smooth pursuit eye movements and functional psychoses; a review*

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In 1908 Diefendorf and Dodge reported an association between schizophrenia and impaired smooth pursuit eye movements. Using a pendulum as a stimulus and photographing corneal reflection of light, they monitored eye movements in chronic schizophrenics, manic-depressives, epileptics, and mentally retarded patients. Deviant pursuit movements characterized the performance of most of the schizophrenic patients, but of only a small proportion of other patients unless their clinical condition was markedly deteriorated. The authors interpreted their findings as evidence of a central nervous system defect in processing perceptual data. They explicitly discounted poor motivation or inattention as the cause of the group differences. Couch and Fox in 1934 repeated that experiment on a sample of 117 patients that included, in addition to schizophrenics, neurotics, psychopaths, manic-depressives, cases of dementia paralytica, mental deficiency, epilepsy, and alcoholism. These authors, too, reported disordered pursuit movements in their most severely ill patients, but the dysfunctions were not limited to the schizophrenic patients. Couch and Fox believed that inattention was principally involved in producing the deviant pursuit in their patients.

The two issues of specificity and origin of the deviant pursuit movements remained unresolved. Both studies, however, were rarely cited and they seemed to have been all but forgotten. Forty years elapsed before renewed interest in this phenomenon occurred. In a series of papers (Holzman 1975, Holzman, Proctor, and Hughes 1973, and Holzman et al. 1974) we described a significant association between impairments in smooth pursuit eye movements and psychosis. Like Diefendorf and Dodge, we found the frequency of these pursuit disorders to be significantly higher among schizophrenics than among other psychiatric patients. Further, we found that they occurred in about one-half of the first-degree relatives of the schizophrenics, in contrast to a much lower prevalence in the relatives of nonschizophrenic patients.

Several issues were raised by these findings: (1) How specific is the disorder to schizophrenia? (2) Are not drugs responsible for the disorder? (3) Are attentional factors not the likely explanation for them? (4) Does the high frequency of the findings in the relatives of schizophrenics point to a genetic factor in their appearance among schizophrenic patients? We have explored these issues and we shall discuss them in what follows. But first some general remarks about eye movements and their measurement.

Saccadic and Pursuit Eye Movements

When following a moving object, a person must first center the target on the fovea and then maintain it there. The saccadic system performs the first task. A saccade is a rapid eye movement, the fastest in the repertoire of the oculomotor system. Saccades sometimes attain speeds up to 600 degrees a second. During the saccade, meaningful sensory reception is probably suppressed (Volkman 1962 and Zuber and Stark 1966). Pursuit movements, in contrast, act to stabilize the image on the fovea by coordinating eyeball velocity with that of the target. The pursuit system can approximate the speed of the moving object up to about 30 degrees a second. The saccadic system, acting in response to target displac-
ments, is under voluntary control (Stark 1971). Once the object achieves foveal representation, involuntary minute movements—one type of which are called microsaccades—appear. Their function is to prevent the image from fading. The smooth pursuit eye movement (SPEM) is not, strictly speaking, a voluntary movement. It is affected by the velocity of the object and its position on the retina (Young 1971). Recent studies strongly suggest that both the saccadic and pursuit systems are regulated in the paramedian portion of the pontine reticular formation (PPRF) (Henrikssen and Nilsson 1975, Ron, Robinson, and Skavenski 1972, and Skavenski and Robinson 1973). All forms of slow and fast eye movements can be induced by stimulating the PPRF (Cohen and Komatsuzaki 1972 and Luschei and Fuchs 1972) and lesions in the PPRF abolish all horizontal eye movements (Cohen 1971). Precise execution of horizontal eye movements, however, requires cerebellar modulation of neural signals (Robinson 1975).

There has been some speculation that effective horizontal pursuit movements involve the inhibition of saccadic movements (Heywood and Churcher 1971), a feature we will return to later. Some investigators noted that SPEM improves with the use of efferent signals (Steinbach and Held 1968) and when target motion is predictable (Michael and Jones 1966).

