# Smooth Pursuit Eye Tracking Dysfunction in Schizophrenia: Subcortical Implications

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**The study of smooth pursuit eye tracking behavior in schizophrenics has occupied a prominent position in the search for biological correlates of this mental illness for nearly 20 years. During this time, impairments in this behavior have been shown to be a most robust finding in these patients. Attempts to further characterize the basis for this dysfunction have emphasized the role of cortical — particularly frontal — processes in both the symptomatology of schizophrenia and the eye tracking disturbance. In the present paper, arguments are made in support of a subcortical contribution to smooth pursuit eye tracking dysfunction in schizophrenia. Supporting data include new observations of cortical EEG variations in association with pursuit tracking disruptions, and a review of recent data indicating visual-vestibular and cerebellar-vestibular influences on the tracking disturbances in these patients. On the basis of such data, it is concluded that it is highly unlikely that a single mechanism or process is exclusively responsible for impaired pursuit tracking in schizophrenics, and that there are data to support both cortical and subcortical contributions to this dysfunction.**

*Key Words:* schizophrenia, smooth pursuit tracking, subcortical influence

**Depuis près de vingt ans, l'étude du comportement de poursuite visuelle uniforme chez les schizophrènes a pris une place prépondérante dans la recherche de corrélats biologiques de cette maladie. En effet, les déficiences dans ce comportement se sont révélées être des plus caractéristiques chez ces patients. Les tentatives de mettre en lumière les bases biologiques de cette déficience pointent vers le rôle des processus corticaux — particulièrement frontaux — à la fois dans la symptomatologie de la schizoprhénie et dans les troubles de poursuite visuelle. Des arguments sont présentés en faveur de l'hypothèse d'une contribution sous-corticale au désordre de la poursuite visuelle uniforme chez le schizophrène. Des données venant appuyer cette hypothèse incluent les observations récentes de variations corticales électroencéphalographiques associées à la perturbation de la poursuite visuelle. Un recensement d'autres données récentes indiquent aussi la présence d'influences vestibulaires visuelles et vestibulaires cerébelleuses dans ce désordre. Sur ces bases, on conclut qu'il est peu probable qu'un seul mécanisme ou processus soit responsable de la détérioration de la poursuite visuelle chez le schizophrène, et que les données supportent une implication à la fois corticale et sous-corticale dans cette disfonction.**

*Mots clés :* schizophrénie, poursuite visuelle uniforme, influence sous-corticale

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Our understanding of schizophrenia has undergone many changes over the years, and these variations have modified the definition of this disorder and accordingly directed and redirected research on this topic. Guided by the defining symptomatology and the belief that a biological disorder is fundamental to the disease, investigators have attempted to characterize schizophrenia in terms of neurobehavioral, autonomic, neurophysiological and neurochemical characteristics (Buchsbaum 1990; Holzman 1987; Meltzer 1987). The focus of these attempts has shifted with new observations and advances in technology, but one driving force behind much of this research has been the desire to determine specific central nervous system (CNS) sites which play a major role in the etiology of this disease. Depending on which aspects of schizophrenic symptomatology one wishes to emphasize, e.g., cognitive, integrative, clinical response to medication or linguistic deficits, data accumulated may be interpreted as indicating either cortical or subcortical abnormalities.

Information regarding site-specific CNS involvement in schizophrenia can also be obtained by determining physiologic or neurochemical abnormalities in these patients and noting the CNS localization of substrates for the systems involved. In this regard, the study of eye movement behavior in this disorder has been particularly appealing since: eye movement behavior can be noninvasively accessed using inexpensive technology making it possible for several laboratories to assess large numbers of patients; mechanisms and CNS sites of oculomotor control have been extensively studied in primates; and eye movement dysfunction in schizophrenia is a very robust finding — with no negative reports appearing in a research literature spanning nearly 20 years. In fact, aberrant smooth pursuit tracking has been treated as a possible biological marker for schizophrenia (Clementz and Sweeney 1990; Szymanski et al 1991). While clearly documenting the presence of this dysfunction in these patients, this literature has been less definitive in either clarifying the meaning or indicating the CNS location of the dysfunction. These difficulties can be traced to several interactive factors, including the nature of the measurements used, the interpretation of results, and the general complexity of both cognitive and physiological processes being studied. These issues have been previously addressed (Gooding et al 1990; Iacono 1988; Levin 1984) and will not be the subject of extensive elaboration in this paper.

