2, df=14, p. < .05
Mean No. social behaviors (n=4)	21.3	15.3	*t=3.9, df=14, p. < .05

TABLE 5 (continued)

	HYPERKINETICS	NORMAL CONTROLS	
LABORATORY DATA			
Mean amplitude evoked response (P2 uV)	17.4	14.5	t=1.13, df=5, p. > .1
Mean GSR Conductance (umho)	12.53	19.5	t=1.34, df=5, p. > .1
Mean No. SMR bursts per minute	8.03	11.23	*t=-3.45, df=5, p. > .1
Mean No. EMG criterion per minute	98.75	118.2	t=1.99, df=5, p. > .1

*Significance levels at .05 or greater

hyperkinetic and matched normal control subjects over days are provided for each set of means, and levels of statistical significance are denoted by asterisks. Teacher-reported developmental problems are omitted from statistical analysis because normal controls are insufficiently represented on those items. It should be noted, however, that hyperkinetic subjects frequently display these problems. Otherwise, the overview of findings presented in Table 5 indicates significant differences between the two groups on the remaining questionnaire items and on three classes of behavior categories. With one exception (SMR), however, hyperkinetic and normal subjects do not differ on physiological measures. A more substantive assessment of the differences on questionnaire, behavioral, and laboratory data follows.

Questionnaire Data

The questionnaire findings in Table 5 indicate that hyperkinetic subjects exhibit a greater number of developmental problems and current disruptive symptoms than do normal children. The differences between groups are further illustrated in Table B-1, Appendix B, where individual questionnaire items are ranked according to hyperkinetic difference scores (percent hyperkinetic subjects minus the percent normal control subjects displaying each item). These difference scores reflect the ability of individual items to discriminate hyperkinetic subjects from normal children.

Developmental items. A comparison of developmental items reveals that hyperkinetic subjects more often display current and previous medical problems than do normal children. Examples include the high relative incidence of poor health during the first year and perinatal complications in hyperkinetic subjects as well as more frequent visits to physicians during the year preceding the study. Hyperkinetic children also appear to develop sensorimotor coordination less rapidly than do normal subjects. Parents of hyperkinetic children frequently complain of poor large muscle coordination, poor eye-hand coordination, and associated deficits in fundamental skills (e.g., slow and unintelligible speech, reading and writing problems). Similar deficiencies are reported by teachers in addition to a history of poor deportment in school (e.g., has to be sent to the principle's office more than once a month).

Symptoms. Items most reliably differentiating hyperkinetic from normal control subjects on symptoms reported by parents and teachers are those reflecting overactivity (e.g., restless, can't sit still, impatient) and short attention span (doesn't finish projects, doesn't listen, etc.). In addition, the dominance of items such as "irritable," "fights a lot," "disobedient," "talks a lot," and "shows off," indicates that the hyperkinetic children sampled here tend to be extraverted and aggressive rather than

intraverted. This view is also supported by the comparatively low ranks assigned to items such as "nervous," "tense," "unusual worries," and "day dreams."

Behavioral Data

The behavioral data tend to corroborate questionnaire findings. Table 5 reveals that hyperkinetic children differ from normal subjects in displaying significantly more undesirable and social behaviors to the relative exclusion of desirable ones. Undesirable behaviors include four apparently purposeless or undirected activities in addition to oppositional and out-of-seat behaviors. Desirable behaviors include two indices of sustained school-oriented behaviors as well as cooperation with others. Four social categories differentiate between sustained and brief interactions with either adjults or peers. The relative frequency with which hyperkinetics and normal controls display these individual behaviors appears in Figure 1. With the exception of self-talk, hyperkinetic subjects uniformly exhibit undesirable activities more often and desirable behaviors less often than do normal control subjects. Although no specific advance predictions were made regarding social interactions, hyperkinetic subjects display higher rates in each category than do normal control children. It is noteworthy, however, that hyperkinetic subjects interact more often with peers

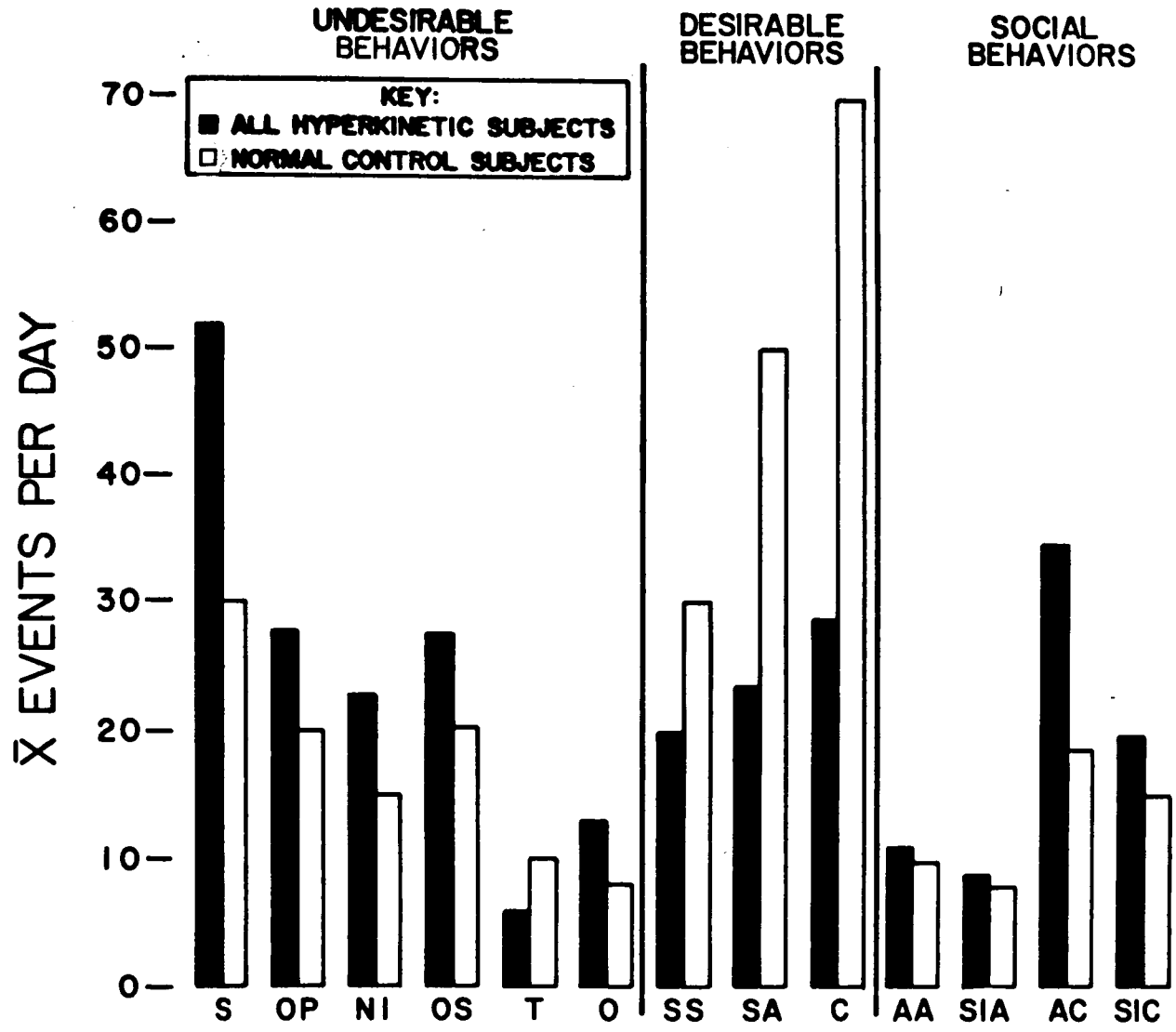


FIGURE 1

Phase 1 (NO DRUG) Performance on 13 Classroom Behaviors: Hyperkinetic vs. Normal Children

than teachers and sustain a smaller proportion of their social approaches than do normal children.

Laboratory Data

In contrast to the questionnaire and behavioral data, overall differences between hyperkinetic and normal children in the laboratory are confined to the relative dearth of SMR bursts anticipated in hyperkinetic subjects. This statistically significant outcome is consistent with the EEG rhythm's noted correlation with behavioral immobility. Other predictions about the two groups were based on low and high CNS arousal differences between hyperkinetic subjects. These were expected to moderate the degree of somatomotor disturbance exhibited by hyperkinetic subjects and to obscure overall physiological differences between hyperkinetic and normal children. In this context, it should be noted that trends in CNS arousal level tend towards the low arousal end of the spectrum in hyperkinetic relative to normal subjects. Although statistically insignificant (see Table 5), this outcome suggests either a disportionately high number of low arousal subjects or the absence of a high arousal group all together.

Phase I (NO DRUG) Differences Between Hyperkinetic and Normal Children As a Function of CNS Arousal Level

Table 6 summarizes Phase I (NO DRUG) differences between hyperkinetic and normal control subjects as a function of CNS arousal level. Otherwise, the format for data analysis and presentation resembles that used in Table 5. Contrary to advance predictions, only those hyperkinetic subjects exhibiting low arousal profiles could be differentiated from the remainder of the hyperkinetic sample. In the absence of high arousal characteristics, the latter group is labelled hyperkinetic control subjects to reflect extensive overlap with normal controls on physiological measures.

Laboratory Differences

Figure 2 presents the mean pre-treatment laboratory performance of individual hyperkinetic and normal subjects on each measure as a function of arousal level. Relative to normal control subjects, the performance of hyperkinetic children ranges from low to normal arousal. Although approximately half the hyperkinetic sample displayed substantially reduced arousal relative to normal control children on any given measure, a comparison of individual subjects across all measures reveals consistent low arousal scores in only four hyperkinetic children (Subjects 1-4). The sporadic performance of hyperkinetic control subjects in the low arousal range appears to account for their intermediate position on the arousal continuum

TABLE 6

PHASE I (NO DRUG) DIFFERENCES IN LABORATORY QUESTIONNAIRE AND BEHAVIORAL INDICES AS A FUNCTION OF SUBJECTS' AROUSAL LEVEL

	LOW AROUSAL ** HYPERKINETICS (n=4)	HYPERKINETIC CONTROLS (n=8)	NORMAL CONTROLS (n=12)
LABORATORY DATA*			
Mean amplitude evoked cortical response (P2 uV)	19.8	15.2	14.5
Mean GSR conductance (umhos)	4.7	17.5	19.5
Mean No. SMR bursts per minute	5.9	9.1	11.23
Mean No. EMG criterions per minute	70.12	113.1	118.2
STEWART QUESTIONNAIRE DATA			
Mean No. parent-reported symptoms (n=47)	24.25	14.125	4.17
Mean No. parent-reported developmental anomalies (n=30)	9.75	11.0	2.25
Mean No. teacher-reported symptoms (n=37)	19.0	10.05	2.05
BEHAVIORAL DATA			
Mean No. undesirable behaviors (n=6)	31.35	21.28	14.41
Mean No. desirable behaviors (n=3)	12.39	27.28	50.83

TABLE 6 (continued)

	LOW AROUSAL ** HYPERKINETICS (n=4)	HYPERKINETIC CONTROLS (n=8)	NORMAL CONTROLS (n=12)
Mean No. social behaviors (n=4)	28.75	14.25	15.3

*Laboratory and behavioral data are assessed over days; consequently, the degrees of freedom are 5 and 14, respectively, for dependent t-tests.

**Low Arousal vs. Hyperkinetic Controls: evoked response amplitudes; t=3.53, df=5, $p < .05$; GSR conductance: t=-3.53, $p < .05$; EMG: T=3.25, df=5, $p < .05$; SMR: t=3.72, df=5, p .05; undesirable behaviors; t = 3.38, df = 14, p .05; desirable behaviors: t=-1.86, df=14, $p < .05$; Social behaviors t=2.62, df=14, $p < .05$; parent-reported developmental problem: t=-6.7, df=10, $p < .05$; parent-reported symptoms: t=2.95, df=10, $p < .05$; teacher-reported symptoms; t=4.4, df=10, $p < .05$;

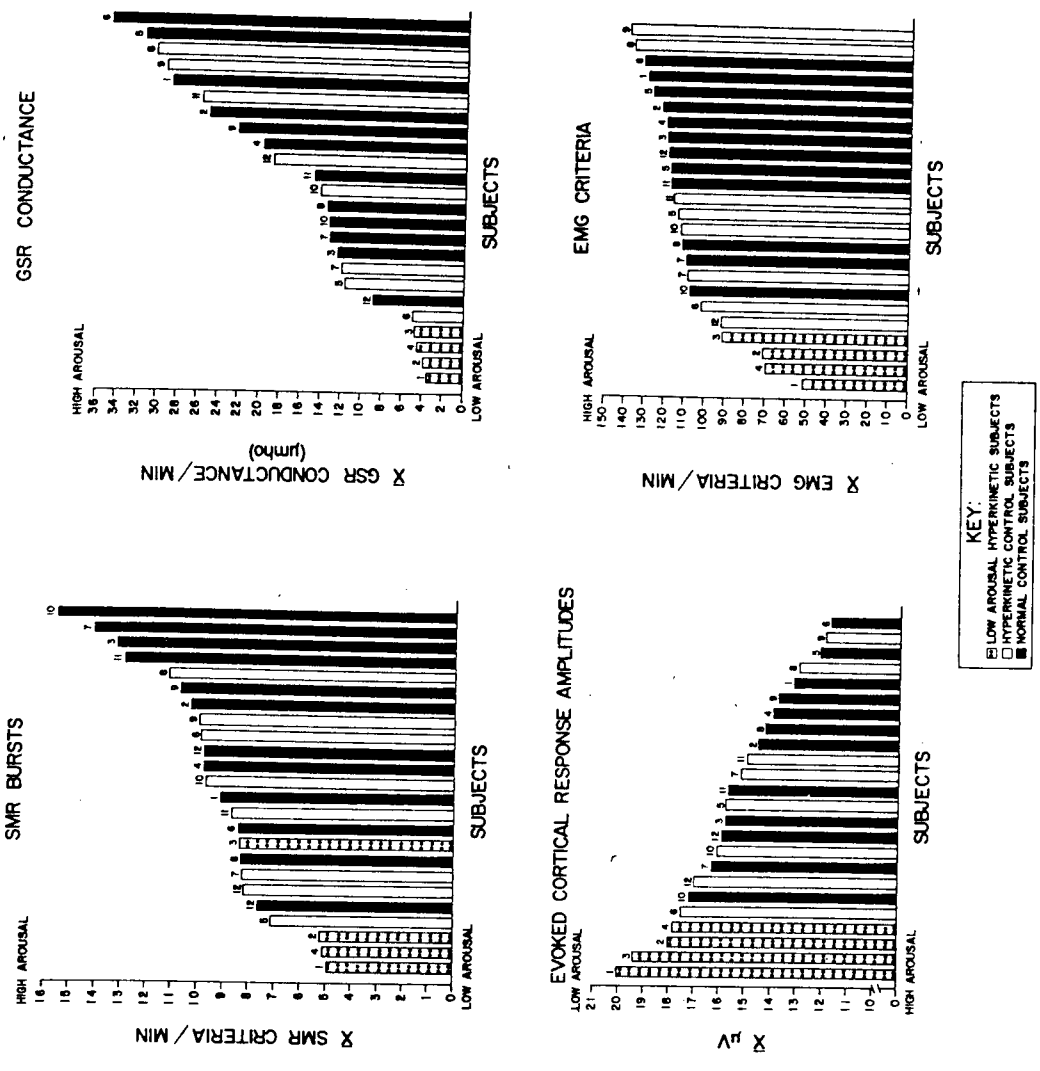


FIGURE 2

Phase 1 (NO DRUG) Laboratory Results: Hyperkinetic vs. Normal Children

relative to low arousal hyperkinetic and normal control subjects. Despite this overlap, statistical analyses presented in Table 6 show significant differences between low arousal and control hyperkinetic subjects on all measures. Consistent with advance predictions, low arousal subjects display significantly higher amplitude evoked cortical responses as well as lower skin conductance, EMG, and SMR levels than do either hyperkinetic or normal control subjects.

In contrast to the laboratory data, it should be recalled that the behavioral and questionnaire assessments effectively differentiated hyperkinetic subjects from normal children irrespective of physiological differences. Nevertheless, systematic differences in the type and degree of somatomotor disturbance are associated with laboratory findings in low arousal and control hyperkinetic subjects.

Behavioral Data

A comparison of the gross behavioral differences reported in Table 6 reveals more severe overall behavioral profiles in low arousal hyperkinetic subjects than in any other group. Relative to hyperkinetic control subjects low arousal subjects differ most radically in excessively producing undesirable and social behaviors and infrequently producing desirable ones.

Figure 3 presents inter-hyperkinetic differences on individual behaviors. Relative to hyperkinetic control subjects, low arousal subjects more often display self-stimulation, object play, out-of-seat, oppositional, and social behaviors. Hyperkinetic control subjects, on the other hand, engage more often in self talk, sustained noninteraction, and cooperative behaviors while sustained school-oriented behaviors are equally uncommon in the two groups. It would appear, then, that low arousal hyperkinetic subjects tend to engage in repetitive, undirected activities (self-stimulation, object play, out-of-seat) that are compatible with other high frequency behaviors which are ostensibly directed towards external environmental events. Illustrations include high frequency social behaviors and overt oppositional reactions to explicit commands and requests from teachers and peers. Hyperkinetic control subjects, on the other hand, appear oblivious to their immediate surrounds. They are more likely to engage in undirected activities such as sustained noninteraction (gazing, day dreaming) and self-talk that are incompatible with social interactions and/or scholastic pursuits. Furthermore, cooperation and oppositional behaviors are typically expressed in a passive fashion. The former usually occurs in response to general rules and instructions outlined at the beginning of class while noncompliance more often follows subsequent changes in routine of which the subjects appear unaware.

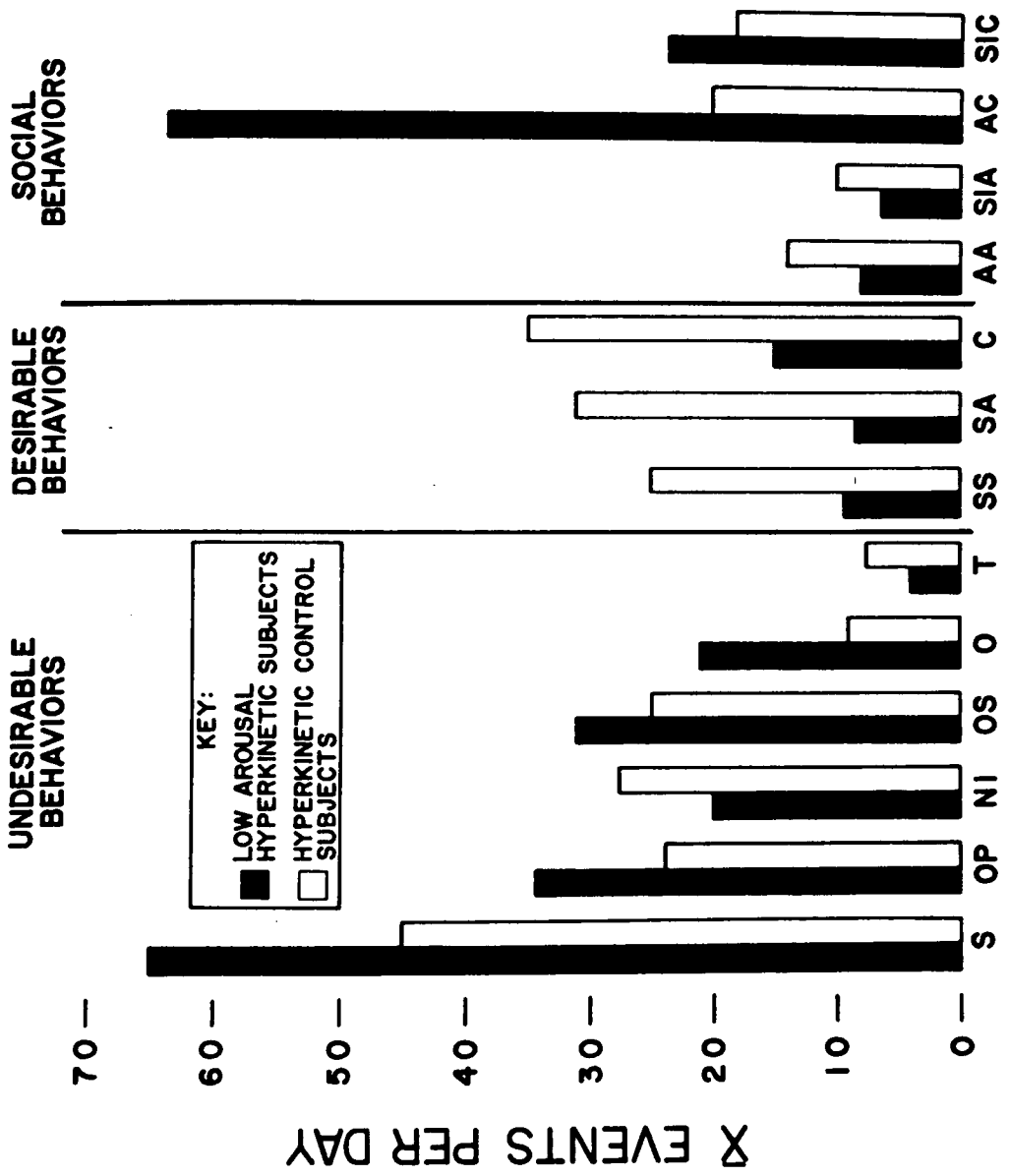


FIGURE 3

Phase I (NO DRUG) Performance on 13 Classroom Behaviors: Low Arousal vs. Control Hyperkinetic Children

Questionnaire Data

The questionnaire data corroborate and extend classroom and laboratory observations. Differences between hyperkinetic groups in the frequency of parent and teacher-reported developmental anomalies and symptoms are statistically significant (see Table 6), and qualitative differences presented in Table B-2, Appendix B clearly implicate motor dysfunction as a central feature in low arousal subjects.

Developmental items. Regarding parent-reported developmental problems, low arousal subjects are more likely than hyperkinetic control subjects to exhibit deficient large muscle coordination (poor at sports, etc.) and disturbed sleep cycles (sleep-walking, night terrors) as well as speech and reading impediments. Aside from an apparent maturational delay affecting sensorimotor coordination, potential precipitating influences during early childhood are confined to miscellaneous accidents requiring ER visits and, to a lesser extent, poor health during the first year. Ironically, hyperkinetic control subjects display fewer maturational deficits, but are more likely to have experienced various medical complications at an early age (troubled pregnancy, prenatal problems, ER visits for head injury).

Nevertheless, the severity of developmental motor deficiencies in low arousal relative to control hyperkinetic subjects is confirmed by teachers who more frequently refer low arousal subjects for speech and reading therapy, tutoring, and LD classes. In addition, the relative gravity of dis-

ruptive behavior profiles in this group is further substantiated by the frequency with which teachers resort to extraordinary disciplinary measures. In contrast to hyperkinetic control subjects, whose misbehavior is generally dealt with in the classroom, 100% of the low arousal subjects must be sent to the principle's office more than once a month, and one-half have been suspended and/or expelled from school on more than two occasions.

Symptoms. Differences in parent and teacher-reported symptoms further dichotimize the two groups on motor-attention span and extraversion-intraversion factors. Relative to hyperkinetic control subjects, low arousal children are more likely to fight a lot, disrupt the class and bother others incessantly, and are more likely to be unpopular with peers. Low arousal subjects also tend more often to be uncoordinated, uncooperative, undisciplined, and intractible. Hyperkinetic control subjects, on the other hand, more often tend to be withdrawn, nervous, tense, and afraid to fight, and to experience episodic depression, usual worries, and nausea. Finally, of the two fundamental characteristics of the syndrome, motor dysfunction is principally associated with low arousal subjects while short attention span is more often designated as the major symptom of hyperkinetic control subjects.

Summary

That excessive overactivity in the absence of tension or anxiety distinguishes low arousal hyperkinetics from hyperkinetic and normal controls is amply documented in the questionnaire and behavioral data. The first feature is consistent with the low arousal subjects' comparatively low baseline levels of SMR while the second is commensurate with their reduced EMG production and GSR conductance. Conversely, hyperkinetic control subjects who exhibit less overactivity and more anxiety, produce higher baseline levels of SMR, EMG, and GSR. Also, since higher amplitude evoked cortical responses are associated with both short attention span and low CNS activation, the fact that low normal subjects exhibit fewer attentional deficits and higher amplitudes evoked responses than do hyperkinetic control children lends credence to an arousal interpretation of this measure. Finally, the fact that low arousal hyperkinetics appear more responsive to extrinsic environmental events is especially indicated by the relative frequency and character of social interactions, cooperation, and opposition with peers and adults. These finding are consistent with the view that excessive overactivity in low arousal subjects reflects an overcompensatory reaction from an otherwise sensory-deprived and sluggish organism.

Phase I (NO DRUG) vs. Phase II (DRUG ONLY) Differences Between Hyperkinetic and Normal Subjects As a Function of Arousal Level

Further evidence supporting the arousal hypothesis of hyperkinesis may be derived from NO DRUG-DRUG ONLY comparisons on each measure. If the hypothesis is valid, low arousal subjects should derive the greatest relative benefit from Ritalin. Increased CNS arousal was expected to accompany a substantial decline in somatomotor disturbance relative to NO DRUG baselines and to hyperkinetic and normal control subjects. Table 7 summarizes the findings in terms of change scores (NO DRUG - DRUG ONLY scores) on each measure. Otherwise, the same format is retained.

