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PHYSIOLOGICAL AND PHARMACOLOGICAL STUDIES IN HYPERACTIVITY: INTERACTIONS BETWEEN EYE MOVEMENTS, ATTENTION AND STIMULANT MEDICATION

A thesis presented to
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ABSTRACT

Hyperactivity (Attention Deficit Disorder with Hyperactivity, DSM-III, 1980) remains the most frequently diagnosed disorder in child psychology. Both CNS hypoarousal and hyperarousal have been posited as the basis for this disorder although the results of psychophysiological studies, while not unequivocal, tend to support the former position. Such investigations have been equally unsuccessful in delineating the basis of attentional deficits observed in hyperactive (HK) children.

One prominent finding from this latter line of inquiry has been that visual information processing, most notably on tasks requiring purposeful and organized visual search strategies, is compromised in these children. The hypothesis that oculomotor control deficiencies contribute to visual processing difficulties led to the investigation of pursuit eye movement (PEM) function in HK children. Despite methodological limitations and procedural differences across studies, evidence of oculomotor dysfunction (superimposed saccades, nystagmoid eye movement, more 'irregular' PEM patterns) has been consistently reported for these children relative to controls. However, these studies have not been in agreement regarding the suspected basis for these oculomotor irregularities. Some have attributed them to physiological

dysfunction, whereas others have considered them reflections of attentional deficits. The present investigation was undertaken: (i) to confirm the presence of PEM dysfunction in HK children using conventional electrophysiological procedures; (ii) to examine oculomotor variables in the same HK children under medicated and nonmedicated conditions; (iii) to examine possible cerebellar involvement in PEM dysfunction by recording trials under both light- and dark-adapted conditions; (iv) to relate variations in these physiological measures to variations in symptomatology; and, (v) to address the question of the basis of these dysfunctions using differential objective measures.

Horizontal pursuit eye movements were examined in 20 HK [DSM-III criteria; recorded under both nonmedicated (HKnm) and medicated (HKm) conditions] and 20 age-matched control children. These eye movements were elicited by a sinusoidally oscillating (0.45 Hz) target light (2mm red LED) subtending a 20 degree arc through the visual field and were recorded electrooculographically (HEOG) under both light- and dark-adapted conditions. Subsequent to electronic conditioning (filtering and differentiating), eye movement data were digitized and scored by computer for aberrations of pursuit tracking patterns, i.e., for velocity arrests (VAs) and saccades superimposed on tracking patterns (number, velocity, amplitude and direction), as well as root mean

square (RMS) error differences between eye and target position. Statistical analyses included examination by repeated measures ANOVAs of group differences and the effects of medication and lighting condition -- variables, respectively, thought to influence attention and cerebellar influences on oculomotor nuclei -- on eye movement measures and the correlation of medication-associated changes in tracking with indexes of behavioural symptomatology.

Results confirmed the presence of PEM dysfunction in nonmedicated HK children as indexed by significantly increased VA scores and greater numbers of intrusive saccades relative to these measures in controls. Dark adaptation or medication, although improving tracking performance (significantly only with dark adaptation), did not normalize PEM performance measures for HK children. However, the combined effect of these manipulations resulted in statistical equality of C and HK groups for VA score, but not for the number of saccades produced. RMS error did not distinguish between groups for any of the recording conditions.

These results are discussed in the context of hypothesized arousal and attentional dysfunctions associated with hyperactivity. Variations in voluntary attention are not supported as the basis of pursuit eye movement difficulties evidenced by HK children. Improvements in PEM performance

after the combined manipulations of dark adaptation and medication suggest a dysfunction based on interactions between cerebellar influences and other medication-responsive processes (possibly decreased CNS arousal). Positive correlations between medication-related improvements in behaviour and tracking performance, as well as between medication dose and increases in saccadic velocity, suggest that oculomotor dysfunction may reflect CNS hypoarousal. This postulated arousal dysfunction would result in an inefficient activation of oculomotor processes, precluding accurate PEM performance.

CURRICULUM STUDIORUM

Frederick Wilburn Bylsma was born in Kingston, Ontario, Canada on January 29, 1957. Mr. Bylsma received the degree of Bachelor of Arts -- Psychology (honours) from the University of Ottawa, Ottawa, Canada in 1979.

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Hyperkinesis (Attention Deficit Disorder with Hyperactivity, ADD-H; DSM-III, 1980) remains one of the most frequently diagnosed disorders in child clinical psychology and psychiatry (Wender, 1975). Diagnoses, according to DSM-III criteria, are based upon behavioural symptomatology as determined from behaviour rating scales (Conners, 1969; 1970; 1973; Goyette, Conners and Ulrich, 1978), interviews with the patient, parent(s) and teacher(s), observations by qualified professionals, and a detailed history of the child's developmental pattern. In North America, prevalence estimates vary from 1 to 20 percent of school-aged children (Lambert, Sandoval and Sassone, 1978; Stewart, Pitts, Craig and Dieruf, 1966), while the generally accepted level is about 3 to 5 percent (Barkley, 1981; Wender, 1971).

It is generally agreed that attention deficits, impulsiveness, low frustration tolerance, inappropriate overactivity and poor academic performance represent the "core" symptoms of hyperactivity. However, the concordance among clinicians and among research laboratories regarding aetiology, necessary and sufficient symptoms for diagnosis, and primary versus secondary symptomatology, has historically been and remains quite low. To some extent, this derives from the fact that the two aspects considered to be of greatest importance in the disorder are "two of psychology's most ambiguous and controversial constructs: arousal and

attention" (Ferguson and Pappas, 1979, p. 62; also see Lacey, 1967).

Neurophysiological basis of hyperactivity

A neurophysiological basis of hyperactivity was first proposed when hyperkinetic behaviour syndromes were initially observed following overt neurological trauma due to head injury, anoxia or neurotoxic reactions. Later, in reports of equally pathological patterns of behaviour in children in the absence of medical or physiological indications of brain damage (Childers, 1935; Still, 1902), a rather tenuous logical jump was made in an attempt to explain these phenomena. It was proposed that since certain cases of overt CNS trauma resulted in pathological behavioural patterns in some individuals, similar pathological behavioural patterns evidenced in the absence of overt neurological trauma must result from some undetectable or subclinical brain trauma, minimal brain damage or dysfunction (Laufer et al, 1957; Tredgold, 1908). It was postulated that this diffuse CNS damage or dysfunction (Clements, 1966) disturbed the child's ability to adequately modulate arousal levels in the face of changing environmental demands (Wender, 1971).

Two opposing theoretical viewpoints have developed to account for the nature of the dysfunctions manifest in

hyperkinesis. One position proposes behavioural overactivation to be a compensatory "stimulus seeking" reaction to subcortical (Bradley, 1937; Dykman et al, 1971) or cortical (Satterfield, Cantwell, Saul and Yusin, 1974; Stewart, 1970; Werry and Sprague, 1970) hypoarousal, with the purpose of this activity being to attain and maintain an optimal homeostatic cortical arousal level (Berlyne, 1960; Hebb, 1955; Zentall, 1975). The alternate position characterizes the increased activity level as a reflection of CNS hyperarousal in either subcortical (Kahn and Cohen, 1934; Laufer, Denhoff and Solomans, 1957; Wender, 1976), cortical. (Buckley, 1972; Dykman, Ackerman, Clements and Peters, 1971) or unspecified (Freibergs and Douglas, 1969) brain sites.

A great deal of psychophysiological research has been undertaken attempting to document these theoretical positions by delineating the nature and site of the CNS dysfunction responsible for the hyperkinetic (HK) syndrome. Various physiological variables purported to reflect CNS arousal levels have been examined for the presence of group differences between normal children, nonmedicated HK children and HK children undergoing treatment with stimulant medications. These variables include basal heart rate (Barkley and Jackson, 1979; Knights and Hinton, 1969; Zahn, Abate, Little and Wender, 1975), phasic heart rate (Porges, Walter, Korb and Sprague, 1975; Sroufe, Sonies, West and

Wright, 1973; Zahn et al, 1975), blood pressure (Knights and Hinton, 1969; Rapoport, Quinn, Bradbard, Riddle and Brooks, 1974; Rie, Rie, Stewart and Ambued, 1976), resting basal skin conductance levels (Conners, 1975; Satterfield and Dawson, 1971; Zahn et al, 1975), nonspecific and specific galvanic skin response (Firestone and Douglas, 1975; Satterfield and Dawson, 1971; Spring, Greenberg, Scott and Hopwood, 1974), electroencephalographic variables (Grunewald-Zubebier, Grunewald and Rasche, 1975; Paine, Werry and Quay, 1968; Satterfield, Cantwell, Lesser and Podosin, 1972; Sheer, 1976), sleep (Busby, Firestone and Pivik, 1981; Nahas and Kryniki, 1977; Small, Hibi and Feinberg, 1971), auditory evoked potentials (Buchsbaum and Wender, 1973; Prichep, Sutton and Hakarem, 1976), visual evoked potentials (Buchsbaum and Wender, 1975; Calliway, Halliday and Naylor, 1983; Conners, 1972), monosynaptic spinal reflexes (Mercier, Firestone and Pivik, 1980; Mercier and Pivik, 1983; Pivik, Bylsma and Margittai, 1986; Pivik and Mercier, 1981) and eye movements (Bala, Cohen, Morris, Atkin, Gittleman and Kates, 1981; Shapira, Jones and Sherman, 1980).

The results of these studies have been quite heterogenous across laboratories, reflecting variations in either operational definitions of the arousal variable studied, dependent variables used, measurement methods, methodological approach, subject selection and exclusion, and/or type and

dose of medication used. In an extensive review of this area, Ferguson and Pappas (1979) concluded that while many studies found no differences between HK and normal children, in those studies where significant differences were found they tended to support the position that the children were hypoaroused. Furthermore, Hastings and Barkley (1978), in a similar review, concluded that while the evidence tends to support a resting level of neither hyperarousal or hypoarousal, hyperkinetic children are "probably underreactive to environmental stimulation or are 'underarousable'" (p. 413; see also Rapoport and Ferguson, 1981).

Attentional deficits and hyperactivity

Recently, research evidence has prompted increased emphasis on attentional deficits of the hyperkinetic syndrome (Conners, 1975; Douglas, 1972; Douglas and Peters, 1979; Dykman et al, 1971; Sergeant and Scholten, 1983). This is reflected as well in the DSM-III (1980) classification of "attention deficit disorder with hyperactvity" (ADD-H) replacing the DSM-II (1968) "hyperkinetic syndrome" classification. Paralleling the difficulties in determining a consistent definition of 'arousal' across studies, researchers dealing with attention have employed a multiplicity of operational definitions, variables and tasks in an effort to tap attentional processes in the study of HK children

(Berlyne, 1970; Douglas, 1972; Rosenthal and Allen, 1978) including reaction time, signal detection rates, discrimination ability, vigilance tasks and distractibility. Although differences in defining important concepts make across-study interpretations and equivalences difficult, relevant and reliable information have nevertheless been produced by these studies.

The concept of a broad, all encompassing attentional deficit in hyperkinesis has not been substantiated (Douglas, 1983) as indicated by abundant evidence that HK children do not perform differently from controls on many tasks requiring attentive behaviour for successful completion, i.e., tests of language ability, intelligence and complex discrimination tasks (Douglas, 1972; Whalen and Henker, 1976). Attention-related tasks which HK children perform significantly worse than controls include those of simple reaction time (Cohen and Douglas, 1972; Porges et al, 1975), simple discrimination tasks (Dykman et al, 1971; Freibergs and Douglas, 1969; Laufer et al, 1957), vigilance tests (Douglas, 1974; Loiselle, Stamm, Maitinsky and Whipple, 1980), selective attention tasks (Loiselle et al, 1980; McIntyre, Blackwell and Denton, 1978) and certain distraction tasks (Odom and Guzman, 1972; Radosh and Gittleman, 1981).

Slower and more variable reaction times and errors of

omission exhibited by HK children on vigilance or selective attention tasks have been interpreted as reflecting either an inability (arousal deficiency) or unwillingness -- because of increased salience of distracting stimuli (Odom and Guzman, 1972) or decreased task stimuli salience (Radosh and Gittleman, 1981) -- to exert the necessary 'effort' to maintain a level of task-oriented attention sufficient to ensure rapid and accurate processing (Douglas, 1983). Similarly, errors of commission or premature responding in selective attention or delayed reaction time tasks in these children have been thought to reflect either increased distraction or an inability to inhibit impulsive responding (Douglas, 1983). Douglas (1983) postulates that all types of errors these children produce on attention-related tasks can be thought of as arising from a "strong inclination to seek immediate reinforcement" coupled with an inherent inability to invest, organize and maintain sufficient attentional energy on task, to inhibit impulsive responding and to modulate arousal levels in response to changing environmental demands. While such a proposition does offer an explanation for the majority of 'deficits' noted in HK children, the statement is so inclusive and general as to render it impossible to test scientifically.

Theories of attention and hyperactivity

Attention, despite its importance in psychology, remains one of the least understood and least adequately defined concepts in psychology. However, there have been attempts to establish theoretical conceptualizations of what 'attention' is and how it relates to our experience. One of the earliest theories of attention was proposed by Broadbent (1957). The initial presentation of this theory depicted attention as essentially a 'filtering' process which filtered out unimportant stimuli and permitted the analysis of important stimuli. The efficacy of our attention is determined by the degree to which we are able to 'filter out' extraneous stimuli and process important ones. This theory spawned a tremendous amount of research but was found to be overly simplistic and inadequate to explain the complexities of the attentional abilities of humans. For instance, the theory was unable to account for why certain stimuli could be detrimental in certain situations and facilitatory in others. A more recent reformulation of the theory (Broadbent, 1971) includes an 'executive' or control mechanism which modifies and modulates the amount of attention directed toward any given stimulus at any given time. The control mechanism plays an extremely important role in determining the efficacy of attention. This 'executive' mechanism maintains a record of the current 'plan of action', i.e., those actions which we

have decided to undertake and the order in which they are to be performed, and modifies the plan according to incoming information received from the environment as we interact with it while carrying out the plan. It is the efficiency of the control process that regulates the efficiency of the ability to attend to a given stimulus at a given time.

