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Dark Condition Normalization of Smooth Pursuit Tracking: Evidence of Cerebellar Dysfunction in Psychosis

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Summary. Smooth pursuit tracking performance was evaluated in psychotic ($n = 20$) and normal control subjects ($n = 20$) during light and dark testing conditions using computer-based analyses of electrographically recorded tracking patterns. Previously reported impaired tracking in psychotics tested under light conditions was reaffirmed. However, the tracking patterns of patients during the dark condition not only resembled those of controls under similar conditions, but were no longer significantly different from controls' light condition performance. Among several possible bases for these results which are considered, the involvement of cerebellar dysfunction in these patients is emphasized.

Key words: Psychosis – Smooth pursuit eye movement – Light-dark conditions

Introduction

The association of aberrant smooth pursuit tracking with psychoses of varying etiologies has been repeatedly demonstrated (Holzman and Levy 1977; Holzman et al. 1973, 1978; Iacono et al. 1981; Jones and Pivik 1983; Pivik 1979; Shagass et al. 1974), but despite the increased attention to it in the psychiatric literature the basis for the dysfunction remains obscure. There is general agreement, however, that this deviant tracking cannot be attributed to either voluntary inattention (Holzman et al. 1974, 1975; Iacono and Lykken 1979; Pivik 1979; Shagass et al. 1974) or – except for patients receiving lithium carbonate (Levy et al. 1985) – to the effects of antidepressant or antipsychotic medications (Holzman et al. 1974, 1975; Jones and Pivik 1985; Pivik 1979; Shagass et al. 1974). Furthermore, reports (Jones and Pivik 1983; Levin et al. 1981; Levy et al. 1978, 1983; Mather and Putchat 1983) indicating that eye movements other than smooth pursuit are not impaired in psychiatric patients have been interpreted as indicating that the oculomotor deficits are restricted to smooth pursuit eye movements and that these deficits involve cortical and not brainstem eye movement control mechanisms (Levin 1984). However, more extensive analyses of eye movements following activation of the vestibular system in psychiatric patients have revealed differences which not only question the normality of vestibular and related brainstem mechanisms of eye movement control in these patients, but also have implications for the basis of smooth pursuit dysfunction prevalent among psychotics. These studies (Cooper 1987; Jones and Pivik 1983, 1985) examined vestibular reactivity and visual-vestibular interactions in combination with smooth pursuit tracking performance in groups of actively psychotic (primarily schizophrenic) patients, schizophrenic outpatients, and normal control subjects following vestibular activation by caloric irrigation. The results corroborated previous reports (Levy et al. 1978, 1983) of normal velocity and bilateral symmetry of the slow phase nystagmus component, but also noted vestibular response abnormalities in the form of dysrhythmic nystagmus, slower fast component velocity, and reduced suppression of vestibular nystagmus by optic fixation in patients with active symptomatology. Moreover, the relative inability of actively ill patients to suppress nystagmus during vi-

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sual fixation, a finding recently independently corroborated by Yee et al. (1987), was related to a higher incidence of disordered smooth pursuit tracking both before and following vestibular activation (Cooper 1987; Jones and Pivik 1985). These data, and studies in neurological patients indicating an association between impaired pursuit tracking and failure of fixation suppression (Chambers and Gresty 1983; Halmagyi and Gresty 1979; Sato et al. 1980), suggest that a disorder in the modulation of vestibuloocular reflexes – more specifically an impairment of the visual suppression mechanism (Sakata and Umeda 1976) – may be integral to abnormal pursuit tracking.

