AN APPROACH TO THE PHYSIOLOGICAL MECHANISM UNDERLYING THE EFFECTIVENESS OF EMG BIOFEEDBACK TREATMENT IN HYPER-ACTIVE ADOLESCENTS

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PREFACE

Previous research has taken a symptomatic approach to the treatment of hyperactivity in children whereby the behavioral and cognitive consequence of EMG biofeedback have been examined and reported. However, the physiological basis for such findings has not been investigated. This study investigated the mechanism underlying the effectiveness of EMG biofeedback for the treatment of hyperactivity in adolescent students during contingent EMG biofeedback training with a noncontingent EMG relaxation condition control on a comprehensive profile of physiological, cognitive, and behavioral changes.

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CHAPTER I

INTRODUCTION

The term "biofeedback" may be defined as

> the use of modern instrumentation to give a person better moment to moment information about a specific physiological process that is under the control of the nervous system but not clearly or accurately perceived. Such information about a biologic process is called biofeedback. In the terminology of servosystem, such information has been called feedback (Miller, 1974, p. 684).

In cybernetic terms, such information about bio-physiological processes is called "closed loop-feedback mechanism" (Smith and Henry, 1967).

The electromygraph is a biofeedback instrument that reveals "what a muscle actually does at any moment during various movements and postures" (Basmajian, 1967, p. 22). It feeds back to an individual the ongoing EMG electrical activity generated by his muscle action. The EMG activity is first picked up off the surface of the skin, then amplified and translated into sound patterns and/or visual stimulation which signals increases and decreases in EMG activity (Autogen 1500 Instruction Manual, p. 2).

Three to four decades ago, the electromyogram was used only to assess and diagnose post-traumatic neuromuscular functioning. However, evidence has accumulated within the last 15 years concerning the use of the EMG in a therapeutic manner with auditory and visual displays as the feedback signals. Consequently, several authors have investigated the therapeutic effectiveness of EMG feedback as a treatment

modality of peripheral nerve-muscle damage, Marinacci and Horandè (1960) were among the first to report the therapeutic use of EMG auditory biofeedback for a number of patients with neuromuscular disorders. Jacobs and Felton (1969) used visual EMG feedback to facilitate muscle relaxation in patients who suffered neck injuries. Numerous case studies of voluntary movement disorders (as, for example, hemiparesis) with sustained damage to the central nervous system, report success through the use of visual and auditory EMG biofeedback treatment (Johnson and Garton, 1973; Brudny, Korein, Levidow, Grynbaum, Lieberman, and Friedmann, 1974; Brudny, Korein, Grynbaum, Friedmann, Weinstein, Sachs-Frankel, and Belandres, 1976). In contrast to conventional physical therapy, Swaan et al. (1974) report greater effectiveness following the presentation of auditory EMG feedback in patients who learned to suppress undesirable hyperactivity of the peroneous longus muscle.

In view of the therapeutic effects of EMG biofeedback by Brudny et al. (1974), most of the results to date suggest that this technique is a useful modality for the treatment of central nervous system disorders of voluntary movement of varying degrees (Brudny et al., 1976). Additionally, Blanchard and Young (1974) report that EMG treatment yields the soundest experimental evidence to the application of clinical problems, despite the criticism that this research has received for inadequate experimental procedures.

Since evidence has accumulated through the years that EMG may be used in a therapeutic manner when auditory and/or visual signals derived from the EMG are fed back to the patient with neurological

disorders, investigators are attempting to understand the relationship of proprioceptive feedback to motor physiology. The mechanism of such therapeutic results is still not well understood, however.

Some neurophysiological and cybernetic concepts as they may pertain to possible physiological bases for the mechanism of this technique will be discussed. Granit (1968), a neurophysiologist, defined neuromotor control as the constant interaction of a triad consisting of muscles, their sensory organs, and motor neurons, all carrying out the automatic and volitional commands emanating from supraspinal centers. Behavioral cybernetic (Smith and Henry, 1967) approaches a feedback mechanism in terms of defining it as self modulating, "closed-loop circuits, possessing specific time characteristics" (p. 381), designed to control "organized, patterned behavior" (p. 380), through "motorsensory processes and physiological regulations" (p. 430). Therefore, neuromotor control appears to be "regulated by a hierarchy of multiple cerebral, brainstem and spinal servosystems of afferent and efferent loops" (Brudny et al., 1976, p. 52). Any disturbances or disruptions in these loops represent displacements in the close-loop control of the organized motor sensory processes (Smith and Henry, 1967).

Brudny et al. (1976) have proposed an hypothetical explanation for the therapeutic mechanism underlying (EMG) sensory feedback therapy; stating that with this technique it may be possible to "transfer motor control from indirect exteroceptive feedback to direct internal feedback in patients with disturbed neuromotor control" (p. 52). Consequently, in an attempt to conceptualize the underlying mechanism

for compensation of disruption in servoloop systems and the translation of visual and auditory stimuli by neural mechanism, these investigators apparently adopt Bach-y-Rita's (1972) concept of plasticity of the central nervous system. He defines the term "sensory plasticity" as "the ability of one sensory system to assume the function of another system" (p. 111). He (1972) further speculates that the function of "sensory substitution" is analogous to the neural mechanism underlying the central nervous system's ability to recover or restore a lesion (Bach-y-Rita, 1972). Hence, in order to transfer neural information on the intensity and rate of muscle contraction, the patterns of sensory signals of integrated EMG activity may be utilized as a "substitution source." In other words, the audiovisual signals are a "reflection of the muscle activity which have matching or resonance characteristics of its neural coding" (Brudny et al., 1976, p. 52).

Typically, proprioception and vision provide direct feedback to the nature of movement (Taub and Berman, 1968). However, in the case of sensory feedback, EMG activity derives information from motor control indirectly through the feedback signals (Brudny et al., 1976; Taub and Berman, 1968). Such information derived from the proprioceptive (kinesthetic) system is

> processed in many central nervous system centers interconnected by a neural network of feedback loops, with eventual relay to the sensorimotor cortex and to the descending pathways of the brainstem reticular system (Brudny et al., 1976, p. 52).

Consequently, in an attempt for the organism to compensate or rehabilitate for a disruption in the servoloop, the auditory and/or visual signals would become part of the feedback loop; i.e., as the signals

substitute for the proprioceptive internal feedback loops, they would form an external feedback loop in the disrupted system. This external loop thus would augment or restore the sensorimotor interaction of the closed loop system of voluntary patterned movements (Brudny et al., 1976). According to Brudny et al. (1976), "audiovisual signals can apparently stimulate and interact with the sensorimotor corticothalamic loops and subcortical systems in a manner that results in a selective pattern of motor outflow" when "the appropriate degree of modulation is derived through visual and auditory sensory channels, and translated by the neural mechanisms with the aid of the brain's plasticity" (p. 52). In support of the above hypothesis, microelectrode studies in animals performed by Buser et al. (1963) revealed that "teleceptive" (auditory and visual) stimuli can excite the sensorimotor cortex.

Consequently, the above findings on EMG treatment with neuromuscular disorders and Brudny et al. (1976) hypothetical model for its underlying therapeutic mechanism have led the present author to further investigate the effectiveness of biofeedback, particularly, EMG as a treatment modality for hyperactivity in children.

Biofeedback training has been examined as a therapeutic technique for the treatment of hyperactivity in children. For example, Nall (1973) explored the effects of biofeedback alpha training procedures as an aid in therapy in an attempt to modify inappropriate behavior in children with learning disabilities characterized as hyperkinetic. She used both academic and behavioral indices to assess the training. The final assessment of the study indicated significant improvement in both measurements, in specific cases, but few overall significant effects.

As another treatment for hyperactivity and its behavioral components, the reduction in muscular tension has also been used as a therapeutic intervention. In a case study with a six and a half year old extremely hyperactive male, Braud et al. (1975) used electromyographic (EMG) biofeedback technique. The child was taught to reduce activity and tension during the EMG biofeedback sessions. A great improvement was seen in behavior, psychological test scores, achievement tests at school, and self concept and self esteem, while there was a reduction in "psychosomatic symptomatology" (headaches, allergies, asthma, and running nose). Braud (1978) compared hyperactive children (N=15) and nonhyperactive children (N=15) to determine the effects of frontal EMG biofeedback and progressive relaxation upon hyperactivity and its behavioral concomitants. The hyperactive children consisted of three groups with five students in each one (biofeedback, relaxation, and hyperactive control). Six of the hyperactive children who were on Ritalin were randomly placed (two per group) in each of the three groups. Hyperactive children were found to possess significantly higher muscular tension levels and, in addition, presented more behavioral problems and had lower test scores. Both electromyographic (EMG) biofeedback and progressive relaxation exercises were successful in the significant reduction of muscular tension, hyperactivity, distractibility, irritibility, impulsivity, explosiveness, aggressivity, and emotionality in hyperactive children. The greatest improvement was seen in the area of "emotionality-aggression" (irritibility, explosiveness, impulsivity, low frustration tolerance, and aggression). No differences were seen in the EMG

improvement of drug and nondrug hyperactive children; both made progress under these self control techniques. However, nondrug children made greater improvements in the behavioral area. Both EMG biofeedback and progressive relaxation resulted in improvements on the test scores of hyperactive subjects (Bender-Gestalt, Visual Sequential Memory, Digit Span, Coding).

In view of the other bioelectric feedback treatments with hyperactive children, Sterman (1974) maintains that a relationship exists between the general level of motor activity and the occurrence of the corresponding EEG sensorimotor rhythm pattern. In his study (1974) subjects in whom motor behavior was curtailed, either passively or actively, showed high voltage sensorimotor rhythm (SMR) discharge. Conversely, subjects characterized by decreased thresholds for motor discharge (for example, epileptic and hyperkinetic patients) showed a minimal expression of SMR activity in the EEG. Normal subjects appeared to be intermediate between these extremes in terms of SMR manifestations. He further postulates that the motor mechanisms that underlie the appearance of the SMR may be facilitated by inactivity and suppression of somatic hyperactivity.

Lubar and Shouse (1976) explored the effects of SMR biofeedback training technique's applicability to the problem of hyperkinesis, independent of the epilepsy issue. This study reports one subject's data extracted from an ongoing group of 12 hyperkinetic subjects because he has been in (SMR) training for significantly longer periods of time than the others. SMR training involves conditioning the 12 to 14 Hz rhythm appearing over the Rolandic cortex. The subject was an 11

year, 8 months old male. The subject participated in five consecutive experimental phases (I. No Drug, II. Drug Only, III. Drug and SMR trainingI, IV. Drug and SMR reversal training, and V. Drug and SMR training III), and was involved in several months of SMR training. 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 12-14 Hz activity in the absence of 4-7 Hz slow-wave activity. A substantial increase in SMR occurred with progressive SMR training and was associated with enhanced motor inhibition, as gauged by laboratory measures of EMG and behavioral assessment in the classroom. Opposite trends in motor inhibition occurred when the training procedure was reversed and feedback presentation was contingent on the production of 4-7 Hz in the absence of 12-14 Hz activity.

Patmon and Murphy (1978) compared the effects of three forms of biofeedback training (EEG alpha and beta, and EMG) with a no-training control on a comprehensive profile of physiological, cognitive, and behavioral changes of hyperactive adolescents. While the increase EEG frequency training impacted on these hyperactive students' physiology by increasing cortical arousal and reducing muscular tension, this translated to no benefit in reduction of hyperactive behavior or cognitive improvements. The decrease EEG frequency training affected neither physiology nor behavior, but did provide a specific and meaningful reading enhancement, which replicated a finding by Nall (1973). The EMG feedback relaxation training reduced cortical arousal and improved attention span and classroom behavior on the posttest. Apparently, differential feedback training produced an inverse relationship between

both cortical arousal, and hyperactive behavior and muscular tension. The training (beta) group that produced an increased level of cortical arousal also resulted in a reduction of muscular tension, but increased hyperactive behavior, while the training (EMG) that led to a decreased level of cortical arousal produced no reduction in muscle tension, but improved attention and reduced hyperactive behavior.

While most of the above findings on EMG biofeedback treatment with hyperactivity indicate a reduction in muscular tension and in levels of activity, there were apparently paradoxical findings in the relationship between the physiological EEG and EMG changes and clinical manifestations as a result of feedback conditioning. In one case where EMG technique had been considered purely symptomatic at the behavioral level, Braud et al. (1975) produced both cognitive and behavioral improvement in a hyperactive child. Braud (1978) also successfully reduced muscular tension, hyperactivity and its emotional components, and cognitive attentional measure through the use of this biofeedback technique. On the other hand, inconsistent physiological changes associated with EEG and EMG as a function of biofeedback conditioning were suggested in other studies. An inverse relationship between EEG sensorimotor rhythm (SMR) and motor inhibition and EMG muscular tension was indicated by some authors (Lubar and Shouse, 1976; Lubar and Bahler, 1976; Sterman, 1974, Chase and Harper, 1971). In hyperactive patients, SMR production of 12-14 Hz increased with contingent EEG biofeedback training, and showed a corresponding decrease in EMG tension reduction with motor inhibition. SMR counterconditioning (4-7 Hz in the absence of 12-14) produced the reverse effects (Lubar and Shouse, 1976; Lubar and Bahler, 1976).

Patmon and Murphy's (1978) differential feedback training in hyperactive students apparently produced an inverse relationship between occipital cortical arousal (placements: T3, O1, left-occipital-temporal lobe) and levels of hyperactivity and muscle tension. EMG training appeared to be more effective in generalizing its effect across sessions than were the EEG training groups. However on the posttest, EMG training produced the greastest decrement in frequency and hyperactivity, but the least reduction in muscular tension. EEG training group (beta) produced the greatest reduction in muscle tension, the least reduction in hyperactivity, and increased cortical arousal.

Neurophysiologists now believe that neuroelectric patterns may be associated directly with specific neural processes and are found to modify the behavioral functions they mediate through EEG biofeedback conditioning (Sterman, 1974). Evidence indicates that SMR has a functional significance as a conditioned "central state" because this neural rhythm has been associated with motor inhibition and single-unit activity within the ventrobasal thalamic nuclei (Wywicka and Sterman, 1968; Chase and Harper, 1971; Sterman, 1974). Simultaneous recordings of electrical activity were found in the subcortical structures together with the cortical SMR (Howe and Sterman, 1972).

Therefore, based on all of the above results, one may speculate parallel relationships involved in the physiological mechanisms of the following: EMG training and its impact on the occurrence of the corresponding inverse relationship between EEG occipital cortical arousal and level of motor activity and muscular tension; and EEG

sensorimotor training, a conditioned "central state," and its effect on the somatic inhibition and EMG muscular tension.

In summary, the previously mentioned hypothetical model of an underlying therapeutic mechanism for EMG (sensory) feedback as a treatment modality for patients with neuromotor disorders dealt mainly with retraining (rehabilitation) muscles or relearning patterned movement. Moreover, a symptomatic approach to the treatment of hyperactivity in children has been the consensus of most research with an examination of the behavioral and cognitive consequences of EMG biofeedback. However, the physiological bases for such findings have not been investigated. Neurophysiological and cybernetic concepts such as servoloop feedback mechanisms, plasticity of CNS, sensory motor integration related to EMG (sensory) biofeedback, has not been applied to the hyperactive syndrome in an attempt to understand the mechanism underlying EMG effectiveness in hhperactivity. Consequently, the purpose of this study was to investigate the mechanism underlying the effectiveness of EMG biofeedback for hyperactivity in adolescent students during training.

To test the above hypothesis, two conditions were examined. One treatment condition consisted of a contingent EMG biofeedback training group to determine its effect on cortical arousal. The second condition was a noncontingent EMG relaxation condition, to control for subject expectancy in biofeedback training, and to determine if relaxation and muscular tension reduction was actually learned with EMG biofeedback. Both groups were evaluated on a behavioral, cognitive and physiological profile.

