

OCULOMOTOR FUNCTION IN WERNICKE-KORSAKOFF'S SYNDROME: I. SACCADIC EYE MOVEMENTS

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Saccadic eye movements from three patients diagnosed as having Korsakoff's syndrome and one patient who had suffered the acute Wernicke stage of the disorder were examined for changes in saccadic characteristics. The three Korsakoff patients showed increased saccadic latencies; two of the three also had reduced saccadic peak velocities and increased saccadic durations. A higher than expected incidence of hypometric saccades was also found in all four patients; saccadic intrusions were recorded in two Korsakoff patients. These abnormal oculomotor responses were found 2-7 years after the onset of the Korsakoff's syndrome and are consistent with the cerebellar and frontal lobe dysfunction reported in this patient group.

Korsakoff syndrome is the neurobehavioral consequence of long years of alcoholism coupled with a severe deficit in nutrition (i.e., thiamine deficiency). Usually the patient is first seen while in the acute Wernicke state, which is characterized by global confusion with peripheral neuropathies and ophthalmoplegia. The ophthalmoplegia has been well described and is noteworthy for the presence of nystagmus, principally in the vertical plane, and for gaze palsy (Victor, Adams, & Collins, 1971). Following the administration of thiamine and the restoration of a nutritionally adequate diet devoid of alcohol, the confusional state clears, the peripheral neuropathies subside, and within four to six weeks the ophthalmoplegia recovers (Victor *et al.*, 1971). In the majority of cases, the severe, chronic neuropsychological syndrome first described by Korsakoff (Korsakoff, 1887, cited by Victor *et al.*, 1971), remains. The major features of the Korsakoff stage of the disorder are a severe and enduring inability to learn new information (i.e., anterograde amnesia), and an impairment in the ability to recall

information prior to the onset of the illness (i.e., retrograde amnesia), despite relatively intact intellectual capacity (Butters & Cermak, 1980; Victor *et al.*, 1971). Patients with Korsakoff's syndrome suffer virtually no changes in language function, but do have significant visuoperceptual abnormalities (Butters & Cermak, 1980; Butters & Brandt, 1984).

Among the perceptual changes associated with Korsakoff's syndrome are deficits in the ability to process visual information. These patients are impaired in the performance of tests of visual scanning (Talland, 1965), visuoperceptual contour analysis (Kapur & Butters, 1973), and tasks which require perceptual closure (Talland 1965). In addition, the irrelevant superficial features of a visual stimulus, such as hairstyle and clothing in a face perception task, are sufficiently salient to interfere with the Korsakoff patient's stimulus analysis (Dricker, Butters, Berman, Samuels, & Carey, 1978). One factor which may be responsible for these perceptual abnormalities may involve a subtle impairment of eye movement function. Although the Korsakoff patient's clinically apparent oculomotor deficits have long since abated, residual defects in scanning and fixation might have a detrimental effect on the individuals' ability to analyse a visual stimulus.

The purpose of the present experiment was to investigate the oculomotor functions of patients with Korsakoff's syndrome. The emphasis in this report was limited to saccades, the rapid movements of the eye which bring targets into the center of the visual field. Saccades direct the eye during scanning of the visual environment. We chose these particular movements for several reasons. First, they are involved to a large extent in visuoperceptual functions such as scanning. Secondly, they are controlled by brainstem, cerebellar, and frontal lobe areas which may be damaged in patients with Korsakoff's syndrome. Finally, these eye movements are the fastest and most demanding motor acts made by the body and may be particularly sensitive to changes in brain function.

METHODS

Subjects

Three alcoholics with Korsakoff's syndrome, one alcoholic who had suffered a Wernicke's encephalopathy but did not develop Korsakoff's syndrome, and an age-matched nonalcoholic control were the subjects of this investigation. Two of the patients (A.A. and J.M.) developed Korsakoff's syndrome following a Wernicke's encephalopathy and subsequent treatment with thiamine. Subject G.P. developed Korsakoff's syndrome with no documented history of having gone through the acute stage of the disorder. Available demographic data for these subjects are presented in Table I.

The alcoholics with Korsakoff's syndrome (G.P., A.A., and J.M.) all had Wechsler Adult Intelligence Scale (WAIS) Verbal I.Q.s appropriate for their education and socioeconomic backgrounds. Their Wechsler Memory Quotients (MQ), on the other hand, were approximately 30 points lower than their IQs; this discrepancy between IQ and MQ is one of the psychometric characteristics of Korsakoff's syndrome. In contrast, the alcoholic who did not develop Korsakoff's syndrome (C.B.) had an MQ virtually identical to his IQ. The nonalcoholic control was of similar age to the four experimental subjects.