Recent investigations have indicated that cerebellar structures play a significant role in the suppression of caloric nystagmus by ocular fixation (Alpert 1974; Baloh et al. 1979, 1981; Ito et al. 1973; Kato et al. 1982) and that this cerebellar influence is largely inactive in darkness (Hood and Korres 1979; Hood and Waniewski 1984) as a result of either the absence of fixation or a reduction in peripheral visual feedback. Of relevance as well are indications of cerebellar damage in schizophrenic patients from postmortem and computed tomography studies (Dewan et al. 1983; Weinberger et al. 1979, 1980). If a dysfunction in cerebellar mechanisms does exist in psychotics which results in disinhibition of eye movements during fixation, then such a dysfunction could contribute significantly to the disordered tracking performance so notable in these patients. Furthermore, if, as suggested by previous studies (Jones and Pivik 1985), the cerebellar deficiency requires light conditions to become manifest, then presumably the interference with pursuit tracking would be absent or greatly attenuated under dark conditions where the cerebellar influence on eye movement control is reduced. There has been one report of eye tracking performance of schizophrenics tested under illuminated and darkened conditions (Mather and Putchat 1983). In that study it was observed that patients exhibited more saccadic eye movements than controls during tracking in both conditions. However, the purpose of the dark condition in this investigation was not indicated, and it was not specified how long subjects remained in the dark. Furthermore, the velocity of the target light (0.75 Hz) would have required eye movement velocity of 48°/s for accurate tracking, a rate exceeding or approximating the upper limit for the pursuit system (Rashbass 1961). Increased saccadic intrusions expected at this rate could overshadow any effect accruing from the lighting condition. The present investigation controlled for these potentially confounding variables and was conducted to determine if disordered smooth pursuit tracking evident in patients with active psychotic symptomatology when tested under lighted conditions would normalize when subjects were recorded in darkness.

Subjects

In total 20 psychiatric inpatients and 20 normal control subjects were studied. The patient population was recruited from the Department of Psychiatry Inpatient Clinic of the Ottawa General Hospital. Controls were recruited from the hospital staff and local population. Diagnoses of psychosis and the presence of psychotic symptomatology were based on two independent psychiatric evaluations according to DSM-III criteria (American Psychiatric Association 1980), hospital diagnosis, information from patient history charts, and psychological testing. The patient population consisted of subjects with diagnoses of schizophrenia, paranoid type ($n = 12$), schizoaffective disorder ($n = 1$), schizophrenia, undifferentiated type ($n = 1$), atypical psychosis ($n = 2$), and major affective disorders with psychotic features ($n = 4$) (major depression, $n = 2$; bipolar disorder, $n = 2$). This group included 3 patients with no previous history of psychosis, 6 with acute onset, and 14 with chronic conditions. None of the patients had been hospitalized for their present psychotic episode longer than 6 months. All patients were receiving medication (phenothiazines, butyrophenones, or neuroleptics) and none had overt signs of organicity or obvious motor (e.g., dyskinetic) abnormalities. Control subjects were screened to eliminate those with personal or family histories of psychosis and the present or recent use of medication known to affect eye movements.

All subjects met the following criteria for inclusion in the study: 20–60 years of age (group mean ages plus SD: patients = 30.15 ± 11.02 years; controls = 23.45 ± 3.8 years; $t(38) = 2.57$, $P < 0.05$), minimum IQ of 90 (Weschler Adult Intelligence Scale), absence of a history of alcoholism, 20/20 vision (normally or after correction), and no known ocular or oculomotor pathology. Patients had normal EEGs as determined by examination of their neurological records. Each participant signed a form of informed consent.

Procedure

Silver-silver chloride electrodes were attached near the outer canthus of each eye for recording the horizontal electrooculogram (EOG; Princeton Applied Research Model 13 preamplifier; 0.03–300 Hz). Vertical eye movements and blink artifact were monitored from similar electrodes placed above and below the right eye. Electrodes were also attached for re-

