DIFFERENTIAL TREATMENT EFFICACY OF EEG AND EMG FEEDBACK FOR HYPERACTIVE ADOLESCENTS

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PREFACE

Previous investigations have examined the utility of biofeedback for the amelioration of cognitive and behavioral symptoms of hyperactivity. However, these studies either have used different forms of EEG and EMG feedback without comparison treatments or have not investigated both behavioral and cognitive changes that accrue from training. This study is to evaluate the comparison of the effects of three forms of EEG or EMG feedback with no-training controls on a comprehensive profile of physiological, cognitive, and behavioral changes.

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CHAPTER I

INTRODUCTION

Biofeedback is a technique which involves the use of electronic equipment to monitor a subject's physiological processes (which are normally not attended to and not under "voluntary" control). The essence of the technique is to make these physiological processes known to the subject by means of some external stimulus such as a light or tone. This "externalization" of information about internal functioning ultimately allows the subject to gain voluntary control over his/her internal physiological systems (Braud, Lupin and Braud, 1975).

Electromyograph (EMG) is an electronic device for monitoring bioelectrical intramuscular activity through electrodes on various cutaneous body areas. This biofeedback training technique is believed to reduce anxiety, muscular tension and produce a calmness and relaxation state. Electroencephalograph (EEG) is an electronic device for monitoring the frequency and amplitude of electrical brain wave activity. It has been argued that people can selectively control the frequency spectrum of their EEG if they are given a signal indicating their success in producing the desired EEG pattern (Beatty, 1972). Subjects trained to control EEG alpha rhythms often associate a feeling of calmness, pleasant relaxation and increased inner awareness. Conversely, subjects trained to control the EEG beta activity relate feelings of frustration, tension, and mental attentiveness. Also, it has generally

been assumed that as subjects are trained to augment alpha density they concomitantly learn to regulate their level of arousal and also gain control over the degree of experienced anxiety (Paskewitz, 1973). Research findings have supported (Orne and Paskewitz, 1973) and contradicted (Walsh, 1973) this assumption. In spite of these contradictory findings, biofeedback training appears to be a logical technique in the solution of behavioral problems, namely, hyperkinesis (Braud, Lupin and Braud, 1975; Nall, 1973). However, research literature reports only three studies on the use of biofeedback as a treatment technique for hyperactive children. Two unpublished studies have investigated the effects of biofeedback training in the adolescent with learning disabilities, but not the hyperactive adolescent.

Braud, Lupin and Braud (1975) provided findings in which electromyographic (EMG) biofeedback techniques were used to control the hyperactivity of a six and one-half year old male. The electrical activity of the frontalis (forehead) muscle group was recorded as the subject trained to reduce his muscular activity and tension level through the use of EMG biofeedback. Observation of both parents and teachers indicated a general overall improvement in the subject's behavior in class and at home. Noted psychosomatic symptoms (headaches, allergies, asthma, and running nose) were eliminated in laboratory sessions after the fifth biofeedback session. A dramatic change in signs of emotionality was noticed over sessions, frustration decreased as signs of confidence increased. The subject showed a marked improvement on the ITPA, and also improved performance on the WRAT and Stanford Achievement test.

Angie Nall (1973) studied the effects of biofeedback alpha training procedures in an attempt to modify inappropriate behavior in children

with learning disabilities characterized as hyperkinetic. She used both academic and behavioral measures to assess the training. The final assessment of the study indicated significant improvement in both measures, in specific cases, but few overall significant effects. There were no significant differences in the overall achievement of the three groups studied (alpha biofeedback training, placebo, and control), although in reading comprehension the alpha training group showed a substantial increase over the other two groups. Unfortunately though, assessing the gains and losses in achievement in relation to the alpha training, no statistical significance was evident. As a matter of fact, the control group showed fewer losses than the placebo or training group. It was only when both achievement and behavioral indices were compared in gains and losses, that the treatment group showed consistent increment or decrement in both areas at the same time. The control and placebo groups varied in the gains and losses, whereas all but one of the training groups improved or became worse in both areas at the same time.

Angie Nall (1973) questioned if this synchrony of behavior and achievement was specific to alpha training, or were other variables effecting performance, like special attention, the relaxation, or some unknown factor?

Murphy and Darwin (1975) reported similar hypotheses as a result of their findings to the use of EEG alpha training treatment. In one study, they investigated the effects of alpha and beta EEG feedback training in learning disabled adolescent students on measures of achievement tests, in the affective domain, and on teacher ratings of academic and socioemotional behavior. Biofeedback consisted of a total of 15

20-minute sessions, with the first five sessions given to feedback of frontalis EMG and the last 10 sessions for EEG feedback in the left occipital-temporal cerebral hemisphere (five subjects received alpha and four beta EEG training). The EMG relaxation group results indicated a significant reduction in frontalis muscular tension over the five sessions. Training was effective in producing a decreased occipital frequency for the alpha subjects, and an increased occipital baseline frequency for beta subjects. Alpha training produced specific enhancement of arithmetic scores (WRAT, grades), a giving up of interpersonal control, increased felt expressed warmth, significantly greater decrement in projective anxiety, and poor teacher ratings of improvement in both the socio-emotional and academic areas. The beta training produced a decrement in arithmetic, greater behavioral and felt independence, no change in expressed warmth, and high ratings of improvement by their teacher in academic and socio-emotional areas. This differentiation might tentatively be attributed to alpha training's greater ability to produce reductions in anxiety, leading to a greater sense of security but possibly reduced teacher-perceived achievement motivation.

Lubar and Shouse (1976) attempted to explore the potential application of sensorimotor rhythm (SMR) training to hyperkinesis in the absence of a seizure history. This study reports one subject's data extracted from an ongoing group of 12 hyperkinetic subjects because he has been in (SMR) training for a significantly longer period of time than the others. The subject was an 11 year, 8 month old male. The subject participated in five consecutive experimental phases (I, No Drug; II, Drug Only; III, Drug and SMR Training; IV, Drug and SMR training reversal training; and V, Drug and SMR training III) and was involved

in several months of SMR training using a 12 to 14 Hz rhythm appearing over the Rolandic cortex. Changes in motor inhibition were indexed by muscular tension in the laboratory and by behavioral observations in the classroom. The feedback presentation for SMR was contingent on the production of 12 to 14 Hz activity in the absence of four to seven Hz slow-wave activity. A substantial increase in SMR occurred with progressive SMR training and was associated with enhanced motor inhibition, as gauged by laboratory measures of muscular assessment in the classroom. Opposite trends in motor inhibition occurred when the training procedure was reversed and feedback presentations were contingent on the production of four to seven Hz in the absence of 12 to 14 Hz activity.

In another study, Murphy, Darwin and Murphy (1976) examined bilateral EEG integrated amplitude measures or power during verbal and spatial processing in two groups of learning disabled adolescents. The subjects' selection consisted of a Discrepant group who had Wechsler IQ scores 15 points above their Verbal IQ scores, and IQ Similar group whose Verbal and Performance IQs were no more than five points discrepant. On both hemisphere specific functions (left-verbal; right-spatial), the Discrepant group was significantly less aroused than the Similar group. Seemingly, the hypoaroused state in both hemispheres for the Discrepant adolescents led to the presumption of cerebral dysfunction.

Satterfield and Dawson (1971) have found that in general the CNS stimulant-responsive children tend to have high electrodermal resistances and high EEG power in the zero to eight Hz frequency band. Duffy's (1972) study supported the observation that electrodermal measures

correlate well with levels of arousal. This reflects a low level of arousal because high skin resistance is strongly associated with lower levels of arousal. Recently, the Grunewald-Zuberbier, Grunewald and Rasche (1975) electroencephalographic study supported the notion of an underaroused CNS for the hyperkinetic child. They reported that hyperactive children have a higher alpha and beta amplitude, more alpha waves and a smaller amount of beta waves. All this research demonstrates a lower state of EEG arousal in the hyperkinetic child.

In summary, studies on biofeedback training of learning disabled children characterized as hyperactive have led to a series of paradoxical results. Paradox one states, based upon the hyperactive behavioral symptoms of the Hyperkinetic Syndrome, that the assumption has been found that the Hyperkinetic Syndrome occurs as a result of escessive neural excitation or a hyperaroused brain. Empirical data to support this assumption is nonexistent. However, on the contrary, EEG studies do support the position that hyperkinetic children have a hypoaroused or underaroused central nervous system (Satterfield and Dawson, 1971; Grunewald-Zuberbier, Grunewald and Rasche, 1975). The second paradox states, given the hyperkinetic child's brain activity level is in a hypoaroused state, further training the brain to a lower aroused state should worsen his behavior and cognitive functioning. In addition, training the brain to a higher arousal state should improve his cognitive functioning. However, recent research findings in biofeedback report inconclusive evidence. Braud, Lupin and Braud's (1975) study with EMG biofeedback is an example of treatment that is purely symptomatic at the behavioral level; however, the results of such training produce both cognitive and behavioral improvement in hyperkinetic

children. Nall's (1973) study using EEG alpha training (reduced arousal) as a treatment technique exemplified a synchrony effect on both cognitive and behavior function. The treatment groups either consistently gained or lost simultaneously in both measures. Murphy and Darwin's (1975) study showed that EEG alpha training enhanced cognition but worsened behavior, while EEG beta training (increasing arousal) decremented cognition and improved behavior. Murphy and Darwin's findings would explain the paradoxes if EEG alpha training specifically enhanced cognition but decremented behavior. Thus, EEG training would manifest a direct effect upon behavior, and inversely affect cognition. In an attempt to clarify the possible discrepancy between Nall (1973) and Murphy and Darwin (1975) findings, and also to present a judgment of total effectiveness of EMG treatment, the present study proposed to investigate the treatment effect of the three specific biofeedback training conditions (EEG alpha and beta, and EMG) on hyperactivity. One treatment group consisted of EMG biofeedback training, a symptomatic treatment of hyperkinesis, aimed solely at the behavioral level. A second treatment group consisted of unilateral EEG alpha training, to determine its specific hemisphere effect on cognitive enhancement. The third group consisted of no training control and was designed to (1) serve as a control condition for the alpha training group, (2) to determine its effect on a hypoaroused brain by training the brain to a more higher aroused state, and (3) to determine if this training produced at least a behavioral improvement. The fourth group was a no-treatment control for a possible regression to the mean or practice effects. The control group's change scores provided the baseline against which the treatment groups' change would be

assessed. All groups were evaluated on a behavioral and cognitive profile.

CHAPTER II

METHOD

Subjects

The subjects in this study were 28 adolescent learning disabled students characterized as behavioral hyperactive, selected from a population of learning disabled students served by the Oklahoma Title VI-G Child Service Demonstration Center for grades 7 to 12. The learning disabled adolescents had been previously assessed and identified as learning disabled through a psycho-educational evaluation (WISC-R, WRAT and Bender Gestalt Visual-Motor test). The subject participants for this study were identified as hyperactive adolescents by a five-point Likert-type behavioral screening scale (Appendix B). Thirty-two hyperactive adolescents were screened positively from a total population of 128 students. However, four of the 32 students were excluded from the study because of their participation in an earlier research project, leaving a total of 28 (22 males and six females) adolescents (Table I).

This screening device consisted of five items that represent five major symptoms (restlessness or overactivity, aggressivity, distractibility or inattentiveness, antisocial conduct, and socio-emotional immaturity) that seemed to persist from childhood through adolescence for the hyperkinetic child as indicated by Safer and Allen (1975), Maletzky (1974), Minde, Weiss and Mendelson (1972), Mendelson, Johnson

and Stewart (1971), and Stewart et al. (1966). On the screening instrument, only subjects who showed the presence of hyperactive symptoms (mean per item rating of greater than 2.5 on a one to five scale) met the criteria of behavioral hyperactivity.

TABLE I

AVERAGE AND STANDARD DEVIATION OF GROSS DESCRIPTIVE DATA FOR SUBJECTS BY AGE, GRADE, AND SEX

Groups	Sex		Age		Grade Level	
	Male	Female	Mean	SD	Mean	SD
Alpha	6	1	13.9	.92	8.2	.58
Beta	5	2	14.7	.73	8.7	.46
EMG	5	2	14.3	1.17	8.5	.63
Control	6	1	14.2	1.34	8.4	.91

In an attempt to tap the basic symptoms of hyperactivity the scale was constructed from two major studies. Stewart et al. (1966) systematically described the hyperactive child syndrome, basing their report on a study of 37 children of average age seven and one-half years. Between the control (normals) and patient (hyperactive) groups in this study, five symptoms were found to be good discriminators between the patient and control group (Table II). Mendelson et al. (1971) did a follow-up study later on hyperactive teenagers between the ages of 12 and 16 which included children of the earlier study who were diagnosed as hyperactive. In the follow-up study, Mendelson et al.

TABLE II

PERCENT POSITIVE SCORES IN THE PATIENT AND CONTROL GROUPS FOR SYMPTOMS SCORED POSITIVE BY ONE-THIRD OR MORE OF THE HYPERACTIVE PATIENTS

Items	Patients	Controls	Difference
Overactive	100	33	67*
Can't sit still	81	8	73*
Restless in MD's waiting room	38	3	35
Talks too much	68	20	48
Wears out toys, furniture, etc.	68	8	60*
Fidgets	84	30	54
Gets into things	54	11	43
Unpredictable	59	3	56
Leaves class without permission	35	0	35
Unpredictable show of affection	38	3	35
Constant demand for candy, etc.	41	6	35
Can't tolerate delay	46	8	38
Can't accept correction	35	0	35
Temper tantrums	51	0	51
Irritable	49	3	46
Fights	59	3	56
Teases	59	22	37
Destructive	41	0	41
Unresponsive to discipline	57	0	57
Defiant	49	0	49
Doesn't complete project	84	0	84*
Doesn't stay with games	78	3	75*
Doesn't follow directions	62	3	59
Hard to get to bed	49	3	46
Enuresis	43	28	15
Lies	43	3	40
Accident prone	43	11	32
Reckless	49	3	46
Unpopular with peers	46	0	46
Moves from one activity to another in class	46	6	40
Doesn't listen to whole story	49	0	49

*Indicates the five symptoms found as good discriminators.

Source: M. A. Stewart, F. N. Pitts, A. G. Craig, and W. Dieruf, The Hyperactive Child syndrome, American Journal of Orthopsychiatry (1966).

on hyperactive teenagers, items three and four of the screening instrument (categorized as overactivity or restlessness) were still prevalent as a symptom in 71 percent of the hyperactive teenagers. Items one and five of the screening instrument, categorized as distractibility, persisted as a symptom in 77 percent of hyperactive teenagers. Item two of the screening instrument persisted in 52 percent of hyperactive teenagers as an antisocial symptom at follow-up.