CHAPTER II

METHOD

Subjects

The subjects in this study were 14 (11 males and 3 females) adolescent learning disabled students characterized as behaviorally hyperactive, selected from a population of 108 learning disabled students served by the Oklahoma Child Demonstration Center for grades 6-12 (Table I). The sample of students selected were free of medication for hyperactivity. The learning disabled adolescents were previously assessed and identified as learning disabled through a psycho-educational evaluation (WISC-R, WRAT, and Bender Gestalt Visual-Motor test).

Biofeedback Trainer

There was one experienced trainer to carry out the procedures. This included applying electrodes, conducting the training sessions, and giving instructions to the subjects for the biofeedback electronic devices, the Autogen 1500 Electromygraph and the Electroencephalograph-Bandpass Filter.

Apparatus

The apparatus for feedback training consisted of a comfortable chair and footstool, a feedback electromygraph Autogen System 1500, and an EEG Bandpass Filter. EEG data was gathered via switchable electrode

sets feeding into an Autogen 70 biofeedback monitor with bandwidths (4-8, 8-12, 12-16 Hz) and integrating functions by-passed, producing a raw EEG signal, with -3 cB bandwidth of .2 - 25 Hz. This signal was fed into a 3-stage Bandpass Filter and meter amplifier. The three bands monitored were: 4 to 8 Hz, 8 to 12 Hz, and 12 to 16 Hz; .5 Hz overlap was calculated at the -3 dB portion of the filter slope. The filter sections themselves were staggered -3-pole Band-pass filters utilizing multiple-feedback and state-variable pole sections. Each of the sections composed a second order filter pole. Center frequencies of the filters were 5.447 Hz, 9. 590 Hz, and 13.856 Hz.

TABLE I

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

	Sex		Age		Grade Level	
	Male	Female	Mean	SD	Mean	SD
False EMG (A)	6	1	14.9	1.313	9.1	.834
True EMG (B)	5	2	15.6	1.591	9.4	1.591

For the 4 to 8 Hz bandwidth filter, the first pole was set at 4.213 Hz with a Q of 7 and a gain of 7. The center pole was a Salen-Key multiple feedback pole with a Q of 3.5. The third section was a state variable filter with a gain of 7 and a Q of 7. Overall filter gain was 34 dB with initial filter slopes on the leading and trailing skirts of 42 dB/octave. A f_1 and $f_2 = W_c X$ 10 leading and trailing skirt slopes had diminished to 12 dB/octave. The 8 to 12 Hz and 12 to 16 Hz filters were repetitions of the first filter section. Amplification was provided by low-noise, high-gain integrated circuit op-amps. Following Band-pass filtration, the resultant signals were fed into precision full wave rectifier circuits and averaging filters, which resulted in a DC equivalent of the incoming AC signal. The DC signals were led to respective meters on the panel of the instrument. Although these are RMS equivalents, the meters calibrated to read out at peak-to-peak levels in order to allow equivalent readings from biofeedback monitors calibrated in peak-to-peak divisions.

The three frequency bands were recorded in amplified microvolts and monitored periodically during the training of the two groups of subjects from bilateral occipital and parietal brain locations. Standard Autogen electrode sets were affixed with an elastic headband at O1, O2, and C3, C4 with ground electrodes placed at Fp1 and Fp2 (the left and right temples: eye). For the EMG reduction feedback, silver/silver chloride cup electrodes were attached to the forehead at the standard frontalis placements. The same EMG biofeedback relaxation taped recorded instructions were given to all subjects (contingent and noncontingent groups) to reduce the frequency of the clicks they heard.

The feedback mode used for contingent EMG condition was standard click feedback. These clicks, which are proportional to the average integral microvolts recorded from the subject's frontalis, were delivered through the subject's headphone. The feedback mode used for the noncontingent group was tape recorded false decreasing click tones, delivered through the subject's headphone. For each participant, during a 20 minute session, an average of two EEG frequency readings per band was monitored concurrently with EMG levels every six minutes within a phase (early, middle, and late). Baseline measures for the EMG levels were recorded individually and in combination with the three EEG frequency bands. Shaping procedures were used in both conditions to increase the difficulty of the task as the subjects became more successful at control of their physiology. Also, a monetary inducement was provided such that all subjects were informed that each one would receive 25¢ a session, and a dollar bonus for those who trained all seven sessions consecutively without any absence at the end of data collection.

Measures

The subject participants for this study were identified as hyperactive adolescents by a five point Likert type behavioral screening scale (Appendix B). This screening device consisted of five discriminatory items that represent five major systems (restlessness or overactivity, aggressivity, distractibility or inattentiveness, antisocial conduct, and socio-emotional immaturity) that seemed to persist from childhood through adolescence for the hyperactive child as indicated by Safer and Allen (1975); Malezky (1974); Minde, Weiss, and Mendelson

(1972); Mendelson, Meldelson, Johnson, and Stewart (1971); and Stewart, Pitts, Craig, and Dieruf (1966).

On the screening instruments, only subjects who showed the presence of hyperactive symptoms (mean per item rating of greater than 2.0 on a 1-5 scale) met the criteria of behavioral hyperactivity. 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 a half. 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. Mendelson et al. did a follow-up study later (1971) on hyperactive teenagers between the ages of 12 and 16, which included children of the earlier study diagnosed as hyperactive. In the follow-up study (Mendelson, Johnson, and Stewart, 1971) on hyperactive teenagers, items 3 and 4 on the screening instrument categorized as overactivity or restlessness was still prevalent as a symptom in 71% of the hyperactive teenagers. Items 1 and 5 on the screening instrument categorized as distractibility persisted as a symptom in 77% of hyperactive teenagers. Item 2 of the screening instrument persisted in 52% of hyperactive teenagers as antisocial symptoms at follow-up (Table II).

The behavioral hyperactivity screening device (Appendix B) used in this study measured the overall sample items mean score as 2.62 in comparison to 0.53 score for the nonhyperactive population. The selection criteria for hyperactivity was based on a mean item score greater than

TABLE II

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

	Patients	Controls	Difference
Overactive	100	33	67[a]
Can't sit still	81	8	73[a]
Restless in MD's waiting room	38	3	35
Talks too much	68	20	48
Wears out toys, furniture, etc.	68	8	60[a]
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
Constand 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[a]
Doesn't stay with games	78	3	75[a]
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

[a]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).

2.0 out of a possible score of 4.0. The hyperactivity (HA) students vs. nonhyperactivity (NHA) students of the general population mean score for each item on the screening scale were as follows:

		HA	NHA
1.	Does not complete expected classroom work or project.	2.6	0.71
2.	Destructive in regard to his/her own and other's property.	1.3	.01
3.	Restless or overactive.	2.9	.72
4.	Cannot sit still (leaves seat unexcused).	2.8	.56
5.	Flits from thing to thing.	3.8	.59

Item 2 appears especially helpful in eliminating false positives while item 5 is the best one to eliminate false negations. All items discriminate well between the two groups.

For all subjects, a teacher rating scale and parent rating scale was obtained. The original construction of the teenager's behavioral rating checklist was divided into five categorical factors: I) defiance or aggressivity, II) antisocial behavior, III) inattentiveness or distractibility, IV) socio-emotionality, and V) hyperactivity or overactivity. The items under each category were then randomly arranged for rating checklist symptoms. This 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 0, 1, 2, 3, and 4,

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 supportive evidence that summated ratings used to access the hyperactivity of children were internally consistent and a 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 coefficient of the total summate ratings was .92.

The Werry-Weiss-Peters Activity Scale (WWP) was used for parents to rate the activity level of their adolescent child, and was found to be the widest in use for hyperactivity according to Safer and Allen (1975). This scale is also an effective measurement for evaluating the degree of hyperactivity because it offers a means of quantification of activity level (Werry and Sprague, 1970; Safer and Allen, 1975).

Additionally, the parent rating scale of hyperactivity (r-0.6-0.7) was used in support of the teacher's rating scale (Safer and Allen, 1975). It was also arranged in checklist form so that the parents can check each item of behavior either 1) no, 2) some, 3) much activity. This parental questionnaire (WWP), the Behavioral Screening Device, and the Teacher's Behavioral Observation Checklist (BOC) were used to obtain reliable assessments of the hyperactivity and its associated symptoms.

Experimental Procedures

The experimenter utilized identical procedures for all subjects.

Four rating scales were obtained on all subjects, one for the initial screening of hyperactivity (Behavioral Screening Device), and the other three (BOC, WWP, and STAI-A-State Anxiety Scale) before and after the training program. The resource room teachers filled the screening scale and the questionnaire (BOC) on classroom behavior. The Werry-Weiss-Peters Scale was filled out by the subject's parents. In order to determine if EMG biofeedback training influence subject's subjective anxiety level (Spielberger, Gorsuch, and Lushene, 1970), the State-Trait Anxiety Inventory, A-State (STAI-A-State) scale was administered individually.

In addition, pre and posttests also included the Wide Range Achievement Test (WRAT) to measure the arithmetic performance, and the subtests of the Wechsler Intelligence Scale for Children-Revised (WISC-R); the subtests Digit Span and Coding were measures for attention span and concentration ability, respectively. These measures were administered individually.

Three frequency bands (theta 4-8, alpha 8-12, and beta 12-16) were monitored bilaterally from occipital (O1, O2) and parietal (C3, C4) areas through the use of an EEG Band-pass Filter. For the contingent EMG condition the biofeedback activity from the frontalis (forehead) was monitored through the use of the Autogen 1500. The frontalis (forehead) was selected because its tension level is believed to be a good index of general physical and mental activity (Budzynski and Stoyva, 1973). The second group received false decreasing tones through headphones. EMG average integral microvolts were also recorded on the noncontingent EMG group.

Each subject participated in seven, 20 minute sessions, consisting of 12 ninety second trials per training session. Baseline measurements for the EMG levels for frontalis muscles were recorded individually and in combination with the three EEG frequency bands, respectively, theta (4-8), alpha (8-12), and beta (12-16), based on three 10 second ratings.

The brain location measurements were counterbalanced to control for order-of-presentation effects (Table III). That is, during a 20 minute session, every six minutes, an average of two EEG frequency readings per band was monitored, concurrently with EMG levels for a brain area. These readings per band were given an average for the two measurements (EMG and EEG frequency bands) within a six minute phase (early, middle, and late) for each session. Specifically, within a 90 second trial, after the selectable time interval of 30 seconds have elapsed, three concurrent readings of both EMG levels and a frequency band measure were monitored every ten seconds for an additional 30 seconds. The Band-pass Filter was then shifted to the next band, allowing a 60 second waiting period before the next three readings of the two meausres. This procedure allowed band measurement to be recorded within the middle 30 seconds of each 90 second trial (Table III). At the start of each session, baseline measures for the EMG levels were recorded.

All subjects were escorted to the experimental room and given the following instructions for trainings:

> Please sit down here. I am going to place the first set of 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.

TABLE III

COUNTERBALANCE FOR ORDER OF PRESENTATION OF BRAIN LOCATION MEASUREMENTS (EACH 90 SECOND TRIAL PER SESSION)

Trials and Phases		30/Sec. Interval	Brain Location[a]	Physiological Measures: EMG	Theta	Alpha	Beta
Early Phase		1[b]	C1	uv	uv	uv	uv
	I	2	C1				
		3	C1				
		1	O2				
	II	2	O2				
		3	O2				
		1	O2				
	III	2	O2				
		3	O2				
		1	C1				
	IV	2	C1				
		3	C1				
Middle Phase		1	C1				
	V	2	C1				
		3	C1				
		1	O2				
	VI	2	O2				
		3	O2				
		1	O2				
	VII	2	O2				
		3	O2				
		1	C1				
	VIII	2	C1				
		3	C1				
Late Phase		1	C1				
	IX	2	C1				
		3	C1				
		1	O2				
	X	2	O2				
		3	O2				
		1	O2				
	XI	2	O2				
		3	O2				
		1	C1				
	XII	2	C1				
		3	C1				

(Note: 20 minute sessions within a 90 second trial. Three 30 second readings of the four physiological measures were recorded in microvolts (uv).)

[a] C1 = Central Cortex; O2 = Occipital Cortex.

[b] These numbers refer to the order of the three second readings equal to 30 second intervals.

The foreheads of the subjects were then cleaned 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.

> The second sets of electrodes will be placed around your head to monitor the electric activity from your brain. Two elastic bands will be wrapped around your head to hold in place two sets of electrodes. One band will be wrapped around the back of your head, crossing the forehead to hold the first set of electrodes. The second band will cross over the center of the head to hold the second set of electrodes. These electrodes have been saturated in a saline conductive solution to insure good electrical contact.

The hair of the subject was parted to coordinate the ground electrodes at Fp1 and Fp2 (the left and right temples: eye), and the first set of active electrodes were placed across the head at C3 and C4; the second set of electrodes were placed across areas O1 and O2.

Once all the electrodes had been properly affixed, the subject was then given a set of headphones, and asked to sit relaxed in a comfortable chair with legs and feet positioned on a footstool, arms and legs uncrossed. A headphone was placed on the subject's head and tape recorded instructions were played.

The following instructions were played:

> This is an experiment on the effects of biofeedback upon an individual's reaction to internal bodily functions. Through the earphones you will hear a series of clicks. As you decrease the number of clicks, you will be gaining control over certain reactions inside your body, which will help you to become more relaxed. We have found that the following instructions generally produce the most relaxation. Let yourself begin to feel quite relaxed. Close your eyes. Try not to blink, swallow, or move your face but let it feel heavy and sagging. Breathe deeply and rhythmically. Try to settle into a daydreamy type of state. Let relaxing

> images come into your mind. This machine is quite sensitive and often records not only your internal body pattern, but also outside body movements. To control for these movements, we have adjusted the machine to screen them out. Occasionally, the machine may fail to screen out these body movements. When this occurs, you will hear an increase in the clicks. At different times throughout the session, there will be silent periods in which we will be recording different internal body measurements. Therefore, try to remain as still as possible during the session. The session will last approximately 20 minutes. Any questions?

Input to the headphone was then switched from the tape recorder to the Autogen 1500 Electromyograph. At the conclusion of the session subjects were asked to write down any strategies they found helpful during the training session. All subjects received assigned training at approximately the same time of the day for all their individual sessions.

All training and data collection were performed within a single-blind design where only the subjects were unaware of the treatment received. The same instructions were presented to each subject. Subjects were informed that this was a biofeedback experiment in which they were to gain control over their physiological pattern. They were to be informed of the type of feedback that they were to receive.

Three tapes were used for the subject receiving false decreasing tones. The first tape was used for Session 1 and Session 2, the second tape for Sessions 3, 4, and 5, and the third tape for Sessions 6 and 7. The relaxation instruction was the same for all subjects. After the instruction, the tapes were blank for subjects receiving true EMG biofeedback. Tapes for the noncontingent group, after instruction, included decreasing tone feedback rate. After the electrodes were placed, baseline measures for frontalis muscles were recorded individually and in combination with EEG frequency bands. The experimenter was to place

the coded tape either A or B in the tape recorder, set the switch for instruction, and turn on the tape recorder for the pre-recorded instructions. After the instructions, the experimenter was to activate switch A or B for training. Subjects were randomly assigned to either of the two treatment conditions, according to their letter code by an individual not actively involved in the experiment. The tape recorder was played continuously. Thus, the experimenter was to be unaware as to whether the subject was receiving false feedback from the tape recorder or EMG feedback from the Autogen 1500.

Design

Independent Measures

The independent between subject variable used for this study was the treatment condition. Seven subjects were assigned randomly to each of the treatment conditions. There were two biofeedback training treatment groups, a contingent EMG and noncontingent EMG control group. The other independent measures were all the within subjects variables; (pre and posttest) time, training sessions, and trials within training sessions.

Dependent Measures

Training session data was obtained for a total of seven sessions for each subject in the training groups. Pre and post session baseline measures appropriate to each group was obtained for both treatment groups. For the noncontingent EMG training group, session data was based on frontalis EMG levels.