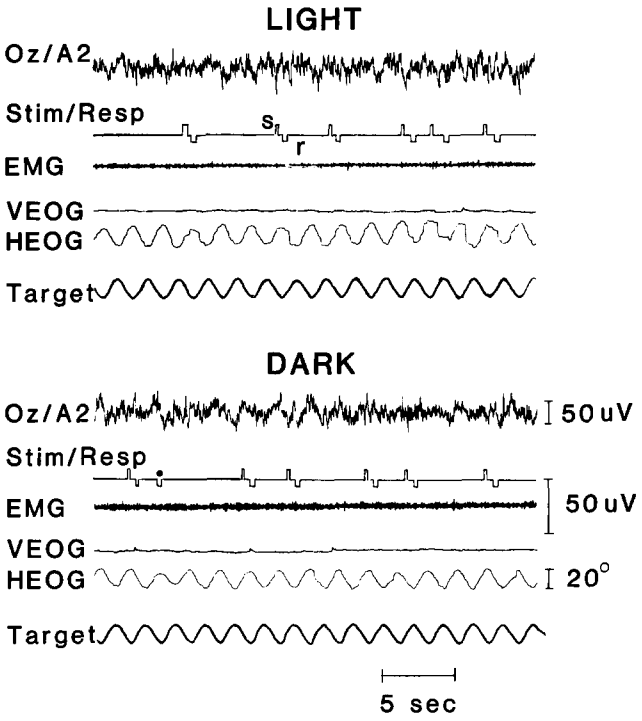

Fig. 1. Electrographic tracings of pursuit tracking and associated measures during light and dark condition testing taken from a paranoid schizophrenic subject illustrating more regular and accurate tracking during the dark. Channel designations: Oz/A2: occipital EEG; Stim/Resp: button-press response channel with indications of target light interruptions (*upward deflections:* s) and subject responses (*downward deflections:* r); EMG: facial electromyogram; VEOG: vertical electrooculogram; HEOG: horizontal electrooculogram; Target: target light excursions. The *filled circle* above the subjects' response (Resp channel, dark condition) indicates a response error

cording EEG (monopolar occipital EEG, Oz/A2; 0.3–35 Hz) and facial electromyographic (orbicularis oris EMG) activities. These activities were monitored to detect variations in level or arousal (EEG), facial muscle tension, or head movement artifact associated with eye movements. Following the attachment of electrodes the subject was positioned 1 m from a light panel with his head stabilized by supports. The light panel consisted of a bank of red light-emitting diodes (LEDs; $n = 128$, 1 mm wide, spaced 1.5 mm apart) embedded in a black background and covered with clear plexiglass. A microcomputer controlling target presentation was programmed to simulate horizontal sinusoidal oscillation of a single target light at 0.45 Hz (2.2 s periods) subtending a 20° arc (10° on either side of midline) through the subject's visual field. Only 1 LED was illuminated at any given time and the duration of illumination (i.e., 5.5 to 30 ms) depended upon the position of the LED in the sinusoid. The time between consecutive LED illuminations was 10 μs. With 128 LEDs extending across a 20° visual angle, each LED subtended 0.16° visual angle. The combined effect of close spacing of the LEDs, brief on/off interval between successive LED illuminations, and brief duration of individual LED illumination effected the perception of continuous motion of a single oscillating target light. Each tracking trial consisted of 15–20 oscillations. Subjects were instructed to visually track the target light, to avoid extraneous head or eye movements, and to depress a hand-held button whenever the target light was interrupted (off cycle 200 ms). This technique has been shown to increase attention to the target and reduce tracking errors (Baloh et al. 1981; Pivik 1979). Within each trial, 4–6 such target light interruptions occurred randomly. A practice trial was given to ensure that the instructions were understood. Tracking trials were recorded under both light and dark (light-tight room) conditions and order of conditions was randomized across subjects. Light and dark exposure periods preceding tracking trials were at least 10 min in duration. Electrophysiological data, target movements, target interruptions, and response data were recorded polygraphically on paper as well as on magnetic tape for later computer analysis (Fig. 1).

To exclude errors associated with initial task orientation, the first three cycles of each trial were excluded from analysis. Also, tracking disruptions related to blinks (deviations in vertical EOG recordings of >1 mm (50 μV) lasting 200–500 ms), eye closure, head movement, or increased facial muscle tension (as determined by the presence of phasic activation of the EMG channel or by direct observation) were excluded from analysis. Artifact-free tracking recordings were then evaluated for pursuit accuracy using two measures of deviation, i.e., the incidence of velocity arrests (VAs, discrete deviations of the eyes from the tracking target) and the general correspondence of tracking and target patterns (root-mean-square error deviations: RMS). Both measures have been previously used in eye tracking analyses, and although they covary closely, they may not reflect the same processes (Iacono and Lykken 1981; Pivik et al. 1985). For determining VAs, tape-recorded horizontal EOG data were filtered (Krohn-Hite Model 3343 filter 5.5 Hz low pass; attenuated 3 dB at cut-off; 48 dB roll off/octave), amplified, and differentiated (Grass 7P21-A differentiator) to obtain the first derivative (velocity) of the sinusoidal tracking pattern. Differentiator output was calibrated at a sensitivity of 20°/s per cm. This information was input to a PDP11/34 minicomputer, digitized at a rate of 200 samples/s per channel, and stored on hard disk. The velocity channels were computer scored for VAs according to established criteria (Pivik 1979; Shagass et al. 1974). Reliability between computerized and noncomputer-