The intent of this paper is to draw attention to data and approaches seeking to more clearly characterize levels of CNS involvement in deviant pursuit tracking in schizophrenia. In this regard, it is important to identify the nature of these aberrations since the specific characteristics of the disruptions will provide more direct clues to the origin of the problem. Although these aberrations have been described in the context of smooth pursuit eye movement recordings, the tracking disruptions have been shown to consist primarily of the intrusion of small saccades on the smooth pursuit eye movements (Iacono 1988; Levin 1984). In part, these saccadic eye movements may occur in response to an increased phase lag present in these subjects (Iacono and Koenig 1983) which necessitates more attempted corrective saccades (Iacono 1988). It is important to note, however, that saccadic eye movement latencies and the dynamic characteristics of these eye movements are normal for the vast majority of schizophrenics studied (Levin 1984). These observations suggest, therefore, that the deviant eye tracking in schizophrenia is a complex phenomenon involving both the pursuit and saccadic eye movement systems.

The CNS representation of the pursuit and saccadic systems is quite diffuse (Clementz and Sweeney 1990; Freund et al 1986). Although different aspects of eye movement behavior may be under the control of specific sites, the nature of the aberrations in schizophrenia, together with the integrative nature of eye movement behavior, would imply both cortical and subcortical involvement. Despite such considerations, emphasis on aspects of schizophrenic symptomatology implicating attention and information processing and the role of the frontal lobes in these processes and in features of eye movement control (Levin 1984), have prompted an emphasis on cortical, particularly frontal, localization of the eye tracking dysfunction in schizophrenia. Evidence for these frontal-behavioral associations, although compelling, has not ruled out contributions to aberrant pursuit tracking from other sites, cortical and subcortical. In examining this issue, our laboratory has gathered data pointing to the possibility of subcortical contributions to this dysfunction. These studies began with a "top-down" approach, in which an initial attempt was made to relate the occurrence of eye tracking disruptions to specific changes in cortical electroencephalographic activity. Subsequent investigations examined subcortical aspects of eye movement control, and included studies of vestibular reactivity and assessment of eye tracking behavior under conditions considered to reflect cerebellar influences on eye movements. Before elaborating on these studies, the general methodology used in these investigations will be presented.

## METHODOLOGY

### Subject Selection

In all of the studies to be described, the subject population consisted of non-hospitalized controls recruited from hospital staff and the local population at large, and of psychotic patients recruited from the Department of Psychiatry Inpatient and Outpatient Clinics of the Ottawa General Hospital. These patients either were exhibiting active psychotic symptomatology or had previously displayed psychotic symptomatology but were in remission at the time of recording. Psychiatric diagnoses were based on DSM-III-R (American Psychiatric Association 1987) criteria independently determined by two psychiatrists. In accordance with these criteria, symptoms were considered in remission if active signs of illness were absent. This decision was also based on independent evaluations by two psychiatrists. Informed consent was obtained from all participants.

Control subjects were characterized by an absence of personal or family histories of psychiatric illness, or personal histories of head injury, and all had normal response profiles on the Minnesota Multiphasic Personality Inventory (Green 1980). Control and patient populations were matched for age, and all subjects had 20-20 vision, normally or after correction. For the investigations involving vestibular activation by caloric irrigation, subjects were determined to have bilaterally intact tympanic membranes. Exclusion criteria included a history of organic disease, alcoholism, motor abnormality, or taking of medication known to affect eye movements or vestibular responses (eg., barbiturates, antihistamines, tranquilizers). Patients were receiving antipsychotic or antidepressant medications, but previous investigations have determined that, with rare exceptions (e.g., lithium), therapeutic dosages of such medication does not account for the marked eye tracking dysfunction in these patients (Abel and Hertle 1988; Levy et al 1984).

