OCULOMOTOR FUNCTION IN WERNICKE-KORSAKOFF'S SYNDROME: II. SMOOTH PURSUIT EYE MOVEMENTS

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Smooth pursuit eye movements were studied in three patients with alcoholic Korsakoff's syndrome, one with Wernicke's encephalopathy, and an age-matched control. Horizontal smooth pursuit eye movements were abnormal in all patients: peak eye velocity and the ability to sustain smooth eye velocity were reduced. Also, smooth pursuit gain began to decrease at relatively low target velocities (i.e., 8–10°). These data demonstrate a severe disturbance in smooth pursuit function long after the clinically apparent oculomotor abnormalities have passed.

To maintain a clear image of a moving target, the oculomotor system uses both its saccadic and smooth eye movement systems. Saccades correct for positional errors between the fovea and the target, while the smooth pursuit system moves the eye at a velocity which matches that of the target. A simplified view of the smooth pursuit system suggests that the velocity of the image as it moves across the retina, the so called retinal slip, initiates smooth pursuit eye movement (Rashbass, 1961). However, smooth movements can be elicited by small retinal errors and by stabilized retinal images (Yasui & Young, 1975; Pola & Wyatt, 1980; Kommerell & Taumer, 1972; Robinson, 1965). Nevertheless, a moving target appears to be the main stimulus needed to elicit smooth pursuit.

When the speed of the target exceeds that of the eye, the position error between the fovea and the retinal image increases. This positional error, if small, can be corrected by an increase in smooth pursuit velocity (Robinson, 1965; Pola & Wyatt, 1980). However, for large errors, saccades are used to refixate the target on the fovea. Thus,

at high velocities, both saccades and smooth pursuit are used to track a moving object (Rashbass, 1961; Fuchs, 1967). Ultimately at very high velocities (greater than 40°/sec for small amplitudes) tracking is almost exclusively saccadic.

The function of the smooth pursuit system can be severely impaired by disease and by exposure to toxic substances such as alcohol. Reduced smooth pursuit effectiveness has been demonstrated in cases of schizophrenia (Holzman, Proctor, & Hughes, 1973; Holzman, Levy, & Proctor, 1976) and dementia (Hutton, 1980). Acute alcohol administration rapidly reduces smooth tracking ability and the smooth movements are replaced by saccadic tracking (Wilkinson, Kime, & Purnell, 1974; Flom, Brown, Adams, & Jones, 1976). In chronic alcoholics, smooth pursuit tracking contains a higher proportion of saccades than does that of normal subjects of the same age (Kobatake, Yoshii, Shimohara, & Takagi, 1983).

Korsakoff's syndrome is the neurobehavioral consequence of long years of alcohol abuse coupled with vitamin deficiency (Victor, Adams, & Collins, 1977). The major neuropsychological symptoms which characterize this disorder are anterograde and retrograde amnesia, problems in concept formation and problem solving, and visuo-perceptive impairments (for reviews see Talland, 1965; Butters & Cermak, 1980). Given this evidence of the patients' impaired processing of visual information, it is important to determine the effects of the Korsakoff's syndrome on basic oculomotor function.

In a previous report we discussed the abnormalities of the saccadic and fixational systems in these Korsakoff patients (Kenyon, Becker, Butters, & Hermann, 1984). However, since the smooth pursuit and saccadic systems are functionally independent (Rashbass, 1961), it is also important to evaluate the oculomotor tracking abilities of these patients. The purpose of this study, therefore, was to examine quantitatively the smooth pursuit eye movements of alcoholics with Korsakoff's syndrome across a wide range of target velocities (4–20°/sec). Patients showed decrements in their ability to produce the peak velocities necessary to track moving targets. Even the slower velocities which they could produce could not be sustained during an entire target movement.

METHODS

Subjects

The subjects of this study were four alcoholics and one nonalcoholic control who have been described in detail in a previous report (Kenyon et al., 1984). Of the alcoholics three had been diagnosed with Korsakoff's syndrome at least two years prior to testing. The fourth subject, C.B., had developed a Wernicke's encephalopathy but did not develop Korsakoff's syndrome. One of the Korsakoff patients, G.P., had no confirmed history of the acute Wernicke's stage of the disorder. Available demographic characteristics of the patients, as well as their verbal IQs (WAIS or WAIS-R) and Memory Quotients (MQs from the Wechsler Memory Scales) are shown in Table I. The patients had been abstinent from alcohol from 2–7 yrs prior to testing (see Table I).