Pre and posttest scores on all three subtests of the WRAT (Reading, Spelling, Arithmetic), and subtests of the WISC-R (Digit Span and Coding) and State Anxiety Inventory were the second set of dependent measures. The third set of dependent measures was the pre and post scores obtained on the teacher's BOC and the parent's questionnaire, the Werry-Weiss-Peters Activity Scale.

For all subjects three EEG frequency bands baseline measures for both occipital and parietal hemisphere were recorded for seven sessions. Baseline measures of frontalis EMG in average integral microvolts and EEG frequency were also recorded for the two testing sessions.

Analysis

A mixed model (1 Between Ss and 1 Within Ss) ANOVA was run on all pre and post measures. The Between Subjects factor was Groups (contingent and noncontingent control). The Within Subject factor was pre and post testing sessions. The dependent measures examined in this design were the following: Behavioral Observation Checklist (teacher's ratings); Werry-Weiss-Peters Activity Scale (parent's rating); WISC-R subtests, Digit Span and Coding; State Anxiety Inventory; and finally, baseline frontalis EMG levels and EEG measures.

A mixed design with 1 Between Ss and 4 Within Ss variables ANOVAS was run on the training data for the two EMG biofeedback groups. The ANOVA was 7 Sessions, 3 Phases (early, middle, and late), 2 Brain Location (central and occipital), and 12 Trials as its Within Subjects Variables. The Between Subject factor was Groups (noncontingent and contingent). The dependent measures in these

ANOVAS were baseline frontalis EMG levels and EEG microvolts in three frequency band widths (theta, alpha, and beta).

To examine the relationships among dependent measures, Pearson product-moment correlation coefficients were computed for EMG training data and EEG frequency band baseline measures.

CHAPTER III

RESULTS

The results of the evaluation of the EMG biofeedback training data and the causal effect on cortical arousal during biofeedback training on measures of physiology, cognition, and behavior is presented in four sections. The first section investigates the training data from the two treatment groups (contingent EMG biofeedback and noncontingent EMG biofeedback). The relationship between EMG training data and EEG frequency band baseline measures is also examined. The second section presents the physiological changes for the two groups, by measuring pre and post-baseline means of EMG integrated microvolts and EEG frequency bands. The third section inspects the cognitive changes on the pre- and post-measures of the Wide Range Achievement (WRAT) and the Wechsler Intelligent Scale for Children-Revised (WISC-R) subtests, Digit Span and Coding. Finally, the fourth section analyzes the behavioral changes between the pre- and post-measures of the Behavioral Observation Checklist and the Werry-Weiss-Peters Activity Scale.

Training Data

To determine the effectiveness of EMG on cortical arousal during biofeedback training, four mixed design analyses of variance on Group (2) x ID (14) x Session (7) x Brloc (2) x Phase (3) x Trial (12) were performed on EMG and on three EEG frequency bands (theta, alpha, and

beta) separately. The between Ss variable was the contingent and non-contingent EMG biofeedback groups, and the within Ss variables were 7 Treatment Sessions, two Brain Locations (central and occipital), 3 Phases (early, middle, and late), and 7 Trials.

The analyses of variances on EEG Frequency Bands for theta and beta revealed no significant main nor interaction effects (Tables IV and V). Therefore, no evidence of change between groups across Training Sessions, Trials, Phases, or Brain Locations was present for these two measures.

However, significant effects did appear in the alpha band. A Phase (Session) main effect, $F(14, 168) = 3.86$, $p < .0001$) on the alpha frequency band showed a significant change in microvolts within the three phases acorss the seven training sessions (Tables VI and Figure 1). Alpha band appeared to show its greatest reduction in microvolts between the early and late phases within the sessions, particularly 1, 4, 5, and 7. In general, there was little microvolt change between middle and late phases for all seven sessions of training in both treatment groups. Group x Session x Brain Location interaction showed a strong significant effect, $F(6, 72) = 6.16$, $p < .0001$) on alpha during EMG training (Table VI and Figure 2). A simple effects test was run on the observed interaction (Group x Session x Brloc) to investigate the relationship among the three factors. The simple effects test showed significant group difference (A at bc) at session one and seven in central and a significant difference at session 3 in the occipital brain area (Table VII). Looking across sessions (B at ac), simple effects test further revealed significant

TABLE IV

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X
ID (SUBJECT CODE) X SESSION X BRLOC
(BRAIN LOCATION) X PHASE X TRIAL
ON THETA FREQUENCY BAND

Source	SS	df	MS	F
Group	68.1430	1	68.1430	0.15
ID (Group)	5341.7705	12	445.1478	
Brloc	2.8605	1	2.8605	0.04
Group*Brloc	83.7512	1	83.7512	1.03
ID*Brloc (Group)	973.5387	12	81.1282	
Session	264.0238	6	44.0039	0.44
Group*Session	666.6503	6	110.1084	1.10
ID*Session (Group)	7225.2046	72	100.3501	
Session*Brloc	510.3745	6	85.0624	1.61
Group*Session*Brloc	392.5838	6	65.4306	1.23
ID*Session*Brloc (Group)	3814.9301	72	52.9851	
Phase (Session)	250.9157	14	17.9225	0.85
Group*Phase (Session)	195.7411	14	13.9815	0.66
ID*Phase (Group*Session)	3561.1011	168	21.1970	
Brloc*Phase (Session)	258.8130	14	18.4866	1.00
Group*Brloc*Phase (Session)	349.2308	14	24.9451	1.36
ID*Brloc*Phase (Group*Sess)	3091.0329	168	18.3990	
Trial (Sess*Brloc*Phase)	436.9208	42	10.4029	0.80
Group*Trial (Session*Brloc*Phase)	612.0644	42	14.5730	1.15
ID*Trial (Group*Sess*Brloc*Phase)	6380.4246	504	12.6596	

TABLE V

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X SESSION X BRLOC (BRAIN LOCATION) X PHASE X TRIAL ON BETA FREQUENCY BAND

Source	SS	df	MS	F
Group	10.7143	1	10.7143	0.14
ID (Group)	939.0565	12	78.2547	
Brloc	3.3661	1	3.3661	0.05
Group*Brloc	100.4309	1	100.4309	1.41
ID*Brloc (Group)	855.6776	12	71.3065	
Session	345.1399	6	57.5233	1.08
Group*Session	68.7714	6	11.4619	0.22
ID*Session (Group)	3820.8438	72	53.0673	
Session*Brloc	349.2077	6	58.2013	1.41
Group*Session*Brloc	206.8302	6	34.4717	0.84
ID*Session*Brloc (Group)	2968.4568	72	41.2286	
Phase (Session)	59.2274	14	4.2305	0.90
Group*Phase (Session)	81.4324	14	5.8166	1.24
ID*Phase (Group*Session)	790.9175	168	4.7078	
Brloc*Phase (Session)	42.4639	14	3.0331	0.66
Group*Brloc*Phase (Session)	83.0972	14	5.9355	1.30
ID*Brloc*Phase (Group*Session)	769.1562	168	4.5783	
Trial (Session*Brloc*Phase)	101.5647	42	2.4182	0.85
Group*Trial (Session*Brloc*Phase)	118.9155	42	2.8313	1.02
ID*Trial (Group*Sess*Brloc*Phase)	1398.6396	504	2.7751	

TABLE VI

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X SESSION X BRLOC (BRAIN LOCATION) X PHASE X TRIAL ON ALPHA FREQUENCY BAND

Source	SS	df	MS	F
Group	418.6532	1	418.6532	0.16
ID (Group)	31710.7423	12	2642.5619	
Brloc	145.2173	1	145.2173	0.89
Group*Brloc	135.2620	1	135.2620	0.83
ID*Brloc (Group)	1967.2852	12	163.9404	
Session	1694.4187	6	282.4031	1.30
Group*Session	783.2937	6	130.5489	0.60
ID*Session (Group)	15585.4659	72	216.4648	
Session*Brloc	1244.6793	6	207.4466	1.48
Group*Session*Brloc	5185.3301	6	864.2217	6.16[a]
ID*Session*Brloc (Group)	10109.0410	72	140.4033	
Phase (Session)	2025.9546	14	144.7110	3.86[a]
Group*Phase (Session)	363.5818	14	25.9701	0.69
ID*Phase (Group*Session)	6292.7688	168	37.4570	
Brloc*Phase (Session)	506.8774	14	36.2055	1.44
Group*Brloc*Phase (Session)	526.9409	14	37.6386	1.50
ID*Brloc*Phase (Group*Sess)	4220.0509	168	25.1194	
Trial (Session*Brloc*Phase)	1250.3189	42	29.7695	1.38[b]
Group*Trial (Sess*Brloc*Phase)	791.8643	42	18.8539	0.90
ID*Trial (Group*Sess*Brloc*Phase)	10573.4453	504	20.9791	

[a] $p < .0001$; [b] $p < .06$.

TABLE VI (Continued)

Corresponding Means for Group*Session*Brloc Interaction on Alpha

	Brloc	Sessions						
(Noncontingent EMG) Group A	Central	19.15	11.76	10.30	12.65	9.98	12.82	14.24
	Occipital	11.15	12.72	16.50	10.12	8.55	10.40	11.67
(Contingent EMG) Group B	Central	9.43	10.89	14.10	13.50	9.30	14.50	5.98
	Occipital	14.39	9.20	7.27	10.00	9.30	11.50	15.93

Corresponding Mean for Phase (Session) Main Effect on Alpha

Phase	Sessions						
Early	14.67	12.78	14.10	13.58	11.16	13.28	13.77
Middle	14.03	10.o6	12.o8	10.73	8.36	11.08	11.10
Late	11.89	10.52	9.86	10.40	8.32	12.59	10.10

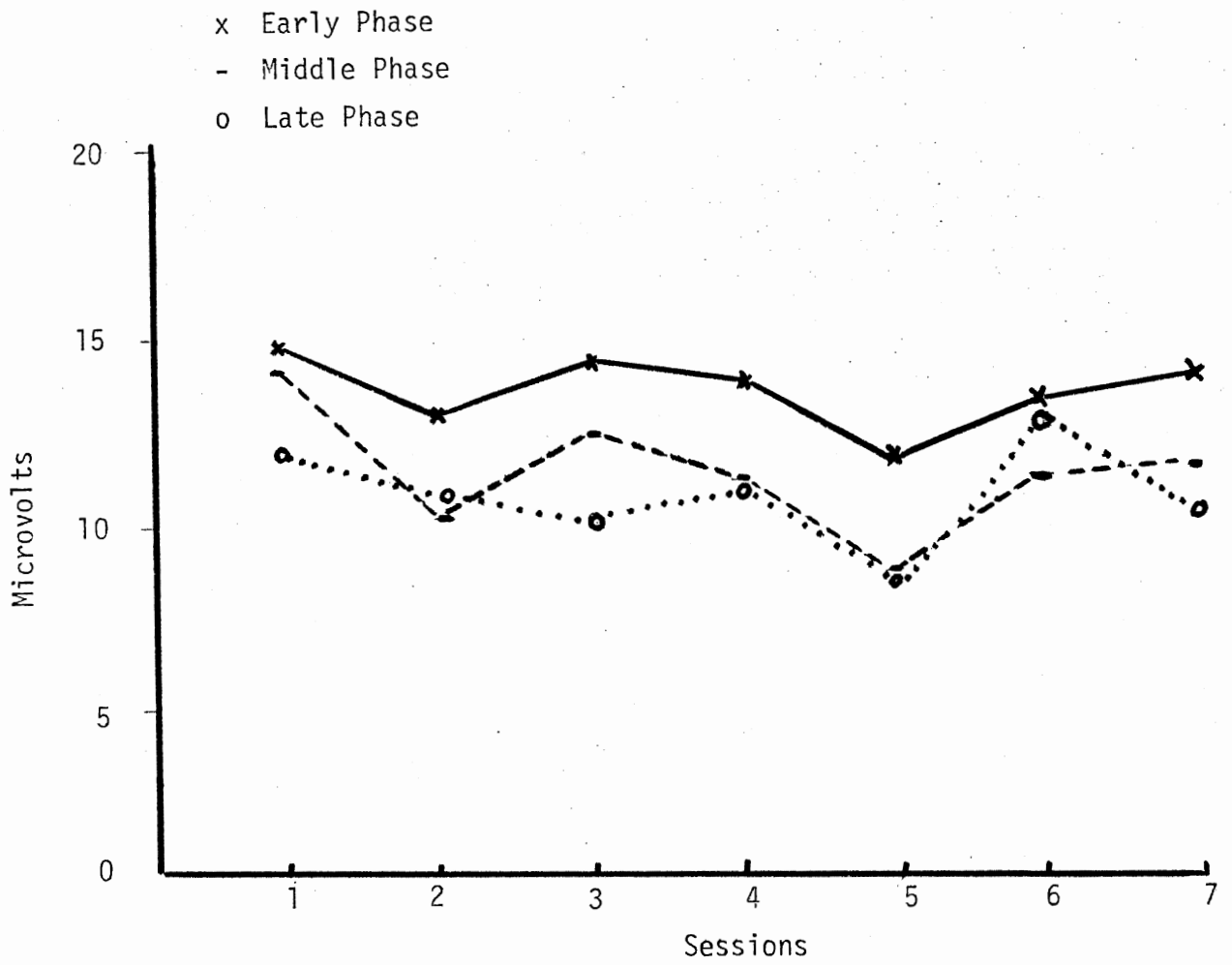

Figure 1. Phase Nested Within Session for Alpha Frequency Band for Both Treatment Groups (Contingent and Noncontingent)

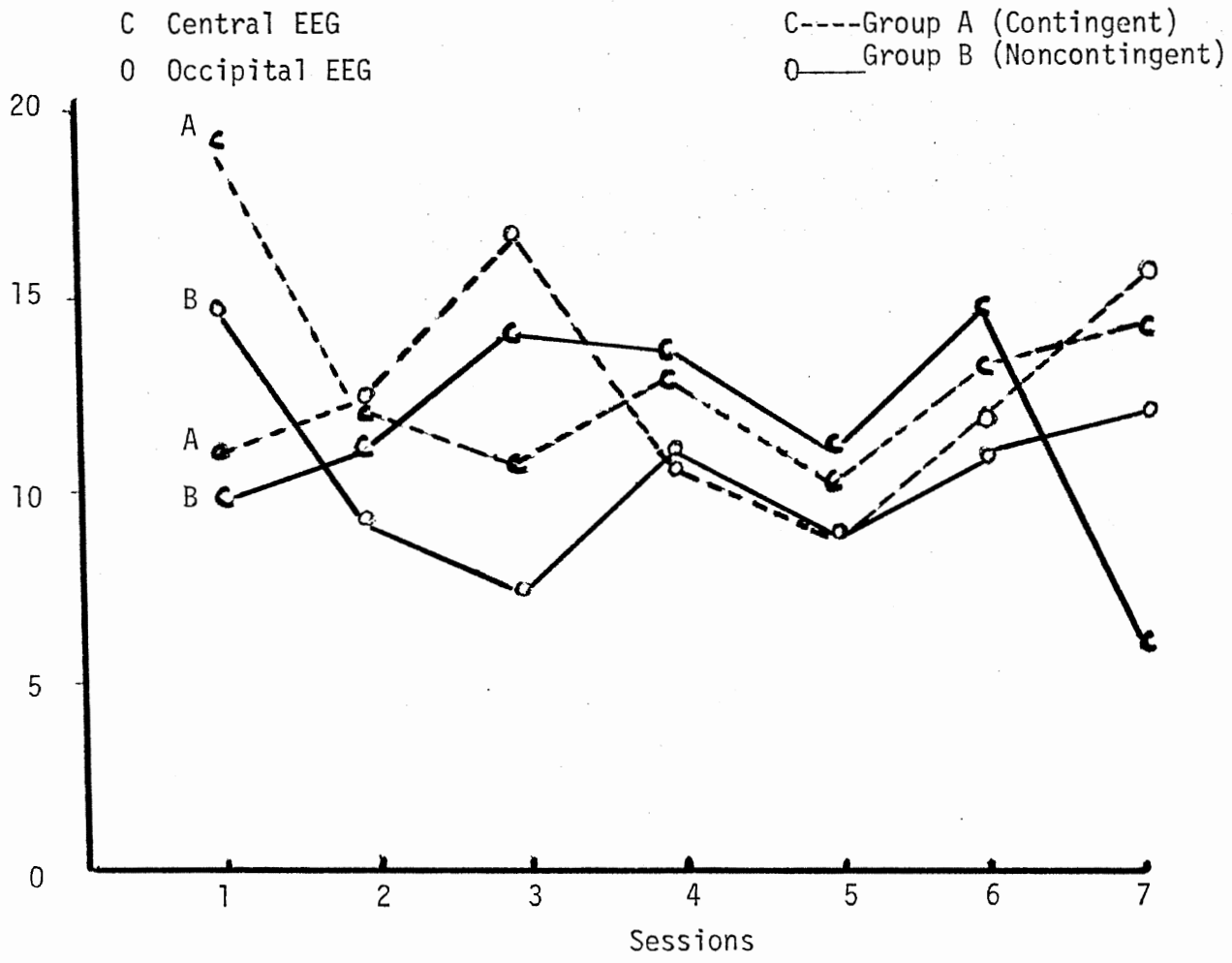

Figure 2. Group x Session x Brloc Interaction on Alpha Frequency for Contingent (B), Noncontingent (A) EMG for Brain Location Central (C) and Occipital (O)