An alternate model views attention as a limited resource or amount of energy that must be distributed among the various tasks to be performed (Kahneman, 1973). This conceptualization of attention is referred to as the 'capacity theory' of attention since it emphasizes the limits on the amount of concurrent processing that can take place before performance begins to deteriorate. Attention is a limited commodity that can be allocated to those tasks or stimuli that are important. To the extent that the demand for 'attention resources' does not exceed the available 'free supply', task performance will not be adversely affected. If, however, it becomes necessary to concurrently perform tasks that require a total of attentional resources that exceeds the total capacity, then performance on one or all tasks will be adversely affected.

The nature of the attention deficit in hyperactivity is such that it can be explained by either model of attention outlined above. The rapid shifts of attention from stimulus

to stimulus could be explained as an inability to maintain or execute the current plan of action, i.e. an 'executive' dysfunction. Alternately, it could be proposed that HK children have a more limited attentional capacity and as a result cannot process as much concurrent information as efficiently as normal children. However, until a more adequate conceptualization is developed of what attention is, and how it can be accurately measured, this unfortunate degree of confusion surrounding attention and its role in the symptomatology of hyperactive children will persist.

Stimulant medications and hyperactivity

For the majority of HK children (approximately 75%) the administration of stimulant medication (methylphenidate, d-amphetamine, pemoline) tends to ameliorate deviant responses. Biochemically, these medications facilitate the action of catecholamines (dopamine, norepinepherine) in the CNS. Physiological indicators of arousal suggesting reduced levels of activation in nonmedicated HK children (i.e., basal heart rate - Knights and Hinton, 1969; phasic heart deceleration in anticipation of a stimulus - Porges et al, 1975; skin conductance levels and nonspecific galvanic skin response - Satterfield and Dawson, 1971; EEG variables - Satterfield et al, 1972) tend to normalize after the administration of stimulant medication. Similarly, stimulant

medication acts to increase attending behaviour while inhibiting impulsive responding in HK children (Douglas, 1983). According to the CNS hypoarousal theory of hyperactivity, these data suggest that the effect of stimulant medication for HK children is to increase arousal levels resulting in behaviour patterns more similar to those of normal children. Whether this effect results from direct action upon attention-regulating brain mechanisms or secondarily through increases in CNS arousal level remains to be determined.

Although the concepts of arousal (defined as level of CNS activation) and attention (defined as the amount of energy invested in a given stimulus; Douglas, 1983) are not identical, they are highly interdependent (for example, decreased levels of arousal preclude highly attentive behaviour). It is impossible to study attention or arousal in isolation since variations in either will necessarily effect associated changes in the other. However, by assessing a behaviour which has measurable indexes that reflect physiological dysfunction and attentional influences differentially and on which HK children have a deficit, the possibility of more accurately determining the relative contributions of attention, arousal and their interrelationship to both behavioural and attentional deficits observed in HK children is enhanced. Oculomotor behaviours

involved in the processing of visual information may be effectively investigated in this context.

Visual processing deficits in hyperactive children

Accurate detection and processing of visual information is critical for efficient and effective interaction with the environment. The extraction and processing of visual information during visual field search depends extensively upon precise oculomotor control to maintain target stimuli on the fovea until sufficient processing has occurred. That HK children experience difficulty in visual information processing requiring purposeful and organized visual search strategies has been well established. For example, these children evidence significant difficulty relative to control children on two tests which assess visual scanning and analysis (Messer, 1976), i.e., the Matching Familiar Figures Test of Reflection and Impulsivity and the Embedded Figures Test of Field Dependence-Independence (Aman, 1978; Douglas and Peters, 1979; Messer, 1976; Sandoval, 1977). Also, Ain (1980) found that HK children were less accurate than controls in recognizing previously presented pictures despite the absence of significant group differences in the amount of time spent viewing the picture on initial presentation. In another portion of the same study, HK children required more time than controls to code and match visual stimuli at a given accuracy

level. Similarly, Barkley (1977) found HK children less able than controls to extract information from videotaped lessons. Noting the evidence indicating that these children have no visual memory difficulties (Benezera, 1980; Corsi, 1972; Kimura, 1963; Osterrieth, 1944), Douglas (1983) suggested that these data support the hypothesis that HK children extract information presented to the visual system less accurately than control children (McIntyre et al, 1978).

The observation that in a task situation HK children spend more time 'off-task' (i.e., not attending to or performing the task when required to do so) than controls (Barkley, 1977) may provide a possible explanation for their difficulty with visual information processing. However, despite focusing only on those times when children appeared to be looking at the target stimuli, the significant difference between HK and control groups for the efficacy of visual information processing was not eliminated (Ain, 1980). These data were interpreted as suggesting that HK children do not use their 'looking time' (time when eyes are directed at the target) as efficiently as controls. Efficient 'looking time' involves rapid and accurate extraction of visual information and implies foveation of visual stimuli until sufficient processing has occurred. Since visual information processing deficits might be explained by inefficient use of looking time, and since accurate looking requires precise oculomotor

control, disturbances of oculomotor functioning of HK children may play an important role in the generation of visual information processing deficits.

Two studies have assessed eye movement control in HK children using electrooculographic (EOG) techniques. Shapira et al (1980) found that nonmedicated HK children had significantly less accurate pursuit eye movements than control children on two tracking paradigms. HK children evidenced more nystagmoid, positive (in the direction of target motion) and reversal (in the direction opposite target motion) saccadic intrusions superimposed upon pursuit tracking patterns. Saccadic reversals were also evident in the eye movements of HK children during a reading task. These authors interpreted the finding of nystagmoid intrusions as suggesting a possible cerebellar dysfunction as the basis for these aberrations and suggested further that aberrant eye movement control may contribute to reading difficulties in these children.

Bala et al (1981) reported evidence of similar PEM deficits in two groups of nonmedicated HK children while tracking targets moving at a constant velocity. Reported deficits included both 'irregular' PEM tracking patterns as well as more and larger saccades (both positive and reversal) superimposed upon PEM patterns. Repeating the task reduced

the number of saccades in controls but not in nonmedicated HK subjects, suggesting that the latter group did not adapt to, or learn, the task. The addition of an attention-enhancing task (button press to target light dimming) reduced the number of saccades for both groups to an equal degree, but failed to eradicate the significant group difference. Administration of stimulant medication was associated with reduced numbers of saccadic intrusions during tracking in a group of medicated HK children relative to a separate group of nonmedicated HK children. Although the medicated group continued to produce more saccades than controls, a significant group difference was no longer present. The pairing of attention-enhancing tasks and stimulant medication further reduced the difference between HK and control childrens' PEM performance such that numbers of saccadic intrusions no longer differentiated the groups. The authors suggested their results indicated the presence of attention-related deficits in oculomotor control in HK children which could be attenuated by combining the administration of stimulant medication with an attention-enhancing manipulation.

There are several methodological features of these studies that either bring into question the validity of the results or which restrict the extent to which these results can be generalized to the HK population at large; for example: (i) Shapira et al (1980) utilized only rating scales

and visual detection of saccades to analyse their data without providing indications of rater reliability; (ii) although saccadic interruptions were noted in both studies, the authors were inconsistent in interpreting the data with regard to the basis of saccadic interruptions of PEM (i.e., attentional variations versus physiological dysfunction); (iii) medication effects on tracking aberrations were not adequately addressed. Bala et al (1981) studied two separate groups of HK children, i.e., one nonmedicated and the other medicated, not the same children under both conditions. While group differences can be studied in this manner, within-subject medication-related improvements in behavioural symptomatology or PEM performance cannot be determined. Additionally, since no indication is given of the degree to which behavioural symptoms were ameliorated by medication, it is impossible to relate improvements in PEM performance to medication-related improvements in behaviour. Furthermore, no indication is given as to why the nonmedicated children were not following a medication treatment schedule (e.g., absence of positive response to medication versus parental or physician's decision to discontinue medication despite good response). Consequently, the possibility cannot be discounted that the group differences noted between medicated and nonmedicated HK children by Bala et al (1981) may have resulted from subject variables unrelated to their ability to control eye movements (i.e., medication response); and, (iv) an additional factor

limiting the interpretation of these data is that the authors did not employ two of the standard indexes of PEM performance, i.e., velocity arrests scores (VAs; slowing of eye velocity to less than or equal to 2 degrees per second; Holzman et al, 1973) and root mean square error scores (RMS; a global measure of the discrepancy between eye and target position; Iacono and Lykken, 1979a, 1979b). In view of the group differences found in the above two studies and noting the shortcomings outlined above, an investigation of PEM performance of HK children, utilizing conventional indexes of PEM performance (VAs, RMS error) as well as indexes of saccadic interruption, controlling for these factors is warranted.

The studies of oculomotor activity in HK subjects by Bala et al (1981) and Shapira et al (1980) were consistent in reporting two types of saccadic interruption of PEM patterns which are distinguishable by their direction, i.e., positive (in the direction of the target motion) and reversal (in the direction opposite target motion) saccades. Similar distinctions have been made for saccades superimposed upon PEM patterns recorded from normal (Iacono and Koenig, 1983; Schalen, 1980) and schizophrenic adults (Holzman, Proctor and Hughes, 1973). Positive saccades have been thought of as correcting for retinal slip (i.e., how far the target image is from the fovea on the retina; Robinson, 1965) due to large

eye-target positional differences or resulting from variations in level of attention (Holzman et al, 1973). Reversal saccades are thought to result from random pontine cellular firing (Schalen, 1980) or lack of visual control (inability to utilize visual feedback) of eye movement (Kornhuber, 1974) and are not considered to serve any corrective function. They have, therefore, been postulated to be an indicator of a physiological dysfunction in the smooth pursuit eye movement control system (Holzman et al, 1973). However, Iacono and Koenig (1983) argue that since some reversal saccades occur closely following a positive saccade which had taken the eye off target, at least a portion of reversal saccades appear to serve a corrective function, i.e., repositioning the eye on target. Conversely, Schalen (1980) considers the entire complex of a postive saccade with a subsequent reversal saccade aberrant within the context of pursuit eye movements.

If reversal saccades do reflect a true physiological dysfunction of PEMs, then some componten(s) of the CNS involved in generation of pursuit eye movements must be malfunctioning. Recent investigations of PEMs in neurological patients with lesions involving cerebellar structures suggest that the cerebellum may play an important role in PEM dysfunction. Pursuit eye movements are thought to have evolved from the retinal stabilization system (stabilizing stimuli on the fovea of the retina) the efficacy of which

relies heavily upon the integrity of the floccular region of the cerebellum (Robinson, 1965). In effect, pursuit eye movements serve to stabilize or fixate a moving target on the fovea. Patients with lesions of the cerebellum, when tested for PEM performance under light-adapted conditions, demonstrate a marked disruption of pursuit tracking patterns (Hood, 1975). Interestingly, pursuit tracking performance of these patients is greatly improved in darkness (Hood, 1975) when cerebellar influences on oculomotor nuclei, thought to be effected through feedback to the cerebellum from the retinal periphery, are markedly reduced (Hood and Korres, 1979; Hood and Wanieski, 1983). Kornhuber (1974) suggests that disruption of such a feedback loop may be important for the occurrence of reversal saccades during PEMs.

There is limited evidence suggesting that cerebellar dysfunction may play a role in hyperactivity. Maiste (1986), in a study of vestibuloocular interaction in HK children, noted a significant reduction in the ability of nonmedicated HK children, relative to control children, to suppress nystagmus by optic fixation -- and fixation suppression is a process that relies heavily on cerebellar mechanisms (Hood and Korres, 1979; Hood and Wanieski, 1983; Zee, 1982). Of relevance to these observations are those by Shapira et al (1980) who, in their study of PEMs in HK children, noted the presence of nystagmoid eye movements superimposed on tracking

patterns and interpreted this finding as indicative of possible cerebellar dysfunction. Thus, cerebellar dysfunction could be a factor in hyperactivity and could play a role in the reported pursuit dysfunction associated with this disorder.

The purpose of the present investigation will be to assess, using standardized indexes (VAs, RMS and numbers of superimposed saccades), the degree of dysfunction of smooth pursuit tracking performance in HK children relative to control children. In addition, by assessing both behavioural symptomatology and PEM performance of the same HK children both when they are nonmedicated and again when medicated, the effect of medication on both variables can be determined. Also, comparing medication-related changes in both behaviour and PEMs will allow the determination of the degree to which these measures parallel each other. If a strong relationship between measures is noted, then PEM performance could serve to aid in the diagnosis of hyperactivity and in assessing the efficacy of stimulant medication treatment of this disorder. Varying lighting conditions under which PEMs are assessed will allow investigation of variation of both cerebellar and attentional influences on pursuit tracking. Dark adaptation is a simple manipulation which allows the investigation of PEMs under a condition which both attenuates cerebellar influences (reduces peripheral retinal feedback to the

cerebellum) and which simultaneously decreases visual environmental distractors--a consequence of which may be decreased distraction (i.e., involuntary inattentiveness) from the tracking target. Consequently, this investigation will provide new information regarding the relative contributions of medication, attentional and cerebellar influences to PEM performance of hyperactive children.