Biofeedback Trainers

The trainers were one undergraduate and two graduate students in psychology. These students were trained specifically in carrying out the procedures for applying electrodes, conducting the training sessions, and in giving instructions to the subjects for both biofeedback electronic devices, the Autogen 120 Electroencephalograph and Autogen 1500 Electromyograph.

Trainers received practice on mock students until they could apply the electrodes for both biofeedback devices accurately, quickly, and smoothly. For the Autogen 120, it was necessary to obtain the subject's help each time the electrodes were applied. The subject held the electrodes in place while the trainer secured them with an elastic headband. Therefore, it was necessary for the trainer to understand how to effectively enlist this help from the subject. Trainers were then observed during at least one complete training session by an experienced teacher. When it was judged that the novice trainer understood each aspect of the sessions, he/she was allowed to conduct a session under the observation of an experienced teacher. If the observing trainer judged the novice trainer competent in all phases of a session, the novice trainer was

allowed to conduct a session without supervision. However, a novice trainer was not allowed to conduct his/her first solo session with a first session subject.

For the Autogen 1500, the novice trainer had to meet the same number and kind of training experiences as for the Autogen 120. However, instead of enlisting assistance from the subjects, the trainer was required to become skillful in appropriately placing the Autogen 1500 three silver/silver chloride electrodes to the subject's frontalis (forehead) muscle.

Apparatus

Apparatus for training consisted of a large, cushioned chair and hassock, a feedback-electromyograph Autogen Systems 1500, and a feedback encephalograph Autogen Systems 120. Brainwave biofeedback was given to two groups of subjects (alpha and beta) via the use of the Autogen 120 feedback unit from the left-occipital-temporal-hemisphere. The feedback was delivered to the subject by a set of stereo headphones. In the case of a decrease frequency condition, the upper threshold was set at the subject's baseline and lower threshold was set at four Hz, the lowest frequency cut off on the theta range. For an increase frequency condition, the lower threshold was set at the baseline and the upper threshold was set at 20 Hz, the highest frequency graduation on the Autogen 120. With the former setting, the subject was required to decrease brain wave frequency in order to move into the band and turn the feedback sound on (down training). With the latter setting, the subject was reminded to increase his brain wave frequency in order to move into the band and turn the sound on (up training).

During the feedback sessions, the Spectrum was set at seven, Integration at six, lower Amplitude threshold at zero, and upper Amplitude threshold at 100 with the Scale at 11.

The third treatment group received bioelectrical feedback from the Autogen 1500 Electromyograph. Baseline value of the frontalis EMG was obtained at the start of each session. The feedback mode used was a standard click feedback. These clicks which were proportional to the average integral microvolts recorded from the subject's frontalis, were delivered through the subject's headphones. The subject received instructions to reduce the frequency of the clicks that he heard.

Experimental Procedures

For all subjects, a teacher rating scale and parent rating scale were obtained. The original construction of the teacher's behavioral rating checklist was divided into five categorical factors: (1) defiance or aggressivity, (2) antisocial behavior, (3) inattentiveness or distractibility, (4) socio-emotionality, and (5) hyperactivity or overactivity. The items under each category were then randomly arranged for rating checklist symptoms. Such strategy was implemented to alleviate selection bias by the rater since some of these items may be found under more than one category of symptoms. The rating scale for teachers consisted of 45 items of classroom behavior arranged in checklist form so that the teacher could check off whether the child exhibited each individual item of behavior: (1) not at all, (2) a little bit, (3) moderately, (4) quite a bit, or (5) extremely. These individual items of behavior were given numerical scores of zero, one, two, three, and four

respectively, and then summed to give a total rating score across all behavior items. This teacher's Behavioral Observation Checklist (BOC) contained items adapted from both Conner's (1969) and Peterson-Quay (Wender, 1973) Behavioral Checklist for classroom teachers. Burns and Lehman (1974) provided supporting evidence that summated ratings used to assess the hyperactivity of children were an internally consistent and reliable normative technique for measuring hyperactivity. An analysis of the internal consistency of summated ratings revealed coefficients of .87 and .94. The test-retest reliability coefficients of the total summated ratings has been found to be .92.

The Werry-Weiss-Peters Activity Scale was used for parents to rate the activity level of their adolescent child. The Werry-Weiss-Peters Activity Scale was found to be the widest in use for hyperactivity according to Safer and Allen (1975). This scale is a useful measurement to evaluate the degree of hyperactivity because it offers a means of quantification of activity level (Werry and Sprague, 1970; Safer and Allen, 1975). The parent rating scale of hyperactivity was found to correlate moderately ($r = .6$ to $.7$) with the teacher's rating scale. The Werry-Weiss-Peters Activity Scale was also arranged in checklist form so that the parents could check each item of behavior as: (1) no activity, (2) some, or (3) much activity. Three measures (Behavioral Screening Device, Teacher's Behavioral Observation Checklist, and the parental questionnaire--Werry-Weiss-Peters Activity Scale) were to provide reliable assessment of the degree of hyperactivity and its associated symptoms.

In addition, the present study used the Wide Range Achievement Test (WRAT) to measure arithmetic, spelling, and reading performances. The

subtests of the Wechsler Intelligence Scale for Children-Revised (WISC-R), Digit Span and Coding, were measures for attention span and concentration ability, respectively.

There were two EEG biofeedback conditions that monitored the unilateral left occipital temporal hemisphere through the use of the Autogen 120 feedback encephalograph. The alpha EEG training required the subject to decrease the frequency activity (down training) of the left hemisphere, and the beta EEG training required the subject to increase the frequency (up training) of the left hemisphere. The third treatment condition, EMG electromyographic biofeedback, involved monitoring the biofeedback activity of the frontalis (forehead) through the use of the Autogen 1500. The frontalis was selected because its tension level is believed to be a good index of general physical and mental activity (Budzynski and Stoyva, 1973).

Pre- and post-tests were administered to all subjects. These tests included the Behavioral Observation Checklist (BOC), the Werry-Weiss-Peters Activity Scale parent questionnaire, the WRAT, and the Digit Span and Coding subtests from the WISC-R. Resource room teachers filled out the questionnaire activity scale before and after the training treatment. The WRAT and subtests from the WISC-R were administered individually for pre- and post-test measures.

Seven subjects were assigned to each of the three biofeedback conditions and the control condition which consisted of pre- and post-testing sessions without intervening biofeedback training. Each biofeedback subject received eight 20-minute sessions with appropriate instructions, according to their respective treatment technique. The subject was

informed as to the general nature of the study, but she/he was not told of the three differential treatment techniques.

Before the first training session, all subjects for the alpha and beta groups were familiarized with the feedback sound which was a type of white noise. She/he was also shown the sound that muscle artifact produced, a crackling sound, plus the noise produced by a misplaced electrode, a buzzing sound. She/he was instructed to keep the sound on as much as possible in both ears by any internal strategy that worked. The subject was also told that if at any time during the session she/he was able to keep the sound on easily the experimenter would move the criterion threshold so as to make it more difficult. If this happened, the subject would hear a burst of feedback sound followed by a quiet period and this would mean that she/he was doing exceptionally well. These are the instructions:

> The purpose of this procedure is to teach you biofeedback training so that you can learn to control the electrical activity of your brain. This electrical activity is called EEG signals. I will know how well you are controlling the EEG activity by monitoring the electrical activity with these electrodes. You will hear a sound through these headphones. It will be a swishing or a bumping sound followed by silence, and your task will be to make the swishing sound stay on as much as possible. If you hear a hum, your electrode contact is inadequate and you should ask for your electrodes to be rechecked. The session will last 20 minutes. There will be a one minute break every 10 minutes.

After these initial instructions and at the start of all subsequent training sessions the subject was given further instructions as follows:

> Please sit here in a comfortable position with your spine straight and your head drooped slightly forward. Remove your shoes, place your feet on the floor, arms and legs uncrossed, while I take your baseline readings. Two readings will be taken before each session, one with your eyes opened and one with your eyes closed. Please do not blink your eyes or move about while the baseline readings are being taken. With your assistance, I am going to place three electrodes around your

> head to monitor the electrical activity from your brain. An elastic band will be wrapped around the back of your head, crossing the forehead to hold the electrodes in place. There will be no chance for you to receive a shock from these electrodes. These electrodes have been saturated in a saline conductive solution to insure good electrical contact. If you use any common hair oils, please clean your hair before I place these electrodes on your scalp. Remember to keep your body as still as possible and do not talk during the training session.

The hair of the subject was parted at Coordinate T3 (above the left ear) and one active electrode placed in the middle of the part, with the sponge side down. The second active electrode was placed at Coordinate O1 (the left occipital lobe), sponge side down. The elastic band was then wrapped around the first active electrode (TS), crossing the forehead approximately one-half inch above the eyes, continuing around the head in a counter-clockwise direction, covering the second electrode, until both ends of the elastic band overlap. The third electrode (ground electrode) was placed underneath the elastic band above the left eye, sponge side down.

Baseline measures of frequency and amplitude were taken as average readings over a time interval of 50 seconds for the left hemisphere by opening the lower and upper thresholds on the frequency and amplitude dials. The value recorded as the frequency baseline was used as the starting reference point for training in that session. The percent time interval selector was set at 100 seconds as the subject was instructed to begin trying to control the EEG feedback by making the sound stay on as much as possible. If at any time during the sessions the subject was able to keep the percent time meter above 90 percent for at least 30 seconds, the reference was reset using the same procedure outlined above for setting the frequency baseline.

All subjects receiving EMG frontalis biofeedback were escorted to the experimental room and given the following instructions:

> Please sit down here. I am going to place three electrodes on your forehead to monitor the level of tension in your forehead muscle. There is no chance for you to receive a shock from these electrodes. I will also clean your forehead with alcohol to insure good contact.

The foreheads of the subjects were then cleansed with alcohol, and the three electrodes positioned. The two active electrodes were placed one inch above the eyebrows and spaced four inches apart. The third electrode, the ground electrode, was placed in the center of the forehead. Once the electrodes had been properly placed, the subject was then given a set of headphones, and asked to sit relaxed in a cushioned chair with legs and feet positioned on a hassock, arms and legs uncrossed. The subject was then instructed how they might use the sound feedback in learning to relax. The instructions were as follows:

> The purpose of this procedure is to teach you biofeedback training so that you can better learn to relax. I will know how relaxed you are by monitoring the forehead muscle with the electrodes. You will hear a sound through these headphones. It will be a crackling sound, and your task will be to reduce the rate of the popping sounds. As you are reducing this popping noise rate, you are actually reducing the level of tension in your forehead muscle--the muscle we are monitoring. This session will last for 20 minutes. There will be a one minute break every 10 minutes. Remember to keep your eyes closed, and do not talk or move except during the one minute breaks.

Following the relaxation instructions, a baseline value was recorded in average integrated microvolts at the beginning of each EMG training session. Then following the initial baseline, microvoltage variation was recorded at three minute intervals during the session.

Finally, throughout all three training conditions, all subjects were encouraged and supported in their efforts to consciously learn to

control the EEG brain wave or EMG tension level. All subjects received the assigned training at the same time of the day for all their individual sessions.

Design

Independent Measures

The independent between subject variable used in the study was the treatment condition. Seven subjects were assigned randomly to each of the treatment conditions. There were three biofeedback training groups and a no-training control group. The three biofeedback treatment conditions were left-occipital-temporal EEG alpha training, left-occipital-temporal EEG beta training and frontalis EMG relaxation training. The other independent measures were all within subjects variables: pre- and post-tests, time, training sessions, and trials within training sessions.

Dependent Measures

For all subjects, left hemisphere EEG amplitude and frequency baseline measures and baseline measures of frontalis EMG in average integral microvolts were recorded for the two testing sessions. At the start of each testing session, two recordings of each measure were obtained. Training session data were obtained for a total of eight sessions for each subject in the training groups. Pre-session baseline measures, appropriate to each group, were obtained for all three treatment groups. For the two EEG training groups, training sessions data were based on EEG frequency and amplitude. For the EMG training group, training session data were based on frontalis EMG levels.

The pre- and post-test scores on all three subtests of the WRAT (reading, spelling, and arithmetic), and subtests of the WISC-R, Digit Span and Coding, were the second set of dependent measures. The third set of dependent measures was the pre- and post-test scores obtained on the teacher's BOC and the parent's questionnaire, the Werry-Weiss-Peters Activity Scale.

Analysis

Three sets of ANOVAs were performed on the following data. First, a Mixed Model (one Between Ss and two Within Ss) ANOVA were run on all pre- and post-measures. The Between Subjects factor was Groups (EMG, EEG down training, EEG up training, and no-training control). The Within Subjects factors were pre- and post-testing sessions. The dependent measures examined in this design were as follows: Behavioral Observation Checklist (Teacher's Ratings), Werry-Weiss-Peters Activity Scale (Parent's Ratings), Wide Range Achievement Test (WRAT), WISC-R subtests, Digit Span and Coding, left EEG frequency and amplitude, and finally baseline frontalis EMG levels. The results of this ANOVA were to evaluate the differential effects of the biofeedback training on these measures of hyperactivity and achievement.

Second, a Mixed Model (one Between Ss and two Within Ss) ANOVA was performed on the training sessions for the two EEG biofeedback groups. The Between Subjects factor was the two EEG training groups--alpha (down training) and beta (up training). The Within Subjects factors were the eight sessions and seven trials within each session. A trial was a two minute recording period. The dependent measures examined in this design were the baseline frequency and amplitude measures.

Third, a Repeated Measures design (two Within Ss) was run on the training session data for the EMG biofeedback group. The ANOVA had eight sessions and seven trials as its Within Subjects variables. The dependent measures in this ANOVA were the baseline frontalis EMG levels.

CHAPTER III

HYPOTHESES

The following hypotheses were investigated:

I. The EMG biofeedback training group, a symptomatic treatment of Hyperactivity, was expected to enhance arithmetic performance on the WRAT due to a behavioral relaxation effect which would consequently increase attention and concentration and calm behavior.

II. Unilateral EEF alpha biofeedback training, producing a significant hemispheric effect, was predicted to enhance specifically the arithmetic performance over reading and spelling scores and not effect behavioral improvement.

III. Unilateral EEG beta biofeedback training was expected to effect no change on arithmetic performance, but was predicted to cause a positive change in behavior.

The specific predictions under Hypothesis III were:

1. For the Behavior Observation Checklist and Werry-Weiss-Peters Scale measures, a Group by Time interaction effect was predicted such that EMG and EEG beta (up training) would show a greater reduction than the control and alpha (down training) groups.

2. For the Digit Span and Coding measures, a Group by Time interaction effect was predicted such that EMG and EEG alpha (down

training) groups would show greater increments than the control and EEG beta (up training) groups.