TABLE VII

SIMPLE EFFECTS TEST ON GROUP X SESSION X BRLOC INTERACTION FOR THE ANOVA TREATMENT GROUP X ID (SUBJECT CODE) X SESSION X BRLOC (BRAIN LOCATION) X PHASE X TRIAL ON ALPHA FREQUENCY BAND

Source	SS	df	MS	F
Between Subject				
Group (A)				
at bc11				
Session/Central	338.674	1	338.674	8.7232[a]
at bc21	2.625	1	2.625	.068
at bc31	51.529	1	51.529	1.327
at bc41	2.619	1	2.619	.068
at bc51	1.557	1	1.557	.040
at bc61	10.151	1	10.151	.261
at bc71	238.334	1	238.334	6.139[a]
Group (A)				
at bc12				
Session/Occipital	36.832	1	36.832	.949
at bc22	42.508	1	42.508	1.095
at bc32	301.090	1	301.090	7.755[a]
at bc42	.046	1	.046	.001
at bc52	1.845	1	1.845	.048
at bc62	3.786	1	3.786	.098
at bc72	63.487	1	63.487	1.635
Within Cell	6522.539	168	38.825	
B (Session)				
at ac11				
Noncontingent EMG/ Central	403.587	6	67.264	2.175[b]
at ac12 (Occipital)	271.059	6	45.176	1.461
at ac21				
Contingent EMG/ Central	411.225	6	68.538	2.216[b]
at ac22 (Occipital)	398.790	6	66.465	2.149[b]
Within Cell	8907.722	288	30.930	
AB (Group/Session)				
at c1				
Central	899.872	6	149.979	3.618[a]
at c2				
Occipital	677.346	6	112.891	2.724[b]
Within Cell	5968.624	144	41.449	

TABLE VII (Continued)

Source	SS	df	MS	F
C (Brain Location)				
at ab11				
Noncontingent EMG/ Sessions	224.224	1	224.224	5.614[b]
at ab12	3.219	1	3.219	.081
at ab13	138.034	1	138.034	3.456
at ab14	22.332	1	22.332	3.456
at ab15	7.157	1	7.157	.179
at ab16	18.995	1	18.995	.476
at ab17	23.063	1	23.063	.577
at ab21				
Contingent EMG/ Sessions	86.195	1	86.105	2.156
at ab22	18.928	1	18.928	.474
at ab23	163.068	1	163.068	4.083[b]
at ab24	42.833	1	42.833	1.072
at ab25	- .125	1	- .125	.003
at ab26	32.388	1	32.388	.811
at ab27	346.091	1	346.091	8.665[a]
Within Cell	6710.488	168	39.944	
AC (Group/Brain Location)				
at b1				
Sessions	382.964	1	382.964	6.046[b]
at b2	46.462	1	46.462	.734
at b3	353.152	1	353.152	5.576[b]
at b4	66.317	1	66.317	1.047
at b5	7.168	1	7.168	.113
at b6	65.364	1	65.364	1.032
at b7	398.395	1	398.395	6.290[b]
Within Cell	5320.592	84	63.340	
BC (Session/Brain Location)				
at a1				
Noncontingent EMG	718.368	6	119.728	2.681[b]
at a2				
Contingent EMG	810.029	6	135.005	3.023[a]
Within Cell	6430.009	144	44.653	

[a] $p < .01$; [b] $p < .05$.

results between the two groups within Brain Location; i.e., the non-contingent EMG group showed a significant effect in the central location only, while the contingent EMG training group revealed differences across sessions in both central and occipital. For the simple effects tests across sessions, a significant group by session (AB at c) effect showed at both central and occipital. In simple effects tests across brain locations (c at ab), the control group yielded significant difference in session one only, while the training groups' difference appeared at session three, and also at session seven. Between the two groups and the two brain locations (AC at b), sessions one, three, and seven were significant. Finally, simple effects test indicated significant results among sessions and between brain locations (BC at a) for both groups (Table VII). These findings thus conclude that within the control group alpha was greater in central than occipital on session one; session three showed the reverse effect. However, after session three there was no difference in central alpha vs. occipital alpha. For the training group, there was no difference in alpha between the two brain locations in session one, but by session three, central alpha was greater than occipital alpha, and then by session seven, an inverse relationship occurred between brain location and alpha; occipital alpha was again significantly greater than central alpha.

The analysis on EMG integrated microvolts during training produced a Group x Session interaction effect, $F(6, 72) = 2.49$, $p < .03$; and a Group x Phase (Session) interaction effect, $F(14, 168) = 1.78$, $p < .05$ (Table VIII and Figure 3). Simple effects test calculated for Group x Session interaction produced significant differences between

TABLE VIII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X SESSION X BRLOC (BRAIN LOCATION) X PHASE X TRIAL ON EMG LEVELS

Source	SS	df	MS	F
Group	67.3286	1	67.3286	0.81
ID (Group)	994.7204	12	82.8934	
Brloc	.2527	1	.2527	1.29
Group*Brloc	.0121	1	.0121	0.06
ID*Brloc (Group)	2.3421	12	.1952	
Session	272.5640	6	45.4273	1.52
Group*Session	446.3176	6	74.3863	2.49[a]
ID*Session (Group)	2147.5454	72	29.8270	
Session*Brloc	2.2645	6	.3774	1.28
Group*Session*Brloc	.9666	6	.1611	0.55
ID*Session*Brloc (Group)	21.2408	72	.2950	
Phase (Session)	39.2644	14	2.8046	1.48
Group*Phase (Session)	47.2318	14	3.3737	1.78[b]
ID*Phase (Group*Session)	318.8782	168	1.8981	
Brloc*Phase (Session)	4.7851	14	.3418	1.41
Group*Brloc*Phase (Session)	3.0304	14	.2165	0.90
ID*Brloc*Phase (Group*Sess)	40.6172	168	.2418	
Trial (Sess*Brloc*Phase)	23.6909	42	.5641	1.11
Group*Trial (Sess*Brloc*Phase)	28.1096	42	.6693	1.35[c]
ID*Trial (Group*Sess*Brloc*Phase)	249.5569	504	.4952	

[a] $p < .03$; [b] $p < .05$; [c] $p < .08$.

TABLE VIII (Continued)

Corresponding Means for Group*Session Interaction							
Group							
A (Noncontingent EMG)	2.29	2.50	2.04	2.63	1.94	3.96	2.48
B (Contingent EMG)	4.17	2.28	2.00	1.69	1.64	1.50	1.22

Corresponding Means for Group*Phase (Session) Interaction on EMG							
Group (A)	Phase	Sessions					
Noncontingent EMG	Early	2.41	2.10	2.20	2.60	2.14	4.58	2.71
	Middle	2.24	2.10	2.10	2.70	1.93	3.85	2.38
	Late	2.22	3.37	1.86	2.60	1.75	3.47	2.36
Group (B)	Phase	Sessions						
Contingent EMG	Early	4.57	2.47	2.50	1.70	1.59	1.60	1.34
	Middle	3.89	2.40	2.03	1.65	1.70	1.36	1.29
	Late	4.06	1.90	1.46	1.68	1.61	1.54	1.04

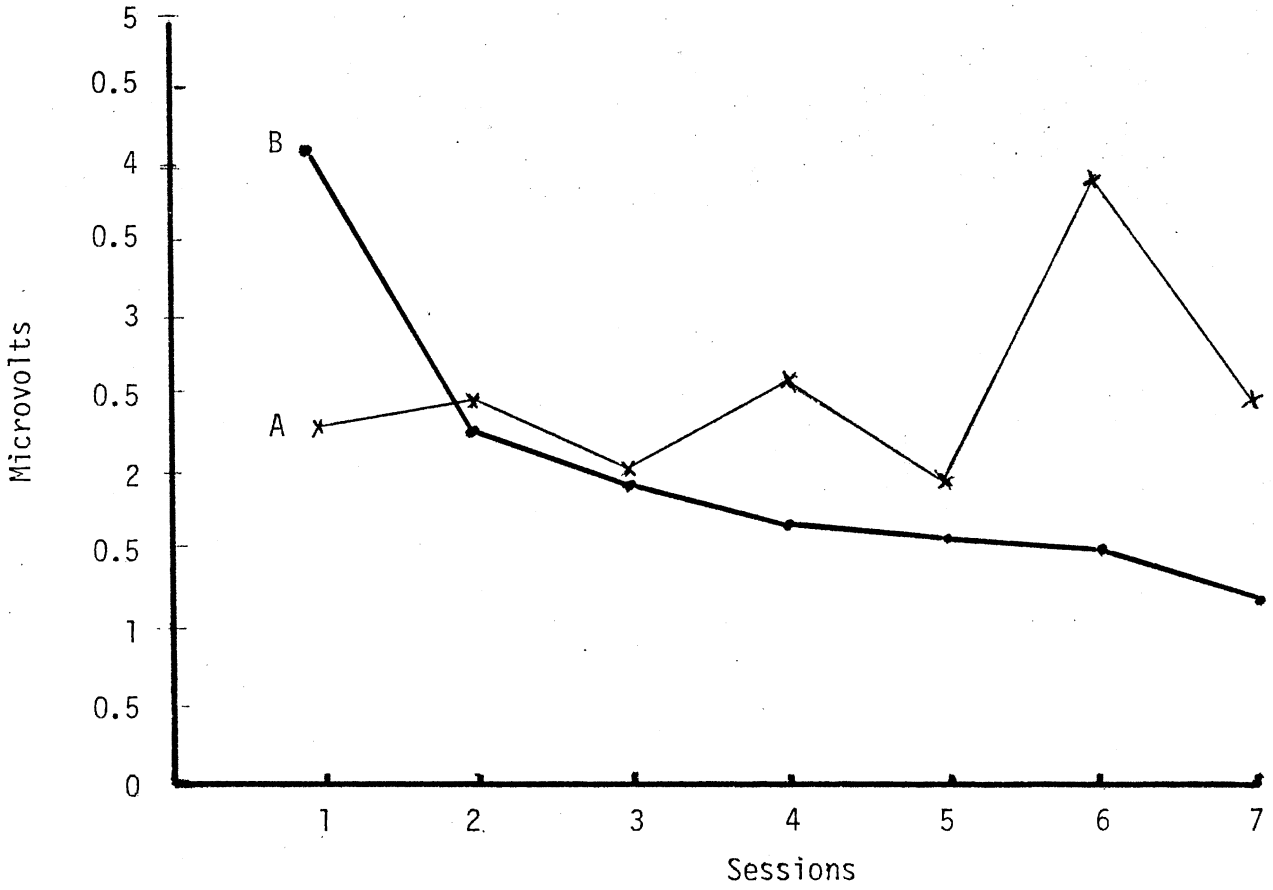

Figure 3. Group by Sessions for EMG Levels for (B) Contingent EMG and (A) Noncontingent EMG

groups at session 6 (Table IX and Figure 3), with the training group having a lower EMG (1.50 uv) than the control group (3.96 uv). Simple effects test run on Group x Phase (Session) interaction (Table X and Figure 4) indicated significant differences between the groups for early, middle, and late phases within sessions one, four, six, and seven, such that on session one, the control group had lower EMG but by session four, this was reversed with the training group showing a lower EMG. Also, for Late Phase within sessions, EMG was higher for the control group than for the training group. Phase within Session showed significant results for both groups (Table X). Figures 3 and 4 graphically illustrate the two factor relationship for each interaction (Group x Session, and Group x Phase (Session), respectively), on EMG integrated microvolts. Such results show (Figure 3) that true (contingent) EMG feedback started out with higher EMG baseline levels at session one, and then produced a general decline in muscular tension throughout training with a significantly greater reduction in EMG level at session six over control. Conversely, false (noncontingent) EMG feedback group began with low EMG levels and produced a general increase in muscular tension, peaking at session six during training. Adding the variable of Phase within Sessions, (Figure 4) the above training effect showed as early as the late phase of session two, and across all three phases, in sessions four, six, and seven. Therefore, the contingent feedback group showed the expected effect on reducing EMG levels across time.

In order to examine the relationship between EMG training data and EEG frequency band measures, Pearson product-moment correlation

TABLE IX

SIMPLE EFFECTS TEST ON GROUP X SESSION X BRLOC INTERACTION FOR THE ANOVA TREATMENT GROUP X ID (SUBJECT CODE) X SESSION X BRLOC (BRAIN LOCATION) X PHASE X TRIAL ON EMG LEVELS

Source	SS	df	MS	F
Between Subjects				
Group A				
at b1 (Session)	12.3967	1	12.3967	2.0273
at b2	.1756	1	.1756	.0287
at b3	.0075	1	.0075	.0012
at b4	3.0926	1	3.0926	.5058
at b5	.3129	1	.3129	.0512
at b6	21.2496	1	21.2496	3.4751[a]
at b7	5.5654	1	5.5654	.9101
Within Cell	513.6463	84	6.1148	
Within Subject				
B (Session)				
at a1 (Noncontingent EMG)	18.9918	6	3.1653	.6340
at a2 (Contingent EMG)	40.9021	6	6.8170	1.3655
Within Cell	718.8817	144	4.9922	

[a] $p < .01$.

TABLE X

SIMPLE EFFECTS TEST ON GROUP X PHASE (SESSION) INTERACTION FOR THE ANOVA TREATMENT GROUP X ID (SUBJECT CODE) X SESSION X BRLOC (BRAIN LOCATION) X PHASE X TRIAL ON EMG LEVELS

Source	SS	df	MS	F
Between Subjects				
Group (A) at bc11 Early Phase (Session)	16.36	1	16.36	27.6267[a]
at bc12	.6006	1	.6006	1.0142
at bc13	1.72	1	1.72	2.9045
at bc14	2.6668	1	2.6668	4.5034[a]
at bc15	1.0396	1	1.0396	1.7555
at bc16	31.0814	1	31.0814	52.4864[a]
at bc17	6.5979	1	6.5979	11.1417[a]
Group (A) at bc21 Middle Phase	9.4021	1	9.4021	15.8771[a]
at bc22	.4781	1	.4781	.8074
at bc23	.0018	1	.0018	- .0030
at bc24	3.62	1	3.62	6.1130[b]
at bc25	.1787	1	.1787	.3018
at bc26	21.7003	1	21.7003	36.6448[a]
at bc27	4.1279	1	4.1279	6.9707[a]
Group (A) at bc31 Late Phase	11.8753	1	11.8753	20.0535[a]
at bc32	7.3283	1	7.3283	12.3751[a]
at bc33	1.4697	1	1.4697	2.4818
at bc34	2.835	1	2.835	4.7874[b]
at bc35	.0652	1	.0652	.1101
at bc36	13.0309	1	13.0309	22.0049[a]
at bc37	6.1354	1	6.1354	10.3607[a]
Within Subjects				
BC (Phase w. Session) at a1 (Group A-Noncontingent EMG)	71.1827	14	5.0845	8.5861[a]
at a2 (Group B - Contingent EMG)	154.7398	14	11.0528	18.6646[a]
Within Cell	106.5930	180	.5922	

[a] $p < .01$; [b] $p < .05$.

