EYE MOVEMENTS OF HYPERKINETIC CHILDREN

by

STANLEY P. BALA

A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York.

1976

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

August 20,1976
date

Howard Cohen
Chairman of Examining Committee

August 26, 1976
date

Florence D. Denmark
Executive Officer

Morton Nathanson M.D.
Daniel V. Caputo Ph. D.
John Stamm Ph. D.
Walter B. Essman M.D. Ph.D.

The City University of New York

Abstract

The objectives of this research were to study the eye movements of normal and hyperkinetic children and compare them under controlled conditions. The goal of the research was to gain insight into the pathophysiology of the hyperkinetic syndrome with the hope of developing quantitative techniques for use in diagnosing hyperkinesis.

Twenty normal and 14 hyperkinetic children were tested on a pursuit tracking task. The stimulus was a 1° white dot projected onto a dark background. The pursuit target traveled at a constant velocity of 22½° per second, making 30°-amplitude swings in both the horizontal and vertical planes. Two measures of horizontal pursuit were analyzed: number of eye movements and mean amplitude of saccadic eye movements. Both measures were obtained for two separate pursuit trials, one occurring at the beginning of the test session, the other at the end of the test session.

Results showed that:

(1) There was saccadization of pursuit in both the normal and hyperkinetic groups.

(2) Normals made significantly smaller saccadic eye movements than did hyperkinetics on both the first and second pursuit tasks. Hyperactives frequently made large saccades away from the path of the pursuit target.

(3) Normals made significantly fewer eye movements than did hyperactives on the second pursuit task.

(4) As a group, normals showed a significant decrease

in the number of eye movements from first to second pursuit task. Hyperactives showed no change in the number of eye movements from first to second pursuit task.

(5) High-IQ normals (range 118-138) made significantly more eye movements on the second pursuit presentation than did the middle-IQ normal group (range 96-117).

(6) There was no significant effect of age on the number or amplitude of eye movements made during the pursuit tasks in both the normal and hyperkinetic groups.

(7) Both the number and mean amplitudes of eye movements were significantly correlated for the hyperkinetic group between the first and second pursuit tasks.

(8) The number of eye movements but not their mean amplitudes was significantly correlated between the first and second pursuit tasks in the normal group.

Results were discussed in terms of the physiology of the pursuit system as well as those pathophysiological conditions and related factors which might affect pursuit performance.

Acknowledgements

I dedicate this manuscript to my late mother, Mrs. Hedwiga Jankowska Bala, whose patient guidance and example in part made this work possible. I also thank my father, Francis P. Bala, whose love and encouragement provided strength in times of doubt. I am especially grateful to my wife, Rochelle J. Bala, whose dedication and service enabled this thesis to become a reality. I wish to acknowledge and thank my sponsor, Dr. Bernard Cohen, for his guidance and advice during every step in the planning and execution of this project, and Drs. Daniel Caputo, William S. Battersby, and David Brown for their criticisms and suggestions in the writing of the manuscript. A special note of thanks goes to Mr. Charles Lasner, without whose professional advice on computer programming this work would have been impossible, and to my colleague and friend Mr. Steven Solomon for those many occasions when he gave his time and energy to help this experiment reach its conclusion. Finally, an open acknowledgement of thanks goes to those in SFG groups (especially SFG Flushing No. 2) for their thoughts, consideration, and concern during the unfolding of this project.

This work was funded by City of New York Health Research Council Grant No. HRC U2379.

Table of Contents

List of Tables

List of Figures

Chapter I Introduction

Definition of the Problem.

The "hyperactive child" syndrome has become a widely used diagnostic category within the last 20 years. A hallmark of the disorder is compulsive hypermotility, impulsivity, and distractibility (Wender, 1971). Hyperactive individuals may be of either normal or superior intelligence and manifest several if not all of the following symptoms: (1) perceptual problems (figure-ground difficulties); (2) perceptual motor problems; (3) disorders of attention, including perseveration; (4) short attention span; and (5) distractibility (Wender, 1971). Often these children exhibit soft or equivocal neurologic signs, including abnormal EEG and the inability to perform relatively complex motor tasks such as hopping, tandem walking, or rapid alternating movements (Peters, Romine, & Dykman, 1975; Quitkin & Klein, 1969; Satterfield, Lesser, Saul, & Cantwell, 1973). Impulsivity and emotional lability are often chief complaints (Campbell, Douglas, & Morgenstern, 1971). Specific learning disorders, speech difficulties, and disorders of memory and thinking, although not always associated with this syndrome, seem to appear nevertheless very frequently in this population. Problems in methematics and reading are especially prevalent (Wender, 1971). In general, the scholastic difficulties reported seem to be related to marked perceptual deficit and short attention span, as well as to hyper-

motility (Freiberg & Douglas, 1969; Laufer, Denhoff, & Solomons, 1957; Palkes & Stewart, 1972; Wender, 1971).

Recently, hyperactivity has been labeled minimum brain dysfunction (MBD) by the U.S. Department of Health, Education and Welfare (1966, 1969). Wender (1971) pointed out the confusion in the field regarding the labeling of the core disorder. He presented a variety of names used in classifying this disorder, including hyperactive or hyperkinetic child syndrome, minimal brain dysfunction, maturational lag, postencephalitic behavior disorder, and minimal cerebral palsy.

In 1971, the Office of Child Development and the Office of the Assistant Secretary for Health and Scientific Affairs conservatively estimated the incidence of hyperkinetic disorder to be 3% of the elementary school population (Report of the Conference..., 1971). Due to the relatively high incidence of this syndrome, it is important to develop clear diagnostic criteria for it and to attempt to determine its causes and manifestations.

Individuals classified under the rubric of hyperactive are characterized by involuntary overactivity. Recently, however, Dykman, Peters, & Ackerman (1973); Pope (1970); and Shaffer, McNamara, & Pincus (1974) have presented evidence indicating that some MBD children are hypoactive. They also stress the point that it is not the amount of activity the child evidences but the fact that the activity occurs in inappropriate situations which can lead to

the child's being labeled as hyperactive. However, two lines of evidence suggest that not all hyperactivity can be explained as inappropriate behavior. The first derives from the fact that many hyperactive children are hyperactive from birth (Wender, 1971). In these cases, hyperactivity does not seem to be situationally determined and appears to be related to a naturally high rate of motoric activity. Secondly, experimental evidence by Christensen & Sprague (1973) showed that hyperactives had significantly fewer seat movements after drug treatment and conditioning procedures. Such a decrease might be expected if hyperactives were indeed more active than normals.

Factors in the Etiology of Hyperkinesis.

Introductory remarks. The etiology of this syndrome remains a matter of speculation. Wender (1971) has implicated a variety of etiologic factors. These are: (1) organic brain damage, (2) genetic transmission, (3) extreme placement on the normal distribution, (4) intrauterine "random" variation in biologic development, (5) fetal maldevelopment, and (6) psychogenetic determinants. The presence of any one of these etiologic factors does not necessarily insure the expression of hyperactive behavior. For instance, a child with known organic brain damage may not express hyperactive behavior at home but may demonstrate hyperactivity when placed in a psychologically stressful environment such as a classroom.

Organic brain damage. Theories relating the hyperkinetic

syndrome to underlying organic brain pathology are mainly based upon the observations of similar behavioral aberrations in children following known organic cerebral disorders, e.g., encephalitis lethargica (Kahn & Cohen, 1934).

A review of the literature yields general agreement that many of the symptoms and signs exhibited by these children may be due to brain damage. A number of studies have shown a higher percentage of EEG abnormalities in hyperkinetics.

Burks (1955) found that 50% of 137 hyperactive children had abnormal EEGs, two-thirds of which were of the paroxysmal, nonfocal variety. In comparison, only 9.6% of the control group demonstrated EEG abnormalities. Satterfield et al. (1973) found that 31% of their hyperkinetic population exhibited EEG abnormalities, concluding that the EEG examination played a significant role in the assessment of the hyperactive child. Additional findings of slow diffuse dysrhythmias in hyperkinetics reported by Werry, Weiss, & Douglas (1964) add further support to the high incidence of EEG abnormalities in hyperkinetic children. Furthermore, Klinkerfuss, Lange, & Weinberg (1965) found that the diffuse abnormal slow brain wave activity seen in their hyperkinetic population did not improve with age.

Based upon the fact that EEG abnormalities are seen in cases of known organic brain damage (Meyer, Falconer, & Beck, 1954), the high rate of occurrence of such abnormal-

ities in the hyperkinetic samples studied could indicate some underlying organic pathology. Further evidence of possible organic pathology comes from Prechtl & Stemmer's (1962) study of 50 hyperkinetic children. All subjects evidenced choreiform movements defined as slight jerky movements which occurred irregularly and arhythmically in different muscle groups. Typically, muscles of the tongue, neck, face, and trunk were involved. The results are limited in that a control group of children was not included in this study.

Several recent findings are of interest as possible mechanisms for an organic basis of hyperactivity. A model of hyperkinesis secondary to lead poisoning was developed by Sauerhoff & Michaelson (1973) and Silbergeld & Goldberg (1974). In the latter study, mice were exposed to lead acetate from birth and their activity was measured between 40 and 60 days of age. Results showed mice treated with lead acetate to be more than three times as active as age-matched controls. The second part of the study involved the administration of drugs currently prescribed for hyperkinetic children, including d- and l-amphetamine, methylphenidate, phenobarbital, and chloral hydrate. The treated hyperactive mice responded "paradoxically" to stimulant drugs while phenobarbital increased their level of motor activity. Additional evidence implicating lead poisoning as a possible factor in hyperactivity comes from a study by Oliver (1972) comparing a group of hyperactive

children with controls on two measures of body lead: (1) blood lead levels, and (2) urine lead levels (after administration of an oral dose of a lead chelating agent). Results showed hyperkinetics to have significantly higher lead levels on both measures, with 60% of the urine lead levels for this group being in the toxic range. However, a subsample of hyperkinetic children used in this experiment did not significantly differ from normals on both measures of lead levels. This finding indicates that body lead levels are not the only factor associated with hyperkinesis.

Sauerhoff & Michaelson (1973) found a 20% decrease in dopamine in the brains of their lead-treated animals relative to controls. There was no change in norepinephrine content, however. They hypothesized, as did Wender (1971), that hyperkinesis may represent an abnormality in catecholamine metabolism. This deficiency may be genetic in nature or, as suggested by the aforementioned studies, acquired as a function of pathologic agents. Either way, the findings are of interest, especially in light of the fact that: (1) the incidence of pica is highest in lower socioeconomic groups, and (2) the occurrence of the hyperkinetic syndrome is greatest in lower socioeconomic groups (Silverman & Metz, 1973). As yet there is no positive evidence of a defect in dopamine metabolism, however.

Asphyxia has also been implicated as a possible cause of brain damage which might be expressed as hyperactive

behavior. Windle (1968) found that monkeys who had suffered mild degrees of asphxia exhibited a syndrome characterized by incoordination, especially eye-hand incoordination, and clumsiness. He found that hyperactivity and aggressiveness were early components of this syndrome.

Fetal malnutrition coupled with low birth weight has been of particular interest as regards the hyperkinetic syndrome. In an early study of the effect of low birth weight on development, Pasamanick & Knobloch (1959) posited a "continuum of reproductive casualty." The study compared 500 low-birth-weight and 492 full-sized infants at 40 weeks of age. Results indicated that neurologic pathology increased as birth weight decreased. Denhoff (1973) reported that low birth weight was associated with his hyperkinetic sample.

Lead poisoning, abnormalities in catecholamine metabolism, asphyxia, and fetal malnutrition are only some of the posited mechanisms which could contribute to an organic basis for hyperactivity.

Genetic transmission. Current research also lends support to Wender's (1971) genetic transmission hypothesis for this syndrome. Willerman (1973) asked mothers of 93 sets of same-sexed twins to evaluate both their activity level and zygosity. Results indicated that intraclass correlations for activity level were substantially higher for monozygotic twins than for dizygotic twins. Moreover, in twin sets, if the evaluation of one of the members

indicated hyperactivity, the monozygotic partner also showed a similar activity level. This correlation of activity level was not present with dizygotic twins.

Morrison & Stewart (1973a) studied all of the available natural parents of 59 children classified as hyperactive. They found that those subjects who had a hyperkinetic sibling or a parent who had a history of hyperkinesis were more likely to have a cousin, aunt, or uncle similarly affected. The evidence was interpreted as lending support to a polygenetic hypothesis for inheritance of hyperactivity. Stewart & Morrison (1971) interviewed the parents of 59 hyperkinetic and 41 control children. Relative to controls, results demonstrated a higher incidence of sociopathy, hysteria, and alcoholism in mothers and fathers of hyperactive children. Furthermore, a significantly greater number of the parents of hyperactive children had themselves been hyperactive as children. They raise the possibility that there was: (1) a link between alcoholism, sociopathy, hysteria, and childhood hyperactivity, and (2) the genetic and/or social transmission of this syndrome from parent to child.

Morrison & Stewart (1973b), using psychiatric profiles, compared the legal parents of 35 adopted hyperactive children with the biologic parents of hyperactive children and with a control group of parents. Results showed that the high prevalence of hysteria, alcoholism, and sociopathy found in the biologic parents of hyperactive

children was not found either in the adoptive parents or in the control parents. Adoptive parents were not especially likely to have been hyperactive themselves.

The high incidence of hyperkinesis occurring within the same family, the greater incidence of other disorders such as alcoholism, hysteria, and sociopathy in the parents and relatives of hyperkinetics, and the greater prevalence of the hyperkinetic syndrome in sibs as opposed to half-sibs appear to provide support for a hypothesis of genetic transmission in hyperkinesis. The high incidence of alcoholism and sociopathy found in the parents and relatives of hyperkinetics may represent different attempts to handle similar genetic endowments in themselves. This approach does not assume the genetic transmission of alcoholism and sociopathy; however, it does suggest that there may be different behavioral responses which can occur in an attempt to cope with similar genetic endowments responsible for hyperkinesis.

To recapitulate, current studies lend support to the hypothesis that hyperkinetic behavior may have some underlying genetic component. Acceptance of the genetic transmission hypothesis, however, requires further research. For example, data on monozygotic twins of a hyperkinetic parent(s) who are separated early in life and reared in different foster homes would yield important information regarding the genetic component in this syndrome. To date, no such study has been reported.

Sociocultural factors. Occurrence of MBD syndrome, including hyperactivity, varies as a function of socio-economic class. Silverman & Metz (1973) reported that secondary schools in low-income areas had estimated rates of occurrence of minimal brain damage among their students that were fully 2.8 times higher than those of secondary schools not in low-income areas and about twice as high as the estimated rates for all secondary schools. In a study of 48 MBD children, Denhoff (1973) reported that his hyperkinetic sample had parents who were in the lower social classes while children classified as hyporeactive were found to come from higher social classes. Tarnopol (1970), studying the relationship between MBD and delinquency, found that a significant degree of minimal brain dysfunction existed in minority-group delinquent school dropouts. She stated that in lower-class populations there are more predisposing or contributing factors to MBD such as poor environment, poor nutrition, and poor physical conditions than in higher social class populations. It would appear that socioeconomic factors play a role in the MBD syndrome.

Hyperactive behavior may be to some extent culture-bound. Poor impulse control, aggressiveness, hyperactivity, and emotional lability are rarely seen in some American subcultures. Sollenberger (1968) studied the behavior of Chinese-American children of New York. He reported a total lack of aggressiveness and crying in these children.

One would expect to find, purely on the basis of prevalence figures for this syndrome, hyperkinetics among Chinese children. That this does not seem to obtain presents a strong argument for cultural relativity in the expression of hyperactive behavior. It would appear, therefore, that even with minimal organic damage, the adequate internalization of familial and cultural controls may be enough to curb hyperactive behavior.

Thus, the MBD syndrome may be viewed as a resultant of many variables, including organic, socioeconomic, behavioral, emotional, and cultural.

Physiologic Correlates of Hyperactivity.

Many recent studies have compared normals and hyperkinetics on a measure of autonomic arousal, basal skin resistance (galvanic skin responses).

Dykman, Ackerman, Clements, & Peters (1971) found lowered autonomic reactivity in MBD children when compared to normals. This discrepancy was most marked on measures of skin resistance.

Spring, Greenberg, Scott, & Hopwood (1974) found a significant difference between hyperactive and normal boys on measures of specific (GSR) responses. Normals had larger mean amplitude specific GSRs than hyperactives. The investigators concluded that, as a group, hyperactive subjects were less aroused than normals. However, not all studies reported underarousal.

Knopp, Arnold, Andros, & Smeltzer (1973) reported that

23% of their hyperkinetic sample was underaroused while 36% was defined as overaroused. Furthermore, Satterfield & Dawson (1971) reported that some of their hyperactives were overaroused when compared with normals. Finally, Zahn, Abate, Little, & Wender (1975) reported no significant differences in baseline autonomic arousal levels between normals and hyperactives. In general, however, MBD children were found to be less reactive autonomically to all types of stimuli.

In view of the conflicting results obtained in these well-constructed studies, it is not clear at this time whether hyperactive children are autonomically over- or underaroused.

Overview of the Theoretical Approaches to the Hyperkinetic Syndrome.

There are, as has been shown, a variety of theories as to the physiological and psychological mechanisms underlying the hyperkinetic syndrome. Abrams (1968) felt that this syndrome reflects a delayed and irregular maturation pattern manifesting itself through metabolic, chemical, genetic, emotional, or other unknown means. Laufer et al. (1957) suggested that the cortex does not adequately modulate the diencephalon. Luria (1960) viewed hyperkinesis as a delay or disruption in the development of the inhibitory processes in the brain due to brain damage. His interpretation follows the classical Pavlovian model of behavior as produced by the subtle interplay of excita-

tory versus inhibitory processes. Zaporozhets (1957) postulated that hyperkinetic children are unable to advance beyond the external and investigatory behavior of early life and remain dependent on tactile inputs in order to respond to external stimuli. Wender (1971) viewed the etiology of the syndrome in terms of an abnormality in the metabolism of monoamines. Sokolov (1963) saw hyperkinesis as an inability to inhibit the orienting response, i.e., to habituate to stimuli. Relevant to Sokolov's failure-to-habituate argument are the findings by Hunt (1965) and Berlyne (1966) indicating that even apparently familiar scenes or objects are consistently treated as a new perceptual situation by the hyperkinetic child. Stimuli possessing a moderate degree of novelty for normal subjects evoked high levels of attention and exploration in the hyperkinetic child.

Satterfield & Dawson (1971) have suggested that hyperactivity may be viewed as secondary to a condition of low arousal. This theory views hyperactive behavior as an attempt to increase proprioceptive and exteroceptive sensory input.

Recently, Zahn et al. (1975) proposed a modified version of the underarousal hypothesis. They hypothesized that the optimum arousal level (i.e., the level that produces optimum performance) is higher in hyperkinetics than in controls.

Brief Review of the Oculomotor System.

Although abnormalities of body movement, ocular mobility, and visual-oculomotor coordination have been analyzed quantitatively by standard physiologic techniques in a variety of nervous and learning disorders, most of the early studies of hyperkinetic children have been largely qualitative or descriptive in nature. In particular, there have been no quantitative studies of visual-oculomotor function in hyperkinetic children despite the fact that their visual-motor or perceptual deficits seem to be an important cause for their learning disorders and poor school performance. The following is a discussion of aspects of the oculomotor system which have relevance to the present approach to the study of the hyperkinetic syndrome.

