Smooth Pursuit Eye Movement Dysfunction in Chronic Schizophrenics and Their First Degree Relatives

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This study had been done on 15 medicated chronic schizophrenics, 12 of their first degree relatives and 12 normal subjects. Smooth pursuit eye movements (SPEM) study was the target of this work. The results showed that schizophrenics definitely do poorly on this task. Incidence was around 90% on most of SPEM measurement parameters. First degree relatives of schizophrenics showed nearly similar performance to the sample of normal people. SPEM dysfunction in schizophrenics was not correlated to the age of patient, age of onset of illness, duration of illness, nor to the degree of neuropsychological impairment. Various possibilities of the interpretation of SPEM dysfunction in schizophrenia was discussed and a conclusion of the selective attention impairment is most probably behind this dysfunction was reached.

(Egypt.J. Psychiat.,1994,17:193-202).

Introduction

Smooth pursuit eye movements (SPEM) are the slow movements that are used to follow a continuously moving object as contrast with the high velocity saccadic movements (shifts) that fixate the image of an object onto the fovea (Nuechterlein and Asarnow, 1989). The presence of the moving stimulus is essential for the SPEM to occur. Any attempt to move the eyes at a slow velocity in the absence of a target results in a saccade. In man, the SPEM system is most efficient at low target velocities with a gain (eye velocity / target velocity) of approximately 0.9. This ratio declines with increasing velocities of the target (Sweeney et al, 1992).

The control of SPEM is distributed widely in the brain areas (brain stem, cerebellum and motor cortex) (Bender, 1980), so it is difficult to use its impairment as a localizing sign in CNS diseases.

SPEM impairment can occur in normal people in case of fatigue, distraction or uncooperativeness and it is estimated that nearly 8% of normal people exhibit SPEM abnormalities (Holzman, 1987).

Very early in the history of schizophrenia in 1908 patients were found to have inaccurate pursuit eye movement (Mather and Putchat, 1982). In relatively recent studies, Holzman et al (1973) confirmed this finding and reported the interruption of slow SPEM by saccadic eye movements. The same authors reported the abnormality in first degree relatives of schizophrenics more than in general population. Since that time, a postulation that this abnormality could be a genetic marker for schizophrenia was put. As regards the incidence of this abnormality, Holzman (1987) had reported that 50% to 85% of schizophrenics show this abnormality. Around 40% of the

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first degree relatives of schizophrenics show it in comparison to only 8% of the normal general population.

Research with twin pairs who were chosen because one twin had schizophrenia showed that concordance for poor SPEM is higher among MZ twins than DZ twins. This finding supports the genetic transmission of the eye tracing dysfunction. It was also found that SPEM dysfunction is found in relatively remitted schizophrenics, i.e. it is a trait marker (Nuechterlein and Asamow, 1989).

The effect of neuroleptics on SPEM has been studied extensively. The cumulative results of the various works provided strong evidence of no effect of neuroleptics on the SPEM (Holzman et al., 1975; Levy et al., 1983; Siever et al. 1986).

Despite the numerous studies confirming the presence of SPEM dysfunction in schizophrenics and their relatives, there has been little study of the relationship between this dyfunction and many of the clinical and neuropsychological variables in schizophrenia. An example of these studies is that of Sweeny et al., (1992) who examined the correlation between SPEM and neuropsychological tests of frontal and temporal lobes functions. They found a correlation between impairment of these functions and poor SPEM. Our hypothesis is that it is possible to find a correlation between cognitive impairment in schizophrenia as manifested in WAIS and intellectual processing scale of Luria - Nebraska battery and the SPEM. So the aim of this work was: (a) to study SPEM in Egyptian schizophrenic patients and their first degree relatives; (b) to find out the relationship between SPEM dysfunction, if any, and some of the clinical and neuropsychological variables. The WAIS and intellectual processing scale of Luria - Nebraska Battery.

Subjects and Method

Fifteen chronic schizophrenic subjects, 13 of their first degree relatives and 12 controls were included in this work. The patients and their first degree relatives were recruited from Kasr El-Aini outpatient clinic during the period of May to July 1993. Diagnosis was made by two psychiatrists using DSM-III-R criteria. Patients were subjected to the following:

(a) Clinical interview with the aim of

i) Diagnosis according to DSM-III-R

ii) Determining approximately the age of onset of the illness.

iii) Determining approximately the duration of the illness.

(b) neurological examination; (c) visual assessment; (d) neuropsychological assessment (Wechsler Adult Intelligence Scale) (WAIS) and Intellectual processing scale of Luria-Nebraska Battery (IPS-LNB); (e) smooth pursuit eye movement assessment. They were selected to fulfill the following criteria: (i) neurologically free; (ii) no visual problems; (iii) cooperative to a good extent to complete the SPEM test and neuropsychological tests. Patients were on their conventional neuroleptic treatments.