A common clinical test uses a pendulum to study eye movements. Most normal persons can track the sinusoidal motion of the pendulum within a narrow critical range. Deviations from normal tracking have been reported to be associated with various pathological conditions of the central nervous system such as brain-stem and cerebellar disease (Benitez and Bouchard 1974, Dichgans and Jung 1975, Hoyt and Daroff 1971, and Jongkees and Oosterveld 1973), hemispheric lesions (Mayuzumi and Tsutsui 1974), presenile dementia, anoxia-related brain damage, and cerebral vascular disease (Rodin 1964), disorders involving the basal ganglia (Hoyt and Daroff 1971), Parkinson's disease (Slatt, Loeffler, and Hoyt 1966), and some drug intoxications such as barbiturate and Dilantin (Von Noorden and Preziosi 1966) and ethyl alcohol (Corvera, Torres-Courtney, and Lopez-Rios 1973).

Methods of Measurement

Many techniques have been used to study eye movements. These include the use of afterimages, direct observation with magnification of the image of the eye, mechanical recordings of eye movements, motion and still photography, electrooculography (EOG), and photomonitoring. Yarbus (1967) has discussed the merits of more than a dozen different methods. Of the more sophisticated techniques that yield permanent records of eye movements, photoelectric and EOG techniques are probably in greatest use for experimental and clinical purposes. Each method has its drawbacks and its advantages. The photocell method gives greater accuracy and

Figure 1. Smooth pursuit record of normal tracking

Note.—Channel 1 displays direct eye movements. Channel 2 displays the velocity of the eyes in the right (up) and left (down) directions. Calibration: 10 mm of pen deflection corresponds to 10° of eye movement.
Figure 2. Four examples of SPEM

*Note.*—Subjects are tracking a pendulum. In each figure, channel 1 records direct eye movements and channel 2 records eye velocity. Segment "a" shows normal sinusoidal SPEM with 5.6 velocity arrests per cycle. In segment "b" saccadic shifts replace smooth tracking with 14.9 velocity arrests per cycle. Segment "c" shows cogwheeling pursuit with 14.5 velocity arrests per cycle. Segment "d" is an example of a spiky pattern with 13.2 velocity arrests per cycle.

is free from artifacts that could contaminate the record since no part of the recording apparatus touches the skin. But it requires a special laboratory setup and is not easily portable. EOG, on the other hand, is portable and can therefore be used "in the field"—that is, in people’s homes. But its accuracy is lower than that of the photocell method.

In our work we have used EOG recordings because of the convenience it offers in working with disturbed patients and the ease of transporting it and of setting it up quickly in homes or in different hospital wards. We acknowledge the loss of accuracy with EOG as a trade for its portability. We have reasoned, however, that if results emerge that merit followup, it will then be possible to study selected subjects with greater precision using other apparatus in a laboratory.

Our procedure had the subject sit in front of a moving pendulum, which made a target excursion of 20 degrees of visual angle and oscillated at 0.4 Hz. A head restraint held the subject’s head still. We placed silver-silver chloride electrodes at the outer canthus of each eye and a ground at midforehead. Placement of electrodes was such as to minimize the appearance of blinks on the records. A polygraph displayed actual eye movements as well as the derivative of the eye movement signal as velocity in the right and left directions. In some experiments we used an instrumentation recorder driven by a differential preamplifier.

There are few acceptable quantitative scoring schemes for eye movements. We consequently devised two scores of our own. One is a qualitative assessment of the curve, which permits one to classify it as “good” or “bad” SPEM. The second score is a quantitative index of the degree to which eye speed departs from that of the target. We called this score “velocity arrests.” We scored velocity arrests on the derivative channel and counted all eye movements that returned to at least within 2 mm of the zero line. Velocity arrests that were obligatory (occurred at each end of the pendulum swing) were not counted. It is important to state that this score does not constitute a literal translation of actual eye movements. This is an arbitrary score, with many drawbacks, not the least of which is its dependence upon the sensitivity of the recording apparatus. It nevertheless has a fairly good repeat reliability (r from .52 to .79) and correlates .70 with the qualitative score of “good” or “bad” tracking patterns (Holzman, Proctor, and Hughes 1973 and Holzman et al. 1974). Figure 1 shows an eye-tracking record of a normal subject. The velocity arrests are labeled as c.