SUMMARY AND HYPOTHESES

Hyperactive children are purported to have visual information processing deficts that may be related to arousal and/or attentional dysfunctions. Furthermore, HK children are thought to be deficient on measures of PEM accuracy. The possibility that deficiencies in oculomotor control may contribute to the noted visual information processing deficits noted in HK children has not been adequately addressed. Distinctive aberrations in PEMs are thought to differentially reflect attentional factors (saccades in the direction of target motion) or physiological dysfunction (saccades in the direction opposite target motion). There are no reports in the literature comparing PEMs in HK and control children attempting to determine the relative contributions of these different factors to disordered PEMs. Furthermore, medication effects have not been studied in this context nor have variations in behaviour and in PEMs been related after

administration of stimulant medication. It is the purpose of this study to address these issues of physiological, medication and attentional influences on PEMs in HK children. Based on the preceding literature review, it is hypothesized that:

1) Relative to either control children or themselves while medicated, nonmedicated HK children:

   i) will evidence significantly more deviant PEMs as indexed by increased VA scores, RMS error scores and numbers of saccades;

   ii) will produce more superimposed positive saccades (reflecting attentional dysfunction) and reversal saccades (reflecting physiological dysfunction).

2) Relative to the nonmedicated condition, stimulant medication will reduce but not normalize (i.e., equate to control group levels), VA scores, RMS error and numbers of saccades of the HK children.

3) Under dark-adapted conditions:

   i) VA scores, RMS error values and numbers of positive saccades will be reduced to an equal extent for both HK children while nonmedicated and control children;

   ii) reversal saccades will be reduced to control group levels for the HK children while nonmedicated;

iii) all indexes of PEM tracking performance of HK children while medicated will be normalized.

METHODOLOGY

Subjects: Twenty 8-12 year old children (M = 9.63, SD = 1.71 years; 17 males, 3 females) diagnosed as hyperactive (Attention Deficit Disorder with Hyperactivity) served as the experimental (HK) group. Twenty age-matched volunteer children (M = 9.45, SD = 1.67 years; 8 males, 12 females) served as the control (C) group. Hyperactive children were referred from the Psychology Department of the Children's Hospital of Eastern Ontario, the Family and Child Unit of The Ottawa General Hospital and The Child Study Centre of the University of Ottawa. Control subjects were recruited from the general population.

Inclusion in the HK group was based on the following criteria: (i) scores of greater than 1.5 on the Hyperactivity/Impulsivity Index (HI) of the revised Connors Parent and/or Teacher Behaviour Rating Scales (Connors, 1969; Connors, 1976, Goyette et al, 1978) while nonmedicated; (ii) adherence to the criteria outlined in the DSM-III for Attention Deficit Disorder with Hyperactivity, i.e., exhibiting motor restlessness, short attention span, distractability, impulsiveness, labile emotions and poor

academic performance; (iii) presently receiving and responding favourably (based upon parents and physicians assessment of improved behaviour and ability to attend) to stimulant medication for the treatment of hyperactivity; and, (iv) symptoms must have been present prior to the age of 3. In addition, both control and HK children were living at home with at least one parent and were not receiving medication other than stimulants for hyperactivity (HK group). Additional exclusion criteria included diagnoses of major psychosis, overanxious reaction, unsocialized aggressive reaction, peripheral sensory loss, epilepsy, normal constitutional hyperkinesis, mental retardation, post-traumatic organic brain syndrome, encephalitis or toxic organic brain syndrome.

Pursuit eye movements of control subjects were assessed once, whereas this measure was assessed in HK children under both nonmedicated [HKnm] medicated [HKm] conditions. Only one recording session was conducted per day. For the HK children, the order in which the medicated and nonmedicated sessions took place was left to the discretion of the parents and physician. The HK children were normally medicated (methylphenidate: $M = 0.72$, $SD = 0.39$ mg/kg) and therefore, for the nonmedicated condition, all HK subjects stopped their daily medication regimen for one week prior to the recording session to allow for an adequate 'wash-out' of drug effects.

Since the order of medicated and nonmedicated trials was determined by the child's parent(s) and physician, the time period for which the HK subjects were on an uninterrupted daily medication regimen prior to the 'medicated' recording session varied across subjects. For those who were recorded initially while medicated (n = 13) the time period ranged from several months to one year. For those children recorded initially while nonmedicated (n = 7), the medication regimen had been reinstated for a minimum of seven days to a maximum of five weeks prior to the 'medicated' recording session.

Connors' Revised Parent and Teacher's Behaviour Rating Scale scores were obtained for each child (controls and HK subjects - the latter both on and off medication) near the time of each recording session. Control children's HI scores were found to be age appropriate according to norms established by Goyette et al. (1978) and were significantly lower than those of the HK group, either nonmedicated (C: $M = 0.68$, $SD = 0.32$; HKnm: $M = 1.83$, $SD = 0.52$; HKnm > C, $p < .001$) or medicated (HKm: $M = 1.53$, $SD = 0.68$; HKm > C, $p < .001$). Within the HK group, the decrease in HI scores in the medicated relative to the nonmedicated condition was not statistically significant.

Experimental procedures and techniques were explained to each child and his parent or guardian and forms of informed

consent were signed by both individuals.

Physiological recordings: Grass Instruments' gold-plated surface electrodes were placed at midline frontal, central and occipital sites (International 10-20 system; Jasper, 1958) and referenced to the right mastoid bone (M2) for recording of electroencephalographic activity (EEG, Grass Model 7P122 ac amplifiers, 0.3 Hz low rolloff to 100 Hz high rolloff, 60 Hz notch filter engaged). Activity from these derivations was monitored for signs of drowsiness during recording sessions. Beckman miniature silver-silver chloride (Ag-AgCl) electrodes were applied near the outer canthus of each eye and above and below the left eye to obtain measures of horizontal and vertical eye movements (HEOG and VEOG; Princeton Applied Research Model 113 preamplifier; 0.03 Hz low rolloff to 300 Hz high rolloff), respectively. Two Ag-AgCl miniature electrodes were applied to monitor variations in facial muscle activity (orbicularis oris muscle group) and to indicate head movement (EMG, Grass model 7P122 ac amplifier, 10 Hz low rolloff to 10 KHz high rolloff, 60 Hz notch filter engaged). Both the VEOG and EMG data were scrutinized for artefactual intrusions on PEM tracking pattens.

Experimental procedures: Subjects were positioned 1 meter from the target (2mm diameter red light emitting diode) with their heads stabilized by lateral and posterior

restraints to minimize head movement during the tracking trials. The task required that the subject track, with pursuit eye movements only, the movement of the target light through a sinusoidal pattern (0.45 Hz, i.e., 2.2 second period) subtending a 20 degree arc through the visual field (+/- 10 degrees from centre) for fifteen to twenty oscillations (33 to 44 seconds total trial duration). To ensure that subjects were attending to the target during the experimental task, the target light extinguished (200 msec off-cycle) in a semirandom sequence four to six times during every ten oscillations. Subjects were required to indicate detection of these interruptions by pressing a button held in the dominant hand (see Fig. 1). A practice trial was given to ensure that subjects understood all aspects of the task. Experimental trials were preceded and followed by a 30 second eyes closed condition during which EEG channels were monitored for signs of drowsiness or sleep (disappearance of alpha and replacement by theta and vertex sharp waves; the presence of rolling horizontal eye movements).

During each recording session the tracking task was presented in both light- and dark-adapted (subject in darkened light-tight room with eyes open for a period of at least ten minutes before the task was presented) conditions. The order of light- and dark-adapted conditions within a session was randomized across subjects, and also across recording sessions

for the HK children.

All physiological data as well as indicators of target motion, target light interruptions and subject responses were simultaneously displayed on Grass model 78 polygraph paper writeout (see Fig. 1) and recorded on magnetic media (Hewlett Packard model 8228A FM 8 track instrumentation recorder) for later computer analysis.

DATA ANALYSIS

Velocity Arrests: Beginning with the fourth oscillation in each trial (to allow for initial task orientation) ten consecutive oscillations of HEOG data were filtered (5.5 Hz low pass, attenuated 3 dB at cutoff, 48 dB rolloff per octave) and subsequently input to a Grass Model 7P21 electronystagmographic differentiator to yield indications of eye velocity. The differentiator was calibrated such that a velocity of 20 degrees/sec equalled 1 cm of pen deflection on polygraphic writeout. Two halfwave outputs were taken from the differentiator, one indicating velocity during eye motion from left to right, the other for right to left motion (see Fig. 2). These three channels of data (i.e., filtered HEOG and two differentiated HEOG signals) as well as the target motion indicator were simultaneously input to a PDP11/34 computer via A/D conversion at a sampling rate of 200 points

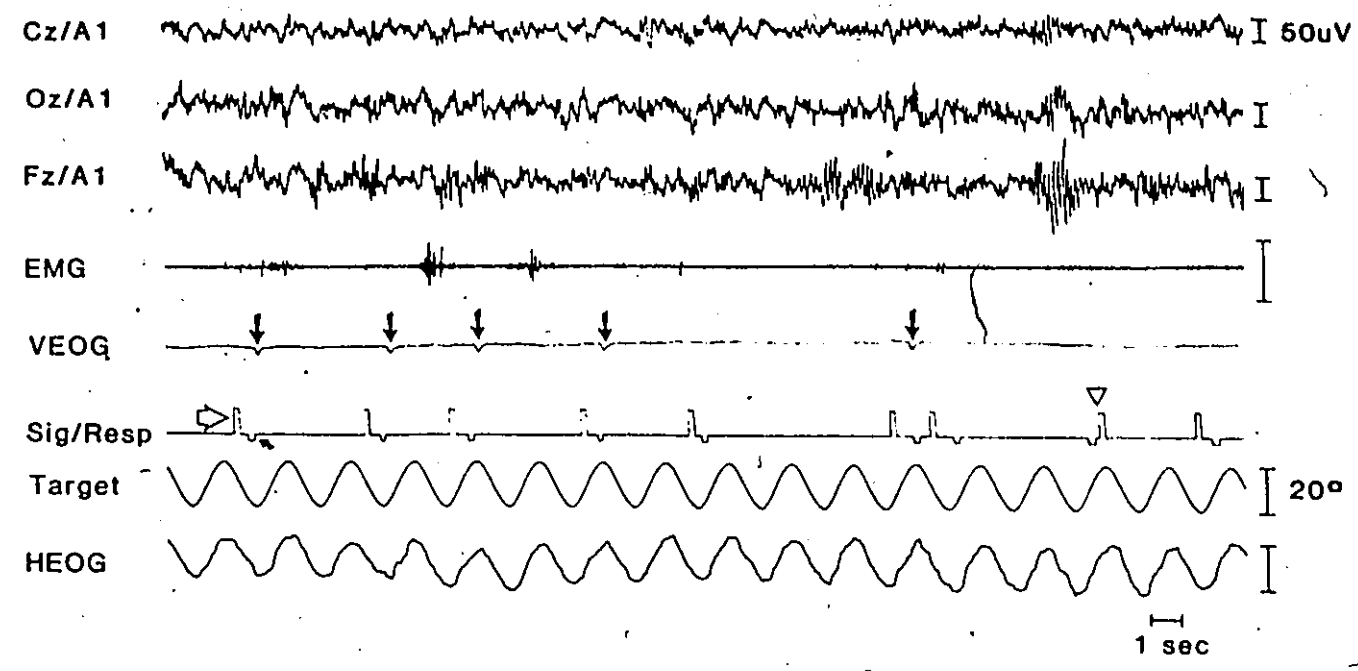


Figure 1. Polygraphic tracings of physiological and non-physiological data recorded during the experimental procedures. Derivations: Cz/A1 - central EEG; Oz/A1 - occipital EEG; Fz/A1 - frontal EEG; EMG - facial muscle activity; VEOG - vertical eye movement (arrows above the VEOG tracing indicate blinks); Sig/Resp - target 'off-cycle' indicator (upward deflection, open arrow), subject response (arrow below tracing) and an incorrect response (open inverted triangle); Target - target oscillation; HEOG - subjects' pursuit tracking eye movements.

per second per channel and stored on hard disk.

The two differentiated HEOG channels were computer scored for velocity arrests (VAs) according to previously established criteria, i.e., those instances where eye velocity falls to less than 2 degrees/sec for longer than 40 msec (Holzman et al, 1973; Pivik, 1979). The VA score associated with a given aberration depends upon its duration. Deviations with durations less than 40 msec were not considered. Deviations greater than 40 but less than 90 msec received a VA score of 1, and each subsequent increase in duration by 45 msec increased the VA score by 1. VAs detected within 200 msec of a blink (VEOG deflection greater than 2mm on paper writeout returning to baseline within 150 msec) or EMG artefact were deleted from further analysis. Mandatory VAs due to the halfwave analysis and target turn around points were eliminated from statistical analyses (n = 40 for each trial). The total VA score over 10 oscillations, after correction for artefact-related aberrations (blink or EMG associated) and mandatory VAs, served as an index of the accuracy with which subjects tracked the target, with higher VA scores indicating decreased accuracy.

Saccadic eye movements: The HEOG data utilized for the VA analyses were also scored for the occurrence of superimposed saccades. Saccades were operationally defined as

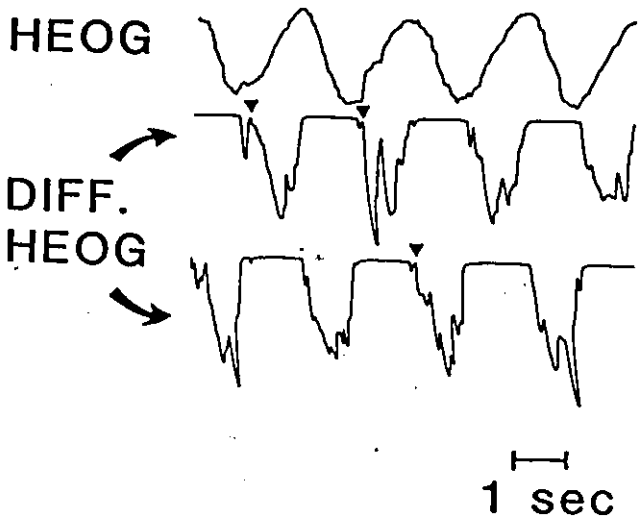


Figure 2. Illustration of eye movement data scored for VAs. The filtered horizontal eye tracking pattern (HEOG) is electronically processed for velocity [two bottom tracings (DIFF. HEOG)]. The filled triangles above these latter tracings index VAs. See text for discussion.

rapid eye movements greater than 2 degrees in amplitude with velocities greater than 40 degrees/sec. Saccades were computer scored and categorized according to whether they were 'positive' (same direction as the target) or 'reversal' (opposite target motion) saccades. The total number of saccades over 10 oscillations, the average amplitude and average velocity of each type were determined and stored for later statistical analysis. Saccades which occurred coincidentally with blinks or EMG artefact were deleted from analyses.