Overall Differences Between Phase I and Phase II in Hyperkinetic and Normal Control Subjects

A comparison of Phase I (NO DRUG) and Phase II (DRUG ONLY) performance in normal control subjects reveals no significant differences over time in any measure. (teacher-reported symptoms: $t = .73$, $df = 14$, $p \triangleright .1$; undesirable behaviors: $t = 1.22$, $df = 14$, $p \triangleright .1$; desirable behaviors: $t = -.72$, $p \triangleleft .1$; social behaviors: $t = 1.01$, $df = 14$, $p \triangleright .1$; evoked response amplitude: $t = .96$, $df = 5$, $p \triangleright .1$; GSR: $t = .39$, $df = 5$, $p \triangleright .1$; EMG: $t = 1.0$, $df = 5$, $p \triangleright .1$; SMR: $t = -.2$, $df = 5$, $p \triangleright .1$). Similar analyses performed on the hyperkinetic sample as a whole reveal

TABLE 7

PHASE I (NO DRUG) VS. PHASE II (DRUG ONLY) CHANGES ON LABORATORY, QUESTIONNAIRE, AND BEHAVIORAL INDICES AS A FUNCTION OF SUBJECTS' AROUSAL LEVEL

	LOW AROUSAL * HYPERKINETIC	HYPERKINETIC CONTROL	NORMAL CONTROL
LABORATORY DATA			
Mean amplitude evoked cortical responses (P2 uV)	Subjects -3.0	Subjects +.9	Subjects +.35
Mean GSR conductance (umhos)	+2.9	+.9	+1.25
Mean No. SMR Bursts per minute	+4.42	+1.52	-1.6
QUESTIONNAIRE DATA			
Mean No. teacher-reported symptoms	-5.75	-1.97	+2.25
BEHAVIORAL DATA			
Mean No. undesirable behaviors (n=6)	-7.36	-2.24	+.51
Mean No. desirable behaviors (n=3)	4.68	1.69	-.94
Mean No. social behaviors (n=4)	-14.0	-.25	+.75

*Low Arousal vs. Hyperkinetic Controls: Evoked Responses: $t = -2.12$, $df = 5$, $p < .05$; GSR Conductance, $t = .99$, $df = 5$, $p > .1$. EMG: $t=2.5$, $df=5$, $p < .05$; SMR: $t=5.9$, $df = 5$, $p < .05$; Undesirable behaviors: $t = -4.15$, $df = 14$, $p < .05$; desirable behaviors: $t = 1.98$, $df = 14$, $p < .05$; social behaviors: $t = 5.8$, $df = 14$, $p < .05$; teacher-reported symptoms: $t = 3.28$, $df = 19$, $p < .05$

significant improvement in the number of teacher-reported symptoms ($t = 4.6$, $df = 11$, $p < .05$) and an associated increase in the production of SMR ($t = 3.42$, $df = 5$, $p < .05$). Since no other physiological measure originally differentiated hyperkinetic and normal subjects,the absence of significant changes in them following medication is not surprising (Evoked response: $t = 1.01$, $df = 5$, $p > .1$; GSR: $t = -.99$, $df = 5$, $p > .1$; EMG: $t = -1.5$, $df = 5$, $p > .1$). It is noteworthy, however, that significant changes in behavioral measures failed to accompany those in the questionnaire and SMR data (Undesirable: $t = 1.13$, $df = 11$, $p > .1$; Desirable: $t = 1.22$, $df = 14$, $p > .1$; Social: $t = 1.4$, $df = 14$, $p > .1$).

Phase I vs. Phase II Changes As a Function of Arousal Level

A comparison of NO DRUG-DRUG ONLY change scores in low arousal and control hyperkinetic subjects reveals statistically significant differences in the predicted direction on questionnaire, behavioral, and laboratory indices (see Table 7). Relative to hyperkinetic control subjects, low arousal subjects display the following changes under medication: 1) fewer teacher-reported symptoms; 2) fewer undesirable and social behaviors in conjunction with more frequent desirable classroom behaviors; 3) physiological changes reflecting increased CNS arousal level accompanied by a reduction in SMR production.

Figure 4 presents NO DRUG-DRUG ONLY change scores on individual behaviors as well as the relative reinforcement frequencies for the two groups. In general, decreases in undirected, social, and oppositional behaviors are accompanied by increases in school work and cooperation. The concomittant decline in social reinforcement from peers principally results from a reduction in social approaches towards peers with which it correlates .61 in the NO DRUG condition and .74 in the DRUG ONLY condition. Thus, although substantial social reinforcement accompanies high frequency behaviors that were incompatible with school work in the NO DRUG condition, the improvements sustained following medication cannot be accounted for in terms of changed reinforcement frequencies. If anything, post-treatment reductions in reinforcement discourage the observed modifications in behavioral profiles.

Finally, medication-induced improvements in the teacher-reported and behavioral symptoms of low arousal subjects are also accompanied by the expected changes in physiological measures. With the exception of slight, but statistically significant, increases in GSR conductance, low arousal subjects display significant reductions in evoked cortical response amplitudes and increases in EMG and SMR frequencies relative to hyperkinetic control subjects GSR and evoked cortical response changes reflect an increase in CNS arousal level while the increase in SMR

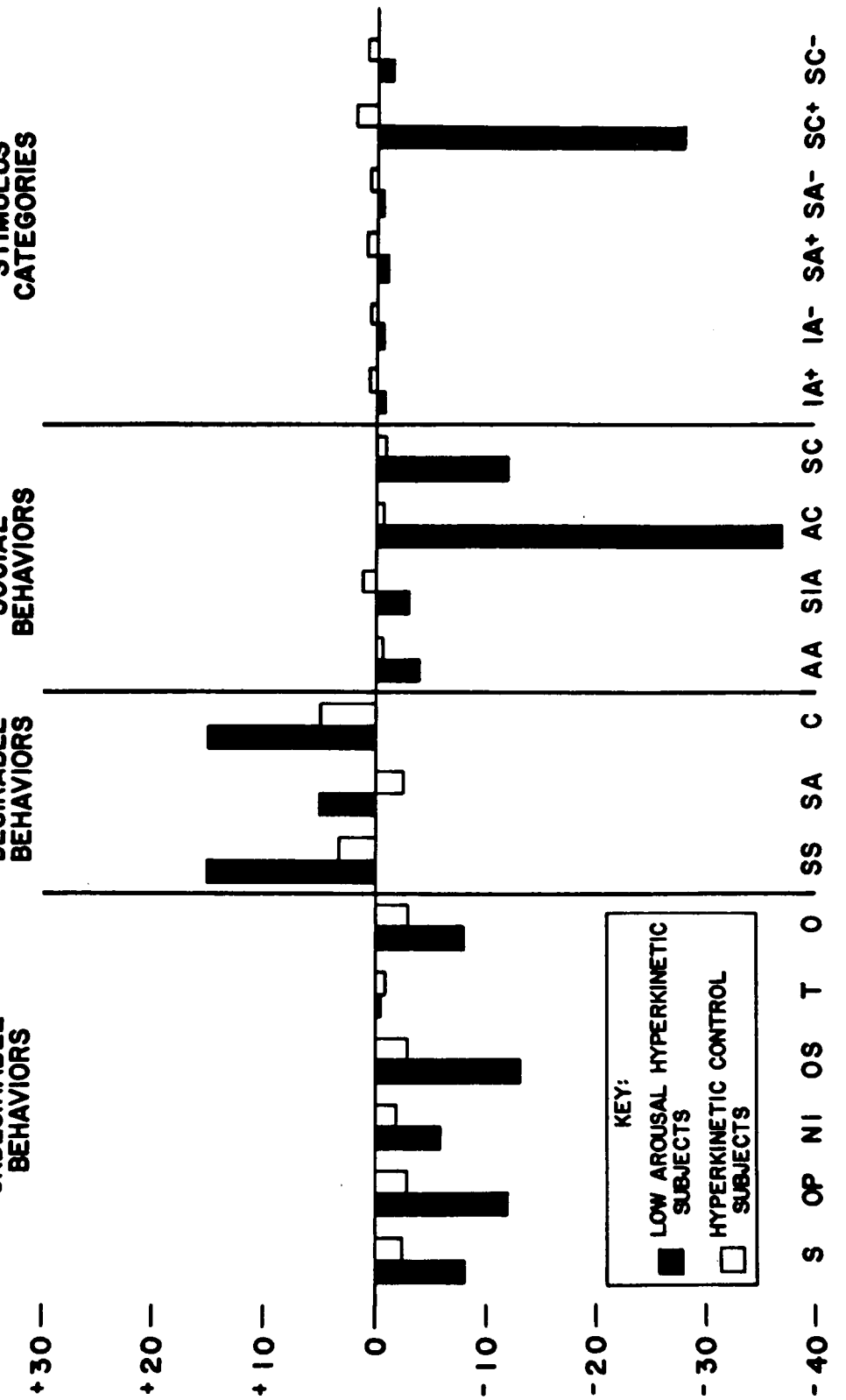


FIGURE 4

Phase I (NO DRUG) vs. Phase II (DRUG ONLY) Changes in Behavior and Stimulus Categories in Low Arousal and Control Hyperkinetic Children

production again confirms its customary association with enhanced behavioral immobility. The fact that EMG increases rather than decreases with medication corroborates a general increase in physiological arousal level; at the same time, this change contradicts EMG's usual negative correlation with SMR production. This finding is consistent with the view that behavioral improvements associated with stimulant drug medication result from increasing general physiological arousal rather than developing as a secondary outcome from enhanced internal motor control. The latter effect has been attributed to conditioned increases in SMR production and its role in alleviating various epileptic and sleep disorders (Sterman, 1976).

CHAPTER IV

TRAINING DATA

In the absence of a high arousal group of hyperkinetic subjects, all four subjects in the low arousal group were selected for training. Physiological and behavioral profiles are presented individually for each training subject before, during, and at the end of the EEG biofeedback sequence.

Case 1

Subject 1, a male Caucasian of normal intelligence (Otis Lennon, form KE2, IQ=104), was 11 years, 8 months 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 (11 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.

Laboratory Findings

Figure 5 represents evidence supporting the notions that contingent EEG feedback produces orderly changes in SMR production relative to daily and pre-training baselines and that corresponding changes in EMG occur in the opposite direction as training progresses. SMR and EMG values during the training sequence represent the following ratio:

$$\frac{\bar{x} \text{ criterions per minute during initial 15-minute feedback period}}{\bar{x} \text{ criterions per minute during initial 5-minute baseline period}}$$[1]

Comparable values are provided for pre-training baseline sessions when no feedback is presented.

Relative to pre-training baseline, negligible training effects occur during early SMR training sessions and are successively replaced by substantial increases in SMR ratios. Pre-training performance level are temporarily resumed following a three-week lapse in SMR training and the contingency reversal phase. Original training effects are recovered when the SMR contingency is reinstated and are

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[1]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 carryover 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 studies in other laboratories (e.g., Sterman and Friar, 1972; Sterman, 1974). EMG values are restricted to the same time intervals.

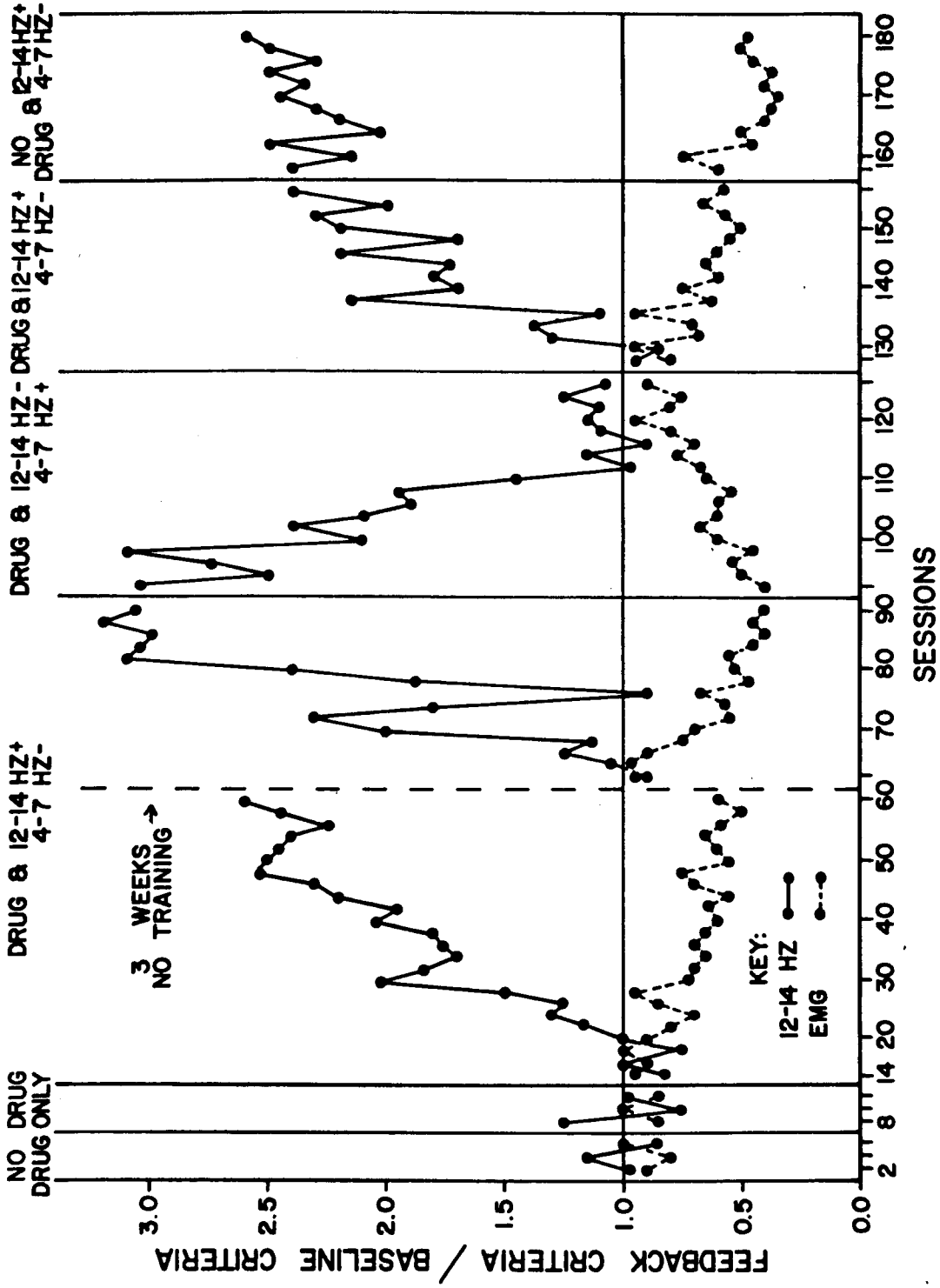


FIGURE 5

SMR and EMG Ratios over Six Phases for Subject 1

sustained following some variability in Phase VI when medication is gradually withdrawn (5 mg/week). Finally, the inverse relationship between SMR and EMG is supported by moderate but statistically insignificant outcomes in the brief pre-training phases (I: $r = .31$, $t = .66$ $df = 4$, $p > .1$, II: $r = .33$, $t = .63$, $df = 4$, $p > .1$)[2] and by statistically significant ones during the three SMR training phase (III: $4 = -.48$, $t=3.43$, $df=37$, $p < .05$; V: $r=.63$, $t=2.63$, $df=1-$, $p < .05$; VI: $r = -.75$, $t = 3.26$, $df = 10$, $p < .05$).

Figure 6 includes additional data supporting the conclusion that SMR production increases with biofeedback training in subject 1. SMR values for the initial and final 5-minute baseline periods and the initial 15-minute feedback period represent average percentages of seconds of 12-14 Hz activity and are derived from the following formula:

$$\bar{x} \quad \frac{\text{instances of 12-14 Hz activity/minute}}{13} \times \frac{100}{60}^{3}$$

Even though biofeedback was not presented for individual SMR instances, trends in this measure correspond to those for feedback criteria (SMR bursts) in Figure 5.

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[2]Correlations were computed across daily sessions in each pre-training phase.

[3]This conversion is partly justified on the basis of our detection system, which requires a signal duration from 1/12th-1/14th second for any wave to qualify as a discrete SMR instance. Assuming the center frequency of 13 Hz, each instance represents 1/13th second of 12-14 Hz activity.

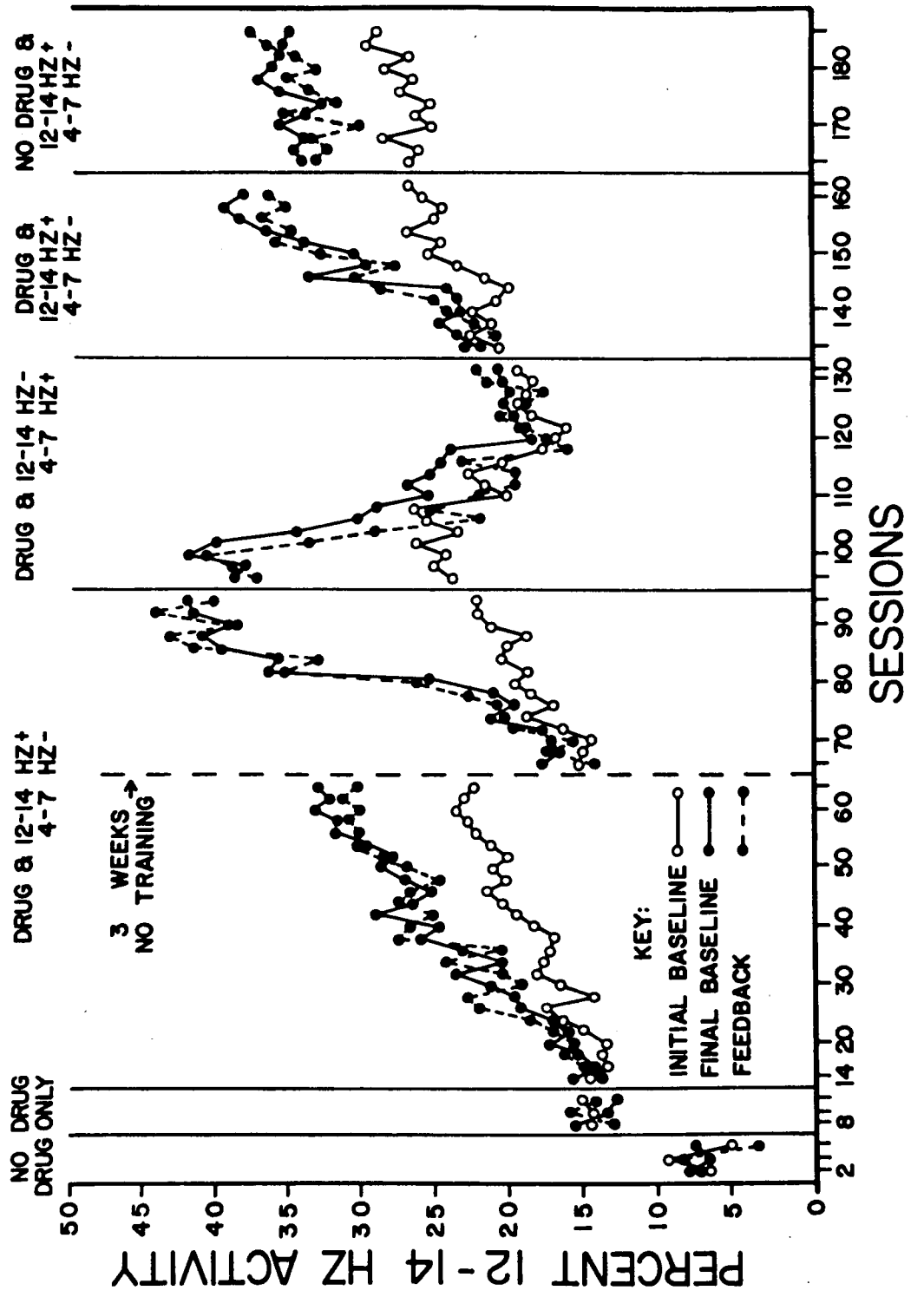


FIGURE 6

Mean Percent 12-14 Hz activity during Initial Baseline, Feedback, and Final Baseline Periods over Six Phases for Subject 1

Relative to pre-training sessions, training effects in Figure 6 are absent in the first few training sessions, after which feedback values regularly exceed those for the initial baseline period. This corresponds to the trends evidenced in SMR ratios in Figure 7, and the difference between the two periods is statistically significant only during SMR training phases (I: $t = 1.11$, $df = 4$, $p \not> .1$; II: $t = .91$, $df = 4$, $p > .1$; III: $t = 10.5$, $df = 37$, $p < .05$; IV: $t = 2.43$, $df = 19$, $p < .05$; V: $t = 7.89$, $df = 12$, $p < .05$; VI: $t = 3.96$, $df = 10$, $p > .05$). It should be noted that a reduction in the percent of 12-14 Hz activity during the contingency reversal phase is accompanied by a reduction in the differences between the two periods even though this discrepancy remains statistically significant.

In contrast to the initial five-minute baseline, the percent of 12-14 Hz activity during the final five-minute baseline period regularly exceeds feedback values after training effects are established. However, the difference between the two periods does not progressively increase with training time and is not statistically significant across any of the SMR training phases (III: $t = 1.59$, $df = 37$, $p > .1$; IV: $t = .59$, $df = 19$, $p > .1$; V: $t=1.33$, $df=12$, $p > .1$; VI: $t=1.45$, $df=10$, $p > .1$). Nevertheless, the fact that the initial percentage of SMR activity is virtually tripled in both feedback and final 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. A similar but less striking shift occurs during the initial five-minute baseline period. Baseline shifts are further illustrated by the overall decline in 12-14 Hz activity by the end of the contingency reversal phase during both baseline and feedback periods. A demonstrable shift in baseline SMR appears essential if valid extrapolations from the laboratory to general experience are to be made.

Corresponding changes in the predicted direction are also evidenced in the remaining CNS arousal measures. Representative samples of EEG power spectra during the six phases are presented in Figure 7. Each sample represents 14, 8 second epochs assessed during the initial baseline period Relative to the NO DRUG phase, the anticipated decline in slow wave activity between 4-7 Hz during the DRUG ONLY phase is associated with a moderate increase in power in the 12-14 Hz frequency range. This trend is noticeably accentuated during the DRUG AND SMR training phases. A temporary reversal in this trend during SMR counter-conditioning (Phase IV) reinforces the view that the observed redistribution of power reflects treatment influences above and beyond those expected to accompany maturation alone. Furthermore, the fact that these changes are sustained during SMR conditioning in the absence of medication (Phase VI) reinforces the significance of the training procedure as a moderating influence.

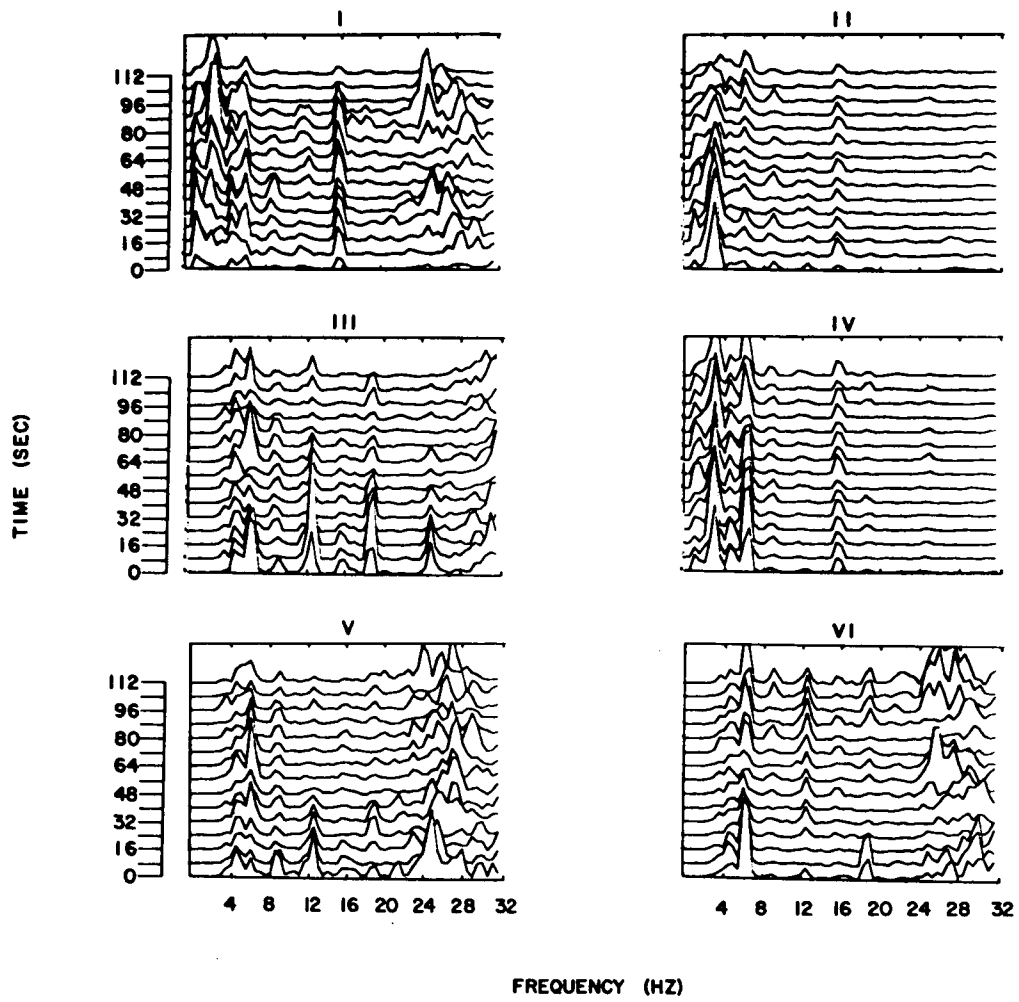


FIGURE 7

Changes in EEG Spectra over Six Phases for Subject 1

Similar trends are evidenced in evoked cortical response amplitudes and in GSR conductance, presented in Figures 8 and 9, respectively, in four of the six experimental conditions. Measures of these indices were not obtained during the contingency reversal and SMR retraining phases (IV, V). Evoked cortical responses from each hemisphere are averaged for each session,and a representative sample for each phase is presented in Figure 8. GSR conductance is averaged and presented in Figure 9 for each session. In both cases, pre-training trends established following the administration of drugs alone are enhanced following DRUG AND SMR training and are sustained by the training procedure when medication is withdrawn.