Figure 2 shows four qualitatively different kinds of
SPEM. Figure 2a shows good SPEM, and examples of deviant eye tracking are shown in figures 2b, 2c, and 2d. Fig. 3 is part of the record of a 46-year-old woman whose condition was diagnosed as "psychotic depression." The tracing in channel 1 represents fairly good pursuit movements, and the number of velocity arrests is 7.4 per cycle, which is within the range of a sample of 72 normal control subjects. In contrast, a portion of the record of a woman who was approximately the same age as the patient with psychotic depression but whose condition was diagnosed as schizophrenic is shown in figure 4. Here the qualitative pattern is noticeably disrupted and the number of velocity arrests is 13.2 per cycle.

Figure 3. SPEM of a psychotically depressed patient

Note.—Channels 2 and 3 display the eye velocity in the right (channel 2) and left (channel 3) directions.
SPEM and Schizophrenia

How specific are SPEM disorders to schizophrenia? Table 1 shows the prevalence of eye-tracking dysfunction found in the sample of patients we had tested. Among schizophrenics, the rate of occurrence is high. Among the manic-depressives, it is considerably lower. Shagass, Amadeo, and Overton (1974) have essentially replicated these findings, but they found SPEM dysfunctions among patients with other psychotic conditions. In further testing we, too, found that large numbers of non-schizophrenic psychotic patients showed deviant SPEM.

In our earlier work we reported that the association between schizophrenia and SPEM dysfunctions was much sharper when psychological test criteria distinguished the patient groups. That is, when the groups were divided not on the basis of diagnosis by symptoms but on the basis of the specific quality of thought disorder as described in Holzman et al. (1974), the split was much more dramatic, and the specificity for schizophrenia was much more definite.

The diagnosis of schizophrenia on the basis of thought disorder used the following pathognomonic signs: queer or absurd verbalizations, autistic logic, contaminations on the Rorschach test, and content of verbalizations suggesting preoccupation with deterioration and fragmentation. Manic psychosis was diagnosed when there was an absence of typically schizophrenic thought disorder in a context of massive denial, hyperactivity or playfulness, excitement, overproductivity, counterphobic recklessness, and extravagant interpretations leading even to confabulations. Depressive psychosis was diagnosed likewise if schizophrenic thought disorder was absent and if strong evidence was present of depressive retardation in motor, perceptual, and cognitive functioning in a setting of self-depreciation, inertia, or potential delusional thinking. It would be important to repeat this study using diagnostic information from psychological tests in order to understand the issue of specificity.

Drug Effects

In our earlier studies it seemed to us that neuroleptic agents did not account for the SPEM deviations. As we previously had noted, a significant portion of the family members of schizophrenic patients, none of whom were receiving medication, showed the tracking dysfunction. Four of six nonpsychotic patients who were receiving neuroleptic drugs did not show the SPEM dysfunction. Of four recent schizophrenics who were not receiving neuroleptic drugs at the time of testing and who had not been receiving the drugs for at least 2 weeks previous, one showed SPEM deviations. And statistical tests of the relationship between poor eye tracking and medicated patients were not statistically significant. Shagass, Amadeo, and Overton (1974) also reported no relationship, irrespective of diagnostic groups, between drug treatment and SPEM dysfunctions.

We have tested normal volunteer subjects before and after ingestion of single doses of diazepam, chlorpromazine, and secobarbital (Holzman 1975). The only drug that showed significant effects on SPEM was secobarbital. All drugs produced sedative effects that were judged to be approximately equal. Sedation, therefore, was not the principal factor producing the dysfunctions in smooth pursuit movements. We are continuing the
Table 1. Frequency of good and bad SPEM and average velocity arrests per cycle

<table>
<thead>
<tr>
<th>Group</th>
<th>Hospital diagnosis</th>
<th>Psychological test diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( N )</td>
<td>Good SPEM</td>
</tr>
<tr>
<td>Schizophrenics (recent)</td>
<td>40</td>
<td>19</td>
</tr>
<tr>
<td>Schizoaffectives</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Schizophrenics (chronic)</td>
<td>29</td>
<td>4</td>
</tr>
<tr>
<td>Relatives of schizophrenics (recent)</td>
<td>34</td>
<td>19</td>
</tr>
<tr>
<td>Manic-depressives</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>Nonpsychotics</td>
<td>19</td>
<td>15</td>
</tr>
<tr>
<td>Relatives of nonschizophrenics</td>
<td>19</td>
<td>17</td>
</tr>
<tr>
<td>Normal controls</td>
<td>72</td>
<td>66</td>
</tr>
<tr>
<td>Totals</td>
<td>228</td>
<td>150</td>
</tr>
</tbody>
</table>