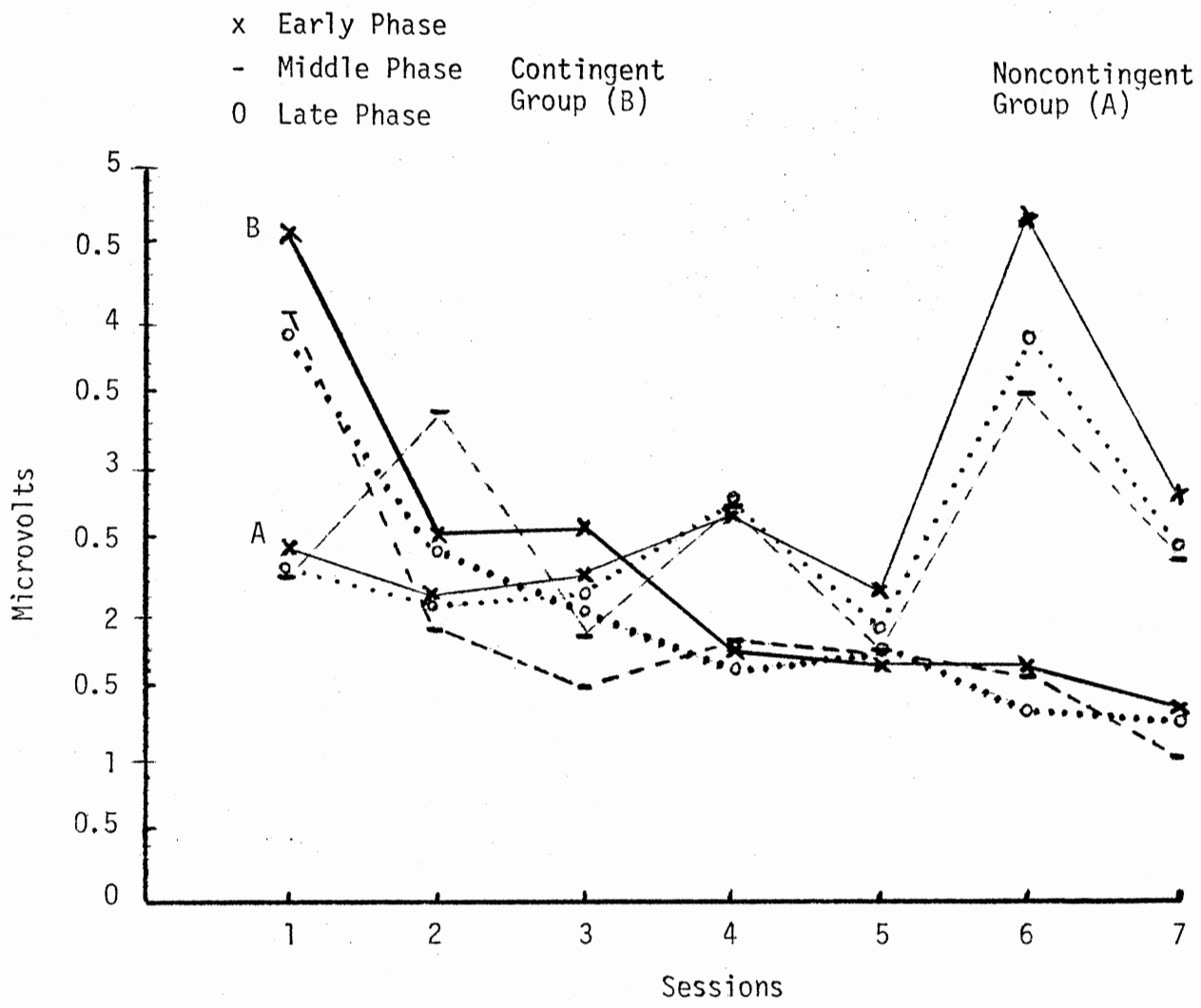

Figure 4. Group x Phase (Session) on EMG Level for Early (x), Middle (-), and Late (o) Phase Within Session for Contingent (B) and Noncontingent (A) Groups

coefficients between EMG and each EEG Band were computed across the Groups, Sessions, and Brloc (Brain Location) variables. The three EEG frequency bandwidths (theta 4-8, alpha 8-12, and beta 12-16) were recordings of peak-to-peak amplitude measured in microvolts, or millionths of a volt. EEG activity of consistently low amplitude is called synchronous or dysynchronous EEG activity, indicating high cortical arousal. EEG activity of consistently high amplitude is called synchronous EEG activity and indicates low cortical arousal. Thus, a decrease in microvolt values means a lower amplitude dysynchronized wave; i.e., high cortical arousal. Conversely, an increase in microvolt values means higher amplitude synchronized waves; i.e., low cortical arousal. Table XI depicts the amplitude of correlations matrix between EMG uv levels and the three EEG bandwidths. To further analyze the correlation data in Table XI, each subject's correlation coefficient value for each session was transformed into a Fisher Z score. Then the Z scores for each subject were averaged across the seven sessions, providing a mean Z value. These values were then placed into the analysis of variance depicted in Tables XII through XV. The first set of three Fisher Z analysis of variances was performed on each of the three frequency bands. The alpha band analysis of variance yielded no significant main nor interaction effects (Table XII). However, the analysis of variance on beta frequency band produced a marginally significant Group x Brloc crossover interaction effect, $F(1, 12) = 3.497$, $p < .10$ (Table XIII and Figure 5). For the training group, Figure 5 shows a positive relationship between amplitude in central beta and EMG and no relationship in the occipital area. The findings were reversed for the

TABLE XI

CORRELATION COEFFICIENTS BETWEEN THE EMG LEVELS AND EEG FREQUENCY BANDS (THETA, ALPHA, AND BETA) ACROSS TREATMENT GROUP, SESSIONS, AND BRLOC (BRAIN LOCATION)

	EEG Frequency Bands											
	Theta				Alpha				Beta			
BRLOC	Central		Occipital		Central		Occipital		Central		Occipital	
Sess	r	p<	r	p<	r	p<	r	p<	r	p<	r	p<
1	-.099	.27	-.027	.76	.073	.42	.039	.66	-.176	.05	-.078	.39
2	-.032	.73	-.169	.06	-.019	.84	-.072	.42	.191	.03	.116	.19
3	-.32	.0003	-.125	.16	-.102	.26	-.039	.66	-.321	.0002	-.037	.68
4	-.155	.08	-.124	.16	-.200	.03	-.151	.09	-.124	.17	-.095	.29
5	-.131	.73	-.164	.07	-.091	.31	-.395	.0001	-.097	.28	-.219	.01
6	-.09	.32	-.212	.02	-.096	.28	-.370	.0001	.320	.0003	.160	.07
7	-.156	.08	-.080	.37	-.051	.57	.251	.005	.347	.0001	-.12	.18
Group A												
Sess												
1	-.306	.0005	-.32	.0003	-.422	.0001	-.373	.0001	.111	.22	-.021	.81
2	-.086	.34	.015	.87	-.41	.0001	-.169	.06	-.184	.04	-.097	.28
3	.116	.19	-.062	.49	-.063	.48	-.120	.18	.148	.10	.09	.32
4	-.22	.01	-.594	.0001	-.316	.0003	-.598	.0001	-.273	.002	-.273	.002
5	-.192	.03	.288	.001	-.243	.006	-.04	.657	-.034	.70	.232	.009
6	-.272	.002	.481	.0001	-.189	.03	.038	.67	-.141	.12	.379	.0001
7	.068	.45	-.555	.0001	-.07	.43	-.351	.0001	-.088	.33	.231	.009
Group B												

TABLE XII

FISHER Z ANALYSIS OF VARIANCE FOR TREATMENT GROUP X BRLOC (BRAIN LOCATION) X ID (SUBJECT CODE) ON ALPHA FREQUENCY BAND CORRELATED TO EMG

Source	SS	df	MS	F
Group	.0042	1	.0042	.05
ID (Group)	.9790	12	.0816	
Brloc	.0197	1	.0197	.411
Group*Brloc	.00002	1	.00002	.0005
Brloc*ID (Group)	.5759	12	.0480	

TABLE XIII

FISHER Z ANALYSIS OF VARIANCE FOR TREATMENT GROUP X BRLOC (BRAIN LOCATION) X ID (SUBJECT CODE) ON BETA FREQUENCY BAND CORRELATED TO EMG

Source	SS	df	MS	F
Group	.004	1	.004	.08
ID (Group)	.6199	12	.0517	
Brloc	.0069	1	.0069	.1034
Group*Brloc	.233	1	.233	3.496[a]
Brloc*ID (Group)	.7994	12	.0666	

Group	Central	Occipital
A (Noncontingent EMG)	.01014	.16114
B (Contingent EMG)	.21643	.00257

[a] $p < .10$.

TABLE XIV

FISHER Z ANALYSIS OF VARIANCE FOR TREATMENT GROUP X BRLOC (BRAIN LOCATION) X ID (SUBJECT CODE) ON THETA FREQUENCY BAND CORRELATED TO EMG

Source	SS	df	MS	F
Group	.0232	1	.0232	0.63
ID (Group)	.4425	12	.0369	
Brloc	.0454	1	.0454	3.20[a]
Group*Brloc	.0704	1	.0704	4.96[b]
Brloc*ID (Group)	.1704	12	.0142	

[a] $p < .10$; [b] $p < .05$.

Means for Theta Frequency Band on Brloc (Brain Location)

Central	.00207
Occipital	-0.07850

Corresponding Means for Group x Brloc Interaction

Group	Brloc	
	Central	Occipital
A (Noncontingent EMG)	-0.0769	-0.0571
B (Contingent EMG)	0.0810	-0.0999

TABLE XV

SIMPLE EFFECTS TEST ON GROUP X BRLOC (BRAIN LOCATION) INTERACTION FOR ANOVA TREATMENT GROUP X BRLOC X ID (SUBJECT CODE) ON THETA FREQUENCY BAND

Source	SS	df	MS	F
Between Subjects				
A (Group) at b1 (Central)	.08734	1	.08734	22.39[a]
A (Group) at b2 (Occipital)	.0064	1	.0064	1.64
Within Cell	.0936	24	.0039	
Within Subjects				
B (Brloc) at a1 (Non-contingent EMG)	.00135	1	.00135	.28
B (Brloc) at a2 (Contingent EMG)	.00124	1	.00124	.257
Within Cell	.11584	24	.00483	

[a] $p < .01$.

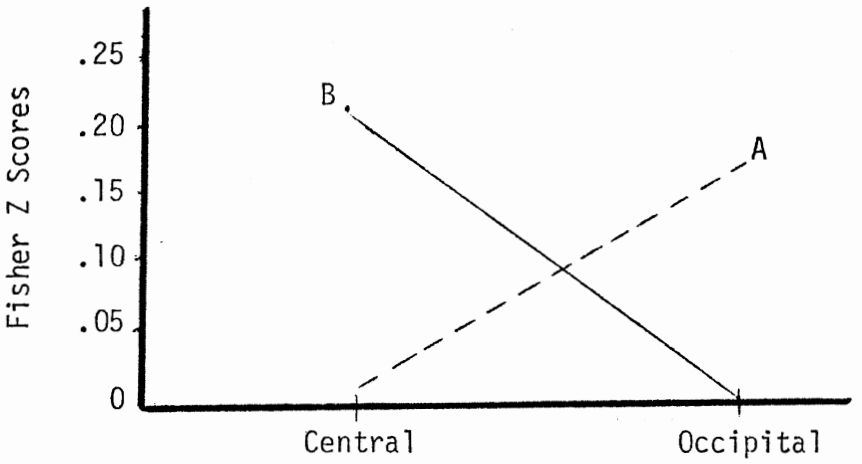

Figure 5. Group x Brloc (Brain Location) on Beta Frequency Band Correlated to EMG for Noncontingent EMG (A) and Contingent EMG (B)

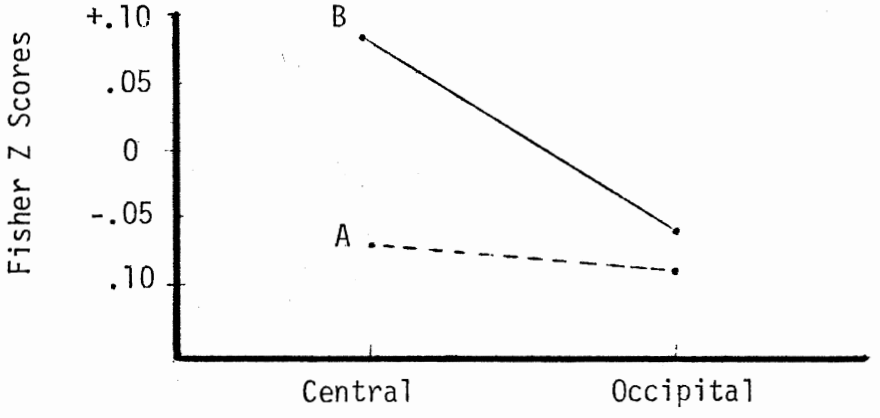

Figure 6. Group x Brloc (Brain Location) on Theta Frequency Band Correlated to EMG for Noncontingent EMG (A) and Contingent EMG (B)

control group (noncontingent) such that central beta showed no relationship to EMG, but occipital beta showed a positive relationship. As a result, the above interaction can be best explained in the following manner. For the training group (contingent), the two locations differ; i.e., in central cortex, the less aroused the central EEG, the higher the muscle tension. Occipital location showed no relationship between EEG and muscle tension. For the control group, the two locations differ in an opposite way from the training group; in occipital the less aroused the occipital EEG, the higher the muscle tension. Central brain areas showed no relationship between EEG and muscle tension.

The theta band Fisher Z analysis of variance showed a marginally significant Brloc main effect, $F(1, 12) = 3.2$, $p < .10$, such that EMG was positively correlated with theta in central; i.e., as central cortex is aroused, muscle tension is reduced. In occipital cortex, EMG is negatively correlated with theta implying that as occipital cortex becomes aroused, EMG increases (Table XIV). The Group x Brloc interaction was significant at the $p < .05$ level, $F(1, 12) = 4.95$ (Table XIV). Simple effects test run on this interaction for theta frequency's relationship to EMG showed a significant difference between groups at the central location only; i.e., true EMG training produced a positive relationship between central theta and EMG. The control group showed a negative relationship between these two variables (Table XV and Figure 6). These findings indicate that EMG treatment produced different relationships between theta and EMG in the two brain locations, such that EMG training couples low amplitude

central theta with low EMG; i.e., it couples central arousal with low muscle tension. Even though simple effects test did not find occipital theta significant, Figure 6 shows occipital theta coupled with low EMG. The EMG training produced a differentiated state between central and occipital areas. The control group showed, for both locations, high amplitude theta with low EMG levels. Across both brain sites, the control group showed low cortical arousal coupled to low muscle tension.

The second set of Fisher Z analysis involved only one analysis of variance which was calculated on microvolt reading of the total EEG frequency bands (i.e., power reading which is the amount of electrical energy contained in all three bandwidths (theta, alpha, and beta). A marginal Group x Brloc interaction effect, $F(1, 12) = 2.16$, $p < .10$ (Table XVI) was found. Figure 7 graphically shows this interaction. While there was no difference found between the two brain locations for the control, EMG training produced a more positive relationship in the central brain than the occipital cortex.