Classroom Observations

The behavioral and stimulus categories assessed in the classroom are described in Table A-1, Appendix A. Figures 10 and 11 present the average number of daily events scored per week for these categories under the 6 conditions. Reliabilities were computed for two 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%, 85% and 81% in the 6 conditions, respectively.

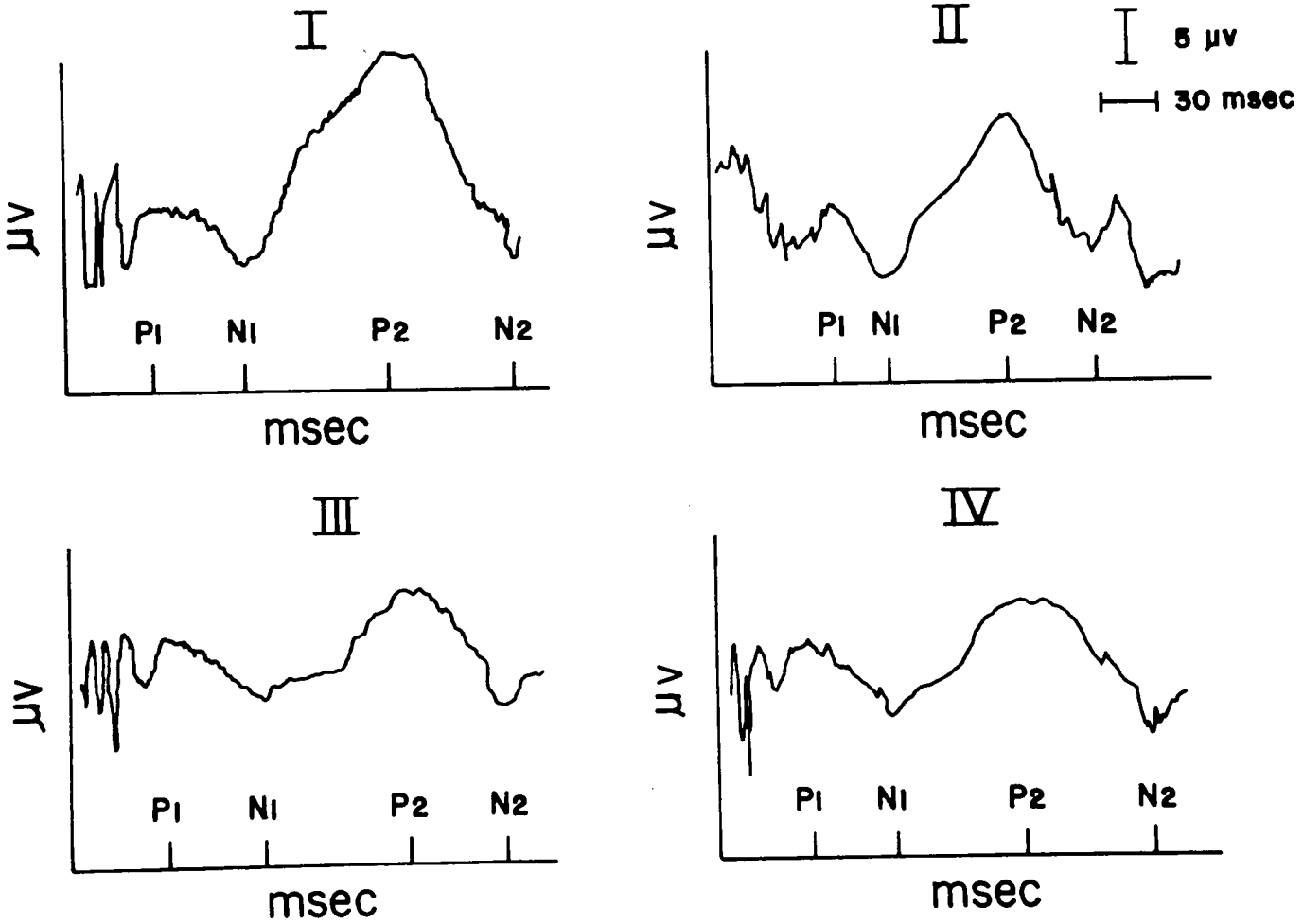


FIGURE 8

Changes in Cortical Evoked Response Amplitudes over Four Phases for Subject 1

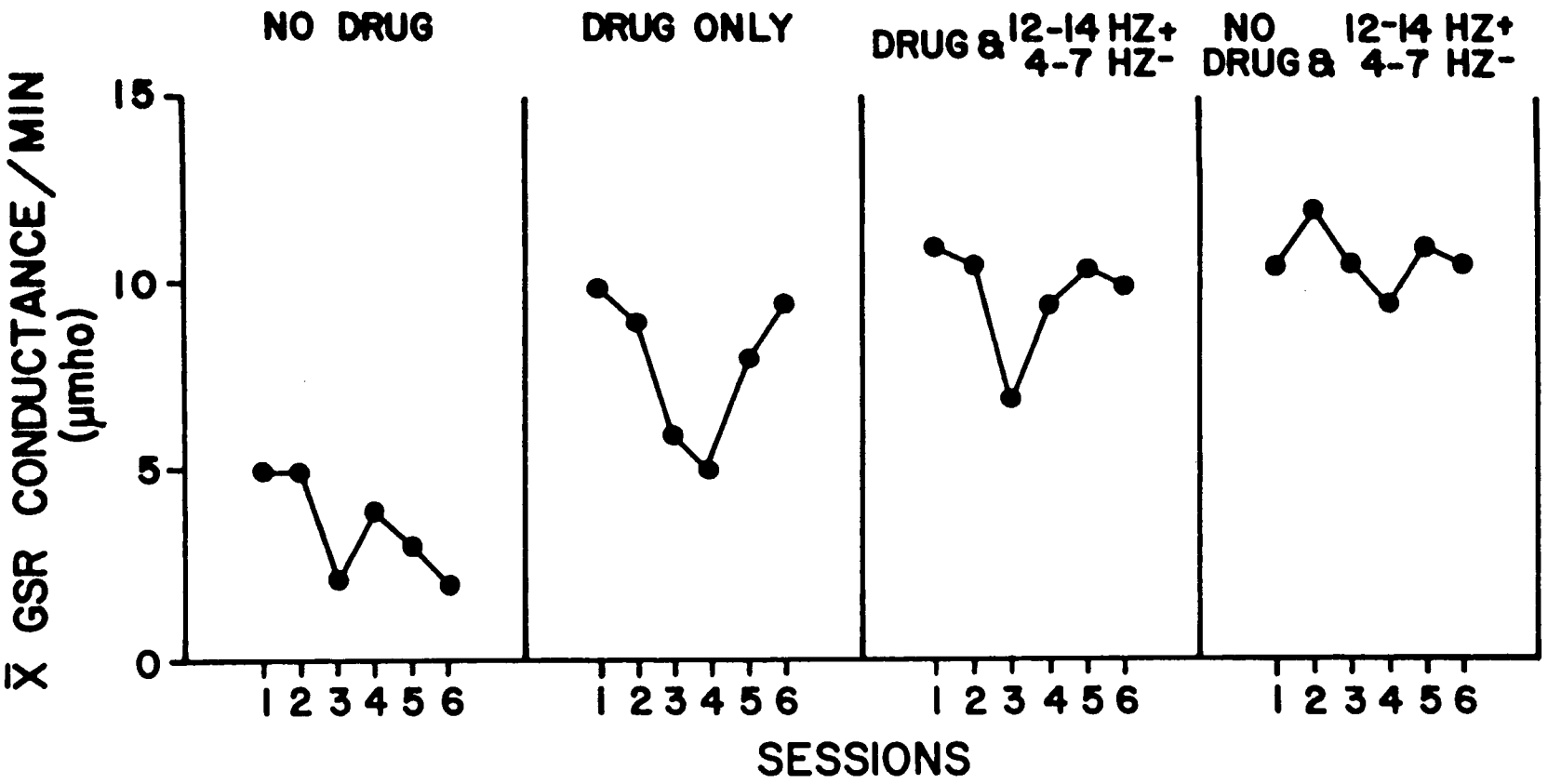


FIGURE 9

Changes in GSR Conductance over Four Phases for Subject 1

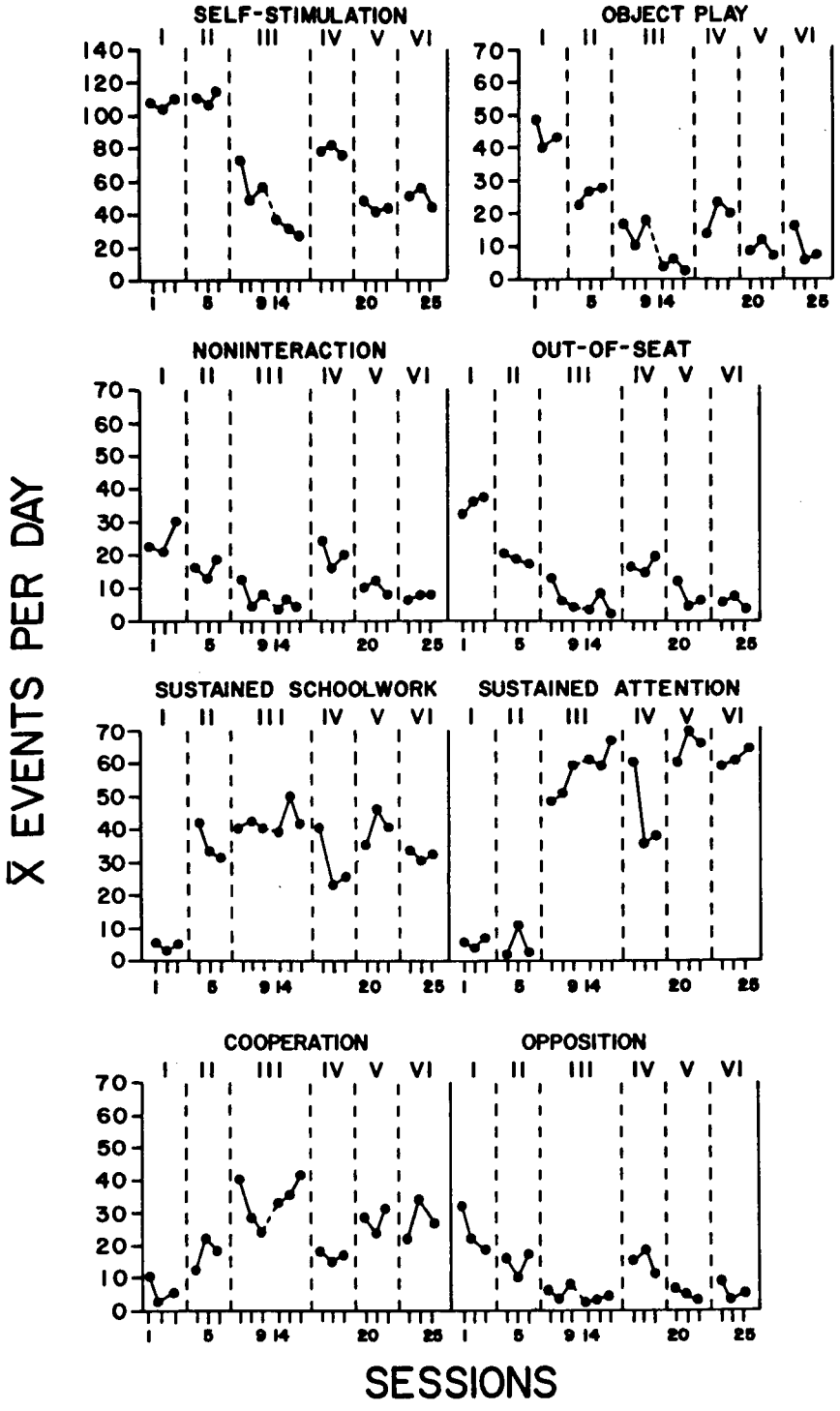


FIGURE 10

Predicted Changes in Eight Behaviors over Six Phases for Subject 1

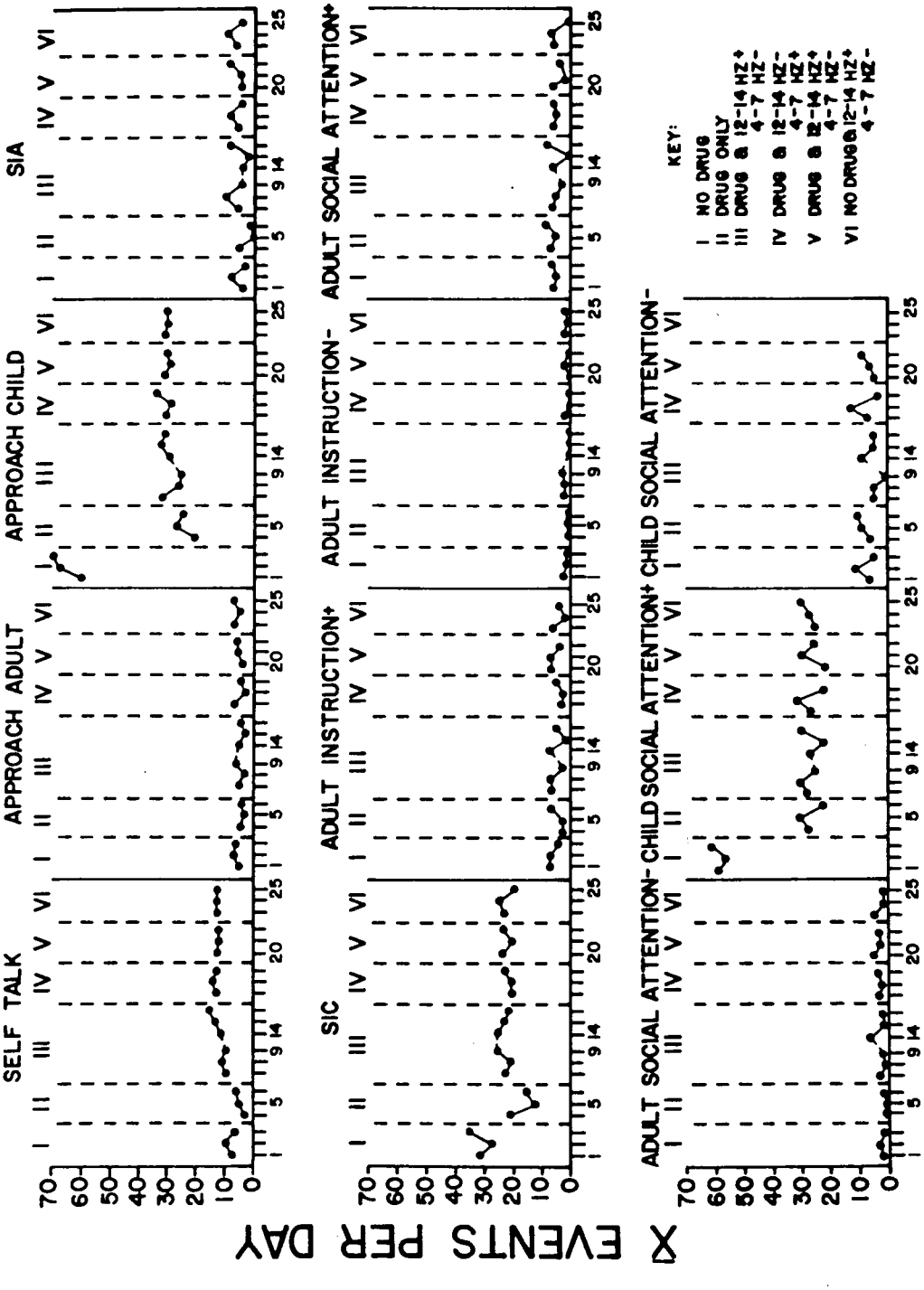


FIGURE 11
Unpredicted Changes in Five Behaviors and Changes in Stimulus Categories over Six Phases for Subject 1

Combining medication and SMR training was intended to enhance the level of improvement already achieved within drugs alone. The 8 behaviors included in Figure 10 exhibit predicted changes during SMR training. Relative to the NO DRUG phase, improvement in six of those categories is displayed during the DRUG ONLY phase. The most dramatic effect occurs in classwork, although the subject focuses on academic materials (sustained school work) to the relative exclusion of attending to lectures (sustained attention). The slight, but unanticipated increase in self-stimulation in this subject may be accounted for by the rather exclusive increase in classwork with which it usually correlates positively (Wahler, 1975). When drug and SMR are combined, even further improvement occurs in the behaviors benefitted by medication, and substantial changes are evidenced in the two that were not (self-stimulation and sustained attention).

The remaining 5 behaviors included in Figure 11 failed to change in the predicted direction during SMR training. One undirected activity (self-talk) and all four social behaviors occur at moderate or high frequencies during the NO DRUG phase. Uniform decreases in them following DRUG ONLY are reversed during SMR conditioning although pre-training levels under NO DRUG are not resumed. No explanation is currently available for this trend in self-talk. However, the unanticipated increase in this

subject's social behavior may have resulted from his being reinstated into work groups with other children during all training phases. Under the first two conditions, the child was isolated from the remainder of the class in order to restrict his social interactions.

Changes in stimulus categories are also presented in Figure 11. Positive and negative instructions as well as negative social attention from both teachers and peers occur infrequently during the study regardless of the experimental condition. In contrast, relative to the NO DRUG phase, positive social attention from peers decreases during both treatment conditions, varying inversely with improvement in the child's hyperkinetic behaviors in general and as a direct function of the child's reduced social approaches (AC) in particular. The average correlation between the two events is .64 in the six conditions with a range of .51 to .72 in individual conditions, indicating that the relationship between them is fairly consistent, despite overall reductions in their frequency during DRUG ONLY and SMR training phases. On the basis of these data, then, it would be difficult to conclude that changes in peer reinforcement contribute directly, or even positively, to the observed changes in target indices of hyperactivity.

Case 2

Subject 2 is also a male Caucasian of normal intelligence (WISC=92) and was 10 years, 7 months at the inception

of the training period in March, 1975. His parents reported excessive overactivity that was manageable until the child was first enrolled in elementary school (6 years, 1 month). At the school's request, he was referred to a pediatrician who prescribed Ritalin (10 mg./school day). An optimal dosage level was subsequently established (20 mg./school day), and this regimen has been maintained even though the parents contend that his hyperactivity is more pronounced at school than at home.

Laboratory Findings

Figure 12 presents SMR and EMG ratios over the 6 experimental conditions for subject 2. Acquisition of the SMR task is indicated by contingent increases in SMR production during feedback periods (Phases III, V, VI) relative to daily and pre-training baselines and by contingent decreases in SMR output during counter-conditioning (Phase IV). Not unlike subject 1, pre-training baselines under the DRUG ONLY condition are not completely resumed during the contingency reversal phase. The inverse relationship with EMG is again supported (Phase I: $r = -.41$, $t = .89$, $df = 4$, $p > .1$; II: $r = -.29$, $t = .60$, $df = 4$, $p > .1$; III: $r = -.45$, $t = 2.65$, $df = 28$, $p < .05$; IV: $r = -.53$, $t = -2.21$, $df = 13$, $p < .05$; V: $r = -.6$, $t = 2.4$, $df = 10$, $p < .05$; VI: $r = -.48$, $t = -2.09$, $df = 10$, $p < .05$). Although absolute differences are masked by the ratio transformation, it is

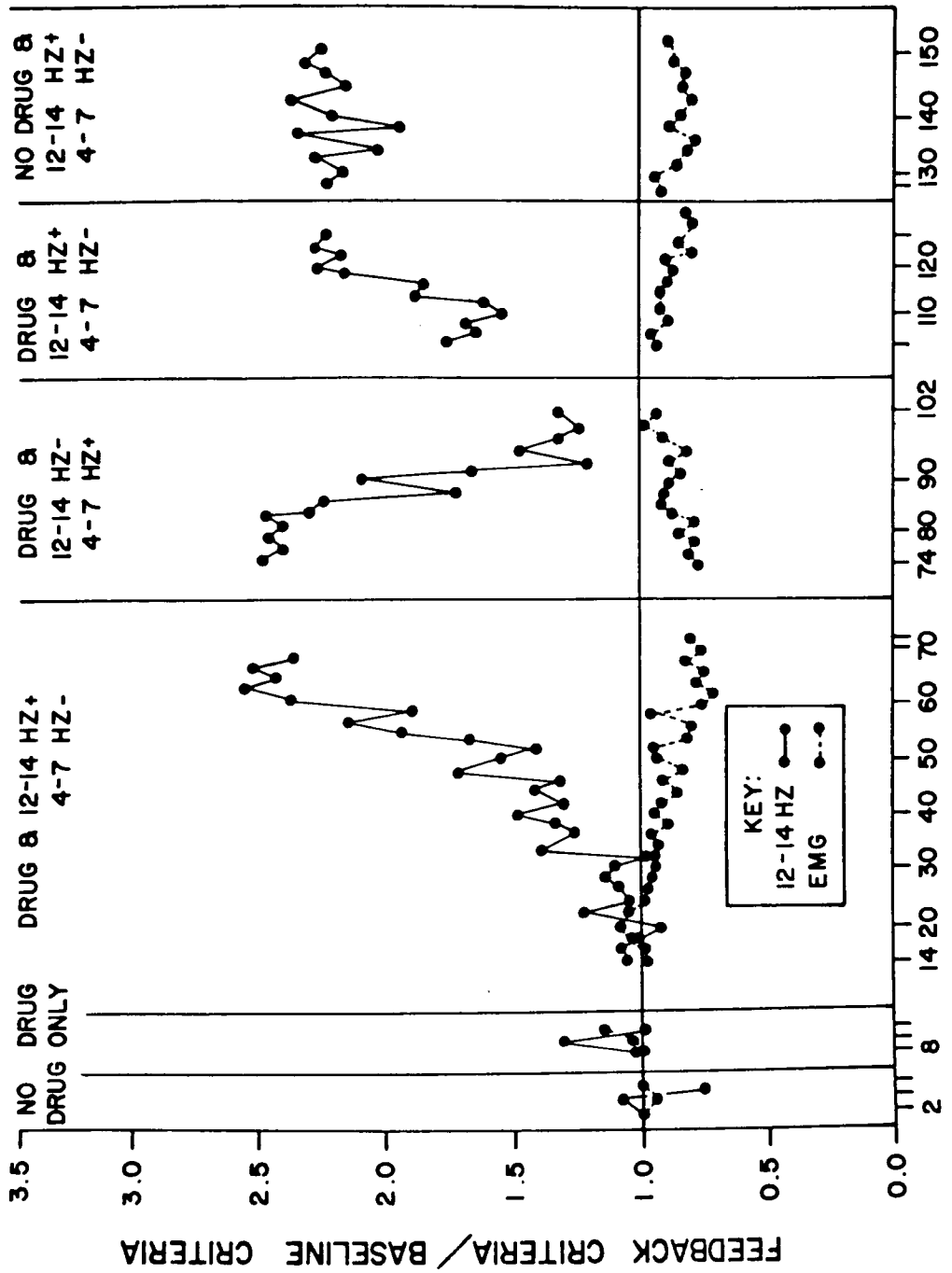


FIGURE 12

SMR and EMG Ratios over Six Phases for Subject 2

noteworthy that dependent change in the production of both responses are of lesser magnitude throughout both treatment conditions in subject 2 than in subject 1.