### Recording Procedures

Eye movements were recorded from Beckman miniature silver-silver chloride electrodes attached to the outer canthus of each eye (horizontal electrooculogram; HEOG: .03 to 100 Hz; 6 dB/octave) and above and below one eye to detect vertical eye movement (VEOG) and blink artifact. Electroencephalographic (EEG) and electromyographic (EMG) activities were also recorded. Recording sites for EEG measures varied across studies, but monopolar occipital activity ($O_z/A_2$) was always recorded to obtain a general measure of level of arousal as indexed by variations in alpha activity. Facial EMG activity was monitored (orbicularis oris muscle) for detection of changes in facial muscle tension and movement artifact.

During eye movement testing, subjects reclined on a cot with their heads elevated 30° and restrained by lateral supports to assume and maintain the proper ventroflexed position for caloric testing. In this position, subjects were 1 m from a light panel, the primary plane of which was parallel to the coronal plane of the subject's head. All eye movement recordings were conducted while the subjects were in this position. The target light panel was a bank of red light-emitting diodes (LEDs: N=128, 1 mm wide, 1.5 mm apart) imbedded in a black background and covered with clear plexiglass. This panel was programmed by a micro-computer to simulate horizontal sinusoidal oscillation of a single target light at 0.45 Hz (2.2 sec.) describing a 20° arc (10° on either side) in the subject's visual field. Close spacing of the LEDs and brief on/off intervals between successive LED illuminations effected the perception of continuous motion of a single oscillating target light. During tracking trials, subjects were instructed to visually track the target light (15-20 oscillations/trial) and to depress a hand-held button whenever the target light was interrupted (off cycle — 200 msec, 4-6 randomly occurring interruptions/trial). A practice trial was undertaken to ensure the instructions were understood.

For procedures involving activation of the vestibular system, a variation of the Fitzgerald-Hallpike technique of caloric irrigation (Fitzgerald and Hallpike 1942) was used. Deviations from that method included: (a) electrooculographic recording of eye movements; and, (b) bilateral irrigation with cool water only. Water cooled and maintained at 30°C by a Grant Instrument circulator was delivered via a double-walled hose from the circulator to the external auditory canal. Each irrigation (250 ml of water) extended over a 30 sec. period. For investigations involving caloric irrigation, spontaneous eye movements with eyes closed were recorded for 30 sec. to permit screening for spontaneous nystagmus. Subjects performed a continuous performance task (substracting serially by 3s) during eyes closed portions of the vestibular testing procedure to maintain attention.

### Analysis of Eye Movement Data

Analogue EOG signal values and tracking signal data were digitized at the rate of 200 samples/sec and analyzed by computer to determine eye tracking or vestibular nystagmus parameters. Pursuit tracking patterns were analyzed for the incidence of discrete deviations (velocity arrests: VAs), as well as for global deviations of tracking from target patterns (root-mean square error:RMS). For both measures, established analytical procedures were used (VA: Holzman et al 1978; Pivik 1979; Shagass et al 1976; RMS: Iacono and Lykken, 1979). A VA was scored when eye velocity slowed to less than 2°/second for greater than 40 msec. Mandatory VAs detected within 200 msec of a blink or associated with EMG artifact were deleted from these analyses. Although this measure consistently discriminates schizophrenics from control subjects (with significantly more VAs in schizophrenia), it has been criticized because it does not correlate well with other objective indices of tracking performance (Iacono 1988) and is considered to be vulnerable to influence by EEG and muscle activity. In the present investigations filtering and artifact removal minimized EEG and EOG influences.

The more global RMS error measure was determined by first correcting for phase lag between the target and eye tracking signals, and then executing a point-by-point determination of percent deviation of eye from target position by subtracting eye tracking values from target signal values and dividing the difference by the target signal value. These deviations were squared and the mean of these squared deviations (or error) determined across the total number of points. The square root of this mean is the RMS error value.

In most cases, group and subject differences across conditions and testing sessions were analyzed using repeated measures analysis of variance (UCLA Medical School BMDP2V Program adjusted using the Greenhouse-Geisser procedure and Bonferroni corrections; Dixon 1983). Indicated post-hoc analyses were conducted using the Newman-Keuls procedure.