Behavioral Changes

Mixed analyses of variances were to be conducted on the pre- and post-scores of the Behavioral Observation Checklist (BOC), the teachers' rating measurement, and the Werry-Weiss-Peters Scale (WWP), the parents' questionnaire. Because of an unequal number of WWP questionnaires on post returns (5 for contingent EMG; 6 for noncontingent EMG), for both groups, and unweighted means analysis was run on pre- and post-scores, yielding no significant main nor interaction effects (Table XVII). The same nonsignificant effects were found for the analysis of variance run on the BOC variable (Table XVIII).

TABLE XVI

FISHER Z ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X BRLOC (BRAIN LOCATION) X BAND ON READING (TOTAL MICROVOLTS ON THREE FREQUENCY BANDS: THETA, ALPHA, AND BETA) CORRELATED TO EMG

Source	SS	df	MS	F
Group	.0076	1	.0076	.20
ID (Group)	.4552	12	.0379	
Brloc	.0636	1	.0636	1.09
Group*Brloc	.1841	1	.1841	3.16[a]
ID*Brloc (Group)	.6998	12	.3499	
Band	.2746	2	.1373	2.08
Group*Band	.0237	2	.0119	.18
ID*Band (Group)	1.5863	24	.0661	
Brloc*Band	.0085	2	.0042	.12
Group*Brloc*Band	.1192	2	.0596	1.69
ID*Brloc*Band (Group)	.8461	24	.0353	

[a] $p < .10$.

Corresponding Means for Group x Brloc Interaction

Group	Brain Location	
	Central	Occipital
A (Noncontingent EMG)	.0108	.0494
B (Contingent EMG)	.1234	.0252

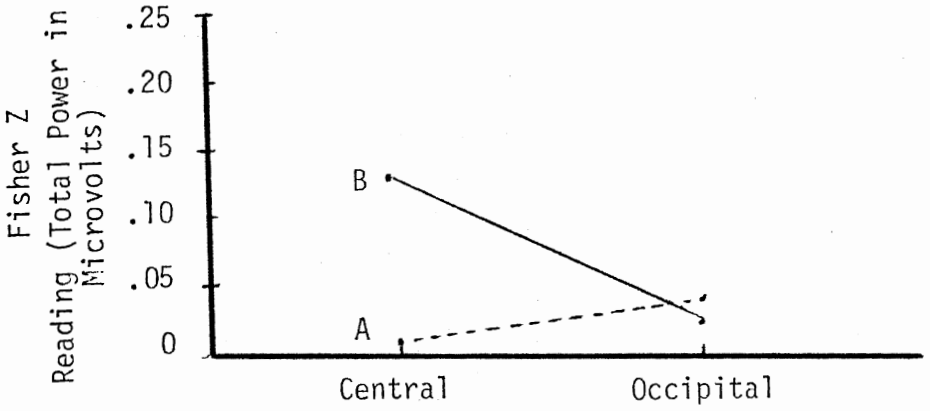

Figure 7. Group x Brloc (Brain Location) on Reading (Total Microvolts on Three Frequency Bands) Correlated to EMG for Noncontingent (A) and Contingent (B) Treatment Groups

TABLE XVII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X TEST (PRE-POST) WITH UNWEIGHTED MEANS ANALYSIS ON WWP (WERRY-WEISS-PETERS) QUESTIONNAIRE

Source	SS	df	MS	F
A (Group)	25.88	1	25.88	.59
B (Test)	23.104	1	23.104	.51
AB Group x Test	22.97	1	22.97	.53
W. Cell	952.03	21	45.34	

TABLE XVIII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON BOC (BEHAVIORAL OBSERVATION CHECKLIST)

Source	SS	df	MS	F
Group	28.0000	1	28.0000	.08
ID (Group)	4040.0000	12	336.6666	
Test	63.0000	1	63.0000	0.49
Group*Test	2.2857	1	2.2857	0.02
ID*Test (Group)	1539.7143	12	128.3095	

The STAI-A-State Anxiety Inventory, a measure of the students' subjective anxiety level as a result of EMG relaxation training yield a significant Test main effect, $F(1, 12) = 8.34$, $p < .01$ (Table XIX). The anxiety level decreased for both groups (contingent and noncontingent EMG biofeedback training) from a pre score of 41.57 to a post score of 35.00 after training.

TABLE XIX

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON STAI-A-STATE ANXIETY SCALE

Source	SS	df	MS	F
Group	120.1429	1	120.1429	1.83
ID (Group)	786.5714	12	65.5476	
Test	302.2857	1	302.2857	8.34[a]
Group*Test	11.5714	1	11.5714	0.32
ID*Test (Group)	435.1429	12	36.2619	

[a] $p < .01$.

Corresponding Means for Test Main Effect

Pre	Post
41.57143	35.00000

Cognitive Changes

The effects of EMG biofeedback training on achievement was evaluated by mixed model analyses of variance performed on pre and posttest measures of the three subtests (Reading, Arithmetic, and Spelling) on the WRAT. The results of two analyses of variance for the WRAT Reading and Arithmetic subtests yielded no significant main nor interaction effects (Tables XX and XXI). For Spelling, a Test (pre-post) main effect was found significant, $F(1, 12) = 16.87$, $p < .002$, and Group x Test interaction effect, $F(1, 12) = 3.10$, $p < .10$, was marginally significant. In general, the total score for spelling dropped from pretest (47.07) to posttest (43.57). The Group x Test interaction produced a 5.0 drop in performance for the experimental group and a 3.0 drop for the control students (Table XXII).

The analyses of variance on Digit Span Forward and Backward yielded no significant main nor interaction effects (Tables XXIII and XXIV). Total raw scores on Digit Span indicated a nonsignificant main effect but a marginally significant Group x Test interaction, $F(1, 12) = 3.08$, $p < .10$. The contingent EMG group showed an improvement of .439 from pre to post time, while the students in the control group scores decreased -.859 (Table XXV). The analysis of variance for coding yielded only a significant Test effect, $F(1, 12) = 7.64$, $p < .02$. The mean score for pretest was 11.07 and posttest was 10.86 (Table XXVI). Thus, the Group x Test interaction effect implies that attention span improved differentially over time between the two groups, but concentration ability did not. Both group findings on Spelling and Digit Span were marginal. Caution is required in interpretation of these two effects.

TABLE XX

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON READING SUBTEST FOR WIDE RANGE ACHIEVEMENT TEST

Source	SS	df	MS	F
Group	63.0000	1	63.0000	0.15
ID (Group)	4972.7143	12	414.3929	
Test	11.5714	1	11.5714	0.31
Group*Test	89.2857	1	89.2857	2.38
ID*Test (Group)	450.1429	12	37.5119	

TABLE XXI

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON ARITHMETIC SUBTEST FOR WIDE RANGE ACHIEVEMENT TEST

Source	SS	df	MS	F
Group	11.5714	1	11.5714	0.19
ID (Group)	725.2857	12	60.4405	
Test	17.2857	1	17.2857	2.02
Group*Test	0.0000	1	.0000	0.00
ID*Test (Group)	102.7143	12	8.5595	

TABLE XXII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON SPELLING SUBTEST FOR WIDE RANGE ACHIEVEMENT TEST

Source	SS	df	MS	F
Group	15.75	1	15.75	0.09
ID (Group)	2195.8571	12	182.9881	
Test	85.75	1	85.75	16.87[a]
Group*Test	15.75	1	15.75	3.19[b]
ID*Test (Group)	61.0000	12	5.0833	

[a] $p < .002$; [b] $p < .10$.

Corresponding Means for Test Main Effect

Pre	Post
47.0714	43.5714

Corresponding Means for Group*Test Interaction

Group	Pre	Post
A (Noncontingent EMG)	45.5714	43.5714
B (Contingent EMG)	48.5714	43.5714

TABLE XXIII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON WISC-R SUBTEST DIGIT SPAN F (FORWARD)

Source	SS	df	MS	F
Group	.0357	1	0.0357	0.00
ID (Group)	122.4286	12	10.2024	
Test	.3214	1	.3214	0.34
Group*Test	.8929	1	.8929	0.95
ID*Test (Group)	11.2857	12	.9405	

TABLE XXIV

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON WISC-R SUBTEST DIGIT SPAN B (BACKWARD)

Source	SS	df	MS	F
Group	1.7500	1	1.7500	0.60
ID (Group)	34.7143	12	2.8929	
Test	.0357	1	.0357	0.06
Group*Test	.8929	1	.8929	1.42
ID*Test (Group)	7.5714	12	.6310	

TABLE XXV

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON WISC-R SUBTEST DIGIT SPAN TOTAL SCORE (FORWARD+BACKWARD)

Source	SS	df	MS	F
Group	1.7500	1	1.7500	0.09
ID (Group)	226.7143	12	18.8929	
Test	.3214	1	.3214	0.34
Group*Test	2.8929	1	2.8929	3.08[a]
ID*Test (Group)	11.2857	12	.9405	

[a] $p < .10$.

Corresponding Means for Group*Test

Group	Pre	Post
A (Noncontingent EMG)	11.1429	10.2857
B (Contingent EMG)	11.0000	11.4286

TABLE XXVI

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON WISC-R SUBTEST CODING

Source	SS	df	MS	F
Group	206.2857	1	206.2857	0.55
ID (Group)	4487.1428	12	373.9286	
Test	217.2857	1	217.2857	7.64[a]
Group*Test	.5714	1	.5714	0.02
ID*Test (Group)	421.1429	12	28.4286	

[a] $p < .02$.

Corresponding Means for Test Main Effect on Coding

Pre	Post
11.07143	10.857143

Physiological Changes

To examine the changes in physiological baselines before and after treatment due to EMG relaxation training, EMG microvolt levels were recorded separately and in combination with EEG frequency bands. Mixed model analyses of variances were performed on two sets of data. Tables XXVII, XXVIII, XXIX, and XXX show the first four EMG baseline measures recorded individually, and EMG levels measured in combination with each frequency band as dependent variables. Three analysis of variances calculated on EMG levels separately, and on EMG levels measured under alpha and theta frequency bands failed to yield any meaningful statistical significant findings (Tables XXVII, XXVIII, and XXX). However, the baseline EMG levels recorded while monitoring beta frequency wave (Table XXIX) revealed no main effects, but a significant Group x Brloc interaction effect, $F(1, 12) = 5.40$, $p < .04$. The experimental group showed a higher EMG baseline level (4.183) than the control group (2.0507) during central beta monitoring, and likewise, in occipital cortex; i.e., while monitoring beta wave in the occipital cortex, the experimental group's EMG level was 4.069 and control's was 2.213. Thus, before and after EMG relaxation training, the experimental group's EMG baseline levels were higher than the control in both brain areas while monitoring in the beta bandwidth.

The second set of analyses of variance used baseline means of three EEG frequency bands (theta, alpha, and beta) as dependent measures recorded in combination with EMG levels (Tables XXXI, XXXII, and XXXIII). Only the alpha band showed a marginally significant difference in Brain Location (Brloc), $F(1, 12) = 3.64$, $p < .08$. In the occipital area of the brain, alpha was 15.491 microvolts and in central, 10.737 microvolts (Table XXXIII).

TABLE XXVII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) ON EMG-1

Source	SS	df	MS	F
Group	20.9353	1	20.9353	1.48
ID (Group)	170.2041	12	14.1837	
Test	16.5463	1	16.5463	1.02
Group*Test	19.4936	1	19.4936	1.21
ID*Test (Group)	193.8443	12	16.1537	

TABLE XXVIII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST X (PRE-POST) X BRLOC (BRAIN LOCATION) ON EMG-A (EMG LEVELS RECORDED IN COMBINATION WITH ALPHA FREQUENCY BAND)

Source	SS	df	MS	F
Group	59.2046	1	59.2046	2.78
ID (Group)	255.8219	12	21.3185	
Test	63.0064	1	63.0064	2.23
Group*Test	61.0281	1	61.0281	2.16
ID*Test (Group)	339.1681	12	28.2640	
Brloc	.1046	1	.1046	1.60
Group*Brloc	.0803	1	.0803	1.23
ID*Brloc (Group)	.7855	12	.0655	
Test*Brloc	.0469	1	.0469	0.29
Group*Test*Brloc	.0413	1	.0413	0.26
ID*Test*Brloc (Group)	1.9097	12	.1591	

TABLE XXIX

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) X BRLOC (BRAIN LOCATION) ON EMG-B (EMG LEVELS RECORDED IN COMBINATION WITH BETA FREQUENCY BAND)

Source	SS	df	MS	F
Group	55.6605	1	55.6605	2.52
ID (Group)	264.7056	12	22.0588	
Test	65.2536	1	65.2536	2.33
Group*Test	63.2400	1	63.2400	2.26
ID*Test (Group)	335.8333	12	27.9861	
Brloc	.0080	1	.0080	0.16
Group*Brloc	.2674	1	.2674	5.40[a]
ID*Brloc (Group)	.5943	12	.5943	
Test*Brloc	.1273	1	.1273	1.53
Group*Test*Brloc	.0039	1	.0039	0.05
ID*Test*Brloc (Group)	.9960	12	.9960	

[a] $p < .04$.

Corresponding Means for Group*Brloc (Brain Location)

Group	Central	Occipital
A (Noncontingent EMG)	2.0507	2.2129
B (Contingent EMG)	4.1829	4.0686

TABLE XXX

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) X BRLOC (BRAIN LOCATION) ON EMG-T (EMG LEVELS RECORDED IN COMBINATION WITH THETA FREQUENCY BAND

Source	SS	df	MS	F
Group	40.6813	1	40.6813	1.89
ID (Group)	258.8524	12	21.5710	
Test	50.8254	1	50.8254	1.91
Group*Test	47.1962	1	47.1962	1.77
ID*Test (Group)	320.1208	12	26.6767	
Brloc	1.3734	1	1.3734	0.92
Group*Brloc	.1863	1	.1863	0.12
ID*Brloc (Group)	17.9875	12	1.4989	
Test*Brloc	2.0713	1	2.0713	1.47
Group*Test*Brloc	0.7849	1	0.7849	0.56
ID*Test*Brloc (Group)	16.9485	12	1.4124	

TABLE XXXI

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) X BRLOC (BRAIN LOCATION) ON THETA FREQUENCY BAND (RECORDED IN COMBINATION WITH EMG-T)

Source	SS	df	MS	F
Group	.02791	1	.02791	0.00
ID (Group)	1374.6652	12	114.5554	
Test	.0279	1	.0279	0.00
Group*Test	10.0725	1	10.0725	0.22
ID*Test (Group)	562.1652	12	46.8471	
Brloc	46.9029	1	46.9029	1.62
Group*Brloc	61.6350	1	61.6350	2.13
ID*Brloc (Group)	346.5402	12	28.8783	
Test*Brloc	14.7600	1	14.7600	0.85
Group*Test*Brloc	.0279	1	.0279	0.00
ID*Test*Brloc (Group)	209.0401	12	17.4200	

TABLE XXXII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) X BRLOC (BRAIN LOCATION) ON BETA FREQUENCY BAND (RECORDED IN COMBINATION WITH EMG-B)

Source	SS	df	MS	F
Group	4.0179	1	4.0179	0.21
ID (Group)	230.1340	12	19.1778	
Test	7.1429	1	7.1429	0.31
Group*Test	3.3761	1	3.3761	0.15
ID*Test (Group)	275.2232	12	22.9353	
Brloc	8.0636	1	8.0636	0.44
Group*Brloc	28.5714	1	28.5714	1.55
ID*Brloc (Group)	221.7634	12	18.4080	
Test*Brloc	7.1429	1	7.1429	0.43
Group*Test*Brloc	17.4386	1	17.4386	1.05
ID*Test*Brloc (Group)	199.4419	12	16.6202	

TABLE XXXIII

ANALYSIS OF VARIANCE FOR TREATMENT GROUP X ID (SUBJECT CODE) X TEST (PRE-POST) X BRLOC (BRAIN LOCATION) ON ALPHA FREQUENCY BAND (RECORDED IN COMBINATION WITH EMG-A)

Source	SS	df	MS	F
Group	4.3597	1	4.3597	0.03
ID (Group)	1759.7935	12	146.6495	
Test	45.7659	1	45.7659	0.66
Group*Test	10.6097	1	10.6097	0.15
ID*Test (Group)	836.6908	12	69.7242	
Brloc	316.4690	1	316.4690	3.64[a]
Group*Brloc	189.9065	1	189.9065	2.19
ID*Brloc (Group)	1042.9408	12	86.9117	
Test*Brloc	108.9914	1	108.9914	2.64
Group*Test*Brloc	3.0762	1	3.0762	0.07
ID*Test*Brloc (Group)	495.0614	12	41.2551	

[a] $p < .08$.