Figure 13 provides evidence that changes in percent of 12-14 Hz activity accompany both drug-induced and reinforced changes in SMR criteria. Again, subject 2 displays higher pre-treatment levels of SMR and smaller relative increases following medication and SMR training than does subject 1. Nevertheless, evidence of a baseline shift clearly emerges during both five-minute baseline periods although less substantial than that observed in subject 1 and in spite statistically negligible differences between feedback and final baseline period levels during training (III: $t = 1.11$, $df = 28$, $p > .1$; V: $t = .98$, $df = 10$, $p > .1$; VI: $t = 1.21$, $df = 10$, $p > .1$) Differences between the percent of 12-14 Hz production during feedback and initial five-minute baseline period are nevertheless significant (III: $t = 2.31$, $df = 28$, $p < .05$; V: $t = 2.98$, $df = 10$, $p < .05$; VI: $t = 1.95$, $df = 10$, $p < .05$)

The impact of trained SMR changes on the EEG is further corroborated by corresponding tends in EEG spectra and in evoked cortical response amplitudes, presented in Figures 14 and 15 respectively. Relative to the low frequency, high voltage spectral components and high amplitude evoked cortical responses during the NO DRUG phase, progressive trends towards higher resting frequencies in the background EEG and lower voltage evoked

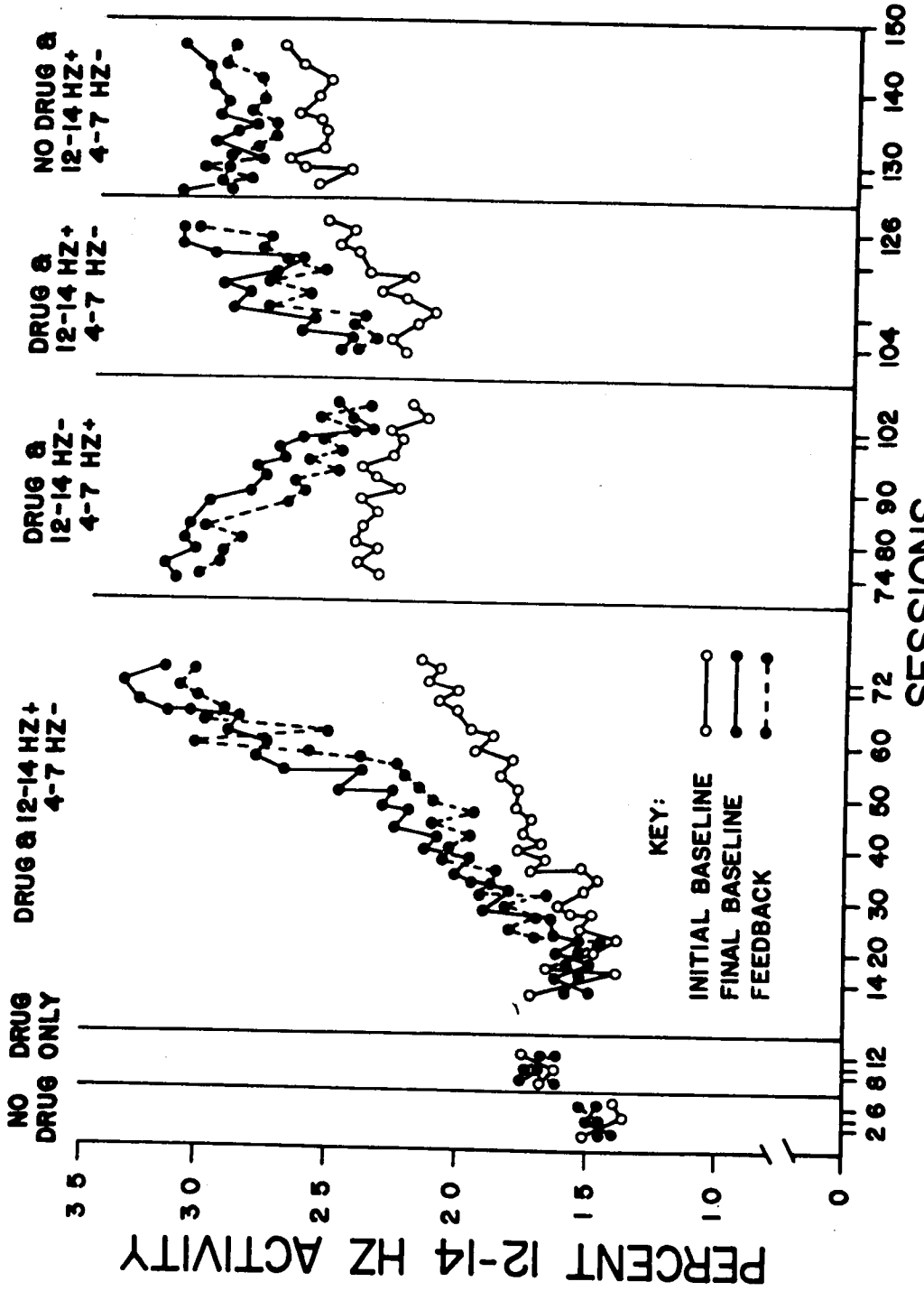


FIGURE 13

Mean Percent 12-14 Hz Activity during Initial Baseline, Feedback, and Final Baseline Periods over Six Phases for Subject 2

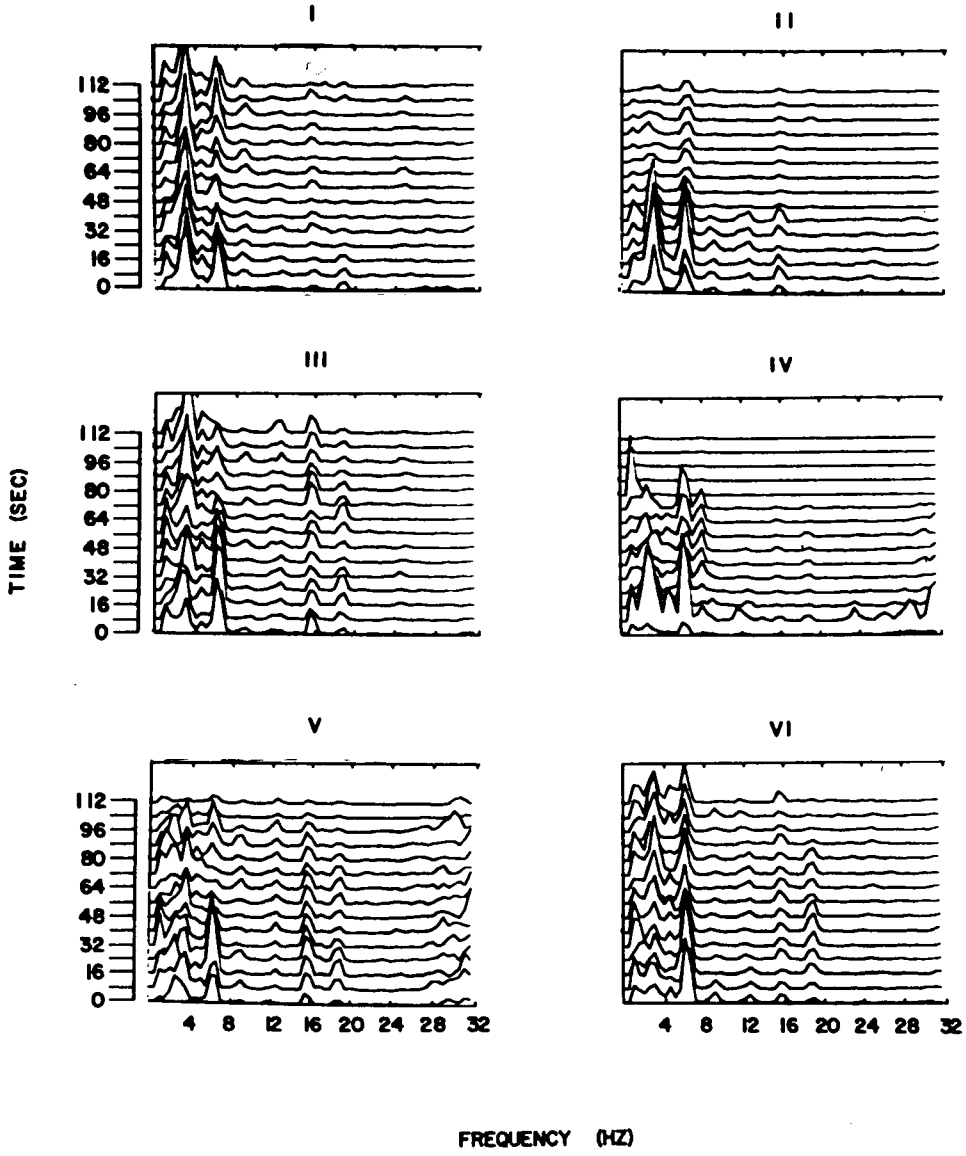


FIGURE 14

Changes in EEG Spectra over Six Phases for Subject 2

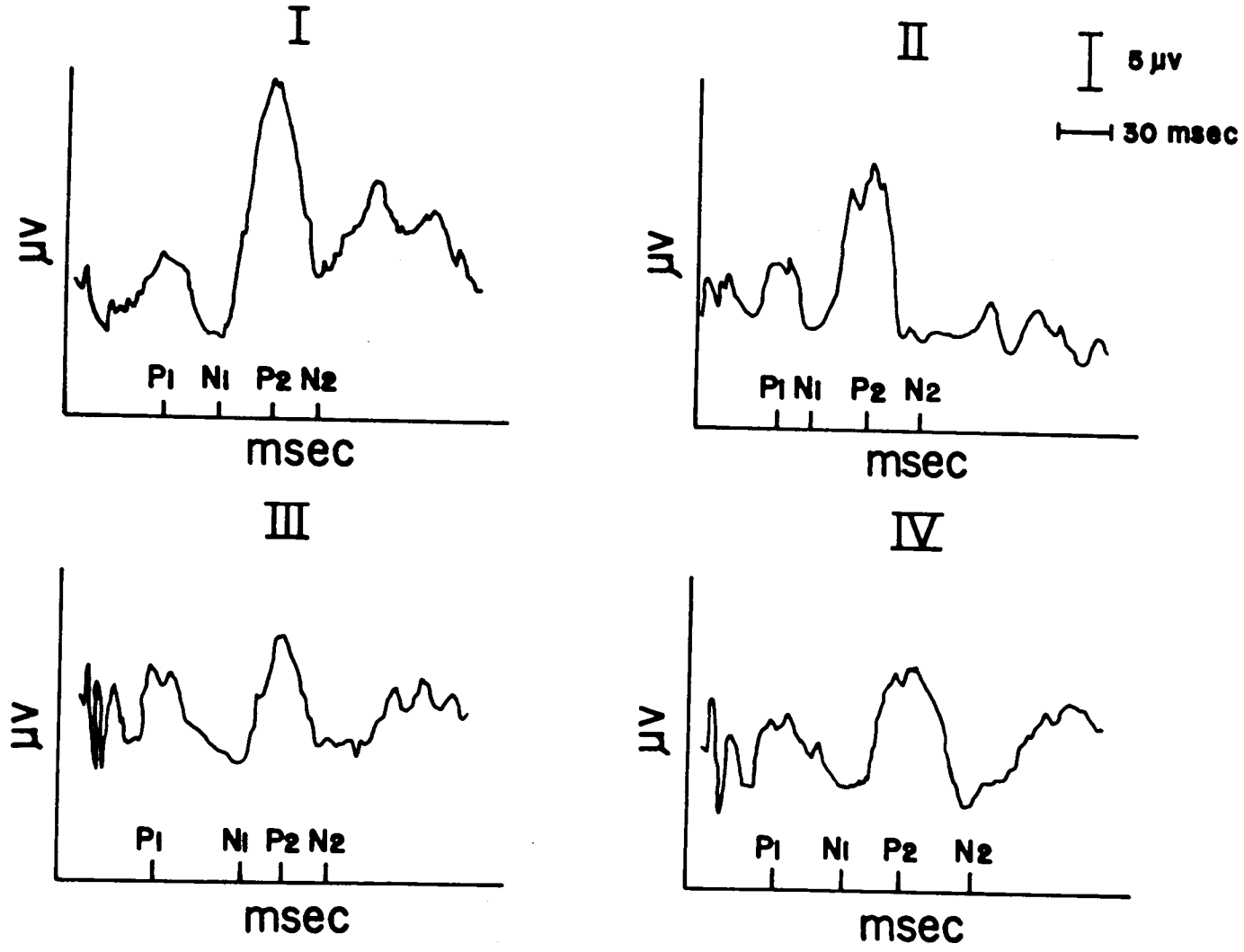


FIGURE 15

Changes in Cortical Evoked Response Amplitudes over Four Phases for Subject 2

cortical responses are exhibited during subsequent treatment conditions. Normalization of the subject's pretreatment low arousal profile is also indicated by the treatment-induced increases in skin conductance shown in Figure 16.

Classroom Observations

Figures 17 and 18 present changes in the category system for subject 2 throughout the experiment. Average interobserver reliabilities, computed as previously described, were 80%, 92%, 95%, 93%, 96% and 84% for the 6 conditions, respectively.

Figure 17 includes the 8 behaviors showing predicted changes during SMR training. They are the same categories in which subject 1 showed improvement. Relative to the NO DRUG condition, moderate increases in both forms of sustained classwork occur in conjunction with the anticipated decline in self-stimulation during the DRUG ONLY phase. These features differentiate the two subjects. Otherwise, principal changes following drug administration in subject 2 consist of the expected reduction in purposeless motor activities, especially object play and out-of-seat behaviors. The combination of drug and SMR training results in further improvements in these behaviors and produces stable reductions in oppositional behavior and noninteraction, the two undesirable behaviors that are largely unaffected by medication.

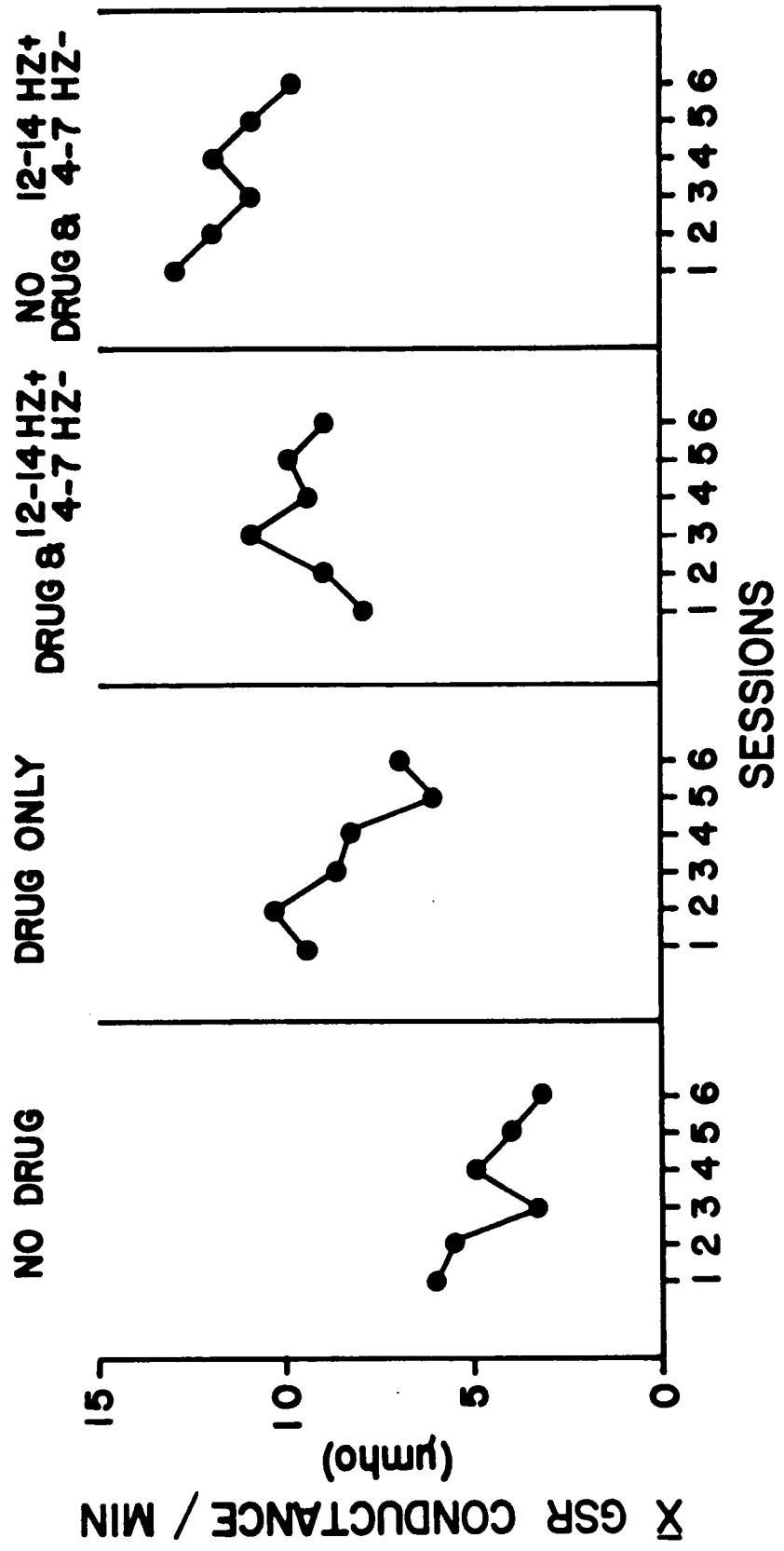


FIGURE 16

Changes in GSR Conductance over Four Phases for Subject 2

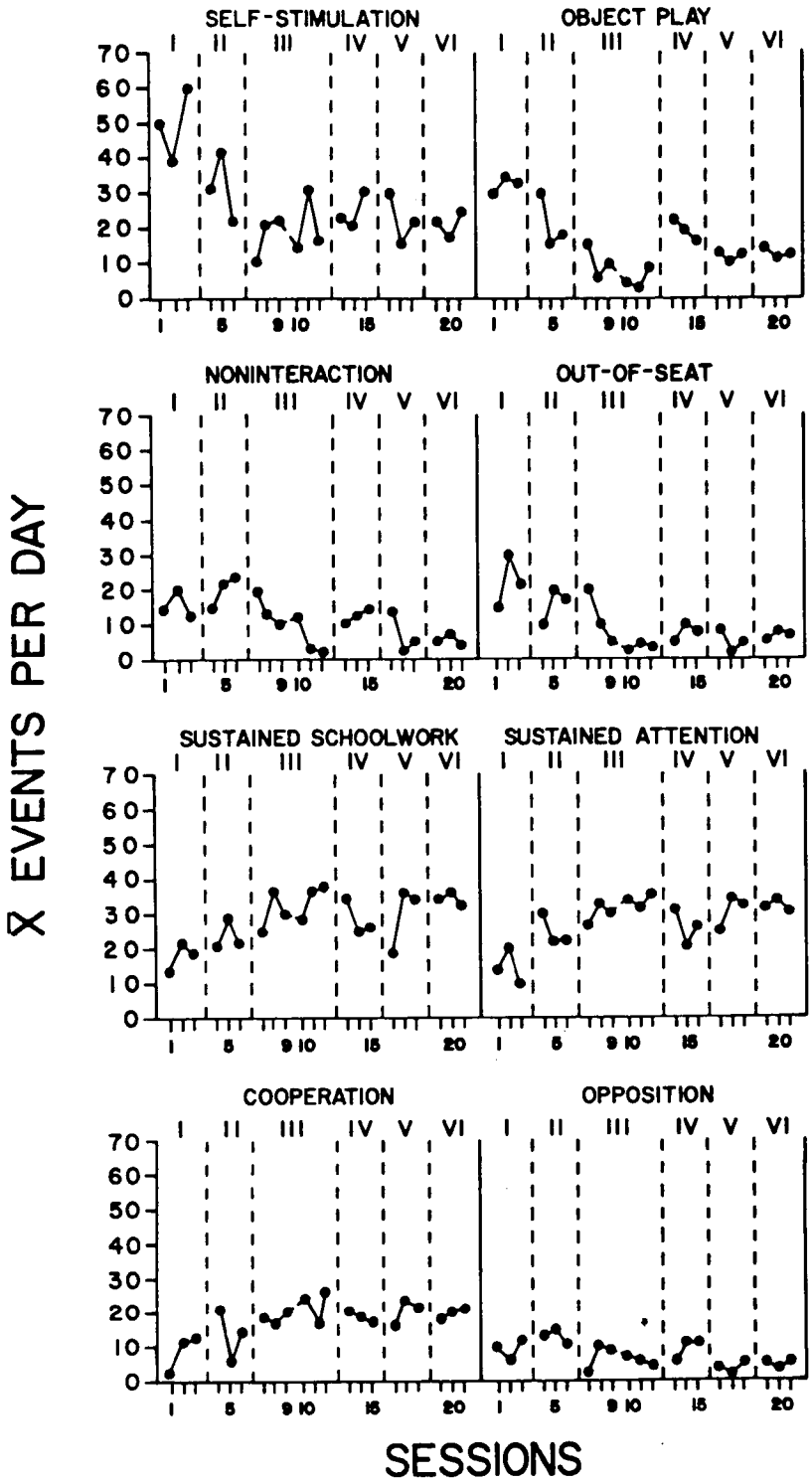


FIGURE 17

Predicted Changes in Eight Behaviors over Six Phases for Subject 2

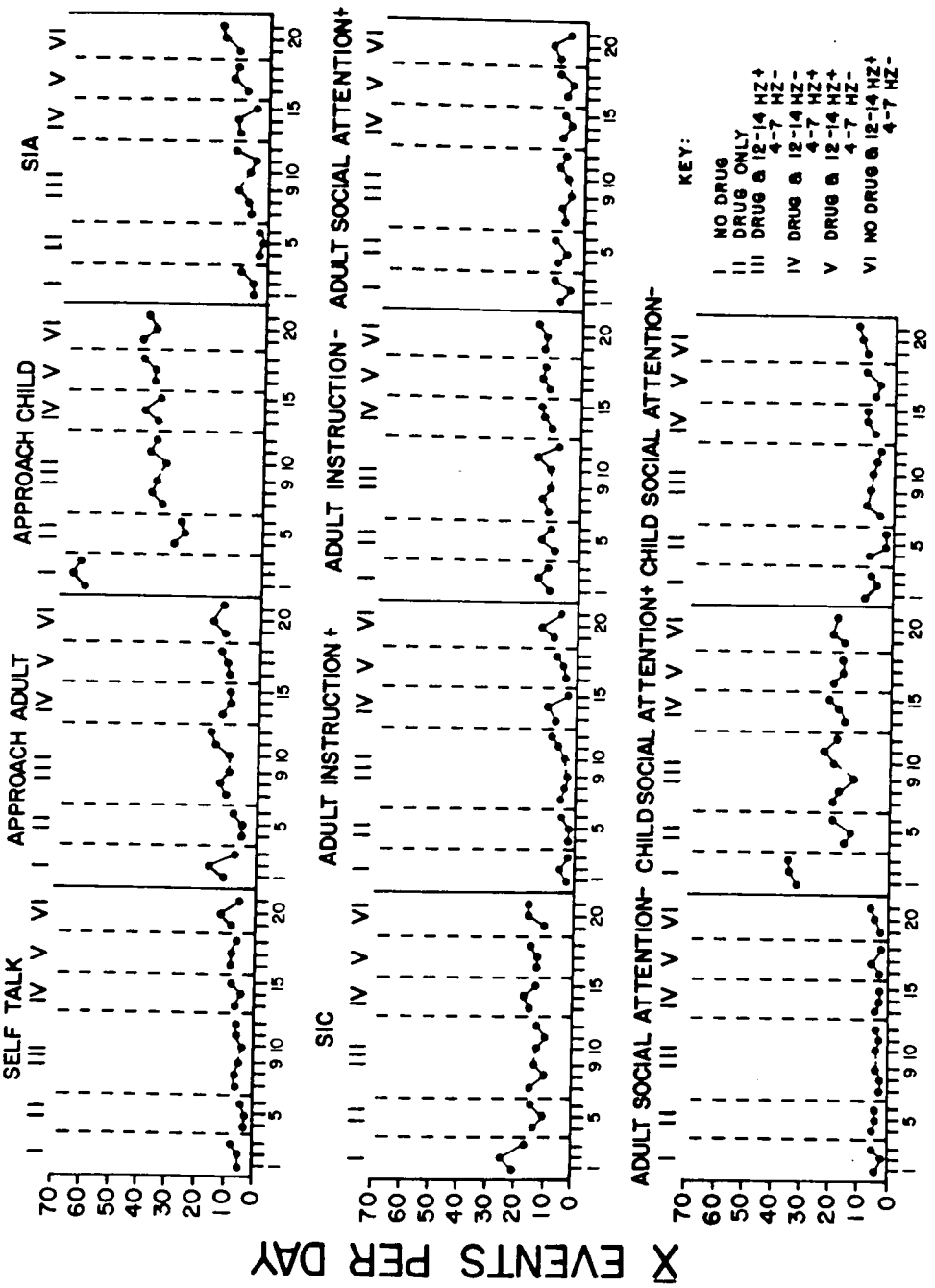


FIGURE 18

Unpredicted Changes in Five Behaviors and Changes in Stimulus Categories over Six Phases for Subject 2

The remaining behaviors, self-talk and the four social behaviors, fail to change in the predicted direction during SMR training although stable decreases in them are displayed with medication alone. These data are provided in Figure 18. No satisfactory explanation is currently available to account for the partial return of these symptoms. Subject 2 had not been segregated from his classmates, for example, so that, unlike Subject 1, obvious changes in classroom routines do not appear responsible.

Figure 18 also includes the stimulus categories. The most substantial drug-induced changes is revealed in the highest frequency pre-treatment reinforcer, positive social attention from peers. Also, although it is not apparent from the viewing Figures 17 and 18, high frequency peer reinforcement in the pre-treatment condition (Phase I) is most regularly associated with the target's social approaches ($r = .73$, $t = 4.8$, $df = 13$, $p < .05$), and the subsequent decline in these behaviors appears responsible for corresponding reductions in peer attention.

Case 3

Subject 3 (WISC IQ=95) is the younger brother of subject 1 and was 10 years, 10 months at the beginning of the EEG biofeedback training sequence in June, 1975. This subject's behavior problems differ from his sibling's in remaining relatively unnoticed until he entered elementary school. Although both parents and teachers report excessive

"fidgeting" the subject's inability to focus his attention for more than a few seconds has been their most serious concern. Signs of overactivity are, in their opinion, most reliably revealed in his apparently purposeless wandering about both home and classroom settings. His regular placement in semi-open classroom situations has not discouraged these problem behaviors, and he has been suspended from school twice and expelled once in the past 3-year period. He was admitted to the Riverbend program for 9 months following the first suspension in February of 1974. A complete return of symptoms following his release resulted in his being placed on 10 mg. Ritalin (February, 1975) and referred for this study. He participated unsuccessfully in the training program for six months, at which time he was suspended and then expelled from elementary school (January, 1975). He was terminated in the training program at the same time and is currently attending a local school for the emotionally handicapped.