Materials

Eye movement system. Horizontal eye movements were measured using an infrared reflection method (Stark, Vossius, & Young, 1962), with a linear range of ±7°, a noise

TABLE I

Demographic and psychometric characteristics of the subjects

Patient	Diagnosis	Age	Years of education	WAIS Verbal IQ	Wechsler MQ	Medication	Years since diagnosis
M.B.	Nonalcoholic	56	12	n.a.	n.a.	none	n.a.
C.B.	Wernicke's encephalopathy	58	12	120	122	none	4
J.M.	Korsakoff's syndrome	55	12	95	80	Dilantin/Elavil	7
G.P.	Korsakoff's syndrome	60	14	134	90	Phenobarbitol	2
A.A.	Korsakoff's syndrome	55	9	99	69	none	5

level of two minutes of arc, and a bandwidth of 1000 Hz. The eye position information was entered into a PDP 11/34 minicomputer with a sampling rate of 1000 Hz.

Stimulus. Eye movement stimuli were presented on the screen of a dual channel Tektronix oscilloscope. The screen was 57 cm from the subjects eyes and the beam placed at eye level. The luminous spot on the oscilloscope, the target, was focused to a point of light which subtended 6 minutes of visual arc, with luminance at least 1 log unit above threshold. Target motion was produced by a Kron Hite function generator which was controlled by the experimenter. Ten degree target movements using triangle ramps of constant velocity elicited smooth eye movements. Calibrations were performed before and after each run to ensure no changes in calibration during the experiment. Target velocities ranged between 4 and 20°/sec.

The sequence of target velocities was random such that slow and fast tracking velocities were presented in an unpredictable manner to the subjects. The purpose of this type of presentation was to reduce the reliability of the subjects' predictions of target movement; prediction can significantly modify oculomotor behaviour (Stark, *et al.*, 1962).

Procedures

The five subjects were tested while seated in front of the oscilloscope screen. The eye movement instrument was clipped to the subject's prescription glass frame, or to blank frames, which were then secured to the head with an elastic headband. Following adjustment of the eye movement sensors, the subject's head was placed in a head and chin rest to reduce head movement during testing. The room illumination was reduced to mesopic level prior to the start of testing. The eye movement system was then calibrated by having the subject fixate on the target in the center of the screen and then following the target with the eyes as it moved five degrees to the right or left of the central location. Each test session began and ended with a calibration test of the instruments to ensure that the linearity and scale factor for each eye had not changed.

Complete oculomotor testing lasted approximately 20 min. The subjects were each told to look at the target as it moved across the screen. All the subjects were tested using the same procedures except that two subjects were seen in their residences (A.A. and J.M.) and tested under low photopic rather than mesopic illumination.

Data Analysis

The eye movement responses to the moving target stimulus were hand analyzed from stripchart recordings. The resolution of the stripchart records were ± 10 ms and had a bandwidth of 100 Hz. To reduce the effects of the subjects' prediction of the target movement on the data further, only the first four ramp responses (i.e., the first two complete cycles) following a target velocity change were measured and averaged. Peak velocity was calculated by measuring the slope of the eye movement records during the pursuit. Total *saccadic* amplitude during each ramp response was calculated by adding up the amplitudes of the individual saccades made during each ramp response.
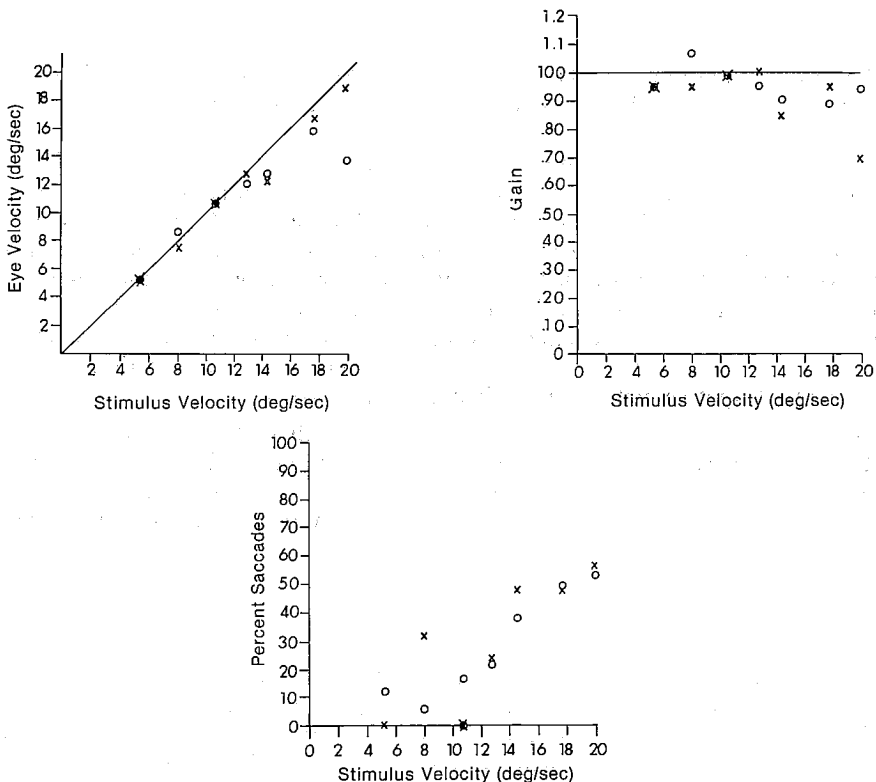