Description of eye movements. Dodge (1903) described five types of eye movements which occurred in the horizontal plane. Two of the five types he specified were the saccadic (quick eye movement) and the pursuit (slow eye movement).

Saccades occur when a subject quickly looks between two stationary points in the visual field (Westheimer, 1954b). Forward saccades made during the pursuit of a moving stimulus serve to bring the eye onto target and, as Rashbass (1961) has indicated, are stimulated by the position of the target. Saccadic eye movements can be voluntary. Maximum attained velocity of a saccade may be

upwards of 700° per second (Fuchs, 1971; Robinson, 1964).

Pursuit movements are elicited in response to movement of a stimulus across the retina and are not, as far as is currently known, under voluntary control. The function of pursuit movements is to match eye velocity to stimulus velocity, thereby creating a stationary image on the retina. Rashbass (1961) indicated that pursuit movements alone could not bring the image of the target to the retina. Dodge et al. (1930) and Young (1971) reported the maximum velocity capabilities of the pursuit system to be on the order of 40° per second. Above this velocity, tracking becomes more saccadic in nature.

Saccadic system. Wagman (1964) and Wagman, Werman, Feldman, Sugarman, & Krieger (1957) used stimulation techniques to evoke eye movements in monkeys. Findings indicated that bilateral conjugate deviations could be elicited from many areas of the cerebrum including the frontal and occipital eye fields as well as subcortical areas such as the hippocampus, pulvinar, thalamus, superior colliculus, putamen, caudate nucleus, and globus pallidus.

More recently, Robinson & Fuchs (1969) studied eye movements evoked by stimulation of frontal eye fields in monkey. Stimulation of various locations elicited contralateral conjugate saccades whose amplitudes ranged from 1° to 70°. Typically, these saccades occurred 25 msec after the onset of stimulation. Changes in stimulus param-

eters or initial eye position were found to have very little effect on the amplitude and direction of the evoked saccadic eye movement.

Bender & Shanzar (1964) defined the oculomotor pathways in the brain stem of the monkey using electrical stimulation and lesion techniques. They concluded that there was a convergence of oculomotor pathways originating from various parts of the cerebrum onto the pretectum and midbrain areas. In particular, the lower midbrain and paramedian pontine tegmentum were stressed as being sites of convergence for various impulses which acted in the initiation and control of saccadic eye movements. The investigators also stressed the fact that both the midbrain and pontine portions of the oculomotor pathways occupy the same general location as does the excitatory portion of the reticular formation.

Cohen & Henn (1972), recording in the brain stems of monkeys, found unit activity in the paramedian zone of the pontine reticular formation (PPRF) which was associated with saccadic eye movements. Units were found whose firing preceded saccades by 12 to 20 msec. More recently, Büttner-Ennever & Henn (1976) traced anatomical connections from the PPRF to the ipsilateral abducens nucleus.

Pursuit system. Less is known about the pursuit system than about the saccadic system. Based upon stimulation and lesion data, Cohen (1971) arrived at the conclusion that partially separate neural groups in the paramedian

pontine reticular formation were responsible for the production of both slow and rapid eye movements. Bizzi (1970), recording in unanesthetized monkeys, found cells in the frontal eye fields which fired during smooth eye movements and slow phases of nystagmus.

Physiology of Pursuit.

It has been experimentally shown that normals make corrective saccades during pursuit of a visual stimulus (Dodge, 1903; Westheimer, 1954a). Rashbass (1959, 1961) experimentally demonstrated that smooth eye movements were responses to stimulus movement and not to stimulus position. By causing a stationary spot (stimulus) to change position suddenly in one direction (i.e., to the left) while subsequently giving it a velocity in the opposite direction (i.e., to the right), he observed that the subject's eyes responded by moving in a direction that was away from the position change of the stimulus (i.e., to the right). Results indicated that the function of the smooth pursuit mechanism was to attain a stationary image on the retina by matching eye movement velocity with stimulus velocity. Velocity matching was performed regardless of errors in position of fixation. Errors in target fixation were corrected by saccades which occurred within 200 msec after the beginning of slow movement. The latency between successive corrective saccades was on the order of 200 msec. During normal pursuit performance (not preceded by a change of stimulus position), smooth

pursuit movements occurred after a reaction time of approximately 150 msec. Subsequently, eye velocity reached stimulus velocity in approximately 400 msec. Due to the initial reaction time, the point of fixation of the eye would always be trailing the target even when they were both traveling at the same speed. This lag was corrected for by one or more saccadic eye movements which served to bring the point of fixation of the eye onto the pursuit target.

Corrective saccades made during smooth pursuit movements were characterized as being forward saccades (Rashbass, 1959, 1961; Robinson, 1965). That is, they occurred along the track of the pursuit stimulus always in the direction of stimulus movement and served to correct for discrepancies between eye position and target position. Saccades occurring in the direction opposite that of stimulus movement (backward saccades) were not seen, even under barbiturate administration.

Present Experiment.

Related research. There is a dearth of quantitative research on eye movements in hyperkinetic children although an examination of the literature yields many references to problems involving figure-ground perceptions, visual motor integration, perceptual organization, and visual processing (Denhoff, 1973; Klatskin, McNamara, Shaffer, & Pincus, 1972; Strauss & Werner, 1941; Tarnopol, 1970; Wender, 1973).

One of the presumed functions of eye movements is to bring features of the stimulus onto the most sensitive part of the retina, the fovea centralis. Fleming (1969) stated that the perception of an event can be characterized as a process consisting of an extensive series of eye movements which serve to explore a visual event, thereby analyzing that event. Fleming assumed that a synthesis of that visual event is then constructed from this analysis. Presumably, any defects in the eye movements of hyperkinetics could contribute to problems in visual analysis and consequent synthesis of visual events.

Cantor (1971), Denhoff (1973), and Strauss & Werner (1941) have reported strabismus and saccadization of pursuit movements in hyperkinetics. The reports of saccadization of pursuit are qualitative and do not include quantitative analysis of comparisons between hyperkinetic and normal groups as to the number or amplitude of eye movements.

Bogacz, Mendelaharsu, & Mendelaharsu (1974) reported saccadic pursuit in a group of children diagnosed as developmental dyslexics. In comparison to controls, the dyslexic children made more saccadic eye movements during horizontal pursuit. In view of these findings, it is of interest to determine whether pursuit problems also exist in children who are hyperactive.

Laufer et al. (1957) characterized their hyperkinetic subjects as showing poor powers of concentration as well

as short attention spans. Grassi (1970), using an auditory vigilance paradigm, demonstrated that brain damaged and behavior disordered children missed more signals than control subjects. According to Grassi, the behavior disordered and brain damaged children exhibited a greater buildup of reactive inhibition during the task, which resulted in a breakdown of attention. This inability to sustain periods of attention may be directly related to the restless hypermotility demonstrated by hyperkinetics.

Sykes, Douglas, & Morgenstern (1973) found that, relative to controls, hyperactives showed no difference in their ability to direct attention for brief periods of time. However, they found hyperactives to be significantly inferior to controls in their ability to sustain attention, particularly on the experimenters' paced task. The inability to sustain attention appears to be due to a general lack of, or inability to control, inhibitory processes.

Many studies have indicated a high level of motor activity in hyperkinetic children (Laufer et al., 1957; Satterfield et al., 1973; Wender, 1973). Recent findings by Pope (1970) and Shaffer et al. (1974) have questioned the concept of hyperactivity in these children. There has also been disagreement as to what such devices as ballistographs, pedometers, actometers, accelerometers, etc, measure. The classification of a child as hyperactive would seem, therefore, to depend on the question

asked, e.g., gross activity level versus restlessness, and on the type of measuring instrument used. In view of this, the question arises as to whether the putative hypermotility of these children might also be reflected in their extraocular physiology.

Hypotheses. In view of the foregoing experimental evidence, the present study was undertaken to test the following hypotheses:

(1) Hyperkinetics evidence trouble with smooth pursuit movements:

(a) The number of eye movements is greater for hyperkinetics than for normals.

(b) Eye movement amplitudes are larger for hyperkinetics as compared to normals.

(2) Hyperkinetics are not able to sustain long periods of attention to the visual stimulus and evidence many movements unrelated to the task.

(3) Hyperkinetics show a developmental lag as regards normal age-related developmental changes in eye movements relative to controls.

Chapter II Methods and Materials

Subjects.

Twenty male control children were compared with 14 male hyperkinetic subjects. Each subject received $10.00 for participation in the study. Both clinical and control groups were matched for age. Age matching was accomplished using group distributions rather than yoked controls. The children ranged in age from 5 to 10 years. The mean age of the hyperkinetics was 88.4 months (range, 68-125 months; sd, 19.1 months) and of the control children 95.7 months (range, 60-124 months; sd, 18.7 months). A t test yielded no significant differences in age between the two groups (t = 1.104, $p < .90$). Normal children were recruited from private schools in New York City which served middle-middle- to high-middle-class environments. No scholarship cases were included in the normal sample. Those normals exhibiting nervous or psychiatric disorders were excluded. Those normals demonstrating physical or visual problems were also excluded from the present study. Originally, it was planned that the control sample would be drawn from the public school population, thereby yielding a greater heterogeneity of IQs and socioeconomic classes. However, due to administrative procedures, it would have taken at least a year and a half to first secure permission from the Board of Education to enable collection of such a sample. Consequently, private school populations were used, resulting

in a group that had a mean IQ which was clearly above average and which was of a higher socioeconomic status than that of the hyperactives. Hyperkinetic children were drawn mainly from the Pediatric Neurology Clinic at Elmhurst Hospital, which services a middle-class and lower-middle-class population. In addition, a small segment of the clinical population (five subjects) came from: (1) a private school for minimally brain damaged and learning disordered children, and (2) private practice. This subgroup of five children came from middle-class to high-middle-class environments. A test for significance between the mean socioeconomic status of the control and hyperkinetic groups was not performed, but data indicated that the number and amplitude of eye movements were determined by diagnosis rather than by social class. The most striking case was that of a set of dizygotic twins, one diagnosed as hyperactive and the other as normal (KW and MW). The twins came from the Elmhurst sample and, while they both had the same socioeconomic status, their performances on the test were widely disparate. The normal member of the twinship (KW) showed data that fell well within the normals' distribution (private school sample) while the hyperactive member of the set (MW) demonstrated data for the same test which fell into the upper part of the hyperkinetics' distribution.

The diagnosis of hyperkinesis was based upon: (1) history, (2) neurologic examinations, (3) psychiatric evalua-

tions, and (4) psychological testing. Two hyperkinetic subjects had experienced seizures. In the first case, the seizure (tonic-clonic with eyes deviated to the right) occurred at 3 months of age (present age, 68 months) and was associated with H flu meningitis. In the second case, the seizure occurred at 5 years of age (present age, 103 months) concomitantly with high fever. According to Chusid (1970), convulsions in the presence of high fever are commonly encountered in young children. Since both subjects showed no further convulsive activity or evidenced any EEG abnormalities, the presence of the single convulsive episode was not considered as an influencing or biasing factor in the present study. In addition, two subjects evidenced strabismus (esotropia) which was subsequently surgically corrected. In one case it was alternating in character. None of the other subjects exhibited hard neurologic signs, or definite signs of brain dysfunction or organicity. No abnormal EEGs were found in the hyperkinetic group. As is typical for hyperkinetics, most displayed "soft" neurologic signs such as poor motor coordination, poor rapid alternating movements, and poor fine motor control. A summary of the findings in the hyperkinetic children is given in Table 1. In all cases, soft signs were of no specific help in localizing possible dysfunctions.

Since initial testing took place during the academic year, the parents of the four subjects on drug were con-

TABLE 1

SUMMARY OF MEDICAL FINDINGS

IN THE HYPERKINETIC POPULATION

Medical Finding	No. of Cases	% of Population
Birth problems	4/14	28
Right/left confusion (no dominance pattern)	3/14	21
Abnormal fine motor coordination	7/14	50
Abnormal gross motor coordination	6/14	42
Hyperactivity onset at birth	4/14	28
Hyperactivity onset at 0-4 years of age	5/14	35
Hyperactivity onset after 4 years of age	5/14	35
Strabismus	2/14	14
Delayed developmental milestones	1/14	8
Normal EEG	14/14	100
History of convulsions	2/14	14

cerned that a drug-free period would adversely affect their child's academic and interpersonal school adjustment. As a result, they refused to remove their children from drugs for the 1-week period prior to testing.

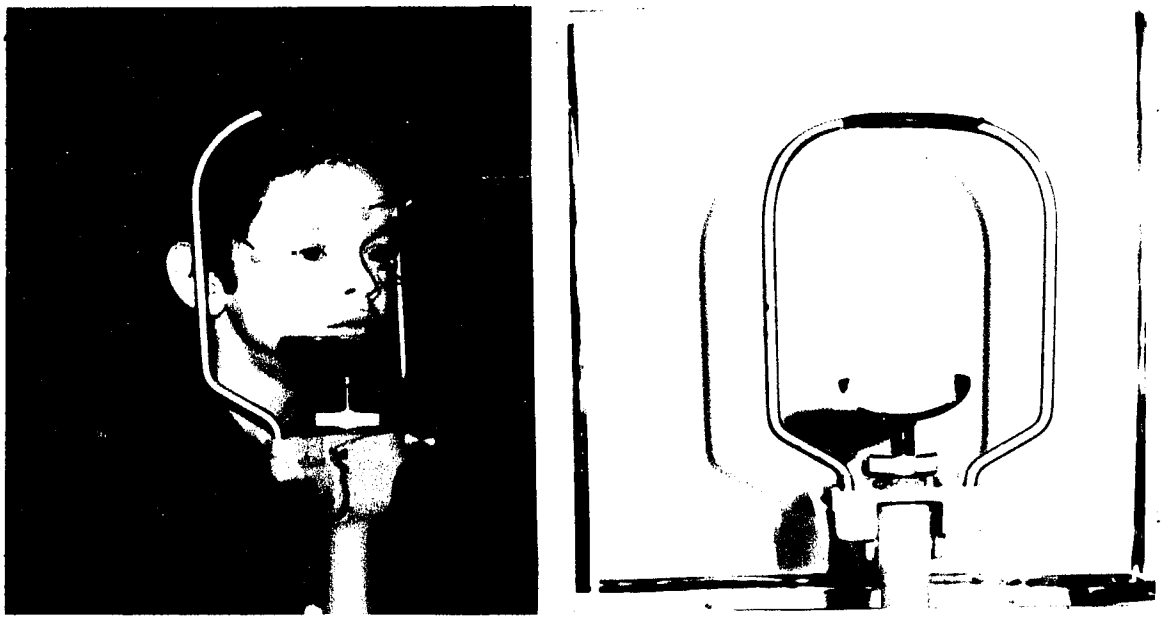
Gross & Wilson (1974), in a study of 817 MBD children, obtained WISC Performance and Verbal IQ data on 420 patients. They reported the Performance IQ to be higher in more cases than the Verbal IQ. Because it was considered advantageous to measure the child's best performance and because each subject would have to return for a lengthy test session if the Full Scale IQ were to be obtained, it was decided to use the WISC Performance IQ measure in both populations. WISC Performance IQs were obtained on 16 of the 20 control subjects, yielding a mean Performance IQ of 118 (range, 96-138; sd, 11). For two of the 14 hyperkinetics, no IQ data were available. WISC Performance IQs were obtained on eight hyperkinetic subjects, yielding a mean IQ of 98 (range, 82-123; sd, 17). It was not possible to obtain WISC Performance IQs on six of the 14 hyperkinetics because either the parents objected to further testing of their child or, after repeated attempts, the parents could not be reached. However, four hyperkinetics had been given other measures of IQ (three subjects scored 78, 82, 83 on the Stanford-Binet, one subject scored 130 on the Peabody). Only one subject scored in the borderline defective category (78 on Stanford-Binet). An attempt to restrict the hyperactive group to an IQ

range comparable to that of the normals would have resulted in the loss of over 65% of the sample. It was therefore decided to use the discrepant IQ samples.

Procedure.

Testing was conducted in a light-sheltered, air-conditioned laboratory at Mount Sinai Hospital. During the test sessions, the subjects sat in a chair with an adjustable head and chin support (see Figure 1). Head restraint, if necessary, was provided by the examiner's holding the subject's head lightly. Both horizontal and vertical eye movements were recorded using electrooculography (EOG), which is accurate for eye movements between 2° and 40°. In the horizontal channel, eye movements to the right produced upward trace deflections, while eye movements to the left resulted in downward trace deflections. In the vertical channel, both upward and downward eye movements caused simultaneous trace deflections in the direction of the eye movement. Since the EOG is subject to drift, the data were analyzed, and an average rate of drift was calculated to be on the order of .36° per second; range, .004° per second to .76° per second. A large proportion of records demonstrated no drift for the duration of the task being analyzed.

Eye movements were induced using various visual stimuli projected onto a white translucent screen by rear projectors. The projection screen was 1 meter from the subject and covered about 40° of the central field. The



A B

Fig. 1. A, Typical subject during test. Subjects sat in a chair with an adjustable head and chin support. B, Subject's view of the adjustable head and chin support facing a white translucent projection screen 1 meter away. The screen covered about 40° of the central field.

test procedure was as follows:

First, visual stimuli consisting of white dots were presented using a film. The white dots were in the midposition, and 15° and 20° from the midposition. The subject was instructed to look at these dots. The dots were first presented in the horizontal plane and secondly in the vertical plane. In this way, horizontal and vertical saccades of known amplitude were made. These were used for calibrating the record. The onset of spot movement was coded using a digital code (BCD) and recorded by a photoelectric cell in the lower-left-hand portion of the field.

After saccades were recorded, a pursuit target was presented using a white dot moving at 22½° per second. Previous experimental evidence (Dodge et al., 1930; Young, 1971) has shown that the pursuit system is capable of tracking visual targets moving up to 40° per second. Based upon this evidence, it was felt that a stimulus velocity of 22½° per second would be well within the range of pursuit capability of children. Because the pursuit target was presented by film, it was not continuous, but had a flicker rate of 24 frames per second. However, although discontinuous, the stimulus was of equal length for all subjects. The movement of the spot on the screen induced smooth or "ramp" eye movements of 30° in the horizontal plane (15° right, 15° left) and in the vertical plane (15° up, 15° down). At the end of each 30° excursion

of the stimulus, stimulus movement was halted for approximately 750 msec. A graph of spot movement to the right and left against time is shown in Figure 2. The duration of stimulus movement was 9.3 seconds, and total task time was 14.5 seconds.

Optokinetic nystagmus (OKN) was also induced by moving stripes. An ambiguous, heart-shaped figure similar to that used in the Russian studies (Zaporozhets, 1957) was then presented in order to promote scanning and pursuit movements. First, the subject viewed the figure for 30 seconds; then, a white pointer appeared on the screen and the child was instructed to watch the pointer as it moved around the edge of the figure. Calibration was then repeated as previously described.