Laboratory Findings

SMR and EMG ratios for the three experimental conditions appear in Figure 19. In contrast to both previous cases, subject 3 clearly failed to demonstrate acquisition of the SMR task despite extensive exposure to the training procedure. The inverse relationship between SMR and EMG is again revealed although the moderate correlations coefficients merely reinforce the often reported systematic

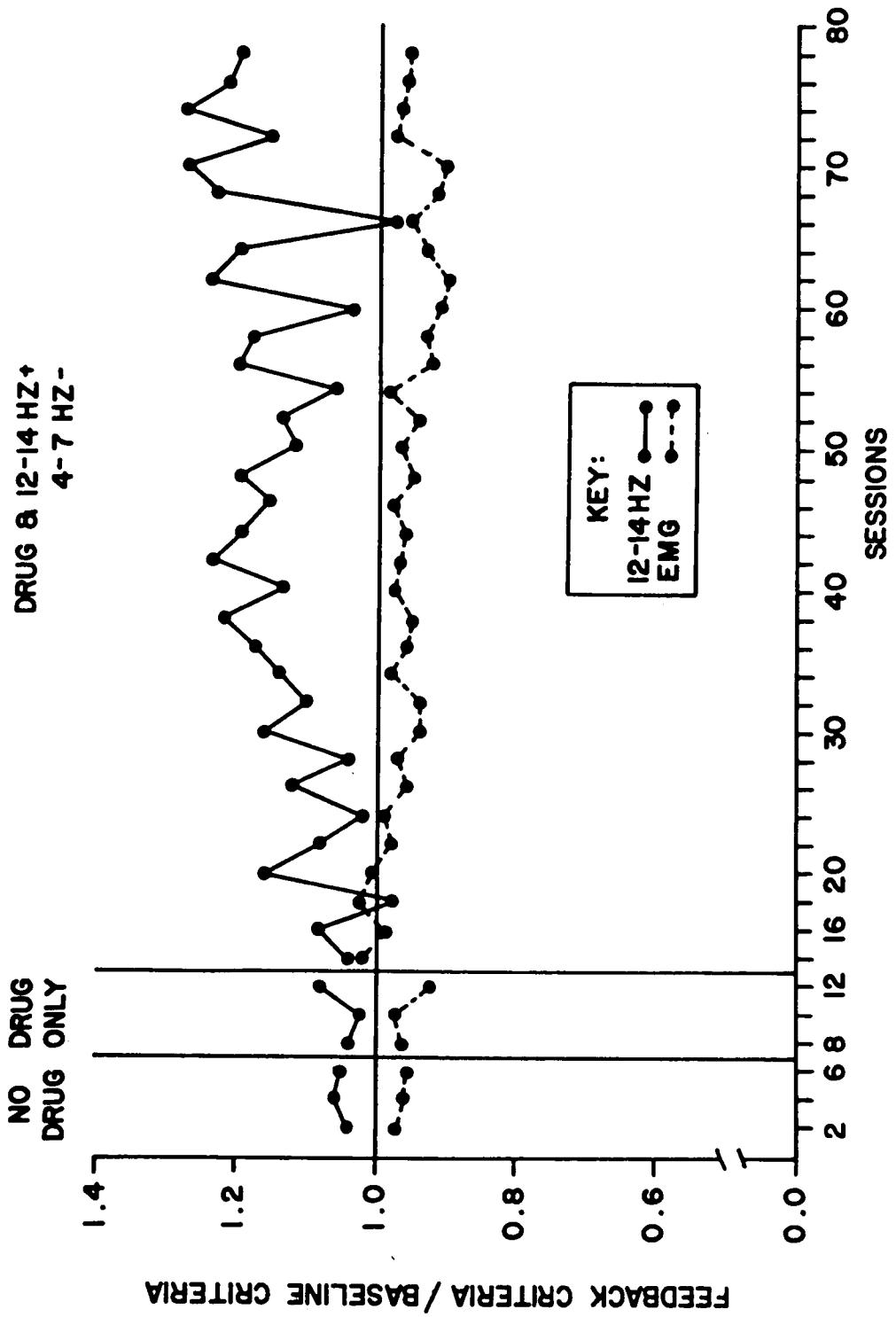


FIGURE 19

SMR and EMG Ratios over Three Phases for Subject 3

relationship between spontaneous changes in these two indices (I: $r = -.29$, $t = .6$, $df = 4$, $p > .1$; II: $r = -.31$, $t = .6$, $df = 4$, $p > .1$; III: $r = -.34$, $t = 2.13$, $df = 35$, $p > .05$).

The remaining physiological indices indicate that moderate changes in the predicted direction following medication alone are sustained but not enhanced during unsuccessful SMR training. Changes in the percent 12-14 Hz activity, presented in subject 3 in Figure 20 show pre-treatment levels of approximately 20% that are increased to 27% following Ritalin administration and are maintained at that level after the addition of the feedback treatment. As indicated in Figures 21, 22 and 23, corresponding trends towards a normalization of arousal level in this subject are displayed in EEG spectra, evoked cortical response amplitudes, and CSR conductance, respectively.

It is noteworthy that pre-treatment levels of SMR productions are higher in this subject than in the other low arousal children since overactivity per se is reportedly less severe a problem for him than is distractibility. This observation is indirectly supported by the subject's higher amplitude evoked cortical responses, which double as indicators of low arousal and short attention span. Taken together, these findings suggest attention span as the central deficit, a fact which may account for this subject's difficulty in task acquisition.

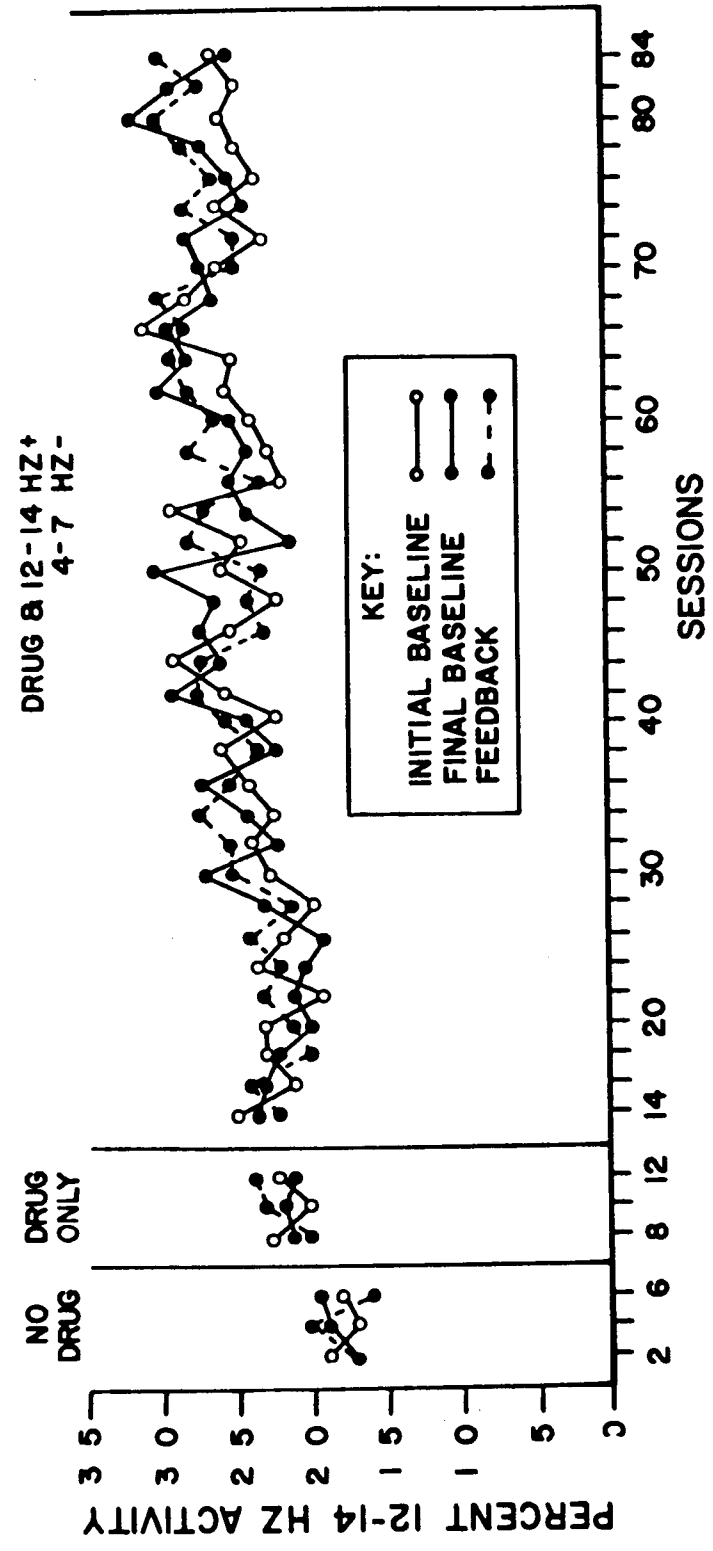


FIGURE 20

Mean Percent 12-14 Hz Activity During Initial Baseline, Feedback, and Final Baseline Periods over Three Phases for Subject 3

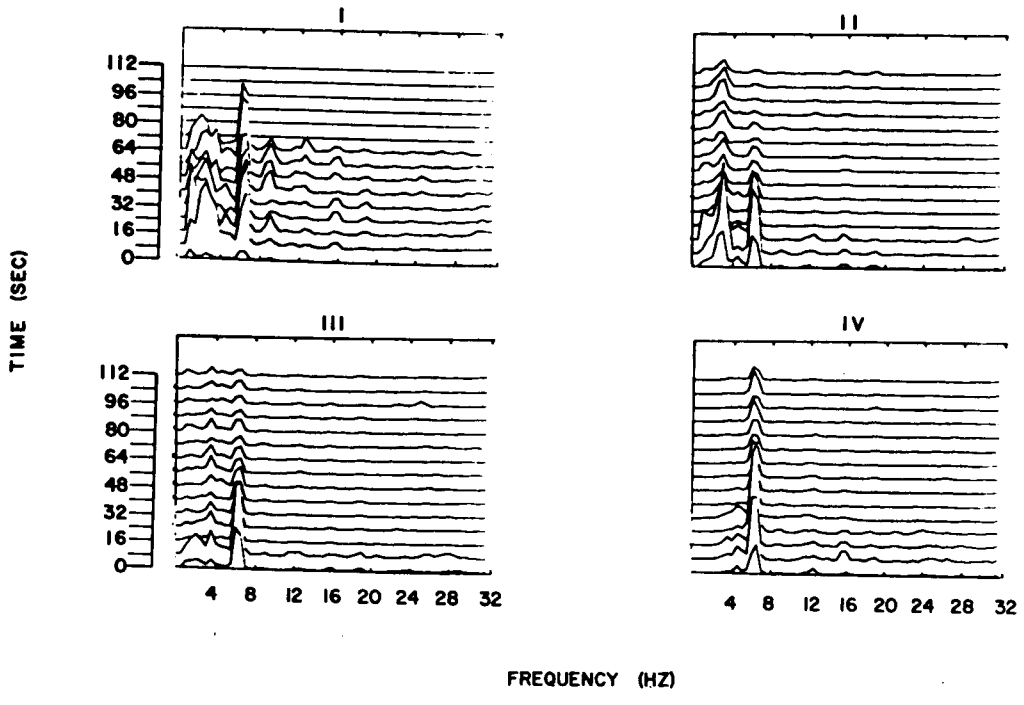


FIGURE 21

Changes in EEG Spectra over Four Phases for Subject 3

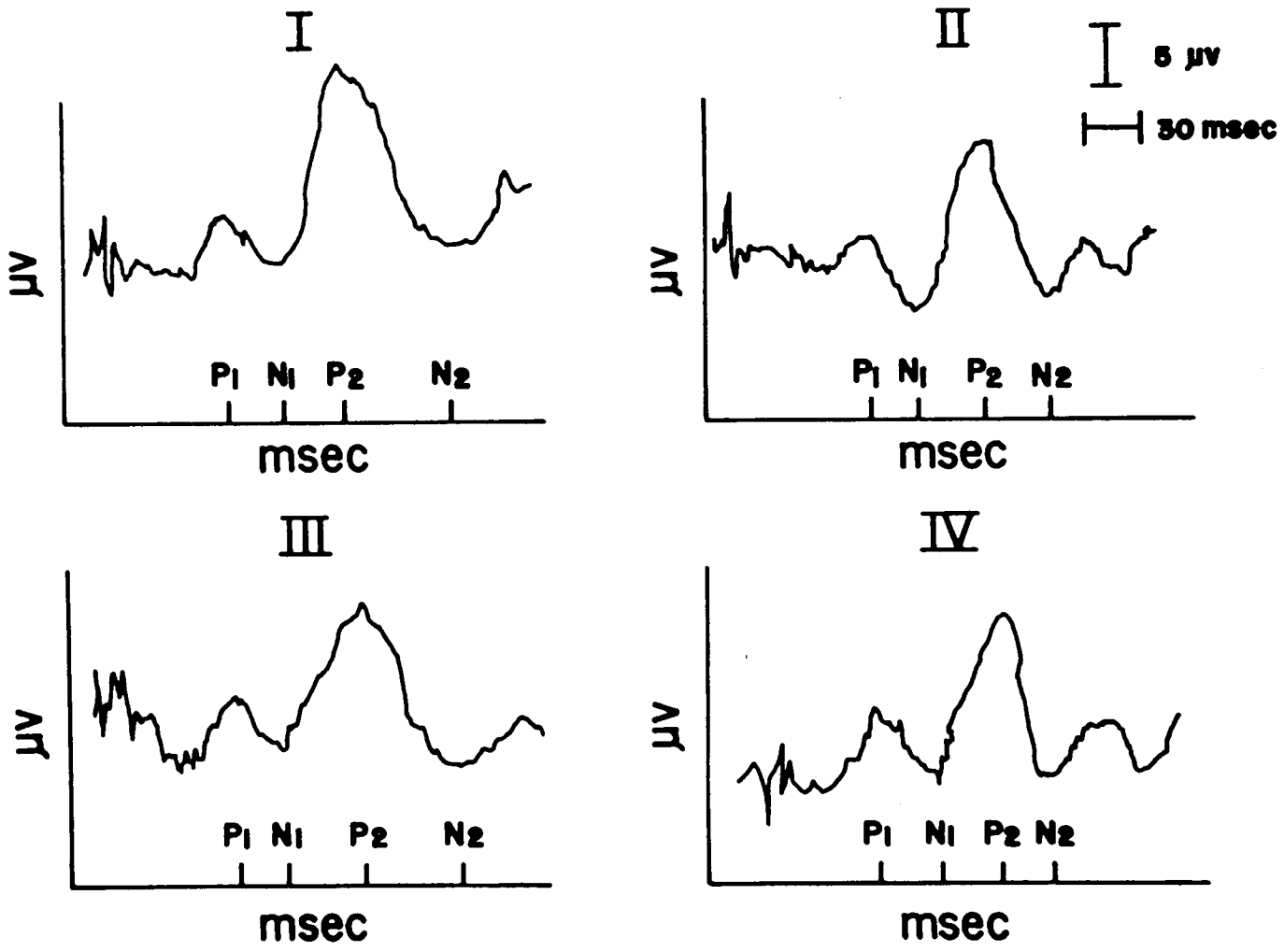


FIGURE 22

Changes in Cortical Evoked Response Amplitudes over Four Phases for Subject 3

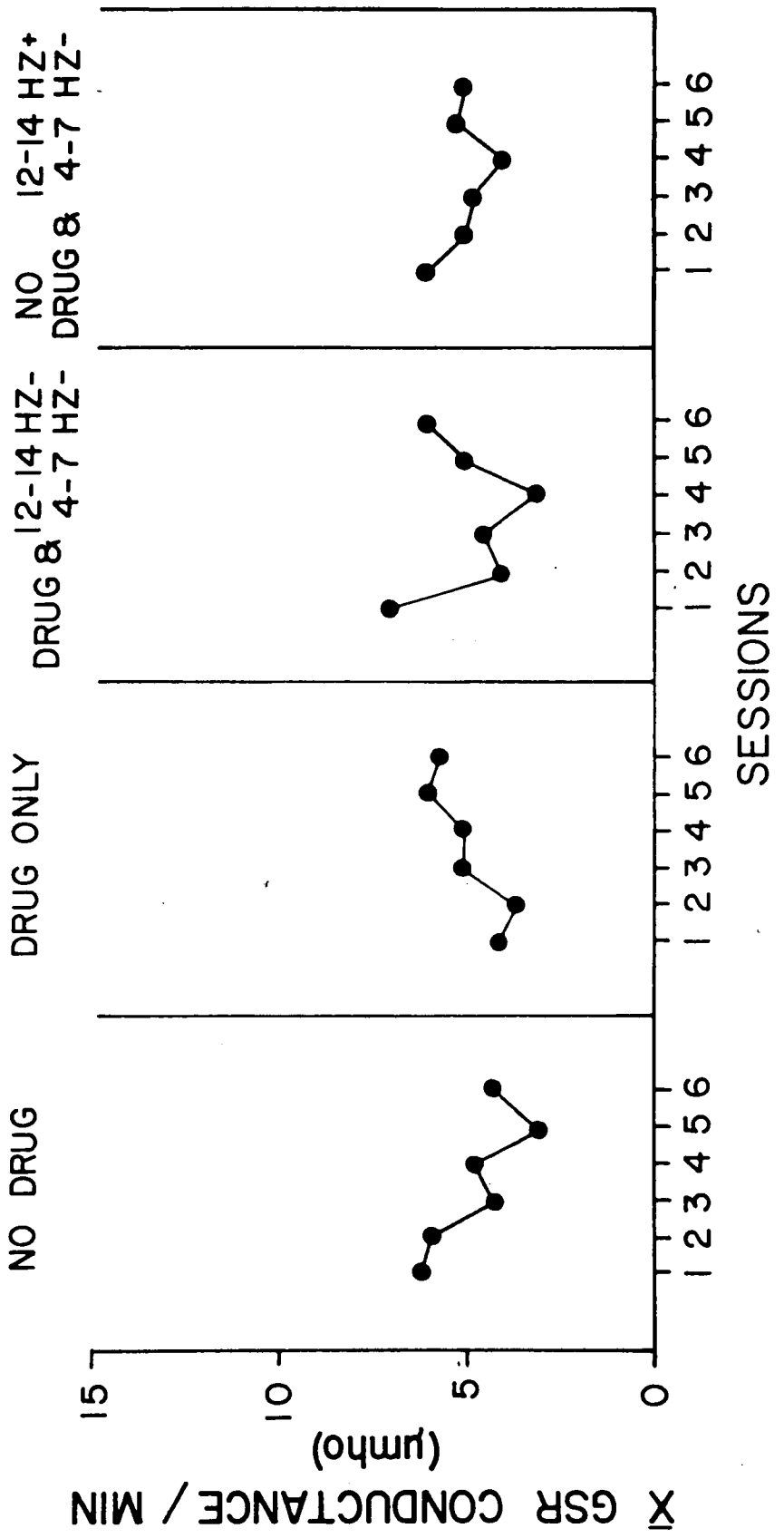


FIGURE 23

Changes in GSR conductance over Four Phases for Subject 3

Classroom Observations

Average interobserver reliabilities per session for subject 3 are 80%, 83% and 82%, respectively, for the 3 conditions. Figure 24 displays changes in the 13 target behaviors and 6 stimulus categories. Pre-treatment and post-treatment profiles in the laboratory conform to the laboratory data. During the NO DRUG condition, subject 3's disruptive behaviors appear to reflect attentional deficits more reliably than overactivity per se. This is suggested by excessive gazing about the classroom (non-interaction), infrequent sustained periods of classwork (sustained school work and attention), and wandering about the classroom (out-of-seat). The lengthy duration of the subject's walks around the classroom may be contrasted to out-of-seat behaviors in other low arousal hyperkinetics who typically left and returned to their seats several times each period. On the other hand, the fidgeting implied in frequent self-stimulation and object manipulations are less characteristic of subject 3 than of the other training subjects.

Relative to the NO DRUG phase, the type changes in this subject following medication resembles those in the other low arousal subjects, and similar trends are also revealed in reinforcers, especially positive social attention from peers. The major difference is that no further change is evidenced following the admission to the training program.

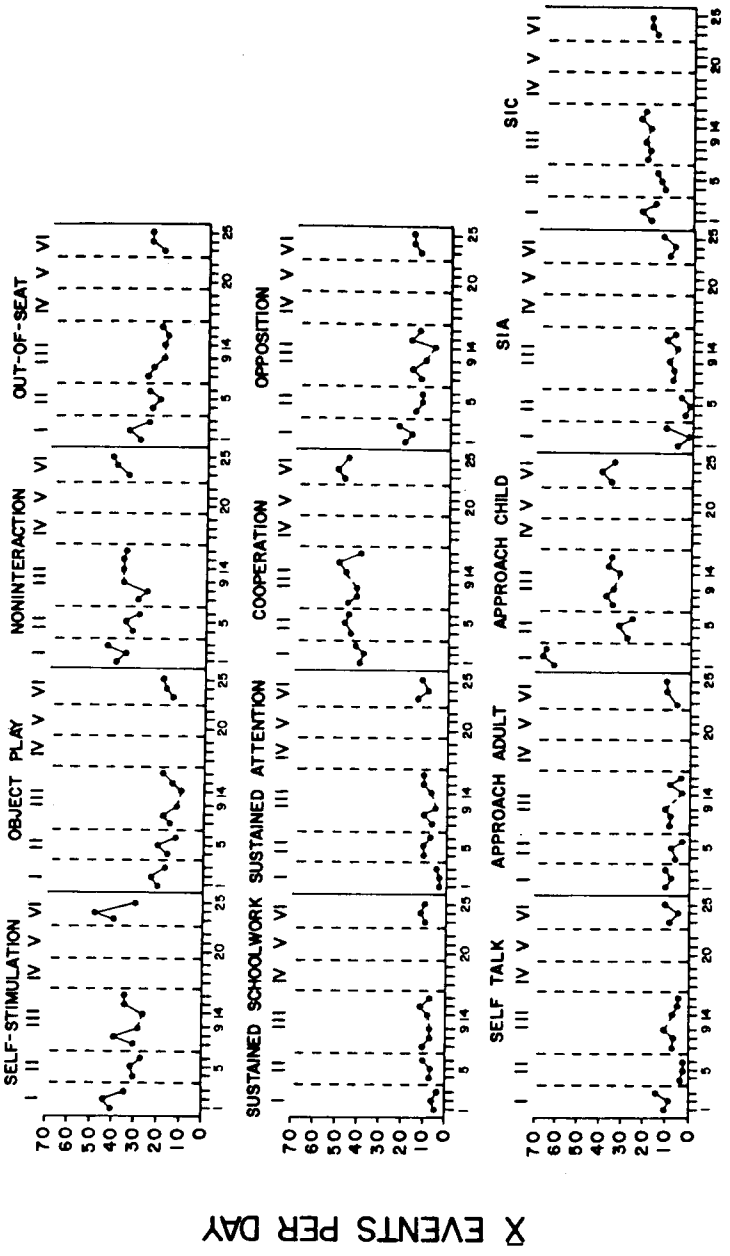


FIGURE 24

Changes in 13 Behaviors Categories over Four Phases for Subject 3

Case 4

Subject 4 (IQ=98) was 10 years, 0 months at the beginning of training in April, 1975. The mother has considered the child overactive, aggressive, and intractible since he began walking at 20 months. His deportment at school, although semi-controlled on a stabilized drug regimen (25 mg/day) since January, 1973, has resulted in 2 suspensions, both of which were followed by expulsion. He has been in a classroom employing a token economy system for all pupils since the beginning of training.