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/Elvail	7
G.P.	Korsakoff's syndrome	60	14	134	90	Phenobarb.	2
A.A.	Korsakoff's syndrome	55	9	99	69	none	5

Materials

Eye movement system. Horizontal eye movements were detected using an infrared reflection method (Stark, Vossius, & Young, 1962). An infrared light-emitting diode (FPE100) diffusely illuminated the eye while two infrared sensitive phototransistors (TI-LS400) were pointed at the limbus on each side of the orbit. These measured the light reflected from the eye and their output was differentially amplified to produce a signal proportional to eye position. This signal was sampled by the computer at a rate of 1000 Hz. The system used had a linear range of $\pm 7^\circ$ and had a noise level of 2 min arc; the instrument's bandwidth was 1000 Hz.

The eye position information was entered on-line into a PDP 11/34 minicomputer for subjects G.P., C.B., and M.B. Two of the patients, A.A. and J.M. were tested in their residences, so their raw data were first stored on an F.M. instrumentation tape-recorder and then played back into the same computer used in gathering the other subjects' data. These F.M. recordings were taped at 3 3/4 *ips*, resulting in a recorder bandwidth of 1250 Hz. Because these bandwidths are all well above the power of saccadic eye movements (Zuber, Semmlow, & Stark, 1968), there would be no important changes in the characteristics of the eye movements between the data recorded in the laboratory and that recorded in the residences.

Stimulus. Eye movement stimuli were presented on the screen of a dual channel Tektronix oscilloscope. Control signals were fed exclusively to the X-axis amplifier so that the beam moved only in the horizontal plane. The oscilloscope was placed so that the screen was 57 cm from the subject's eyes, and the beam was at eye level. The small luminous spot on the face of the oscilloscope screen, the target, was focused to a point which subtended 6 minutes of visual arc, and its luminance was at least 1 log unit above threshold. Target motion was produced by a Kron Hite function generator which was controlled by the experimenter. Five and ten degree target movements which occurred at irregular intervals were used to elicit saccades. The different target amplitudes along with the irregular interstimulus intervals made it difficult for the subject to predict the future motion of the target. The interval between successive target movements ranged from 50 to 4000 ms.

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 (if needed) 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 low 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 follow the target as it moved five degrees to the right or left of this central point. Each test session began and ended with a calibration test of the instrument to ensure that the linearity and scale factors for each eye did not change during testing. Complete oculomotor testing lasted approximately 20 min.

The subjects were each told to look at the target as it moved across the screen. The eye movement stimuli were generated by having the target move from one side of the screen (e.g., five degrees to the right) to the other side (e.g., five degrees to the left) instantaneously. The subjects generated saccades to bring the target back into central view. Five degree stimuli were generated by moving the target either from the central position to one side (e.g., five degrees to the right), or from a side position to the center. All of the subjects were tested using the same procedures, except that the two subjects seen in their residences (A.A. and J.M.) were tested under low photopic rather than mesopic illumination.

Data Analysis

The eye position information was processed off-line with the help of a minicomputer and analysis software. The data were visually displayed and then transformed with a

TABLE II
Summary of quantitative characteristics of the saccades

Subjects	Mean saccadic latency ^a	Peak velocity ^b		Duration ^a	
		5°	10°	5°	10°
M.B.	187 (± 45) (N=8)	244.7	397.8	33.4	39.8
C.B.	322 (± 103) (N=18)	266.1	387.5	34.8	51.9
J.M.	307 (± 44) (N=32)	247.8	389.9	29.9	37.5
A.A.	250 (± 74) (N=31)	189.1	297.4	42.1	54.5
G.P.	393 (± 112) (N=29)	175.0	297.3	52.9	64.8
Normative values	200 (± 30)	261 (± 42)	410 (± 67)	42 (± 8)	51 (± 8)

^amilliseconds

^bdegrees per second

The normative values for the saccadic latencies were taken from Saslow, (1967), those for the saccadic velocities and durations from Bahill *et al.* (1981).

differentiator program so that saccadic velocity could be determined along with duration and amplitude. The bandwidth of the differentiated data was 250 Hz (Bahill & McDonald, 1983). Saccadic latencies were only measured for unpredictable target movements, and this was done from a strip chart record of the eye movements. To analyze saccadic latencies we selected out those saccades which followed target movements with interstimulus intervals of at least 1 sec; all other analyses used the entire range of saccadic responses. The resolution of the strip chart record was ± 10 ms and its bandwidth was 100 Hz. This time resolution of the latency data was more than adequate given the magnitude of the effect needed for results to be statistically significant.