3. For arithmetic scores on the WRAT, a Group by Time interaction effect was predicted such that EEG alpha (down training) and the EMG (relaxation) groups were expected to show a greater enhancement than the control and EEG beta (up training) groups.
4. For Reading and Spelling scores on the WRAT, a Group by Time interaction effect was predicted such that EEG alpha (down training), EMG (relaxation) and EEG beta (up training) groups would show a greater increase than the control group.
5. A specific enhancement in Arithmetic score over both Reading and Spelling scores was expected such that a Group by Time interaction effect would show a greater significance for the EEG alpha (down training) and EMG groups than for the EEG beta (up training) groups and control groups.
6. For the frequency baseline measure, a Group by Time interaction effect was predicted such that EEG beta (up training) group would show a greater increase than the control and EMG group; the control and EMG groups were expected to show a greater increase than the EEG alpha (down training) group.
7. For amplitude baseline measure, a Group by Time interaction effect was predicted such that the EEG alpha (down training) group would show a greater increase than the control and EMG groups; the control and EMG groups were expected to show a greater increase than the EEG beta (up training) group.
8. For the frontalis EMG baseline measure, a Group by Time interaction effect is predicted such that the EMG relaxation group

would show a greater reduction than the other three groups.

IV. The frequency and amplitude measures for EEG (alpha and beta) training groups were expected to differ across the training sessions, such that training the left hemisphere down (alpha) would result in a decreased frequency and an increased amplitude, and training in the left hemisphere up (beta) would result in an increased frequency and decreased amplitude. A Group by Session and Group by Trial interaction effect was predicted on both frequency and amplitude measures such that EEG beta (up training) would increase in frequency and decrease in amplitude across both sessions and trials in relation to EEG alpha group; EEG alpha would decrease in frequency and increase in amplitude across both sessions and trials in relation to EEG beta group.

V. The integrated microvolt measures for the EMG frontalis training group were expected to differ across training sessions. A Main Session and a Main Trial effect was predicted such that EMG relaxation group would show reduction in EMG level across both sessions and trials.

CHAPTER IV

RESULTS

To evaluate the biofeedback training data and to determine the differential effects of this biofeedback training on measures of physiology, cognition, and behavior, the results will be presented in four sections. The first section will investigate the training data from the three treatments, EEG (alpha and beta) and EMG (relaxation). The second section presents the physiological changes for the four groups, by measuring pre- and post-baseline means and ranges of left occipital-temporal EMG amplitude (p-p uv), left occipital-temporal EEG frequency (Hz) and frontalis EMG (integrated uv). The third section examines the cognitive changes on the pre- and post-measures of the WRAT and the WISC-R subtests, Digit Span and Coding. Finally, the fourth section will analyze the behavioral changes between the pre- and post-measures of the Behavioral Observation Checklist and the Werry-Weiss-Peters Activity Scale.

Training Data

Two mixed model ANOVAs were performed on the alpha and beta biofeedback groups' EEG data. The Group (2) x Session (8) x Trials (7) ANOVAs were performed separately on the frequency and amplitude measures. On the frequency data, a marginally significant main Group effect,

$F(1,12) = 3.7707$, $p < .09$, was found such that the beta group had a significantly higher mean baseline frequency (10.90714 Hz) than the alpha group's 9.70561 Hz. The Group by Trials interaction was also marginally significant, $F(6,12) = 1.9121$, $p < .10$, as hypothesized (Table III). The beta group increased in frequency across trials within each session in relation to the alpha group (Figure 1). There was a significant result obtained for the Session by Trial interaction, $F(42,504) = 1.7133$, $p < .025$, on frequency. Since this (Session by Trial) interaction effect did not involve the group variable, its importance for this study is minimal.

The ANOVA on EEG amplitude data revealed no significant main nor interaction effects (Table IV). Therefore, no evidence of change between the two training groups across both training sessions and trials was present for the EEG amplitude measure.

For the EMG (relaxation) treatment, the original planned procedure to teach subjects how to relax on the feedback myograph was to reduce the rate of auditory clicks produced by the frontalis muscle tension. However, during some of the training sessions for five of the seven subjects in this EMG group, equipment failure of the myograph's auditory output forced the trainers to substitute visual eyes open feedback for the original auditory eyes closed feedback. Thus, the originally planned Repeated Measures ANOVA to be run on the data for 8 Sessions x 7 Trials for the EMG treatment group was changed to a 5 Session x 6 Trials ANOVA. This Repeated Measures ANOVA was performed only on those five subjects with the first five training sessions that included auditory eyes closed feedback. The Main Sessions effect was found to be significant $F(4,120) = 6.6113$, $p < .01$ (Figure 2); and a marginal effect was obtained

TABLE III

ANALYSIS OF VARIANCE SUMMARY TABLE FOR EFFECTS OF EEG (BETA AND ALPHA) GROUPS (G) X SESSIONS (X) AND TRIALS (T) ON FREQUENCY MEASURES

Source	SS	df	MS	F	p
G	282.9580	1	282.9580	3.7707	$p < .09$
X	69.79097	7	9.970139	1.0681	NS
T	10.09588	6	1.682646	0.4128	NS
S(G)	900.4983	12	75.04152		
GX	99.32695	7	14.18956	1.5201	NS
GT	46.76349	6	7.793915	1.9121	$p < .10$
XT	130.5751	42	3.108930	1.7133	$p < .025$
SX(G)	784.0901	84	0.334405		
ST(G)	293.4854	72	4.076185		
GXT	101.6712	42	2.420743	1.3341	NS
SXT	914.5447	504	1.814572		

Corresponding Frequency Means for Groups by Trials Interaction

Beta	10.3393	10.5536	11.0857	11.2232	11.0263	11.2143	10.9071
Alpha	10.1250	9.8036	9.8125	9.6786	9.4554	9.5911	9.4732

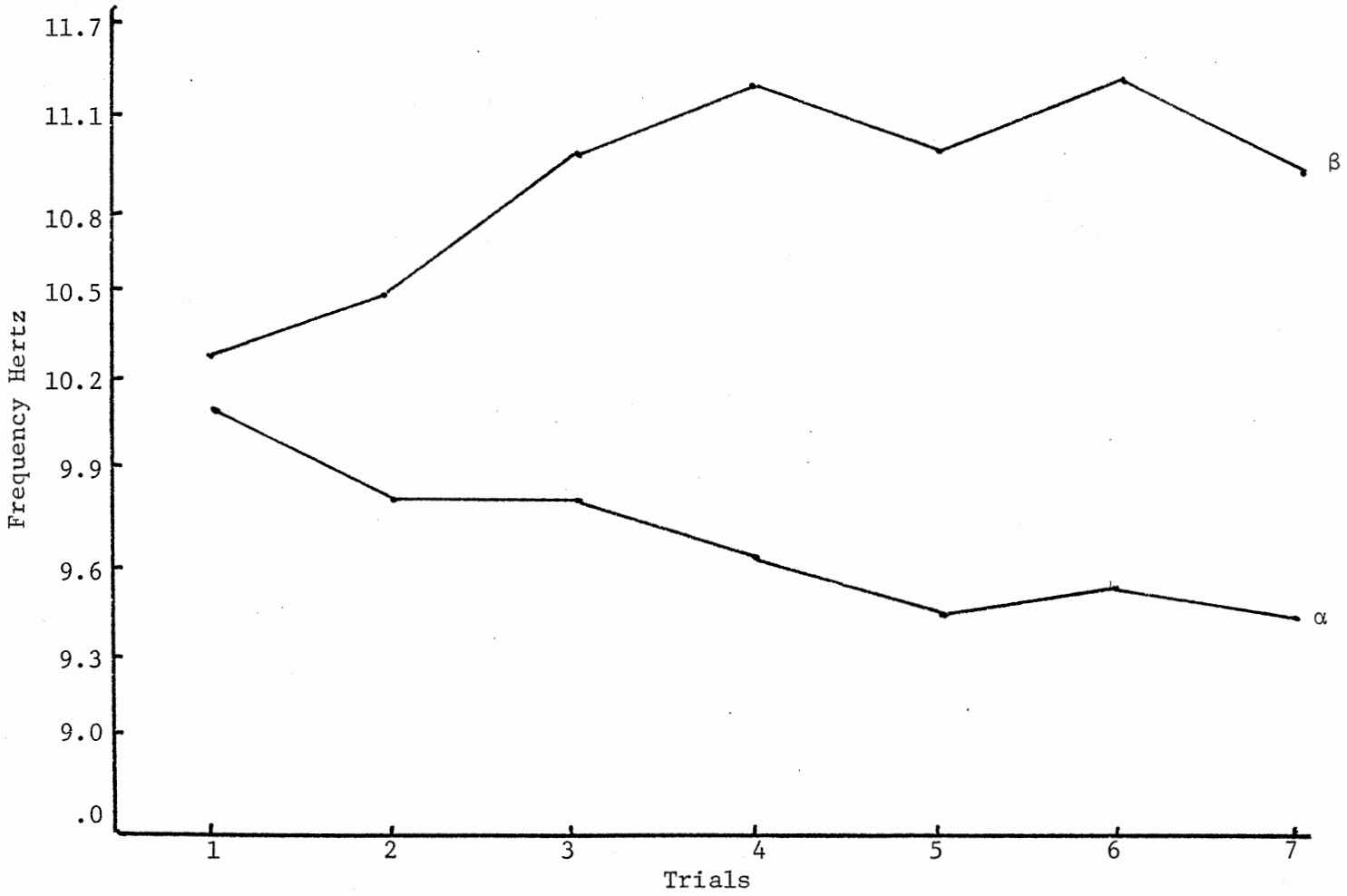

Figure 1. Group x Trials for EEG Frequency Training for EEG Alpha (α) and Beta (β)

TABLE IV

ANALYSIS OF VARIANCE SUMMARY TABLE FOR EFFECTS OF EEG (BETA AND ALPHA) GROUPS (G) X SESSIONS (X) AND TRIALS (T) ON AMPLITUDE MEASURES

Source	SS	df	MS	F	p
G	1237.532	1	1237.532	0.1277	NS
X	9761.668	7	1394.524	0.9366	NS
T	1388.028	6	231.3380	1.0821	NS
S(G)	116311.4	12	9692.617		
GX	13587.32	7	1941.046	1.3037	NS
GT	353.8472	6	58.97452	0.2759	NS
XT	1905.687	42	45.37347	0.5520	NS
SX(G)	125069.8	84	1488.926		
ST(G)	15392.34	72	213.7825		
GXT	3955.583	42	94.18054	1.1458	NS
SXT	41426.76	504	82.19595		

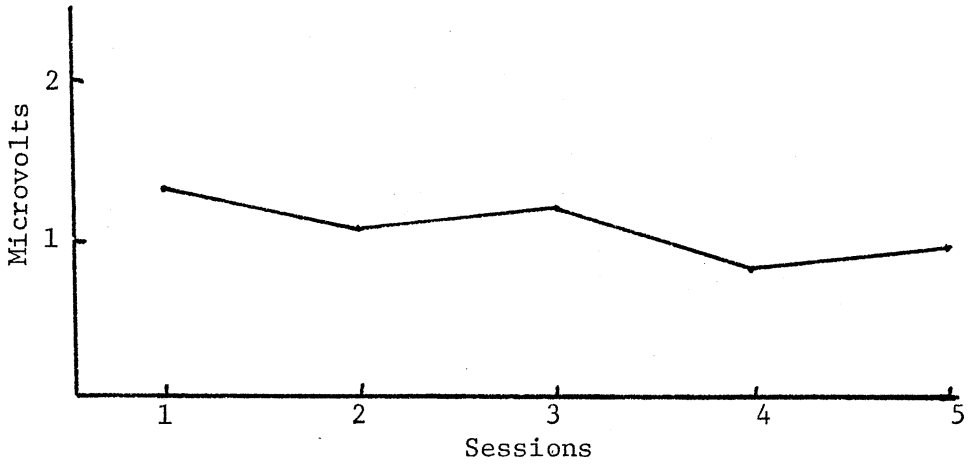

Figure 2. Training Sessions for EMG Relaxation Group on EMG Integrated Microvolts

for the Trials factor, $F(5,120) = 2.0198$, $p < .09$. Both significant outcomes were in support of Hypothesis V. Results of this analysis are presented in Table V. Two subsequent trend analyses performed on both Sessions and Trials effects indicated that the mean reductions on EMG frontalis level showed no significant linear trends for either the main Sessions effect, $F(1,4) = 2.3553$, n.s., or the main Trials effect, $F(1,24) = 1.8575$, n.s. Nevertheless, a general decrease in the two sets of means was reflected across the trial and session factors. The means ranged from 1.295 uv in the first session to .91533 uv on the fifth session; and from trial one, 1.22960 uv to .882 uv in trial six (Figure 3). Nonsignificant means for training data ANOVAs are given in Appendix E.

Physiological Changes

To examine the detrimental effects of the three biofeedback treatments and the no-training control groups on physiological baselines of EEG amplitude and frequency and EMG level from pre- to post-tests, two sets of mixed model (one Between Subjects and two Within Subjects variables) ANOVAs were performed. The first set of ANOVAs used the ranges of values for EEG frequency, EEG amplitude and EMG levels, as the dependent measures on the three ANOVAs. The second set of ANOVAs used the means of the values for these three physiological measures as the dependent variables. For the three ANOVAs performed on the range values, all main and interaction effects failed to yield statistical significance (Tables VI, VII and VIII). See Appendix E for nonsignificant means.