Next, age-appropriate test slides from the Raven's Progressive Matrices were used to elicit scanning movements during visual problem solving. Many of these eye movements were vertical or oblique. The slides were projected onto the screen using a carousel projector. After each slide, the child was instructed to look at a center spot. The head was restrained lightly, if necessary, by the examiner. The child's verbal response to the test was recorded separately. After viewing the slides from the Raven's Progressive Matrices, older children were shown reading slides appropriate for their age and development (California Reading Test, Form W, Grades 1-6). These slides induced horizontal scanning movements. After each

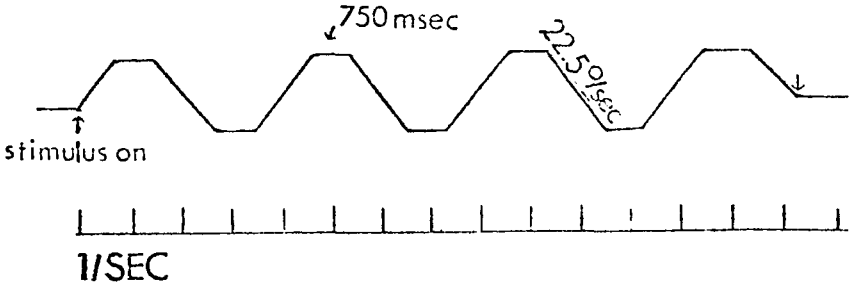


Fig. 2. Graphic representation of stimulus movement versus time. The 1° pursuit stimulus initially appeared in the midposition (see arrow indicating stimulus onset) and traveled 15° to the right at 22½° per second. Subsequent stimulus excursions at the same velocity covered 30° of the visual field (15° right to 15° left). At the end of each 30° excursion, stimulus movement was halted for approximately 750 msec. Total duration of stimulus movement was 9.3 seconds and total task time was 14.5 seconds.

slide, comprehension was tested and recorded. The children were also given a visual motor coordination task, the Seguin Form Board (Merrill-Palmer Scale of Mental Tests). This test was given with the head free, and eye movements were recorded during two trials.

A white rotating drum 36 inches in diameter and 24 inches high with 3° black stripes, each at 45° intervals, was then lowered over the child's head. The drum was internally lighted and filled the field of vision. It provided a standard environment and was rotated to provoke optokinetic nystagmus. First, with the drum still, spontaneous eye movements were recorded for 60-second periods, both in light and in darkness. Next, the child was instructed to stare at a spot in front of him for as long as he could without moving his eyes. The length of time to the first eye movement was measured on several trials. Optokinetic nystagmus was then induced using constant-velocity drum rotation of 22½°, 45°, and 67½° per second.

The drum was then lifted and the calibration film previously described was again presented, concluding the session. The entire test took about 30 minutes, and the test session, with application and removal of electrodes, took from 40 to 45 minutes.

Analysis of Data.

To analyze the data, portions of magnetic tape associated with different test conditions were identified

both by simultaneous voice comments on one channel of tape and from the BCD code which was separately recorded on another channel. In this thesis, analysis is restricted to horizontal eye movements which occurred during the pursuit portion of the film presentation.

Analysis of eye movements was performed using a small, general-purpose digital computer (Digital Equipment Corporation, PDP 8/E). Data of interest were digitized and stored on digital magnetic tape (Dectape). During digitization, the horizontal and vertical positions of the eyes were sampled 600 times each second. One hundred and forty-five seconds of consecutive data could be stored on one Dectape. After the eye movements were digitized, the beginning and ending of eye movement and the occurrence of blinks were identified and marked by the computer.

Programs for marking and identifying eye movements in primate data were operational at the beginning of this experiment. These programs accurately identified and marked 95% of these eye movements. However, for human data, these same programs failed to mark adequately the beginning and ends of eye movements and at times failed to mark movements at all. Currently, as a result of this research, programs for identifying and marking eye movements recognize 97% of eye movements and 95% of blinks. The general character of the marking program is as follows:

The computer continuously samples the digitized eye movement data at a sampling rate of 1.6 msec and writes

the digitized values on digital magnetic tape. Each 1.6-msec sample consists of an X (horizontal EOG) and a Y (vertical EOG) channel voltage. In order to determine the beginning of a movement in either the horizontal or vertical plane, four X and Y values are taken in the X and Y channels. For each channel they are then arranged according to increasing values. The median value for the 6.4-msec sample is calculated. These median values are then compared to the median X and Y values obtained for the next 6.4-msec sample. If the difference between them is equal to or exceeds a threshold value, the computer recognizes that a movement has begun. It marks the eye movement beginning on tape using an offset time, which accounts for error in detecting the exact beginning of the eye movement. The error arises from the sample time intervals used in determining the beginning of the eye movement. If the difference is less than threshold, the program discards these values, moves up the tape 1.6 msec, and begins again.

When a beginning of an eye movement is found and marked in either the X or Y channel, a blink check routine is enabled. A rapid up followed by a rapid down movement occurring in the vertical channel within a specified time interval (112 msec) is defined as a blink. If the movement is not a blink, the computer then samples both the X and Y channels for the occurrence of the end of the movement. A movement end is defined as a difference be-

tween sample medians which is equal to or less than the end threshold value. The end value subroutine is different than the beginning value subroutine in that it requires both the X and Y channels to fall below the specified threshold value. The end of a movement is marked on tape after introducing another offset to correct for error introduced by use of the median routine. The marked digitized data are then displayed on an oscilloscope and visually confirmed.

"Focal" programs were then run on the marked data, which yielded amplitudes, durations, and numbers of eye movements marked. Focal is a higher-level interactive programming language. Figure 3 presents a replay of digitized data from the computer. The downward deflection of the traces in A and B shows two movements to the left which occurred during horizontal pursuit. The beginning of each movement is marked by a small upward tic and the end by a small downward tic. This shows that the program accurately identifies and marks both small and large saccades. C shows horizontal and vertical EOG recordings during two horizontal movements to the left of about 2°. There was no deflection in the vertical EOG (the lower trace), indicating that the eyes did not move up or down during the movements. Also of importance is the fact that although the movements were of different durations, the beginnings and ends were correctly marked.

Initially, the data were plotted and correlations

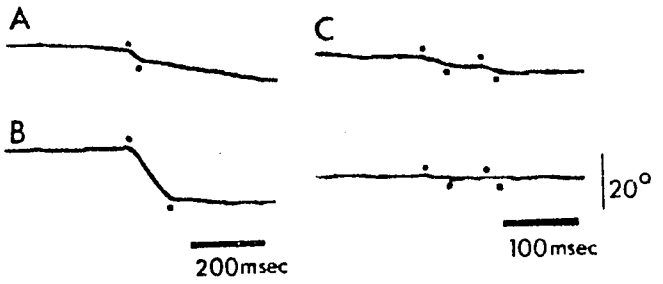


Fig. 3. Display of digitized data from the computer. The downward deflection of the traces in A and B show two movements to the left occurring during horizontal pursuit. The beginning of each movement was marked by a small upward tic and the end by a small downward tic. These traces demonstrate the accurate identification and marking of both small and large saccades. C depicts horizontal and vertical EOG recordings during two horizontal movements to the left of about 2°. There was no deflection in the vertical EOG (lower trace), indicating that the eyes did not move up or down during the movements. Even though the movements were of different durations, both the beginning and ends of the movements were correctly marked. The vertical bar to the right of C represents 20° of deviation for both the horizontal and vertical EOGs.

between dependent and independent variables were calculated. In the case of significant correlations, regression equations were computed using least-squares techniques; *t* tests were calculated when appropriate. Analyses of variance and covariance were run on an IBM 370 computer using the SPSS (Statistical Package for Social Sciences, New York: McGraw-Hill, 1975). One-way analyses of variance (ANOVAR) were performed to describe the relationships between variables. An analysis of covariance (ANCOVAR) was used to study the effects of factors and covariates on dependent measures using regression techniques resident within the ANCOVAR program. This analysis adjusts for the effect on the dependent variable of each factor and covariate when assessing the effect of any one given variable, thereby yielding a measure of the unique variance associated with any given factor or covariate.

Chapter III Results

Qualitative.

Two questions arose at the beginning of this study: Would hyperactive children sit still long enough for the collection of meaningful data, and could EOG be used to record their eye movements? A small number of hyperactive children initially reacted strongly to the test by crying and refusing to pay attention or sit still, but most were cooperative. Data were taken from agitated children only after they had calmed down and were able to attend to the tasks. None of the normals exhibited these behavior patterns. Experience in testing these children demonstrated that EOG could be used to record eye movements of hyperkinetics. We were able to complete testing in all the children.

The decision to analyze saccadic eye movements during horizontal pursuit was made on theoretical and practical grounds. Theoretically, pursuit is known to be a very sensitive index of central nervous system disorders (e.g., Parkinson's disease), and it was expected that if hypermotility were manifested in eye movements it might be present as saccadic interruption of smooth pursuit. Furthermore, pursuit performance requires a sustained focus of attention to the stimulus. Therefore, distractibility could result in saccadization of pursuit. In addition, pursuit performance was viewed as a measure of oculomotor performance with a minimum of cognitive in-

volvement in comparison to eye movements made during reading or on the Raven's Progressive Matrices Test. Simplification was considered an advantage because of the many variables already present. Practically, inspection of EOG tracings showed that there were qualitative differences between normals and hyperkinetics during pursuit.

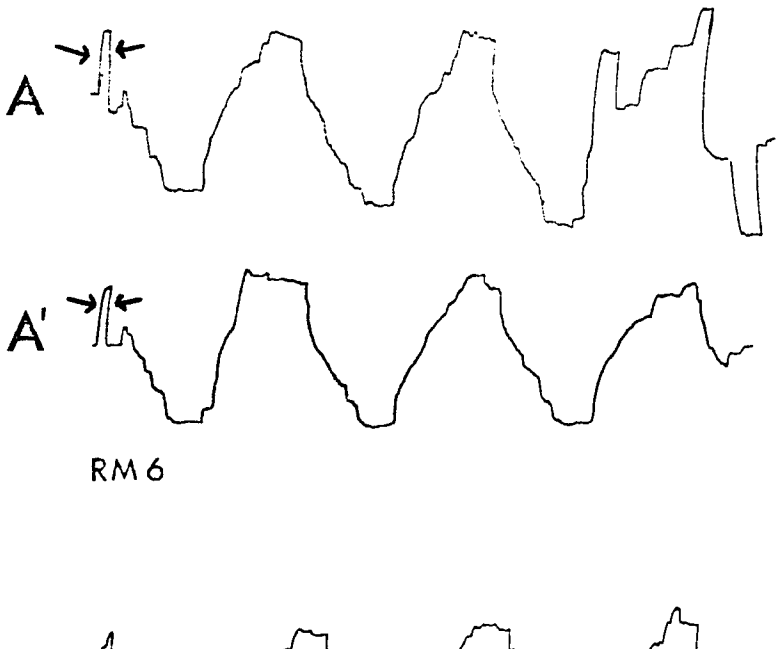
In assessing pursuit performance, a first question was whether the children could or would follow the 1° dot moving in the horizontal plane at 22½° per second. Results indicated that both normal and hyperkinetic subjects did follow adequately at some time during the pursuit task. The graph of stimulus movement versus time shown in Figure 2 can be compared to eye movement tracings shown in Figures 4-6, which were recorded in response to this stimulus.[1] There was a demonstrable synchrony between eye and stimulus movements and between periods of fixation when there was no stimulus movement. In each subject, it can be seen that at least for several cycles, pursuit eye movements were in phase with stimulus movements and slopes of slow eye movements appeared to correspond to slopes of pursuit movements. These tracings demonstrate that both normals and hyperkinetics followed directional

[1]It will be seen that the ocular responses to the 30° stimulus excursions are larger in some records than in others. This is due to different EOG gains caused by variations in electrode placements and to individual resistance differences. To analyze the records, each subject was calibrated independently and an individual conversion factor (bits per degree) was calculated for use in quantitative analyses.

Fig. 4. First and second pursuit tasks of normals. The stimulus is shown in Fig. 2. Periods of fixation are separated by 30° of horizontal deviation. Note: 1. Many normals had large forward saccades occurring within the first second after stimulus onset. In some records, these saccades occupied the entire initial 15° pursuit segment to the right (arrows, B', D, E, E'). 2. In some records, large to and fro saccades occurred during the initial pursuit segment (arrows, A and A'). 3. Normals made fewer and smaller saccades on the second pursuit presentation than on the first (compare A' to A and B' to B). 4. Despite a decrease in eye movements from first to second pursuit tasks, saccades tended to occur in the same place (arrows, D and D'). 5. Normals with high WISC Performance IQs (118-138) made more saccadic eye movements than normals with lower WISC Performance IQs (96-117). Compare D and D' from an 8-year-old with an IQ of 96 to E and E' from a 9-year-old with an IQ of 135.

NORMALS

FIRST VS SECOND PURSUIT



Continued on next page

NORMALS

FIRST VS SECOND PURSUIT

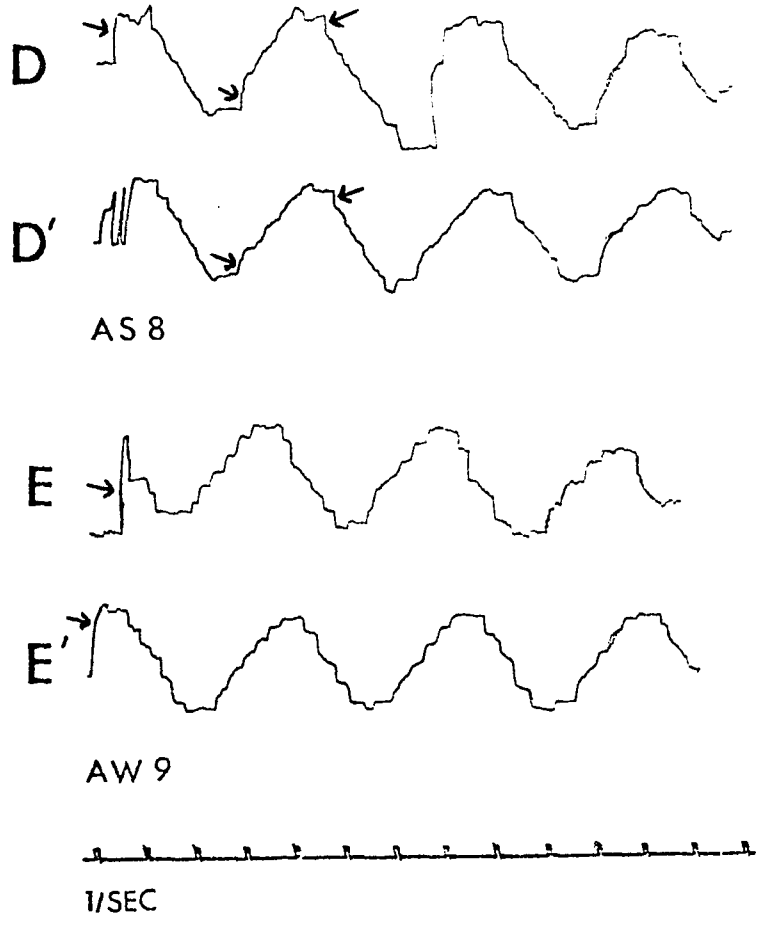
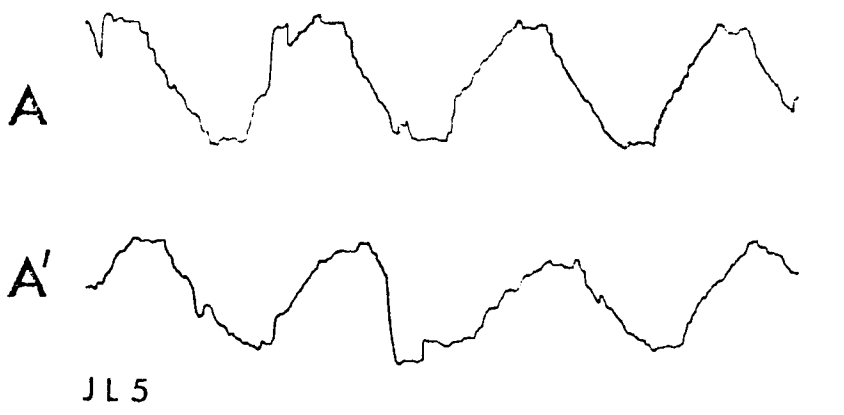


Fig. 5. First and second pursuit tasks of hyperkinetics. Note: 1. Large forward saccades occurring within the first second after stimulus onset (arrows, B, E, F, F'). 2. Large to and fro saccades during the initial pursuit segment (C, C', D, E, F). 3. Large-amplitude saccades away from the pursuit path (A', B, B', C, C', E, E'). 4. Some hyperkinetics made fewer eye movements on the second pursuit task than on the first (B and B'). However, pursuit performance in other hyperkinetics was worse on the second presentation (C and C').