FIGURE 1   Smooth pursuit function of a normal age-matched control. (a) Average eye velocity from four ramp responses is plotted as a function of target velocity. (b) Gain of the smooth pursuit system (eye velocity/target velocity) is plotted as a function of target speed. (c) The proportion of the total eye movement composed of saccades is plotted against target velocity. All plotted points represent the average of four ramp velocities to the right (o) and left (x). The straight solid line represents ideal system performance.

RESULTS

Three measures of smooth pursuit performance were taken for each of the subjects. The results for M.B., the nonalcoholic control, are shown in Figure 1. In part A of the figure the peak velocity of her smooth tracking movements is plotted as a function of the velocity of the target across the oscilloscope screen. Movements to the left are marked with an 'x' and movements to the right with a circle (o). At low velocities, (i.e., 12°/sec), M.B.'s eye velocity matched that of the target. However, as the speed of the target increased, her ability to match the target velocity declined in a fashion consistent with other reports on normal smooth pursuit eye movements (Rashbass 1961; Fuchs, 1967). While her data deviate from the 45° line representing "perfect" tracking, it falls, well within the bands of normal responses expected from a healthy subject (Nemet & Ron, 1977; Lisberger, Evinger, Johanson, & Fuchs, 1981; Stark *et al* 1962; Fender & Nye, 1961; Schalen, 1980).

A second measure of tracking ability was the smooth pursuit gain: peak eye velocity divided by the target velocity. Gain values of less than 1.00 indicated that eye velocity lagged the target velocity; gain of 1 indicated perfect tracking. For subject M.B., shown in Figure 1b, the gain was approximately 1.00 in the range of target velocities under 12°/sec. At higher speeds, M.B.'s eyes did not match the target velocity, and the gain fell below 1.00. However, the tracking gains for the range of velocities studied were within the limits for a normal subject.
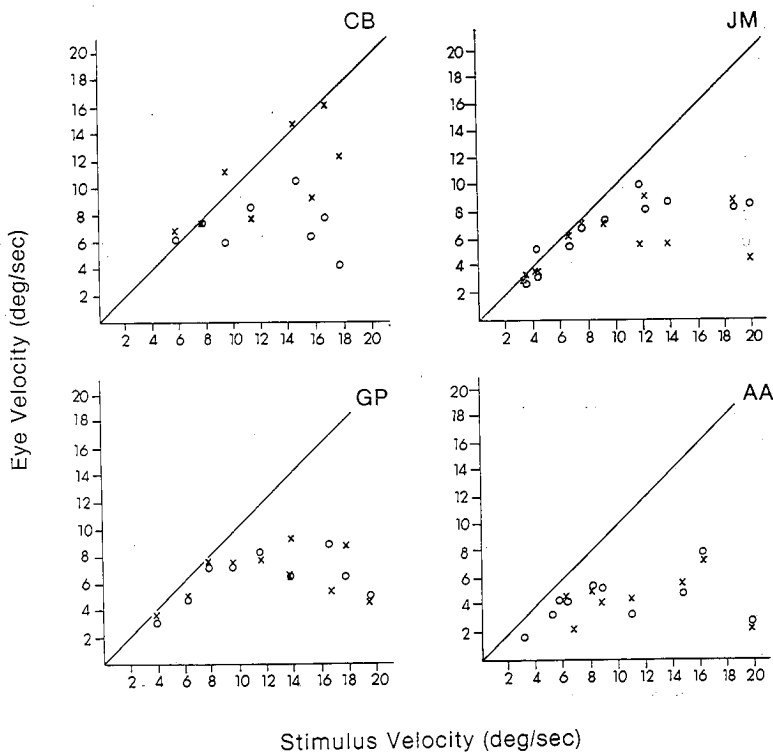