The main sequence characteristics of our subjects were compared to that of a normal population (Bahill, Brockenbrough, & Troost, 1981). Peak velocities and saccadic duration at five and ten degrees were predicted for each of our subjects from linear regression equations based on Log (10) transformed scores. These "best fit" estimates were used because even though our subjects generated saccades of nearly five and ten degrees, they never generated saccades of *exactly* five or ten degrees. Comparison to data from Bahill (Bahill *et al.*, 1981) was chosen because they most closely resembled the instrumentation and the bandwidth of both our position and velocity data from our subjects. Latency data were compared to Saslow's (1967) population scores.

RESULTS

Saccadic Latency

In Figure 1 are shown the distribution functions of saccadic latencies for each of the subjects. The distribution of latencies for subject M.B., the control, are shown with open circles. The saccadic latency following a five or ten degree target movement was 187 msec for subject M.B., the nonalcoholic control. This was well within the limits established for both young and old normal subjects (Becker, 1972; Saslow, 1967; Stark *et al.*, 1962) (see Table II).

The mean latency for subject A.A. was 250 msec not significantly different from that of M.B. ($t(37) = 3.78, p < 0.05$), but which was 1.7 standard deviation units about the mean of the normative population (Saslow, 1967). The mean saccadic latencies for the remaining three patients were all significantly longer than that of M.B. (J.M., $t(38) = 6.31, p < 0.001$; C.B., $t(24) = 8.31, p = 0.003$; G.P., $t(35) = 4.84, p < 0.001$) and well above the mean for the normative population. These long latencies did not appear to be related either to the size of the saccade or to its direction. However, small secondary corrective saccades were often necessary to bring the eyes to final target position and the latencies of these eye movements were within normal limits (i.e., 150–200 ms after cessation of principle saccade; see Figure 5). Although not included in the latency data analyzed for each subject, we did observe that the corrective saccades after a hypometric saccade had latencies one would expect for a normal subject given the size of the saccade correction (Becker, King, Fuchs, Jurgens, Johanson, & Kornhuber, 1981).

Main Sequence

The relationship between the amplitude of a saccade and its duration and peak velocity is called the main sequence (Bahill, Clark, & Stark, 1975; Zuber, Stark, & Cook, 1965). The main sequence is a quantitative description of the manner in which saccadic peak velocity and the duration of the movement change as a function of the size, or amplitude of the saccade.

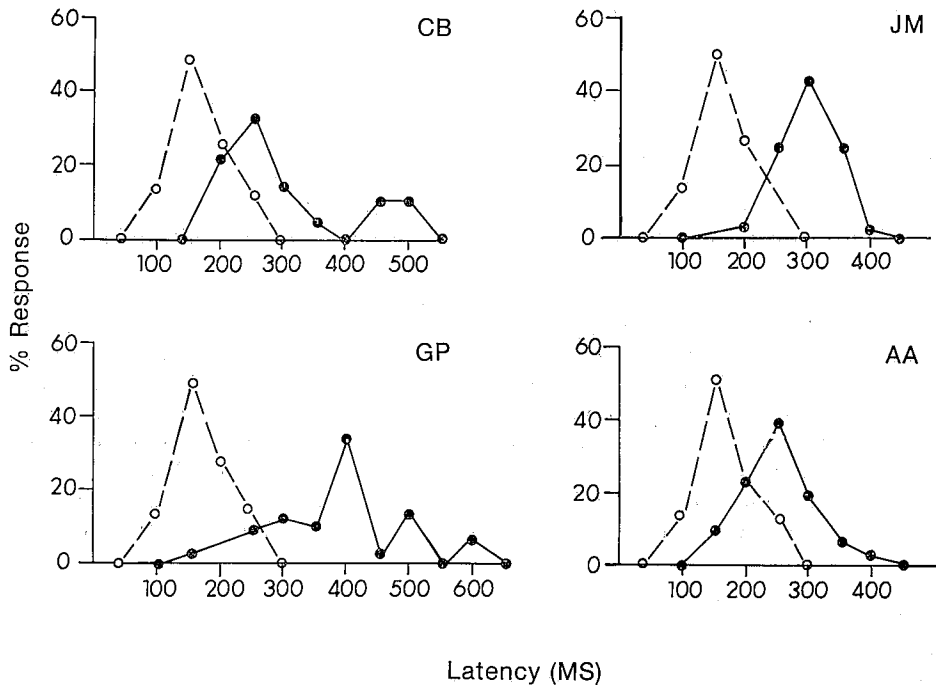


FIGURE 1 Saccadic latency distributions for the four patients (●) plotted with that of the normal control subject (○). Each point represents the percentage of saccades with latencies ± 25 ms about the data point. (i.e., value at 100 ms includes latencies between 75 ms–125 ms).