Root Mean Square Error: An analysis of positional differences between eye and target within each oscillation, i.e., root mean square error, was calculated to provide a global measure of tracking accuracy sensitive to the occurrence of deviations during ocular pursuit (Iacono and Lykken, 1979a, 1979b). Using the same ten consecutive oscillations upon which the VA analyses were based, HEOG and target indicator data were filtered (Khron-Hite model 3343, 10.0 Hz low pass, attenuated 3 dB at cutoff, 48 dB rolloff per octave), digitized at a sampling rate of 400 points per second per channel and stored on hard disk for later computer analysis.

The HEOG and target signals were equated for amplitude and adjusted for eye lag time (by minimizing the average time

discrepancy between eye and target zero cross, maximum and minimum occurrences). Subsequently, a point-by-point determination of eye position error relative to target position was made for each of the ten oscillations. These error values were squared and summed within each oscillation. An average of the squared error values was determined for each oscillation, and by taking the square root of this average, the average RMS value for each of the ten oscillations was obtained. The RMS error value for each oscillation and an average of these 10 error values (average error for all oscillations) was output and used for statistical analysis (see Fig. 3).

In order to explore the degree to which the global RMS error score is influenced by the discrete intrusions (VAs and saccades), RMS error scores were calculated for: (i) the time period between the beginning (time at which eye velocity falls to 2 degrees/sec.) and end (time at which eye velocity increases to greater 2 degrees/sec.) of each VA; (ii) during each saccade. Subsequently, the global RMS error was recalculated after subtracting RMS error coincident with the occurrence of VAs and saccades, singly and together. These latter values represent the global RMS error as adjusted for discrete intrusions.

Corneoretinal Potential: Electrooculographic measurement

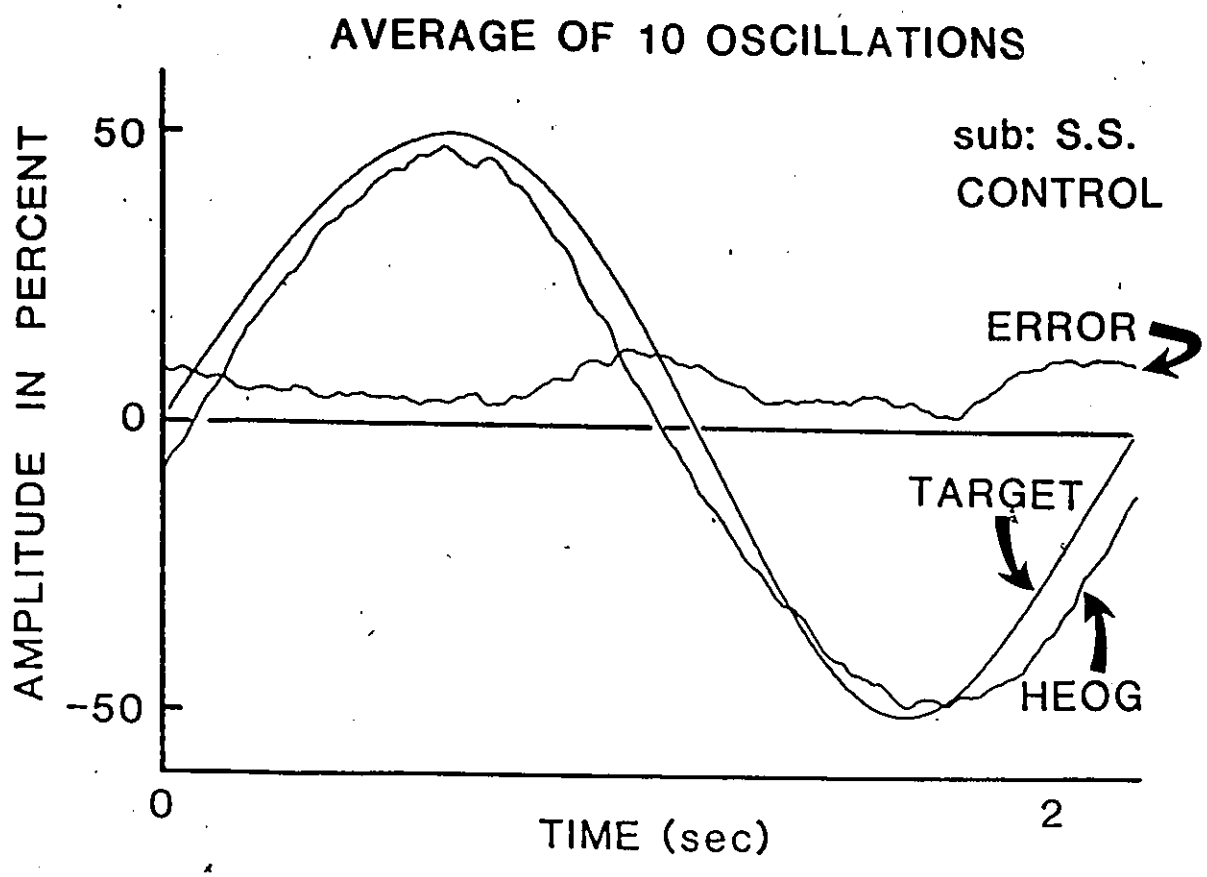


Figure 3. Depicted are the averages of the 10 TARGET and HEOG oscillations which constitute a trial. The ERROR tracing indicates the position error at each point along the oscillation averaged across the 10 oscillations. To obtain the RMS error score for the trial, these position error values are squared, summed across 10 oscillations and averaged to yield a single value indicating global RMS position error for that trial.

of eye movements is based on variations in corneoretinal potential (CRP) with respect to recording electrodes. Relative to light-adapted conditions, during dark adaptation a time-dependent variation in CRP has been reported (Dodt and Baier, 1984; Hickson, 1983; Jacobs, Feldman, Rabinowitz and Bender, 1973) characterized by a 10-20% CRP decrease in the first 15-20 minutes of darkness, followed by a marked (20-50%) increase after 20 minutes. This large phasic increase dissipates after approximately 1 hour, following which a slight (3-8%) tonic increase in CRP is maintained in darkness (Jacobs et al, 1973). Depending, then, on how long the subject was in darkness before recordings were taken, a given subject could show either an increase or decrease in CRP in the dark- relative to the light-adapted condition. In the present investigation, a time-dependent reduction in the amplitude of HEOG signals recorded under dark-adapted conditions could, if of sufficient magnitude, preclude reliable detection of small tracking aberrations and confound the light-dark condition analyses. To address this possibility, the peak to peak amplitude of the oscillatory HEOG signal for each subject in both light and dark conditions was converted to microvolts by comparing them with a sine wave of equal frequency (0.45 Hz) at a peak to peak amplitude of 1000 microvolts. A difference score, in microvolts, was obtained by subtracting the dark-adapted HEOG amplitude for a 20 degree eye movement from the HEOG amplitude for a 20 degree

eye movement recorded under light-adapted conditions. Subjects were categorized with regard to whether they were recorded either early (less than 20 minutes in darkness at the time of recording) or late (longer than 20 minutes in darkness before recording) in the dark-adapted condition. This group breakdown was made since after 20 minutes in darkness, the switch from cone to rod vision is complete. Breaking the groups into those who were recorded before or after 20 minutes in the darkness allows the determination of distinct influences of cone versus rod vision on CRP. CRP change scores (light score - dark score) were compared within and across groups and were correlated (Pearson product moment correlations) with similar change scores for VAs, RMS error values and numbers of saccades detected.

STATISTICAL ANALYSIS

Rationale for statistics used: While VAs, RMS error and the number of saccades superimposed on PEM are intercorrelated to a significant degree (see Appendix), these measures represent and index different aspects of PEM performance. Therefore, a plan of data analysis was employed in an attempt to determine the unique contribution of each type of deviation indexed by each measure to the PEM dysfunction of HK children. To that end, VA scores, numbers of saccades and RMS error scores were analysed using repeated

measures analysis of variance. The control group was compared to the HK group (both while nonmedicated and again in a separate analysis, while medicated) for each variable, with the repeated factor being lighting condition. Within the HK group, both lighting condition and medication condition served as repeated factors.

Analyses of variance: The fact that C children were recorded once while HK children were recorded twice precluded the analysis of all group, medication and lighting condition effects simultaneously. To assess the main dependent variables, three analyses of variance for repeated measures (BMDP program P2V) were performed for each variable (VA score, RMS error and the number of saccades). The first ANOVA compared C and HKnmed groups with lighting condition as a repeated factor. A similar analysis (2 groups, 1 repeated factor) was employed to compare C and HKmed groups across lighting conditions. A third ANOVA (repeated across medication and lighting conditions) assessed within-group effects for the HK group (means, standard deviations and ANOVA tables are given in detail in the Appendix). Due to the nature and number of these comparisons, a correction on the alpha level necessary to reach significance (.05) was employed to guard against the probability of increased Type I errors. Following the method of Kirk (1982), a Bonferroni correction was calculated for each source of variance -- groups (C vs

HKnm; C vs HKm), lighting condition (C - light vs dark; HKnm - light vs dark; HKm - light vs dark) and medication condition (HKnm vs HKm). With this correction applied, the alpha level for group effects was .025 (yielding a 'familywise error' of .05), .035 for lighting condition effects (yielding a 'familywise error' of .105) and remained unoorrected at .05 for medication effects. Post hoc Newman-Keuls tests were performed where warranted.

While the primary focus of the investigation was upon VA scores, RMS error scores and numbers of superimposed saccades, exploratory analyses of the amplitude and velocity of superimposed saccades and the contribution of VAs and saccades to overall RMS error were also performed. These secondary analyses were also corrected for alpha levels.

Additional ANOVAs were performed to assess possibly confounding lighting condition order effects within each group [1 grouping factor [light first vs dark first) repeated over lighting condition], medication condition order effects within the HK group [1 grouping factor (medicated first vs nonmedicated first) and repeated across lighting condition], as well as gender differences (1 grouping factor, repeated across lighting condition).

Correlations of Connors HI score and daily medication

dose with indexes of PEM dysfunction. The degree to which Connors HI scores, medication dose (in mg and expressed in mg/kg of body weight) and medication-associated changes in behaviour are correlated (Pearson product moment correlations; SPSS program) with similar changes in VA scores, RMS error and numbers of saccades was assessed for the HK children. In addition, partial correlations (SPSS program) of these variables were determined after correction for HI values in the nonmedicated state.

RESULTS

Subjects were cooperative during the experimental procedures and performed the tracking task as requested. For all subjects, statistical analyses of eye movements were based on scores calculated from 10 consecutive artefact-free oscillations of tracking data.

Groups were presented with equal numbers of target interruptions during tracking trials, and detection accuracy ranged from 80 to 90% across groups (see Table 1). Across lighting conditions (L: light-adapted; D: dark-adapted), the HKnm group was least accurate (L: 80%; D:82%), and HKm most accurate (L: 86%; D: 90%). Control subjects responded with intermediate accuracy (L: 84%; D: 86%). There were no statistical differences between groups or across lighting or

Table 1
Summary of Target Interrupt Detection Data

		C	HKnm	HKm
light adapted				
interrupts	M	5.16	5.15	5.25
	SD	0.60	0.93	0.79
responses	M	4.32	4.10	4.50
	SD	1.73	1.77	1.32
accuracy		84%	80%	86%
dark adapted				
interrupts	M	5.15	5.50	5.45
	SD	0.75	0.83	0.76
responses	M	4.45	4.50	4.85
	SD	1.15	1.64	1.53
accuracy		86%	82%	90%

medication conditions on this measure.

Order and gender effects: The possibility was examined of whether the order of lighting or medication condition presentation or the gender of the subjects confounded the results of this investigation. Analysis of variance across gender found no significant differences for any variable. Similarly, no significant order effects were noted for lighting condition for either controls or HK children. Medication condition order did not discriminate across subjects in the HK group. Therefore, order effects are discounted as the basis of differences across groups for PEMs performance accuracy.

Velocity Arrests: To determine possible differential group effects of lighting condition on the incidence of VAs, repeated measures ANOVAs across groups (i.e., C vs HKnm or C vs HKm) and repeated over lighting conditions were performed. In comparisons between C and HKnm subjects, a marginally significant main effect for group was noted, with the HKnm group producing more VAs than C subjects [$F(1,38) = 3.42$, $p < .055$]. Despite a large difference between group mean scores, post hoc Newman-Keuls analysis failed to find significant differences in either the light (C: M = 18.15, SD = 19.77; HKnm: M = 30.05, SD = 29.17) or dark (C: M = 7.30, SD = 7.76; HKnm: M = 15.55, SD = 14.27) condition. Hartley's

test of homogeneity of variance revealed that these groups were not homogenous for variance ($p < .01$), a violation of the assumptions underlying the ANOVA procedure. Although ANOVAs are relatively robust and are not markedly affected by violations of the homogeneity of variance assumption (Keppel, 1982), it was thought that this condition may have contributed to the inability to demonstrate statistically significant differences between groups. In an effort to reduce variability, a logarithmic transform of VA data was effected, and these transformed data were subjected to analysis of variance. Significant group differences were demonstrated [$F(1,38)= 6.15$, $p < .018$] and post hoc analyses revealed that HKnm children had significantly more VAs than C children in both light and dark conditions ($p < .05$, both conditions).

A similar analysis comparing HKm and C groups on VA scores, either nontransformed or transformed indicated no significant group differences (HKm - light: $M = 27.32$, $SD = 18.98$; dark: $M = 10.00$, $SD = 12.64$).