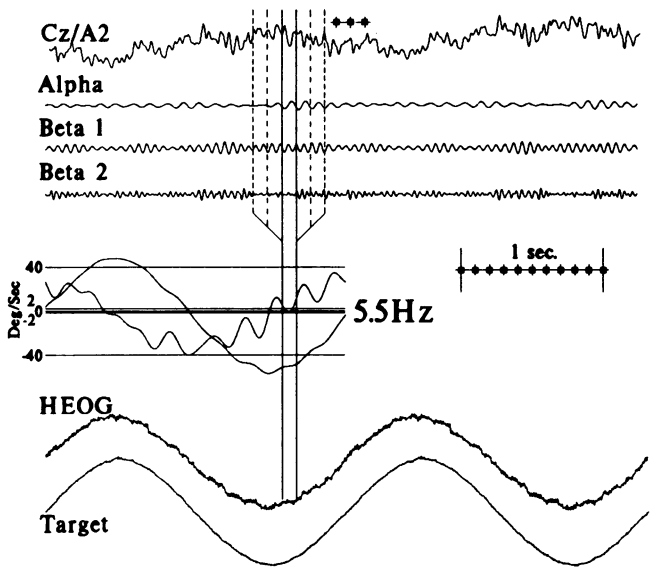


**Fig. 1. Schematic diagram illustrating intervals of EEG activity to be analyzed (Fast Fourier Transform and power spectral analysis) for possible variations in conjunction with eye tracking aberrations (velocity-arrests). The interrupted vertical parallel lines designate 100 msec bins before and after a central bin (continuous parallel lines) capturing the velocity arrest. Upper tracing: midline central EEG; next three tracings: band-pass filtered alpha (8-12 Hz) and beta (1: 15.1-20 Hz and 2: 20.1-50 Hz, respectively) components of the upper central EEG recording; center tracings show the horizontal electrooculogram (HEOG) filtered at 5.5 Hz (smooth tracing) and differentiated for velocity (sinusoidal variations). The last channel depicts target movement.**

## EEG CORRELATES OF SMOOTH PURSUIT TRACKING DYSFUNCTION

It has been previously postulated that smooth pursuit tracking interruptions may index CNS events effecting or reflecting "the phasic interrupting of centering of focus" (Holzman et al 1973). In view of the relationship between brainwave activity and behavior, it might be expected that variations in cortical EEG activity at sites generating or affected by such disruptions would occur in close association with tracking disruptions. Furthermore, it has been demonstrated in other contexts that short-lived endogenously generated activity in, for example, the oculomotor (Miyauchi et al 1987) or auditory (Fazen et al 1985) systems is accompanied by variations in cortical EEG activity. To date, there are no reports in the literature of attempts to determine the co-variation between EEG and ocular dysfunction in schizophrenia. The data to be described were gathered in an investigation designed to determine if: (a) EEG activity from different cortical sites varied in power spectral characteristics in association with phasic interruption of smooth pursuit tracking performance in schizophrenic patients; and, (b) the time relationship of any variations which were present relative to the occurrence of the VA. In this regard, cortical generation of the pursuit tracking disruption would be expected to be signaled by EEG changes prior to onset of the disruption. In this investigation, EEG activity was recorded from frontal, central and occipital midline placements referenced to the right mastoid. The frontal and occipital sites were selected because these areas have been implicated in eye movement control (Daroff and Hoyt 1971; Daroff and Troost 1978; Schiller et al 1979) and attention (Cohen et al 1987; Crowne 1983; Wurtz et al 1982). The central derivation served as a comparison site relatively uninvolved in eye movement control or attentive processes. For these analyses, the computer-detected VA onset and offset times were used as guides for analysis of filtered EEG data (50 Hz low pass, 48 db roll off per octave; digitized at 500 samples/sec/channel). Only VAs isolated from the adjacent VAs by more than 200 msec were selected. EEG data were analyzed for two 100 msec bins immediately preceding VA onset, one 100 msec bin encompassing the VA, and two 100 msec bins immediately following VA offset (Figure 1). The 100 msec bin size was selected to maximize detection of EEG variations in association with discrete eye tracking disruptions and still allow for meaningful EEG band analyses. For each EEG derivation, the 5 data bins were individually subjected to Fast Fourier Transform and power spectral analysis. Spectral data were determined for frequency bands corresponding to alpha (10-12.0 Hz) and beta activities (15.1-50 Hz). Only EEG frequencies $\geq$10 Hz could be examined since bin size (i.e., 100 msec) limited the frequency resolution of analysis. Data presented are from 7 control subjects, 7 actively-ill schizophrenics, and 6 patients with affective disorder.