ized scoring of VAs on an independent set of data (400 half-wave velocity tracings from 10 subjects, 5 patients and 5 controls) was previously determined and found to be acceptable (93%). For RMS analyses, tape-recorded horizontal EOG and target movement channels were filtered (10 Hz low pass; attenuated 3 dB at cut-off; 48 dB roll off/octave) and input to the computer for digitization at a sampling rate of 600 samples/s per channel. Following correction for phase lag between target and pursuit tracking patterns, a global estimate of position error between target and pursuit tracking patterns was calculated for each oscillation and an average RMS error score for each trial was determined. The EEG activity associated with tracking patterns was quantified. These data were then coded for subject and light/dark condition and scored for the number of resets. Data were analyzed using correlational procedures and repeated measures analysis of variance [2 groups, with 1 repeated factor (lighting condition)] with post hoc Newman Keuls tests performed where appropriate.

Results

Subjects generally complied with task instructions and a database was established consisting of 10 artifact-free oscillations/subject per condition. Patients failed more than controls to signal detection of target light interruptions under both light and dark testing conditions (average response failures: light, patients: 1.2%; controls: 0.1%; dark, patients: 0.7%; controls: 0.5%), but attention to the target as determined by this measure was good for both groups.

Analyses for both VA and RMS measures revealed significant effects for group and light-dark conditions (Fig. 2), but interaction effects were present only for the VA measure. For both measures under the light exposure condition, patients' tracking performance was significantly impaired relative to that of controls (VA: $F(1.38) = 16.67$, $P < 0.0002$; RMS: $F(1.38) = 5.96$, $P < 0.02$), replicating, once again, the finding of pursuit tracking dysfunction in psychotics. The light-dark condition comparisons revealed that subjects' tracking performance was superior when they were tested in the dark ($F(1.38) = 12.45$, $P < 0.002$). This relative improvement in performance, present in both subject groups and evident across tracking measures was, however, most impressive for patients and most strikingly reflected in VA scores. For example, relative to tracking performance in the light condition, control subjects' VA and RMS tracking scores improved in the dark by 37% and 20%, respectively. Similar comparisons for patients revealed decreases in tracking errors of 60% and 31% for VA and RMS

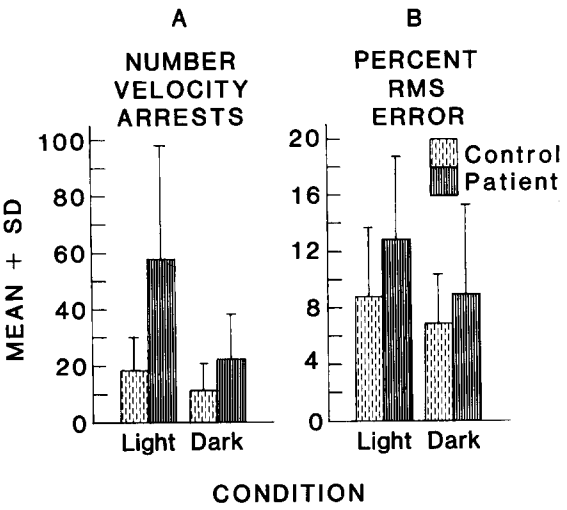

Fig. 2. Variations in velocity arrest and root-mean-square (RMS) indexes as a function of lighting condition and group

measures.[1] The parallel relationship between VA and RMS measures of tracking accuracy was reflected in significant correlations between these measures across groups in both light (controls, $r = 0.56$, $P < 0.005$; patients, $r = 0.91$, $P < 0.001$) and dark (controls, $r = 0.61$, $P < 0.002$; patients, $r = 0.66$, $P < 0.001$) conditions. The light to dark condition improvement in tracking performance was generally accompanied by marked reductions in performance variability as well, i.e., variability in VA scores was reduced 14% and 62% for controls and patient groups, respectively, and RMS variability was decreased by 42% for controls, but only changed slightly (increased 4%) for patients.

Within group comparisons of tracking performance across lighting conditions were of borderline significance for controls (VA: $F(1.19) = 2.95$, $P < 0.10$; RMS: $F(1.19) = 3.02$, $P < 0.10$), but were highly significant for patients [VA: $F(1.19) = 24.98$, $P <$

[1] Although the serial illumination of LED target lights effected the perception of continuous motion of a single target light, the unlighted LEDs constituted a potentially distracting set of stimuli during the light condition. To test this possibility 10 additional actively psychotic patients with diagnoses of schizophrenia ($n = 5$) and major affective disorder ($n = 5$) were recorded once using the clear plexiglass covering over the light panel, i.e., as described in the procedure section, and a second time using a translucent cover. The latter condition minimized the visibility of nonlighted LEDs, thereby decreasing the influence of these lights as potential distractors.