Corresponding Means for Brloc (Brain Location)	
Central	Occipital
10.7366	15.4911

CHAPTER IV

DISCUSSION

This study proposed to investigate the mechanism underlying the effectiveness of EMG biofeedback training for hyperactivity in adolescent students. Hypotheses were designed to answer the following questions: What effects does EMG have on EEG cortical arousal? What impact does EMG training have on the inverse relationship between occipital EEG arousal and level of motor activity and muscular tension (Patmon and Murphy, 1978)? How do these results parallel with the function of central EEG sensorimotor rhythm (SMR) training and its effects on the inhibition of motor activity and muscular tension (Lubar and Shouse, 1976; Sterman, 1974)? What effects does EMG biofeedback have on cortical arousal during training and on physiological, behavioral, and cognitive indices?

To determine the effectiveness of EMG biofeedback training on cortical arousal, two treatments (true EMG biofeedback, contingent, and false biofeedback, noncontingent) were administered to hyperactive adolescents. The results indicated that of the three EEG frequency bands, theta 4-8, alpha 8-12, and beta 12-16, only the alpha band showed significance. More specifically, the true (contingent) EMG training group showed no difference in alpha between the two brain locations in session one, but by session three, central alpha was greater than occipital alpha, and then by session seven, the trend

reversed with occipital alpha greater than central alpha. Within the false (noncontingent) EMG control group, alpha was greater in central than occipital on session one; session three showed the reverse effect. Finally, after session three, there was no difference in central vs. occipital alpha.

Therefore, EEG frequency band did show a difference in cortical arousal, particularly for alpha wave in the two brain locations for the two treatment groups during the training sessions. The amplitude increased more in occipital alpha than central for the true EMG group. Consequently, occipital EEG showed less cortical arousal than central for the true EMG training group, especially during the last session.

EMG relaxation training procedures produced evidence of effectiveness for both across and within sessions in muscle tension reduction. A reverse relationship was exhibited by the two treatment groups. True EMG feedback started with higher EMG levels at session one, producing a general decline in muscular tension throughout training with a significantly greater reduction in EMG level at session six over control. Conversely, false EMG feedback group began with low EMG levels and produced a general increase in muscular tension, peaking at session six during training. Apparently, the true EMG training group was more effective in generalizing its effects across sessions than the false biofeedback group. Adding the variable of phase within session, the expected training effect on reducing EMG levels across time showed as early as the late phase of session two, and across all three phases (early, middle, and late) in sessions four, six, and seven for the contingent (true) feedback group.

In summary, true EMG feedback produced less cortical arousal in occipital cortex than it did in central cortex. Additionally, the true EMG biofeedback with relaxation instruction produced the expected training effect on reducing muscular tension. True EMG biofeedback training was more effective in generalizing its effects within early, middle, and late phases and across sessions than false EMG biofeedback with relaxation instructions.

When the correlations between cortical arousal and EMG levels were investigated in each brain location a significant difference between the brain locations (central and occipital) held for theta; marginally significant differences were found for beta band and total power. However, alpha band showed no differences between brain locations on this measure.

The Group x Brain Location interaction indicated that EMG treatment produced different relationships between theta and EMG in the two brain locations. The control (noncontingent) condition showed negative relationships for both locations between EMG and theta frequency. Across both brain sites, the control group showed low cortical arousal coupled with low muscle tension. For the experimental (contingent) group, EMG training produced a positive relationship between central theta and EMG such that it coupled low amplitude central theta with low EMG; i.e., central arousal was associated with low muscle tension. This result gives support to Lubar and Shouse's (1976) findings. Although their work was with sensorimotor rhythm (SMR) biofeedback (12-14 Hz) over a specific central area (Rolandic), a substantial increase in EEG arousal and reduction in muscle tension

occurred as a result of SMR training: the EEG became more dysynchronized as muscle tension reduced. Additional evidence for this effect comes from the marginal effect on the relationship between EEG total band power and EMG levels. This Group x Brain Location interaction mimics the above theta findings for the true EMG training group. This correlation was more positive in the central brain than in occipital. No difference between brain locations in this relationship occurred between total power and EMG for the control group.

As the beta frequency band was monitored, the brain locations differed for the true EMG biofeedback group and the false EMG biofeedback group. For the former group, as beta amplitude decreased, central cortex showed more EEG arousal and less muscle tension; occipital cortex showed no relationship between EEG and muscle tension. The false EMG group's cortical arousal differed in an opposite way in the two brain locations from the training group; as amplitude decreased in occipital, the more aroused was occipital EEG, and the higher the muscle tension. Central beta showed no relationship between EEG and muscle tension for this control group.

Such findings may imply that during true EMG training, as central EEG becomes more dysynchronized, muscle tension decreases. Conversely, during false EMG training, as occipital EEG becomes more synchronized in occipital, muscle tension increases. Patmon and Murphy's study (1978) using occipital-temporal EEG measures does not match the true EMG results; i.e., EMG biofeedback relaxation reduced cortical arousal (beta) but showed the least reduction in muscle tension. Perhaps the difference between the present study and Patmon

and Murphy's is the more localized occipital readings here relative to more diffuse EEG readings in the first study on hyperactive students.

Overall, the correlation between cortical arousal and EMG levels showed two general trends. One occurred for the training group for all three interactions (Group x Brain Location) on the following measures; beta, theta, and total power: a positive correlation was revealed between central cortex and EMG (see Figures 5, 6, and 7). Secondly, the variables of theta and total power showed the same graphic trend for the relationship between the two locations and groups. Beta measures differed by displaying a crossover interaction. As a result, one may conclude, in general, that in central beta and central theta, EMG biofeedback training affects cortical arousal such that as central beta becomes more dysynchronized, muscle tension decreases, and if central theta increases in amplitude, muscle tension increases. Furthermore, the relationship being more positive in the central over the occipital cortex implies an indirect relationship between EMG and "central state" as a result of EMG biofeedback training. That is, perhaps the EMG audio signals are substituting for the proprioceptive internal feedback loop through stimulation and interacting with the "sensorimotor corticlothalamic loops" (Brudny et al., 1976), thus serving to derive information as a result of EMG activity from motor control indirectly through feedback signals.

Various studies have reported (Budzynski and Stoyva, 1969; Green, Green, and Walters, 1970; Braud, Lupin, and Braud, 1978; and Braud, 1978) the use of electromyographic biofeedback techniques as an effective method in modifying hyperactive behavior and improving cognition. In this study, measures of behavioral changes for hyperactivity

showed no significant effect. The STAI-A-State Anxiety Inventory Scale, however, indicated that anxiety level decreased for both treatment groups (contingent and noncontingent EMG biofeedback training). True EMG biofeedback training did not specifically in this case influence subjects' subjective anxiety level.

Assessing the effect of EMG biofeedback training on cognition, only WRAT Spelling changed significantly, dropping from pre to post-test. WRAT Reading and Arithmetic showed no significant results. The attentional measure for attention span improved differentially over time between the two groups, but concentration ability did not. Since both findings in spelling and digit span was marginal, further interpretation of data is unadvisable. The physiological measurements demonstrated no meaningful significant changes as a result of EMG relaxation training examined separately or in combination with EEG frequency bands. Apparently, the training session effect did not transfer outside the training situation.

The external validity in this study may have been jeopardized, particularly for the above cognition and behavioral findings. Extraneous variables such as the physical arrangement of the classroom as a feedback training facility probably affected the experimental outcome. The acoustical dimensions were not adequate to filter out the variability of noise levels outside the classroom during the training which effected the volume of the feedback clicks. External emotional stimuli were not controlled at a minimal during the training period for some of the students. Their resource teacher, whom they were closely attached to, officially resigned nearing the administration

of posttest time. The resource teacher reported that several participants' behavior and classwork performance had been unusually sporadic during training periods because of emotional traumas that had taken place at home. Two students in particular, known as "highly stressed" with "high test anxiety" were among these students. These factors may have inhibited the transfer of benefit outside the training session.

The double-blind methodology originally designed for this study to evaluate a control for placebo and treatment expectations was not successfully carried through because of the experimenter's difficulty in monitoring ongoing physiological activity and conducting biofeedback training with hyperactive subjects who were prone to be talkative and inquisitive. An isolated facility for the experimenter and subject would have been more appropriate for this kind of subject. A single-blind design was employed, however, to control for subject expectations.

A suggested criticism of the data analysis involves a more conservative approach. The ANOVA F test is robust with respect to violation of the assumption of homogeneity of variances, provided that the number of observations in the samples is equal as was the case in this study. However, within-subject effects with such variables as Sessions, Brain Location, Phases, and Trials in repeated measures designs involves observations for a given person that are dependent or correlated. The assumption of equality of variances and covariances must be sufficiently met in this case (Kirk, 1968). The ANOVA F test is not robust to the violation of this assumption, and when it is violated, the univariate F ratio has a positive bias yielding significant results too often. If it is questionable as to the equality of variances and

covariances which may be the case in this data analysis, too many Type I errors are made for the F test. In the latter instance, the usual F ratio is not distributed, as F with the given degrees of freedom, but approximately distributed as F with new degrees of freedom smaller than the usual values. One way to determine the new degrees of freedom is to use a correction factor, the Greenhouse-Geisser conservative F test, to change the original degrees of freedom such that the new F gives approximate estimation of significance (Kirk, 1968).

The usual univariate F test suggested the analysis of variance table tends to give results closer to the nominal significance levels than do results under the Greenhouse-Geisser conservative approach, provided the degree of heterogeneity of covariance is relatively moderate. However, this assumption may not have held in this data analysis. Thus, the Greenhouse-Geisser test procedure yields a negatively biased or "conservative" test in the sense of not rejecting the hypotheses being tested as often as it should be rejected (Kirk, 1968). In applying the Greenhouse Geisser effect to the repeated measures analyses in this study and opting for a .10 alpha level rather than a usual .05 alpha level of significance, the Group x Brain Location x Session effect on alpha amplitude would be upheld. The Group x Session and Group x Phase (Session) effects on EMG value could not be upheld. The results of the Fisher Z analyses would not be affected.

Shaping procedures were used in both conditions to increase the difficulty of the task as the subjects became more successful at controlling their physiology. Also, a monetary inducement was provided such that all subjects were informed that each one would receive .25¢

a session, and a dollar bonus for those who trained all seven sessions consecutively without any absence at the end of data collection.

Finally, what effects does EMG have on EEG cortical arousal? EMG training produced less EEG cortical arousal within the alpha band (8-12 Hz) in occipital cortex. How does this result parallel with the function of EEG sensorimotor rhythm (SMR) training, a conditioned "central state," and its effect on somatic inhibition and EMG muscular tension. EMG training parallels with the function of EEG SMR training only in the reduction of muscular tension during training. The measures of behavioral changes for motor inhibition (hyperactivity) showed no indication of transfer outside the training session. This occurrence may have been attributed to extraneous variables previously mentioned in this study.

What impact does EMG training have on the occurrence of its corresponding inverse relationships between EEG occipital cortical arousal and levels of motor activity and muscular tension? Looking at the relationships between EEG occipital cortical arousal and EMG levels as it parallels the function of EEG sensorimotor training, EMG affected cortical arousal such that as central beta became a less aroused synchronized wave, muscle tension increased; central theta correlated with EMG such that as theta increased in amplitude, muscle tension was increased. Moreover, a positive relationship between EEG total band power and EMG levels was found in the central brain that was not present in occipital for the training group. Studies (Sterman, 1974; Lubar and Shouse, 1976) maintain that relationships exist between general level of motor activity and the occurrence of the corresponding

EEG sensorimotor rhythm (SMR) pattern with hyperactive children. Progressive SMR (12-14 Hz) conditioning reduces EMG muscle tension and hyperactivity. On the other hand, the present study revealed EMG as being closely associated with a function of the central cortex such that central beta (12-16 Hz) decreased muscle tension while central theta increased muscle tension. Again as stated above, because no behavioral changes transferred beyond training sessions, an examination of the relationship between EMG training and its influence on the reduction of motor activity did not occur.

Another point of view involves the parallel relationship in the physiological mechanism of EMG effectiveness in training hyperactive students and the efficacy of EEG (SMR) training of hyperactive children. Investigators (Wyrwicka and Sterman, 1968; Chase and Harper, 1971; and Sterman, 1974) speculate that neuroelectric patterns are associated directly with specific neural processes such as SMR and are found to modify behavioral function such as hyperactivity. However, in the present study no direct relationship is indicative of the results, but one may postulate a similar physiological model formulated from neurophysiological and cybernetic concepts of the servoloop feedback mechanism. The more positive relationship found in central than occipital may imply an indirect relationship between EMG activity and "central state" as a consequence of EMG biofeedback training. EMG audio signals may be substituting for the proprioceptive internal feedback loop through reverbration and interaction with the sensorimotor corticothalamic loops, extracting information from motor control indirectly through feedback signals.

What effect does EMG biofeedback have on physiological, behavioral, and cognitive indices? Few meaningful results stemmed from physiological or cognitive indices. A marginal significance indicated that attention span improved differentially over time between the two treatment groups, but concentration ability revealed no evidence of improvement. Measures of behavioral changes for hyperactivity showed no significant effect. Physiological measures showed no interpretable results due to EMG relaxation training.

Summary

EMG biofeedback training produced two major physiological results: 1) a reduction in muscular tension and less EEG cortical arousal in occipital than in central cortex, 2) a close association was shown between the function of the central cortex and the reduction of muscle tension after examining the functional relationship between EEG (SMR) training and its parallels to EMG training. The treatment effect of both EMG biofeedback and SMR biofeedback (Lubar and Shouse, 1976) reduced muscle tension in hyperactive students. As muscle tension was reduced, EEG activity became dysychronized in central cortex. Likewise, SMR training (Lubar and Shouse, 1976) dysynchronized EEG waveforms over the Rolandic area.

In view of the parallel relationship in the physiological mechanism of EMG effectiveness in training hyperactive students and the efficacy of EEG (SMR) training of hyperactive children, the more positive relationship in central than occipital may imply an indirect relationship between EMG activity and "central state" as a consequence of EMG

biofeedback training. Lastly, this study revealed that EMG biofeedback had no significant impact on cognition and hyperactivity level nor caused any change on physiological measures. The training program, apparently, was not sufficiently powerful to transfer outside the training session.

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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" neurological disturbances (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 symdrome" 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 "hyperactvity" 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 television 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 hperkinetic 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 (over includes all stimuli, unable to screen out or discriminate).
7. Heedless 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 coordination.
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 (Ex.: 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 (Ex.: collaborative games, riding bicycle, gym, etc.).
20. Poor socialization; quarrelsom; 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 appropriate 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 disability 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 differentiation 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 profoundly 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. Sometime 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 preservative 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 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-antisocial 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 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.

Hyperactivity: Adolescence

While most of the clinical literature on hyperactivity reflect informational materials and techniques designed for the younger child, little research has been written on the adolescent group. Moreover, there is a general agreement among many investigators who support the view that the high activity levels associated with hyperactivity decreases in adolescence. However, clinicans who have had extensive experience with the hyperactive child agree that elimination of the activity problem does not alleviate the remaining major problems, particularly those in the areas of educational achievement and social and emotional adjustment (Ross and Ross, 1976).

According to the present author, along with others (Wilcox, 1970; Weiss, Minde, and Werry, 1971; Ross and Ross, 1976), hyperactivity does not disappear in adolescence. Instead, the characteristics remain

much the same for the adolescent as for the child, but the behavioral manifestations change as the person grows older. Some of the particularly noteworthy differences will be discussed below.