1 Subjects were categorized according to hospital diagnosis and psychological test diagnosis.

Investigation of drug effects on SPEM using chronic administration of a variety of drugs in lower primates who have been trained to follow a pendulum smoothly. Studies using drugs with known neural effects may help provide a more specific physiological mapping of SPEM and their dysfunctions. We can, however, conclude that there is no evidence that the dysfunctions reported are a reflection of psychotropic medication received by the patients.

Other Effects

Both Shagass, Amadeo, and Overton (1974) and we noted an age effect—with increasing age SPEM tends to decline. In one sample of 72 normal subjects between 15 and 59, the correlation between age and velocity arrests was .36 (cf. also Miller 1975). Neither Shagass, Amadeo, and Overton (1974) nor we discovered sex differences or interactions between age and sex with respect to SPEM. A correlation between velocity arrests and peak serum CPK (creatine phosphokinase) was not significant.

**SPEM and Attention**

Is attentional dysfunction the likely explanation for SPEM dysfunctions? The task of following a pendulum requires that subjects pay attention to the target. But once the subject looks at the swinging pendulum, the pursuit system is triggered and the eyes follow the target. Occasionally a subject’s interest and motivation flag and we have therefore adopted a procedure of “re-alerting” subjects after 30 seconds of tracking. This re-alerting instruction has no significant effect on the number of eye arrests, but errors of overshooting are indeed minimized. We interpreted this to indicate that the velocity arrest score is unaffected by voluntary attention whereas errors of velocity overshooting are so affected, thus demonstrating that voluntary behavior, motivated behavior, or paying attention are not the principal
issues in the SPEM dysfunctions we described.

In a recent paper Shagass, Roemer, and Amadeo (1976) reported an interesting technique to optimize effective performance. They placed arabic numerals on the pendulum; as the pendulum oscillates it rotates slowly, thus displaying different numbers. The subject's task was to read the numbers silently as he tracked the pendulum. Shagass, Roemer, and Amadeo (1976) reported significant reductions in the number of velocity arrests and a tendency for tracking to become smoother as a result of this procedure, although the differences between patients and normals remained. We repeated Shagass, Roemer, and Amadeo's experiment (Holzman, Levy, and Proctor 1976) and obtained essentially identical results. None of the subjects in either the Shagass group's experiment or in our repetition of that work realized that their tracking was deviant or that it had improved when they were asked to read the numbers. This number-reading task can help us understand the problem of attention in schizophrenia. We have tested our subjects four times—twice in the usual way with the unnumbered pendulum, once with numbers placed on the pendulum and instructions to the subject to read the numbers silently while tracking the pendulum, and finally with the plain, unnumbered pendulum again. We have found improvement with the number maneuver both in patients and in normal subjects when they are given seconal, alcohol, or chloral hydrate. Figure 5 shows these results for patients. But the number-reading task is able to normalize only certain kinds of eye-tracking impairments. We have noted that there are two principal kinds of eye-tracking dysfunctions in our subjects. In type 1 the pursuit movements are almost totally replaced by rapid eye movements or saccades. This is illustrated in figure 1b. In type 2 small amplitude rapid movements intrude upon the smooth tracking, leaving its general form intact but giving the tracings a cogwheel appearance. This is shown in figures 1c and 1d. Reading the numbers silently while tracking dramatically improves type 1 impairment. Figure 6 shows this effect. When the numbers are removed, however, most of the subjects showing this type of dysfunction revert to gross saccadic shifting.