An analysis within the HK group repeated across both medication and lighting conditions found a nonsignificant reduction in VA score associated with medication. It is notable that while medication reduced the group average VA score for the HK group by only 10%, within-group variability was reduced by approximately 35%.

In all of the above analyses a significant main effect for lighting condition was found. For all groups a significant reduction in VA score in the dark- relative to light-adapted condition was noted ($.036 < p < .001$; see Figs. 4 and 5).

Saccadic eye movements: Saccadic eye movements were superimposed upon PEM patterns for all groups under all conditions. The majority of these intrusions were positive saccades in both the light- (C: 95%; HKnm: 84%; HKm: 88%) and dark-adapted (C: 98%; HKnm: 95%; HKm: 92%) conditions. Only 6 control (30%), 9 nonmedicated HK (45%) and 8 medicated HK (40%) subjects evidenced reversal saccades in the light-adapted condition, and these proportions were reduced in the dark-adapted condition (C: n = 2, 10%; HKnm: n = 6, 30%; HKm n = 4, 20% -- see Table 2).

Reversal saccades: For subjects who had reversal saccades, those saccades constituted only a small percentage of the total number produced in either light-adapted (C: 2% to 17%, M = 8.5%; HKnm: 5% to 38%, M = 15.5%; HKm: 7% to 34%, M = 12.8) or dark-adapted (C: 9% to 20%, M = 14.5%; HKnm: 2% to 21%, M = 9.2%; HKm: 4% to 17%, M = 9.5%) conditions. One control, 4 HKnm and 3 HKm subjects had reversal saccades in both lighting conditions, with the remaining subjects exhibiting reversal saccades in either the

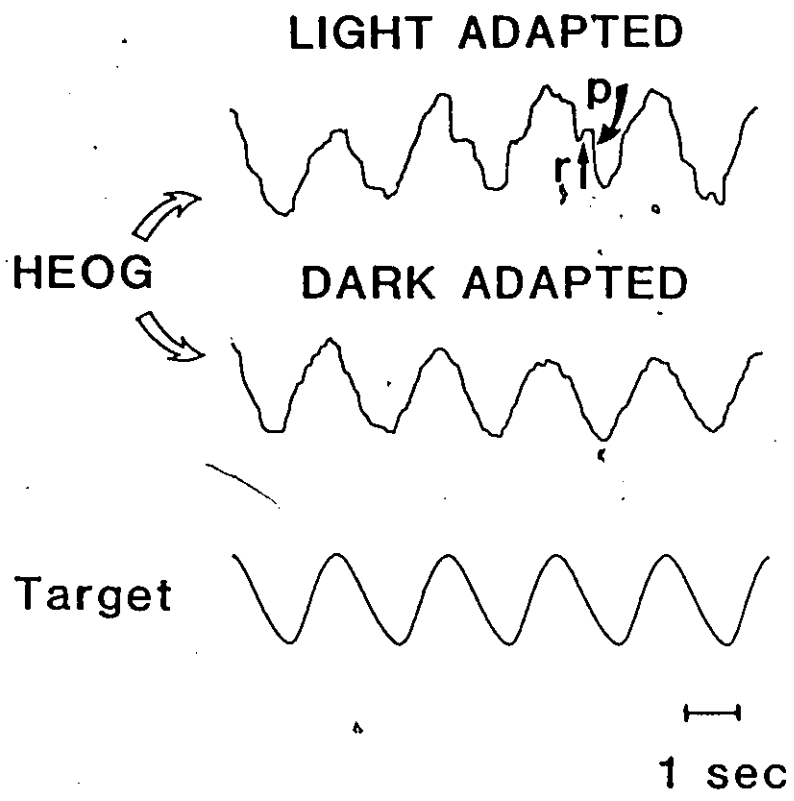


Figure 4. HEOG tracings recorded from the same subject under light- and dark-adapted conditions. Note the marked improvement in tracking accuracy in the dark condition. A reversal and a positive saccade ('r' and 'p', respectively) are indicated on the light-adapted HEOG tracing.

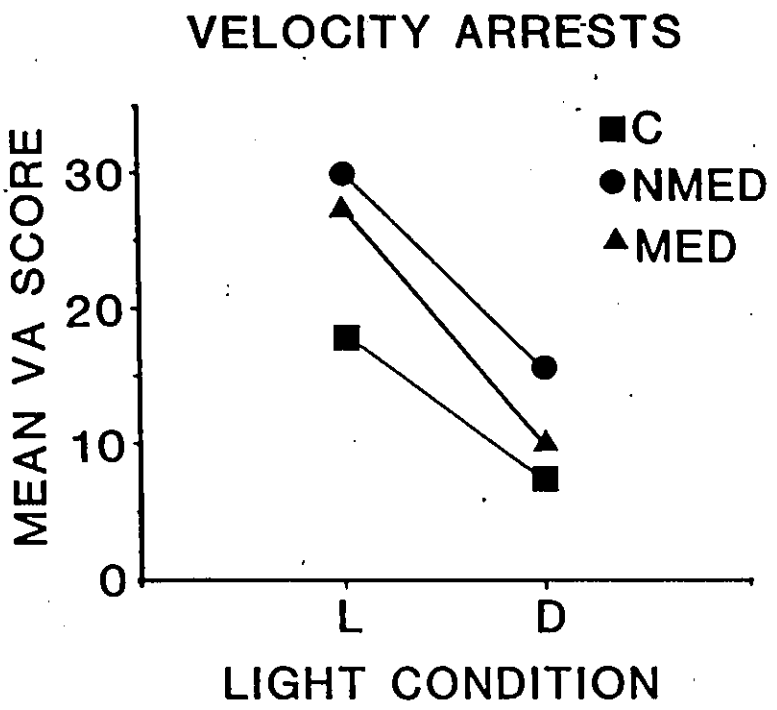


Figure 5. Group average VA scores graphed as a function of lighting condition [Group: C - control; NMED - nonmedicated HK; MED - medicated HK; Lighting condition: L - light-adapted; D - dark-adapted]. See text for discussion.

Table 2
Number of Saccades Superimposed on Pursuit Eye Movements as a Function of Group, Lighting Condition and Type

Group	Type	Light	Dark
Control	- total	332	180
	- % positive	95%	98%
	- % reversal	5%	2%
HKnm	- total	720	477
	- % positive	84%	95%
	- % reversal	16%	5%
HKm	- total	623	489
	- % positive	88%	92%
	- % reversal	12%	8%

light- or dark-adapted condition only.

The discrepant numbers of subjects across group, lighting and medication conditions limited the analysis of reversal saccade data to simple discriptive statistics. Controls had the lowest, HKnm the highest and HKm an intermediate number under light-adapted conditions. Dark adaptation was associated with a reduction in the number of reversal saccades for all groups, but this reduction was most pronounced for the HKnm and HKm groups. Reductions in HK group reversal saccades in the dark-adapted condition were such that the group average score approximated control group levels (see Fig. 6, Reversal column, top panel). Controls were intermediate for velocity of reveral saccades while the HKnm and the HKm groups had slower and faster velocities, respectively. Although saccades for all subjects were reduced in velocity in the dark-adapted condition, relative group position remained unchanged (see Fig. 6, Reversal column, middle panel). Amplitude of reverse direction saccades was the same for the C and HKnm groups and did not change significantly across lighting conditions. The HKm group evidenced slightly larger saccades in the light, but amplitude was reduced in the dark-adapted condition to levels approximating C and HKm levels. Additionally, reversal saccades for both control subjects and hyperactive children regardless of medication condition (see Fig. 6) were fewer in number, smaller in amplitude but of approximately equal

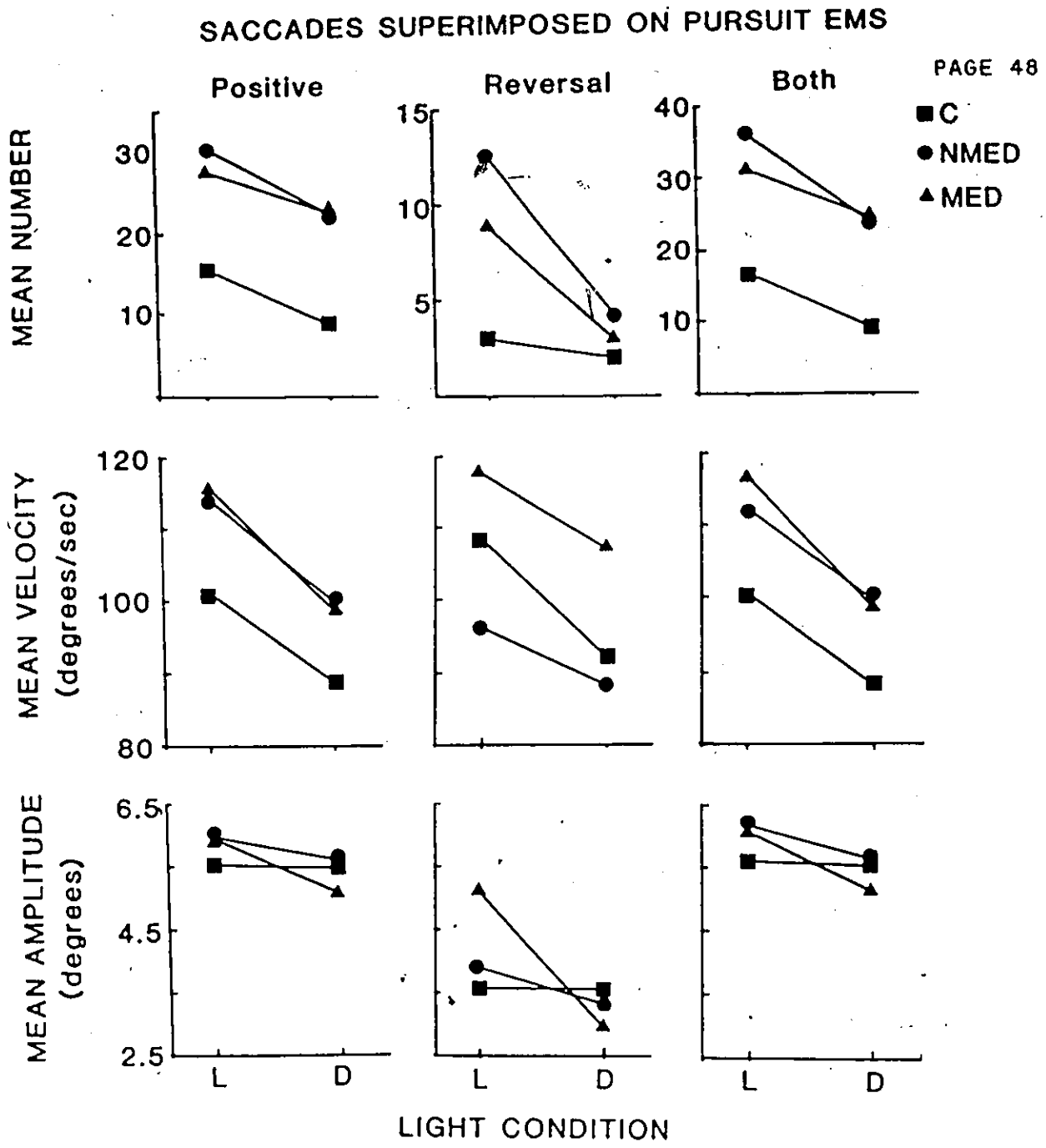


Figure 6. Summary graphs of the number, velocity and amplitude (upper, middle and lower horizontal panels, respectively) of saccades superimposed upon PEMs as a function of group and lighting condition. Saccades were examined according to direction relative to target motion, being either positive (left vertical panels) or reversal (middle vertical panels) saccades. The right vertical panels illustrate the averages after combining saccades in both directions. See text for discussion.

velocity relative to positive saccades.

Positive Saccades: Since all subjects showed positive saccades, a more rigorous analysis of these data was possible. Analyses comparing C to the HK group, either nonmedicated or medicated, for number, amplitude and velocity of positive saccades superimposed on PEMs (ANOVAs across groups and repeated over lighting conditions) revealed significant main group effects for the number of saccades produced but not for amplitude or velocity of these saccades. Therefore, saccades superimposed upon PEMs of HK children are deviant only in that they are more numerous relative to control group levels. Control subjects evidenced significantly fewer positive saccades than either HKnm [$F(1,38) = 6.43$, $p < .02$] or HKm [$F(1,38) = 5.39$, $p < .03$] subjects. Although HK subjects exhibited fewer saccades when medicated than nonmedicated, this reduction was not statistically significant (see Fig. 6, Positive panel).

Main effects for lighting condition were found, indicating significant reductions in the number ($p < .02$) and velocity ($p < .001$) of positive saccades, and trend toward significance for amplitude ($p < .05$), for all groups in the dark-adapted relative to the light-adapted condition. Lighting condition did not interact with group or medication status, and the pattern of group differences found in the

light was maintained during dark-adaptation.

Root Mean Square Error: ANOVAs performed on RMS error data did not indicate significant main effects for group or medication condition or their interaction. However, consistent with the results found for VA scores and positive saccades, a significant main effect for lighting condition was present for all groups. A significant decrement in RMS error was noted in the dark- relative to the light-adapted condition ($p < .01$; see Fig. 7).

The RMS error measure includes in its calculation of global error those instances where discrete aberrations (i.e., VAs and superimposed saccades) are present. In an effort to assess the contribution of these discrete aberrations to global RMS error, this error value was recalculated after removing from the total error that associated with VAs and with saccades separately and in combination. Although these manipulations did reduce the average RMS values, they did so equally for all groups and therefore did not alter the pattern of group means for the uncorrected RMS error measure.