FIGURE 2   Patient smooth pursuit eye velocity versus target velocity (see Figure 1 for symbols).

As target velocity exceeds eye velocity, a positional error occurs between the fovea and the target's position on the retina. Consequently, small corrective saccades are generated during smooth pursuit to realign the fovea with the target. Saccades become a larger portion of the total tracking amplitude as the smooth pursuit system increasingly fails to track the target adequately. Thus, the proportion of the total movement which is composed of saccades can indicate the ability of the system to sustain the needed velocity to track the target. This measure contrasts with peak velocity which only indicates the maximum performance of the smooth pursuit system and not its ability to maintain that performance. The proportion of saccadic eye movements during smooth tracking is shown for subject M.B. in Figure 1c. Notice that when the target moved at slow speeds, and the eye maintained target position on fovea, virtually no saccades occurred in the record. However, as target velocity increased and tracking efficiency decreased, the proportion of the total movement made up of saccades increased. By the time the target was moving at 20°/sec, 50% of the tracking consisted of saccadic movements.

In contrast to the normal dynamics of N.B's smooth tracking responses, the performance of the four alcoholics was significantly impaired. In Figure 2 are plotted the eye velocities as a function of target velocity for each of the four patients. In
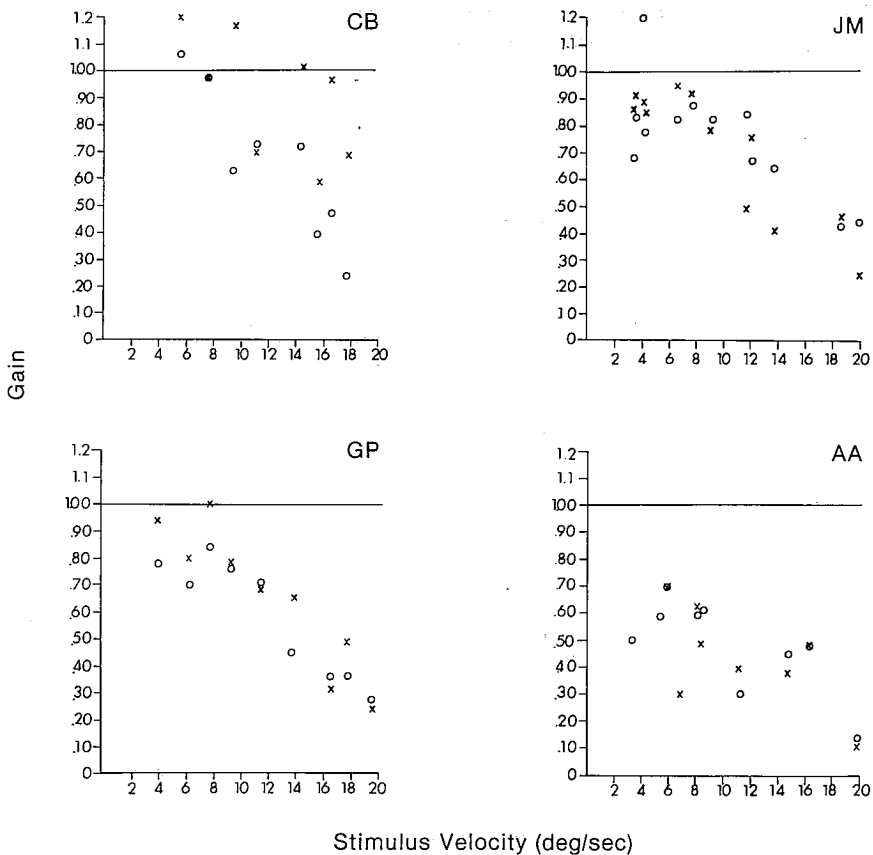

Stimulus Velocity (deg/sec)

FIGURE 3   Smooth pursuit gain versus target velocity in the four patients.

general, the peak velocity of the smooth pursuit eye movements fell below that of the target velocity, even at low target speeds (less than 10°/sec).