Hyperactivity. The hyperactivity adolescent does not engage in the "frantic to-and-fro purposeless motor activity that is characteristic of the five or six year old." His "urge for constant movement" tends to become more sophisticated and is restricted to tapping (fingers, pencils, or feet), grimacing, or tics (Wilcox, 1970). Or he may compensate for his urge in such a forced artifical fashion that he becomes rigidly tense in his whole body.

Other noticeable features that still persist in some but not all hyperactive adolescents are social problems and emotional lability, impulsivity, attentional difficulties, and academic deficiencies.

Social Problems and Emotional Lability. Although these youngsters continue to overract to stimuli, they have developed some ability to respond in an appropriate manner (Wilcox, 1970). They appear to be still characterized by aggression, restlessness, and antisocial behavior. They seem to be less variable in mood than formerly, but are still demanding, still unaware of their impact on others, and still not able to exercise the degree of social adjustment necessary to soften their contacts with others (Wilcox, 1970; Weiss, Minde, and Werry, 1971; Mendelson, Johnson, and Stewart, 1971; Huessy, Metoyer, and Townsend, 1974; Ross and Ross, 1976). THey tend to lack social skills, such as the ability to relate to one's peers, to control one's own behavior, willingness to work and to complete tasks once begun, and a

basic sense of responsibility (Page, 1970). Difficulties in the home, particularly rejection by parents and siblings, poor self-esteem, and depression remain major problems as a result of hyperactivity (Ross and Ross, 1976; Huessy, Metoyer, and Townsend, 1974; Weiss, Minde, and Werry, 1971; Mendelson, Johnson, and Stewart, 1971).

Wilcox (1970) states that the hyperactive adolescents have developed some ability to delay responses in an appropriate manner. However, a residue of impulsivity still seems to remain and manifest itself through continuous overreaction to stimuli. An example of such reaction is sometimes noted "in the hardest clapper, the uncontrolled sneezer, and the one with the loudest laughter" (Wilcox, 1970).

Attentional Difficulties. Although the attention span of these individuals lengthens with maturity, they still lack the ability to sufficiently attend long lecture periods and foreign language classes required of them at the secondary level. Two groups of young people that may be found in such classes are: "the goof-offs" (he who can't pay attention and distracts others), and the anxiety-ridden student (who just freezes and cannot concentrate for long)" (Wilcox, 1970).

Of the students who have memory and thinking disorders, this disability becomes most marked and recognizable at the secondary level. These students do not have the "ability to think things through to completion." Their school assignment and work are disorganized. Time and sequence are their greatest enemies. They lack the inner direction to organize. They may have, however, achieved some degree of workable methods to eliminate the obvious quality of their deficiency in time sense, size differentiation, and distance. A pertinent example of

"where-am-I-in-time-and-space" deficit is the student who fails in class because of a "package of fifty-two tardy slips" (Wilcox, 1970).

It is the opinion of some professionals (Hoy, Weiss, Minde, and Cohen, 1972) that the main contributing influence to these students' difficulties is a cognitive energy deficit, an inability, rather than an unwillingness to perform routine classroom tasks.

As a result of the hyperactive adolescent's learning impediments, he is also characterized as having consistent retardation in school performance. Spelling deficiency is usually the residual difficulty that is seen most often after reading and writing are conquered (Ross and Ross, 1976; Hoy, Weiss, Minde, and Cohen, 1972; Wilcox, 1970).

In summary, the above literature directs attention to the unique complex needs of the hyperactive adolescent to adulthood in order to help him profit from junior high experiences and to save him from the dropout roles during high school. Therefore, further empirical study is required on the hyperactive adolescent for the development of special interventions and techniques to aid him in the development of personal growth and to establish adequate learning environments, differing from those used with hyperactive children.

Etiology of Hyperactivity

The results of many studies designed to determine etiology or the underlying defect of hyperactive children has depended greatly on the definition of hyperactivity. Definitions do vary because of the different schools of thought and the lack of agreement over the concept of hyperactivity.

Etiological explanations for hyperactivity are generally categorized into four major frameworks; medical-clinical, behavioral, psychological, and sociological (Conrad, 1974). The medical-clinical model assumes some type of organic base or dysfunction. Some organic base is postulated in the absence of gross organ dysfunction (brain tumor or mental retardation) or disease (e.g., cerebral palsy) as the cause of deviance from a medical-clinical perspective. Usually, evidence for an organic cause of deviant behavior is inferred from "soft neurological signs" of cerebral dysfunction as related to hyperactivity. Children, however, who are not hyperactive, exhibit "soft signs" and some children who are labeled as hyperactive do not exhibit these signs. Prevailing medical consensus, though, postulates some (although unclear) relation between soft signs and hyperactivity (Conrad, 1974).

The behavioral model assumes that the behavior is either "bad habits," inadequate socialization, or an adaptation to the environment. Psychological model views hyperactive behavior either as a by-product of anxiety or the results of some unconscious conflict. And lastly, the sociological model of etiology is in contrast to the other three. From a sociological perspective, it views hyperactivity in the same manner as any other form of social deviance, relative to norms, levels of tolerance, significance audiences (family or school), and available sanctions (Conrad, 1974).

All three models other than sociological postulates some type of individual "pathology" that is in need of individual remediation. Sociological model views the hyperactive behavior as meaningful within the situation and not in need of individual remediation.

According to Ross and Ross (1976), the review of literature showed that most of the clinical description and experimental studies from the turn of the century to 1970 have treated hyperactivity as a homogeneous phenomenon. Prior to 1970 there had been little evidence of action to the idea that etiological subgroups of hyperactivity exist. Most of the interest focused on etiological classification of an organic-nonorganic dichotomy (Cruickshank, Bentzen, Ratzeburg, and Tannhauser, 1961). Still (1902) established the earliest scientific description of etiological subgroups for hyperactive children. He linked hyperactivity to a variety of etiological factors, including genetic transmission and child rearing procedures. Other than his study, Bender's (1953) categories of organic, constitutional, and environmental hyperactivity was one of the few exceptions.

Considerable evidence accumulated during the late sixties for the importance of additional factors (genetic and psychogenic) other than organic, to enter into the discussion of etiological subgroups. Evidence of empirical support for subgroup etiology was lacking with the exception of two comprehensive review articles published--Werry (1968) and Werry and Sprague (1970). Both articles gave supporting evidence for genetic, organic, and psychogenic etiologic factors for hyperactivity.

Although insufficient empirical data was in existence during this period, valuable description of subgroups of hyperactive children through clinical observation was obtained. Howell, Rever, Scholl, Trowbridge, and Rutledge (1972) suggested a treatment-oriented classification for assigning excessively active children to two classes: primary hyperactivity, in which excessive movement is the root of the

child's difficulty, so that the child is best treated by focusing on the activity itself; and secondary hyperactivity, in which the high level of activity is a symptom of a more basic problem, or a reaction to it, so that therapeutic efforts should be directed to the underlying case (psychogenic).

Two patterns of hyperactivity have also been identified by Marwit and Stenner (1972). In the first of these the child consistently exhibits a high level of activity that is often inappropriate. He is clumsy, often has perceptual and learning deficits, is unable to stay with a task, and has poor peer relationships. The etiological factors that this pattern is often associated with are organic brain damage and maturational lag, but the pattern can also occur as a normal variant of temperament. The second pattern represents a learned response (behavioral) and is essentially a life style developed by the child as a means of coping with his environment. The child who is characterized by this pattern is clearly capable and does not have the basic learning and behavioral deficits of the first pattern, although he may be anxiety-ridden as a result of precipitating social and nonsocial enviromental factors.

On the basis of clinical histories and tests, Ney (1974) categorized hyperactivity into four types to determine the difference among them. Genetic (constitutional) included 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. Behavioral (conditioned) refers to hyperactive children where parents were responding with attention selectively to their active distracting behavior. Minimal

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

Although the descriptive labels differed for the various categories, some, like Howell et al.'s secondary hyperactivity, Marwit and Stenner's learned response pattern, and Ney's reactive and behavioral categories were similar in nature.

The following discussion will include four major features of etiological influence that have been investigated: genetic, organic, psychogenic, and nonsocial environmental factors. In the search for knowledge and understanding of hyperactivity, some direct and indirect empirical evidence have originated from the investigation of these four factors. In other cases, the results were contradictory.

Genetic Factors. In examining the role of genetic factors in the transmission of hyperactivity, no studies have established a direct link between the two. Two sets of well-documented findings have suggested that such a relationship may exist. Human twin studies have suggested a genetic component of activity level (Scarr, 1966). Willerman and Plomin (1973) found a significant parent-child resemblance in activity level when parents of children within the normal range of activity level were asked to report on their own childhood activity level. None of the foregoing data can be regarded as conclusive, but they have important implications for the etiology of the hyperactivity problem. On the other hand, Stewart (1973) determined that the hypothesis

of genetic transmission was doubtful since he found no difference in the frequency of hyperkinesis in the family of those who were hyperkinetic than in families of the controls.

Organic Factors. The earliest behavioral description as being a possible causative link between hyperactivity and brain damage occurred in 1902. Still's (1902) description of children deficient in "moral control" was remarkably similar to present day hyperactivity. The view that the hyperactive behavior pattern was linked to actual damage done to the brain received support from Holman (1922), Ebaugh (1923), and Strecker and Ebaugh (1924). They noted that children, when recovered from the acute phase of encephalitis, rarely showed evidence of cognitive impairment but often underwent a "catastrophic change" in personality, becoming hyperactive, distractible, irritable, antisocial, destructive, unruly, and unmanageable in school. Because of a series of studies conducted by Strauss and his associates (Strauss and Lehtinen, 1947; Strauss and Kephart, 1955) on the difference in the behavior patterns of brain-injured and non-brain-injured retarded children, brain damage come to be inferred from behavioral signs alone.

Although hyperactivity does occur in children with severe and demonstrable brain damage, there is little empirical evidence to support the view of brain damage as a major etiological factor in hyperactivity. 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.

Evidence from the Kauia pregnancy study (Werner, Bierman, French, Simonian, Conner, Smith, and Campbell, 1968) showed little relationship between severe perinatal stress and later performance.

Werry et al. (1964), using a group of children aged 7-12, 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.

Werry and Sprague (1970) found that when the criteria for subject selection are either demonstrable brain damage or brain damage inferred from noxious events, such as severe perinatal anoxia that carry a high probability of causing significant damage, the research produces little evidence that brain damage causes hyperactivity. However, when the criteria for subject selection is hyperactivity, there is a higher incidence of minor abnormalities in the experimental group than is usually the case for the normal control children.

Psychogenic Factors. One explanation of the etiology of hyperactivity centers around the traditional view that the "mother's behavior with her child is primarily a function of her attitudes, motives, and philosophy of child rearing, and as such, is relatively independent of the infant's characteristics" (Ross and Ross, 1976, p. 74). Bettelheim (1973) proposed a child's constitutional predisposition as an etiological factor for hyperactivity. When the child is stressed with environmental pressures that exceed his tolerance level, he reacts with hyperactivity.

There is both theoretical and empirical support for the early acquisition of hyperactivity as a function of direct reinforcement or through observational learning processes. Prenatal activity of a fetus characterized by a high level of arousal to maintain an optimal level of stimulation, subsequently diagnosed as hyperactive, (Zentall, 1975; Berlyne, 1960; and Leuba, 1955) exemplifies the case for the development of a hyperactive behavior pattern through early direct reinforcement.

Development of a hyperactive behavior pattern through coping responses is also strengthened through social reinforcement. Activity level transmitted through modeling processes (Bandura, Ross and Ross, 1961; Kaspar and Lowenstein, 1971) represent an example of such developmental behavior pattern. Other cases of observational learning are findings from genetic investigations of hyperactivity (Cantwell, 1972; Morrison and Stewart, 1971), longterm drug studies (Gross and Wilson, 1974), and individual case studies (Daniels, 1973) which have consistently shown that the parents of hyperactive children were often hyperactive as children themselves.

Environmental Factors: Etiological influence of hyperactivity has been explained also in terms of lead poisoning (Byers and Lord, 1943; Wiener, 1970; and Needleman, 1973), food additives in the diet (Feingold, 1973), and radiation stress, a constitutional result of exposure to conventional fluorescent lighting and to certain conditions of television viewing (Ott, 1974).

In summary, the results from the above research studies involving the four major etiological influences on hyperactivity (genetic,

organic, psychogenic, and nonsocial environmental) reflect the research at large on hyperactive children. The organic hypotheses as well as the psychogenic hypotheses presently are still unestablished and undistinguishable. From a psychiatric viewpoint, 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 co-existence of parental psychopathology and hyperactivity in the child can be just as easily encompassed within a genetic hypothesis (Werry, 1968). Conclusively, it is noteworthy to mention that, although the available data on the etiology of hyperactivity are not adequate to establish an unequivocal cause-effect relationship, the present data for some of the factors and the essential potentiality of others, qualifies them for serious consideration.

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

STAI-A-STATE ANXIETY FORM X-1

Self-Evaluation Questionnaire

Developed by C. D. Spielberger, R. L. Gorsuch, and

R. Lushene

STAI FORM X-1

Name_____ Date_____

Directions: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you feel right now; that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to best describe your present feelings. best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm.	(1)	(2)	(3)	(4)
2. I feel secure.	(1)	(2)	(3)	(4)
3. I am tense.	(1)	(2)	(3)	(4)
4. I am regretful.	(1)	(2)	(3)	(4)
5. I feel at ease.	(1)	(2)	(3)	(4)
6. I feel upset.	(1)	(2)	(3)	(4)
7. I am presently worrying over possible misfortunes.	(1)	(2)	(3)	(4)
8. I feel rested.	(1)	(2)	(3)	(4)
9. I feel anxious.	(1)	(2)	(3)	(4)
10. I feel comfortable.	(1)	(2)	(3)	(4)
11. I feel self-confident.	(1)	(2)	(3)	(4)
12. I feel nervous.	(1)	(2)	(3)	(4)
13. I am jittery.	(1)	(2)	(3)	(4)
14. I feel "high strung."	(1)	(2)	(3)	(4)
15. I am relaxed.	(1)	(2)	(3)	(4)

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
16. I feel content.	(1)	(2)	(3)	(4)
17. I am worried.	(1)	(2)	(3)	(4)
18. I feel over-excited and "rattled."	(1)	(2)	(3)	(4)
19. I feel joyful.	(1)	(2)	(3)	(4)
20. I feel pleasant.	(1)	(2)	(3)	(4)

APPENDIX F

PRE-TRAINING TABLES

TABLE XXXIV

PRE-TRAINING BASELINE CORRELATION AMONG THE THREE EEG BANDS AMPLITUDE VALUES

		EEG Bands		
		Theta	Alpha	Beta
EEG Bands	Theta	x	+.4859[a]	+.5610[b]
	Alpha	x	x	+.157
	Beta	x	x	x

[a] $p < .05$.
[b] $p < .025$.

Pearson product-moment correlation coefficients r computed with 12 degrees of freedom.

TABLE XXXV

PRE-TRAINING BASELINE MEANS OF THE THREE EEG BANDS AMPLITUDE VALUES FOR BOTH BRAIN LOCATIONS AND GROUPS

Group	Brain Location	EEG Bands		
		Theta	Alpha	Beta
		uv	uv	uv
Noncontingent EMG (Control)	Central	5.1785	15.3571	5.4464
	Occipital	3.9285	14.1071	5.1786
Contingent EMG (Experimental)	Central	2.3214	10.7143	5.1786
	Occipital	5.1786	15.8929	5.5357

Note: uv = Microvolts.

VITA

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Doctor of Philosophy

Thesis: AN APPROACH TO THE PHYSIOLOGICAL MECHANISM UNDERLYING THE EFFECTIVENESS OF EMG BIOFEEDBACK TREATMENT IN HYPERACTIVE ADOLESCENTS

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