Comparisons of error scores for transparent [$\bar{x} = 36.60$ (VA); $\bar{x} = 8.89$ (RMS)] and translucent [$\bar{x} = 41.60$ (VA); $\bar{x} = 10.76$ (RMS)] trials were not significantly different (paired t-tests), thereby providing no support for an explanation of light-dark differences in patients' tracking scores resulting from enhanced distractibility for these subjects by the light stimulus panel.

0.0001; RMS: $F(1.19) = 6.89$, $P < 0.002$). Although patients continued to make more errors than controls while tracking under the dark condition, group differences, either for comparisons of tracking performance in the dark or patients' dark condition performance compared with control performance under the light condition, were no longer statistically significant.

Light-dark comparisons of quantified EEG activity indicated a slight (6%), nonsignificant increase in alpha activity during the dark condition for control subjects and, for patients, a change of similar degree (7%) which was significant ($F(1.19) = 5.61$, $P < 0.03$) but opposite in direction (decreased during the dark). Except for the positive correlation for control subjects between VA and alpha activity measures in the dark condition, alpha activity was not associated to a statistically significant degree with VA (light: controls, $r = 0.23$, NS; patients, $r = -0.06$, NS; dark: controls, $r = 0.42$, $P < 0.03$; patients, $r = -0.18$, NS) or RMS (light: controls, $r = 0.27$, NS; patients, $r = -0.07$, NS; dark: controls, $r = -0.03$, NS; patients, $r = -0.12$, NS) measures for either group.

The corneoretinal potential (CRP) is reported to decrease in the dark (Kris 1958; Yarbus 1967) and this reduction could conceivably affect tracking errors and, accordingly, contribute to the improved tracking associated with testing in the dark. To assess this possibility, analyses were conducted to determine how variations in this potential across conditions were related to associated changes in tracking performance. For these analyses, light-dark condition differences in the CRP, as measured by the amplitude of the 20° tracking eye movement patterns, were determined for each subject and then correlated with the between condition tracking difference scores for both VA and RMS indexes. The CRP analyses revealed decided between group differences and within group variability for this measure across conditions. As a group, control subjects demonstrated the expected CRP reduction during the dark condition (mean reduction relative to light adapted condition = 65.5 μV), and the CRP data for 77% of these subjects followed this pattern. However, 66% of the patient population exhibited CRP increases during the dark, resulting in a mean relative CRP increase of 37.9 μV during this condition. When the light-dark differences in the CRP and tracking measures were correlated, instead of a positive relationship between these variables, which would be expected if tracking errors had been eliminated because of a reduction in the CRP, negative correlations were obtained for both control (VA: −0.43, NS; RMS: −0.31, NS) and patient (VA: −0.28, NS; RMS: −0.46, NS) groups.

There is generally a reduction in tracking accuracy as a function of age (Holzman et al. 1974; Sharpe and Sylvester 1978) and since patients were older than control subjects in this investigation (6.7 years, on average) it was important to determine if group performance differences might be age-related. However, age did not correlate significantly with tracking performance regardless of measure, group, or light-dark condition.

Discussion

The observation that a functional manipulation may statistically normalize the smooth pursuit tracking dysfunction in patients showing active psychotic symptomatology is unprecedented. Previous investigations have reported reductions in tracking errors in both patients and controls following manipulations designed to enhance task-related attention (Cegalis and Sweeney 1979; Holzman et al. 1976; Levin et al. 1981; Shagass et al. 1976), but none effected reductions which eliminated patient-control differences in tracking performance, i.e., significant residual impairment of pursuit persisted in patients. A normalizing effect on tracking errors while having subjects silently read numbers affixed to the tracking target was reported by Holzman et al. (1976), but this effect was restricted to tracking errors associated with large saccadic intrusions. Furthermore, those results were based on analyses of the tracking performance of a mixed population of schizophrenic patients and their first-order relatives, in which patient-nonpatient results were not differentiated and comparisons were not made with data from normal controls. The normalization of patient tracking performance observed in the present investigation is notable because of the unequivocal nature of the effect and because it occurs in a clinical population in which pursuit impairment is common enough to have been considered as a putative biological marker (Holzman et al. 1984; Iacono 1985). The finding is all the more impressive because smooth pursuit tracking behavior is notoriously vulnerable to disruption, to the extent that an integral part of investigations examining this behavior consists of strategies to control experimental sources of tracking error.