Subjects with type 2 dysfunctions do not generally improve with the number-reading task although there are reductions in the number of eye arrests. The extent of the changes never results in normal smooth pursuit movements. Figure 7 shows this effect. Type 1 tracking may represent a pursuit system that does not switch on easily. Type 2 may represent a pursuit system in which other interferences do not switch off. Type 1 tracking in which smooth pursuit is almost completely replaced by saccades could reflect an incapacity to begin a pursuit movement. The number-reading task, by providing a more commanding stimulus than the pendulum alone, overrides this initial sluggishness and normalizes the SPEM. For type 2 dysfunctions, the superimposed cognitive task lends only a minor degree of help to reestablish some degree of suppression of the intrusive activity by invoking another functional system. When number reading ceases and the visual inhibitory controls are again suspended, however, the earlier level of disinhibition of the saccadic system returns. This view accords with the recent evidence that effective SPEM involves inhibition of saccades (Heywood and Churcher 1971).

In a previous paper we used an analogy to help understand the phenomenon of tracking improvement with the number-reading task. Many organic defects can be overcome or circumvented by introducing other intact functions that may compensate for the dysfunction. For

Figure 5. Effects of reading numbers silently while tracking a pendulum

![Graph showing effects of reading numbers silently while tracking a pendulum.]

Note. — Velocity arrests decrease significantly for schizophrenics and return to the previous high level after removal of numbers. Differences between schizophrenics and normals remain significant for all conditions.
example, the Parkinsonian patient with an intention tremor stops or diminishes his tremor during a purposeful act, such as reaching for a wanted object. The stutterer can speak more fluently by markedly slowing his rate of speaking or by singing. These maneuvers temporarily normalize the impaired functions. But one cannot reason that the original cause of the dysfunction is the absence of such a special maneuver. Likewise, it would seem questionable to attribute SPEM dysfunctions to failures in the deployment of voluntary attention simply because they are to some extent correctable by a superimposed dominating cognitive task. It seems to us that what is involved is a failure to maintain a visual-attentive focus that is continually locked onto the oscillating pendulum, a failure of cognitive centering in spite of the intention to do the task.

At this juncture, then, we believe that a disorder of nonvoluntary attention accounts for the eye-tracking dysfunction and is thus involved in schizophrenia. Accordingly, the SPEM dysfunctions reveal not simply a disorder of the oculomotor system, but a more general central nervous system disorder. SPEM dysfunctions implicate a neurophysiological substrate of impaired nonvoluntary attention in schizophrenia. This may be described as a failure of inhibitory, synchronizing, or integrating systems that may be located in the brain stem, for (as we have noted) smooth pursuit eye movements as well as other types of eye movements are regulated in the cerebellum and in the pontine paramedian reticular formation, just ventral to the medial longitudinal fasciculi.

**Genetic Implications**

Our earlier work showed that 45 percent of the relatives of schizophrenic patients had SPEM disorders in contrast to about 10 percent of the relatives of nonschizophrenic patients. Schizophrenics and their families accounted for about 82 percent of all deviant SPEM found in that sample. Moreover, the velocity-arrest scores of the relatives were equal to those of the probands. When one used psychological test criteria, there was an even stronger association between SPEM and schizophrenia and family members of schizophrenics. To study the meaning of this familial association, we have continued to test families of schizophrenics and of other psychiatric patients. We have also conducted a study of monozygotic and dizygotic twins who were

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**Figure. 6. Effects of reading numbers silently on type 1 SPEM impairment in which saccadic shifts replace smooth pursuit**

*Note.*—(a) Poor tracking with 10 velocity arrests per cycle. (b) Normal SPEM occurs during silent number reading with 3 velocity arrests per cycle. (c) Pursuit movements return to saccadic shifts after removal of numbers from pendulum, with 11 velocity arrests per cycle.
Figure 7. Effects of reading numbers silently on type 2 SPEM impairment.

Note.—(a) Poor SPEM with 16.8 velocity arrests per cycle. (b) Improvement, but still impaired tracking during silent number reading, with 10.8 velocity arrests per cycle. (c) Return to previous poor quality of SPEM after removal of numbers, with 18.4 velocity arrests per cycle.

discordant for schizophrenia.