For the second set of ANOVAs performed on the baseline means, the frequency measure ANOVA showed no significant main nor interaction effects (Table IX). But, t-tests for dependent samples used to assess

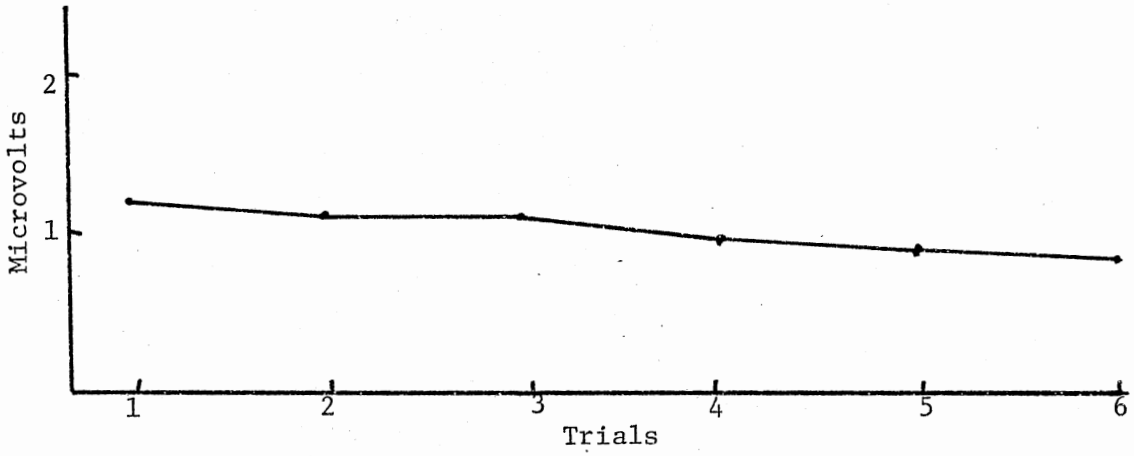

Figure 3. Trials for EMG Relaxation Group on Integrated Microvolts

TABLE V

ANALYSIS OF VARIANCE FOR EMG TRAINING WITH EYES CLOSED FOR SESSIONS (X) AND TRIALS (T) ON EMG FRONTALIS LEVELS

Source	SS	df	MS	F	p
X	5.613900	4	1.403475	6.6113	p < .01
T	2.143806	5	.4287612	2.0198	p < .09
XT	.9395676	20	.4697838E-01	.2213	
S(XT)	25.47408	120	.2122839		

TABLE VI

ANALYSIS OF VARIANCE FOR TREATMENT GROUPS (G) AND PRE-POST TIME (T) ON EEG FREQUENCY BASELINE RANGES

Source	SS	df	MS	F	p
G	.3148215	3	.1049405	.1996	NS
T	.4287500	1	.4287500	1.1520	NS
S(G)	12.61850	24	.5257707		
GT	.5933914	3	.1977971	.5314	NS
ST(G)	8.932521	24	.3721883		

TABLE VII

ANALYSIS OF VARIANCE FOR TREATMENT GROUPS (G) AND PRE-POST TIME (T) ON EEG AMPLITUDE BASELINE RANGES

Source	SS	df	MS	F	p
G	28.28571	3	9.428568	.2066	NS
T	3.500000	1	3.500000	.0771	NS
S(G)	1095.140	24	45.63083	.0771	
GT	53.35715	3	17.78571	.3919	NS
ST(G)	1089.135	24	45.38062		

TABLE VIII

ANALYSIS OF VARIANCE FOR TREATMENT GROUPS (G) AND PRE-POST TIME (T) ON EMG BASELINE RANGES

Source	SS	df	MS	F	p
G	.3029256E-01	3	.1009752E-01	.0256	NS
T	.3332512	1	.3332512	.9140	NS
S(G)	9.455601	24	.3939834		
GT	.2222767	3	.7409221E-01	.2032	NS
ST(G)	8.750269	24	.3645945		

TABLE IX

ANALYSIS OF VARIANCE FOR TREATMENT GROUPS (G) AND PRE-POST TIME (T) ON EEG FREQUENCY BASELINE MEANS

Source	SS	df	MS	F	p
G	10.36644	3	3.455481	0.3738	NS
T	.4828933	1	.4828933	.1426	NS
S(G)	221.8363	24	9.243179		
GT	19.88423	3	6.628077	1.9578	NS
ST(G)	81.24992	24	3.385413		

t-Test Values for EEG Frequency Baseline Means on Pre-Post Change Scores

Treatment Groups	Mean Difference	Standard Deviation	t	p
Alpha	0.0714	2.0470	-0.0923	NS
Beta	1.4286	2.0616	-1.8334	$p < .10$
EMG	-1.9286	2.5611	-1.9923	$p < .05$
Control	-0.25	2.2276	-0.2904	NS

change scores for individual groups, found the EMG relaxation treatment to produce a significant reduction in frequency (t (6 df) = 1.9923, $p < .05$); also the beta group showed a marginally significant increment in frequency (t (6 df) = 1.8334, $p < .10$, one-tailed test). The other two groups showed no reliable change in EEG frequency (Table IX).

A marginally significant main time effect, $F(1,24) = 2.8640$, $p < .10$, was observed on EEG amplitude baseline means. The amplitude showed a reduction across all groups from pre-test ($\bar{x} = 46.4256$ uv) to post-test ($\bar{x} = 39.16071$ uv). No other main nor interaction effects were significant (Table X). This ANOVA failed to meet predictions for Hypothesis III-7, which stated that for the amplitude baseline measures, a Group by Time interaction effect was predicted such that the EEG Alpha (down training) group would show a greater increase than the Control and EMG groups. The Control and EMG groups were expected to show a greater increase than the Beta (up training) group.

The ANOVA performed on the frontalis EMG baseline measure yielded a significant main time effect, $F(1,24) = 33.5320$, $p < .001$, and a significant Group by Time interaction effect, $F(3,24) = 2.25574$, $p < .08$ (Table XI, Figure 4). In general, the EMG levels dropped from pre-test (2.14785) to post-test (1.4875). To further investigate the Group by Time interaction, a set of planned comparisons were run on the four pairs of pre-post test means for each group. Contrary to the hypothesized prediction, alpha, beta and control groups showed significant reductions in EMG frontalis levels while the EMG group's change was not significant, $t(24$ df) = 1.3152, n.s. The EEG beta group yielded the strongest reduction, $t(24$ df) = 5.1048, $p < .001$; EEG alpha next,

TABLE X

ANALYSIS OF VARIANCE FOR TREATMENT GROUPS (G) AND PRE-POST TIME (T) ON EEG AMPLITUDE BASELINE MEANS

Source	SS	df	MS	F	p
G	2750.049	3	906.6829	1.6493	
T	739.5044	1	739.5044	2.8640	$p < .10$
S(G)	13193.67	24	549.7361		
GT	479.5491	3	159.8497	.6191	
ST(G)	6196.938	24	258.2056		

Corresponding EEG Amplitude Baseline Means for Pre- and Post-Time

Pre-Mean	Post-Mean
46.42856	39.16071

TABLE XI

ANALYSIS OF VARIANCE FOR TREATMENT GROUPS (G) AND PRE-POST TIME (T) ON EMG BASELINE MEANS

Source	SS	df	MS	F	p
G	.7638928	3	.2546309	.2900	
T	6.104994	1	6.104994	33.5320	p .001
S(G)	21.07150	24	.877979		
GT	1.396855	3	.4656184	2.5574	p .08
ST(G)	4.369548	24	.1820645		

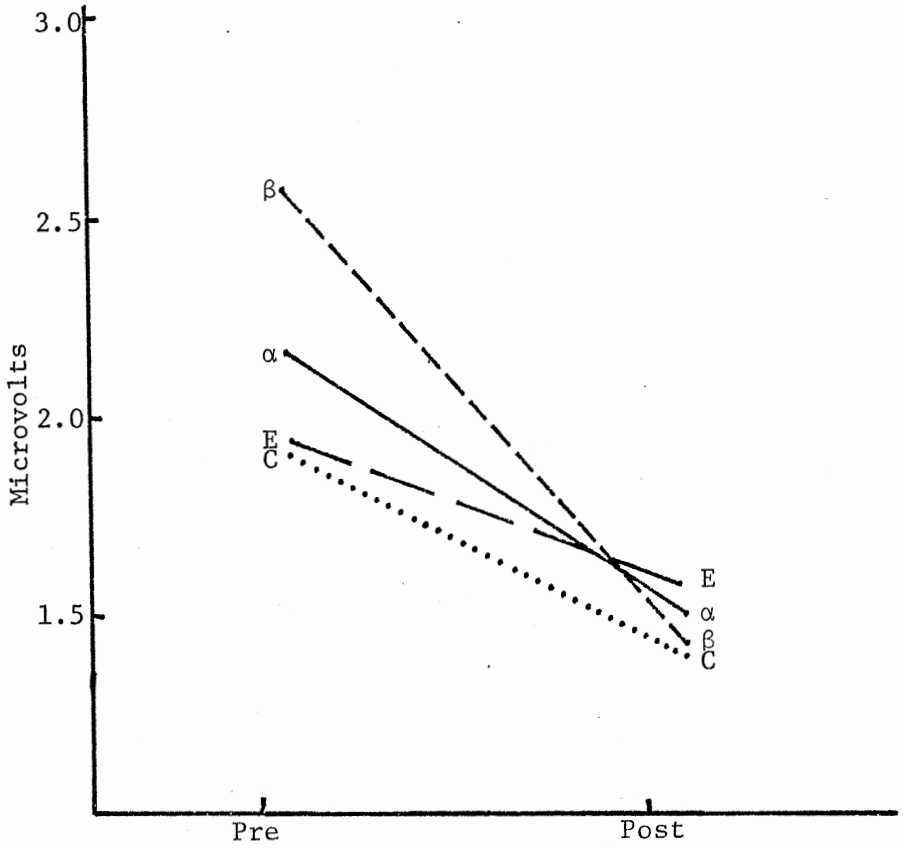

Figure 4. Group x Time Interaction on EMG Integrated Microvolts for EMG (E), Beta (β), Alpha (α), and Control (C)

$t(24\ df) = 2.7998$, $p < .01$; and the control group third, $t(24\ df) = 2.3614$, $p < .05$ (Table XII).

Cognitive Change

To evaluate the effects of biofeedback training on achievement, a mixed model (one Between Ss and two Within Ss variables) ANOVA was to be performed on pre- and post-test measures of the Wide Range Achievement Test (WRAT). However, a Hartley's Fmax test of homogeneity of variance rejected the null hypothesis of equal variances ($F(6)$, $K = 12$, 38.5714, $p < .01$). Therefore, a set of t-tests for dependent samples was used to assess the presence of reliable change for the four groups. In the area of reading, only EEG alpha training produced a significant increase in WRAT reading, $t(6\ df) = 3.16$, $p < .02$ (two-tailed test). Over the three week pre-post test interval, students showed a reliable improvement of .429 months in word recognition grade level on the WRAT. This finding partially supports Hypothesis III-4. However, the expected enhancement in arithmetic score over both spelling and reading scores for the EEG alpha and EMG groups over the EEG beta and control groups was not found on the WRAT scores. Further, in both arithmetic and spelling, no reliable changes were shown for any group (Table XIII).

Subtests from the Wechsler Intelligence Scale for Children-Revised (WISC-R) were used to measure the effects of the four biofeedback treatments on Digit Span and Coding. Two mixed model ANOVAs were to be used to analyze the pre-post WISC-R subtest data. The ANOVA on Digit Span was excluded because a Hartley's Fmax test of homogeneity of variance rejected the null hypothesis of equal variances ($F(6)$, $K = 4$, 12.15, $p < .05$). Therefore, a set of t-tests for dependent samples was computed

TABLE XII

T-TEST VALUES FOR PLANNED COMPARISONS OF PRE-POST EMG BASELINE MEANS FOR TREATMENT GROUPS

Groups	Microvolts (uv) Mean Difference $\bar{x}$	t Values	p	df
EMG	.3 uv	1.3152	NS	24
Beta	1.1642 uv	5.1048	$p < .001$	24
Alpha	.6386 uv	2.7998	$p < .01$	24
Contact	.5386 uv	2.3614	$p < .05$	24

TABLE XIII

SETS OF T-TESTS FOR DEPENDENT SAMPLES ON WRAT CHANGE SCORES FOR TREATMENT GROUPS

Treatment Group	Mean Difference	t Values	p	df
		Spelling		
EMG	-.186	.62	NS	6
Beta	.643	.95	NS	6
Alpha	.043	.378	NS	6
Control	.071	.56	NS	6
		Arithmetic		
EMG	-.086	.69	NS	6
Beta	.000	.00	NS	6
Alpha	-.071	.46	NS	6
Control	-.056	.22	NS	6
		Reading		
EMG	.029	.15	NS	6
Beta	.371	1.25	NS	6
Alpha	.429	3.16	$p < .02$	6
Control	.229	1.29	NS	6

to assess the presence of reliable change for the four groups. Only the EMG training improved performance on Digit Span, $t(6\ df) = 2.483$, $p < .05$. The other three groups showed no reliable change in these scores (Table XIV).

The second ANOVA, Treatment Groups (4) x (pre-post) Time on Coding revealed a marginally significant main Group effect, $F(3,24) = 2.1005$, $p < .10$; and a significant main time effect, $F(1,24) = 8.6471$, $p < .01$, on the Coding scale scores (Table XV). However, the Group x Time interaction effect was not significant implying that concentration did not improve differentially over time among the four groups. Thus, these tests failed to meet prediction III-2 stating that the EMG and EEG alpha groups would show a greater increment than the control and EEG beta groups.

Behavioral Change

For the Behavioral Observation Checklist (BOC) and the Werry-Weiss-Peters Activity Scale (WWP), a Group by Time interaction effect was predicted such that the EMG and EEG beta (up training) groups would reveal a greater reduction in Hyperactive behavior than the control and alpha (down training) groups. A mixed model ANOVA was performed on the pre- and post-scores of the BOC, the teacher's rating measurement, for all four treatment groups. The Group and Time main effects were significant ($F(3,24) = 2.4006$, $p < .10$; $F(1,24) = 5.3193$, $p < .05$, respectively) on the BOC variable (Table XVI). The Group by Time interaction effect was also significant ($F(3,24) = 4.5740$, $p < .05$) on the BOC measure (Figure 5), supporting Hypothesis III-1. Nevertheless, the greatest reduction

TABLE XIV

T-TESTS FOR DEPENDENT SAMPLES ON THE WISC-R SUBTEST DIGIT SPAN CHANGE SCORES FOR TREATMENT GROUPS

Treatment Groups	Mean Difference	t Values	p	df
EMG	1.14	2.483	$p < .05$	6
Beta	1.43	1.37	NS	6
Alpha	.857	.9998	NS	6
Control	.29	.214	NS	6

TABLE XV

ANALYSIS OF VARIANCE FOR TREATMENT GROUPS (G) AND PRE-POST TIME (T) ON WISC-R CODING SCALE SCORES

Source	SS	df	MS	F	p
G	106.3393	3	35.44643	2.1005	$p < .10$
T	24.44643	1	24.44643	8.6471	$p < .01$
S(G)	404.9968	24	16.87484		
GT	3.196426	3	1.065475	.3769	NS
ST(G)	67.85069	24	2.827112		

Corresponding Means for Each Treatment Group on Coding Scores

EMG	Beta	Alpha	Control
9.64286	6.64286	8.64286	10.28571

Corresponding Means for Pre- and Post-Time on Coding Scores

Pre	Post
8.14286	9.46428

TABLE XVI

ANALYSIS OF VARIANCE FOR TREATMENT GROUPS (G) AND PRE-POST TIME ON BEHAVIORAL OBSERVATION CHECKLIST (BOC) MEASURE

Source	SS	df	MS	F	p
G	5296.141	3	1765.380	2.4006	$p < .10$
T	604.5713	1	604.5713	5.3193	$p < .05$
S(G)	17649.54	24	735.3972		
GT	1559.573	3	519.8577	4.5740	$p < .05$
ST(G)	2727.739	24	113.6558		

Corresponding Pre-Post Time Means on Behavioral Observation Checklist

Pre	Post
59.7143	53.1429

Behavioral Observation Checklist Means for the Four Treatment Groups

EMG	Beta	Alpha	Control
41.5000	55.7143	60.2143	68.2857

Group by Time Interaction Means on BOC

Groups	Pre	Post
EMG	49.4286	33.5714
Beta	51.0000	60.4286
Alpha	62.0000	58.4286
Control	76.4286	60.1429