Relatives were the parents and sisters and brothers of the patients who agreed to participate in the research and were assessed prior to the testing by psychiatric interview to exclude any diagnosable mental disorder. No visual or neurological problems were also present in this group. Control group were selected from audiology clinic from people coming for other reasons than vestibular problems.

Relatives group was not matched with patients group as regards age because the relatives group included parents of the patients group. Control group was selected to match with patient group as regards age.

The three groups were not matched with each other as regards sex or social class because these factors are reported in the previous researches not to affect the SPEM. (Holzman et al., 1987). The schizophrenic patients, were of different types and of different duration of illness and different ages.

SPEM tracking was done in the audiology unit of Kasr El-Aini by one of the authors (Shabana, M.). WAIS and IPS-LNB were done in the clinical psychology unit of psychiatry clinic.

Eye movement testing procedure:

Subjects were tested in a quiet darkened room. Targets were presented as moving dots horizontally in front of the subject who sits on one meter distance from the target screen. Subjects were actively encouraged to focus his attention on the moving target (light dot) and the performance was monitored simultaneously, so that the subject could be realerted if he stops tracking the target. Chin, forehead and head supports were used to minimize head movements. Eye tracking was evaluated by the use of an electro-ocullograph (EOG) which records the eye position while the subject follows the moving object. To begin the test, we had to perform calibration. The purpose of this calibration is was measure the strength of the EOG signal coming through the electrodes against known calibration angle, i.e., the EOG signal needs to be standardized for each subject. The subject is then asked to look back and forth between two targets that are at known angle. The strength of the resulting signal is then divided by the angle that the eye actually moves to give calibration rates. This ratio is then used in the actual testing and analysis and equate the amplitude in degrees of movement.

The velocity of the moving dot is changed in three different velocities 10, 20, 30. Then the results are analyzed for each testing frequency at different velocities. Several measures are obtained according to Sweeney et al, (1992).

(1) **Phase:** *This is measured in degrees.* Positive phase indicates that the degree to which the patient is ahead in tracking the target. The converse is true of the negative phase.

(2) **Pursuit gain:** The primary function of pursuit eye movement is to match the angular velocity of the eye to that of the slowly moving objects. The accuracy of this velocity match is referred to as the 'gain' of pursuit eye movement and is calculated as the ratio of average pursuit eye movement velocity over target velocity. The extent to which gain ratio drops below 1.0 indicates the severity of pursuit eye movement disturbance.

(3)**Saccadic eye movements:** an evaluation of saccadic eye movements that interrupt SPEM provides further information about visual tracking performance. There are three types of saccades: (a) **Catch up saccades.** These are corrective rapid eye movements that compensate for pursuit error by abruptly shifting the eyes towards the target. The frequency and size of catch up saccades typically increase as pursuit gain decreases, reflecting the need for more frequent and longer corrections for pursuit tracking error. (b) **Square wave jerks:** These are intrusive saccades that disrupt pursuit by shifting the focus of the eyes away from the target. During pursuit, square wave jerks the eyes away from the target for up to 450 msec, during which time pursuit continues until a second saccade refocuses on the target. (c) **Anticipatory saccades.** These are saccadic movements that move the eye ahead of the target after which pursuit velocity is immediately and markedly reduced. These movements are considered

anticipatory because the eyes appear to suddenly move ahead to where the target is expected and then wait for it to arrive at the anticipated location.

As regards neuropsychological assessment, all patients were subjected to WAIS (Arabic version) and the IPS-LNB (Arabic version) (Raslan, 1989). The data were subjected to statistical analysis.

Results

Sociodemographic and clinical data: The number of subjects was 15, out of them only three were females. The diagnostic subtypes included three paranoids, seven disorganized and five undifferentiated. Control group included two females. The first degree relatives group included four females.

Mean age of patients was 29.9±8.6, while mean age of relatives group was 40.9±14.4 and the mean age of normal control group was 28.5±7. It was found that patients group was matched as regards age with normal control group, but not matched with relatives group (because of the inclusion of parents of