Results summarized in Figure 2 illustrate the presence of differences in levels of EEG activity across subject groups in association with VAs and reveal relative increases in alpha activity and decreases in beta during and following the VA. The profile of VA-associated EEG variations are similar across groups. These findings demonstrate the presence of EEG changes in association with pursuit tracking disruptions, and further speak to the possible role of these electrographic variations in the generation of eye tracking disruptions. Specifically, increases in alpha activity subsequent to the occurrence of the VA argues against alpha intrusion as a source of artifact for this measure (Iacono and Lykken 1983). Furthermore, relative decreases in beta activity are not consonant with suggestions that VA detections represent increases in muscle artifact, since such artifacts would be associated with increases in beta activity. Finally, the timing of the EEG changes are not what would be expected if the disruptive oculomotor events were triggered cortically — in the latter case EEG changes should be evident prior to the onset and not coincidentally with or following the tracking disruption. At best, however, these

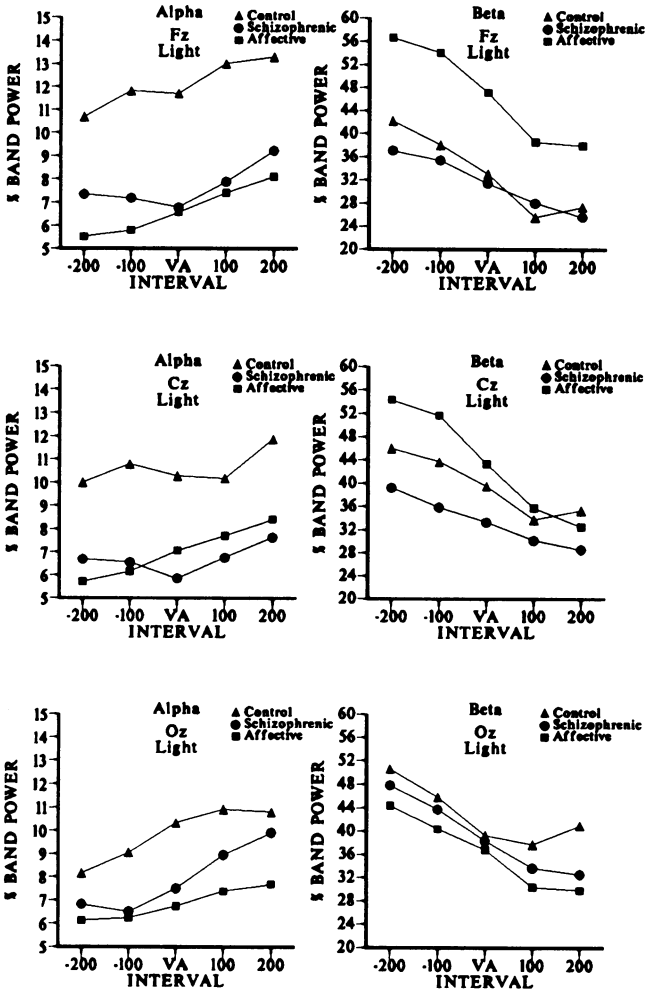


**Fig. 2. Results of VA-associated EEG analyses. The graphs provide data for alpha and beta activity bands across frontal, central and occipital midline sites. See text for discussion.**

data can provide only indirect support for arguments favoring subcortical involvement in the generation of pursuit tracking dysfunction in schizophrenics. A more direct assessment of subcortical involvement in this dysfunction was required, and for this we turned to the vestibular system — a subcortical system involved in eye movement control which can be accessed non-invasively, and for which there is some history of dysfunction in schizophrenia.