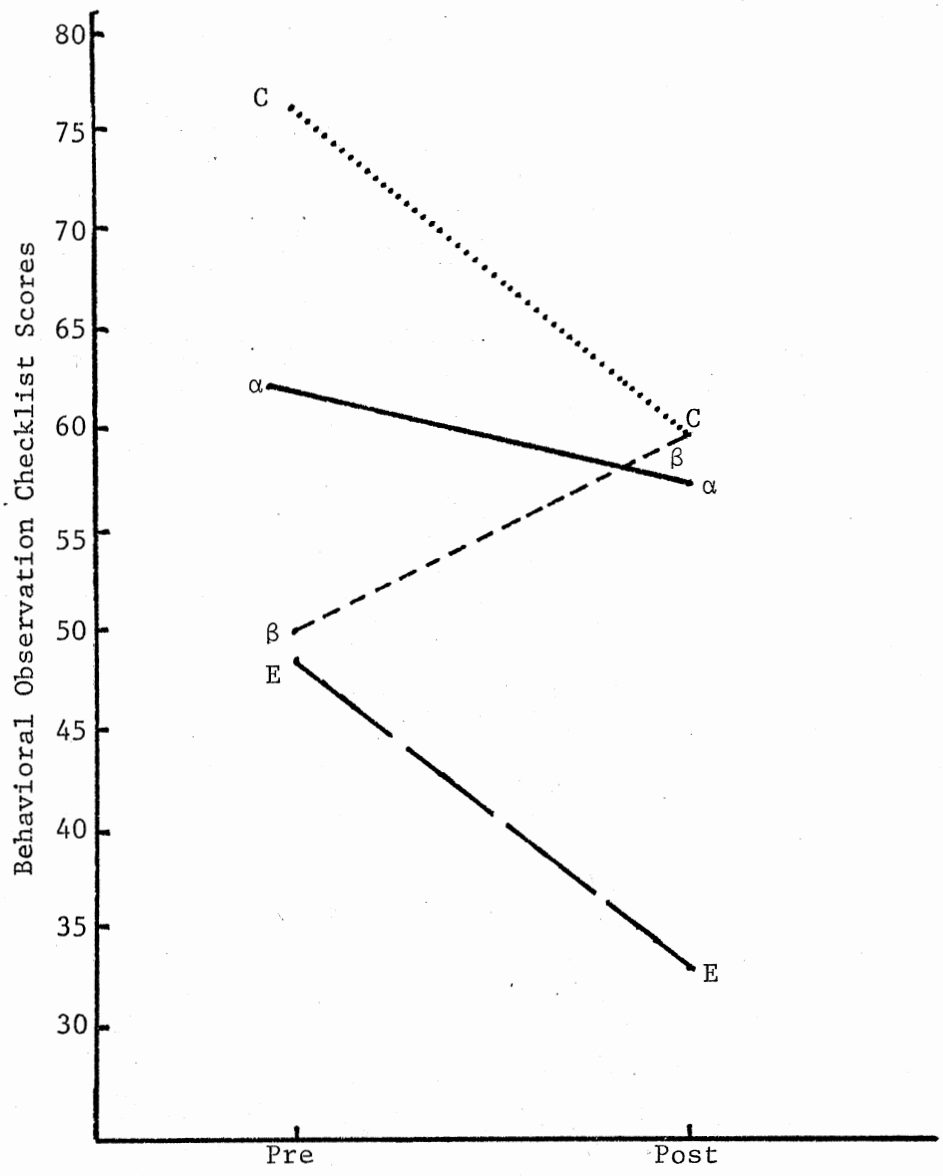

Figure 5. Group x Time Interaction on Behavioral Observation Checklist for EMG (E), Beta (B), Alpha ( ), and Control (C) Groups

(pre-post) scores were indicated by the EMG and Control group means (EMG = -15.86 and Control = -16.29) rather than the EEG beta and EMG as predicted. The behavior reduction for the alpha group was -3.57, and the beta group revealed a +9.43 increase in hyperactive behavior. A simple effects test was run on the observed interaction (Group by Time) to investigate the relationship between the two factors. The simple effects test showed significant group differences in the pre-test (Table XVII). In order to insure equivalence among the groups at the pre-test, a one way analysis of covariance (ANACOVA) was performed. The covariate for the ANACOVA was the pre-test value for the BOC on the adjusted post-test means of the BOC. The covariate and main group effects were both found to be significant ($F = 5.522$, $p < .026$; $F = 2.395$, $p < .093$), respectively (Table XVIII). A variation (15.5 percent) in the BOC post-test score was accounted for by the pre-test values on the BOC.

A set of six planned pairwise comparisons was performed on the adjusted BOC post-test means which indicated that EMG group ($t(24\ df) = -4.6117$, $p < .001$) showed a significantly greater post-test reduction over the other three groups for two-tailed tests (Table XIX). Hypothesis III-1 was partially supported.

An ANOVA was not performed on the WWP scales because of the unequal and small cell sizes due to the insufficient number of returned parent questionnaires for the pre- and post-measures. However, return on the pre-test WWP questionnaires was sufficient to provide a comparison among the four groups' pre-treatment levels of parent-rated hyperactivity. No significant differences were found among the four treatment group means on the WWP pre-test scores (Table XX). t-Tests for related samples computed separately for EMG, beta, and alpha groups on the returned pre- and

TABLE XVII

SIMPLE EFFECTS TEST FOR BEHAVIORAL OBSERVATION CHECKLIST: GROUPS (G) BY TRIAL (T) INTERACTION

Source	SS	df	MS	F	p
Bet. G at T1	3264.28	3	1088.093	9.5736	p < .05
Bet. G at T2	3591.423	3	1197.144	10.53331	p < .05
Bet. T at g1	880.070	1	880.070	7.7433	p < .0125
Bet. T at g2	311.142	1	311.142	2.7376	
Bet. T at g3	44.643	1	44.643	.3928	
Bet. T at g4	928.282	1	928.283	8.1675	p < .0125
ST(G)	2727.739	24	113.6558		

TABLE XVIII

ONE WAY ANALYSIS OF COVARIANCE OF BEHAVIORAL OBSERVATION CHECKLIST (BOC) POST-TEST BY GROUP WITH BOC PRE-TEST

Source of Variation	SS	df	MS	F	p
Covariates Pre-BOC	2153.874	1	2153.874	5.522	p .026
Main Effects Group	2802.056	3	934.019	2.395	p .093
Explained	4955.930	4	1238.982	3.176	p .032
Residual	8971.461	23	390.063		
Total	13927.391	27	515.829		

Adjusted Post-Test Means for BOC from Analysis of Covariance

EMG	Beta	Alpha	Control
36.06	62.34	56.25	57.91

TABLE XIX

T-TEST VALUES ON PAIRWISE COMPARISONS AMONG THE FOUR ADJUSTED POST-TEST MEANS FOR BOC

Groups	F	p	df
EMG vs. Beta	-4.6117	p .001	24
EMG vs. Alpha	-3.5430	p .01	24
EMG vs. Control	-3.8343	p .001	24
Beta vs. Alpha	1.0687	NS	24
Beta vs. Control	.7774	NS	24
Alpha vs. Control	.2913	NS	24

post-mean change scores for the BOC were found to be nonsignificant. The mean and standard deviation for the three groups on the change scores were as follows: EMG $\bar{x}$ = -4.75, S.D. = 5.56; beta $\bar{x}$ = -1.17, S.D. = 10.9; and alpha $\bar{x}$ = -3.75, S.D. = 14.04. Change scores for the control group were not reported because of the poor return on the post-test questionnaires (Table XX).

TABLE XX

T-TEST VALUES ON COMPARISON OF PRE-TEST WERRY-WEISS-PETERS ACTIVITY SCALE (WWP) SCORES FOR TREATMENT GROUPS

Groups	Mean	SD	t	p	df
EMG	21.00	18.78	2.236	NS	3
Beta	21.00	19.19	2.682	NS	5
Alpha	16.71	17.1	2.589	NS	6
Control	19.5	15.79	2.470	NS	3

Number of Returned Pre- and Post-Questionnaires (WWP) for Each Treatment Group

Group	Pre	Post
EMG	4	5
Beta	6	7
Alpha	7	4
Control	4	3

CHAPTER V

DISCUSSION

This study proposed to clarify some discrepancies in research found on the effects of biofeedback training on behavior and cognition. Hypotheses were designed to answer the following questions: Will biofeedback treatments (i.e., left occipital-temporal EEG alpha, down-training arousal; EEG beta, up-training arousal; and frontalis EMG, relaxation) be learned by hyperactive adolescents? What differential effects will the specific biofeedback training groups produce before and after treatment? Does EEG training manifest a direct effect upon behavior and inversely affect cognition such that EEG alpha training will enhance cognition but have no effect on behavior, while EEG beta training effects no change on cognition and improve behavior? What is the total effectiveness of the EMG biofeedback, a symptomatic treatment?

To answer the first question, training effectiveness for the three treatment groups was assessed on hyperactive adolescents. For the two left occipital-temporal EEG (alpha and beta) biofeedback treatment conditions, the study proposed that subjects who learned to control beta brainwaves would increase EEG frequency and decrease EEG amplitude activity in relation to the alpha group, and the controlling of alpha rhythm would be indicated by decreased EEG frequency and increased EEG amplitude in relation to beta across both trials and sessions. The results showed a higher frequency mean for the beta training group over the alpha

training group. While no changes from session to session were found in frequency measures, the beta group showed a marginally significant increase in left occipital-temporal EEG frequency across the six training trials within each session in comparison to alpha training. Therefore, EEG training was effective in producing a higher cortical arousal state in the beta subjects over that of the alpha subjects, which partially confirms the above hypothesis. In addition, the results are consistent with Murphy and Darwin's (1975) findings for left occipital-temporal EEG frequency. The lack of across sessions changes in EEG frequency may be accounted for by the large amount of intragroup variability on this measure. This lack of session to session change in frequency was found also by Stroebel et al. (1976), who showed no across sessions changes in frequency after 20 sessions of EEG feedback in adults. In addition, the latter finding might best be explained by the fact that biofeedback was specifically contingent upon changes in frequency and not amplitude.

For the third treatment condition, the study proposed that the EMG relaxation group would learn to control EMG frontalis muscular tension level by reducing the EMG integrated microvolts across both sessions and trials. The EMG group results indicated that there was stronger evidence of session to session reductions in the EMG integrated microvolts than across trials reductions. Thus, hyperactive adolescents successfully learned to relax by reducing their frontalis muscular tension levels.

In summary, the EMG relaxation training procedures produced clear cut evidence of effectiveness for both within and across sessions reduction in muscle tension. The differential EEG biofeedback also showed evidence of training effectiveness, but this was limited to EEG frequency

changes within sessions. Apparently the EMG training was more effective in generalizing its effects across sessions than was the EEG training.

Baseline readings of the trainees' EEG frequency and amplitude and EMG integrated microvolts were taken immediately before each testing session to provide evidence of changes in EEG and EMG measures that occurred as a result of treatment. The nonsignificant change between the pre- and post-treatment for these three physiological baseline range values implied that variability of these measures were not affected by treatment. Also, EEG amplitude mean baseline values showed no change pre to post for the four groups. The absence of change on all range measures and the amplitude means might be best explained by the specificity of biofeedback training. Physiological variability was not the measure fed back to the subjects; biofeedback was based only on physiological level. EEG amplitude was not a response on which feedback was contingent.

Since EEG beta activity is associated with a high brain arousal level, the beta group frequency training was expected to increase in frequency between the two baseline readings. Only the beta group's pre-post change score mean (1.4285) showed an increase in frequency baseline mean. All other change score baseline means showed a reduction. However, the alpha group showed no evidence of reduction on this measure. Their change score was equivalent to the control group, but EMG relaxation did show a reliable reduction on change in frequency mean baseline from before to after treatment. Therefore, the beta group's training did transfer to the post-test situation, but the alpha group's training did not. The EMG relaxation which had showed the strongest training effects resulted in the only reliable reduction in EEG baseline frequency

on the post-test. Therefore, to differentially change the EEG frequency measure in hyperactive adolescents, up frequency training and frontalis EMG reduction appear to be the best training procedures.

Turning to the results on changes in baseline muscular tension, only the EMG relaxation group showed no reliable change on muscle tension. The other three groups all showed significant reductions in frontalis EMG with the greatest reduction for the beta group, then the alpha group and the least reliable reduction for the control group.

This order of degree of frontalis reduction is the exact opposite as that of the degree of frequency reduction for the four groups. EEG beta training produced frequency increase and the greatest EMG reduction. The EMG relaxation training produced the greatest frequency decrement but the least EMG reduction. Alpha training and the control group were second and third in order of frequency increment and EMG decrement. One might interpret that biofeedback training can effect a reciprocal relationship in arousal between frontalis muscular tension and the cortex in hyperactive adolescents.

Assessing the effect of biofeedback training on cognition, the EEG alpha and EMG relaxation groups were expected to show a greater enhancement in arithmetic performance on the WRAT, and on the WISC-R subtests, Digit Span and Coding, than the control and beta groups. Digit Span was taken as an index of the subject's attentional span, and Coding as an index of concentration. EMG biofeedback relaxation condition improved attention span as shown by a significant increase in the WISC-R Digit Span subtest over the other three treatment groups. Unilateral EEG alpha biofeedback training showed more improvement in reading abilities over arithmetic and spelling than EMG, EEG beta and control. Unilateral EEG

beta biofeedback training produced no effect on cognitive performance.

Did EEG training inversely affect cognition such that EEG alpha training enhanced cognition, while EEG beta effected no change in cognition? The results partially support the above position in that the EEG beta training produced no effect on cognition and occipital alpha training increased the reading level for hyperactives. Consistent with these findings, alpha training (according to previous research by Nall, 1973) has produced success in reading. One contrary finding reported that occipital alpha training enhanced arithmetic performance (Murphy and Darwin, 1975). However, the latter study was not based on hyperactives. Furthermore, the assumption to explain the second paradox which previously stated that further training the hyperactive's brain activity level to a lower arousal state, should worsen his cognitive functioning, was not fully met as a result of this study's findings on cognitive change. While only the alpha group showed a reliable increase in WRAT reading scores, all cognitive areas showed some indication of improvement for the treatment groups, except for the arithmetic area in which performance decremented across all treatment groups.

Since research reports by Budzynski and Stoyva (1969) and Green, Green and Walter (1970) considered EMG as an effective method to produce relaxation and reduce anxiety, electromyographic training in hyperactives should enhance attention and concentration ability thus improving arithmetic performance. Only attention span improved under EMG relaxation training. Neither EMG nor alpha groups' concentration ability and arithmetic performance became better. Failure of improvement in both cognitive areas, attention and concentration (qualities required for

arithmetic tasks), may be a possible explanation for no improvement on WRAT arithmetic.