The smooth tracking ability of patient C.B. is shown in Figure 2a. Tracking in the leftward direction (shown by 'x' was relatively normal with eye velocity generally equal to that of the stimulus. However, when the target moved to the right (shown by 'o') eye velocity was consistently below that of the stimulus. For the leftward movements, maximum eye velocity was approximately 16°/sec while maximum velocity to the right was only 10°/sec.

The tracking ability of the three patients with Korsakoff's syndrome was consistently poorer than that of M.B. and C.B. For subject J.M. the peak velocity of his eye movements began to depart from the velocity of the target at approximately 8°/sec, at which point eye velocity saturated. That is, in spite of the increases in target speed, and the resultant changes in input to the smooth pursuit system, the eyes did not move any faster than 8°/sec.

A similar pattern of performance was seen in patient G.P. Although he was able to track the target at speeds slower than 8°/sec, his ability was impaired for velocities between 8 and 20°/sec. Maximum velocity was reached at approximately 10°/sec, with similar saturation effect.
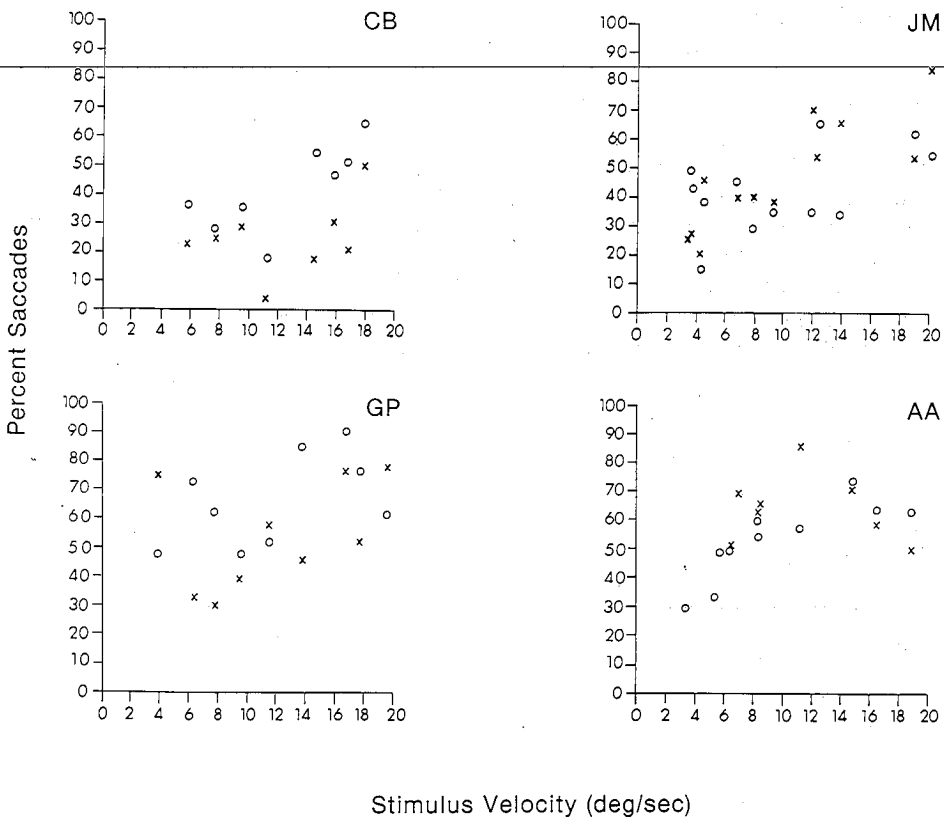

FIGURE 4   Percentage of total tracking amplitude composed of saccades plotted against stimulus velocity in the four patients.

Finally, the results of the smooth tracking task from patient A.A. are even poorer than those of G.P. and J.M. He was unable to follow the moving stimulus adequately even at very slow target velocities (less than 8°/sec). A.A.'s maximum velocity was approximately 8°/sec at target velocity of 16°/sec.