Figures 2 and 3 present the main sequence data for all of the subjects. Each of the four patient's data are presented as individual points and *solid* regression lines, while the data from control subject M.B. are presented as a dashed regression line. Notice that as the amplitude of the saccades increase, the peak velocity and saccadic duration increase as well. The relationships between amplitude and duration ($r=0.94, p<0.001$) and amplitude and peak velocity ($r=0.96, p<0.001$) are large and highly significant in subject M.B. The predicted saccadic duration and peak velocity for M.B. at five and ten degrees are very close to the mean value for a normal population (see Table II).

The results of the main sequence analysis differed in the four patients. For C.B. and J.M., the relationship between amplitude and saccadic duration and between amplitude and peak velocity were very close to the mean value for normal population at 5 and 10° (see Table II).

The main sequence analysis for A.A. and G.P. revealed several important abnormalities. The peak velocities of saccades of five and ten degrees were well below those of a normal population (see Table II). An example of one of these abnormal saccades appears in Figure 4. The upper trace is the record of eye position and the lower trace is eye movement velocity. The saccade presented in Figure 4A has an abnormally long duration and a lower peak velocity than the normal saccade shown in 4B.

Hypometric Saccades

The qualitative characteristics of the saccadic eye movements of the nonalcoholic control were essentially normal. M.B. was able to attain the final target position and maintain fixation without difficulty. Twelve percent of her saccades (3 of 26) were hypometric. That is, the initial eye movement was not sufficiently large to bring the eyes in line with the target and a second corrective saccade was generated. Approximately 10% of all saccades in a normal population are hypometric (Becker, 1972). There were no clear abnormalities in M.B.'s eye movements.

Patient C.B. also showed no overt saccadic instabilities. Fixation on the target was normal and stable and saccades were accurate. Thirty-eight percent (9 of 24) of his saccades were hypometric, a significantly greater proportion than seen in M.B. ($\chi^2(1)=4.69, p=0.03$). However, subjects J.M., G.P., and A.A. had the greatest