## SMOOTH PURSUIT TRACKING: VISUAL-VESTIBULAR AND CEREBELLAR-VESTIBULAR INTERACTIONS

To date we have conducted several studies examining vestibular functioning in schizophrenic and affective disorder patients (Jones and Pivik 1983; 1985; Cooper and Pivik 1991). The results from these studies have indicated that in many respects, vestibular functioning is normal in these patients. This statement is based on the presence of normal maximum slow phase-velocity, normal duration and latency of nystagmus, and normal peak nystagmus frequency. There was one index, however, which did discriminate among subject groups; specifically, unlike control subjects, actively-ill schizophrenic patients were unable to suppress vestibular nystagmus during fixation of a visual target (Figure 3). This failure or impairment of fixation suppression has been replicated both in our laboratory and by others as well (Yee et al 1987). A relationship between failure of fixation suppression and presence of tracking deficits has also been repeatedly demonstrated (Cooper and Pivik 1991; Jones and Pivik 1983). Across these studies, 40-60% of actively-ill patients have shown significantly reduced fixation suppression relative to controls. In the absence of other signs of vestibular pathology in these patients, it is likely that these abnormalities involve dysfunction of interactions between the vestibular and other cortical and/or subcortical oculomotor mechanisms. Among such mechanisms, cerebello-vestibular interactions must be

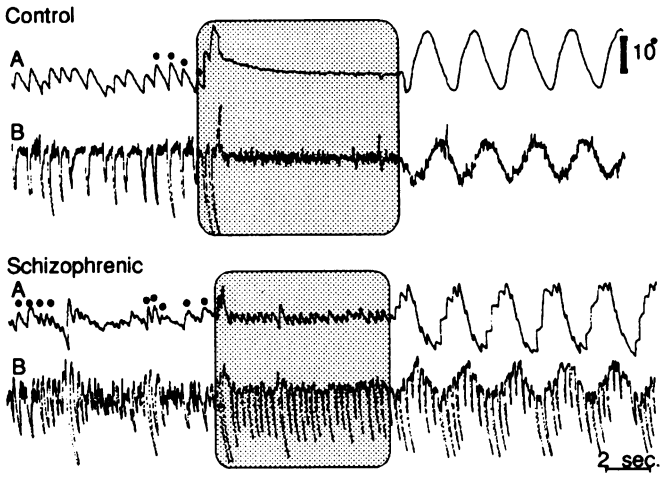


**Fig. 3. Illustrations of the fixation suppression procedure in a control subject (upper tracings) and in an actively-ill schizophrenic (lower tracings). In each pair of tracings the upper channel (A) is the horizontal EOG and shows the presence of nystagmus in the initial portion (examples of individual nystagmus beats designated by filled circles), followed by a period with eyes opened during which the subject fixates a stationary target (indicated by the shaded square), and finally tracking a slowly moving target. In each pair of tracings, the lower channel (B) is a recording of the HEOG (channel A) differentiated for velocity. In the schizophrenic patient's tracings, note the failure to suppress nystagmus during the fixation period with the consequent continued intrusion of nystagmoid eye movements on the pursuit tracking pattern.**

considered prominent because of the acknowledged role of cerebellar mechanisms in smooth pursuit and saccadic eye movement control and in the suppression of vestibular nystagmus (Freund et al 1986; Kato et al 1979; Miles and Lisberger 1981; Zee 1984) and suggestive evidence of cerebellar dysfunction in psychosis (Heath et al 1982; Snider 1982). In this regard, parallels between oculomotor behavior in cerebellar patients and actively-ill schizophrenics, namely the presence of eye tracking dysfunction and impaired fixation suppression, are worthwhile underscoring. Furthermore, impaired pursuit tracking present in cerebellar patients during light-adapted testing conditions is effectively normalized when these subjects are tested under darkened conditions. This effect had been attributed to the removal of the cerebellar influence, which is inactivated in the dark where optimal visual fixation is precluded (Guedry et al 1979; Hood and Waniewski 1984). We investigated the effects of lighting conditions on pursuit tracking in schizophrenic patients and demonstrated not only that eye tracking disruptions in these patients are diminished when recordings are conducted in dark-adapting conditions (Figure 4), but that in the dark-adapting conditions, data from schizophrenic patients and those of normal controls are no longer statistically significantly different (Cooper and Pivik 1991; Pivik et al 1987, 1988; Figure 5). Consideration was made of other factors which could conceivably influence tracking performance in the dark-testing condition (e.g., decreased distractors, or variations in the corneo-retinal potential), but evidence did not support either of these possibilities as primary contributors to the observed effect (Cooper and Pivik 1991; Pivik et al 1988).