Corneoretinal Potential: There was a small average increase in CRP during dark adaptation for control (M = 28 uV or 7% of light-adapted value) and HKm children (M = 20 uV or 6% of light-adapted value). Nonmedicated HK children,

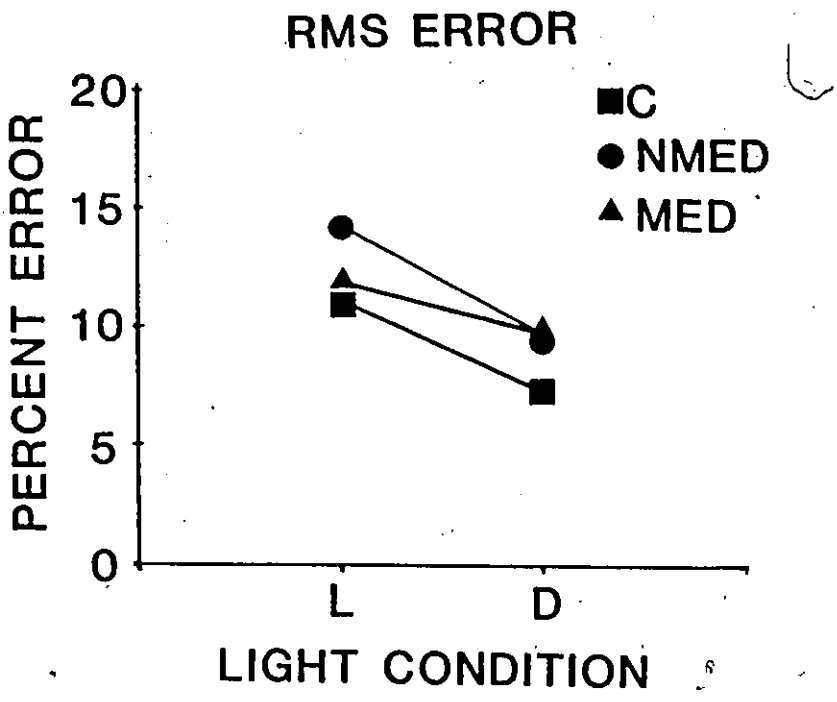


Figure 7. Variation in group average RMS error data across lighting condition. See text for discussion.

however, showed a slight CRP decrease during dark adaptation (M = -18 uV or 5% less than light-adapted value). There were no significant differences across groups or within the HK group across medication conditions on this measure. Eight (8) control, 8 HKnm and 10 HKm subjects showed CRP decreases in the dark, while the remaining subjects evidenced either an increase (12 controls, 11 HKnm, 9 HKm) or no change (1 HKnm, 1 HKm) from light- to dark-adapted conditions. Control children, whether recorded early or late in the dark-adapted condition, showed an increase in CRP in darkness (early, n = 12, 5.8% increase; late, n = 8, 9.3% increase). HK children recorded early in the dark adaptation condition -- regardless of medication -- had slightly smaller CRPs (HKnm, n = 13, 2.3% decrease; HKm, n = 11, 4.3% decrease) than in the light condition, whereas those recorded after longer periods of dark adaptation consistently showed slightly larger CRPs (HKnm, n = 7, 7.5% increase; HKm, n = 9, 6.8% increase). However, CRP light-dark change scores correlated negatively with comparable change scores for VAs, RMS error and the number of saccades superimposed on PEMs (See Table 3).

Correlations between Connors HI, medication and PEM performance indexes: To determine whether the 'baseline' degree of behavioural symptomatology affected the medication-related change in indexes of PEM performance, Connors HI scores of the HK group obtained for the

Table 3
Group Correlations (and associated significance levels) Between Changes in Corneoretinal Potential (CRP) and Indexes of Pursuit Eye Movement Accuracy from Light- to Dark-adapted Conditions

	Control	HKnm	HKm
CRP with			
VA	-0.17 ns	-0.31 p < .10	-0.21 ns
RMS	-0.31 p < .10	-0.14 ns	-0.50 p < .02
Saccades	-0.47 p < .02	-0.17 ns	-0.52 p < .01

nonmedicated condition were correlated with medication-related changes in VA scores, RMS error values and numbers of saccades. In addition, a partial correlation of medication dose with medication-related changes in VA, RMS and saccades measures controlling for the influence of HI scores from the nonmedicated condition was performed. Connors HI scores of nonmedicated HK children did not correlate significantly with medication-related changes in any of the indexes of pursuit tracking performance. However, medicated HK children's Connors HI scores showed a significant positive relationship with medication-associated changes of both positive ($r = 0.50$, $p < .05$) and reversal ($r = 0.42$, $p < .05$) saccades, but were not meaningfully related with similar changes in other indexes of pursuit performance.

Daily dosage of medication (expressed in mg) was not significantly related to changes in pursuit performance. Furthermore, partialling out the influence of HI scores did not alter the correlations of medication dose with medication related changes in PEM performance indicators. However, when expressed as a function of body weight (i.e., mg/Kg), medication dose was correlated with medication-related decreases in VA scores ($r = 0.45$, $p < .05$) and increases in velocity of positive saccades ($r = 0.54$, $p < .02$). Medication-associated changes in the Connors HI were weakly correlated with similar changes in RMS error values ($r = 0.41$,

$p < .07$) and showed a stronger correlation with medication-associated changes in positive saccade velocity ($r = 0.54$, $p < .02$).

DISCUSSION

The observation that nonmedicated HK children evidence disordered smooth pursuit tracking corroborates previous reports (Bala et al, 1981; Shapira et al, 1980) and confirms a major hypothesis of the present investigation. Indexes of aberrant tracking included significantly greater VA scores for HK children while nonmedicated and significantly greater numbers of positive saccades under both medication conditions relative to control subjects. In addition, although the incidence of reversal saccades was not remarkable in light-adapted conditions, more HK than control subjects exhibited this kind of intrusion, lending partial support to the hypothesized physiological basis of tracking dysfunction in HK children. RMS error scores, however, did not discriminate between groups. Stimulant medication effected only a slight, nonsignificant reduction in tracking errors relative to those present in the nonmedicated state, failing to confirm an expected significant medication-related reduction of tracking dysruption in HK children. However, a marked reduction in the variability of HK group data was noted in the medicated condition. Dark adaptation was associated

with the anticipated significant reductions in VA scores, positive saccades, and RMS error values relative to scores in the light-adapted condition for both subject groups. For those subjects who evidenced reversal saccades, incidence of these aberrations was equal across groups in this condition. There was only partial confirmation of the hypothesis that combining medication and dark adaptation effects would normalize tracking in HK children. In this condition, HK group VA scores approximated control group values, representing a larger decrease on this measure than was noted with either darkness or medication alone. Furthermore, these combined manipulations were no more effective than dark adaptation alone in influencing upon the number saccades -- positive or reversal -- evidenced by the HK children. There are a variety of potentially confounding variables which could have influenced these results and which must be addressed before discussing the implications of the data. Such factors include methodological limitations, subject drowsiness, inattention or artefact in physiological recordings.

Electrooculography is an accurate, reliable and accepted psychophysiological technique for recording eye movements. Eye displacements of 1 degree visual angle can be detected by this method. The operational definition of a saccade utilized in the present investigation (2 degrees visual angle) is well within this range and therefore the EOG recording technique

cannot be considered a limiting factor in the ability to detect saccadic intrusions. Furthermore, the VA and RMS scoring methods employed have been utilized in numerous studies and have been shown to distinguish between groups of normal and experimental subjects (Holzman et al, 1973; Iacono and Lykken, 1979a, 1979b; Pivik, 1979; Shagass et al, 1974). Similarly, computer scoring of saccades is a reliable technique (Bala et al, 1981; Maiste, 1986) and this measure has been shown to distinguish between control and HK groups (Bala et al, 1981). Order effects can also be excluded as possible confounds since the order of occurrence of light- and dark-adapted trials, as well as that of nonmedicated and medicated recording sessions, was randomized and nonsystematic across groups.

It is known that younger children execute PEMs less efficiently than older children (Gilligan, Mayberry, Stewart, Kenyon and Gaebler, 1981; Kowler and Martins, 1982). Since the two groups in the present investigation were age matched this factor cannot account for the results noted. In addition, subject gender was found not to be a significant factor contributing to the results. Similarly, the order of presentation of lighting and medication conditions had no statistically significant effect on the results.

The monotony of the tracking task and the use of dark

adaptation are conditions which can induce drowsiness and therefore electroencephalographic data were monitored (beginning 30 seconds before and extending until 30 seconds after each trial was complete) to detect signs of drowsiness. When such indications were detected, the subject was realerted and experimental procedures were repeated.

Normal physiological events such as blinking or movement can introduce artefact into HEOG recordings. To ensure across-group variations in these events did not contaminate the results, VEOG and EMG data were monitored for blinks and movement-associated influences on HEOG patterns. Tracking data found to be associated with such artefact were eliminated from statistical analyses.

A reduced level of voluntary attention is a major behavioural symptom of hyperactivity and has been found to be associated with increased pursuit tracking errors (Holzman et al, 1976; Pivik, 1979; Shagass et al, 1974). To direct attention toward the target during tracking trials, an attention-enhancing task (button-press response to randomly presented target light interruptions) was included on all tracking trials. Across groups and recording conditions subjects responded with equal accuracy to these target interruptions suggesting that subjects were equally attentive to the task.

An additional influence that could compromise the validity of the dark-adapted data is the variation in CRP induced by darkness. However, for both groups changes in CRP were found to be inversely correlated with changes in indexes of tracking aberration. This pattern of correlation is inconsistent with an explanation of the noted tracking improvements based on systematic variations in CRP.

In the preceding paragraphs, the possibility that technical, methodological or procedural confounds significantly influenced the results of this investigation has been discounted, and it must therefore be assumed that the data reflect real differences between groups and across conditions. In the following discourse, these data will be examined with reference to the current literature -- in the areas of both hyperactivity and eye movement -- in order to explore and perhaps better define the nature of the dysfunction which results in PEM aberrations in HK children.

The results of this investigation are in general agreement with those of Shapira et al (1980) and Bala et al (1981) who reported oculomotor performance deficits in HK children in the form of more irregular tracking patterns and more saccadic interruptions of pursuit eye movement. Additional indexes of tracking performance in this investigation included velocity arrests and RMS error

measures. VAs differentiated groups, RMS error measures did not. In investigations of PEMs in adults, both measures have been shown to discriminate between normal and patient groups (Iacono and Lykken, 1979a, 1979b, 1981; Iacono, Tauson and Johnson, 1981; Pivik, Bylsma and Cooper, 1986). However, the VA index has been shown to be more sensitive to group differences (Pivik et al, 1986), an observation corroborated by the present results where VA but not RMS error scores discriminated statistically between C and HK groups. In this study, this observation may be accounted for by the fact that although HK children evidenced large numbers of discrete tracking aberrations, analysis of RMS values calculated during VAs (i.e., from VA start to VA end) did not discriminate between groups. These data suggest that, while being more numerous, HK subjects' VA tracking errors did not result in larger average eye-target position differences, as indexed by RMS error scores, than those of control children. In addition, for any subject group the total duration of brief disruptions (VAs) per trial did not constitute more than 5% of the total time over which global RMS error scores are calculated (10 oscillations each of 2.2 second duration, or 22 seconds). This implies that the tracking patterns of the HK children were largely accurate, but were frequently interrupted by discrete aberrations.

There is a marked difference between what the VA and RMS

error indexes measure. The VA score, and similarly the saccade score, indicates the presence and number of discrete deviations in pursuit eye movements. Alternately, the RMS measure incorporates into its calculations the entire tracking trial -- regardless of whether performance is accurate or inaccurate -- and yields a single score which indicates the average eye-target position difference across 10 oscillations. Unless tracking patterns show a marked and relatively consistent morphological variance from the target pattern, increased RMS error calculated during periods of inaccurate tracking would be computationally offset by less extreme RMS error during periods of accurate tracking. In this manner, increased RMS error during discrete aberrations (VAs and saccades) would be averaged together with, and effectively negated by, RMS error during accurate tracking. This would reduce the sensitivity of the RMS measure to real differences between groups for prevalence of discrete aberrations. If only the RMS error measure is examined, investigations could fail to detect significant and informative group differences on measures of discrete PEM aberration. In this light, it would appear imperative that future investigations of eye movement performance include discrete scoring methods (VA and/or saccade) in addition to the global RMS error method.

In the literature, deficits in PEM performance have been reported: (i) in normal young adult subjects under conditions

of reduced attentiveness, increased distraction, after administration of certain medications (alcohol, barbiturates, chloral hydrate), and in older (60+ years) relative to younger (20-35 years) normal adults; (ii) in adult psychiatric patients evidencing psychotic symptoms (primarily schizophrenic patients) and their first order relatives; (iii) in patients with major affective disorders (depression, bipolar disorder) and in association with treatment of affective disorders with lithium carbonate; and, (iv) in neurological patients with cerebellar lesions. While not an exhaustive list of conditions with which PEM dysfunction has been associated, these studies implicate levels of attention (voluntary and involuntary), the anatomical and functional integrity of cerebellar structures and certain medication effects as important factors related to PEM performance. These factors will now be discussed with respect to the results of the present investigation.

Attention effects on PEM performance in HK children

In the present investigation two methods of increasing attention were employed, i.e., inclusion of the button-press task during the tracking trial and reducing potentially distracting stimuli by dark adaptation. The two methods differ in the manner in which they enhance attention. The button press task engages attention by increasing salience of

the tracking target and implies a voluntary, active facilitation of attentive behaviour. In contrast, increasing attention by reducing distractors in the dark-adapted condition is a passive, nonvoluntary process. Assessing differential influences of the button press task and dark adaptation on PEMs aberrations may yield evidence regarding the nature -- active versus passive -- of attention-related involvement in PEM dysfunction noted in HK children.