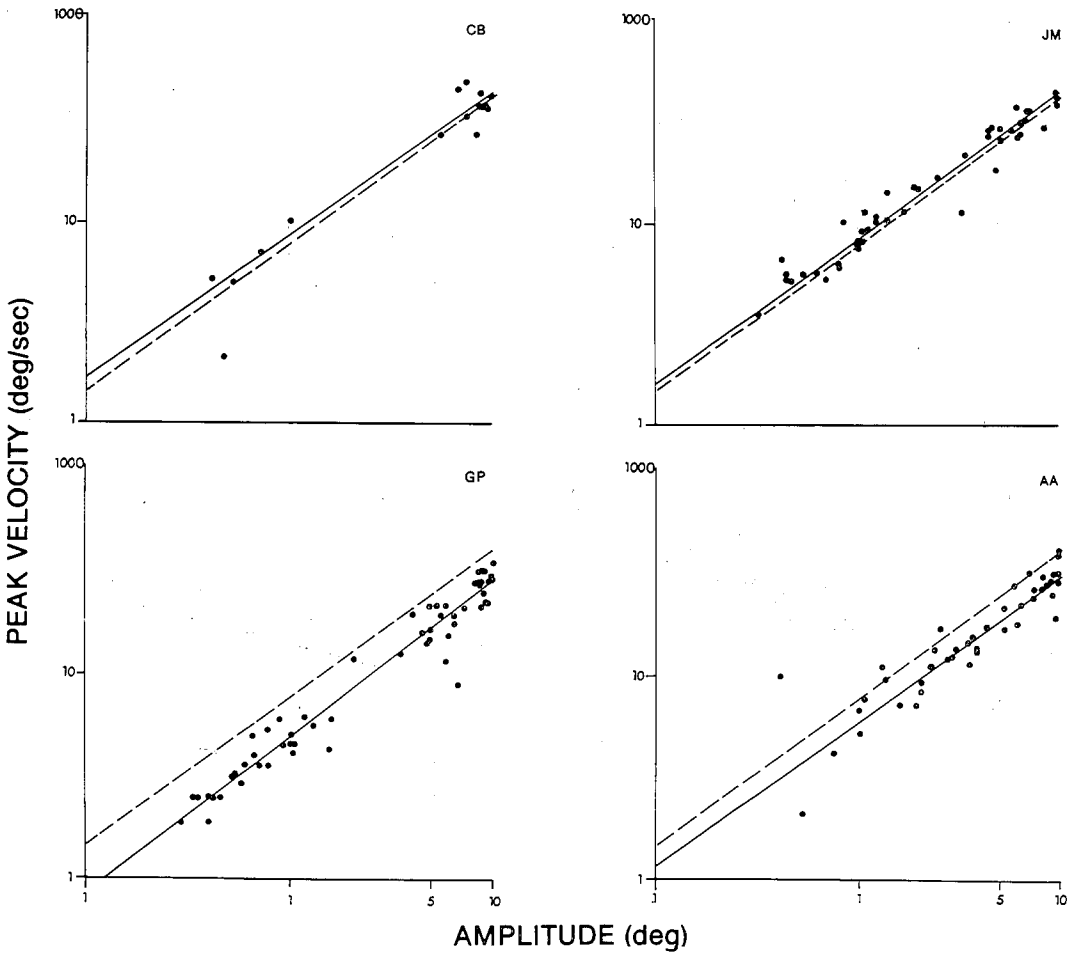


FIGURE 2 Peak velocity of the saccades plotted as a function of saccadic amplitude on log-log coordinates. The solid line represents the best-fit regression line through the subjects' data points. The broken line represents the best fit regression line for the main sequence of the control subject, M.B.

proportion of hypometric saccades. Approximately half the saccades generated by J.M. (59%, 39 of 66), G.P. (49%, 24 of 49) and A.A. (57%, 29 of 51) were hypometric. These are significantly greater proportions than that seen in M.B. (J.M.: $\chi^2(1)=17.13$, $p=0.0002$; G.P.: $\chi^2(1)=10.54$, $p=0.0016$; A.A.: $\chi^2(1)=14.73$, $p=0.0003$) and are more frequent than in the normal population (Becker, 1972). An example of a hypometric saccade in Figure 5 shows eye position for both the right (upper trace) and left (middle trace) eyes as well as the position of the stimulus target (lower trace). This response was typical for this patient and the other two Korsakoff patients. Two or three saccades were often needed for the eye to acquire the target. Notice that the latency to initiate the primary saccade is approximately 320 ms while the subsequent refixation saccades have latencies of 120 ms.