Not surprisingly, the performance of the four patients was equally poor when assessed with the index of tracking gain (see Figure 3). Normal smooth pursuit function has a gain of approximately 1.00 up to velocities of 15–20°/sec (Rashbass, 1961; Fuchs, 1967). In these four patients, however, tracking gain began to fall below 1.00 at target velocities as low as 8–10°/sec. Patient C.B. showed a normal velocity gain for tracking leftward movements but a much reduced gain tracking rightward movements. Unlike C.B., the three Korsakoff patients showed reduced gains for both right and left tracking targets. Once again patient A.A. showed the most tracking impairment, never reaching a gain of 1.0. Both G.P. and J.M. showed similar declining functions with increased target velocity. Each showed good tracking only at very low velocities.

For the Korsakoff patients, and to a lesser extent C.B., saccades were always a part of the smooth pursuit record. That is, even at low target velocities, the retinal error was such that small corrective saccades were needed to keep the fovea aligned with the target as it moved across the screen (Figure 4). Notice that for all patients, the proportion of the tracking movements that were made up of saccades was higher than that for M.B. (see Figure 1c), and increased as the velocity of the target increased. When target velocity reached 16°/sec, fully 50% of the patients' tracking was composed of saccadic eye movements.

Eye movement records from each subject are shown in Figures 5 and 6. For each subject, records are shown at both low (6°/sec) and high target velocities (12°/sec). Notice that for subject M.B. (Figure 5), smooth tracking at the low velocity was quite sufficient to keep the eye aligned with the target. At the higher target speed, however, one saccade was needed in each ramp to help correct for retinal position error. This performance is similar to that seen in other healthy normal subjects.

The tracking records for the four patients, however, show grossly abnormal oculomotor function (Figure 6; note differences in time calibrations). The records for all
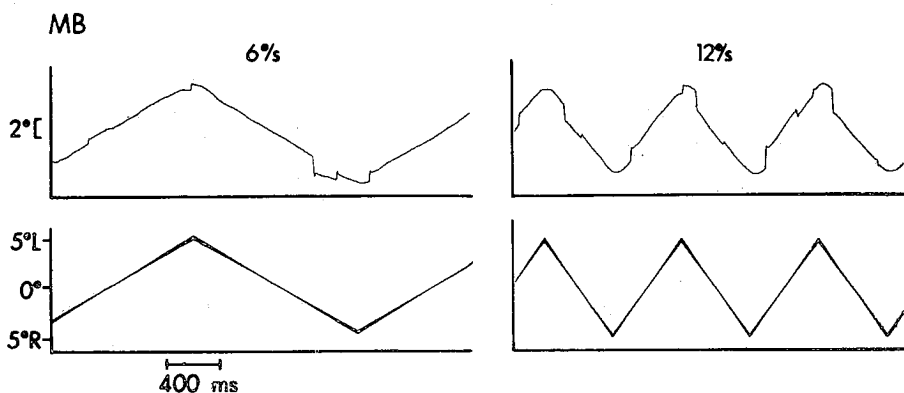