The button-press task during tracking is comparable to the 'voluntary-attention-enhanced' conditions utilized in other investigations of PEM dysfunction (Holzman et al, 1976; Pivik, 1979). In the present investigation, HK children evidenced significantly increased VA scores and numbers of saccades relative to control subjects despite responding to target interruptions with accuracy equal to control group levels (indicating equal across-group levels of voluntary attention). This pattern of results parallels comparisons of normal and schizophrenic adults (Pivik, 1979) and suggests that the PEM aberrations of HK children do not result from a dysfunction of directed attention.

In the dark-adapted condition, inclusion of the button press task ensured that voluntary attentiveness was maintained. Dark adaptation was associated with significant reductions in VA scores, RMS error and numbers of positive

saccades for both subject groups. However, significant differences remained between control and nonmedicated HK subjects for VA score and incidence of positive saccades. Residual tracking errors of adult schizophrenic patients noted after voluntary attention had been enhanced are posited to result from 'phasic interruptions of nonvoluntary attention' (Holzman et al, 1976). In the present investigation, the observation that increasing nonvoluntary attention by dark adaptation reduced tracking errors but did not normalize PEMs performance of HK children argues forcibly that while attention may be an important factor in these results, it is not the primary cause of oculomotor deficits for these children.

Cerebellar influences on oculomotor function

In addition to the presumed attentional effects of dark adaptation noted above, this manipulation is also known to reduce cerebellar influences on oculomotor nuclei. Interruption of this influence by lesions has been shown to result in severe disruption of PEMs (Hood, 1975), and the attenuation of deviant cerebellar influence by darkness is associated with marked improvement in PEM performance (Hood and Korres, 1979; Hood and Waniewski, 1984).

Recent investigations of vestibulo-ocular interactions in

schizophrenic patients have provided data suggesting that cerebellar dysfunction may contribute to PEM deficits observed in these patients. The ability to effect suppression of nystagmus by visual fixation of a stationary target (fixation suppression) is intimately related to cerebellar function and is sensitive to cerebellar dysfunction (Hood, 1975; Hood and Waniewski, 1984). Cerebellar mechanisms are thought to be involved in effecting both fixation suppression and PEMs (Benson and Barnes, 1978). Coexistent deficiencies in these oculomotor behaviours have been reported in both patients with cerebellar lesions (Hood, 1975; Hood and Korres, 1979) and in schizophrenic patients (Jones and Pivik, 1983; Pivik et al, 1986). For both patient groups pursuit tracking performance is compromised in light-adapted conditions, while in dark-adapted conditions control-group levels of performance are achieved. These tracking improvements in darkness have been attributed to attenuation of deviant cerebellar influences on oculomotor nuclei (Hood and Waniewski, 1984; Pivik et al, 1986).

There is limited evidence that cerebellar dysfunction may contribute to hyperactive symptomatology and therefore may contribute to PEM dysfunctions observed in these children. Shapira et al (1980) reported that HK children exhibited nystagmoid interruption of pursuit tracking and interpreted these data as indicative of possible cerebellar dysfunction.

Maiste (1986) found that nonmedicated HK children evidenced significantly less fixation suppression of caloric nystagmus than control children and suggested these findings indicate cerebellar dysfunction in the former group. If tracking aberrations in HK children are related to deviant cerebellar influences, then dark adaptation should be associated with a normalization of tracking performance for these children. However, only numbers of reversal saccades were reduced to control group levels in the Hknm group by this manipulation. Significant group differences remained between control and HKnm groups for VA score and numbers of positive saccades. These results are in contrast with darkness-related effects noted in both schizophrenic patients--for whom this manipulation statistically normalized RMS error and VA scores--and cerebellar patients whose tracking performance was greatly improved in darkness. Taken together these findings suggest that either cerebellar dysfunction is not a major factor contributing to PEM dysfunctions of HK children, or that the ameliorative effects of reduced cerebellar influence are somehow overridden -- perhaps by optogenetic differences in cerebellar function or by some other age-related dysfunction which influences PEM performance. However, the failure of HK children to effect normal fixation suppression, a sensitive index of cerebellar function (Zee, 1982), argues for the presence of some form of cerebellar dysfunction associated with hyperactivity. It should be noted, however,

that despite a significant reduction from control group levels on this measure, the reported level of suppression attained by the HK group (approximately 72%; Maiste, 1986) is well within the range of normality for adult subjects (50-60%; Takemori, Aiba and Shiozawa, 1981). Cerebellar influences on eye movement are modulatory, suggesting neurochemical involvement. Thus, PEM-related cerebellar dysfunction in HK children may result from a lowered degree of neurotransmitter efficiency relative to control children and not a structural abnormality -- a contention reinforced by the absence of signs commonly associated with cerebellar lesions (i.e., ataxia). Neurochemical imbalance, particularly of catecholaminergic transmitters, has been posited as an important factor underlying hyperactivity and serves as the logical and theoretical basis for treatment of hyperactivity with stimulant medications (Shaywitz, Cohen and Shaywitz, 1978; Shaywitz, Hunt, Jatlow, Cohen, Young, Pierce, Anderson and Shaywitz, 1982; Wender, 1976).

Medication effects on PEM performance of HK children

Administration of stimulant medication is associated with reductions in indexes of behavioural symptomatology, a trend toward normalization of responses on variables thought to indicate hypoarousal, and increased attentive behaviour in HK children. Stimulant medications appear to have no direct

effect on oculomotor structures involved in PEM since the administration of a prominent and commonly used stimulant, i.e., amphetamine, has not been associated with change in PEM performance (Tedeschi, Bittencourt, Smith and Richen, 1983 -- 15 mg oral dose) or in incidence of positive or negative saccades or overshoots superimposed on tracking patterns of normal adult subjects (Filip, David and Filipova, 1978 -- 20 mg oral dose). However, these medications do increase levels of arousal, and this effect is reflected in the oculomotor system as reduced saccadic reaction times and the attenuation of fatigue effects (Tedeschi et al, 1983). There is evidence as well of similar activating effects of stimulant medications on oculomotor behaviour in HK children. Maiste (1986) reported medication-related increases in vestibular reactivity (increased slow and fast velocity components of nystagmus and increased nystagmus frequency) and similar enhancements in parameters of voluntary saccades (increased peak velocity and reduced saccadic reaction time) in a group of hyperactive children. A similar increase in velocity of nystagmus slow phase found in adults in conjunction with mental alerting by serial subtraction during ongoing nystagmus (Jones and Pivik, 1983) was attributed to increased arousal induced by the mental alerting task. Taken together, these data suggest that the facilitatory influence of stimulant medication on oculomotor functioning, as reflected by reductions in saccadic reaction time and less susceptability to fatigue, could be-

either a direct influence on the oculomotor nuclei, or a secondary effect due to increased CNS arousal.

In the present investigation, stimulant medication was associated with small, nonsignificant reductions in VA score, numbers of saccades and RMS error in light-adapted conditions. Although in the right direction, these results do not corroborate those of Bala et al (1981) who reported a normalization of saccade incidence in association with administration of medication. In that study medication effects were compared across two separate unequally sized groups of HK children, one nonmedicated and the other medicated. Furthermore, within the medicated group not all subjects were recorded under all experimental conditions. This within-group difference across medication conditions could limit the ability to reliably detect true group and condition effects. In the present investigation, to ensure that the results were not compromised by subject variables, within-group subject characteristics were held constant by recording the same HK children both under nonmedicated and medicated conditions.

A direct relationship was noted between daily dose of medication and amount of reduction in VA score ($r = .45$, $p < .05$). * Similarly, Connors HI scores obtained from HKm children were found to vary directly with medication-related reductions

in both positive (r = .50, p * .05) and reversal (r = .42, p * .05) saccadic intrusions. Taken together these data suggest that medication-associated reductions in tracking dysfunction may parallel medication-related reductions in behavioural symptomatology in HK children. Stimulant medications are thought to exert their ameliorative effect on behaviour indirectly by increasing CNS arousal levels, and may affect PEM performance in a similar manner. This position is supported by the positive correlation between amount of increase in saccadic velocity in the medicated condition with daily dose of medication (mg/kg; r = .54, p * .02) and similarly, with medication related changes in Connors HI index (r = .54, p * .02). These data argue that PEM dysfunction in HK children may derive from underarousal which results in a secondary, disruptive influence on the oculomotor system.

In the present investigation, saccades of HK children were not significantly different from control group saccades for amplitude or velocity, corroborating similar findings reported by Maiste (1986) who noted no significant group differences between HK and control children for latency, amplitude, velocity or duration of voluntary saccades. Together these data suggest that saccadic eye movements are not deficient in HK children. These data are paralleled by the results of an investigation of saccades and pursuit in remitted psychotic adults (Iacono, Tauson and Johnson, 1981).

These authors reported pursuit abnormalities in the absence of evidence of deficiencies in the saccadic system. In the present investigation, saccades are considered aberrant only because they intrude upon PEM tracking patterns more frequently for HK children than control children, and not because of across-group differences in saccade characteristics. These data could be interpreted as indicating that the deficiency in oculomotor performance of HK children is limited to the pursuit system. However, under certain circumstances, saccades superimposed upon pursuit eye movements are both normal and beneficial. For instance, when the eye lags the target by too great a margin, a corrective 'catch-up' saccade repositions the target on the fovea. Therefore, since the pursuit and saccadic systems are so intimately interrelated, it is impossible from the present data to state without qualification whether only the pursuit, the saccadic or both systems are affected. An 'overdriven' saccade generation system, or alternately, decreased control -- volitional on nonvoluntary -- over this system, are conditions which could result in an increased number of saccades exhibited during pursuit by HK children.

The greatest opportunity for attentiveness to the task and for accurate PEMs would appear to be created when the button-press task, dark adaptation and medication are combined. Attention is enhanced by the button-press task, the

reduction of distraction by dark adaptation and the administration of stimulant medication. Physiologically, dark adaptation reduces cerebellar influences on PEMs while medication acts to increase arousal levels. In this condition VA scores were reduced to control group levels in the HK children while reductions in positive or reversal saccades produced by HK children did not exceed reductions associated with dark adaptation alone. The combined effect of medication and dark adaptation on VA scores of HK children is comparable to the normalization of VA and RMS error scores in inpatient schizophrenic adults under similar conditions (Pivik et al, 1986). In the present investigation, the normalization of VAs suggests that combining the effects of medication (reduced variability) and darkness (significant decrease of group mean scores) on PEM performance are synergistic since neither manipulation alone normalized tracking dysfunctions in HK children. Stimulant medications are thought to correct for decreased levels of CNS arousal and thereby ameliorate behavioural symptoms and increase attentiveness in HK children. It is conceivable that medication-associated increases in CNS arousal could contribute to the observed normalization of VA scores -- increased arousal could allow a more normal engagement of the PEM system, reducing the probability of interruption.

Although VAs and positive saccades are not identical

aberrations, it is surprising that HK group positive saccade incidence remained unchanged while VA scores normalized when dark adaptation and medication are combined since both measures are thought to reflect a dysfunction of attentional systems. Since numbers of reversal saccades were reduced to control group levels by dark adaptation alone, medication would not be expected to effect a further significant reduction. The lack of effect on positive saccades by this manipulation can be explained if stimulant medication did not completely normalize arousal levels and, secondarily, performance of the pursuit system. Eye-target position errors (retinal slip, Robinson, 1965) are corrected by positive saccade eye movements which reposition the target on the fovea. If the PEM activation signal is not sufficient to ensure complete accuracy of eye-target position, small corrective saccades would be necessitated, and this could explain the continued prevalence of significantly more positive saccades exhibited by the HK children.

The differential response across measures of PEM performance to attention, dark adaptation and medication manipulations, alone and in combination, suggests a dissociation of aberrations. However, the intricate interconnections between the pursuit, saccadic and cerebellar systems make it difficult to conceive of three separate dysfunctions acting independently and simultaneously, but in a

discrete manner, to effect VAs, or positive or reversal saccades during tracking trials. A more parsimonious conception is that a single dysfunction with widespread CNS representation influencing pursuit, saccadic and cerebellar systems serves as the basis for all types of PEM aberrations. The results of this investigation lend themselves to this interpretation regarding PEM aberrations, specifically with regard to attention, cerebellar influences and arousal levels.

Decreased attentiveness to the target -- either intentional or involuntary -- can be discounted as the primary basis of PEM deficits in HK children since: (i) all groups performed the button-press task with equal accuracy and can therefore be thought of as equally attentive to the task -- yet varied in PEM performance; and, (ii) dark adaptation reduced both measures of PEM dysfunction believed to reflect nonvoluntary attentiveness (VA score and positive saccade incidence) for both control and hyperactive children, but did not normalize HK group scores. Similarly, cerebellar dysfunction alone cannot serve as the basis of tracking dysfunction in HK children since the attenuation of cerebellar influences on oculomotor nuclei in darkness effected a normalization of only reversal saccade incidence (the least prevalent aberration) while reducing, but not normalizing, VA scores or numbers of positive saccades in HK children.

Noting that groups were equally attentive to the tracking task, the synergistic action of stimulant medication -- thought to increase both arousal (Ferguson and Pappas, 1979) and attention (Douglas, 1983) -- and darkness to effect a normalization of VA scores suggests that both inadequate arousal levels and aberrant cerebellar function may be involved in PEM dysfunction in HK children. The abnormal interaction between arousal mechanisms, the PEM system and cerebellar structures -- due perhaps to some neurochemical deficiency which stimulant medications correct -- could result in an inability to accurately modulate pursuit eye movements.

There is controversy in the eye movement literature regarding the localization of the CNS dysfunction responsible for aberrant PEMs dysfunction in schizophrenic patients, and these considerations have relevance to the results of this investigation. Levin (1984) cites a body of literature which implicates frontal cortical structures as the site of this disturbance. That author reasons that in the absence of evidence indicating damage to or dysfunction of brainstem structures involved in eye movement regulation, cortical structures implicated in maintaining goal directed behaviours must be at fault. There is evidence that the frontal eye fields are involved in suppression of saccades during PEM tracking and Levin (1984) suggests damage or dysfunction in this area may be associated with saccadic interruption of

tracking patterns in schizophernic patients. However, recent demonstrations that actively ill schizophrenics who exhibit impaired PEMs also have deficient fixation suppression of vestibular nystagmus (a process mediated by the cerebellum) and that these two measures are highly correlated in these subjects (Jones and Pivik, 1983) implicate subcortical (cerebellar) structures as important in PEMs dysfunction. In addition, as in neurologic patients with cerebellar lesions, dark adaptation has the effect of normalizing tracking patterns of schizophrenic inpatients (Pivik et al, 1986). These data argue for brainstem involvement in the CNS dysfunction responsible for PEM deficits.