An alternative explanation for the insignificant improvement in cognition, specifically WRAT arithmetic for both the alpha and EMG group, may be related to the assymetrical functioning of the brain via unilateral EEG biofeedback training. A considerable amount of positive evidence has been acquired (Kimura and Doreen, 1973; White and Murray, 1969). This evidence suggests that even though there appears to be a general interaction that exists between cortical hemispheres, the left occipital temporal lobe dominates the right parieto-occipital temporal lobe in the perception or apprehension of verbal material, whereas the right hemisphere dominates the left temporal lobe in the perception of nonverbal or visuo-spatial tasks. Therefore, unilateral EEG training of the left occipital temporal lobe would most likely have shown improvement in WRAT reading rather than WRAT arithmetic because arithmetic may have a greater visuo-spatial component than reading.

Teacher observation of classroom behavior and parent's observations at home were behavioral indices to measure the occurrence of change in activity level for subjects following biofeedback treatment. The EMG training produced a significant reduction in teacher rating of hyperactivity. Both the alpha and beta groups were not significantly different from the control. But the EEG beta group's behavior actually did worsen from pre- to post-test.

The parents' ratings on subjects' pretreatment activity levels showed no significant differences among the four biofeedback treatment groups. Over the experimental period, the change in behavior reduction was evaluated for only three groups (EMG, EEG beta and alpha) because

of the lack of full cooperation from the parents on the returned post questionnaires. Looking at the numerical mean differences on pre-post change scores, the behavior reduction pattern results from parents' ratings were similar to teachers' behavior ratings. EMG group's reduction (-4.75) was more than alpha's (-3.75) and beta's (-1.17) decrease, with beta showing less improvement than the other two groups. On the other hand, the alpha group's behavior at change substantiated the study's expected outcome by not exhibiting a significant effect on behavior improvement.

The behavior indices (the Behavioral Observation Checklist and the Werry-Weiss-Peters Activity Scale) were expected to show greater improvement for the EMG and EEG beta groups over the control and alpha groups. On the contrary, EMG and the control received high ratings of behavior improvement. Teachers and parents rated the EMG group high. This result contradicts recent electroencephalographic research findings by Lubar and Shouse (1976) and Murphy and Darwin (1975) which support the assumption that beta frequency training is associated with behavioral improvement, such that motor inhibition and socio-emotional areas are enhanced. In like manner, as stated previously, hyperactives are associated with a hypoaroused or underaroused CNS (Grunewald-Zuberbier, Grunewald and Rasche, 1975). Therefore, paradox two (as previously stated), suggests that hyperactives with hypoaroused brain activity, trained further to a lower state, should increase their hyperactive behavior. Murphy and Darwin's (1975) findings were consistent with the paradox. However, the present result found alpha training not worsening behavior but affecting no significant change in behavior overall.

Did EEG training manifest a direct effect upon behavior such that alpha training effected no change in behavior while beta training improved behavior? Results report that EEG beta training worsened rather than improved behavior, alpha training caused no effect upon behavior and EMG relaxation training enhanced behavior in hyperactives.

Practical Implications

This study suggests practical implications for the educational training of hyperactive adolescent students. The basic learning principle used in biofeedback training may be academically applied to facilitate a positive learning environment for these students in the classroom.

A continuous reinforcement schedule is built into the biofeedback training system so that an immediate sensory stimulus is fed back to the subject in order to make him become aware of his desired behavior. He eventually learns to develop control of the feedback and thus the specified behavior through exploring various internal strategies. The reward is based on a self-control mechanism (i.e., voluntary control over the function).

The above concept of a voluntary feedback system can be particularly applicable for the hyperactive adolescent in shaping academic behavior in the classroom, by allowing the student the opportunity to develop strategies for learning curriculum materials through trial and error of continuous correction of error until he masters the skill or concept. Moreover, it must be noted that the learning material should be programmed so as to minimize the student's opportunity for error, subsequently producing positive continuous reinforcement (Freiberg and

Douglas, 1969). Such a schedule will also serve to reinforce attention in the student and act as a continuous counterforce to his maladaptive responses such as distractability, overanxiousness and physical restlessness. The student's attention would thus be drawn back to the task by the immediate appearance of a visible, audible or tangible reinforcement which would be in view of the student until he makes the response (Freiberg and Douglas, 1969).

The student will also become aware of his strengths and weaknesses by measuring himself continuously against levels of cognitive difficulty. At the same time he is gaining a sense of self-accomplishment and self-esteem through taking on self responsibility and setting his own expectations for academic learning.

Thus, the student is most likely to begin learning because by so doing he can satisfy his need in building and forming attitudes of self confidence. Therefore, one may expect him to become "transfer-conscious" and begin to apply what he knows since all education is predicted on the assumption that learning experiences will transfer or generalize to further learning (Mouly, 1968). Consequently, this approach should facilitate learning and create a relaxed atmosphere for the student because it focuses attention, arouses interest, reduces boredom, and is self-paced for the hyperactive adolescent.

Since research indicates that hyperactivity has been so ambiguously and incorrectly used with regard to diagnosis and definition, this study took a more conservative approach in identifying and screening hyperactive subjects which subsequently led to a diminution in sample size. However, a less stringent behavioral definition would have increased the likelihood of a greater sample size, greater statistical power, and

consequently converted the marginal findings into definitely significant results. Therefore, an alpha level of .10 might be a more appropriate criteria of statistical significance for this study, given the reduced cell size of only seven subjects. This would permit clear interpretation of some of the physiological findings and the EMG group's reduction of classroom hyperactivity.

Summary

With respect to the left occipital-temporal EEG beta training, the results indicated that the beta group was effective in producing a higher cortical arousal state in its subjects. The beta group's training transferred to the post-test situation and showed the strongest reduction in frontalis muscular tension. Furthermore, EEG up training produced no effect on achievement performance and actually increased hyperactive behavior over the experimental period. Consequently, one might infer that left occipital-temporal EEG beta training in hyperactive adolescents enhances cortical arousal and reduces muscular tension in the frontalis, but produces no benefits in cognition or classroom behavior.

The left occipital-temporal EEG alpha treatment decreased cortical arousal in each session, but showed no evidence of producing a reliable reduction over time due to alpha training. However, alpha training improved reading levels in hyperactive adolescents at the end of the treatment.

Experimental data further showed that EMG treatment produced a strong reduction in frontalis muscle tension during training; but, during the post-test the EMG group revealed the least reduction in muscular tension relative to the other three treatment groups, and the greatest

reduction in EEG frequency. Moreover, the EEG relaxation condition improved attention and classroom behavior in hyperactive adolescents. Consequently, EMG appears to be an effective treatment technique for improving cognition and behavior in hyperactive adolescents.

Therefore, the following will integrate the above findings to answer the initial questions of the study. First, what specific training effects did biofeedback treatments (left occipital-temporal EEG alpha and EEG beta and frontalis EMG) have on these adolescents? The EMG relaxation training produced clear cut evidence of effectiveness for both within and across sessions reduction in muscle tension. The differential EEG biofeedback also showed evidence of training effectiveness, but this was limited to EEG frequency changes within sessions. Evidence revealed that the EMG training seemed to be more effective in generalizing its effects across sessions than did the EEG training.

With respect to the second inquiry, what differential effects did the specific biofeedback training groups produce following treatment? To differentially change the EEG frequency measure in hyperactive adolescents, up frequency training and frontalis EMG reduction are the best training procedures. The assumption that biofeedback training can effect a reciprocal relationship in arousal between frontalis muscle tension and the cortex in hyperactive adolescents was upheld.

Did EEG training inversely affect cognition such that EEG alpha training enhanced cognition, while EEG beta training effected no change in cognition? EEG beta training produced no effect on cognition and occipital alpha training increased the reading level at the secondary level for hyperactives. Moreover, did EEG training manifest a direct effect upon behavior such that alpha training effected no change in

behavior while beta training improved behavior? Results indicated that EEG beta training actually worsened (though nonsignificantly) rather than improved behavior. Alpha training caused no effect upon behavior and EMG relaxation training enhanced behavior in hyperactives.

In regard to the fourth question, what was the total effectiveness of the EMG biofeedback symptomatic treatment? Overall, EMG enhanced behavior and cognition on attention span in these adolescents.

To comment on the paradox stated earlier, given that the hyperactive child's brain activity is in a hypoaroused state, further training the brain to a lower arousal state should worsen his behavior and cognitive functioning. However, studies by Nall (1973) and Murphy and Darwin (1975) did not uphold this expectation. The results from this study indicated that down training (alpha) the brain improved rather than worsened one area of cognition functioning (WRAT, reading) and had no effect on the behavioral aspect of hyperactive adolescents. EMG relaxation training showed a marked decrease in cortical arousal on the post-test, and produced improvements in cognition on attention span and classroom behavior. This result is also consistent with Braud, Lupin and Braud's (1975) findings reported on EMG relaxation training; both cognition and behavior improved in hyperactive children. Training the brain to a higher arousal state (beta) did not improve cognitive functioning or behavior. Instead, EEG beta training worsened behavior. Therefore, the paradox that decreasing cortical arousal enhances, rather than worsens, cognition and behavior in hyperactive students, is upheld. Further research is needed to understand this paradox. The practical implications for the educational training of hyperactive adolescent students, as a result of the study, have been discussed.

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APPENDIXES

APPENDIX A

LITERATURE REVIEW

Hyperactivity

Definition and Diagnostic Terms

Hyperactive behavior investigated in the present study falls along a continuum in the literature research. A myriad of terms and associated symptoms to describe the concept of hyperactivity are used depending upon the context. Hyperactivity may be viewed as only one symptom in a constellation of symptoms constituting a syndrome, or as a primary disorder coexisting with other characteristics.

Hyperactive children are known by many different diagnostic names. Labels such as "hyperkinetic child" or the "hyperactive child" have appeared frequently in educational, scientific, and general literature since the 1950's. These labels have been overused, ambiguously used, and incorrectly used. Ambiguity and exaggeration have resulted from lack of clear definition in description and diagnosis of these label terms (Renshaw, 1974). Most of the emphasis on the many diagnostic names either differ in the aspects of the children's behavior or differ in theories of the origin of hyperactivity.

Some synonyms of hyperactivity are "maturational lag," hyperkinetic," "immaturity of the nervous system," "hyperactive child," "impulsive disorder," and "perceptual-motor problems." Two names often misunderstood by parents are "minimal brain dysfunction" and "minimal cerebral dysfunction." Finally, two fairly common names are usually incorrect: "minimal brain damage" and "minimal brain injury" (Wender, 1973). The terms hyperactive and hyperactivity refer to all these conditions.

The first diagnostic term, Minimal Brain Dysfunction (MBD), describes the phenomena of disturbances of cognition, perception, and learning, which is commonly associated with hyperactivity and inattentiveness. A behavioral difficulty is sometimes added as a diagnostic feature of MBD (Clements, 1966).

Secondly, "Minimal Brain Damage" is a term attempting to describe presumptive underlying pathology within the brain of the child which might have occurred in utero, during delivery, or during early life (Renshaw, 1974).

Minimal brain dysfunction differs from minimal brain damage in that MBD attempts to describe the functioning deficiency between thought processes and learning and motor execution. On the other hand, minimal brain damage implies a clear knowledge that there is indeed damaged brain tissue, which at this point is merely speculative, or sometimes hypothesized from clinical findings where neurological signs are detected. The implications may be that dysfunction can occur without actual tissue damage, or that if there is tissue damage, it is not massive since there are no "hard" neurological signs present in most cases (Renshaw, 1974).

Ounsted (1955), in discussing his study with epileptic children, listed the following signs manifested in the behavior of "brain injured" children: (1) distractibility, (2) short attention span, (3) wide scatter on the test results when given formal intelligence tests, (4) fluctuation of mood with euphoria as the abiding background, (5) aggressive outbursts, (6) diminution or absence of spontaneously affectionate behavior, (7) lack of shyness, and (8) lack of fear.

Jasper, in 1938, published the first report demonstrating that in a group of disturbed nonepileptic (i.e., psychogenic origin) children a substantial proportion had an abnormal EEG.

Finally, Clements and Peters (1962), reporting on brain dysfunction of school age children, listed 10 common characteristics: (1) hyperactivity, (2) specific learning defects in the presence of normal intelligence, (3) perceptual motor deficits, (4) impulsivity, (5) emotional instability, (6) short attention span, (7) coordination deficits, (8) distractibility, (9) equivocal neurologic signs, and (10) frequent abnormal EEG.

Conclusively, the similarities between the list of Clements and Peters' MBD children and Ounsted's brain injured children with epilepsy (i.e., children with proven organic brain disease) are striking. Thus, similarities between behavioral deviations exhibited by children with known brain malfunction (brain damaged or dysplasia) and a large subgroup of children with problems of behavior or learning or both led to the concept of "minimal brain dysfunction." This concept assumes that these latter children have some dysfunction of their brain that is not severe enough to be manifested by the usual "hard" neurological disturbances (such as motor weaknesses, spasticity, abnormalities in sensation, or pathologic reflexes), but is marked rather by minimal "soft" neurologic distrubances (such as clumsiness, nystagmus, mixed or confused laterality) (Gross and Wilson, 1974).

Therefore, at present it is not known if the subgroup of hyperactive children who do have supposedly brain damage are subject to a developmental cause that is different from that experienced by other hyperactive children.

The third diagnostic term is "hyperkinetic syndrome." It is a medical label sometimes used synonymously with "hyperactivity." Hyperkinetic Syndrome (HK) is a collection of clinical behavioral manifestations, forming a clinical entity with a wide spectrum from mild to severe (Renshaw, 1974). Furthermore, hyperkinesis is commonly noted as one of the cardinal characteristics of MBD. The terms "hyperkinetic impulse disorder" and "hyperkinetic behavior syndrome" are among the many labels used to designate this condition (Kenny and Clemmens, 1975).

The Diagnostic and Statistical Manual of Medical Disorder (American Psychiatric Association, 1968) gave the following definition under 308.0, Hyperkinetic Reaction of Childhood (or Adolescence):

> This disorder is characterized by hyperactivity, restlessness, distractibility, and short attention span, especially in young children; the behavior usually diminishes in adolescence. If this behavior is caused by organic brain damage, it should be diagnosed under the appropriate non-psychotic Organic Brain Syndrome (p. 50).

This definition did not clearly differentiate from those children with other behavior disorders who may also show the symptoms of hyperactivity. The term "hyperactivity reaction" is used to describe the behavioral component of the syndrome--namely the hyperactivity, distractibility, short attention span (Renshaw, 1974).

Wender (1973) refers to a combination of problems that are seen among hyperactive children as a "syndrome" in medical terminology. A syndrome is a group of difficulties that tend to clump, cluster, or move together. It is characteristic of medical syndromes for a given individual not to have all the problems associated with the syndrome. The term "syndrome" according to Safer and Allen (1976), however, limits its application to hyperactivity. The major reason for this is that

hyperactive children share no specific learning or perceptual-cognitive problem. On the other hand, a child could qualify as learning disabled for inclusion in the MBD category with perceptual-cognitive problems in any of a number of areas.