FIGURE 5 Smooth pursuit responses from age-matched control while tracking target at 6°/sec and 12°/sec. The upper trace slows target position and the lower trace eye position. Calibration and time marks are indicated on each trace.
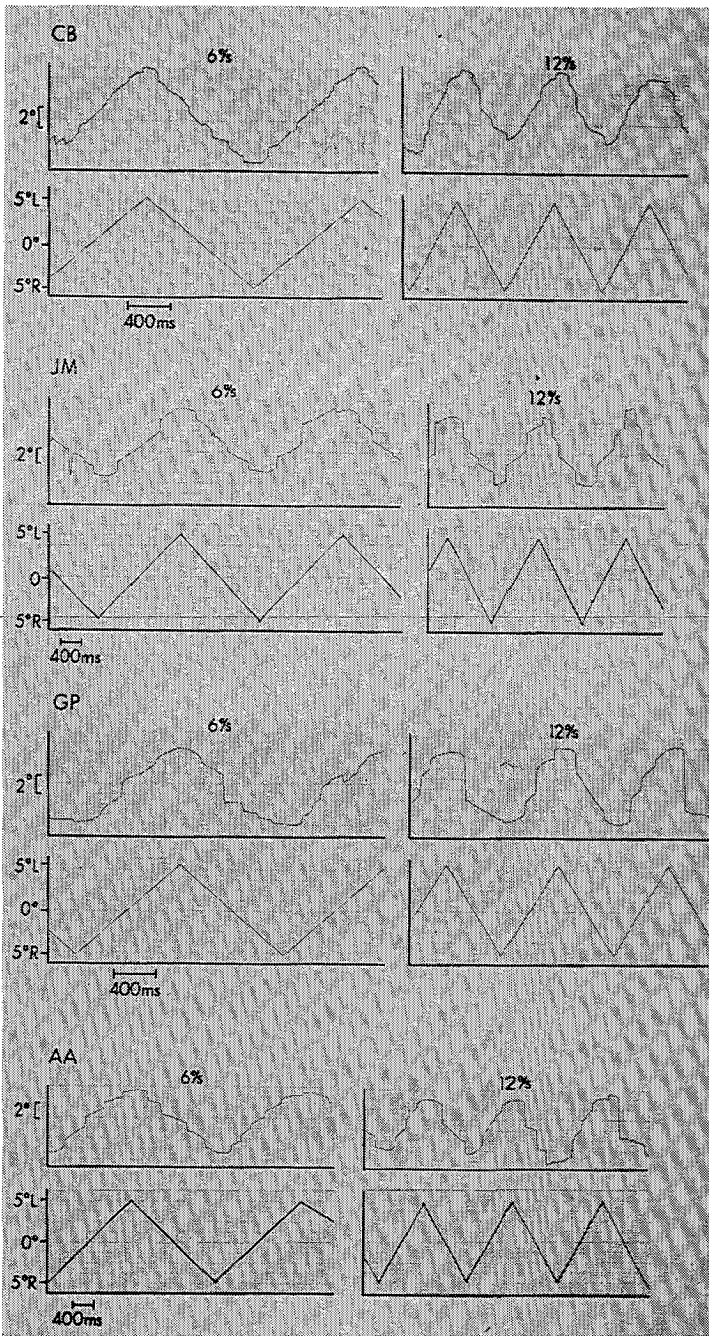
FIGURE 6   Smooth pursuit responses representative of tracking behavior from each patient at 6°/sec and 12°/sec constant velocity ramps. Calibration and time marks are indicated on each trace

four patients include corrective saccades, even at the low target speed. The quality of the smooth pursuit was clearly poor for A.A. and G.P. even at low target velocities. Their tracking, shown in Figure 6, has a staircase pattern with reduced eye velocity between the refixation saccades. However, somewhat better pursuit was seen in the other two patients, C.B. and J.M. Smaller yet more frequent saccadic movements occurred at low target speeds, while larger amplitude and less frequent saccades were observed at the higher target velocities. Within the range of velocities tested we did not observe pure (i.e., exclusively) saccadic tracking at any time.

DISCUSSION

These data demonstrate significant changes in the smooth pursuit tracking ability of patients with alcoholic Korsakoff's syndrome. The tracking performance of the Korsakoff patients was marked by reduced maximum eye velocity, reduced velocity gain, and a decrease in the ability to sustain peak eye velocity. Similar abnormalities have been reported for detoxified chronic alcoholics (Kabatake et al., 1983). The alcoholics had a larger proportion of saccadic movements during smooth pursuit than did age-matched nonalcoholic controls. The present data confirms this finding, and also indicate that the increased saccadic frequency is the result of lower velocity gain and reduced peak velocity.

These chronic changes in smooth pursuit function are similar to those seen in nonalcoholic individuals who have received an acute dose of alcohol. When blood alcohol levels reach approximately 25 mg/ml, smooth pursuit ability begins to deteriorate and declines rapidly with increasing blood alcohol levels. At approximately 40 ml/mg blood alcohol smooth tracking stabilizes in this deteriorated state (Wilkinson, et al., 1974; Flom et al., 1977). The performance of the Korsakoff patients reported here appears to be as poor as that of nonalcoholics with blood levels of 40 mg/ml of alcohol.

Patients with dementing syndromes, such as presumed Alzheimer's Disease, also have significantly impaired oculomotor function (Hutton, 1980). Hutton (1980) found an elevated number of velocity arrests during smooth tracking which increased with the severity of the dementia. The correlation between the severity of the dementia and tracking deficiency was larger at higher target velocities (18.4°/sec) which were more demanding of the sensory/motor system.