The evidence that schizophrenia, mania, and depression run in families has long suggested a genetic component in these disorders. Specific genetic hypotheses about the mode of transmission have not been particularly fruitful. Twin studies have established a concordance rate to schizophrenia between 30 percent and 40 percent for monozygotic twins and between 8 percent and 10 percent for dizygotic twins. Adoption studies have found that the number of schizophrenic offspring of biological parents who are schizophrenic is approximately the same whether or not those children have been reared in adoptive or biological homes. Although these and other studies have established a genetic component in the propensity to become schizophrenic, they have left ambiguous the answers to questions about the mode of transmission, what is transmitted, and the moderating or inhibiting effects of environment or constitution. A significant handicap in attempting to map an alleged genetic transmission of schizophrenic predisposition is the absence of an objective indicator of schizophrenia that can be reliably reproduced and associated with schizophrenia and that does not require clinical assessment of schizophrenic behavior itself. The presence of SPEM dysfunctions in the families of schizophrenic patients suggests that SPEM dysfunctions may qualify as a genetic indicator of the predisposition for schizophrenia.

Twin Data

To test whether SPEM disorders might be a genetic indicator, we examined the concordance of poor eye tracking in monozygotic (MZ) and dizygotic (DZ) twins who were discordant for schizophrenia. Kringlen (1967) had previously studied such a group. In that study concordance figures for schizophrenia ranged from 25 to 38 percent in MZ twins and from 4 to 10 percent in DZ twins, depending upon whether a strict concept of schizophrenia was employed. That sample of twins with its low clinical concordance seemed ideally suited for exploring the concordance of disordered eye tracking. We were fortunate indeed in having Dr. Einar Kringlen as a collaborator in this study, a description of which can be found in Holzman et al. (in press).

We selected from among Kringlen's sample those twin pairs who were available for testing in 1975. Twins were tested without knowledge of their zygosity, diagnosis, or relationship to each other. The identities of the
Figure 8. SPEM records of two sets of twins

Note.—The first record (a) of each pair is that of the proband (i.e., 5005 and 1511); the second record is that of the co-twin (i.e., 8826 and 6142). The figure illustrates concordance in SPEM patterns within twin pairs.

Subjects were retained by Dr. Kringlen. We recorded the eye movements with a differential preamplifier driving an instrumentation magnetic recorder. Most subjects were tested in their own homes which were located throughout the southern half of Norway. After all the data had been gathered, the tapes were sent to the United States where the signals were then played back through a polygraph for visual analysis and scoring. Three people scored the records independently for good or bad tracking and for the number of velocity arrests per cycle. On a prearranged date these scores were sent to Norway to Dr. Kringlen, who on the same date sent to Chicago the names, relationships of the subjects, and zygosity determinations. The data could be evaluated and processed only after this mutual exchange of codes and scores had taken place.

Eleven sets of MZ and 15 sets of DZ twins were tested. Of the MZ pairs, two sets were concordant and two sets were partially concordant for schizophrenia. One pair of DZ twins was partially concordant for schizophrenia. Our results showed that 68 percent of the schizophrenic persons in this sample had disordered SPEM. This prevalence is within the range of our previous findings. Within the discordant co-twins, 54 percent showed disordered SPEM, which is similar to our previous findings of about 45 percent of the first-degree relatives of schizophrenics who showed deviant SPEM.

A Pearson product-moment correlation between twin pairs (with respect to the quantitative index of SPEM) and the velocity arrest scores is .77 for MZ twins ($p < .005$) and .40 ($p = .15$) for DZ pairs. These values are reasonably close to those theoretically predicted for genetic association, which would be 1.00 and .50, but because of small sample size and large variances, they are not significantly different from each other.

With respect to the concordance on the qualitative typing of eye tracking, whether A or B, 5 of 7 (71 percent) of the MZ sets, and 7 of 13 (54 percent) of the DZ sets were concordant for poor eye tracking, using pairwise concordance. The concordance rates for clinical schizophrenia in these samples are 18 to 36 percent for MZ and 0 to 13 percent for DZ twins, depending upon the broadness of the diagnostic criteria. Figure 8 shows examples of SPEM concordance in each of two sets of twins. The tracking patterns are strikingly similar within twin pairs. The data thus show a considerably higher concordance for poor eye tracking than for clinical schizophrenia.