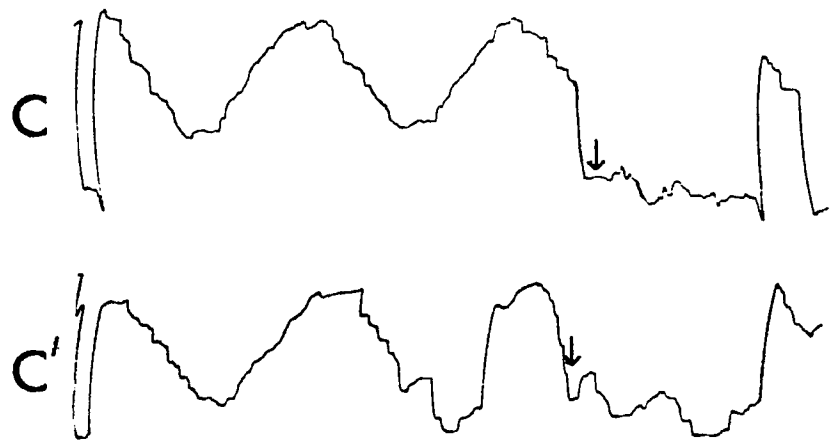
HYPERKINETIC

FIRST VS SECOND PURSUIT

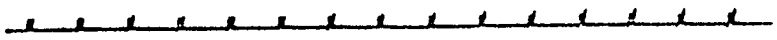


JL 5

MW 5



TG 5



1/SEC

Continued on next page

HYPERKINETIC
FIRST VS SECOND PURSUIT

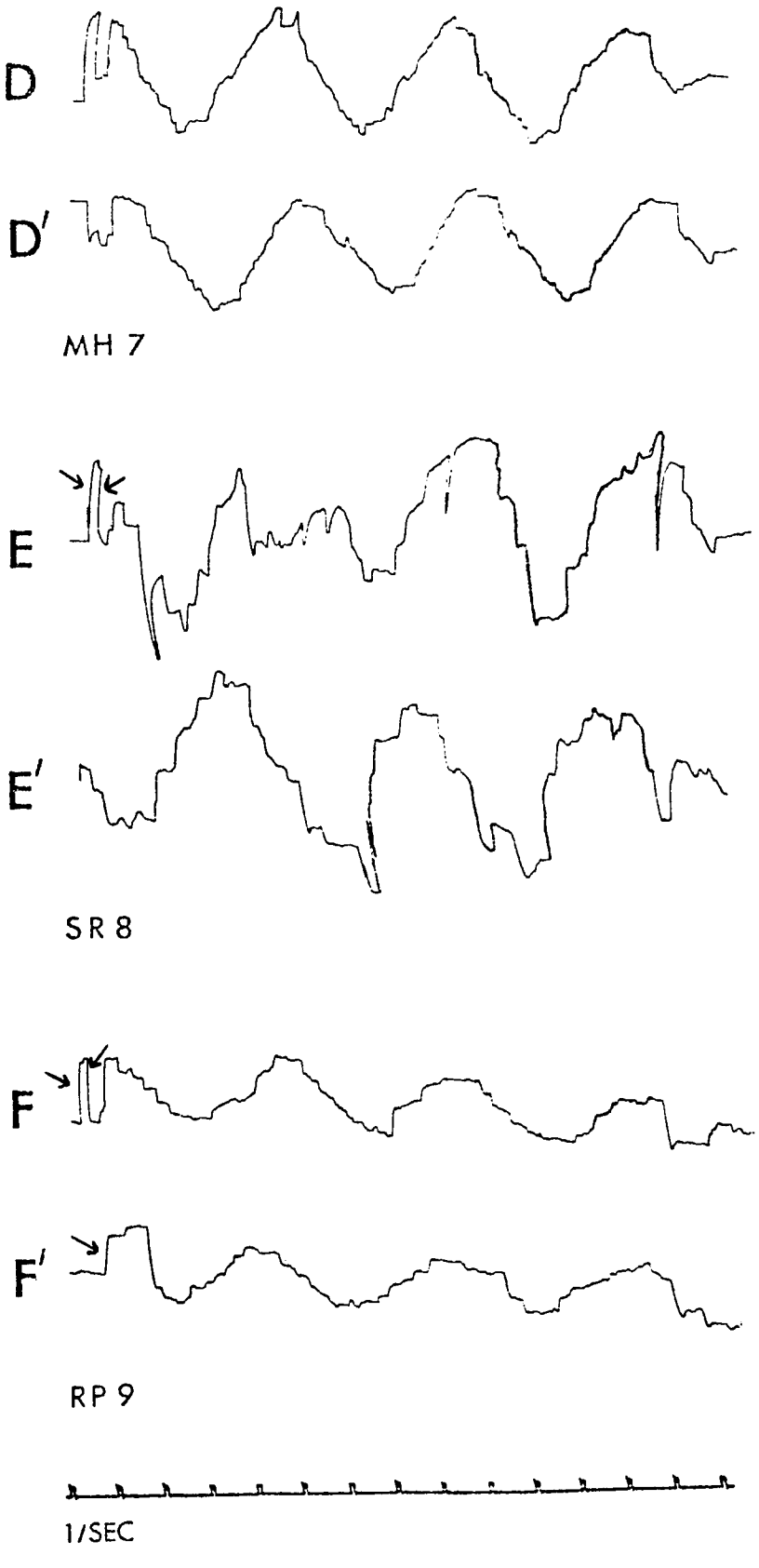
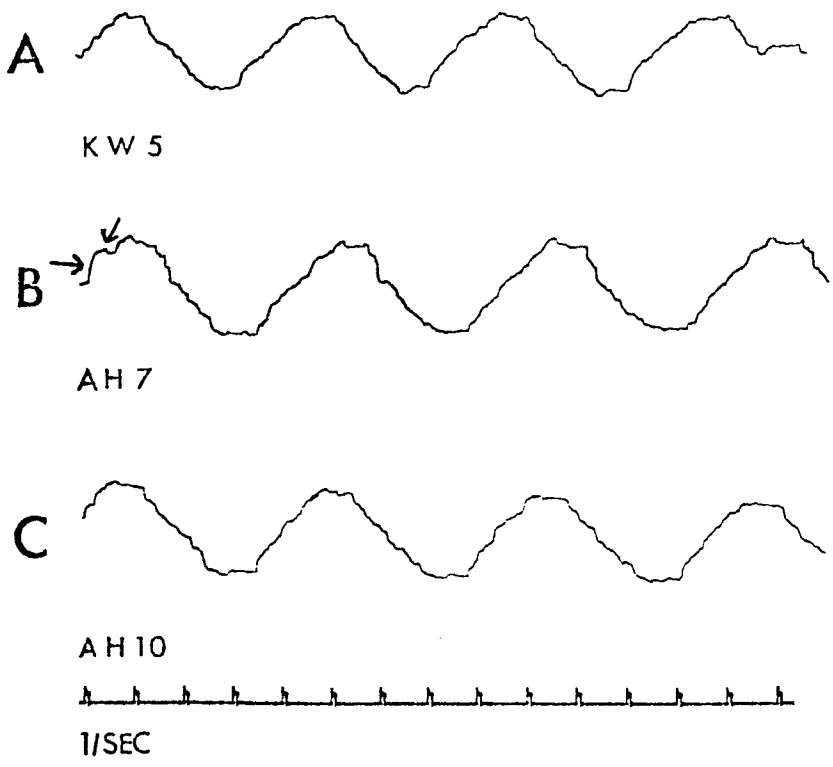
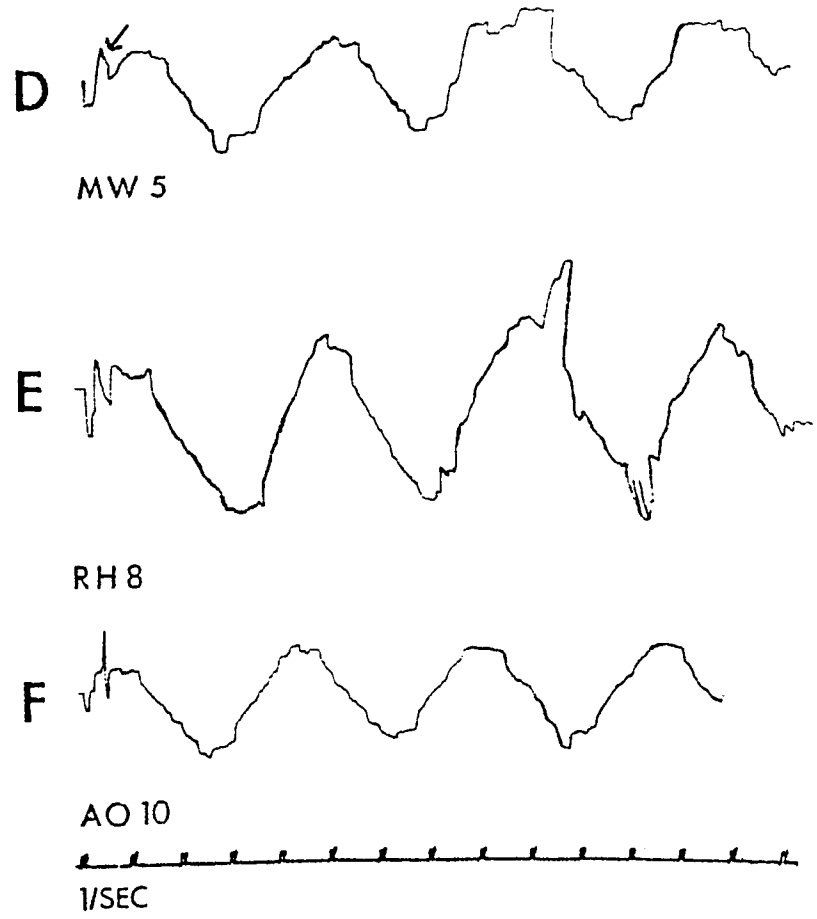


Fig. 6. Typical pursuit records of normal and hyperkinetic subjects for the second pursuit task. Normals made few large saccadic eye movements away from the pursuit track. Their pursuit performance was characterized by many small saccades along the target path and periods of smooth following (A-C). Hyperkinetics often had large saccades away from the pursuit track (D, E). Hyperkinetics frequently paused longer after the stimulus had changed direction and then made larger forward saccades towards the target path (D, E).

NORMAL SECOND PURSUIT



HYPERKINETIC SECOND PURSUIT



changes in the stimulus with appropriate pursuit movements for at least part of the test. Furthermore, both normals and hyperactives showed periods of fixation whose times of occurrence and durations corresponded with the actual times the stimulus was known to stop its movement. Therefore, although tracking was not performed equally well by all children, there was indication that both normal and hyperkinetic children could track appropriately.

Qualitative analysis of the pursuit records showed that many normal and hyperkinetic children had large forward saccades occurring within the first second after stimulus onset. In some records, these saccades occupied the entire initial 15° pursuit segment to the right (arrows, Figure 4B', D, E, E'). In other records a forward saccade that overshot the target was followed by a backward saccade onto target. In these records, a larger proportion of the slow pursuit performance during the initial 15° segment was preserved (arrows, Figure 5B; Figure 6B, D). Finally, a distinct group of records evidenced large to and fro saccades occurring during the initial pursuit segment. This type of response was seen more often in hyperkinetic children than in normals and usually occurred during both pursuit tasks (Figure 4A, A'; Figure 5C, C', D, D', F).

In part, these initial responses may be linked to an element of surprise. That is, the calibration task immediately preceding the first pursuit presentation had re-

quired the subject to make 15° and 30° saccadic eye movements in the horizontal and vertical planes, and the subjects had no prior information about the pursuit task. However, the initial saccades could also have been an individual response to a new stimulus since many of the children made almost exactly the same response when the test was repeated 20 to 30 minutes later.

The pursuit task was given twice, once at the beginning and once at the end of the test session in order to replicate the findings of the initial test. It seemed reasonable to suppose that the children might modify their tracking behavior so that it was smoother and more efficient on a second occasion. Figure 4 contrasts pursuit performance between first and second pursuit tasks for the normal group. The records shown in this figure represent the most erratic pursuit of the normals. In these and other normals, a consistent finding was that they made fewer and smaller saccades on the second pursuit presentation than on the first. Record A of Figure 4 is the first pursuit test of a 6-year-old child. There were many large saccades away from the pursuit track and in the third cycle of Figure 4A the child no longer pursued the stimulus. However, on the second presentation (Figure 4A'), pursuit was smoother and there was not the same interruption of tracking during the third stimulus cycle. Record 4B is from a 7-year-old boy. It was punctuated by many saccades which occurred during all cycles of the first

pursuit task. During the second pursuit task (Figure 4B'), there were fewer saccades, especially during the last two stimulus cycles. The same was true for the records shown in Figure 4C-E. It is of interest to note that despite the decrease in eye movements, there were basic similarities between the first and second tests and saccades tended to occur in the same place. The arrows in records D and D' of Figure 4 from an 8-year-old show similar saccadic movements occurring in the second pursuit cycle, when the subject was changing pursuit direction from left to right and again from right to left. Many other examples of saccadic interruptions occurring at the same place during smooth pursuit in other normals can be seen in Figure 4A-E.

Another interesting trend which was apparent on examination of records was that the normal subjects with the highest IQs, i.e., those from 118 to 138, made more saccadic eye movements than children with somewhat lower IQs, i.e., those from 96 to 117. This was true on both the first and second pursuit tasks. However, both groups of normal children showed a drop in the number of saccades on the second pursuit task. Records 4D and D' are from an 8-year-old with an IQ of 96. There were 15 saccades on the first pursuit task and 14 on the second. In comparison, records from a child with an IQ of 135 are shown in Figure 4E and E'. He had 30 saccades on the first pursuit task and 24 on the second.

Figure 5 shows typical examples of pursuit for the hyperkinetic group. During the first pursuit task many of the hyperactives made a large number of saccades. Some of these were large-amplitude saccades which clearly took the eyes away from the pursuit path, sometimes necessitating backward saccades to bring the eyes back onto the target (Figure 5A', B, B', C, C', E, E'). Further inspection of the records showed a greater frequency of backward saccades in hyperkinetics than in normals. A forward saccade is a saccade in the direction of pursuit, usually to bring the eye on target. A backward saccade is a movement in the direction opposite to the target movement. While it can be used to bring the eye onto the target, more often it tends to shift the direction of gaze entirely away from the target (Figure 5B, second cycle).

Some boys in the hyperkinetic group showed habituation from first to second pursuit task, i.e., they made fewer movements and pursuit was somewhat smoother. Figure 5B and B' from a 5-year-old depicts a marked decrease in the number of large saccadic eye movements away from the target path from first to second pursuit. However, in others the second pursuit performance was worse than the first. Figure 5C and C' from a 5-year-old illustrates such an instance. During the first pursuit presentation, the subject followed the stimulus for about two complete cycles. This was somewhat more than during the second pursuit task. Again, it is of interest to note that a

leftward sustained deviation of the eyes occurred in approximately the same place on both records (see arrows, Figure 5C and C'). Figure 5E and E' from an 8-year-old shows an example of improvement from the first to the second pursuit task. During the first presentation, the subject did not appear to follow the stimulus until about the third cycle. On the second presentation, he started pursuing the stimulus almost immediately. Although there was generally improvement in the hyperactives' pursuit performance, it fell considerably short of that shown by age-matched normals. Figure 5D and F shows the best pursuit among hyperactives.

Typical pursuit records of both normal and hyperkinetic subjects (age range 5-10 years) for the second pursuit task are shown in Figure 6. The normals made very few large saccadic eye movements away from the pursuit track (Figure 6A-C). Their pursuit performance was characterized by many small saccades along the target path and they had many periods of smooth following. Pursuit efficiency did not appear to be age dependent. Figure 6A from a 5-year-old shows very little saccadization of pursuit during the second and fourth pursuit cycles to the right. Figure 6B from a 7-year-old and 6C from a 10-year-old show similar pursuit performances. They made few saccades during the third and fourth pursuit cycles to the right. Records in Figure 4A'-E' from normal children also show similar characteristics. In contrast, hyperactives ranging from

5 to 10 years of age often had large saccades away from the pursuit path (Figure 6D and E). Additional examples are shown in Figure 5A', B', C', and E'. None of the normal children had similar large saccades away from the target in the second presentation, particularly towards the end of pursuit. In hyperkinetics, as in normals, pursuit efficiency did not appear to be age dependent.

One other characteristic of following which was present in both normals and hyperkinetics was that the eyes often did not begin to move at stimulus velocity concurrently with the reversal in stimulus direction. Frequently there was a short period of fixation at the time when the stimulus reversed direction followed by a forward saccade onto the target. This tendency can be seen at least once in almost every one of the normal records. An example is shown in Figure 6C each time the stimulus moved from left to right. This tendency was exaggerated in the hyperkinetic children, who frequently paused longer after the stimulus had changed direction and then made larger forward saccades towards the target path. Figure 6D shows an example of this. Saccades occurred on directional change during the first, second, and third stimulus cycles. Record E depicts a large saccade occurring during a direction change in the third cycle of pursuit. Record F from a 10-year-old approximates more closely a normal record, with smaller saccades occurring during directional changes.

In summary, qualitatively there were the following

differences in results:

(1) Normals appeared to have fewer and smaller saccadic eye movements during pursuit than did hyperkinetics.

(2) As a group, normals tended to make fewer saccadic eye movements in the second pursuit presentation.

(3) Overall, hyperkinetics did not show a marked decrease in eye movements from the first to second presentations.

(4) Hyperkinetics had more eye movements away from the pursuit path than normals.

(5) Some hyperkinetics showed more difficulty in tracking during the third and fourth pursuit cycles.

(6) Relative to normals, hyperkinetics seemed to make more and larger eye movements just following a change in stimulus direction.

Quantitative.

Data distributions. Two factors were considered for data analysis, the number and the amplitude of saccadic eye movements made during pursuit. As detailed in the methods section, the EOG voltages during pursuit were read into the computer, which digitized them, identifying and marking each movement. The digitized data were subsequently displayed on a storage scope to verify that the movements had been marked correctly. Then the number and amplitudes of horizontal movements were processed by the computer using appropriate statistical procedures.

Figure 7 displays the distribution in the number of

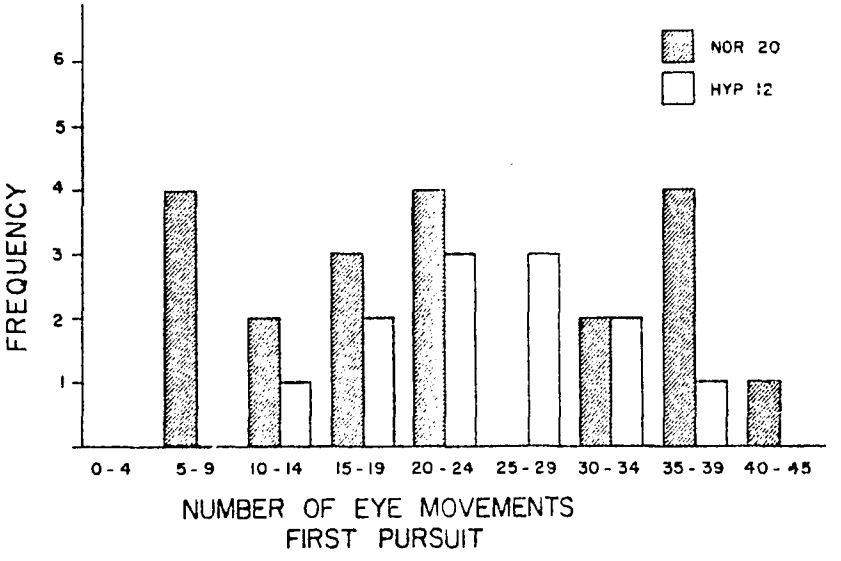


Fig. 7. Distribution of the number of saccadic eye movements made by normals and hyperkinetics during the first pursuit task. The abscissa is divided into nine equal-width intervals with each interval being equal to an increment of five eye movements. Each bar represents the frequency of cases which fell into that interval. The normals' distribution (n = 20) had a range of 7-41; mean, 22; sd, 11. The hyperkinetics' distribution (n = 12) had a range of 12-39; mean, 25; sd, 7.

saccadic eye movements made by normals and hyperkinetics during the first pursuit. The abscissa is divided into nine equal-width intervals, with each interval being equal to an increment of five eye movements. Each bar represents the frequency of cases which fell into that interval. In the normal group (n = 20) the distribution of eye movements has the following characteristics: range, 7-41; mean, 22; sd, 11. The hyperkinetic group's (n = 12) distribution is shifted to the right and has the following characteristics: range, 12-39; mean, 25; sd, 7. Thirty percent (six cases) of the normal distribution falls below the hyperactives' distribution and the mean is less although there is no significant difference ($\underline{t}$ = .63, $\underline{p}$< .8).