Alcoholic Korsakoff patients, chronic alcoholics, and patients with Alzheimer's disease share many visuoperceptual deficits (Rosen, 1983) and in addition, many of the oculomotor abnormalities in these patient groups are similar. Although the oculomotor changes may correlate with the degree of cognitive impairment (Pirozzolo & Hansch, 1981; Hutton, 1980), the present data cannot be interpreted as representing the basis for the perceptual abnormalities. Although the fixational (Kenyon et al., 1984) and tracking abilities of Korsakoff patients are impaired, no clear relationship has been shown between these types of oculomotor abnormalities and perceptual dysfunction. Further work, especially that which would be directed at visual scanning and scan-path mechanisms (Stark & Ellis, 1982) would help elucidate the relationship between oculomotor functions and perceptual abnormalities.

The anatomical basis for the smooth pursuit dysfunction among the alcoholic Korsakoff patients may lie in the cerebellum. Patients with Korsakoff's syndrome have been found to have a loss of Purkinje cells in the region of the flocculus and

paraflocculus (Victor, Adams, & Collins, 1971). Studies with monkeys have shown that these cells encode information about eye velocity, and may be involved in the early phases of smooth pursuit movements (Lisberger & Fuchs, 1978; Miles & Fuller, 1975). In addition, while ablation of the flocculus and paraflocculus in monkeys (Westheimer & Blair, 1973; Zee, Yamazaki, Butler, & Gucer, 1981; Burde, Stroud, Roper-Hall, Wirth, & O'Leary, 1975) and man (Nemet & Ron, 1977) does not eliminate the ability to make smooth pursuit movements, eye tracking is significantly impaired (Zee et al., 1981).

The functional basis of the smooth pursuit tracking deficiency lies in the inability of the patients to generate smooth movements of high velocity. Normal peak eye velocity can be as high as 150°/sec if the target amplitude is increased sufficiently (Lisberger et al., 1981). That is, when the eyes are permitted to move 30–49°, they are capable of very high velocities because they can apply maximum smooth pursuit acceleration for a longer time before reaching the limits of the target movement (Lisberger et al., 1981). The peak smooth pursuit eye velocity is thus controlled by two factors: (1) the time available for the eye to accelerate to a particular velocity (which is related to the distance the target moves), and (2) the maximum acceleration signal of the smooth pursuit system (Lisberger et al., 1981). If either the time available, or the maximum acceleration is limited, the eyes will not be able to attain high velocities. In the present case, the abnormal smooth pursuit velocities might be best understood by assuming a lower than normal acceleration limit in the patients' smooth pursuit system. With such a reduced acceleration ability the eyes must have a relatively longer period of time to accelerate in order to reach a particular target velocity. At lower speeds (e.g., 6°/sec), the subjects had a relatively long time during each ramp movement to accelerate their eyes to target speed. Thus, their tracking is relatively good at low velocities. However, at higher target speeds (e.g., 12°/sec) sufficient time was not available during each ramp for the eyes to accelerate to target speed, and thus, performance deteriorated at high target velocities. Furthermore, according to this view, as the target speed increases, and the amount of time available for acceleration during each ramp decreases further, the velocity of the eye movements should actually decrease. In fact, this was evident to some extent in each of the patient's records, but was most obvious in patient G.P.

Age is a factor which reduces smooth pursuit functions (Keuchenmuster, Linton, Mueller, & White, 1977; Sharpe & Sylvester, 1978; Spooner, Sakala, & Baloh, 1980; Kobatake et al., 1983). Normal subjects above 50 years of age show a 10% reduction in peak pursuit velocity with target velocities of 22°/sec (Spooner et al., 1980). However, the changes shown in the Korsakoff patients are much more severe than those of normal elderly subjects. At 20°/sec our patients had reductions of 70% (G.P.), 90% (A.A.), 60% (J.M.) and 45% (C.B. at 18°/sec) in peak smooth pursuit velocities. While some tracking deficiency may be attributable to normal aging effects, these are relatively minor and do not represent the basis of these patients' severe oculomotor problems.

Various medications have been shown to change smooth tracking function. Phenobarbital severely limits the subjects ability to maintain smooth tracking function (Rashbass, 1959; Norris, 1971) and this may account for some of G.P., oculomotor abnormalities. However, the smooth pursuit eye movements of A.A., who was not medicated at the time of testing were as affected as those of G.P. Thus, these oculomotor abnormalities probably do not reflect simply the effects of medication.

In summary, alcoholics with Korsakoff's syndrome have significantly abnormal smooth pursuit eye movements. These findings coupled with our previous report of

abnormal saccadic eye movements indicates that eye movement disorders are a continuing feature of Korsakoff patients' oculomotor system, and the diffuse damage this condition inflicts on the nervous system.

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