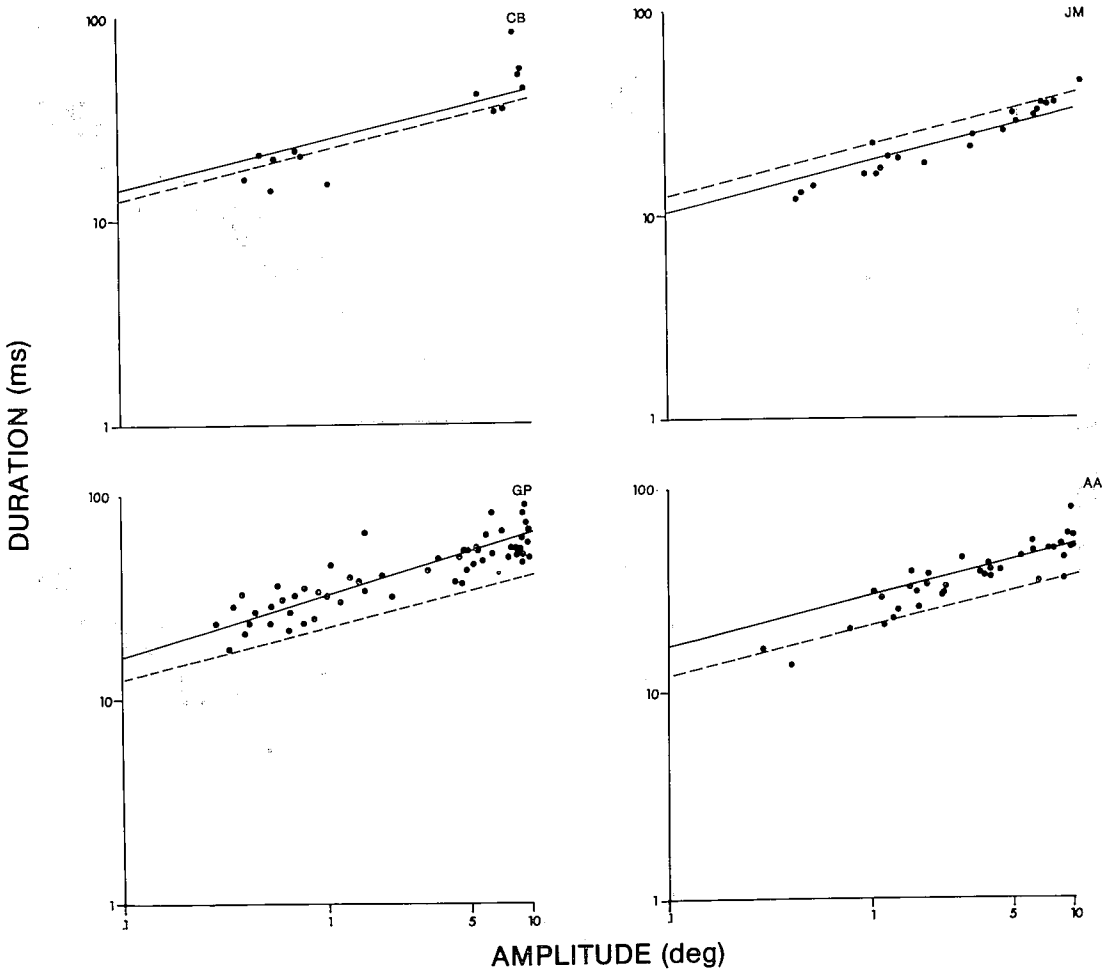


FIGURE 3 Duration versus amplitude main sequence plots for the four patients.

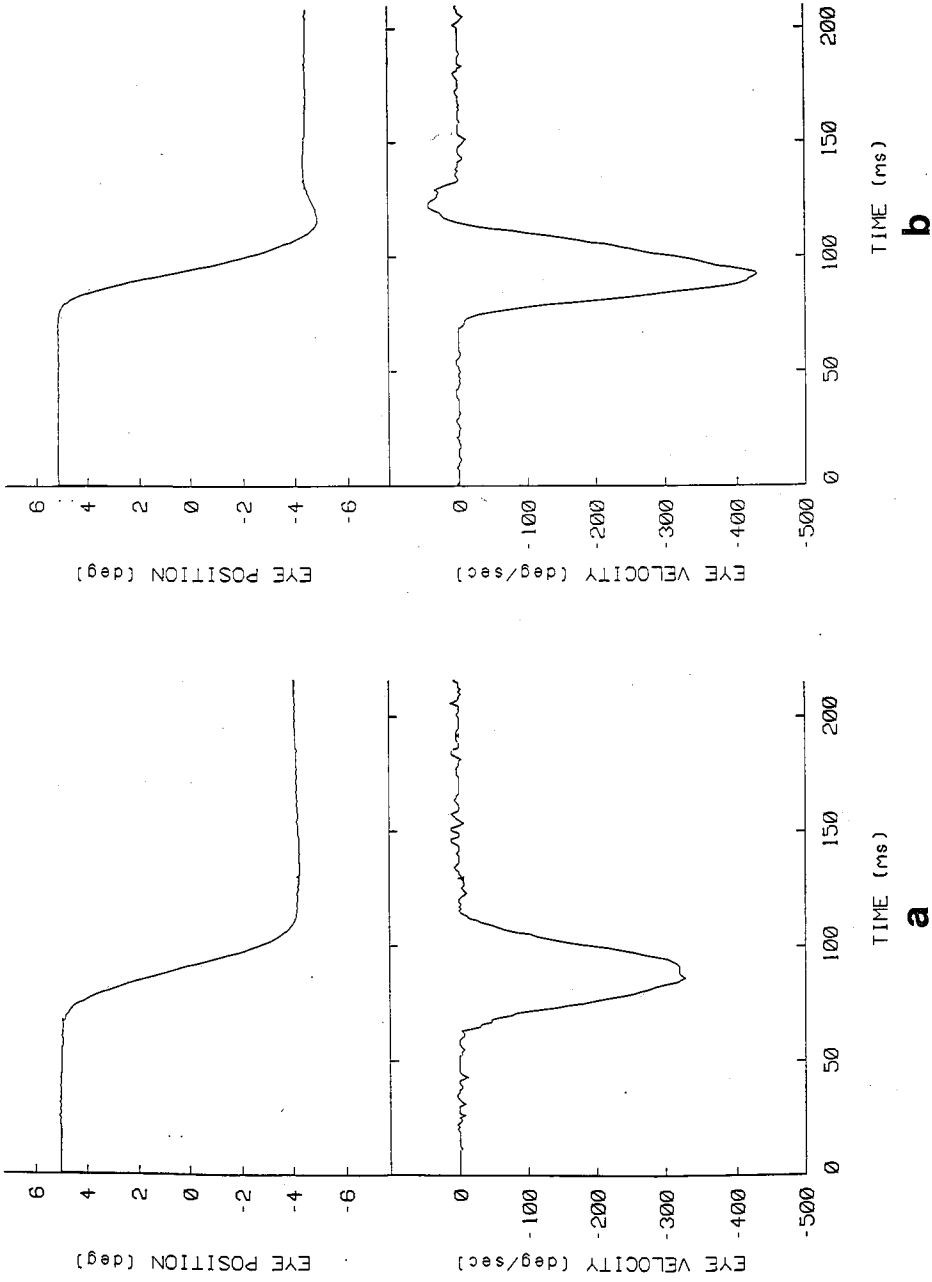


FIGURE 4 Two representative saccades taken from computer-recorded data. The upper trace represents eye position and the lower trace is eye velocity. In the left-hand portion (A) a 9.8° saccade made by patient G.P. is contrasted with a 9.6° saccade (B) by the normal control subject. Scales are shown on the figures.