In attempting to account for the present results, it is logical to look first at the possible contribution of these potentially confounding influences from the dark condition results. Such influences fall into two general categories, namely those which may be identified as artifact which, where possible, are to be excluded (e.g., movement, blinking, EEG interferences), and those which may be considered state or condition variables (level of arousal, condition-related variations in the CRP, attention) which, although not

completely subject to experimental control, should be evaluated and their impact assessed. Regarding the first category, the requirement that the database consists of an equal number of artifact-free oscillations/subject per condition excludes differences based on variations in the incidence of blinking or movement during tracking. EEG activity, particularly in the 8–12 Hz (alpha) band, has been suggested as a source of interference during tracking. Iacono and Lykken (1981) based their suggestions on observed covariations between a spiking EOG pattern and increased alpha activity (as determined by power spectral analysis). However, these findings may not generalize to the present investigation since the noted EEG-EOG covariation was observed only in normal control subjects under nontracking conditions. In the present investigation, activity in the alpha band was excluded by filtering activity above 5.5 Hz from horizontal EOG recordings prior to VA analyses. However, this activity was not completely filtered from recordings analyzed for the RMS index, and yet the RMS and VA results closely paralleled one another. Furthermore, if alpha interference did account for elevated tracking error indexes in patients, then such activity should markedly decrease during the dark condition. Patients did exhibit a slight (7%), but statistically significant, decrease in alpha activity in this condition, but alpha activity in patients did not correlate significantly with tracking indexes in either light or dark conditions. The above considerations uniformly argue against attributing the observed experimental effects on tracking performance to relative decreases in artifact during the dark testing condition.

Although alpha activity can be excluded as a source of artifactual interference with EOG recordings, this measure can also be considered as a state variable since it may index levels of cortical arousal (Bunnell 1982; Pfurtscheller and Aranibar 1977). Variations in level of arousal can influence tracking performance and might be expected to differ between light and dark conditions. Iacono and Lykken (1979, 1981) suggested that tracking improvement associated with a monitoring task in normal subjects might be due to enhanced arousal, a condition which should be associated with decreased alpha activity. In support of this relationship quantified alpha activity has been shown to decrease, but not to a significant degree, in actively ill psychotic patients under conditions promoting increased attention to a tracking task (Pivik 1979). In the present investigation, the absence of group differences in alpha activity and the slight variations in quantified alpha activity from light to dark conditions suggest that variations in cortical arousal did not contribute significantly either to group differences in the light condition or to the absence of group differences during the dark condition.

A reduction in the CRP commonly occurs during dark adaptation, and this variation could attenuate EOG sensitivity and, consequently, effect an apparent reduction in tracking aberrations. However, the light-dark variations across groups in this measure were negatively associated with changes in tracking performance indexes. This relationship was determined in part by the within and between group variability in extent of CRP reduction during the dark condition. Individual differences in CRP dark adaptation variations have been previously reported for normal subjects (Anderson and Purple 1980) and a recent report has indicated that psychoactive compounds may influence the extent to which the CRP decreases by dark adaptation in psychotic patients (Kaschka et al. 1987). Also, of relevance to the negative light-dark CRP tracking performance index relationships is the fact that five control subjects and two patients did not exhibit reduced tracking errors during the dark condition. These results and considerations do not support an explanation of the dark-related reduction in tracking errors resulting from reduced CRP values.

Attention to the tracking target is critical for pursuit performance and since previous attention-enhancing manipulations have failed to eradicate significant tracking performance differences between groups of actively ill patients and control subjects, the tracking deficit has been attributed to a nonvoluntary attentional disorder. This suggestion fits well with the general consensus of a disturbance of attentional processes in psychiatric disorders, particularly schizophrenia (Hemsley and Zawada 1976; McGhie et al. 1965; Silverman 1964). In the present investigation, monitoring the tracking signal for interruptions and the dark adaptation procedure may both be viewed as attentional manipulations. Perhaps disordered tracking in psychiatric patients derives primarily from a voluntary attentional deficit and previous attention-enhancing procedures simply have not been as effective in restricting attention to the tracking target as the combined effects of darkness and requiring subjects to signal the presence of target light interruptions. It is likely that these manipulations in concert produce a greater cumulative task-related attentiveness than either does alone. However, since responding to target-light interruptions alone results in only moderate improvement in tracking performance (Pivik 1979), it follows that the marked reduction in tracking errors must be attributable largely to other effects associated with tracking in the dark.