There is evidence that frontal lobe dysfunction may play a role in hyperactivity (see Mattes, 1980 for a review). The two previous reports of oculomotor function in HK children are not in agreement regarding the physiological basis of the dysfunction causing pursuit eye movement aberrations. Shapira et al (1980), in attributing nystagmoid interruptions to cerebellar dysfunction, implicate brainstem structures, while Bala et al (1981) are nonspecific as to the location of the dysfunction. Maiste's (1986) investigation of the vestibulo-ocular interaction which indicated significantly reduced fixation suppression in HK children, points to cerebellar dysfunction in hyperactivity. In the present investigation, subcortical involvement is suggested since

significant reductions in indexes of PEM performance were found only in association with darkness, i.e., where cerebellar influences are removed from oculomotor nuclei. In addition, where normalization of tracking errors does occur brainstem structures are implicated, i.e., (i) for those HK subjects who showed reversal saccades, numbers of these aberrations were normalized in darkness; and (ii) VAs were normalized by a combination of darkness and stimulant medication (thought to increase arousal by increasing brainstem reticular activity). These data strongly suggest subcortical structures play a major role in PEM dysfunction of HK children.

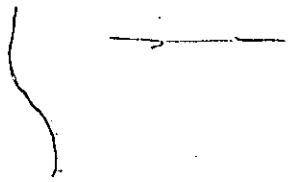
CONCLUSIONS

In the present investigation nonmedicated HK children were shown to exhibit disordered PEM, as indexed by significantly increased VA scores and numbers of positive saccades. In addition, more HK than C subjects evidenced reversal saccadic interruptions, and for those subjects who did show reversals, this type of aberration was present in larger numbers for HK relative to C subjects. These oculomotor disturbances may contribute to the visual information processing difficulties that HK children exhibit. Variations in level of attention are discounted as the primary basis of PEM aberrations since significant differences in

tracking performance between control and HK children persist after levels of both voluntary (as assessed by the button-press task) and nonvoluntary (as effected by reduced distraction in dark-adapted condition) attentiveness are enhanced. Cerebellar dysfunction cannot be discounted as a factor contributing to aberrant PEM in HK children since dark adaptation did significantly reduce (but did not normalize) VAs and positive saccades for HK children and, for those HK subjects who did show reduce reversal saccades, these aberrations were reduced to control group levels in the dark adapted condition. However, failure to normalize all tracking aberrations suggests that neither attention nor cerebellar dysfunction is the sole cause of PEMs deficits of HK children and that other deficits exist which compromise PEM performance.

Administration of stimulant medication was associated with only slight, nonsignificant reductions in indexes of tracking dysfunction in the light-adapted condition. However, medication-associated reductions in PEM aberrations were correlated with the degree to which behavioural symptoms were reduced by these medications. Furthermore, a positive relationship between medication-related increases in saccadic velocity and daily dose of medication suggest that medication-associated reductions in PEM dysfunction are related to the arousing or activating effect of these

medications. Medication and dark adaptation interact to effect a normalization of VA scores in HK children. These data suggest that medication has the effect of promoting normal interactions between PEM control structures and cerebellar structures. This medication-related effect may be mediated through arousal mechanisms or by direct action of medication on PEM control mechanisms or cerebellar structures or both.



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APPENDIX A
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VELOCITY ARREST SCORES

	C	HKnm	HKm
light M	18.15	30.05	27.32
SD	19.77	29.17	18.94
dark M	7.30	15.55	10.00
SD	7.46	14.27	12.64

ANALYSIS OF VARIANCE TABLES

C vs HKnm (2 groups, 1 repeated factor -- light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
GROUP	19674.875	1	2030.112	3.92	.0550
LIGHT	3213.112	1	3213.112	13.82	.0006
L x G	66.612	1	66.612	0.29	.5965
ERROR	8837.775	38	232.573		

C vs HKm (2 groups, 1 repeated factor -- light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
GROUP	685.933	1	685.933	2.05	.1603
LIGHT	3864.852	1	3864.852	26.25	.0001
L x G	203.672	1	203.672	1.38	.2470
ERROR	5447.327	38	147.225		

HKnm vs HKm (2 repeated factors -- medication, light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
MEDS	29.066	1	29.066	0.21	.6486
LIGHT	4320.118	1	4320.118	20.54	.0003
M x L	95.066	1	95.066	0.44	.5170
ERROR	3916.184	19	217.566		

TRANSFORMED VELOCITY ARREST SCORES

	C	HKnm	HKm
light M	00.94	01.28	01.27
SD	00.64	00.45	00.44
dark M	00.66	00.99	00.79
SD	00.47	00.47	00.44

ANALYSIS OF VARIANCE TABLES

C vs HKnm (2 groups, 1 repeated factor -- light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
GROUP	2.232	1	2.232	6.15	.0177
LIGHT	1.607	1	1.607	9.74	.0034
L x G	0.001	1	0.001	0.01	.9264
ERROR	6.271	38	0.165		

C vs HKm (2 groups, 1 repeated factor -- light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
GROUP	1.118	1	1.118	3.08	.0874
LIGHT	2.958	1	2.958	20.03	.0001
L x G	0.256	1	0.256	1.73	.1963
ERROR	5.465	38	0.147		

HKnm vs HKm (2 repeated factors -- medication, light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
MEDS	0.059	1	0.059	0.37	.5524
LIGHT	3.021	1	3.021	29.81	.0001
M x L	0.211	1	0.211	1.20	.2869
ERROR	3.153	19	0.175		

ROOT MEAN SQUARE ERROR - RMS

	C	HKn	HKm
light M	11.10	14.25	11.91
SD	5.93	11.74	7.77
dark M	7.79	9.75	9.82
SD	3.45	4.13	8.75

ANALYSIS OF VARIANCE TABLES

C vs HKn (2 groups, 1 repeated factor -- light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
GROUP	130.892	1	130.892	1.95	.1712
LIGHT	304.395	1	304.395	9.02	.0047
L x G	7.146	1	7.146	0.21	.6480
ERROR	1282.214	38	33.742		

C vs HKm (2 groups, 1 repeated factor -- light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
GROUP	40.328	1	40.328	0.66	.4218
LIGHT	145.260	1	145.26	4.70	.0365
L x G	7.405	1	7.405	0.24	.6272
ERROR	1174.000	38	30.895		

HKnm vs HKm (2 repeated factors -- medication, light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
MEDS	25.912	1	25.912	0.97	.3374
LIGHT	216.844	1	216.844	5.35	.0322
M x L	29.101	1	29.101	0.52	.4798
ERROR	1063.975	19	55.999		

NUMBER OF SUPERIMPOSED SACCADES

	C	HKnm	HKm
light M	16.60	36.00	31.15
SD	16.81	41.30	31.56
dark M	9.00	23.85	16.73
SD	9.16	23.19	24.37

ANALYSIS OF VARIANCE TABLES

C vs HKnm (2 groups, 1 repeated factor -- light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
GROUP	5865.313	1	5865.313	6.18	.0174
LIGHT	1950.313	1	1950.313	5.47	.0247
L x G	103.513	1	130.513	0.29	.5931
ERROR	13541.675	38	365.360		

C vs HKm (2 groups, 1 repeated factor -- light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
GROUP	4500.000	1	4500.000	5.49	.0244
LIGHT	1022.450	1	1022.450	6.09	.0182
L x G	4.05	1	4.050	0.02	.8774
ERROR	6377.500	38	167.829		

HKnm vs HKm (2 repeated factors -- medication, light condition)

SOURCE	SUM OF SQUARES	DF	MEAN SQUARE	F	p
MEDS	90.313	1	90.313	0.39	.5395
LIGHT	1776.613	1	1776.613	3.21	.0890
M x L	145.513	1	145.513	0.52	.4795
ERROR	5423.738	19	285.460		

CORRELATIONS OF BEHAVIORAL SYMPTOMATOLOGY INDEXES AND MEDICATION DOSAGE WITH MEDICATION-RELATED CHANGES IN INDEXES OF PURSUIT TRACKING PERFORMANCE OF HYPERACTIVE CHILDREN

	NMED CONNORS INDEX	MED CONNORS INDEX	MED RELATED CHANGE IN CONNORS INDEX	MEDS (mg)	MG/KG
LIGHT					
VA	-0.15 p=.28	0.04 p=.44	-0.10 p=.36	-0.31 p=.12	-0.45 p=.06
RMS	-0.06 p=.41	-0.28 p=.15	0.41 p=.06	-0.18 p=.26	-0.25 p=.20
SAC	-0.13 p=.31	-0.26 p=.17	0.31 p=.13	-0.06 p=.41	-0.10 p=.37
DARK					
VA	0.26 P=.16	0.37 P=.08	-0.25 P=.18	0.05 P=.43	0.28 P=.18
RMS	0.15 P=.28	0.36 P=.08	-0.33 P=.12	0.18 P=.26	0.35 P=.12
SAC	0.18 P=.25	0.52 P=.02	-0.43 P=.05	0.07 P=.40	-0.03 P=.46

INTERCORRELATIONS OF PURSUIT EYE MOVEMENT PERFORMANCE MEASURES ACROSS LIGHTING CONDITIONS

CONTROL GROUP

	LIGHT VA	LIGHT RMS	LIGHT SAC	DARK VA	DARK RMS	DARK SAC
LIGHT VA	--	0.71 p=.01	0.70 p=.01	0.43 p=.03	0.29 p=.11	0.22 p=.18
LIGHT RMS		--	0.69 p=.01	0.30 p=.10	0.35 p=.06	0.10 p=.34
LIGHT SAC			--	0.40 p=.04	0.36 p=.06	0.51 p=.01
DARK VA				--	0.36 p=.06	0.42 p=.03
DARK RMS					--	0.18 p=.22
DARK SAC						--

INTERCORRELATIONS OF PURSUIT EYE MOVEMENT PERFORMANCE MEASURES ACROSS LIGHTING CONDITIONS:

HYPERACTIVE GROUP - NONMEDICATED

	LIGHT VA	LIGHT RMS	LIGHT SAC	DARK VA	DARK RMS	DARK SAC
LIGHT VA	--	0.93 p=.01	0.69 p=.01	0.53 p=.01	0.40 p=.04	0.25 p=.15
LIGHT RMS		--	0.83 p=.01	0.66 p=.01	0.54 p=.01	0.24 p=.16
LIGHT SAC			--	0.63 p=.01	0.53 p=.01	0.54 p=.01
DARK VA				--	0.56 p=.01	0.44 p=.03
DARK RMS					--	0.13 p=.29
DARK SAC						--

INTERCORRELATIONS OF PURSUIT EYE MOVEMENT PERFORMANCE MEASURES ACROSS LIGHTING CONDITIONS:

HYPERACTIVE GROUP - MEDICATED

	LIGHT VA	LIGHT RMS	LIGHT SAC	DARK VA	DARK RMS	DARK SAC
LIGHT VA	--	0.60 p=.01	0.79 p=.01	0.74 p=.01	0.40 p=.04	0.40 p=.04
LIGHT RMS		--	0.49 p=.01	0.40 p=.04	0.34 p=.07	0.15 p=.26
LIGHT SAC			--	0.56 p=.01	0.32 p=.08	0.73 p=.01
DARK VA				--	0.84 p=.01	0.62 p=.01
DARK RMS					--	0.58 p=.01
DARK SAC						--

INTERCORRELATIONS OF BEHAVIORAL SYMPOMATOLOGY INDEX, MEDICATION DOSAGE AND MEDICATED CHANGES IN BEHAVIOR INDEX WITHIN THE HYPERACTIVE GROUP

	NMED CONNORS INDEX	MED CONNORS INDEX	MED RELATED CHANGE IN CONNORS INDEX	MEDS (mg)	MG/KG
NMED CONNORS INDEX	--	0.59 p=.02	0.33 p=.13	0.24 p=.22	0.49 p=.06
MED CONNOR INDEX		--	-0.57 p=.02	0.04 p=.46	0.23 p=.26
MED-REL CHNG IN CONNORS INDEX			--	-0.08 p=.41	-0.02 p=.48
MEDS (mg)				--	0.87 p=.01
MG/KG					--

PARTIAL CORRELATIONS OF MEDICATION DOSE WITH
CHANGES IN INDEXES OF PEM PERFORMANCE IN HK CHILDREN
CONTROLLING FOR CONNORS HYPERACTIVITY INDEX SCORES (HKnm)

	MEDICATION (mg)	MG/KG
LIGHT		
VA	-0.42 p=.12	-0.33 p=.18
RMS	-0.17 p=.32	-0.21 p=.28
SAC	0.02 p=.48	0.17 p=.32
DARK		
VA	0.04 p=.45	0.20 p=.29
RMS	0.20 p=.29	0.27 p=.23
SAC	-0.01 p=.49	0.10 p=.40

CORRELATION OF DARKNESS-RELATED CHANGES IN CORNEORETINAL POTENTIAL WITH DARKNESS-RELATED CHANGES IN INDEXES OF PURSUIT EYE MOVEMENT PERFORMANCE

L - D	C (L-D CRP)	HKnm (L-D CRP)	HKm (L-D CRP)
VA	-0.17 p=.24	-0.31 p=.09	-0.21 p=.19
RMS	-0.31 p=.09	-0.14 p=.27	-0.50 p=.02
SAC	-0.44 p=.03	-0.16 p=.25	-0.39 p=.05