Peters et al. (1973) illustrate and list characteristics for three types of disorders: (1) Pure Hyperkinetic Type, (2) Mixed Types, and (3) Pure Learning Disability Type. They specify that a number of severe (Pure Hyperkinetic) cases do exist but they are rare, although moderate to mild hyperkinesis is fairly common. They say that one will not mistake the severe cases of hyperkinesis--those that justify the term hyperkinetic syndrome. But, it is possible to overlook some moderate and all of the mild cases, especially if judgments of the child's behavior were made only in an office setting. On the contrary, Renshaw (1974) declares there is no such specific entity as the "hyperkinetic child."

The fourth diagnostic term, "Hyperactivity," is defined by Safer and Allen (1976) as a long-term childhood pattern characterized by excessive restlessness and inattentiveness. It is a developmental disorder which begins in early to mid-childhood (ages two to six), and begins to fade during puberty. During childhood, the pattern is consistent year after year (i.e., it is not observed for one year but absent for the next two years). The term "hyperactivity" is somewhat limited in itself. Hyperactive children have no more total daily body activity than nonhyperactive children. In many settings, they have a normal activity level. However, when they are expected to sit quietly at their seats and pay attention in the classroom, they are unusually active. Thus, a better way of viewing the activity problem these

children have is to state that they have difficulty modulating their activity level, particularly when they are expected to perform an abstract task (Safer and Allen, 1976).

The clinical signs and symptoms of developmental hyperactivity, unlike the "hyperkinetic behavior," have only a modest degree of inherent unity, but not enough at this time to technically merit the tag syndrome. The major reason for this is that hyperactive children share no specific learning or perceptual-cognitive problem (Safer and Allen, 1976).

Physicians who have treated hyperactive children over a period of years have repeatedly noted that the problems tend to change, become less severe, and to disappear with age. It is this sort of progress that has caused some physicians to label the problem a "developmental lag" (Wender, 1973). The only necessary feature of the hyperactive pattern is developmental hyperactivity. Hyperactivity is best determined by history. It is the persistent pattern of excessive activity in situations requiring motor inhibition. Persistent means extreme (i.e., the most restless three to five percent) (Safer and Allen, 1976).

Hyperactivity is most clearly brought out in the classroom, but it is also notable at the meal table, during visiting, in church, and whenever attention and the sedentary position are expected. The child may be hyperactive in a gross way, as when he leaves his seat constantly to meander around the classroom. Or, he may be able to stay in his seat (e.g., while watching cartoons on television) but he will show his restlessness by fidgeting constantly. Both qualify as hyperactivity (Safer and Allen, 1976).

Signs and Symptoms

The syndrome of "Hyperkinetic reaction of childhood" seems to be a recognizable entity in a sense. When its signs are very gross, the problem is easily defined by age two years (with development of not only walking, but also of running skills) according to Renshaw (1974). She states that usually by around five years, expectable age-related "normal" hyperactivity should begin to noticeably decrease. Attention and concentration improve to where the child participates in games with peers, watches TV programs that interest him, finishes a meal (with one or two interruptions), and entertains himself up to 30 to 60 minutes at a time. How, then, to differentiate normals from hyperkinetic children?

Recognition of hyperkinetic reaction is not difficult when, by the age of five years, at least half of the following signs are persistently and recurrently (not occasionally) present:

1. Ceaseless, purposeless activity
2. Short attention span
3. Highly distractible
4. Highly excitable; labile emotions (from tears to laughter in minutes)
5. Uncontrolled impulses (talks, hits, leaps, etc.)
6. Poor concentration (overincludes all stimuli, unable to screen out or discriminate)
7. Headless of danger/pain
8. Poor response to reward/punishment
9. Destructive; aggressive; lies; steals; has temper tantrums
10. Constant clash with environment (including pets)
11. Accident-prone; clumsy; poor motor-co-ordination
12. Speech problems
13. Strabismus (squint)
14. Perception difficulties; audio-visual problems
15. Mixed L-R dominance (ex: R-handed/L-eyed/R-legged)
16. Irregular developmental milestones (example: no crawling then sudden walking; no babbling then sudden sentences)
17. 'Untidy' drawing, coloring, handwriting (overshooting of lines; unable to draw parallel lines; unable to stay within boundaries)

18. Nothing completed spontaneously, needs excess reminders (eat/dress/task)
19. Inability to cope with phase-related activity (example: collaborative games, riding bicycle, gym, etc.)
20. Poor socialization; quarrelsome; no respect for needs or property of others; friendless; disruptive
21. Sleep disturbance
22. Needs constant supervision (Renshaw, 1974, pp. 82-83).

The cluster of many signs in the child is essential for the diagnosis. From this listing, many variants of the hyperkinetic reaction of childhood are to be expected and indeed are clinically seen. Some hyperkinetic children are well-coordinated. For them sports provide an excellent outlet for their excess activity. Many have no sleep disturbances. Some children with hyperkinetic reaction are exceptionally bright, but are underachievers due to their inability to sustain attention long enough even to be tested or taught. With the help of approiate medication, they may be assisted to settle down, to learn, and do very well academically. Renshaw (1974) feels that if professionals could clearly describe both the behavioral and functional aspects of the hyperkinetic patient, it would enrich the dimensions of understanding him, as well as contribute to cross-discipline comprehension and collaboration. If a child with hyperkinetic reaction shows, in addition to the hyperkinesis, a specific learning diability such as dyscalculia or visual-perceptual difficulty, or poor audio-visual-motor coordination, of sufficient severity to impede functioning, such diagnosis should be carefully added.

Renshaw (1974) states that diagnostic clarity is essential in management; thus, a differentation of hyperkinetic reaction from other conditions should be executed. Hyperactivity is to be distinguished from the restlessness of anxiety states or reactive behavior disorders by its chronicity and by the absence of a clear onset (Werry, 1968).

According to Safer and Allen (1976) hyperactivity is the essential feature of the hyperactive (developmental) pattern. Parents often report that the child was "different" from the beginning of his life. Frequently, such infants are restless and have feeding problems and "colic." They also often have sleeping problems of various sorts: some children fall asleep late and with difficulty, awaken frequently, and arise early; others fall asleep orofoundly and are hard to arouse (Wender, 1973).

As the child grows from an infant to become a toddler, and later grows older, he is incessantly in motion, driven like a motor, constantly fidgety, drumming his fingers, shuffling his feet. He does not stay at any activity long. He pulls all his toys off the shelf, plays with each for a moment and discards it. He cannot color for long. He cannot read to himself without quickly losing interest. Of course, he is unable to keep from squirming at the dinner table; he may not even be able to sit still in front of the TV set. At school his teacher relates that the child is fidgety, disruptive, unable to sit still in his seat; that he jostles, bothers, and annoys his fellow pupils; and that he gets up and walks around the classroom, talks out, and clowns (Wender, 1973). Sometimes the hyperactive child is as overtalkative as he is overactive, talking as ceaselessly as he moves.

It is important to emphasize that what is different about the hyperactive child is not his level of activity while at play. What is so different about the hyperactive child is that when he is requested to turn off his motor, he cannot do so for very long. However, it is to be emphasized that the hyperactive child need not always be moving. Sometimes he can sit relatively still. For whatever reason, this is most apt to occur when he is getting individual attention (Wender, 1973).

There are two additional points to be established about hyperactivity: the first is that not all hyperactive children are overactive, and the second point is that the hyperactivity is often the first symptom to disappear as the child grows older. Often the other problems persist. Therefore, the fact that a child once was overactive but no longer is does not mean that all the problems are resolved. Many of the other problems may persist and require treatment even though the hyperactivity itself is gone.

Inattentiveness is viewed by Safer and Allen (1976) as the most prominent characteristic of the four major features associated with hyperactivity. Teachers report inattentiveness by these descriptive phrases: short attention span and short interest span. Psychologists say that the child is unable to persist at an abstract task. Parents report that the child does not listen to stories for any length of time and that he frequently changes activities (Safer and Allen, 1976).

Wender (1973) divides this major characteristic into two prominent features: attention difficulty and easy distractibility, that seem to almost always be present in the hyperactive child. She noted that, like hyperactivity, distractibility need not be present at all times. Often when the child receives individual attention he can attend well for a while without being distracted. Different experts like the pediatrician and the psychologist may report that the child was not inattentive during his brief office examination or during the testing examination. They may be correct, but what is important is not how the child can pay attention when an adult is exerting the maximum effort to get him to do so. The question is how well he can persevere in a

task on his own and in this most hyperactive children have considerable difficulty.

In some hyperactive children, the distractibility may be concealed by the ability to stick with a particular activity for an unusually long period of time. Usually it is an activity they choose themselves. Sometimes it is a socially useful one (e.g., reading), and sometimes it is not. The child may seem to "lock on" and be undetachable or unusually persistent. The activity may be repeated in a stereotyped and preseverative manner. Such paradoxical behavior in an ostensibly distractible child may be confusing to a parent, because there is really no satisfactory explanation for this paradox (Wender, 1973).

Another major feature of hyperactivity is a learning impediment. According to Safer and Allen (1976), about one-third of hyperactive children have a prominent learning impairment, and another 40 to 50 percent have a notable academic lag. However, the majority of children with notable academic deficiencies have perceptual-cognitive deficits (Safer and Allen, 1976). A learning disability is usually assumed when there is a clear discrepancy between the child's mental and/or chronological age and his age-expected academic achievement. The learning difficulties of the hyperactive child are usually appraised with respect to the three areas of information processing: receptive, integrative, and expressive. These terms respectively refer to the child's ability to grasp sensory detail, organize this input, and utilize or express this information (Safer and Allen, 1976).

As a rule, hyperactive children with learning impediments have great difficulty grasping abstractions, although they may be successful on concrete tasks. Frequently, they have trouble with phonetics; they can

identify the letters but cannot pronounce them correctly. Their spelling is frequently poor. They often add numbers well on their fingers, but do poorly on paper and pencil subtraction. They may memorize their multiplication tables, but do poorly on division. In effect, they have trouble incorporating new information and applying it in the realm of ideas (Safer and Allen, 1976).

Hyperactivity is not in any way related to mental retardation. Hyperactivity does not affect intelligence as ordinarily defined and measured by intelligence tests. The proportions of the bright, normal, and slow are the same among hyperactive children as among children who are not hyperactive. However, even though as mentioned, that the majority of children with academic deficiencies have certain perceptual-cognitive deficits, not all of the hyperactive children do. Some may have an "unevenness" of intellectual development. Intelligence tests measure abilities and skills in a number of separate areas, such as vocabulary, arithmetic, understanding, memory, and certain forms of problem solving. Usually a child's performance is pretty much the same in each of these separate areas. If a child's vocabulary is normal for his age, his memory and problem solving are usually age-normal as well. Hyperactive children seem more likely to have uneven development. The child may be superior in vocabulary, average in memory and somewhat slow in problem solving. His intelligence, which averages his ability in all these areas, may then be average but he may be advanced in some regards and behind in others. If the school does not make allowances for these inconsistent abilities, the problems of such a child will be accentuated (Wender, 1973).

Behavior problems are the third most common feature of the hyperactive pattern (Safer and Allen, 1976). Misconduct is notable in over 80 percent of hyperactive children. The behavior difficulties occur most prominently in the classroom situation. Teachers report that the child disturbs others, speaks out of turn, makes disruptive noise, and often gets into fights (Safer and Allen, 1976). Most hyperactive children manifest interpersonal behavior that has several distinctive characteristics: (1) a considerable resistance to social demands, a resistance to "dos" and "don'ts," to "shoulds" and "shouldn'ts;" (2) increased independence; (3) domineering behavior with other children (Wender, 1973).

The fourth most common feature of hyperactive children is immaturity. Nearly all hyperactive children operate on a less sophisticated level than do their agemates. This is reflected in their wishes, their choice of younger friends, their interests, their difficulty in coping with environmental changes, their frequent temper outbursts, and their low frustration tolerance. Their drawings of people are simplistic even if one considers and corrects for the visual-motor problems which many of these children have. They have a mild tendency to cry more easily, to persist longer in baby talk, and to be more afraid (Safer and Allen, 1976).

A number of emotional and behavioral features occur often in hyperactive children, but less often than the major features of the disorder. One is impulsivity. This is common in hyperactives. It is apparent in tasks. When the hyperactive child is asked to follow a path on a maze test, he goes headlong into blind alleys without stopping to meditate. Likewise, in a playroom, he darts from one activity to another without

much forethought (Safer and Allen, 1976). Impulsivity is also shown in poor planning and judgment. Hyperactive children show less of these qualities than seems to be age-appropriate. Social-impulsivity--anti-social behavior--is sometimes a problem in hyperactive children (Wender, 1973). Peer difficulties are also fairly common for hyperactives. This is in part because their restlessness bothers their classmates and in part because learning-impaired children generally tend to be unpopular. In games, their low frustration tolerance, impulsiveness, and short attention span adversely influence their ability to cooperate (Safer and Allen, 1976).

Many hyperactive children also have low self-esteem. Low self-esteem particularly characterizes learning-impaired children, so that it is by no means a peculiar characteristic of hyperactivity (Safer and Allen, 1976).

As a group, hyperactive children also tend to have more emotional deviance and anxiety than do nonhyperactive children. The nature of the relationship of these symptoms to hyperactivity is somewhat unclear.

The Etiology of Hyperactivity

The results of many studies designed to determine etiology or the underlying defect of hyperactive children have depended greatly on the definition of hyperactivity.

Werry et al. (1964) using a group of children, ages 7 to 12 who were classified as hyperactive on the basis of past history and sustained hyperactivity, found that there was no significant difference between the experimental and control group on four measures of pre-existent maternal factors (maternal age, ordinal position, birth weight and abortion rate)

or on birth complication (such as prematurity or anoxia) or on EEG ratings.

Campbell et al. (1971) included in their study only those whose chief complaint was hyperactivity from early childhood--these children had cognitive style which made them impulsive and field-dependent.

It is commonly recognized that hyperactive children are fidgety, but Stevens et al. (1970) found that although the hyperactive child was tapping his finger more frequently he could not speed the rate of tapping as much as the normals when reinforced.

Pasamanick (1956) and Clements (1962) found evidence of maternal or fetal difficulties during pregnancy and delivery of children with minimal brain dysfunction which produced hyperactivity.

In 1959, Knobel used a wide variety of tests to find that 40 children showed no positive relationships between organicity and hyperkinesis.

Stewart (1973) determined that the hypothesis of genetic transmission is doubtful since there is no difference in the frequency of hyperkinesis in the family of those who are hyperkinetic than in families of the controls.