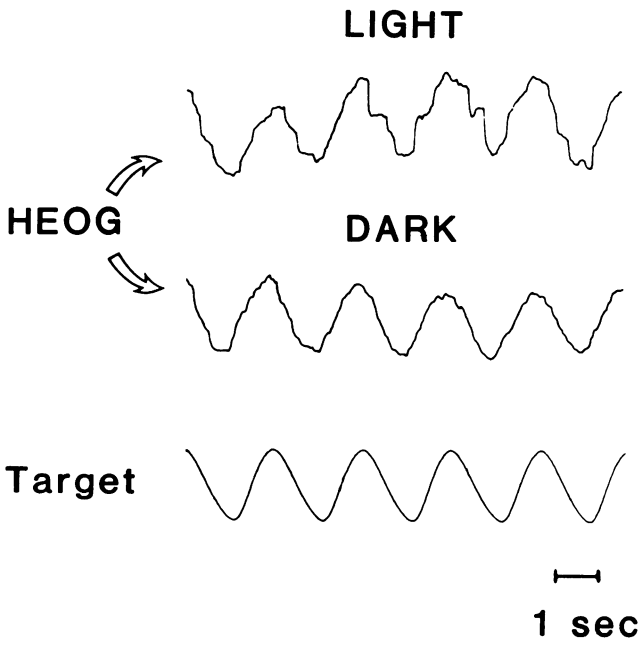


**Fig. 4. Comparisons of pursuit tracking patterns in actively-ill schizophrenics when recorded in the light and dark. Note the marked improvement in tracking accuracy in the dark condition.**

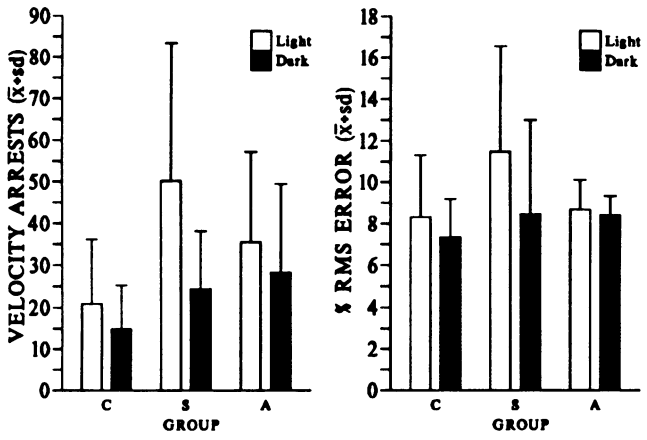


**Fig. 5. Group comparisons of tracking performance in light and dark testing conditions. Illustrated are results for velocity arrest and RMS error measures across control (C: n = 17), actively-ill schizophrenic (S: n = 16) and affective disorder (A: n = 11) subjects. During dark testing, improvement in tracking performance is evidenced in all groups for both measures.**

## CONCLUSIONS

As established in the introduction to this paper, the control of eye movements is an extremely complex process involving both cortical and subcortical mechanisms. Equally complex, and to a great extent less fully explored, are the ways in which schizophrenic symptomatology interacts with oculomotor control. In a review paper which argued strongly for the localization of smooth pursuit tracking disturbances in schizophrenics in the frontal cortex, the statement was made that "there is no evidence that schizophrenic patients, individually or as a group, have ocular-motor or visual deficits which implicate the motor or premotor [brainstem] systems of eye movement control" (Levin 1984). Data presented in this brief overview relating to the presence and timing of eye tracking disruption-related changes in EEG, as well as abnormal visual-vestibular interactions and the implications of cerebellar influences on oculomotor control in these patients, suggest that this statement requires revision. In view of the complexities of the systems and behaviors involved, it seems highly unlikely that a single mechanism or process would be exclusively responsible for the impaired pursuit tracking in schizophrenics. Accordingly, the notion that both cortical and subcortical processes contribute to pursuit tracking dysfunction in this population should be more seriously considered.

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