Laboratory Findings

Figure 25 presents SMR and EMG ratios for the 6 conditions. Acquisition of the SMR task is indicated by the anticipated changes in SMR ratios relative to the pre-training phases. The inverse relationship between EMG and SMR is also reconfirmed (I: $r = -.22$, $t = .45$, $df = 4$, $p > .1$; II: $r = -.36$, $t = .77$, $df = 4$, $p > .1$; III: $4 = -.41$, $t = 2.56$, $df = 32$, $p < .05$; IV: $r = -.5$, $t = 1.92$, $df = 11$, $p < .05$; V: $r = -.44$, $t = 1.57$, $df = 10$, $p > .1$; VI: $r = -.49$, $t = 1.99$, $df = 11$, $p < .05$)

Trends in the percent 12-14 Hz activity in Figure 26 reflect the contingent increases and decreases in SMR with biofeedback training. Even though the differences between the final five-minute baseline and feedback periods are not

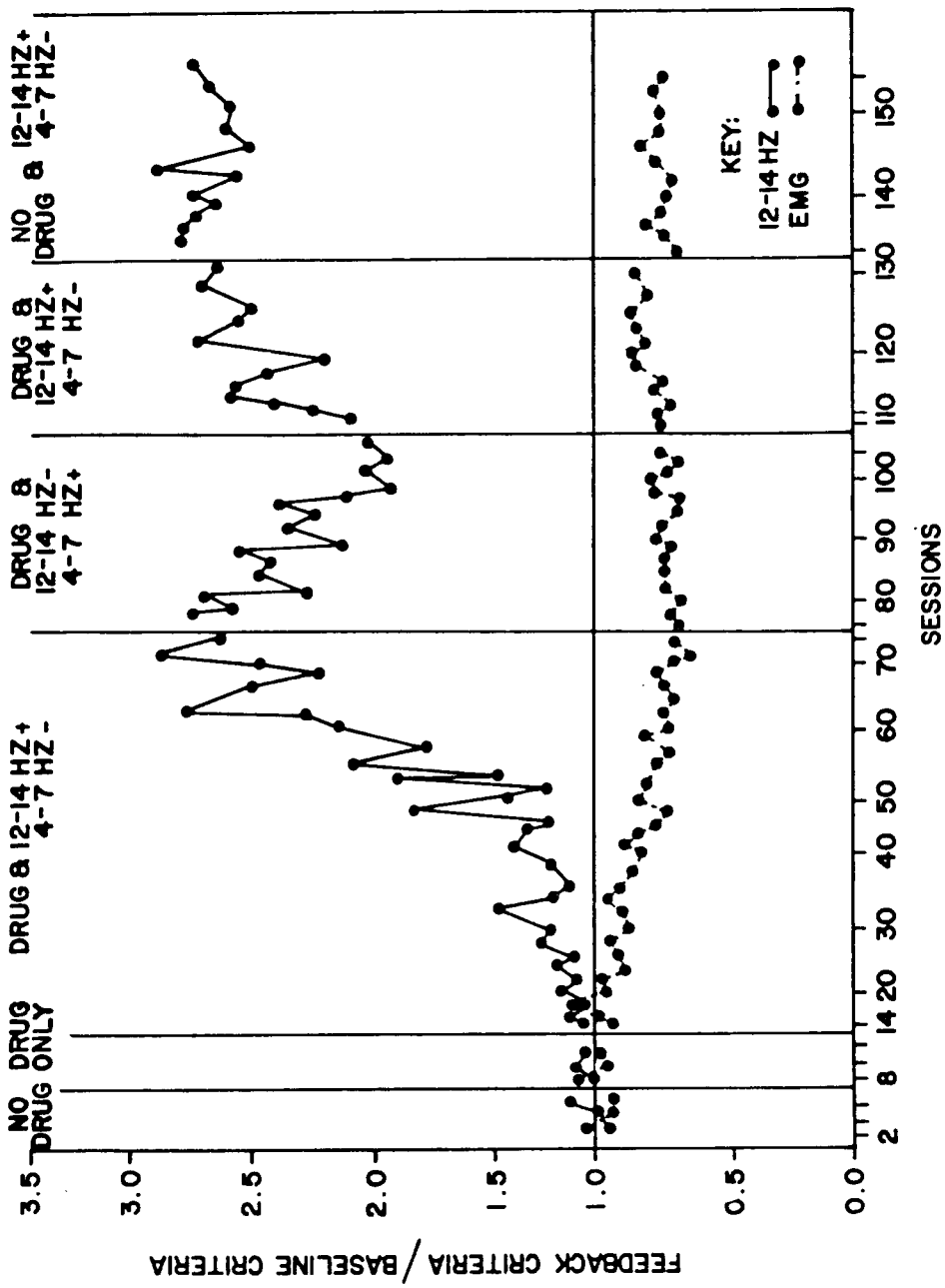


FIGURE 25

SMR and EMG Ratios over Six Phases for Subject 4

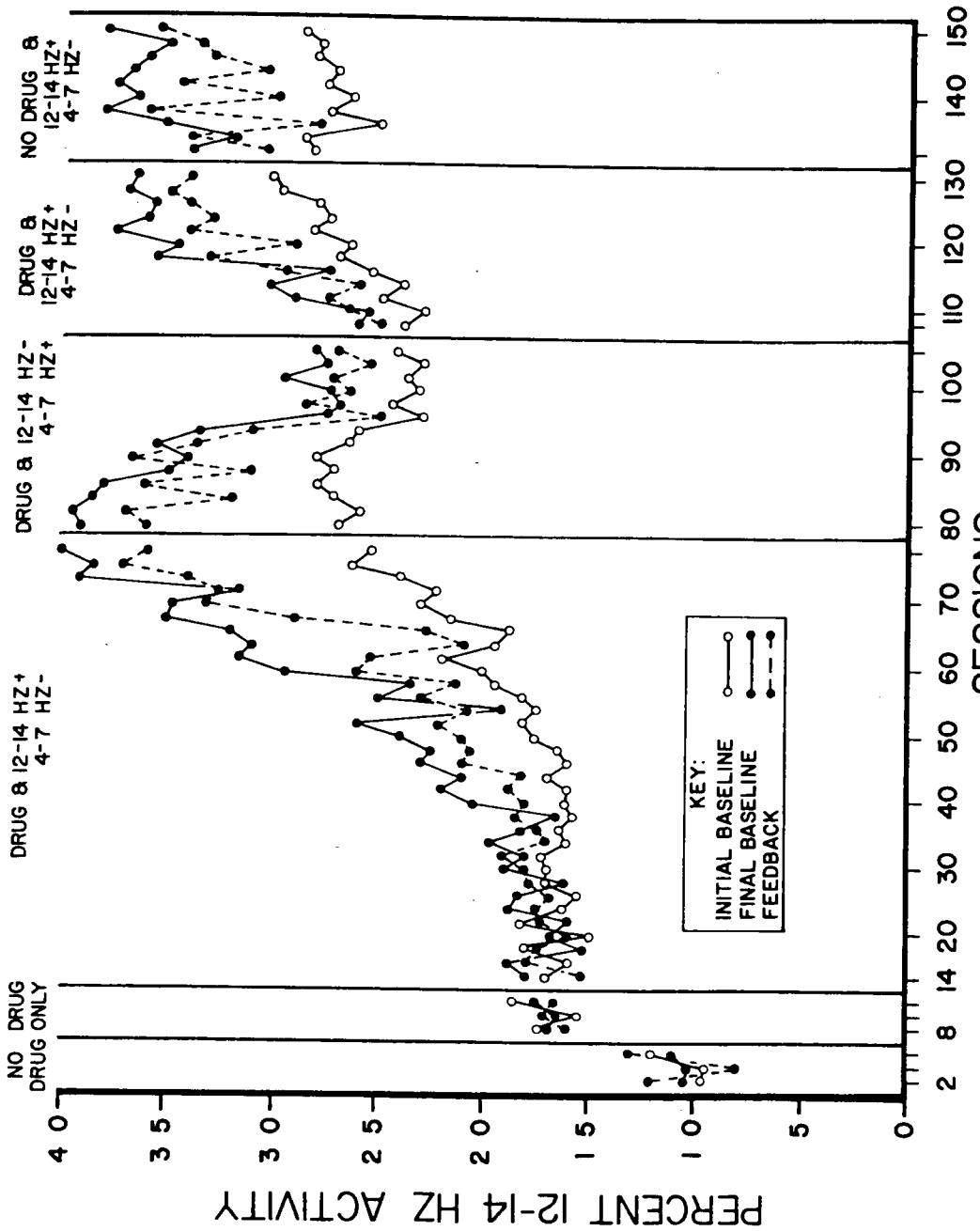


FIGURE 26

Mean Percent 12-14 Hz Activity During Initial Baseline, Feedback, and Final Baseline Periods over Six Phases for Subject 4

statistically significant (III: $t = .68$, $df = 32$, $p > .1$; V: $t = .98$, $df = 11$, $p > .1$; VI: $t = 1.01$, $df = 11$, $p > .1$), a substantial baseline shift is apparent. A somewhat less substantial baseline shift is revealed in the initial five-minute baseline period, and the percent of 12-14 Hz activity produced during this period relative to the feedback period is statistically significant during the three SMR training phases (III: $t = 3.45$, $df = 32$, $p < .05$; V: $t = 2.4$, $df = 11$, $p. < .05$; VI: $t = 2.5$, $df = 11$, $p. < .05$). Corresponding changes during SMR training and counterconditioning occur in EEG spectra, evoked response amplitudes, and GSR conductance and are displayed in Figures 27, 28, and 29 respectively.

This subject's performance is intermediate to the other two successfully trained cases in pre-treatment levels of SMR, amount of acquisition time, and degree to which EEG indices respond to drugs and to biofeedback training. Nevertheless, he ranked third among them in pre-treatment arousal measures other than SMR (see Figure 2, page 58), again suggesting the sensitivity of that measure as a diagnostic and prognistic tool in the disorder.

Classroom Observations

Interobserver reliabilities were somewhat lower for this subject than the others and average 70%, 69%, 78%, 62%, 59%, and 62% respectively, in the 6 conditions.

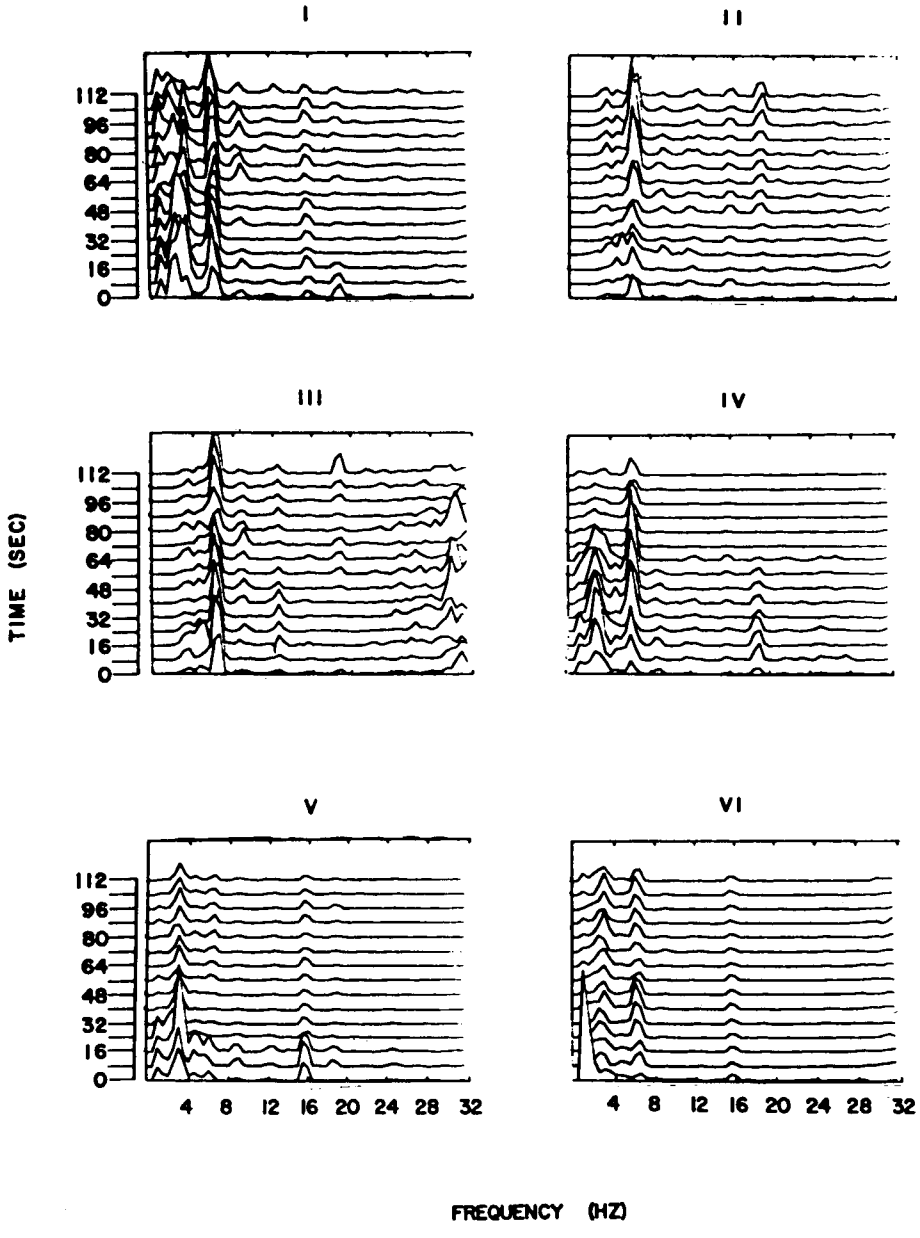


FIGURE 27

Changes in EEG Spectra over Six Phases for Subject 4

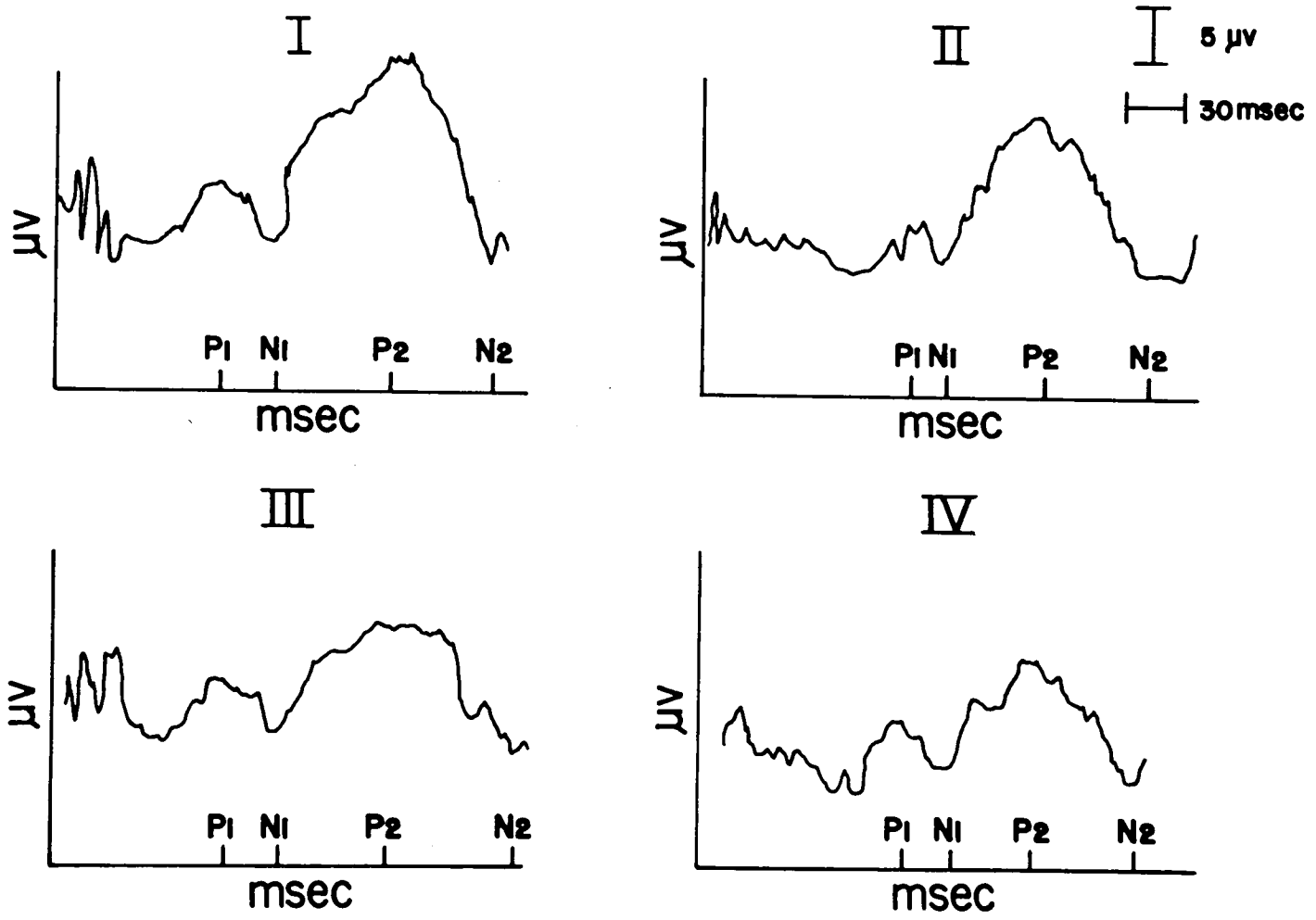


FIGURE 28

Changes in Cortical Evoked Response Amplitudes over Four Phases for Subject 4

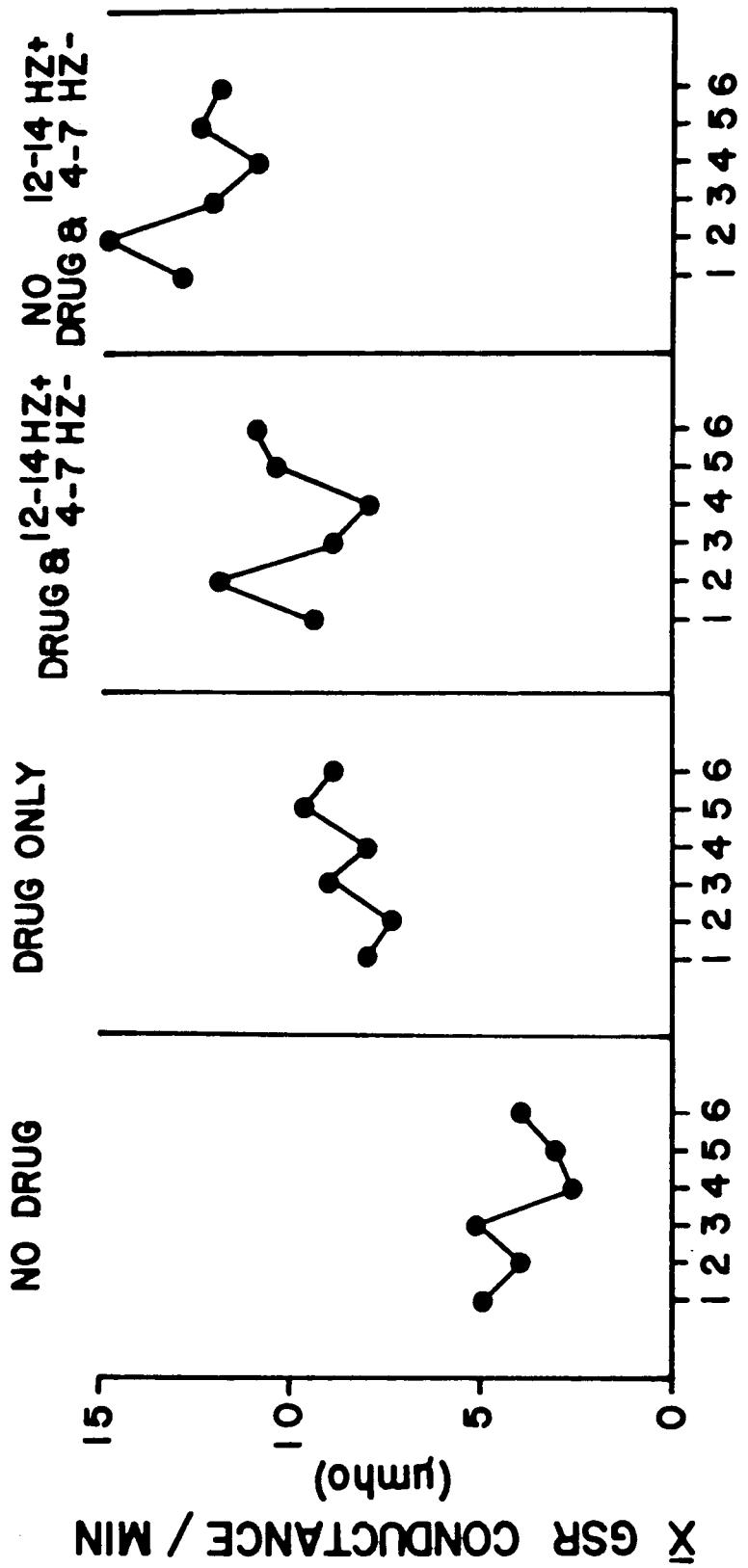


FIGURE 29

Changes in GSR Conductance over Four Phases for Subject 4

Like the other two successfully trained subjects, the same 8 behaviors changed in the predicted directions during SMR conditioning. These data are presented in Figure 30. This subject is distinguished in that, relative to NO DRUG, all these behaviors changed in the predicted direction during both DRUG ONLY and DRUG AND SMR training conditions. Nevertheless, the magnitude of these changes is not striking until the end of the initial DRUG AND SMR training phase. Also, in conjunction with the incomplete reversal in SMR during counterconditioning, these behaviors resumed frequencies more characteristic of early SMR training than of the DRUG ONLY conditions.

Self-talk and the four social behaviors appear in Figure 31. Relative to the NO DRUG conditions, uniform decreases in them are exhibited during the DRUG ONLY condition, and with the exception of self-talk, these levels are sustained during SMR training. Self-talk increases somewhat.

Changes in the stimulus categories for this subject are also reported in Figure 31. Unlike the other three cases, a noticeable increase in positive social attention from teachers and peers during the DRUG ONLY condition. However, further changes in these categories do not accompany the subsequent improvement in undesirable behaviors during SMR training, suggesting that EEG conditioning influences hyperkinetic behavior above and beyond the effects of social reinforcement and medication.

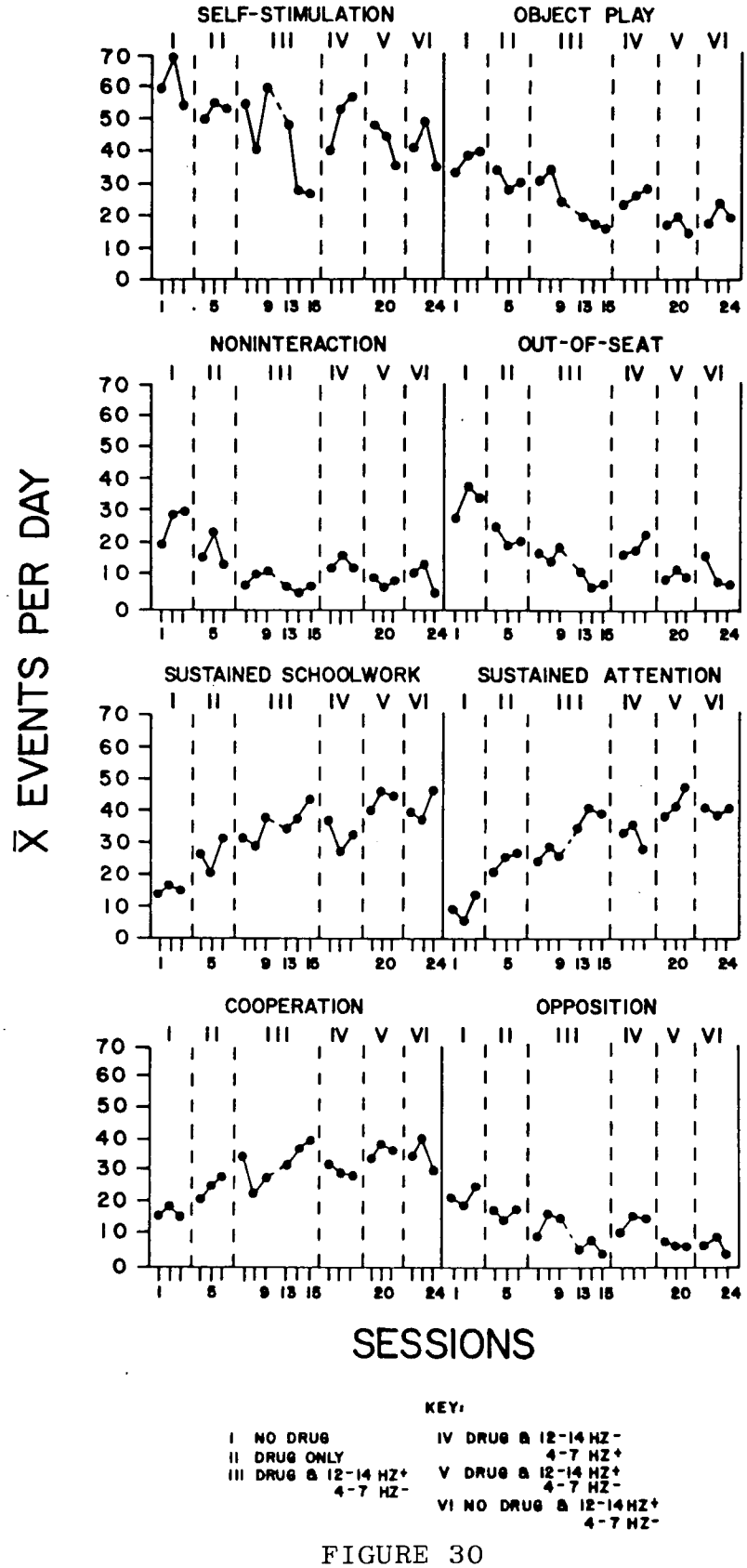


FIGURE 30
Predicted Changes in Eight Behaviors over Six Phases for Subject 4

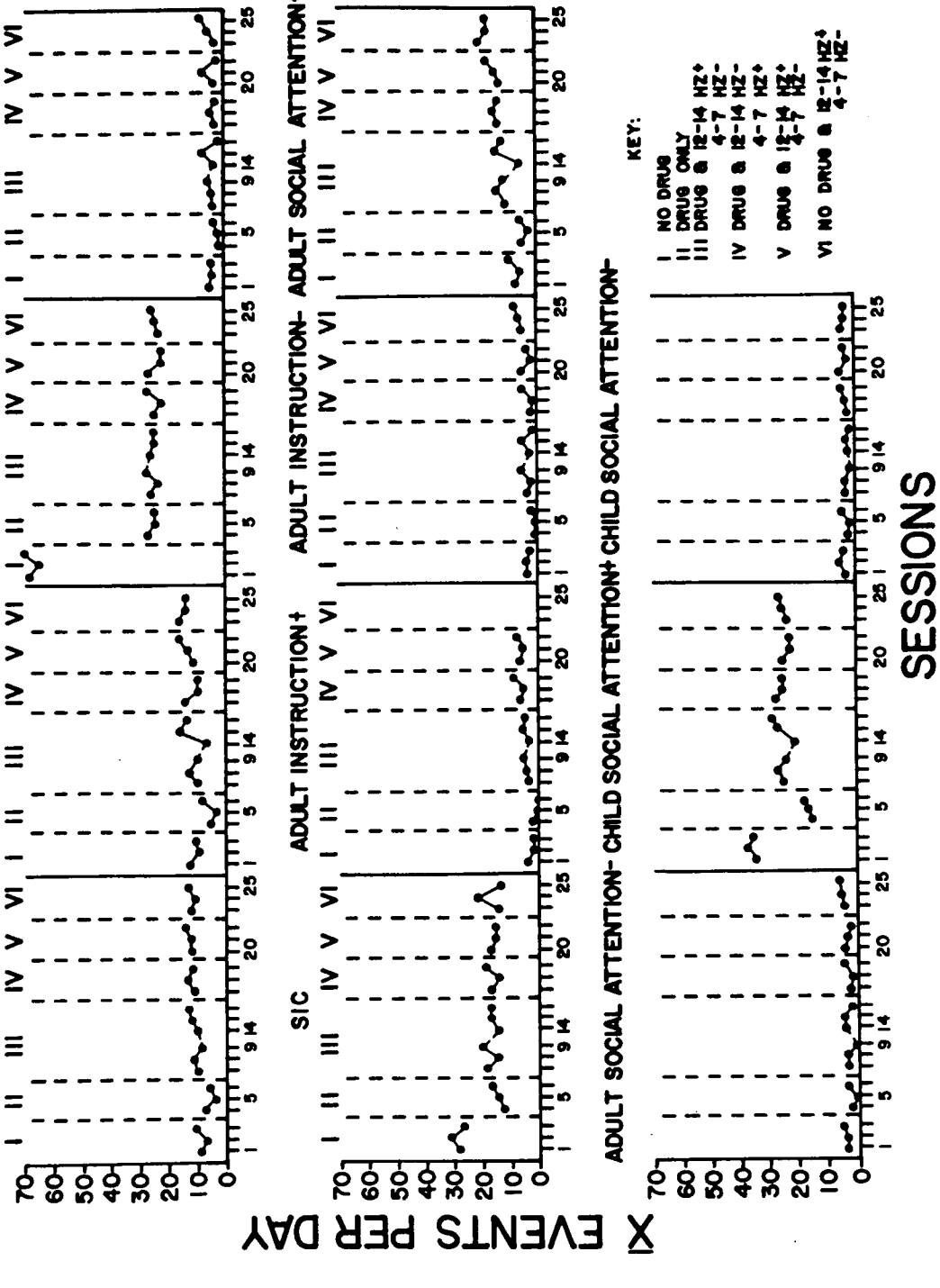


FIGURE 31
Unpredicted Changes in Five Behaviors and Changes in Stimulus Categories over Six Phases for Subject 4

CHAPTER V

PRE-TRAINING VS. POST-TRAINING DATA

An analysis of the training data suggests not only that combining medication and SMR conditioning produces more desirable change in hyperkinetic behavior than that induced by drugs alone, but also that these benefits are sustained following the withdrawal of medication. If these assertions are accurate, physiological and behavior profiles of low arousal subjects at the end of the experiment (Phase VI: NO DRUG AND SMR training) should differ significantly from those displayed during DRUG ONLY. Furthermore, pre- and post-training performance of the arousal subjects should differ significantly from profiles of hyperkinetic control subjects measured during comparable time periods since the latter group did not participate in the training sequence. The fact that control hyperkinetic subjects are assessed under DRUG ONLY and a NO DRUG period at the end of the experiment should not contaminate the outcome of the comparison since Ritalin administration did not significantly affect their pre-treatment symptom profiles. This observation may be reconfirmed by comparing DRUG vs. NO DRUG AND SMR training profiles with similar change scores for normal controls. Finally, if conditioning increases in SMR "normalizes" the performance of low arousal hyperkinetics, as is suggested here, then a

comparison of absolute post-training values in low arousal subjects and normal controls should reflect this resemblance.