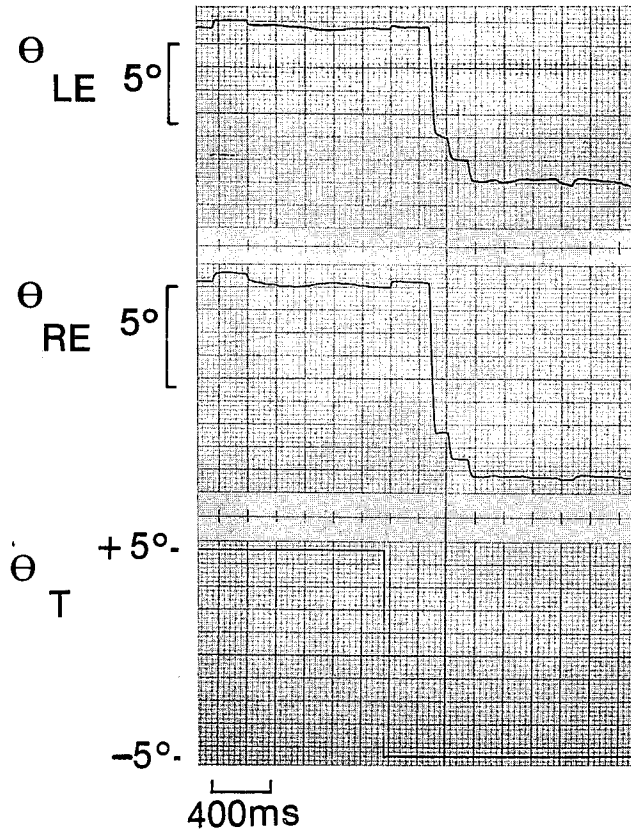


FIGURE 5 A trace of eye position and stimulus movement taken from stripchart record of patient G.P. The upper two traces show left- and right-eye position, respectively. The lower trace shows stimulus position. Scales are shown on the graphs.

Saccadic Intrusions

Saccadic intrusions or square wave jerks were present throughout the records of G.P. and A.A. For G.P., these intrusions were typically 0.5° in size, although they ranged from 0.25 – 1.0° . The intrusions occurred approximately *every two seconds*. For A.A., the intrusions were approximately the same size, but occurred more frequently: *two per second*. These intrusion saccades also fell on the main sequence lines for each subject, indicating that they, too, were slower and took longer to complete than would be expected in a normal individual.

Summary. The saccadic eye movements of the alcoholic and the alcoholics with Korsakoff's syndrome had several abnormalities. First, saccadic latencies were generally increased for movements following target position change, although the latencies to begin corrective saccades were normal. Second, the peak velocity and saccadic duration for five and ten degree saccades were abnormal in two subjects with Korsakoff's syndrome, although the relationship between saccadic amplitude, duration,

and peak velocity was normal in all of the subjects. Finally, all four patients had increased incidence of hypometric saccades, although these increases were greatest in the three Korsakoff patients.

DISCUSSION

The alcoholics with Korsakoff's syndrome have demonstrable abnormalities in their saccadic eye movements which have persisted many years after the clinically apparent ocular dysfunctions have cleared. In none of the cases studied was there any evidence of nystagmus, which is usually seen at the time of the onset of the Wernicke's encephalopathy. Nevertheless, saccadic latencies were abnormally long, saccades were frequently hypometric, and the main sequence of the saccades was affected.

These changes in oculomotor function many years after the onset of the disease parallel the transient increases in saccadic latency and decreases in peak saccadic velocity that have been reported during acute alcohol intoxication (Levett & Hoeft, 1977; Wilkinson, 1974). Indeed, the three Korsakoff patients had longer latencies than normal individuals who had blood alcohol levels as high as 0.1% with resultant saccadic velocities 30 to 50% lower than normal values.