In evaluating the potential contributions to tracking performance of the attention-enhancing manipulations used in the present investigation, it is impor-

tant to note that the effects of the two procedures are mediated differently, and this difference may have a direct bearing on the basis for pursuit impairment in these patients. The target light interruption manipulation enhances target salience relative to other background information by varying a feature of the target. In this situation background information, although deemphasized, nevertheless continues to provide peripheral retinal input and remains as a potential distractor. In the dark condition, however, target salience is enhanced by eliminating background distractors. In this case perceptual isolation of the target is effected by markedly reducing activation of peripheral retinal processes and restricting visual stimulation largely to the foveal area. The inactivation of peripheral retinal elements removes an influence thought to normally facilitate target fixation during both smooth pursuit and suppression of vestibular nystagmus by visual fixation (Guedry et al. 1979; Hood and Waniewski 1984). This effect most likely involves cerebellar mechanisms since in the presence of cerebellar lesions peripheral retinal feedback appears ineffective in suppressing nystagmus (Hood and Waniewski 1984). Cerebellar involvement in eye movement control is well-documented (Zee 1984; Zee et al. 1976, 1981) and there are striking parallels between results from studies of neurological patients with cerebellar disease and psychiatric patients which strongly suggest that cerebellar dysfunction may contribute significantly to disordered pursuit in psychiatric patients. These parallels include: (1) the presence of disordered pursuit and failure of fixation suppression in both pupulations (Baloh 1981; Cooper 1987; Jones and Pivik 1983, 1985; Yee et al., 1987; Zangemeister and Mueller-Jensen 1984); (2) the normalization of impaired pursuit when target background information is attenuated by darkness in both psychiatric (data from the present investigation) and cerebellar patients (Hood and Waniewski 1984); and, (3) postmortem and computed tomography findings of cerebellar damage in schizophrenics (Dewan et al. 1983; Weinberger et al. 1979, 1980). Deficiencies in cerebellar control of saccadic eye movements have been suggested as a source of impaired tracking in schizophrenics (Cegalis and Sweeney 1981; Jones and Pivik 1983, 1985), and the present results strongly reinforce this notion. The confluence of results and effects, although arguing forcibly for cerebellar involvement in schizophrenia-associated pursuit impairment, does not exclude the interactive or independent contribution to disordered tracking from other influences. In this regard, the frontal cortex figures prominently because of the known involvement of frontal areas in eye movement control and visual attention (Stuss and Benson 1986) and indications of compromised frontal functioning in schizophrenics (Franzen and Ingar 1975a, 1975b). On the basis of these considerations, Levin (1984) has speculated that frontal lobe dysfunction may be largely responsible for pursuit impairment in schizophrenia.

It has not escaped our attention that patients' tracking accuracy in the dark condition, although markedly improved and statistically comparable to that of controls, nevertheless remains less accurate than that of controls. Further studies are required to verify and characterize this residual error, but it would not be surprising if such error should persist in view of other structural and neurochemical alterations thought to be associated with psychiatric disorders which may affect oculomotor functioning (Stuss and Benson 1986).

The normalization of impaired pursuit during dark condition testing has implications for the nature and basis of attentional deficits in schizophrenia. As previously stated, investigations in which manipulations have been associated with improved tracking in psychotic, primarly schizophrenic, patients have invoked heightened engagement of attention by the manipulation to explain the improved performance. That proportion of tracking error corrected by such procedures which heighten cognitive sensitivity to the target must be under voluntary control and is secondary to inattention. The remaining, more intractable, proportion of tracking error not subject to voluntary control constitutes the primary source of deviant pursuit tracking in psychotics. The ability to affect the occurrence of this class of tracking errors by testing in the dark, a functional manipulation with documented physiological effects on mechanisms of oculomotor control, suggests that disturbances in attention associated with such errors may be secondary to the tracking impairment. In this case, the postulated failure of cerebellar mechanisms to inhibit saccadic eye movements may result in excessive saccadic intrusions during tracking which involuntarily direct attention away from the target, resulting in the "phasic interrupting of centering of focus" which has been suggested as characterizing deviant tracking (Holzman et al. 1974).

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