Figure 8 displays the distribution of eye movements for the second pursuit presentation. The following characteristics describe the normal distribution: n = 17; range, 3-28; mean, 17; sd, 6. The normal population had its peak frequency in the 15-20 movement interval. The distribution characterizing the hyperkinetics' performance was shifted to the right (n = 14; range, 14-39; mean, 24; sd, 7). The hyperkinetics showed a peak frequency of cases in the 25-30 movement interval. The insert in Figure 8 demonstrates the substantial and significant drop in the number of eye movements from first to second presentation for the normal group (dependent $\underline{t}$ = 2.65, $\underline{p}$< .01). Hyperkinetics showed very little change in the number of eye

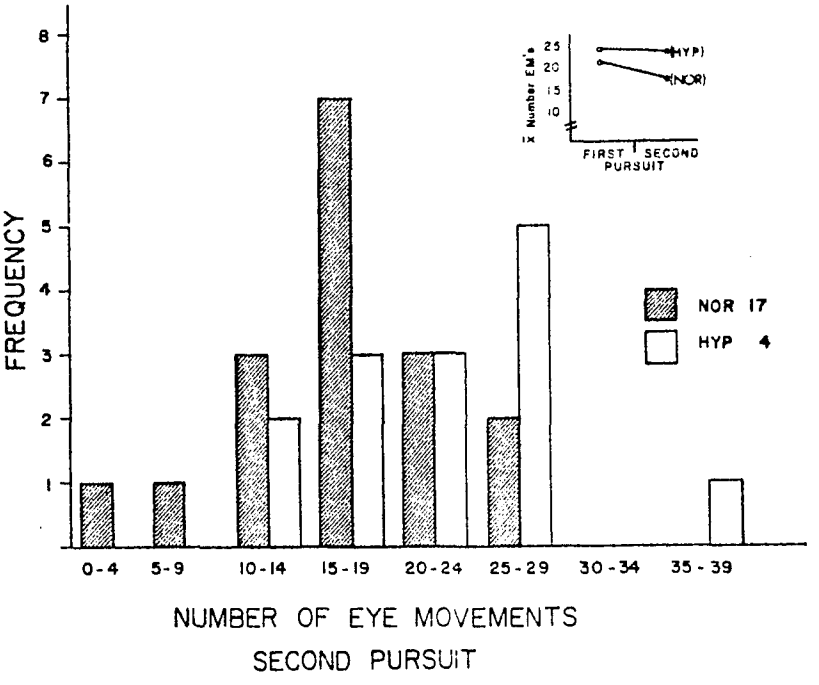


Fig. 8. Distribution of the number of saccadic eye movements made by normals and hyperkinetics during the second pursuit task. Schema as in Fig. 7. The normals' distribution (n = 17) had a range of 3-28; mean, 17; sd, 6. The hyperkinetics' distribution (n = 14) had a range of 14-39; mean, 24; sd, 7.

movements over both presentations.

Thus, normals made fewer eye movements than hyperkinetics and normals had a significant decrease in the number of eye movements made on the second pursuit task. Hyperkinetics did not evidence a similar decrease. Normals demonstrated a substantial drop in variability over tasks while response variability remained the same in hyperactives.

Figure 9 shows the distribution of mean amplitudes for the first pursuit presentation. The abscissa is divided into equal-width intervals, each one representing an increase of 1° over the preceding interval. The normals' distribution has its peak frequency in the 3°-4° category. The distribution is slightly skewed to the right and can be described as follows: n = 20; range, 2.6°-7.2°; mean, 3.9°; sd, 1.2°. The hyperactives' distribution is shifted to the right and shows the following characteristics: n = 12; range, 2.9°-9.3°; mean, 5.2°; sd, 2.1°. As can be seen from the figure, there is some overlap between the tails of the two distributions. Normals had smaller eye movements than did hyperkinetics and showed less response variability ($\underline{t}$ = 2.34, $\underline{p}$< .05).

Figure 10 presents the distributions of mean amplitudes for the second pursuit. The normals' distribution again shows its highest frequency in the 3°-4° category. This group's distribution is characterized as follows: n = 17; range, 2.8°-6.2°; mean, 4°; sd, .8°. The hyper-

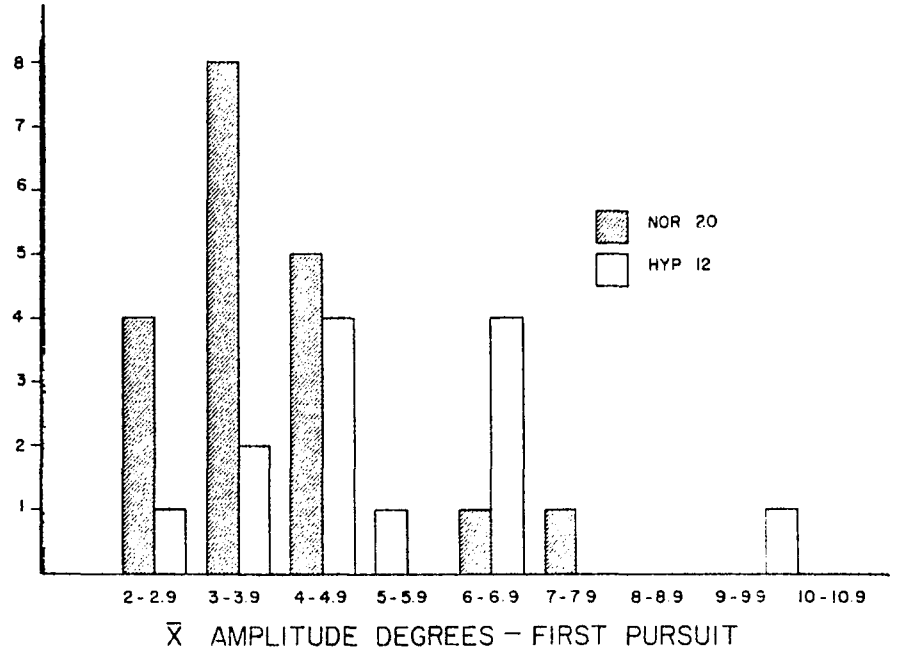


Fig. 9. Distribution of saccadic mean eye movement amplitudes for normals and hyperkinetics during the first pursuit task. The abscissa is divided into equal-width intervals, each one representing an increase of 1° over the preceding interval. The normals' distribution (n = 20) had a range of 2.6°-7.2°; mean, 3.9°; sd, 1.2°. The hyperkinetics' distribution (n = 12) had a range of 2.9°-9.3°; mean, 5.2°; sd, 2.1°.

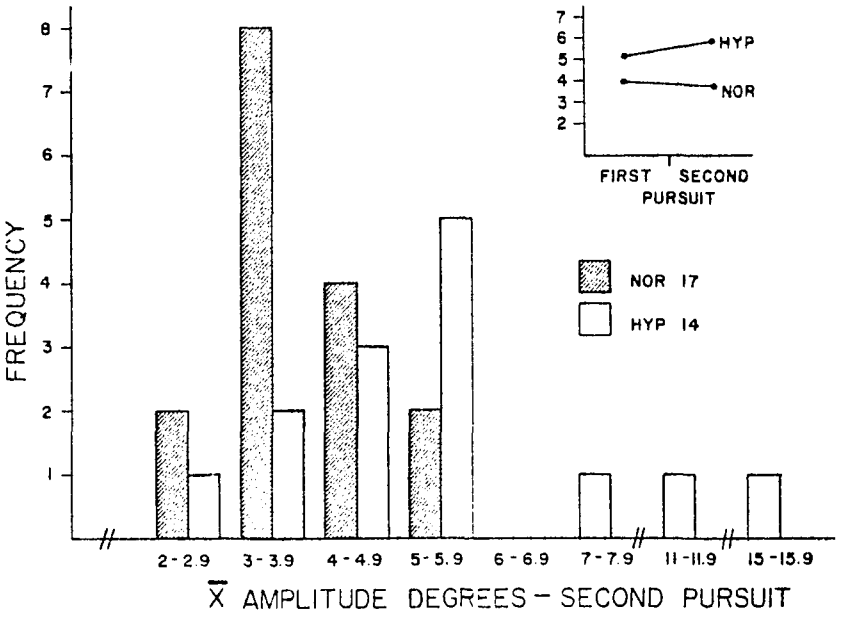


Fig. 10. Distribution of saccadic mean eye movement amplitudes for normals and hyperkinetics during the second pursuit task. Schema as in Fig. 9. The normals' distribution (n = 17) had a range of 2.8°-6.2°; mean, 4°; sd, .8°. The hyperkinetics' distribution (n = 14) had a range of 2.9°-15°; mean, 5.9°; sd, 3.3°.

kinetics' distribution is shifted to the right, as in the first pursuit task. It may be characterized as having one category of peak frequency occurring at 5°-6°. Furthermore, it is characterized by n = 14; range, 2.9°-15°; mean, 5.9°; sd, 3.3°. The insert indicates that the hyperkinetic group increased its mean amplitude of eye movement from the first to the second pursuit. Normals demonstrated a slight decrease in their mean amplitude responses over the two presentations. Increases in both groups failed to reach significance across presentations.

In conclusion, normals made smaller mean amplitude saccades than did hyperactives during both pursuit presentations. Normals also demonstrated less response variability than hyperactives. In fact, hyperactives as a group increased their response variability on the second pursuit task. Finally, unlike the eye movement frequency data, mean amplitude responses were found to remain relatively constant in both groups over test presentations.

Correlational analysis. Relationships between the number of eye movements made on the first and second pursuit tasks as well as the mean amplitudes of eye movements made on the first and second pursuit tasks were studied using Pearson product moment correlation coefficients. The correlation coefficient provides an indication of the degree to which the number and/or amplitudes of eye movements made on the first test are similar to or can be related to the number or amplitudes of

eye movements made on the second test. Furthermore, it was of interest to see if the data shifted from one presentation to the other and, if so, in what direction the shift occurred.

Figure 11 is a plot of number of eye movements on first versus second pursuit for the normal population (n = 17[2]). If there were no response change from first to second pursuit, each data point would fall on the 45° solid line bisecting the graph. Points above this line indicate more eye movements on the second test while points below the line indicate fewer eye movements on the second test. If the 45° diagonal passed through a data circle, whose scaled diameter on the ordinate represented one eye movement, the circle was considered as lying on the 45° line, indicating no change. Results showed that the number of eye movements on the first and second tests were significantly correlated (r = .47, p< .05). The equation for the linear least-squares regression line was y = .30x + 10.5. Thirty-five percent (six of 17 subjects (6/17)) showed no change from first to second pursuit. Fifty-three percent (9/17) decreased their number of eye movements on the second pursuit task while 12% (2/17 subjects) increased their number of eye movements.

Number of eye movements on first pursuit versus number of eye movements on the second pursuit is plotted for the

[2]Three children were not used in this analysis because one of their two records was technically unsatisfactory.

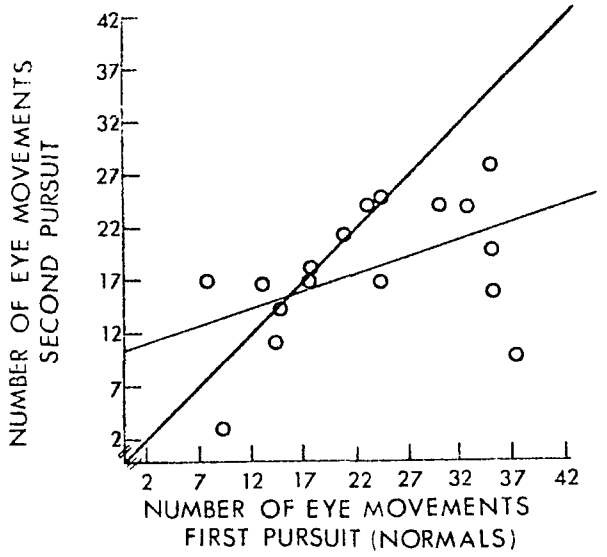


Fig. 11. Number of eye movements on first versus second pursuit for the normal sample (n = 17). The 45° solid line bisecting the graph is a reference line. Points above this line indicate more eye movements on the second than on the first test and vice versa. When the 45° diagonal passed through a data circle, the circle was considered to be on the 45° line, indicating no change. The second line represents the line of best fit and was determined by the least-squares method. A significant correlation was found between the number of eye movements on the first and second tests (r = .47, $p < .05$).

hyperactives in Figure 12 (n = 12). A perfect correlation between the response measures is represented by the solid line bisecting the graph. A significant correlation was also found in this group between first and second pursuit measures ($\underline{r}$ = .65, $\underline{p}$< .05). The second solid line represents the linear regression line calculated by the least-squares method and is described by the equation $y = .62x + 8.95$. Unlike the normals, a dependent $\underline{t}$ test failed to yield a significant difference between first and second pursuit performances. Fifty-eight percent (7/12) of the patients increased their number of eye movements from first to second presentation while 42% (5/12) decreased their number of eye movements.

In summary, these graphs show a correlation between the number of eye movements made on the first and second pursuit tasks for both groups. The correlations indicate that fewer eye movements on the first task are associated with fewer eye movements on the second pursuit task. The correlation was lower in normals than in hyperkinetics, which may reflect in part the greater response variability seen in the group on the first pursuit task. However, a test between the two correlations yielded a value of $z' = .50$, $\underline{p}$< .9, indicating that the two correlations were not significantly different.

The mean amplitude of eye movements on the first pursuit versus the second pursuit for the normal group is plotted in Figure 13 (n = 17). When the 45° line (solid

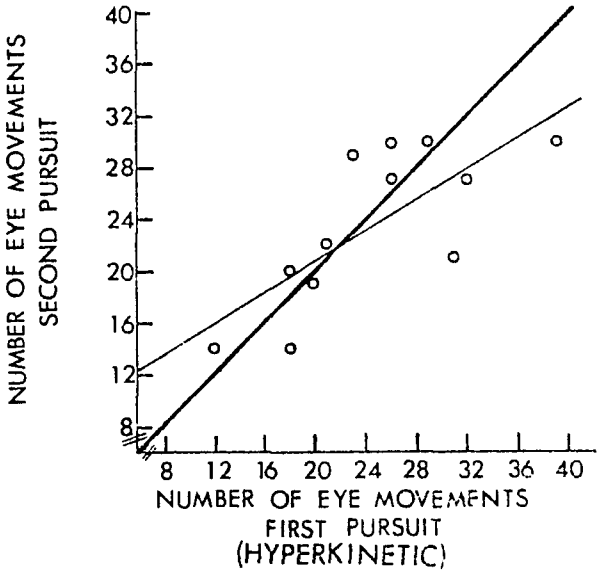


Fig. 12. Number of eye movements on first versus second pursuit for the hyperkinetic sample (n = 12). As in Fig. 11, the 45° solid line bisecting the graph is a reference line. The second line represents the line of best fit and was determined by the least-squares method. A significant correlation was found between the number of eye movements on the first and second tests (r = .65, $p < .05$).

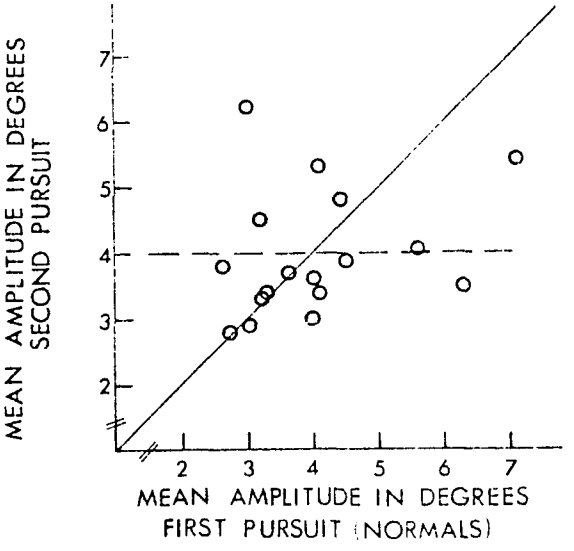


Fig. 13. Mean amplitude of saccadic eye movements on first versus second pursuit for the normal group (n=17). Points above the 45° line bisecting the graph indicate larger saccades on the second test and vice versa. The dashed line at 4° represents the average amplitude for the normal group on both pursuit tasks. Twenty-nine percent (5/17) had saccadic movements of 4° or larger.

diagonal) passed through a data circle, whose scaled diameter on the ordinate represented .2°, that data point was considered as lying on the 45° line, indicating no change. Points above this line indicate larger-amplitude movements on the second test while points below the line indicate that the amplitudes on the second test were smaller. Mean amplitudes on the first and second pursuit performances were not significantly correlated in this group (r = .28, p< .90). Twenty-nine percent (5/17) of the normal population increased their mean amplitude response on the second pursuit while 42% (7/17) decreased their responses. Approximately 29% (5/17) showed substantially no change. As previously shown (see Figure 9 insert), the normal group's mean amplitude response changed very little across pursuit tasks. This average response was used as a reference line for both the normal and hyperkinetic data (dashed line). Data points intersected by the line were considered to fall at the mean (4°). Twenty-nine percent (5/17) evidenced mean amplitude of eye movements greater than 4° on the second pursuit task.

Figure 14 presents the same plot for the patient group (n = 12). There was a significant correlation between mean amplitudes of eye movements for the first and second pursuit tasks (r = .51, p< .05). The least-squares linear regression equation which best fit the data was $y = .89x + .99$. Fifty percent (6/12) decreased their mean amplitude from first to second pursuit while the

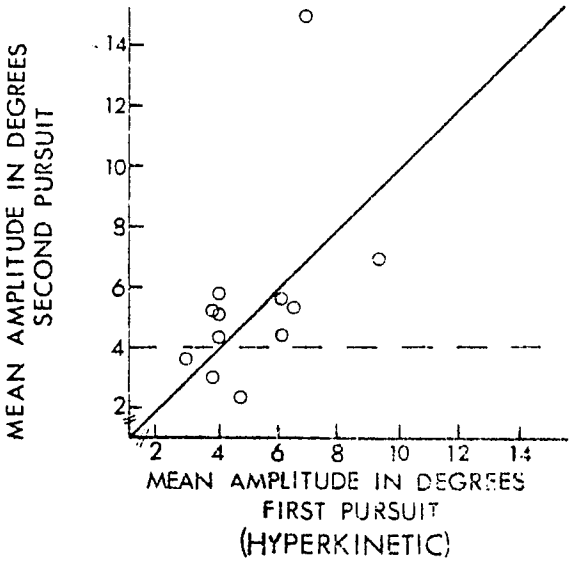


Fig. 14. Mean amplitude of saccadic eye movements on first versus second pursuit for the hyperkinetic group (n = 12). As in Fig. 13, points above the 45° line bisecting the graph indicate larger saccades on the second test and vice versa. There was a significant correlation between mean amplitude of eye movements for the first and second pursuit tasks (r = .51, $p < .05$). As in Fig. 13, the dashed line at 4° represents the average amplitude for the normal group on both pursuit tasks. Seventy-five percent (9/12) had saccadic movements of 4° or larger.

other 50% (6/12) increased their mean amplitude response. Seventy-five percent (9/12) of the mean amplitudes for the hyperkinetic cases were larger than 4° on the second pursuit task.

To summarize, these graphs demonstrate that more normals made eye movements whose amplitudes were below 4° on the second pursuit than did hyperactives. Secondly, they also show a significant correlation between mean amplitude of eye movements across tasks for hyperkinetics but not for normals. That is, for normals, the size of amplitude responses on the first test was not systematically associated with the size of amplitude responses on the second test.

ANOVAR and ANCOVAR analysis. Initially, the use of a single ANCOVAR design including all factors, covariates, and their interactions was considered for analysis of the data. Since there were missing data on some subjects (notably IQ data), an attempt to use an all-inclusive design would have resulted in a large reduction in sample size on which to base differences between groups. In light of this, it was decided to used more limited ANOVAR and ANCOVAR procedures with which to analyze the effects of factors and covariates. Three factors were considered in the ANOVAR-ANCOVAR statistical analysis. They were diagnosis, IQ, and drug status.

Usually, ANCOVAR procedures are used to remove extraneous variation from the dependent variable by

introducing a metric covariate. This procedure results in increasing measurement precision. In the present analysis, however, both covariate and factor effects were of interest. To evaluate both covariate and factor effects, the data were analyzed using regression analysis techniques which are resident within the ANCOVAR program. This approach allowed all effects to be assessed simultaneously, with each effect being adjusted for all other effects. Thus, the proportion of variance in the dependent variable which was the unique contribution of each factor and covariate was isolated. The significance of each factor and covariate effect was then tested by forming the appropriate F ratios.