Increased saccadic latencies may relate to the visual attention and scanning deficits often attributed to Korsakoff's syndrome (Butters & Cermak, 1980; Kapur & Butters, 1977). The increased time needed to perform visual scanning tests (Talland, 1965) may be related to the saccadic latency and the preponderance of hypometric saccades. Increases in saccadic latency (from 200–250 ms to 300–400), and the need for two or three additional corrective saccades would increase the normal time to refixate the target from 200–250 to 550–900 ms per fixation. Previous reports have shown that eye movements which delay or interfere with fixation of words, significantly increase reading time (Ciuffreda, Bahill, Kenyon, & Stark, 1976; Ciuffreda, Kenyon, & Stark, in press). Thus, the abnormal saccadic fixation of these patients with Korsakoff's syndrome may explain, to some extent, their poor performance on test of visual scanning. However, without eye movement recordings during such scanning tests, this proposal remains highly speculative. An alternative explanation is that the increased saccadic latencies may be the result of changes in a higher central function, and not specifically to the oculomotor control system. Thus, the increased scanning time may be the result of a dysfunction of visual information processing, and not changes in saccadic function *per se*.

The hypometric saccades found in these patients are likely the result of damage to cerebellar structures, in particular the vermis. Studies on humans and animals have shown that cerebellar damage results in hypometric and hypermetric saccades (Selhorst *et al.*, 1976a, b; Ritichie, 1976; Aschoff & Cohen, 1971). The behavioral observation of hypometric saccades suggest that the vermis is involved, which is consistent with neuroanatomical observations of alcoholics with Korsakoff's syndrome. The extent of the damage to this area varies, (Victor *et al.*, 1971) as does the severity of the saccadic hypometria.

Changes in the saccadic main sequence were found in two of the four patients. This decrease, while substantial, was not as dramatic as that found in patients with such diseases such a Progressive Supranuclear Palsy, in which saccadic velocities attain only a small fraction of normal velocity values (Zee, Opticon, Cook, Robinson, & Engel, 1976). Nonetheless, these changes in saccadic characteristics are often attributed to brainstem structures in the Paramedian Pontine Reticular Formation (Zee *et al.*, 1976).

The cells that produce the characteristic pulse-step neuronal firing pattern responsible for saccadic eye movements are found in this region of the reticular formation (Cohen & Henn, 1972; Luschei & Fuchs, 1972; Keller, 1974). Damage to cells in this area results in slow saccades, and in cases of severe damage, the loss of saccadic function altogether (Cohen, Komatsuzaki, & Bender, 1968; Keller, 1977). The small change in saccadic velocity and duration in two patients suggest that minor problems in brain-stem area are possible. However, the small sample size and the absence of a truly severe reduction in velocity and duration makes it difficult to reach strong conclusions regarding the anatomical basis of these oculomotor abnormalities.

Two of the four patients in this study were taking anticonvulsant medication at the time of testing. Both showed increased saccadic latencies and one showed both slow saccades and hypometria (G.P.). However, the possibility that these changes were caused by the medications is remote. First, the drugs prescribed do not have any known effects on the saccadic system (Norris, 1971; Rashbass, 1961). Furthermore, subject AA., whose oculomotor abnormalities rivaled those of G.P., was not medicated. Patient C.B., who also had increased saccadic latencies was also not taking any medications.

In conclusion, the characteristics of saccadic eye movements in these patients with Korsakoff's syndrome are clearly abnormal. Latency to begin saccades is extended, the movements tend to be slow, and they are frequently hypometric. These changes in oculomotor function are consistent with known pathological changes associated with this neurobehavioral syndrome. One important question, not addressed here, is whether these abnormalities in eye movement control extend to other oculomotor systems. In the next report (Kenyon, Becker, & Butters, 1984) we describe the changes in smooth pursuit movements in these same individuals.

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The first part of the document discusses the importance of maintaining accurate records of all transactions. It emphasizes that every entry must be supported by a valid receipt or invoice to ensure transparency and accountability. This is particularly crucial for businesses operating in regulated industries where compliance is a top priority.

In addition, the document outlines the various methods used to collect and analyze data. It mentions the use of both manual and automated systems to gather information from multiple sources. The goal is to identify trends and patterns that can inform strategic decision-making and improve operational efficiency.

Another key aspect discussed is the role of communication in the process. It is noted that regular updates and reports are essential for keeping all stakeholders informed. This helps in identifying potential issues early on and allows for a more coordinated response. The document also touches upon the importance of training staff to ensure they are equipped with the necessary skills to handle the data and reporting requirements.

Finally, the document concludes by highlighting the long-term benefits of a robust reporting system. By consistently tracking performance and financial health, organizations can make more informed choices, reduce risks, and ultimately achieve their business objectives. It serves as a guide for anyone looking to streamline their reporting processes and enhance their overall organizational performance.