Ney (1972), on the basis of their etiology, categorized hyperkinetic children into four types to determine the difference among them. The types were as follows:

1. Genetic (constitutional)--children who were hyperactive from a very early age but where the pregnancy for the mother and the perinatal events for the child were normal.
2. Behavioral (conditioned)--hyperactive children where parents were responding with attention selectively to their active

distracting behavior.

3. Minimal Brain Dysfunction (chemical)--children with early and continuous hyperactivity and histories of abnormal pregnancies or perinatal events.
4. Reactive (chaotic)--children from home environments in which there was little agreement on discipline or where there was considerable marital turmoil.

The results found no significant differences between the four types of groups.

Therefore, the results from such research studies quoted above reflect the research at large on hyperactive children. The organic hypotheses, as well as the psychogenic hypotheses, presently are still unestablished. From a psychiatric view point, a large number of the families of the hyperactive children appear to be abnormal, a surprising number also appear to be essentially normal. Thus, hyperactivity can apparently occur in the absence of any parental abnormality and vice versa. Furthermore, the coexistence of parental psychopathology and hyperactivity in the child can be just as easily encompassed within a genetic hypothesis (Werry, 1968). Thus, since the organic or the developmental etiological bases for hyperactivity are not demonstrable, assuming a causal relationship between the two conditions should be discouraged.

APPENDIX B

HYPERACTIVE SCREENING SCALE

Behavioral Screening Scale

Patient Name _____ Date of Birth _____

Information obtained _____
month day year

Screener's Signature _____

Please check the square that seems most appropriate for each behavior trait.

Behavior Traits	Degree of Activity				
	Not at all	A little bit	Moder-ately	Quite a bit	Extremely
1. Does not complete expected classroom work or project.					
2. Destructive in regard to his/her own and other's property.					
3. Restless or overactive.					
4. Cannot sit still (leaves seat unexcused).					
5. Flits from thing to thing.					

APPENDIX C

BEHAVIORAL OBSERVATION CHECKLIST

Observation of Behavior

Student's Name _____ Date of Birth _____
Last First Middle mo./day/year

Questionnaire filled out by _____ Date filled out _____

Please rate the patient on each of the characteristics listed below on the following scales. Place a check mark in the square that indicates your best estimate of the degree to which the child possess the particular behavior characteristic.

Behavior Traits	Not at all	A little bit	Moder-ately	Quite a bit	Extremely
1. Openly defiant.					
2. Destructive in regard to his own and/or other's property.					
3. Daydreams excessively.					
4. Oversensitive, feelings easily hurt.					
5. Restless or overactive.					
6. Impudent.					
7. Steals.					
8. Difficulty in concentrating.					
9. Specific fears (e.g., of dogs, of the dark, etc.).					
10. Excessive demands for teacher's attention.					
11. Overly serious or sad.					
12. Disturbs others (e.g., teasing, interferes with their activities, provokes others nearby, etc.).					
13. Selfish.					
14. Lies frequently.					

Behavior Traits	Not at all	A little bit	Moder-ately	Quite a bit	Extremely
15. Inattentive to what others say.					
16. Does not attend to class-room instructions.					
17. Quarrelsome.					
18. Shyness, bashfulness.					
19. Makes disruptive noise, humming, tapping, etc.					
20. Excitable, impulsive.					
21. Social withdrawal, prefer-ence for solitary activ-ities.					
22. Acts smart.					
23. No sense of fair play.					
24. Has short attention span.					
25. Becomes easily frustrated.					
26. Sits fiddling with small objects.					
27. Temper outbursts.					
28. Truancy from school.					
29. Does not complete ex-pected classroom work.					
30. Falls apart under stress of examination.					
31. Can't sit still (leaves seat unexcused).					
32. Stubborn.					
33. Gets into fights.					
34. Submissive.					

Behavior Traits	Not at all	A little bit	Moder-ately	Quite a bit	Extremely
35. Flits from thing to thing.					
36. Sullen or sulky.					
37. Profane language, swearing, cursing.					
38. Overly anxious to please.					
39. Teases other children or interferes with their activities.					
40. Tension, inability to relax.					
41. Negativism, tendency to do the opposite of what is required.					
42. Passivity, suggestibility, easily led by others.					
43. Nervousness, jittering, jumpiness, easily startled.					
44. Irritability, hot tempered, easily aroused to anger.					
45. Teacher's estimate of student's school performance a) difficulty with reading b) difficulty with spelling c) difficulty with arithmetic					
TOTAL					

Additional comments:

Adapted from Conners' Peterson-Quay.

APPENDIX D

WERRY-WEISS-PETERS ACTIVITY SCALE

Werry-Weiss-Peters Activity Scale

Student's Name _____ Date of Birth _____

Information obtained _____
month day year

Please check the square that seems most appropriate for each behavior trait. If the particular behavior does not apply do not check the square.

	No	Some	Much
DURING MEALS			
Up and down at table.			
Interrupts without regard.			
Wiggling (twists and turns).			
Fiddles with things.			
Talks excessively.			
TELEVISION			
Gets up and down during program.			
Wiggles			
Manipulates body or objects.			
Talks incessantly (constantly).			
Interrupts			
DOING HOMEWORK			
Gets up and down.			
Wiggles (twists and turns).			
Requires adult's supervision or attendance.			
PLAY			
Inability to play quietly with game, listen to records, etc.			
Constantly changing activity.			

	No	Some	Much
Seeks parental attention.			
Talks excessively.			
Disrupts other's activities.			
SLEEP			
Difficulty settling down to sleep.			
Inadequate amount of sleep.			
Restless during sleep.			
BEHAVIOR AWAY FROM HOME (except at school)			
Restlessness during travel.			
Restlessness during church/movies.			
Restlessness when visiting friends, relatives.			
Restlessness during shopping (includes touching everything).			
SUBTOTAL SCORE	x0	x1	x2

TOTAL SCORE _____

APPENDIX E

NONSIGNIFICANT MEANS

TABLE XXI

MEANS FOR MIXED DESIGN ANOVA (GROUP X SESSIONS X TIME)
ON EEG FREQUENCY TRAINING DATA

Sessions

1	2	3	4	5	6	7	8
10.4673	10.8397	10.5796	10.3827	9.9133	10.1612	9.9592	10.1480

Trials

1	2	3	4	5	6	7
10.2321	10.1786	10.4491	10.4509	10.2411	10.4027	10.1902

Sessions

Beta Group

1	2	3	4	5	6	7	8
11.0980	11.8265	10.9245	10.2041	10.6837	10.9694	10.4694	11.0816

Alpha Group

1	2	3	4	5	6	7	8
9.8368	9.8531	10.2347	10.5612	9.1429	9.3531	9.4490	9.2143

Trials

Session	Beta Group						
	1	2	3	4	5	6	7
1.	10.7143	10.7857	11.5000	11.9286	10.7857	11.4286	11.5429
2.	10.7143	11.7857	12.2143	11.7143	12.2857	12.5714	11.5000
3.	11.0000	10.5714	11.0429	12.0714	10.7143	9.8571	11.2143
4.	10.0000	10.0714	10.2857	9.7143	11.0714	10.4286	9.8571
5.	10.4286	9.6429	10.4286	11.1429	11.3571	10.5000	11.2857
6.	10.5714	10.5000	10.5000	11.0714	10.1429	12.2143	11.7857
7.	9.9286	10.3571	11.7857	10.1429	10.6429	11.3571	9.0714
8.	9.3571	10.7143	10.9286	12.0000	11.2143	11.3571	12.0000

Session	Alpha Group						
	1	2	3	4	5	6	7
1.	9.4286	10.0714	11.0000	8.7143	10.1429	9.3571	10.1429
2.	10.4286	10.3571	9.4286	10.4286	9.8571	9.1857	9.2857
3.	10.2857	10.4286	10.7143	10.7143	10.0714	9.3571	9.9286
4.	10.5714	10.3571	10.0714	10.7143	10.0714	10.7143	10.4286

TABLE XXI (Continued)

Session	Trials						
	Alpha Group						
	1	2	3	4	5	6	7
5.	10.1428	9.2143	8.6429	8.5714	9.1429	9.2857	9.0000
6.	10.0000	10.2857	9.5000	9.5000	7.8571	9.1857	9.0000
7.	10.4286	8.8571	9.6429	9.5714	8.0714	10.5714	9.0000
8.	9.7143	8.8571	9.3571	9.2143	9.4286	9.0714	8.8571

TABLE XXII

MEANS FOR MIXED DESIGN ANOVA (GROUP X SESSIONS X TIME)
ON EEG AMPLITUDE TRAINING DATA

Group	
Beta	Alpha
39.791	42.3036

Sessions

1	2	3	4	5	6	7	8
42.0714	39.9184	35.3980	45.1939	41.1429	40.1020	46.9795	37.5714

Trials

1	2	3	4	5	6	7
43.5714	42.5714	40.3303	40.1964	40.3303	39.7232	40.6071

Sessions

Beta Group

1	2	3	4	5	6	7	8
45.5102	43.6531	28.8163	47.3265	40.9388	35.1633	46.6326	30.2857

Alpha Group

1	2	3	4	5	6	7	8
38.6327	36.1837	41.9796	43.0612	41.3470	45.0408	47.3265	44.8571

Trials

Sessions	1	2	3	4	5	6	7
1.	47.8571	46.8571	44.2857	43.7143	46.4286	45.0000	44.4286
2.	47.0000	44.0000	42.2857	43.2857	40.4286	42.2857	46.2857
3.	29.0000	28.5714	31.4286	27.1429	27.1429	30.7143	27.7143
4.	54.2857	50.7143	49.2857	44.2857	46.4286	43.5714	42.7143
5.	49.4286	48.5714	40.7143	42.8571	35.7143	32.8571	36.4286
6.	35.0000	35.7143	32.1429	34.2857	35.0000	34.2857	39.7143
7.	48.5714	37.8571	45.0000	42.8571	54.2857	43.5714	44.2857
8.	31.7143	31.0000	31.8571	25.5714	32.4286	27.0000	32.4286

TABLE XXII (Continued)

Sessions	Trials						
				Beta Group			
	1	2	3	4	5	6	7
1.	47.8571	46.8571	44.2857	43.7143	46.4286	45.0000	44.4286
2.	47.0000	44.0000	42.2857	43.2857	40.4286	42.2857	46.2857
3.	29.0000	28.5714	31.4286	27.1429	27.1429	30.7143	27.7143
4.	54.2857	50.7143	49.2857	44.2857	46.4286	43.5714	42.7143
5.	49.4286	48.5714	40.7143	42.8571	35.7143	32.8571	36.4286
6.	35.0000	35.7143	32.1429	34.2857	35.0000	34.2857	39.7143
7.	48.5714	47.8571	45.0000	42.8571	54.2857	43.5714	44.2857
8.	31.7143	31.0000	31.8571	25.5714	32.4286	27.0000	32.4286
				Alpha Group			
Sessions	1	2	3	4	5	6	7
1.	41.4386	40.0000	35.0000	35.0000	39.2857	40.4286	39.2857
2.	45.0000	38.1429	35.7143	32.1429	31.1429	34.0000	37.1429
3.	49.0000	45.1429	40.7143	41.4286	40.5714	39.1429	37.8571
4.	45.0000	45.7143	40.7143	42.8571	40.0000	44.2857	42.8571
5.	36.4286	36.0000	39.5714	47.1429	41.7143	44.2857	44.2857
6.	47.1429	46.7143	43.8571	47.2857	45.8571	45.1429	39.2857
7.	46.7143	49.7143	48.1429	48.5714	42.4286	47.1429	48.5714
8.	43.5714	46.4286	44.5714	44.7143	46.4286	41.8571	46.4286

TABLE XXIII

MEANS FOR REPEATED MEASURES DESIGN (SESSIONS X TRIALS)
ON EMG FRONTALIS LEVELS

Sessions	Trials					
	1	2	3	4	5	6
1.	1.60	1.42	1.45	1.15	1.21	.95
2.	1.08	1.16	1.11	.99	.99	.83
3.	1.36	1.22	1.36	1.21	1.10	1.22
4.	.91	.87	.82	.73	.73	.66
5.	1.20	1.08	.81	.90	.76	.75

TABLE XXIV

MEANS FOR MIXED DESIGN ANOVA (GROUP X TIME) ON EEG FREQUENCY BASELINE RANGES

EMG	Beta	Alpha	Control
.40000	.53571	.60714	.53571
Pre-test			Post-test
.43214			.60714

Group	Pre-test	Post-test
EMG	.15714	.64286
Beta	.57143	.50000
Alpha	.57143	.64286
Control	.42857	.64286

TABLE XXV

MEANS FOR MIXED DESIGN ANOVA (GROUP X TIME) ON EEG AMPLITUDE BASELINE RANGES

EMG	Beta	Alpha	Control
5.00000	6.71428	6.42857	6.71428
Pre-test			Post-test
6.46428			5.96428

Group	Pre-test	Post-test
EMG	3.57143	6.42857
Beta	7.42857	6.00000
Alpha	7.14286	5.71428
Control	7.71428	5.71428

TABLE XXVI

MEANS FOR MIXED DESIGN ANOVA (GROUP X TIME) ON EMG BASELINE RANGES

EMG	Beta	Alpha	Control
.76071	.78429	.87186	.76143

Pre-test	Post-test
.85821	.70393

Group	Pre-test	Post-test
EMG	.91429	.60714
Beta	.77143	.79714
Alpha	.87143	.76429
Control	.87571	.64714

TABLE XXVII

MEANS FOR MIXED DESIGN ANOVA (GROUP X TIME) ON EEG FREQUENCY BASELINE MEANS

EMG	Beta	Alpha	Control
10.32143	10.71428	10.14286	9.52143

Pre-test	Post-test
10.26786	10.08214

Group	Pre-test	Post-test
EMG	11.28571	9.35714
Beta	10.00000	11.42857
Alpha	10.14286	10.14286
Control	9.64286	9.40000

TABLE XXVIII

MEANS FOR MIXED DESIGN ANOVA (GROUP X TIME) ON EEG AMPLITUDE BASELINE MEANS

EMG	Beta	Alpha	Control
42.50000	41.25000	33.96428	53.46428

Group	Pre-test	Post-test
EMG	44.64285	40.35713
Beta	49.85713	32.64285
Alpha	35.07143	32.85713
Control	56.14285	50.78571

TABLE XXIX

MEANS FOR MIXED DESIGN ANOVA (GROUP X TIME) ON WISC-R CODING SCALE SCORE

Group	Pre-test	Post-test
EMG	9.14286	10.14286
Beta	5.57143	7.71428
Alpha	8.14286	9.14286
Control	9.71428	10.85714

TABLE XXX

MEANS FOR MIXED DESIGN ANOVA (GROUP X TIME) ON FRONTALIS EMG BASELINE MEANS

EMG	Beta	Alpha	Control
1.80571	1.97785	1.83785	1.64928

VITA

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Master of Science

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