The effect of IQ on the number of eye movements made by normals and hyperkinetics during the second pursuit task is shown graphically in Figure 15. The number of eye movements on the second test is plotted on the ordinate and the WISC Performance IQ on the abscissa. Only the normal group showed a significant correlation between the two variables ($n = 14$, $r = .69$, $p < .01$). The equation describing the linear regression line for normals was calculated by the least-squares method, yielding $y = .36x - 22.9$. Data obtained from hyperkinetic subjects were subsequently compared to values predicted by the regression equation. The solid lines connecting some of the data points to the regression line represent the standard deviations of these points after the estimated

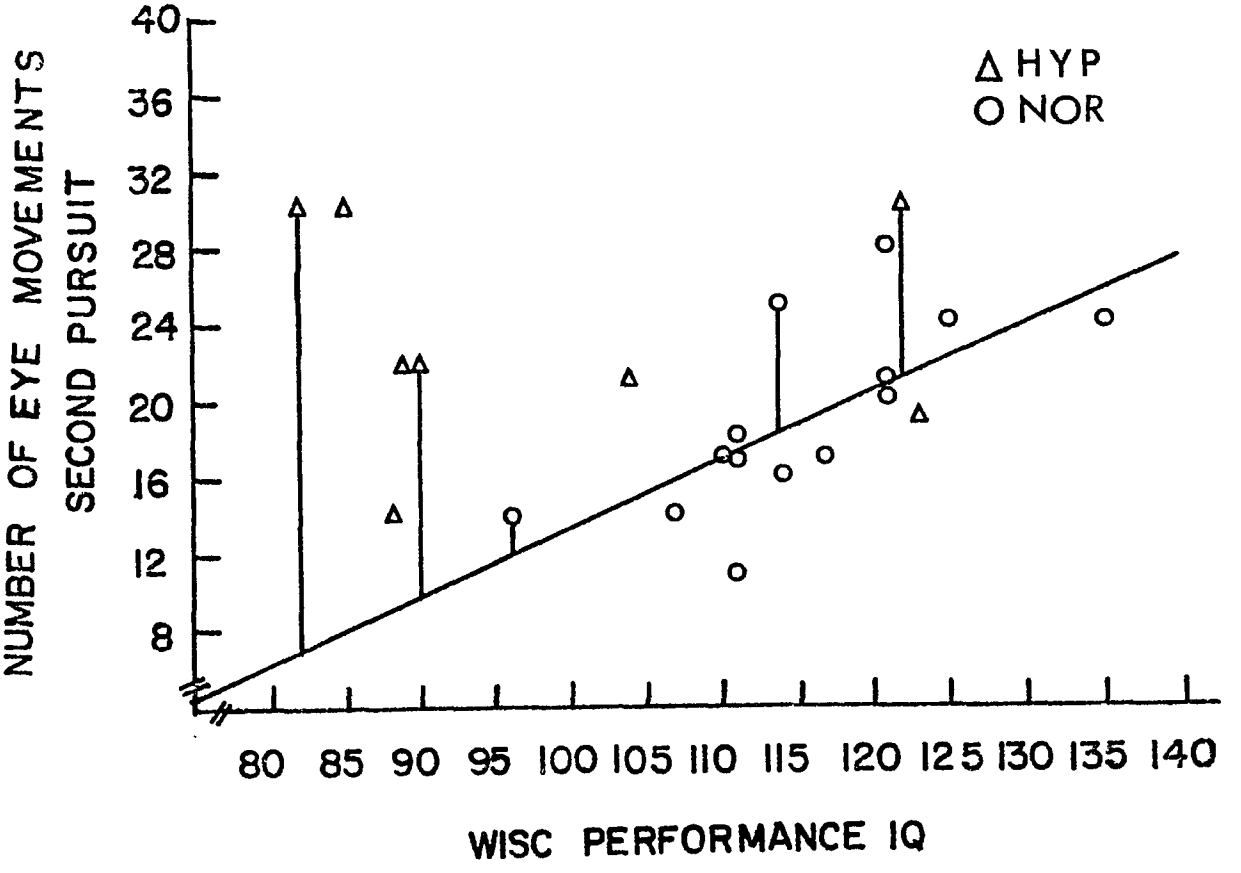


Fig. 15. Number of eye movements made on the second pursuit task by normals and hyperkinetics as a function of the WISC Performance IQ score. There was a significant correlation between the two variables for the normal population ($n = 14$, $r = .69$, $p < .01$). The solid lines connecting some of the data points to the regression line represent the standard deviations of these points after the estimated effect of IQ was removed.

effect of IQ was removed. A *t* test comparing the mean standard deviations between the normal and hyperkinetic groups yielded a highly significant difference ($t = 4.129$, $p < .005$). This finding demonstrates that after the estimated effect of IQ on number of eye movements made on the second pursuit test was removed statistically from the data of both groups, the hyperkinetic sample still demonstrated a highly significant mean deviation from the regression line relative to controls. This finding could be attributed to the factor of diagnosis. In addition, an ANCOVAR was run on the data to determine the effect of diagnosis using both age and IQ as the covariates. Both covariates were found to be nonsignificant while the main effect for diagnosis was significant: $F(1,20) = 5.395$, $p < .03$. Significant interaction between diagnosis and Performance IQ was not supported by these findings.

To study the effect of varying levels of IQ on the response measures for the normal population, a one-way ANOVAR was performed. Results are presented in Table 2. A significant *F* ratio was found for number of eye movements made during the second pursuit task: $F(1,12) = 11.478$, $p < .01$. The high-IQ group (118-138) had a greater mean number of eye movements ($n = 5$; mean, 23.4) than did the middle-IQ (96-117) group ($n = 9$; mean, 16.6). As previously indicated, eye movement amplitudes were identical for both groups. Inspection of the data suggested that age might be an important determining factor of number of

TABLE 2

ANALYSIS OF VARIANCE:

WISC PERFORMANCE--NORMALS

Source	df	MS	F
Between groups	1	150.5742	11.478*
Within groups	12	13.1188	

*$p < .01$.

eye movements on the second pursuit task in the normal group.

To segregate the effect of age on the number of eye movements made on the second pursuit task in both the normal and hyperkinetic samples, an ANCOVAR was performed for each group using age as the covariate. Table 3 shows main effects for IQ to be highly significant in the normal group: $F(1,11) = 11.067$, $p < .01$. The covariate age was found to be nonsignificant. A separate ANCOVAR with IQ as the covariate yielded no significant main effects of age on number of eye movements. No relationship between IQ or age and numbers of eye movements was found in the hyperkinetic group.

The effect of diagnosis on the number and amplitudes of eye movements was studied in three separate ANOVARs. The results are presented in Table 4. The F ratio shows a significant effect of diagnosis on mean amplitudes of eye movements for the first pursuit task: $F(1,30) = 5.5582$, $p < .03$. The hyperactive group had a significantly greater mean amplitude of eye movement (n = 12; mean, 5.2°) than the normal group (n = 20; mean, 3.9°). The F ratio for number of eye movements made during the second pursuit presentation was highly significant: $F(1,29) = 7.939$, $p < .01$. Hyperkinetics made more eye movements (n = 14; mean, 23.9) than did normals (n = 17; mean, 17.4). The calculated F ratio for mean amplitude of eye movements made during the second pursuit presentation shows a sig-

TABLE 3

ANALYSIS OF COVARIANCE:

WISC PERFORMANCE WITH AGE--NORMALS

Source	df	MS	F
Covariate age	1	12.922	.984
WISC	1	145.383	11.067*
Residual	11	13.136	

*$p < .01$.

TABLE 4

ANALYSIS OF VARIANCE: DIAGNOSIS

Dependent Variable	Source	df	MS	F
Mean amplitude eye movement, first pursuit	Between groups	1	12.0967	5.582*
	Within groups	30	2.1671	
Number of eye movements, second pursuit	Between groups	1	326.0508	7.939**
	Within groups	29	41.0708	
Mean amplitude eye movement, second pursuit	Between groups	1	28.2822	5.249*
	Within groups	29	5.3885	

*$p < .03$.
**$p < .01$.

nificant effect of diagnosis: $F(1,29) = 5.249$, $p < .03$. Hyperactives again had larger mean amplitude of eye movements (n = 14; mean, 5.9°) than did normals (n = 17; mean, 4°).

In order to study further the effects of multiple independent variables on the response measures, a 2 (normal, hyperkinetic) X 2 (on-drug, off-drug) with age ANCOVAR using regression techniques was run. Table 5 presents the results of this analysis. For all dependent-variable measures (number of eye movements, mean amplitude of eye movements), the covariate age and the factor of drug status were found to be nonsignificant. There were significant main effects for diagnosis on three dependent measures. The computed F ratio shows a significant effect of diagnosis on the mean amplitude of eye movements made during the first pursuit task: $F(1,25) = 6.461$, $p < .02$. Adjusting the means of both groups for the effects of age and drug showed that the mean eye movements were smaller in normals (n = 20; mean, 3.8°) than in hyperkinetics (n = 12; mean, 5.5°). The F ratio showed there was a significant difference in the number of eye movements on the second pursuit task: $F(1,25) = 4.925$, $p < .04$. The adjusted means of both groups show that normals made a smaller number of eye movements (n = 17; mean, 17.5) than hyperkinetics (n = 14; mean, 24.2). The F ratio for mean amplitude of eye movements made during the second pursuit was significant: $F(1,25) = 4.767$, $p < .04$. Adjusted

TABLE 5

ANALYSIS OF COVARIANCE:

DIAGNOSIS BY DRUG WITH AGE

Dependent Variable	Source	df	MS	F
Mean amplitude eye movement, first pursuit	Covariate age	1	.362	.161
	Drugs	1	5.684	2.530
	Diagnosis	1	14.514	6.461*
	Residual	25	2.246	
Number of eye movements, second pursuit	Covariate age	1	4.258	.092
	Drugs	1	7.115	.154
	Diagnosis	1	228.053	4.925*
	Residual	25	46.309	
Mean amplitude eye movement, second pursuit	Covariate age	1	.844	.172
	Drugs	1	4.923	1.005
	Diagnosis	1	23.353	4.767*
	Residual	25	4.899	

*$p < .05$.

means indicate a smaller mean amplitude response for normals (mean, 3.9°) than for hyperkinetics (mean, 5.9°).

In summary, the results of these analyses show that the factor of diagnosis consistently differentiated between the two groups on three dependent measures: mean amplitude of eye movements made during the first and second pursuit tasks and the number of eye movements made during the second pursuit task. IQ was found to be a significant factor in normals but not in hyperkinetics. Drug and nondrug patients did not differ on the response measures. However, this is a very tentative result since the interaction between referral source and drug status could not be evaluated due to small sample size. Finally, high-IQ normals did not differ from low-IQ hyperkinetics in number of eye movements made during the second pursuit task.

Secondary Findings.

Qualitative analysis of other eye movement tasks demonstrated differences between the hyperactive and control groups. These were not analyzed in detail but will be briefly mentioned. During performance of the Seguin Form Board Test, which was done with the head free, hyperactives tended to show more coupled head-eye movements than did normals. The coupled head and eye movements decreased in both groups as a function of age and number of test presentations. This finding suggests that relative to age-matched controls, hyperactives had an inability

to decouple eye and head movements when orienting to stimuli. Since these observations were qualitative in nature, the effect of IQ on this measure was not studied. It may well be that the number of head-eye coupled movements may be partially attributed to IQ.

It was also of interest to note that, relative to control subjects, some hyperkinetic children evidenced problems with staring. While controls were able to hold their eyes still in forward gaze for periods up to 45 seconds, many hyperkinetics were either unable to stare or demonstrated saccades within the initial 15 seconds of the task.

Chapter IV Discussion

This study shows differences in eye movements made during pursuit between hyperkinetics and normals and among normals of different IQs. Hyperkinetic children made larger saccades than normals during pursuit. They also had many large saccades away from the pursuit path and made more saccades during the second pursuit task than did normals. Normal boys with higher IQs tended to make more eye movements than those with lower IQs on both the first and second pursuit task. The implication of these results will be considered in light of physiological, attentional, arousal, and adaptational factors.

It is clear that forward saccades normally occur during pursuit of a visual stimulus (Dodge, 1903; Westheimer, 1954a). Saccades can therefore be expected as a normal concomitant of smooth pursuit movements. The flickering of the stimulus in the present experiment (24 frames per second) may have partly enhanced some of the saccadization, but similar saccades are also found when the stimulus is continuous (Stark, Vossius, & Young, 1962). In general, one would expect the saccades to be corrective, bringing the point of fixation onto target. These saccades could be either in the forward or backward direction. If the eyes moved exactly at the velocity of the stimulus, no corrective movements would be necessary. This implies that at various times eye velocity of normals and hyperactives was not matched to stimulus velocity.

Results indicated that high-IQ normal subjects made more eye movements than average-IQ normals on the second pursuit task. These results in the high-IQ group could derive from increased attention to stimulus flicker or to increased inspection of the stimulus. Comparison of results obtained using a continuous stimulus would obviate the flicker problem and give a purer measure of response rates of high-IQ versus average-IQ normals on a pursuit task. Regardless, this is an interesting finding for which there is no obvious explanation and which would appear to merit further research.

Most of the saccades made by hyperkinetics were along the target path. However, these saccades were typically larger than those of normal children. There are many lines of evidence to indicate that at least some hyperkinetic children have some brain dysfunction and in some this may be related to actual brain damage (Burks, 1955; Kahn & Cohen, 1934; Prechtl & Stemmer, 1962). Birch, Belmont, & Karp (1967) have reported that damaged nervous systems require more time to process sensory information. Therefore, the larger saccades along the pursuit track made by hyperkinetic children could reflect a neural processing defect in the slow movement system which is associated with minimal brain damage. In hyperactives, this could be due to defects in processing of afferent and/or efferent information. Such dysfunction could directly influence: (1) smooth movement reaction

times, and (2) estimation of eye velocity relative to stimulus velocity, both of which would contribute to saccadization of pursuit.

Evidence of altered reaction times in minimally brain damaged children has been reported by Cohen & Douglas (1972); Dykman et al. (1971); Stevens, Boydstun, Dykman, Peters, & Sinton (1967). The hyperactive children had longer response latencies than normals. If there were reaction time differences between hyperactives and normals, it would be expected to be most evident when the visual stimulus began its constant velocity movement from the initial rest position. Lengthened reaction times could also introduce lag times between stimulus movement and eye movement, necessitating saccades to b ing the eye onto target. In the present pursuit task, the visual stimulus stopped moving for approximately 750 msec before moving with a constant velocity in the opposite direction (see Figure 2). The hyperkinetics made large saccades when the stimulus changed direction more frequently than did normals during both trial presentations. These findings are consistent with the hypothesis that at least in part the poor pursuit performance of the hyperkinetics could be due to slow reaction times. The hyperkinetics also made more backward saccades than did normals during pursuit. This could also be due in part to incorrect estimation of stimulus velocity leading to corrective backward saccades, although it could also be due to dis-

tractibility, leading the subject to look at objects away from the target path.

Deficits in pursuit performance found in hyperkinetics have also been found in patients with Parkinson's disease, cerebellar diseases, drug intoxications, diseases of the retina or eye, and in children afflicted with cerebral palsy (Corin, Elizan, & Bender, 1972; Corvera, Torres-Courtney, & Lopez-Rios, 1973; Hoyt & Daroff, 1971; Jung & Kornhuber, 1964; Rashbass, 1959; Shackel, Davis, & Abercombie, 1962). In some cases the CNS lesions may affect the motor mechanism for pursuit. In other cases there may be difficulties in sensory processing, and in still others the mechanism(s) by which the pursuit system is affected is not well understood. Similar pursuit abnormalities have been reported in schizophrenics (Holzman, Proctor, & Hughes, 1973; Shagass, Roemer, & Amadeo, 1976) where attentional factors have been implicated. Consequently, it is difficult to be certain whether the findings in hyperactives were related to either organic and/or nonorganic factors.

Corvera et al. (1973) analyzed deformation of pursuit curves and associated different types of curves with separate disease processes. All of these types of records were observed in the hyperkinetic children. Figures 5 and 6 contain EOG tracings that correspond to Corvera et al.'s Type 1 curve. For example, in record D, D' of Figure 5 and record F of Figure 6 the waveform is essentially tri-

angular in shape but there are many small saccades which occur along the pursuit track. They associated this type of curve with cerebellar disease. Records B, B', C, C', E, E' of Figure 5 demonstrate the Type 2 curve, which they associated with diffuse cerebral and brain stem lesions. There is a partial loss of the triangular waveform accompanied by large saccades which show no particular pattern of occurrence. Figure 16 presents a record from a patient not in this study who had severe motor deficits. This record illustrates a Type 3 curve. This type of curve was found in patients with lesions involving the oculomotor pathways in the tegmentum of the pons and midbrain. In this record, the subject had no smooth pursuit and substituted many fast movements in both directions for the usual slow triangular tracking pattern. Corvera et al.'s classification is seriously weakened by an absence of comparative data from normals and a lack of anatomical verification in abnormals. Except in cases where there was severe disruption of pursuit such as in Figure 5E and E' and in Figure 16, it is probably not possible to attribute specific types of pursuit defects to structural lesions at the present time. The absence of "hard" neurologic signs in the hyperactives does not rule out brain damage as being a cause for poor pursuit performance. However, processing factors which could be related to chemical or structural brain lesions and attentional factors could account for most of the dif-

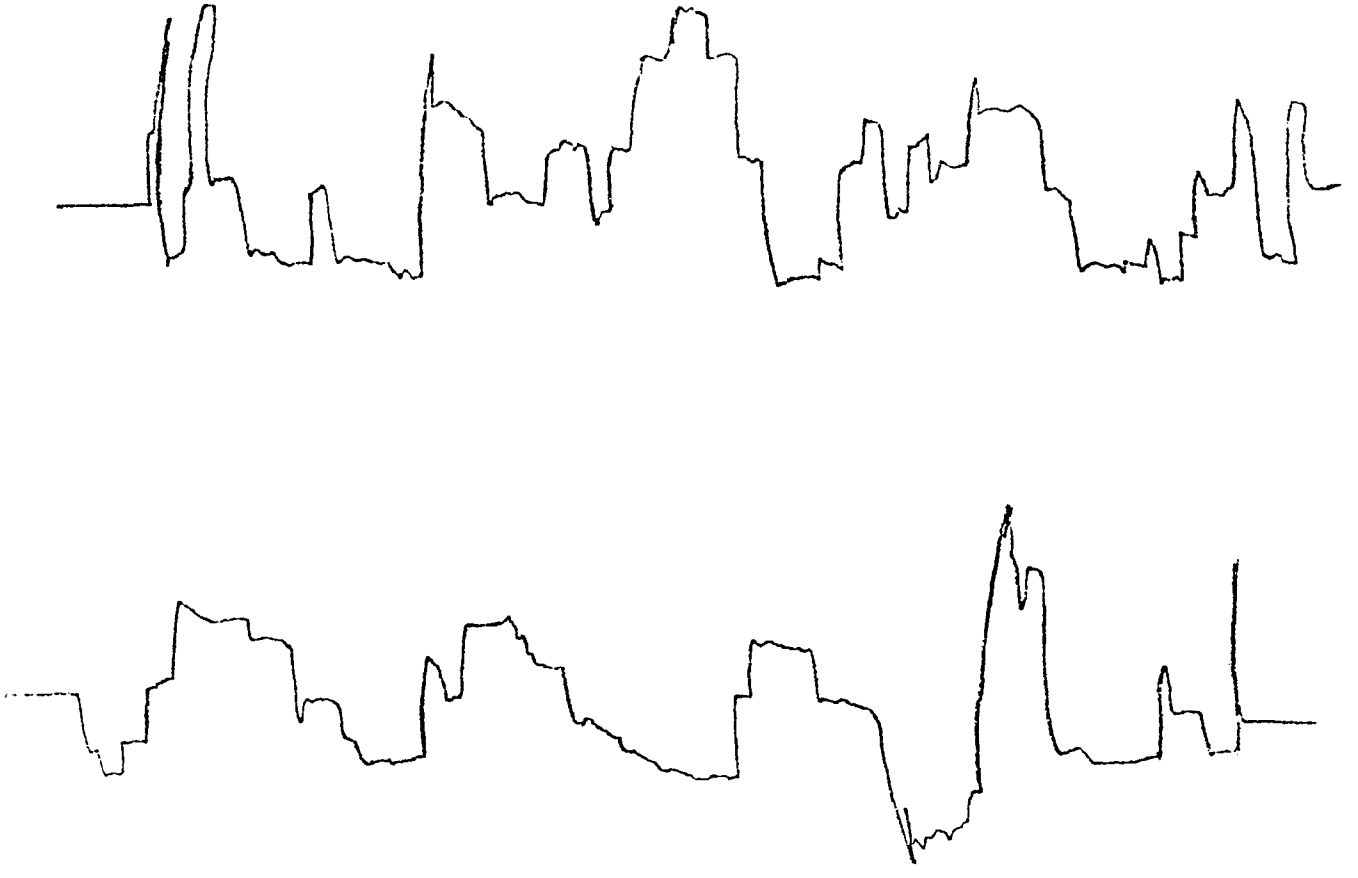


Fig. 16. Pursuit performance of a hyperactive girl with severe motor deficits who was not included in the present study. The subject had no smooth pursuit and substituted many fast movements in both directions for the usual slow triangular tracking pattern.

ferences which were found.