Within the MZ sets there is a significant tendency for tracking performance to be congruent; that is, probands with good or bad tracking tend to have co-twins with the same kind of tracking (Fisher's Exact Test, $p < .045$,
Table 2. Prevalence of good and bad SPEM in probands and their first-degree family members

<table>
<thead>
<tr>
<th>Groups according to diagnosis</th>
<th>Eye-tracking type</th>
<th>Proband</th>
<th>First-degree family members by eye-tracking type</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Mother</td>
</tr>
<tr>
<td></td>
<td></td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>10</td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>B</td>
<td>15</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>Nonschizophrenic psychosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>7</td>
<td></td>
<td>2</td>
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<td>B</td>
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<td>Nonpsychotic</td>
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</tr>
<tr>
<td>B</td>
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<td>0</td>
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<tr>
<td>Normals</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>A</td>
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<td></td>
<td>8</td>
</tr>
<tr>
<td>B</td>
<td>0</td>
<td></td>
<td>0</td>
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one-tailed). But within the DZ sets that tendency is no greater than chance. The significance levels are restrained by the small sample size. It is worth noting, moreover, that there were two DZ probands who had good tracking, but whose nonschizophrenic co-twins had bad tracking, suggesting that SPEM dysfunctions may not necessarily be an outcome of clinical schizophrenia or the treatment of that disorder. Although the small number of twin pairs limits the extent to which one can validly draw firm conclusions, the concordance rates for qualitative classification, as well as the quantitative correlations, are in the correct direction for genetic effects, but these results would also be consistent with environmental influences, such as viral or toxic factors. Another restraint on interpretation is the age characteristics of the twin sample. Their average age is 55. We have seen that SPEM declines with age, and therefore replication of this study is required with a younger group of discordant twins.

**Family Data**

We have continued to gather data on the first-degree family members of schizophrenics and of other patients. Table 2 contains the distribution of normal and deviant SPEM among these samples. The association between poor eye tracking in families of schizophrenics continues to show itself. Where deviant SPEM occurs in the schizophrenic proband, there is a tendency for a familial association with poor SPEM. We are continuing to gather data but have not yet felt that we have sufficient evidence to formulate definite conclusions about the viability of SPEM dysfunctions as a genetic indicator. Continued investigation is nevertheless warranted.

**Conclusions**

This brief review has presented evidence that disorders of SPEM are associated with schizophrenia, and quite likely with other functional psychoses as well as with some central nervous system disorders. The effects are not drug related in the psychiatric population we have studied. A large proportion of first-degree family members also show similar SPEM dysfunctions, and there is a significant concordance among MZ twins who are discordant for schizophrenia. The dysfunction does not appear to be a reflection of the psychosis or its treatment. Nor is it likely to reflect motivational factors or a failure of voluntary attention. It is not a predictor of later psychosis, nor can it be used as a specific diagnostic sign of schizophrenia. Rather it represents an association with a predisposition to some functional psychosis; the predisposition shows itself in a dysfunction of nonvoluntary attention, a disorder of cognitive centering, which has a neurophysiological
substrate not only in the form of eye pursuit dysfunctions, but in hitherto unspecified neural noise.

References


Slatt, B.; Loeffler, J.D.; and Hoyt, W.F. Ocular motor disturbances in Parkinson's disease: Electronystagmographic observations. *Canadian Journal of Ophthal-
selected mental health audiovisuals

A 233-page catalog of currently available films, filmstrips, audiotapes, and videotapes in 27 major mental health categories, Selected Mental Health Audiovisuals, has been published by HEW's National Institute of Mental Health, of the Alcohol, Drug Abuse, and Mental Health Administration. The catalog is designed to aid persons who are engaged in educating both public and scientific audiences about mental illness and mental health, and who need a quick reference source of nonprint materials that can be disseminated.

Approximately 2,300 audiovisuals are listed and described. Subjects covered include aging, animal studies, biochemistry and metabolism, child mental health, cognition and perception, communication, community mental health, crime and delinquency, cultural studies, death and suicide, depression, education, family, group processes, learning, mental retardation, minority groups, motivation, neurosciences, personality, psychology, religion, schizophrenia, sexology, sleep and dreams, social issues, and treatment. Also included in the catalog is a list of sources for free social welfare films, sources for low-cost film rental, and commercial rental libraries. Each listing gives the name of the distributor, his address and phone number, the title of the audiovisual material, the date it will be available, and a short description. The materials are available directly from the distributor, not from the National Institute of Mental Health.