Evidence supporting these views is presented in Figures 32 and 33. Figures 32 and 33 present average daily scores for each of the physiological and behavioral indices in the three groups during the NO DRUG, DRUG ONLY, and NO DRUG AND SMR training phases. Relative to the DRUG ONLY phase, the performance of low arousal subjects over days at the end of the training period reflects the anticipated improvements. Decreases in evoked cortical response amplitudes ($t = 2.8$, $df = 5$, $p < .05$) are associated with increased GSR conductance ($t = 2.27$, $df = 5$, $p < .05$), SMR burst production ($t = -2.96$, $df = 5$, $p < .05$), and EMG criterion output ($t = 2.8$, $df = 5$, $p < .05$). All these changes reflect enhanced physiological arousal and are accompanied by substantial improvement in classroom conduct. Relative to DRUG ONLY teachers report significantly fewer symptoms at the end of SMR training ($t = 2.16$, $df = 13$, $p < .05$). At the same time, a decrease in undesirable activity ($t = -4.5$, $df = 14$, p .05) is evidenced in conjunction with increases in desirable and social behavior (<u>desirable behavior</u>: $t = 3.48$, $df = 14$, $p < .05$; <u>social behavior</u>: $t = 2.37$, $df = 14$, $p < .05$).

Table 8 summarizes the average change scores (DRUG ONLY vs. NO DRUG AND SMR) for each group in each index. Statistical comparisons of daily change scores reveal that

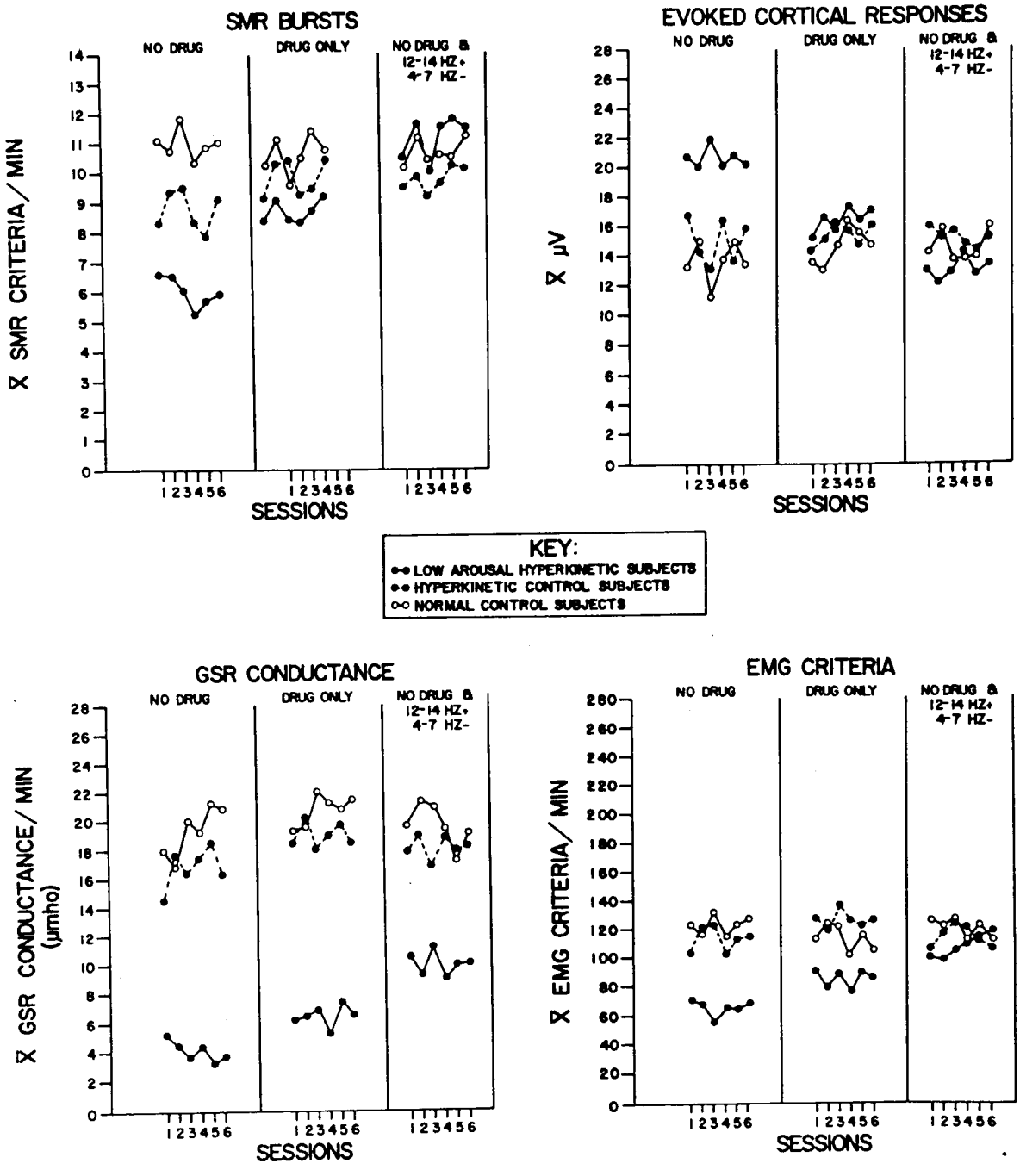


FIGURE 32

Daily Changes in Four Laboratory Measures During Phase I (NO DRUG), Phase II (DRUG ONLY) and Phase VI (NO DRUG AND SMR training) in Low Arousal and Control Hyperkinetic Subjects and Normal Control Children

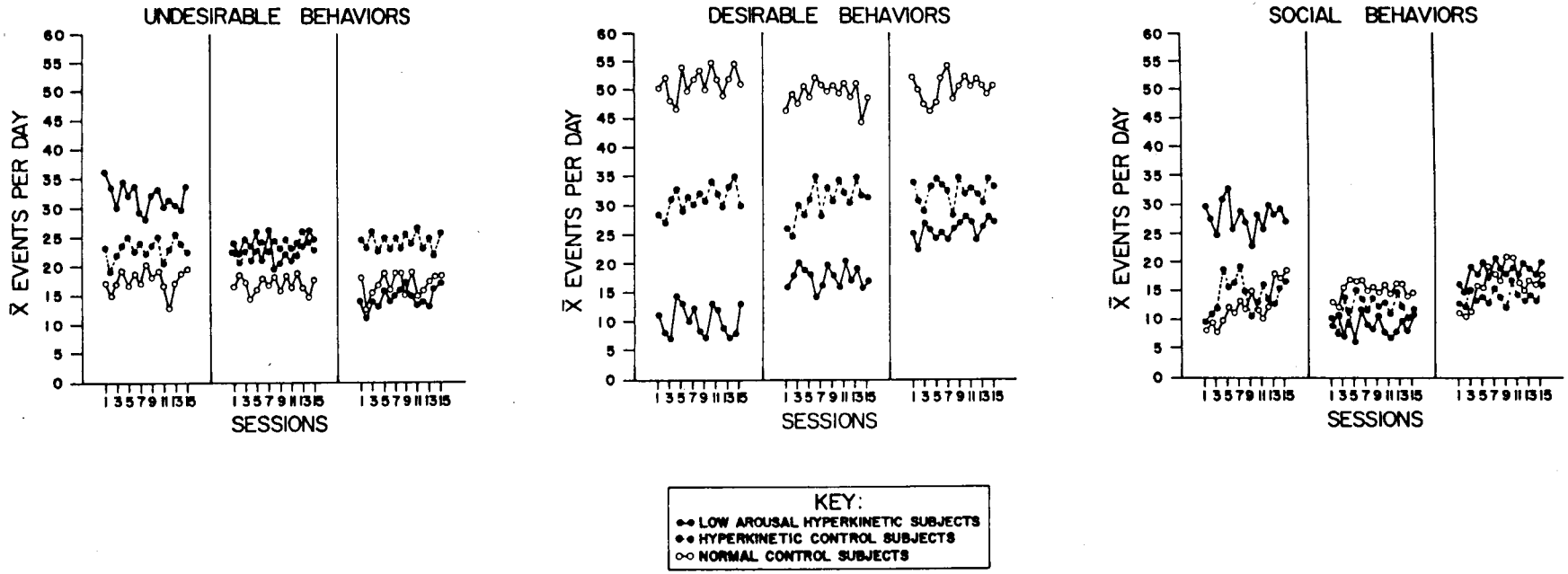


FIGURE 33

Daily Changes in Desirable, Undesirable and Social Behaviors During Phase I (NO DRUG), Phase II (DRUG ONLY), Phase VI (NO DRUG AND SMR TRAINING) in Low Arousal and Control Hyperkinetic Subjects and Normal Control Children

TABLE 8

PHASE II (DRUG ONLY) VS. PHASE VI (NO DRUG AND SMR TRAINING) CHANGES ON LABORATORY, QUESTIONNAIRE, AND BEHAVIORAL INDICES AS A FUNCTION OF SUBJECTS' AROUSAL LEVEL

	LOW AROUSAL HYPERKINETICS (n=3)*	HYPERKINETIC CONTROLS (n=8)	NORMAL CONTROLS (n=12)
LABORATORY DATA			
Mean amplitude evoked cortical response (P2 uV)	*-3.9	**+4.5	***+.27
Mean GSR Conductance (umhos)	+3.6	-.15	-.10
Mea No. SMR Bursts per minute	+2.97	-0.3	-.24
Mean No. EMG Criterions per minute	+14.07	-2.25	+1.8
QUESTIONNAIRE DATA			
Mean No. teacher-reported symptoms	-6.25	+2.0	-.16
BEHAVIORAL DATA			
Mean No. undesirable behaviors	-8.3	+1.3	-.22
Mean No. desirable behaviors	+9.58	+.52	+1.58
Mean No. social behaviors	+4.25	+2.0	+.75

*The low arousal subject (3) who failed to acquire the SMR task is not included in the statistical analyses.

**Low Arousal vs. Hyperkinetic Controls: Evoked Response: t=2, df=5, p .05;

TABLE 8 (continued)

GSR: t=2.27, df=5, p. $\prec$.05; SMR Bursts: t=2.96, df=5, p $\prec$ .05; t=2.8, df=5, p $\prec$.05; teacher-reported symptoms: t=3.C, df=2, p. $\prec$ .05; Undesirable behaviors: t=4.5, df=14, p $\prec$.05; Desirable behaviors: t=3.48, df=14, p $\prec$.05; Social behaviors: t=2.37, df=14, p $\prec$.05

***Hyperkinetic vs. normal controls: Evoked response: t=-.87, df=6, p $\succ$ .1; GSR: t=.96, df=6, p $\succ$ .1; SMR Bursts: t=.86, df=6, p $\succ$ .1; EMG: t=.77, df=6, p $\succ$ .1; Teacher-reported symptoms: t=1.99, df=6, p $\prec$.05; Undesirable behaviors: t=1.10, df=14, p $\succ$ .1; Desirable behaviors: t=1.6, df=14, p $\succ$ .1; Social behaviors: t=1.41, df=14, p $\succ$ .1.

low arousal hyperkinetics are substantially benefitted by SMR training relative to the performance of control hyperkinetics on each index. A comparison of daily change scores for the two control groups, on the other hand, reveals significant differences only on teacher-reported symptoms, reflecting the removal of control hyperkinetic subjects from medication. At the same time, a comparison of the number of symptoms reported by teachers for low arousal subjects and matched normal controls reveals significant differences ($t = 4.7$, $df = 3$, $p < .05$). In conjunction with the overall reduction of teacher-reported symptoms in low arousal subjects during Phase VI relative to DRUG ONLY, these findings suggest that training subjects are more improved than hyperkinetic controls even though teachers can still differentiate training subjects from normal children. Other data indicating incomplete normalization of low arousal subjects include increases in GSR conductance ($t = 6.8$, $df = 5$, $p < .05$) and also in the frequency of desirable behaviors ($t = 2.72$, $df = 14$, $p < .05$). Otherwise, low arousal hyperkinetics and normal controls cannot be differentiated from normal children either physiologically or behaviorally by the end of training (undesirable behaviors: $t = .93$, $df = 14$, $p > .1$; social behaviors: $t = .16$, $df = 14$, $p > .1$ EMG: $t = .11$, $df = 5$, $p > .1$; Evoked cortical response amplitudes: $t = 1.11$, $df = 5$, $p > .1$; SMR bursts: $t = 1.57$, $df = 5$, p .1). Normalization of arousal and behavioral

activity levels is supported by these findings. Also, the fact that undesirable behaviors, which most directly reflect motor activity, changed more substantially than desirable ones, which tend to index attentional deficits, suggests a greater relative role for motor functions in training success.

CHAPTER VI

DISCUSSION AND CONCLUSIONS

A comparison of twelve hyperkinetic and twelve matched normal control subjects revealed statistically significant differences on parent and teacher-reported symptoms of overactivity and distractibility and on associated classroom behaviors. Hyperkinetic subjects also displayed significantly less SMR than did normal children, although no other overall physiological differences between the two groups appeared.

Nevertheless, rank ordering hyperkinetic subjects as a function of CNS arousal level indicated four low arousal subjects differing significantly from remaining hyperkinetic and normal subjects on each physiological measure. Aside from reduced GSR conductance, low arousal characteristics included excessive EEG slow wave activity between 4-7 Hz, reduced EEG activity between 12-14 Hz (SMR), higher amplitude evoked cortical responses, and fewer EMG criterions. It is also significant to note that these group differences were stable ones since each measure was obtained during six sessions conducted over a three-week period.

This low arousal factor also moderated the degree and form of pre-treatment symptom severity reflected both in questionnaire ratings and in classroom behavioral

performance. Relative to hyperkinetic control subjects, low arousal subjects uniformly displayed the most severe pre-treatment symptom profiles, particularly on items reflecting motor disturbances. Thus, the questionnaire data indicated that low arousal hyperkinetic subjects more often revealed maturational deficits in muscular coordination (poor at sports, poor at writing skills, etc.) and were more likely to engage in disruptive motor behaviors (e.g., talkative, overactive, fidgets, etc.) than were hyperkinetic control subjects. On the other hand, low arousal subjects were less likely to exhibit attentional deficits and to be described as nervous, tense, or withdrawn. Similarly, classroom behavioral observations characterized low arousal subjects as actively oppositional, socially aggressive, and as more likely to exhibit repetitive, undirected motor behaviors such as object play and self-stimulation. In contrast, the behavioral profile of hyperkinetic control subjects reflected that these children tended to be more preoccupied (e.g., sustained non-interaction), passively oppositional, and intraverted than low arousal subjects. Finally, relative to pre-treatment (NO DRUG) performance, low arousal hyperkinetic subjects displayed significantly greater improvement following stimulant drug administration than did control hyperkinetic subjects resembling normal children on pre-treatment physiological profiles.

Acquisition of the SMR task in three of four low arousal hyperkinetic subjects was indicated by contingent increases in SMR production during feedback periods relative to daily and pre-training baseline and by contingent decreases in SMR production relative to daily baselines during the contingency reversal phase. Corresponding changes in EMG in the opposite direction tend to support the view that SMR is correlated with motor inhibition. Associated changes in EEG power spectra, GSR conductance, and evoked cortical response amplitudes suggest more complete normalization of CNS arousal profiles than that attained with medication alone. Also, baseline shifts in the percent of 12-14 Hz EEG activity over the training period strengthen the basis for postulating a carry-over effect outside the laboratory setting.

Objective measures of hyperactivity and short attention span in the classroom further corroborate this view. Eight of the total 13 behaviors changed in the predicted direction in 3 of 4 subjects under the 2 treatment conditions. Generally, decreases in undirected activities, out-of-seat, and oppositional behaviors were accompanied by increased cooperation and school work. A marked decline in social behaviors, especially unsustained social approaches to peers, was also noteworthy. 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, 1973).

The effectiveness of the biofeedback technique is supported by the fact that the combined effects of drug administration and SMR training resulted in substantial improvement above and beyond the effects of drugs alone. Further support derives from the maintenance of positive treatment effects with SMR training after medication was withdrawn. In fact, with the exception of desirable behaviors (sustained school work and attention), GSR conductance, and teacher-reported symptoms, training subjects could not be differentiated physiologically or behaviorally from normal control subjects by the end of training period. Also, the loss of improvement following SMR counter conditioning tends to minimize the roles of extraneous influences (e.g. maturation) on treatment outcomes. Finally, the fact that the subject who failed to acquire the SMR task also failed to develop associated physiological and behavioral changes lends additional credence to these assertions.

The similarity between SMR-trained subjects in laboratory and classroom performance is encouraging. However, subject 1 acquired the SMR task more rapidly and extensively and responded more dramatically to both treatment conditions than did subjects 2 and 4. The fact that subject 1 also displayed more severe pre-treatment (NO DRUG) physiological and behavioral profiles suggests

that the severity of original deficits may facilitate treatment outcomes and may restrict the generality of the procedure's application.

It is also noteworthy that subject 4, whose response to medication alone and to SMR training ranked second to subject 1's, also displayed the second lowest frequency of SMR in the NO DRUG condition and the second highest level of pre-treatment symptom severity on behavioral and questionnaire assessments. In contrast, this subject displayed somewhat higher CNS arousal level on measures of GSR conductance, evoked cortical response amplitude and peak frequency component in EEG spectra than did either subject 1 or 2. Pre-treatment levels of SMR, then, most reliably predicted the severity of original motor deficits as well as the success of both treatments in mitigating these symptoms. These findings not only confirm the relationship between SMR and behavioral immobility but also emphasize the EEG rhythm's diagnostic and prognostic value.

All these findings clearly implicate both CNS arousal level and central motor system functions in the disorder's treatment and possibly its development. However, since SMR acquisition, normalization of CNS arousal indices, and behavioral improvement appear to have emerged concurrently, it is difficult to determine whether the observed behavioral outcome reflects primary

changes in CNS arousal or whether the arousal changes represent a secondary effect from enhanced motor control.

In addition to changes in GSR and evoked cortical responses, the fact that overall baseline levels of EMG increased with medication and SMR training further corroborates a generalized, treatment-related increase in physiological arousal level. This change appears to contradict EMG's usual negative correlation with SMR production, although the two do vary inversely within daily training sessions. Finally, this upward trend in EMG is consistent with the view that behavioral improvements associated with stimulant drug medication and SMR training result from increasing general physiological arousal level rather than that they reflect a secondary outcome from enhanced motor control.

At the same time, a number of factors reported here and in related research areas suggest the selective importance of central motor functions. In the study reported here, a primary motor symptomatology, or a symptom profile dominated by motor deficits, characterizes not only hyperkinetic children classified as low arousal subjects but also those low arousal subjects who were effectively trained with the SMR biofeedback procedure. The selective dominance of motor disturbances is illustrated in the developmental backgrounds and in immediate pre-treatment symptom profiles of these subjects. Greater

over-all changes in motor symptoms also occur following both treatments. This trend is indicated by the normalization of undesirable behaviors, which predominantly reflect changes in overactivity, and may be contrasted with the incomplete normalization of desirable behaviors, which predominately reflect changes in attention span. These data are consistent with the fact that pre-treatment differences in SMR production between low arousal subjects more accurately conformed to pre-treatment levels of overactivity, with which SMR is typically associated. Also, the fact that pre-treatment levels of SMR more accurately predicted response to both treatments than did arousal profiles suggests that SMR production may represent a more critical index of the disorder's severity, at least when overactivity is a central feature.

Related research findings also indicate an association between primary motor symptom symptomatology and pre-treatment SMR levels in epilepsy and insomnia (Sterman, 1976; Feinstein et al., 1976); these studies further suggest that the degree to which deficits and motor inhibition are present governs the susceptibility of individuals with these disorders to SMR training. For several reasons, the mechanism involved in the control of this rhythm and in the SMR biofeedback procedure's successful clinical application appears to be one of intrinsic motor control governed at the subcortical

level, either directly or indirectly through proprioceptive influences.

First, considerable evidence indicates that the cortical EEG rhythm and its peripheral motor correlates per se are not causally related. Thus, immobility is a necessary but insufficient condition for SMR production, as indicated by the fact that motionlessness can occur in the absence of SMR (Sterman and Wywricka, 1967). On the other hand, total ablation of Rolandic cortex does not disrupt gamma motor system inhibition following thalamic stimulation sufficient to precipitate 12-14 Hz spindles pre-operatively (Hongo et al., 1973). At the same time, it is clear that the mechanism is a central one since the administration of neuromuscular blocking agents will delay onset of pharmacologically induced seizures (Sterman et al., 1972) while corticospinal tract section abolishes only the peripheral motor manifestations (Witkin and Weatherby, 1955).

Alternatively, there is evidence of discrete subcortical pathways significantly moderating the occurrence of SMR production in conjunction with somatomotor inhibition. Unit recordings in ventroposterolateral (VPL) nucleus of the thalamus reflect the presence and absence of 12-14 Hz activity in conjunction with the production and inhibition of the cortical rhythm (Howe and Sterman, 1972). Also, surface EEG recordings reveal post-central or somatosensory cortex near the postcruciate

dimple as the optimal location for high voltage SMR production in cats. This post-central site receives primary projections from and reciprocally innervates VPL. It would appear, then, that a distinct thalamo-cortico-thalamic pathway is involved in the generation of this EEG rhythm. Furthermore, despite the rather exclusive display of waking SMR within the somatosensory system, the rhythm's appearance in both cortical and subcortical sites is invariably disrupted following any sign of phasic motor activity; on the other hand, the fact that SMR in both sites is never interrupted by sensory stimulation in the absence of movement supports the relative importance of reduced proprioceptive influences in the rhythm's generation (Howe and Sterman, 1972).