Attention has been shown to be important in pursuit performance. Saccadization of pursuit (not unlike that found in the present study) was reported in a mixed group of psychiatric patients (Shagass et al., 1976). With the introduction of a cognitive task during pursuit (i.e., reading numerals off the face of the pursuit stimulus), saccadization was markedly reduced. Similarly, Holzman, Proctor, Levy, Yasillo, Meltzer, & Hurt (1974) found that reminding their schizophrenic subjects to pay attention resulted in better pursuit performance. Therefore, some of the saccadization of pursuit in both normals and hyperactives could be the result of inattention.

Previous experimental evidence has shown that hyperkinetic children are unable to sustain long periods of attention (Grassi, 1970; Laufer et al., 1957). Sykes et al. (1973) found that hyperkinetics performed as well as normals on visual choice reaction tasks requiring the subject to attend for 3 to 4 seconds. However, hyperkinetics did significantly worse than normals on serial reaction tasks requiring sustained attention for up to 9 minutes. In the present study, the subject was required to maintain an uninterrupted focus of attention to the pursuit stimulus for 15 seconds (attention being defined operationally as the occurrence of pursuit and appropriate periods of fixation along the target path). Differences in the number and amplitude of eye movements between

normals and hyperkinetics in the present experiment may have been due in part to the inability of hyperkinetics to sustain attention during the experimental task. This is supported by the finding that hyperkinetics showed progressive deterioration of pursuit over time. That is, pursuit performance during the last third of the task was markedly inferior to earlier pursuit performance (see B, B', C, C', E' of Figure 5). No such decrement was observed in the normal population.

Deterioration of pursuit performance over time in the hyperkinetic sample could be related to Grassi's (1970) hypothesis that minimal brain damaged children accumulate more reactive inhibition than do normals. If correct, then pursuit responses would extinguish faster in hyperactives than in controls, possibly due to the attentional demands of the task. Attentional factors were not directly measured but could have contributed to the present experimental differences (Shagass et al., 1976). Moreover, many hyperkinetics had large saccades away from the pursuit path, some going beyond the limits of the stimulus to the edge of the screen. These saccades were clearly not corrective in nature and could not have been for purposes of stimulus inspection.

Several points suggest that not all saccades during pursuit were due to deficits of attention. For example, the number of eye movements made by high-IQ normal subjects on the second pursuit task was similar to that of

hyperkinetics. A representative record of a high-IQ (135) subject (Figure 4E') indicates that pursuit movements and fixation times occurred in phase with stimulus movements. There is no indication that this subject was not paying attention to the stimulus. Therefore, saccadization must have been related either to stimulus inspection or to target flicker. If the former, it may suggest that under some circumstances information processing is done in a sampled rather than continuous manner even during pursuit. Further studies are warranted to separate the effects of stimulus parameters from processing parameters.

The ability to maintain attention to a stimulus depends to a large degree on the arousal state of the organism. There are data to suggest both that hyperactives are overaroused (Laufer et al., 1957; Luria, 1960; Sokolov, 1963) and underaroused (Boydstun, Ackerman, Stevens, Clements, Peters, & Dykman, 1968; Grünewald-Zuberbier, Grünewald, & Rasche, 1975; Satterfield & Dawson, 1971). Either could be used as an explanation for some of the findings. The overaroused child would be highly distractible and respond to all environmental stimuli. During pursuit such a child could shift its attention to other stimuli, producing saccadic eye movements away from the pursuit path. The underaroused child might have eye movements away from the pursuit path which would serve to produce arousal by bringing new sensory input. These movements might be described as being active searching

movements. With two exceptions, there was no obvious indication of either under- or overarousal in most of the hyperactives. Measures of arousal (such as EEG and GSR) might be of use in future studies to determine the arousal state of the subjects.

One of the most interesting results of the present study was the failure of the hyperkinetic children to decrease significantly the number of saccadic eye movements from first to second pursuit presentation. Neither the normals nor hyperkinetics had a decrease in amplitude of saccades across presentations. There was more response variability in the hyperkinetic than in the control group, and some hyperkinetics, like the controls, did have fewer eye movements on the second than on the first presentation. However, as a group, there was not a significant decrease across trials. The decrease in the number of eye movements was considered to be habituation to the test since some saccadization might represent inspection of novel aspects of the stimulus. On repeated presentation, stimulus novelty should be decreased, resulting in a higher percentage of time in smooth pursuit with fewer saccades. Data suggest that hyperactives approached each test presentation as if it were the first.

Experimental evidence is at variance as to the ability of hyperkinetic children to habituate to stimuli (Cohen & Douglas, 1972; Satterfield & Dawson, 1971; Spring et al., 1974). Satterfield & Dawson (1971) found that, unlike

normals, hyperkinetics failed to show a significant decrease in mean specific galvanic skin response levels across experimental test sessions. Other evidence shows faster habituation in hyperactives (Spring et al., 1974). For the limited pursuit condition, the patient group failed to change significantly their responses across stimulus presentations, supporting the hypothesis that they failed to habituate to stimuli.

As has been frequently observed (Gross & Wilson, 1974; Wender, 1971), hyperkinetics will often display none of the behavior complained about when in a doctor's office. In general, this was the case with the hyperkinetics studied in the present experiment. Most of these children did not exhibit hyperactive behavior and were able to sit still during test procedures. However, despite the successful behavioral adaptation, these hyperkinetics still made more and larger-amplitude eye movements during the test (see records D and E of Figure 6), indicating that adaptation to the test situation was not necessarily correlated with better pursuit performance. Whether the critical factor in producing a larger number of saccades was the subliminal flicker associated with the stimulus or the perceived target movement itself must be separated in future experiments. Regardless of cause, the failure of the hyperactive children to habituate could be of practical value in the diagnosis and treatment of this disorder.

Regarding drug status, four of the hyperkinetic sample were on drugs at the time of testing. Subsequent analysis of on-drug versus off-drug hyperkinetics yielded no differences between the first and second pursuit task in either mean number or mean amplitudes of eye movements. In addition, a scatterplot comparing the performances of both the on-drug and off-drug hyperkinetics on the dependent variables (see Figures A1 and A2 in the Appendix) yielded no clear separation between the two groups. Therefore, for the purposes of this analysis, the groups were judged as being not significantly different and their data were pooled. The finding that drugs had no effect on pursuit performance should be viewed with caution since some of the on-drug and off-drug children came from different referral sources. Removal of cases in either group which did not come from the same source would have seriously decreased the on-drug sample size (n = 4), making comparison impossible.

In conclusion, results of the present experiment show a significant difference between hyperkinetic children and normals on the following response measures: (1) amplitude of eye movements made during the first and second pursuit tasks, (2) number of eye movements made on the second pursuit task, and (3) adaptation with repeated stimulus presentations (defined as a significant decrease in the number of eye movements from the first to the second pursuit task). High-IQ normals did not differ from

low-IQ hyperkinetics as to the number of eye movements made during the second pursuit task. Due to small sample sizes (n = 5), it was felt that the conclusion of no difference between the samples was tenuous. Therefore, a scatterplot was made of the same data (see Figure A3 in the Appendix), which resulted in a strengthening of the statistical finding by showing no clear separation between high-IQ normals and low-IQ hyperkinetics in the number of eye movements made on the second test. In the same scatterplot, a clear separation between groups is seen for the mean amplitude of eye movements made during the second pursuit. Average-IQ normals differed significantly from both groups on the number of eye movements made during the second pursuit task.

Based upon these results, it is not possible at this time to specify clearly one single mechanism responsible for the observed pursuit differences. In fact, these differences may spring from a multiple-factor basis, including both physiological and psychological components. Possible physiological and/or monoamine deficiencies involving the reticular formation and concomitantly the PPRF, a location shown to be a premotor area intimately involved in the integration of eye movement information, could account for these results. Attentional and/or motivational deficits could provide the psychological components necessary to yield these results. It is more likely that there is an interaction between these factors.

It seems improbable, based upon the many diverse experimental findings in these children, that hyperkinesis could have at its core a single-factor etiology which could sufficiently explain both the occurrence of hyperkinesis and its many and variable clinical manifestations. There are further experiments, however, which could be performed in an attempt to separate out the effects of various factors. Separation of motor and attentional deficits could possibly be achieved by using pursuit stimuli of varying velocities such as 6° per second, 15° per second, 22½° per second, and 35° per second. Data from this type of experiment would indicate whether pursuit saccadization would be a constant finding in these children. If it were found that abnormal saccadization occurred during all velocities, an explanation of the findings based on oculomotor deficits involving either peripheral or central mechanisms would appear tenable. This experiment would also yield data on the ability of hyperkinetics, relative to age-matched controls, to pursue targets moving at different velocities. Hyperkinetics may show pursuit deficiencies at lower velocities than do normals.

The effect of attention might be tested by the use of consistent verbal reminders to the child to pay attention during the pursuit task. Effects of attention on pursuit performance may be further studied by introducing random suprathreshold changes in the intensity of the pursuit stimulus over time. These stimulus changes may alert the

child, leading to an increase in attention. Reinforcement contingent upon the child's report of stimulus intensity changes could also be used to study changes in pursuit performance under conditions of increased motivation and attention. The reinforcement condition might be compared to the condition of verbal reminder alone to see if the addition of reinforcement has any effect on pursuit performance. If there were a decrease in the number of eye movements, their amplitude, or both measures under these conditions, then it could be assumed that, in part, the hyperkinetics' pursuit performance deficit found in the present study might be attributable to attentional and/or motivational problems.

The fact that hyperkinetic subjects do not habituate (i.e., make smaller number of eye movements on repeated stimulus presentations) should be further explored. This finding brought up the following questions concerning this group of children: How many pursuit presentations are necessary for the hyperactive group to show a significant drop in number of eye movements? Do habituation effects depend on stimulus velocity? Do subgroups of hyperkinetics such as hyperactives and hypoactives adapt differentially? Repeated presentations of pursuit stimuli both at the same velocities and at different velocities could serve to answer some of these questions.

In the present experiment, drug effects were studied in a very small hyperactive group. Further studies using

larger sample sizes should investigate drug effects on both the number and amplitudes of saccades made during pursuit, as well as adaptation effects.

The buildup of reactive inhibition during pursuit performance could be examined by making the task longer to see during which stimulus epochs the most saccadization occurs. This could answer the question of how many stimulus cycles are necessary to produce optimal pursuit saccadization in normals and hyperkinetics.

IQ findings in normals should be verified. Children of the same age with varying IQ levels should be run under the same pursuit conditions to see if IQ has an effect on the number or amplitudes of saccadic movements made during pursuit. This same experiment might be performed to test the importance of age on pursuit performance; that is, age may be varied while holding IQ constant.

Based upon the qualitative findings that hyperkinetics make more coupled head-eye movements than do normals, these results should be further studied and quantified. Efforts should be made to relate these findings to IQ and age.

In future research a better measure of pursuit performance would be percent time on target. This would greatly strengthen the analysis of pursuit performance.

It is intended that these suggestions for further experimentation will help to further clarify the mechanisms which are involved in oculomotor deficits as seen in the present hyperkinetic population.

Chapter V Conclusions

Hyperkinetic and normal children were tested on a tracking task. The stimulus was a 1° white dot projected on a dark background. The pursuit target traveled at 22½° per second, making 30°-amplitude swings in both the horizontal and vertical planes, although in this thesis analysis was restricted to horizontal eye movements. Two measures of horizontal pursuit performance were analyzed: number of eye movements made and mean amplitude of eye movements. Both measures were obtained for two separate pursuit trials, one occurring at the beginning of the test session, the other at the end of the test session.

The following hypotheses were tested:

(1) The number of eye movements is greater for hyperkinetics than for normals. Results showed that hyperkinetics made significantly more eye movements than did normals on the second pursuit task ($p < .01$). Normals showed a significant decrease in the number of eye movements from first to second pursuit task ($p < .01$). Hyperkinetics did not show a similar decrease in number of eye movements from first to second pursuit task.

(2) Eye movement amplitudes are different in hyperkinetics and normals. Results confirmed this hypothesis. The hyperkinetic group made significantly larger saccadic eye movements than did normals on both the first and second pursuit tasks ($p < .03$). Normals demonstrated a slight increase in the size of eye movements from the

first to the second pursuit task. Hyperkinetics showed a greater increase in the amplitude of eye movements from the first to the second pursuit task than did normals.

(3) Hyperkinetics show a developmental lag as regards normal age-related developmental changes in eye movements relative to controls. There was no clear indication of the effect of age upon the dependent variables in either group. Therefore, evaluation of the developmental-lag hypothesis was not possible.

(4) Hyperkinetics are not able to sustain long periods of attention to the visual stimulus and evidence many movements unrelated to the task. Inspection of records showed that hyperkinetics made many large saccades away from the path of the pursuit target. These large saccades occurred more frequently between the second and third stimulus cycles. In hyperkinetics there may be a breakdown of sustained attention to the visual stimulus.

(5) Hyperkinetics evidence trouble with smooth pursuit movements. There was more saccadization of pursuit in the hyperkinetic group than in the normal group.

Further analysis of the data yielded the following additional findings:

(1) There was a general effect of IQ on number of eye movements made during the second pursuit task ($p < .02$). Both the low- and high-IQ groups evidenced significantly more eye movements than did the middle-IQ group ($p < .05$).

(2) No effect of IQ on the number of eye movements made

during second pursuit was found for the hyperkinetic children. However, the normal-group analysis yielded significant differences between the high- and middle-IQ groups for this task ($p < .01$).

(3) Analysis of covariance (ANCOVAR) demonstrated no significant effect of the covariate age on number of eye movements made by the normal group on the second pursuit task. For the same task, a second ANCOVAR was performed using IQ as the covariate. Covariate effects were found to be highly significant ($p < .01$).

(4) There was no significant difference found between the high-IQ (normal) group and the low-IQ (hyperkinetic) groups regarding the number of eye movements made during the second pursuit task. The amplitude of movement was significantly different in these two groups, however.

(5) No differences were found between the on-drug and off-drug hyperkinetic groups on any response measure. However, these findings are tentative since they are based on small sample sizes.

(6) Both the number and mean amplitudes of eye movements were significantly correlated for the hyperkinetic group between the first and second pursuit tasks ($p < .05$).

(7) The number of eye movements between the first and second pursuit tasks were significantly correlated in the normal group ($p < .05$).

Based upon the experimental evidence it was concluded that: (1) hyperkinetics did not habituate to the pursuit

task, e.g., show a significant change in number of eye movements from first to second pursuit; (2) hyperkinetics made more saccadic eye movements than normals and these movements were consistently larger than in normals; (3) in the normal population IQ was a significant factor as regards number of eye movements, but not in hyperkinetics; and (4) age was found to be an insignificant factor in both control and hyperkinetic populations for all response measures.

It should be emphasized that the differences found between the diagnostic categories are based upon small samples and are therefore tentative in nature. Further research is necessary using large samples to test both the validity and reliability of these findings.

Appendix

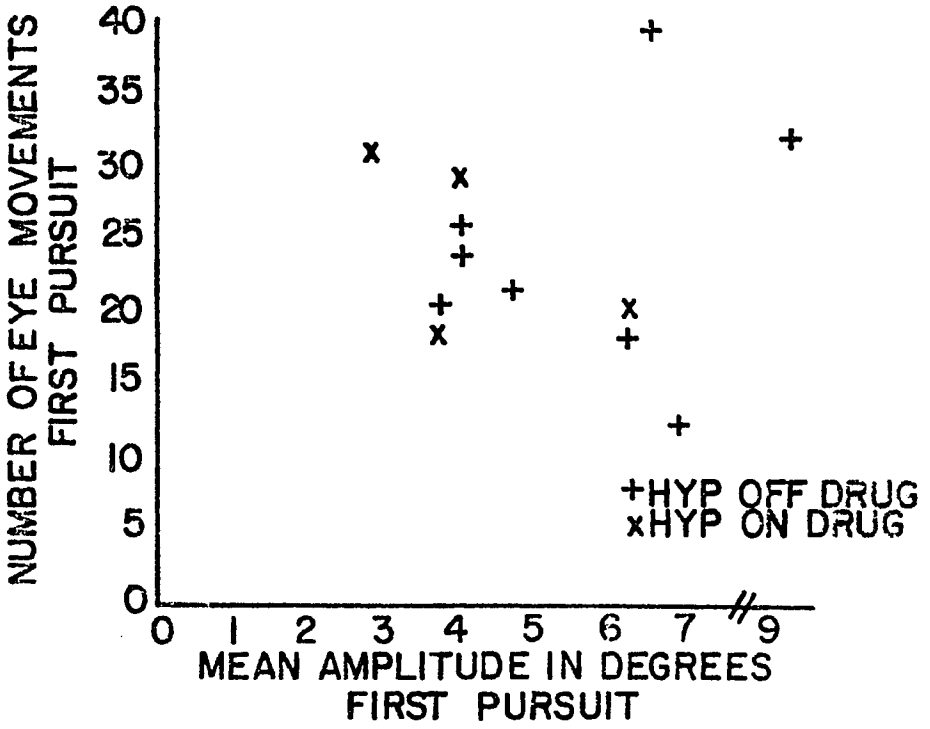


Fig. A1. Scatterplot of the number and amplitudes of saccadic eye movements made by on- and off-drug hyperkinetics on the first pursuit task. No clear separation is shown between the two groups.

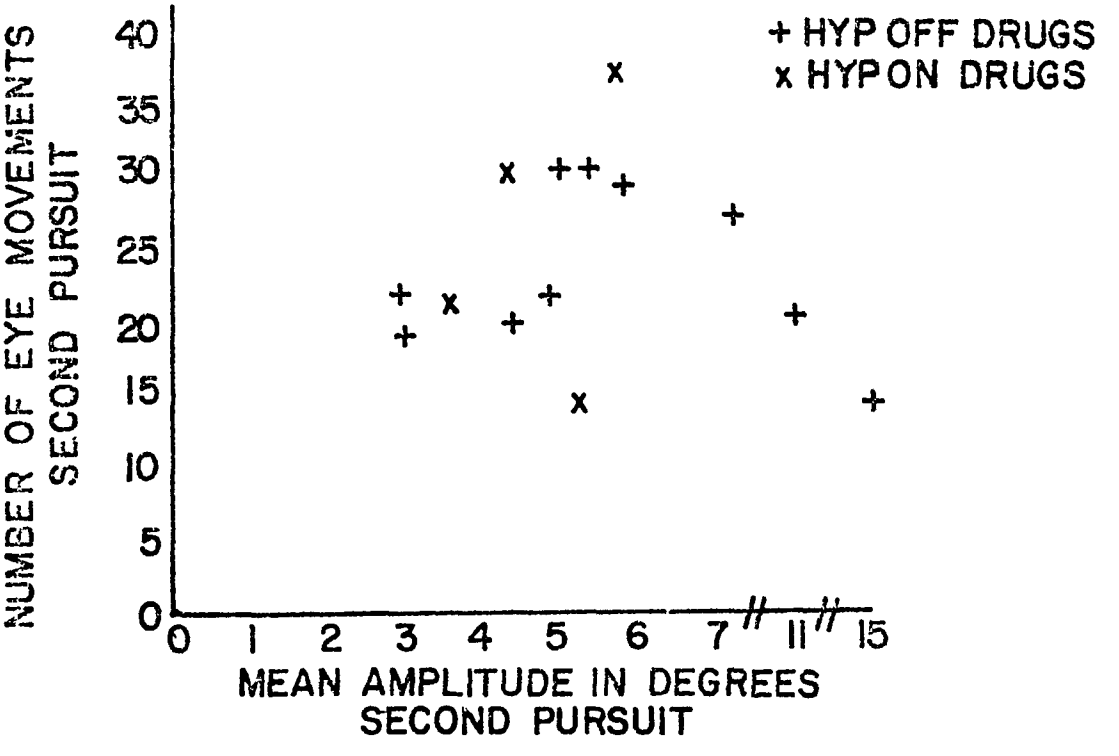


Fig. A2. Scatterplot of the number and amplitudes of saccadic eye movements made by on- and off-drug hyperkinetics on the second pursuit task. No clear separation is shown between the two groups.

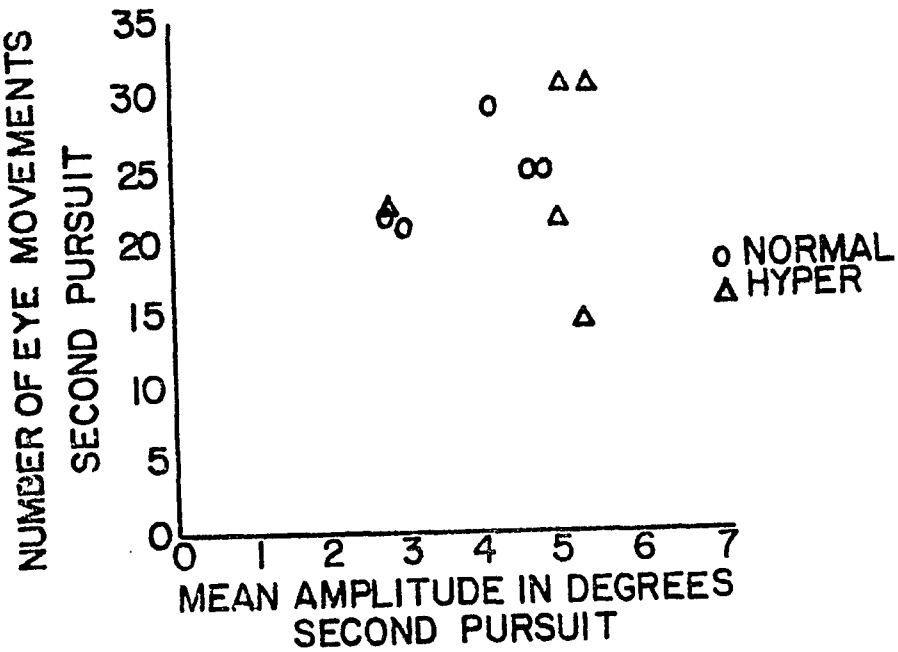


Fig. A3. Scatterplot of the number and amplitudes of saccadic eye movements made by high-IQ normals and low-IQ hyperkinetics on the second pursuit task. No clear separation is shown between the two groups in the number of eye movements made on the second pursuit task; however, a clear separation is seen between groups for the mean amplitude of eye movements made during the second pursuit.

References

Abrams, A. L. Delayed and irregular maturation versus minimal brain injury: Recommendations for a change in current nomenclature. Clin. Pediat., 1968, 7, 344-349.

Bender, M., & Shanzar, S. Oculomotor pathways defined by electric stimulation and lesions in the brainstem of monkey. In M. Bender (Ed.), The oculomotor system. New York: Harper and Row, 1964.

Berlyne, D. E. Curiosity and exploration. Science, 1966, 153, 25-33.

Birch, H., Belmont, I., & Karp, E. Delayed information processing and extinction following cerebral damage. Brain, 1967, 90 (Part I), 113-130.

Bizzi, E. Single unit activity in the frontal eye fields of unanesthetized monkey during eye and head movement. Exp. Brain Res., 1970, 10, 151-158.

Bogacz, J., Mendelaharsu, C., & Mendelaharsu, S. Electro-oculographic abnormalities during pursuit movements in developmental dyslexia. Electroenceph. Clin. Neurophysiol., 1974, 36, 651-656.

Boydstun, J., Ackerman, P., Stevens, D., Clements, S., Peters, J., & Dykman, R. Physiologic and motor conditioning and generalization in children with minimal brain dysfunction. Conditioned Reflex, 1968, 3(2), 81-107.

Burks, H. F. A study of the organic basis for behavior deviations in school children. Unpublished doctoral dissertation, University of Southern California, 1955.

Büttner-Ennever, J., & Henn, V. Pathways from the pontine reticular formation to the oculomotor complex. Paper presented at the German Physiological Society, Spring, 1976.

Campbell, S., Douglas, V., Morgenstern, G. Cognitive styles in hyperactive children and the effect of methylphenidate. J. Child Psychol. Psychiat., 1971, 12, 55-67.

Cantor, H. Role of the neurologist in management of children with learning disabilities. In J. Harstein (Ed.), Current concepts in dyslexia. St. Louis: C. V. Mosby, 1971.

Christensen, D. E., & Sprague, R. L. Reduction of hyperactive behavior by conditioning procedures alone and combined with methylphenidate (Ritalin). Behavior Res. Ther., 1973, 11, 331-334.

Chusid, J. Correlative neuroanatomy and functional neurology. Los Altos, Calif.: Lange Medical Publications, 1970.

Cohen, B. Vestibulo-ocular relations. In P. Bach-y-Rita, C. Collins, J. Hyde (Ed.), The control of eye movements. New York: Academic Press, 1971.

Cohen, B., & Henn, V. The origin of quick phases of nystagmus in the horizontal plane. Bibl. Ophthal., 1972, 82, 36-55.

Cohen, N., & Douglas, V. Characteristics of the orienting response in hyperactive and normal children. Psychophysiology, 1972, 9, 238-245.

Corin, M., Elizan, T., & Bender, M. Oculomotor function in patients with Parkinson's disease. J. Neurol. Sci., 1972, 15, 251-265.

Corvera, J., Torres-Courtney, G., & Lopez-Rios, G. The neurological significance of alterations of pursuit eye movements and the pendular tracking test. Ann. Otol., 1973, 82, 855-867.

Denhoff, E. The natural life history of children with minimal brain dysfunction. Ann. NY Acad. Sci., 1973, 205, 188-205.

Dodge, R. Five types of eye movements in the horizontal meridian plane of the field of regard. Amer. J. Physiol., 1903, 8, 307-329.

Dodge, R., Travis, R., & Fox, J. Optic nystagmus. III. Characteristics of the slow phase. Arch. Neurol. Psychiat., 1930, 24, 21-34.

Dykman, R., Ackerman, P., Clements, S., & Peters, J. Specific learning disabilities: An attentional deficit syndrome. In H. Myklebust (Ed.), Progress in learning disabilities. Vol. 2. New York: Grune and Stratton, 1971.

Dykman, R., Peters, J., & Ackerman, P. Experimental approaches to the study of minimal brain dysfunction: A followup study. Ann. NY Acad. Sci., 1973, 205, 93-109.

Fleming, M. Eye movement indices of cognitive behavior. A. V. Comm. Rev., 1969, 17(4), 383-398.

Freiberg, V., & Douglas, V. I. Concept learning in hyperactive and normal children. J. Abnormal Psych., 1969, 74(3), 388-395.

Fuchs, A. The saccadic system. In P. Bach-y-Rita, C. Collins, J. Hyde (Ed.), The control of eye movements. New York: Academic Press, 1971.

Grassi, J. R. Auditory vigilance performance in brain damaged, behavior disordered and normal children. J. Learn. Disabil., 1970, 3(6), 302-305.

Gross, M., & Wilson, W. Minimal brain dysfunction. New York: Brunner-Mazel, 1974.

Grünewald-Zuberbier, E., Grünewald, G., & Rasche, A. Hyperactive behavior and EEG arousal reactions in children. Electroenceph. Clin. Neurophysiol., 1975, 38, 149-159.

Holzman, P. S., Proctor, L. R., & Hughes, D. Eye tracking in schizophrenia. Science, 1973, 181, 179-181.

Holzman, P. S., Proctor, L. R., Levy, D. L., Yasillo, N. J., Meltzer, H. Y., & Hurt, S. W. Eye-tracking dysfunctions in schizophrenic patients and their relatives. Arch. Gen. Psychiat. 1974, 31, 143-151.

Hoyt, W., & Daroff, R. Supranuclear disorders of ocular control systems in man: Clinical, anatomical and physiological correlations. In P. Bach-y-Rita, C. Collins, J. Hyde (Ed.), The control of eye movements. New York: Academic Press, 1971.

Hunt, J. M. Intrinsic motivation and its role in psychological development. In D. Levine (Ed.), Nebraska Symposium on Motivation. Lincoln: University of Nebraska Press, 1965.

Jung, R., & Kornhuber, H. Results of electronystagmography in man: The value of optokinetic, vestibular, and spontaneous nystagmus for neurologic diagnosis and research. In M. Bender (Ed.), The oculomotor system. New York: Harper and Row, 1964.

Kahn, E., & Cohen, L. Organic driveness: A brain stem syndrome and an experience. N. Eng. J. Med., 1934, 219, 748.

Klatskin, E. H., McNamara, E. M., Shaffer, P., & Pincus, J. Minimal organicity in children of normal intelligence: Correspondence between psychological test results and neurologic findings. J. Learn. Disabil., 1972, 5(4), 213-218.

Klinkerfuss, G. H., Lange, P. H., & Weinberg, W. H. Electroencephalographic abnormalities of children with hyperkinetic behavior. Neurology, 1965, 15, 883-891.

Knopp, W., Arnold, L., Andros, R., & Smeltzer, D. Predicting amphetamine response in hyperkinetic children by electronic pupillography. Pharmacopsychiatry, 1973, 6, 158-166.

Laufer, M. W., Denhoff, E., & Solomons, G. Hyperkinetic impulse disorder in children's behavior problems. Psychosomat. Med., 1957, 19, 38-49.

Luria, A. R. The role of speech in the formation of mental processes. The role of speech in the regulation of normal and abnormal behavior. Bethesda: U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, 1960.

Meyer, A., Falconer, M., & Beck, E. Pathological findings in temporal lobe epilepsy. J. Neurol. Neurosurg. Psychiat., 1954, 17, 276-285.

Minimal brain dysfunction in children. Publication No. 1415, U.S. Department of Health, Education and Welfare, Washington, D.C.: Government Printing Office, 1966.

Minimal brain dysfunction in children. Publication No. 2015, U.S. Department of Health, Education and Welfare, Washington, D.C.: Government Printing Office, 1969.

Morrison, J. R., & Stewart, M. A. Evidence for polygenetic inheritance in the hyperactive child syndrome. Amer. J. Psychiat., 1973, 130(7), 791-792. (a)

Morrison, J. R., & Stewart, M. A. The psychiatric status of the legal families of adopted hyperactive children. Arch. Gen. Psychiat., 1973, 28, 888-891. (b)

Oliver, D. Lead and hyperactivity. The Lancet, Oct 28, 1972, 900-903.

Palkes, H., & Stewart, M. Intellectual ability and performance of hyperactive children. Amer. J. Orthopsychiat., 1972, 42, 35-39.

Pasamanick, B., & Knobloch, H. Syndrome of minimal cerebral damage in infancy. JAMA, 1959, 170, 1384-1387.

Peters, J., Romine, J., & Dykman, R. A special neurological examination of children with learning disabilities. Develop. Med. Child Neurol., 1975, 17(1), 63-78.

Pope, L. Motor activity in brain injured children. Amer. J. Orthopsychiat., 1970, 40(5), 783-793.

Prechtl, H. F. R., & Stemmer, C. F. The choreiform syndrome in children. Develop. Med. Child Neurol., 1962, 4, 119-127.

Quitkin, F., & Klein, D. Two behavioral syndromes in young adults related to possible minimal brain dysfunction. J. Psychiat. Res., 1969, 7, 131-142.

Rashbass, C. Barbiturate nystagmus and the mechanisms of visual fixation. Nature, 1959, 183, 897-898.

Rashbass, C. The relationship between saccadic and smooth tracking eye movements. J. Physiol. (London), 1961, 159, 326-338.

Report of the conference on the use of stimulant drugs in the treatment of behaviorally disturbed young school children. Sponsored by the Office of Child Development and the Office of the Assistant Secretary for Health and Scientific Affairs, U.S. Department of Health, Education and Welfare, Washington, D.C.: January 11-12, 1971.

Robinson, D. The mechanics of human saccadic eye movement. J. Physiol. (London), 1964, 174, 245-264.

Robinson, D. The mechanics of human smooth pursuit eye movement. J. Physiol. (London), 1965, 180, 569-591.

Robinson, D., & Fuchs, A. Eye movements evoked by stimulation of frontal eye fields. J. Neurophysiol., 1969, 32(5), 637-648.

Satterfield, J. H., & Dawson, M. Electrodermal correlates of hyperactivity in children. Psychophysiology, 1971, 8, 191-197.

Satterfield, J. H., Lesser, L. I., Saul, R. E., & Cantwell, D. P. EEG aspects in the diagnosis and treatment of minimal brain dysfunction. Ann. NY Acad. Sci., 1973, 205, 274-282.

Sauerhoff, M. W., & Michaelson, A. I. Hyperactivity and brain catecholamines in lead-exposed developing rats. Science, 1973, 182(4116), 1022-1024.

Shackel, B., Davis, J., & Abercombie, M. Electrooculography in a pilot study of cerebral palsied children. IRE Trans. Biomed. Electr., April 1962, 112-115.

Shaffer, D., McNamara, N., & Pincus, J. Controlled observation on patterns of activity, attention and impulsivity in brain damaged and psychiatrically disturbed boys. Psychol. Med., 1974, 4, 4-18.

Shagass, C., Roemer, R., & Amadeo, M. Eye tracking performance and engagement of attention. Arch. Gen. Psychiat., 1976, 33, 121-125.

Silbergeld, E. K., & Goldberg, A. M. Lead induced behavioral dysfunction: An animal model of hyperactivity. Exp. Neurol., 1974, 42, 146-157.

Silverman, L. J., & Metz, A. S. Numbers of pupils with specific learning disabilities in local public schools in the U.S. Ann. NY Acad. Sci., 1973, 205, 146-157.

Sokolov, E. N. Higher nervous functions: The orienting reflex. Ann. Rev. Physiol., 1963, 25, 545-580.

Sollenberger, R. T. Chinese-American child-rearing practices and juvenile delinquency. J. Social Psych., 1968, 74, 13-23.

Spring, C., Greenberg, L., Scott, J., & Hopwood, J. Electrodermal activity in hyperactive boys who are methylphenidate responders. Psychophysiology, 1974, 11(A), 436-442.

Stark, L., Vossius, G., & Young, L. R. Predictive control of eye tracking movements. IRE Trans. Biomed. Electr., 1962, 3(2), 52-57.

Stevens, D., Boydstun, J., Dykman, R., Peters, J., & Sinton, D. Presumed minimal brain dysfunction in children (relationship to performance on selected behavioral tests). Arch. Gen. Psychiat., 1967, 16, 281-285.

Stewart, M. A., & Morrison, J. R. A family study of the hyperactive child syndrome. Biol. Psychiat., 1971, 3, 189-195.

Strauss, A., & Werner, H. The mental organization of the brain-injured mentally deficient child. Amer. J. Psychiat., 1941, 97, 1194-1201.

Sykes, D. H., Douglas, V., & Morgenstern, G. Sustained attention in hyperactive children. J. Child Psychol. Psychiat., 1973, 14, 213-220.

Tarnopol, L. Delinquency and MBD. J. Learn. Disabil., 1970, 3(4), 200-207.

Wagman, H. Eye movements induced by electric stimulation of cerebrum in monkeys and their relationship to bodily movements. In M. Bender (Ed.), The oculomotor system. New York: Harper and Row, 1964.

Wagman, H., Werman, R., Feldman, D., Sugarman, L., & Krieger, H. The oculomotor effects of cortical and subcortical stimulation in the monkey. J. Neuropathol. Exp. Neurol., 1957, 16, 269-277.

Wender, P. Minimal brain dysfunction in children. New York: John Wiley and Sons, 1971.

Wender, P. Some speculations concerning a possible biochemical basis of minimal brain dysfunction. Ann. NY Acad. Sci., 1973, 205, 18-28.

Werry, J. S., Weiss, G., & Douglas, V. Studies on the hyperactive child. I. Some preliminary findings. Canad. Psychiat. Assoc. J. 1964, 9, 120-130.

Westheimer, G. Eye movement responses to a horizontally moving visual stimulus. AMA Arch. Ophthal., 1954, 52, 932-941. (a)

Westheimer, G. Mechanism of saccadic eye movements. AMA Arch. Ophthal., 1954, 52, 710-724. (b)

Willerman, L. Activity level and hyperactivity in twins. Child Develop., 1973, 44, 288-293.

Windle, W. F. Brain damage at birth. JAMA, 1968, 206, 1967-1972.

Young, L. Pursuit eye tracking movements. In P. Bach-y-Rita, C. Collins, J. Hyde (Ed.), The control of eye movements. New York: Academic Press, 1971.

Zahn, T., Abate, F., Little, B., & Wender, P. Minimal brain dysfunction, stimulant drugs and autonomic nervous system activity. Arch. Gen. Psychiat., 1975, 32, 381-387.

Zaporozhets, A. V. The development of voluntary movements. In B. Simon (Ed.), Psychology in the Soviet Union. Stanford: Stanford University Press, 1957.