Finally, a significant interaction between general arousal level, SMR, and central motor functions underlying the genesis of generalized convulsions associated with epileptic seizures was established relatively early (Sterman et al., 1967). More recent evidence indicates systematic changes in physiological and behavioral arousal level following SMR training in normal subjects (Chase and Harper, 1971; Sterman, 1976) as well as in subjects suffering from waking and sleeping epileptic seizures (Sterman, 1976; Sterman et al., 1972) and from motor restlessness during both states (Sterman et al., 1970; Sterman et al., 1974; Sterman, 1976; Feinstein et al., 1976).Nevertheless, the most pronounced changes always

occurred in physiological and behavioral indices of motor activity.

The findings reported here and in related research investigation suggest, then, that SMR conditioning enhances internal motor control at the CNS level, that an absence of this control may underly the SMR biofeedback procedure's successful clinical application to at lease three otherwise distinct syndromes (epilepsy, insomnia, and hyperkinesis), and that substantial changes in motor activity during training are accompanied by systematic, though less striking, changes in physiological arousal measures that do not directly index motor activity. These findings are consistent with the view that the generalized changes in physiological arousal reported for hyperkinetic subjects in this study are incidental to the enhanced motor control associated with SMR training. Nevertheless, a much larger population of hyperkinetic subjects is required before any substantive conclusion is possible about the technique's therapeutic utility in this disorder or its basis in central mechanisms of arousal or motor control.

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APPENDICES

APPENDIX A

TABLE A-1

DESCRIPTION OF BEHAVIOR AND STIMULUS CATEGORIES EVALUATED IN THE CLASSROOM

CATEGORIES AND NOTATIONS	DESCRIPTION
WAHLER'S BEHAVIORAL CATEGORIES:	
UNDIRECTED ACTIVITIES	
Self Stimulation(S)	Scored for any instance of the subject's manipulation of his own body or for any instance of non-purposive non-directed motor or vocal actions.
Object Play (OP)	Scored for any instance of simple repetitive manipulation of an object, in which the manipulation 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 non-compliance to rules or instructions imposed by adults.
SCHOOL WORK	
Sustained Schoolwork (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.

TABLE A-1 (continued)

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 non-verbal 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)	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 subscript identifies the other person as an adult (SIa) or a child (SIc).

TABLE A-1 (continued)

CATEGORIES AND NOTATIONS	DESCRIPTION
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.
WAHLER'S STIMULUS CATEGORIES:	
INstruction, Non-aversive (1A+)	Scored for direct commands and indirect commands sometime expressed as questions and directed to the subject by an adult (teacher). The instructions must designate an obvious referent or class of referents, and must specify an act of compliance that can be initiated within the interval (10 seconds) in which it was given or in the interval immediately following it; otherwise, it is scored as social attention. When multiple instructions occur in an interval, only one instance is scored, and only the last is considered with respect to the scoring of compliance or opposition.
Instruction, Aversive (IA-)	Scored in the same manner as instruction, non-aversive, but is also judged as aversive because of the content of the instruction, the voice quality of the speaker, and/or the assertive behavior of the speaker. An instruction with aversive content contains ridicule, a threat of punishment, or unpleasant consequences to the child. An instruction with aversive voice quality is spoken loudly or shouted, or is spoken in a "threatening" tone of

TABLE A-1 (continued)

CATEGORIES AND NOTATIONS	DESCRIPTION
Instruction, Aversive (IA-)	voice, which may be high-pitched or low and measure as if the speaker is exercising "control." An instruction with aversive assertive behavior is accompanied by grabbing the child pushing the child aside, striking him, grabbing the object concerned, etc. Non-aversive and aversive instructions can be scored in the same ten-second interval.
Social Attention, Non-Aversive (Sa+) (Sc+)	Scored for any deliberate physical contact made by anyone with the subject, verbalizations directed to the subject, and a kind of looking at each other that entails mutual recognition demonstrated through other non-verbal cues. A sub-script identifies the dispenser of the social attention as an adult (Sa+) or child (Sc+).
Social Attention, Aversive (Sa-) (Sc-)	Scored in the same manner as social attention, non-aversive, but is also judged to be aversive because of the verbal content, voice quality, or the asserted behavior of the dispenser of aversive social attention. "Assertive" denotes the same acts discussed under instruction, aversive.

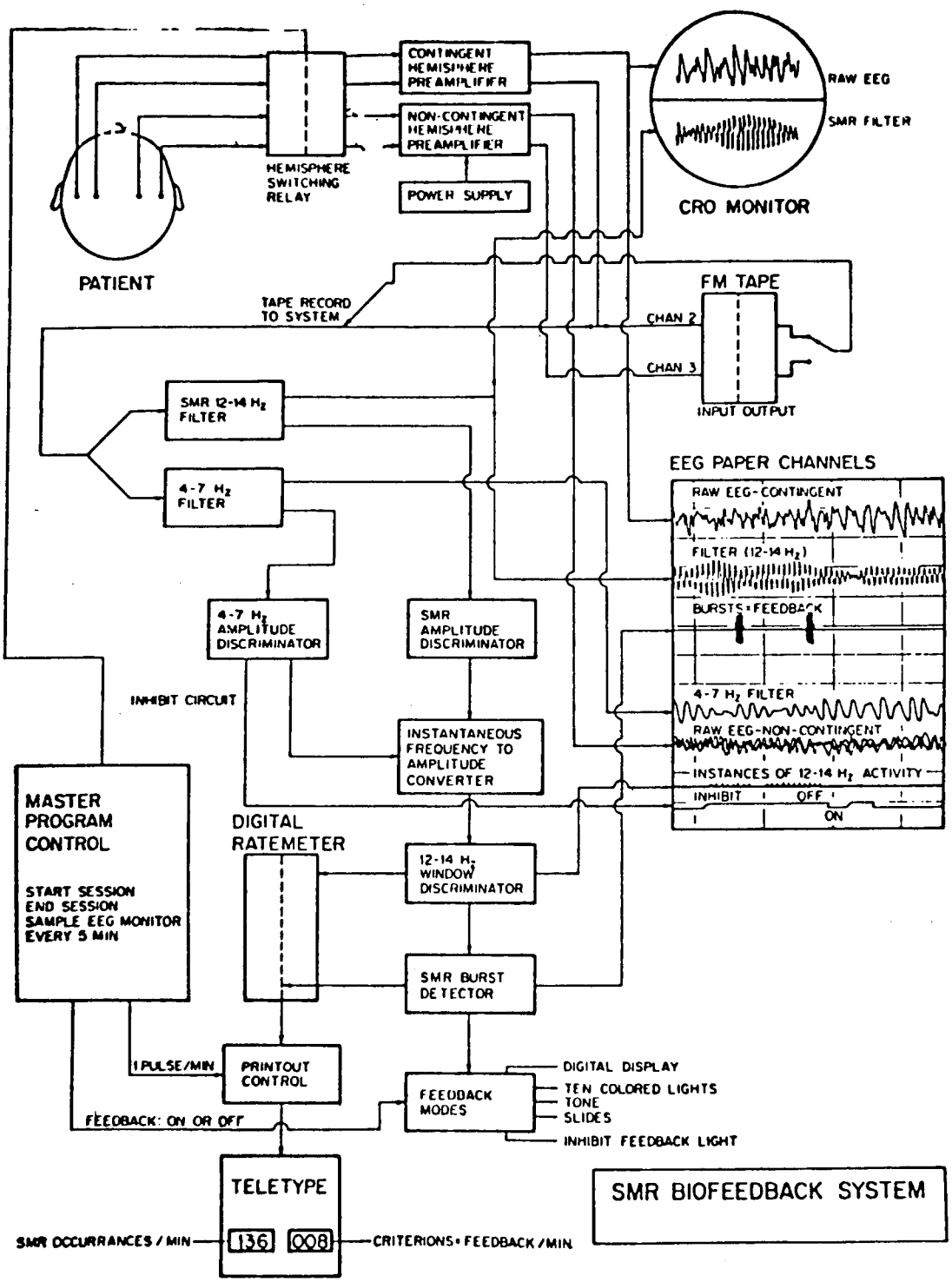


FIGURE A-1

Schematic Drawing of the SMR Biofeedback System

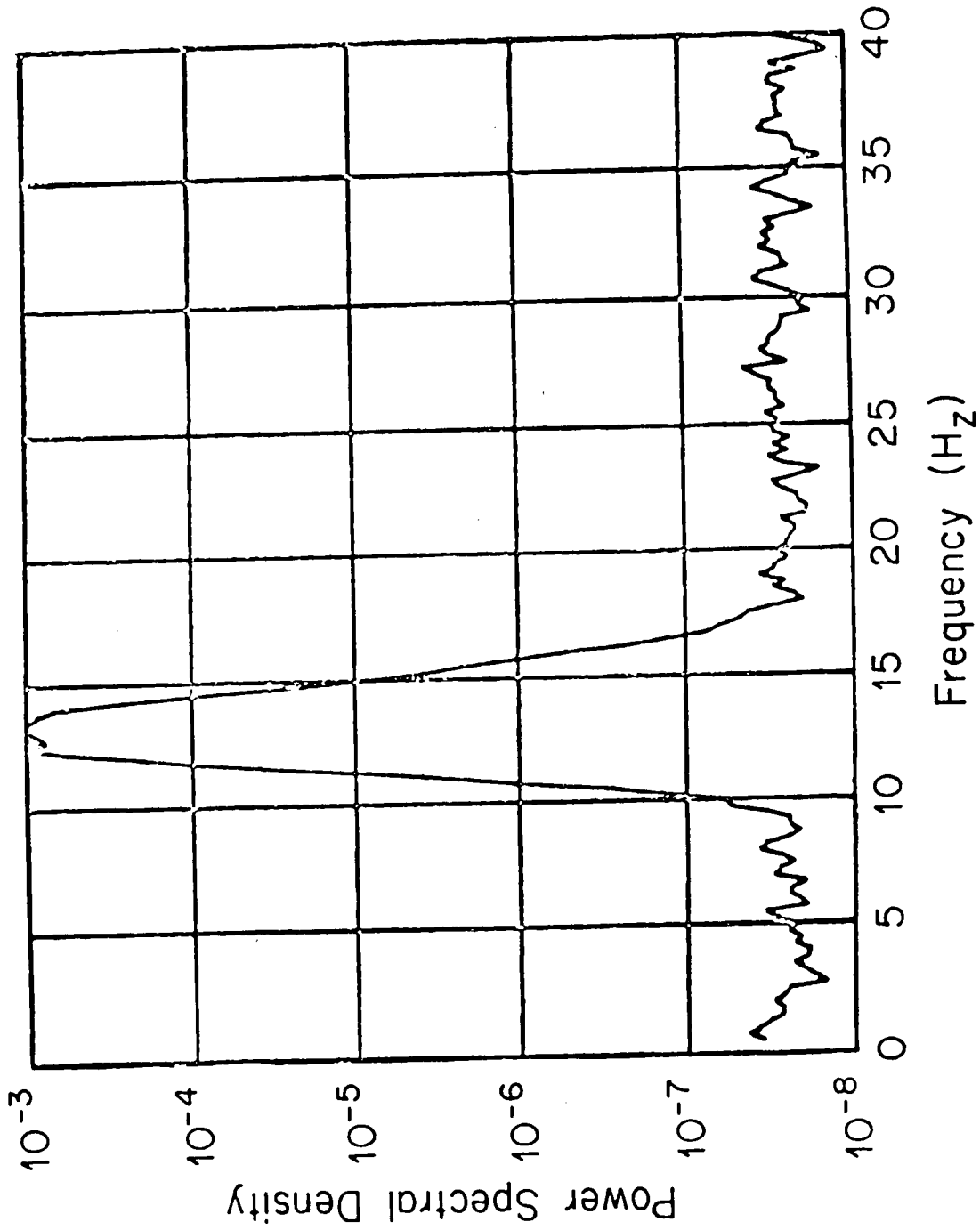


FIGURE A-2

12-14 Hz Filter Response

APPENDIX B

TABLE B-1

PHASE I (NO DRUG) COMPARISON OF PARENT AND TEACHER-REPORTED DEVELOPMENTAL PROBLEMS AND SYMPTOMS: HYPERKINETIC SUBJECTS VS. NORMAL CONTROL SUBJECTS

VARIABLES	HYPERKINETIC SUBJECTS (n=12)		NORMAL SUBJECTS (n=12)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
PARENT-REPORTED DEVELOPMENTAL PROBLEMS: NO DRUG					
NOW TAKING DRUG REGULARLY	12	100	0	0	100
EVER REFERRED TO PHYCHOLOGIST	12	100	1	8	92
SPEECH DIFFICULTIES	8	67	0	0	67
READING DIFFICULTIES	11	92	3	25	67
CNS: EYESIGHT (VISUOMOTOR)	8	67	2	17	50
PRE-NATAL PROBLEMS	6	50	1	8	42
WRITING DIFFICULTY	6	50	1	8	2.0
3 VISITS TO MD IN PAST YEAR	5	42	0	0	42.0
POOR HEALTH IN 1st YEAR	5	42	1	8	34
DIFICIENT LARGE MUSCLE COORDINATION	6	50	2	17	33
TROUBLED DELIVERY	5	42	2	17	25
SLEEPWALKING; NIGHT TERRORS	5	42	2	17	25
ER VISITS (Other Than Head Injury; Fractures; Poisoning)	6	50	3	25	25
TROUBLED PREGNANCY	2	17	0	0	17
ER VISITS (Head Injury)	3	25	1	8	17
3 HOSPITALIZATIONS IN PAST YEAR	2	17	0	0	17
ER VISITS (Fractures)	4	33	2	17	16
CNS: DELERIA	1	8	0	0	8
EPISODIC ILLNESS	1	8	0	0	8
TEACHER-REPORTED DEVELOPMENTAL PROBLEMS: NO DRUG					
REFERRED TO SCHOOL PHYCHOLOGIST	12	100	1	8	92

TABLE B-1 (continued)

VARIABLES	HYPERKINETIC SUBJECTS (n=12)		NORMAL SUBJECTS (n=12)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
SENT TO PRINCIPLE ONCE A MONTH	5	42	0	0	42
REFERRED FOR SPEECH PROBLEM	3	25	0	0	25
REFERRED FOR READING PROBLEMS	3	25	0	0	25
EVER HAD SUBJECT MATTER TUTORING	4	33	1	8	25
SUSPENDED OR EXPELLED	1	8	0	0	8
EVER IN LD CLASS	1	8	0	0	8
TEACHER-REPORTED SYMPTOMS: NO DRUG					
RESTLESS	12	100	1	8	92
FIDGETS	11	92	1	8	83
CAN'T LEAVE OTHERS ALONE	11	92	1	8	83
SHORT ATTENTION SPAN	10	83	0	0	83
EASILY DISTRACTED	11	92	3	25	75
IMPULSIVE	8	67	1	8	66
DEMANDS MUCH ATTENTION	8	67	1	8	66
TALKATIVE	11	92	4	33	58
SHOWS OFF	10	83	4	33	50
DISRUPTS CLASS	6	50	0	0	50
DOESN'T ATTEND TO DIRECTIONS	7	58	1	8	50
HURRIES	4	33	0	0	33
UNCOORDINATED	5	42	1	8	33
FEELS A FAILURE IN SCHOOL	4	33	40	0	33
GENERALLY UNCOOPERATIVE	4	33	0	0	33
DOESN'T FINISH WORK	4	33	1	8	25
DOESN'T FINISH PROJECTS	3	25	0	0	25

TABLE B-1 (continued)

VARIABLES	HYPERKINETIC SUBJECTS (n=12)		NORMAL SUBJECTS (n=12)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
EASILY FRUSTRATED	3	25	0	0	25
IS UNPOPULAR	5	25	0	0	25
UNDISCIPLINED	3	25	0	0	25
FIGHTS A LOT	4	33	1	8	25
DOESN'T DO HOMEWORK	2	17	0	0	16
EASILY AND OFTEN UPSET	2	17	0	0	16
IRRITABLE	2	17	0	0	16
FEELS UNPOPULAR	4	33	1	8	16
LIES	2	17	0	0	16
SLOW AND UNCLEAR SPEECH	2	17	0	0	16
DIFFICULTY IN ADJUSTING	2	17	0	0	16
MANY ACCIDENTS	1	8	0	0	8
STEALS FROM PEERS	1	8	0	0	8
DISINTERESTED IN SCHOOL	2	17	1	8	8
SASSY TO ADULTS	0	0	1	8	-8
SILLY	0	0	1	8	-16
DAYDREAMS	1	8	4	33	-25
PARENT-REPORTED SYMPTOMS: NO DRUG					
RESTLESS AND OVERACTIVE	12	100	0	0	100
SHORT ATTENTION SPAN	12	100	0	0	100
DISOBEDIENT	11	92	0	0	92
LIES OFTEN	11	92	0	0	92
DOESN'T FINISH PROJECTS	12	100	2	17	84
HARD TO RAISE	9	75	0	0	84

TABLE B-1 (continued)

VARIABLES	HYPERKINETIC SUBJECTS (n=12)		NORMAL SUBJECTS (n=12)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
IMPATIENT AND DOESN'T LISTEN	12	100	3	15	75
NAUSEOUS: NO MORNING APPETITE	10	83	1	8	75
DISTURBED SLEEP CYCLE	8	67	0	0	66
IMPULSIVE	8	67	1	8	58
IRRITABLE: TEMPER TANTRUMS	7	58	0	0	58
EXCITABLE: SILLY WITH PEERS	9	75	3	25	50
EASILY UPSET	8	67	2	17	50
FIGHTS A LOT	8	67	2	17	50
DESTRUCTIVE	7	58	1	8	50
MANY HEADACHES	6	50	1	8	41.6
LONELY UNPOPULAR	6	50	1	8	41.6
LOST INTEREST IN FRIENDS	6	50	1	8	41.6
RECKLESS	6	50	2	7	33
PLAYS WITH OR SETS FIRES	5	42	1	8	33
OVERDEPENDENT ON MOTOR	5	42	2	17	25
LOW SELF-ESTEEM: EXPECTS TO FAIL	5	42	2	17	25
EPISODIC DEPRESSIONS	5	42	2	17	25
UNUSUAL WORRIES	4	33	1	8	25
STEALS FROM FAMILY	4	33	1	8	25
ABLE TO GO WITHOUT SLEEP	3	25	0	0	25
PERSECUTION COMPLEX	3	25	0	0	25
NERVOUS, TENSE, SHAKEY	1	42	3	25	16
STEALS FROM OTHERS	3	25	1	8	16
TRIED UNPRESCRIBED DRUGS	3	25	1	8	16
SECUALLY PRECOCIOUS	2	17	1	8	8
UNUSUAL FEARS	2	17	1	8	8
EPISODIC MANIA	2	17	1	8	8

TABLE B-1 (continued)

VARIABLES	HYPERKINETIC SUBJECTS (n=12)		NORMAL SUBJECTS (n=12)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
PARENT-REPORTED SYMPTOMS: NO DRUG					
SAYS LIFE NOT WORTH LIVING	1	8	0	0	8
HYPOCHONDRIACAL	3	25	3	25	0
AFRAID TO FIGHT	3	25	3	25	0
SKIPS CLASS, PLAYS HOOKEY	1	8	1	8	0

TABLE B-2

PHASE I (NO DRUG) COMPARISON OF PARENT AND TEACHER-REPORTED DEVELOPMENTAL PROBLEMS AND SYMPTOMS: LOW AROUSAL VS. CONTROL HYPERKINETIC SUBJECTS

VARIABLES	LOW AROUSAL HYPERKINETIC SUBJECTS (n=4)		CONTROL HYPERKINETIC SUBJECTS (n=8)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
PARENT-REPORTED DEVELOPMENTAL PROBLEMS: NO DRUG					
MORE LIKELY IN LOW AROUSAL SUBJECTS					
DEFICIENT LARGE MUSCLE COORDINATION	3	75	3	37.5	37.5
DISTURBED SLEEP CYCLE	3	75	3	37.5	37.5
ER VISITS FOR MISC. REASONS	3	75	4	50	25
SPEECH PROBLEMS	3	75	4	50	25
POOR HEALTH IN 1st YEAR	2	50	3	37.5	12.5
READING PROBLEMS	4	100	7	87.5	12.5
ER VISIT FOR FRACTURES	3	75	5	62.5	12.5
LESS LIKELY IN LOW AROUSAL SUBJECTS					
BEDWETTING	1	25	6	75	-50.0
TROUBLED PREBNANCY	0	0	3	37.5	-37.5
TROUBLED DELIVERY	1	25	4	50	-25.0
PERINATAL PROBLEMS	2	50	6	75	-25.0
ER VISITS FOR HEAD INJURY	1	25	4	50	-25.0
CNS: EYE (VISUOMOTOR)	2	50	6	75	-25.0
TEACHER-REPORTED DEVELOPMENTAL PROBLEMS: NO DRUG					
MORE LIKELY IN LOW AROUSALS					

TABLE B-2 (continued)

VARIABLES	LOW AROUSAL HYPERKINETIC SUBJECTS (n=4)		CONTROL HYPERKINETIC SUBJECTS (n=8)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
EVER HAD SUBJECT MATTER TUTORING	3	75	1	12.5	62.5
SUSPENDED OR EXPELLED	2	50	0	0	50
EVER BEEN IN LD CLASS	2	50	0	0	50
EVER REFERRED FOR SPEECH PROBLEM	2	50	1	12.5	37.5
EVER REFERRED FOR READING PROBLEM	2	50	1	12.5	37.5
LESS LIKELY IN LOW AROUSALS					
NONE					
PARENT-REPORTED SYMPTOMS: NO DRUG					
MORE LIKELY IN LOW AROUSALS					
STEALS FROM FAMILY	3	75	1	12.5	62.5
FIGHTS A LOT	4	100	4	50.0	50.0
LOW SELF ESTEEM	3	75	2	25.0	50.0
SETS FIRES	3	75	2	25.0	50.0
SKIPS CLASS, PLAYS HOOKEY	2	50	0	0	50.0
OVER DEPENDENT ON MOTHER	3	75	2	25.0	50.0
DISTURBED SLEEP CYCLE	4	100	4	50	50.0
RECKLESS	2	50	1	12.5	37.5
TRIED UNPRESCRIBED DRUGS	2	50	1	12.5	37.5
STEALS FROM OTHERS	2	50	1	12.5	37.5
PERSECUTION COMPLEX	2	50	1	12.5	37.5
IRRITABLE: TEMPER TANTRUMS	3	75	4	50.0	25.0

TABLE B-2 (continued)

VARIABLES	LOW AROUSAL HYPERKINETIC SUBJECTS (n=4)		CONTROL HYPERKINETIC SUBJECTS (n=8)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
DESTRUCTIVE	3	75	4	50.0	25.0
HARD TO RAISE	4	100	6	75	25.0
EASILY UPSET	3	75	5	62.5	12.5
LIES OFTEN	4	100	7	87.5	12.5
LESS LIKELY IN LOW AROUSALS:					
NERVOUS, TENSE, SHAKEY	0	0	5	62.5	-62.5
UNUSUAL WORRIES	0	0	3	37.5	-37.5
AFRAID TO FIGHT	0	0	3	37.5	-37.5
EPISODIC DEPRESSION	1	25	4	50	-25.0
NAUSEA: NO MORNING APPETITE	3	75	7	87.5	-12.5
TEACHER-REPORTED SYMPTOMS: NO DRUG					
MORE LIKELY IN LOW AROUSALS					
FIGHTS A LOT	4	100	0	0	100
GENERALLY UNCOOPERATIVE	4	100	1	12.5	87.5
DOESN'T FINISH WORK	3	75	1	12.5	62.5
DOESN'T ATTEND TO DIRECTIONS	4	100	3	37.5	62.5
IMPULSIVE	4	100	4	50	50
UNCOORDINATED	3	75	3	27.5	37.5
DISRUPTS CLASS	3	75	3	37.5	37.5
DOESN'T FINISH PROJECTS	2	50	1	12.5	37.5

TABLE B-2 (continued)

VARIABLES	LOW AROUSAL HYPERKINETIC SUBJECTS (n=4)		CONTROL HYPERKINETIC SUBJECTS (n=8)		DIFFERENCE SCORE (%)
	No.	%	No.	%	
EASILY FRUSTRATED	2	50	1	12.5	37.5
IS UNPOPULAR	2	50	1	12.5	37.5
UNDISCIPLINED	2	50	1	12.5	37.5
SHOWS OFF	1	25	0	0	25
FEELS UNPOPULAR	4	100	6	75	25
STEALS FROM PEERS	4	25	0	0	25
FEELS A FAILURE IN SCHOOL	1	25	1	12.5	12.5
FIDGETS	4	100	7	87.5	12.5
HURRIES	2	50	3	37.5	12.5
TALKATIVE	4	100	7	87.5	12.5
DEMANDS MUCH ATTENTION	3	75	65	62.5	12.5
CAN'T LEAVE OTHERS ALONE	4	100	7	87.5	12.5
EASILY DISTRACTED	4	100	7	87.5	12.5
SLOW UNCLEAR SPEECH	1	25	1	12.5	12.5
LESS LIKELY IN LOW AROUSALS					
SHORT ATTENTION SPAN	3	75	7	87.5	-12.5

VITA

Margaret Nichols Shouse was born in Memphis, Tennessee on January 6, 1950. Her family subsequently moved to Knoxville, Tennessee where she attended primary and secondary schools. She was graduated from The Webb School for Girls in June, 1967. The following September she entered Rollins College where she majored in Behavioral Science. In June, 1971, she received The Bachelor of Arts degree with distinction.

In the Fall of 1971 she accepted a teaching assistantship at The University of Tennessee and began graduate study in Psychology. She was appointed an NIMH Physiological and Comparative Trainee for the following two years and was an Instructor of Psychology from September, 1974 until June, 1975. She received the Doctor of Philosophy Degree with a major in Psychology in August, 1976.