Table 1

Results of SPEM of Different Groups: Comparison Using ANOVA Test

	Patient Group (n= 15)		Relatives Group (n= 13)		Normal Control Group (n= 12)		F	P
	Mean	±S D	Mean	±S D	Mean	±S D		
Phase	0	2.3	0	1.5	0.3	1.2	1.4	>0.05
Right gain in 10 dls	0.4	0.12	0.65	0.08	0.75	0.1	34.9	0.000**
Right gain in 20 dls	0.3	0.12	0.62	0.1	0.69	0.1	26.9	0.0001*
Right gain in 30 dls	0.3	0.13	0.6	0.11	0.66	0.1	23.0	0.0001*
Left gain in 10 dls	0.4	0.13	0.66	0.1	0.74	0.07	29.3	0.0001*
Left gain in 20 dls	0.4	0.13	0.63	0.11	0.7	0.07	22.7	0.0001*
Left gain in 30 dls	0.38	0.14	0.62	0.12	0.67	0.08	20.7	0.000*
No. of catch up saccades	52.0	20.3	53.6	16.8	43.8	14.3	1.0	0.3
Size of catch up saccades	16.2	27.9	5.5	6.4	4.6	2.5	1.6	0.2
Anticipatory saccades	9.4	4.4	3.2	0.7	2.1	1.4	23.5	0.0001*
Square wave	8.5	8.9	0.9	1.1	3.3	2.2	5.8	0.002*

* Significant difference between patient group and both relatives and normal control group.
* Significant difference between the three groups.

Table 2

Assessment of Correlation Between SPEM Parameters and Clinical Data (Correlation Coefficient Test)

	Age		Age of onset		Duration of illness		Years of education	
	r	p	r	p	r	p	r	p
Phase	0.2	0.4	0.1	0.5	0.2	0.5	0.1	0.7
Right gain in 10 dls	0.3	0.2	0.3	0.2	0.2	0.5	0.4	0.1
Right gain in 20 dls	0.3	0.2	0.3	0.2	0.2	0.5	0.3	0.2
Right gain in 30 dls	0.3	0.2	0.3	0.2	0.2	0.5	0.4	0.1
Left gain in 10 dls	0.3	0.2	0.3	0.2	0.2	0.5	0.3	0.1
Left gain in 20 dls	0.3	0.2	0.3	0.2	0.2	0.5	0.4	0.1
Left gain in 30 dls	0.2	0.2	0.3	0.3	0.2	0.5	0.4	0.1
No. of catch up saccades	0.1	0.5	0.1	0.6	0.1	0.6	0.1	0.5
Size of catch up saccades	0.2	0.4	0.4	0.08	0.2	0.4	0.5	005*
Anticipatory saccades	0.1	0.5	0.2	0.5	0.1	0.6	0.4	0.1
Square wave	0.2	0.4	0.2	0.3	0.1	0.7	0.5	0.05*

* Just statistically significant.

some patients in this group). Patients started their illness at young age (22.6±6.3 years). The duration of illness is relatively long (range 2-20 years; mean 7.2±4.4 years). Educational level was good (range of years of education 0-16; mean 9.7±4.4 years). Only one patient was illiterate.

Neuropsychological results: It was noted that most of the schizophrenics in this study had performed on WAIS in the below average range. Verbal IQ was (84.2±19), Performance IQ was (81.7±18). Full scale IQ was 82.3±18. Deterioration index was 11.1± 11.1. The mean of the intellectual processing scale of Luria - Nebraska battery was 79.9± 10.9.

SPEM results are shown in Table 1. Patients showed marked impairment in different tests of SPEM except for the size and number of catch-up saccades as well as the phase. They did poorly on the three different target velocities and on both sides.They showed significantly more square waves than both groups. They showed also more catch up saccades and of higher size than both

Table 3
Assessment of Correlation Between SPEM Parameters and WIAS and Intellectual Scale of LNB (Correlation Coefficient Test)

	Verbal IQ		Performance IQ		Full Scale IQ		Deterioration Index		Intellectual Processing Scale	
	r	p	r	p	r	p	r	p	r	p
Phase	0.2	5.0	0.03	0.9	0.2	0.5	0.3	0.2	0.4	0.2
Right gain in 10 dls	0.3	0.9	0.08	0.8	0	9.0	0.3	0.2	0.5	0.04*
Right gain in 20 dls	0.4	0.9	0	0.9	0.1	0.8	0.3	0.2	0.5	0.07
Right gain in 30 dls	0.01	0.9	0	0.9	0	0.9	0.3	0.2	0.5	0.06
Left gain in 10 dls	0	0.9	0	0.9	0	0.9	0.4	0.1	0.5	0.05
Left gain in 20 dls	0.03	9.0	0.1	0.8	0	0.9	0.4	0.2	0.5	0.04*
Left gain in 30 dls	0.4	0.05	0.1	0.7	0	0.9	0.4	0.2	0.5	0.03
No. of catch up saccades	0	0.9	0.1	0.7	0.1	0.7	0.2	0.4	0.3	0.1
Size of catch up saccades	0.3	0.2	0.2	0.3	0.3	0.3	0.5	0.1	0.6	0.02*
Anticipatory saccades	0.2	0.4	0.1	0.5	0.2	0.6	0.4	0.1	0.6	0.03*
Square wave	0.3	0.3	0.2	0.5	0.2	0.25	0.4	0.1	0.6	0.01

* Statistically significant.

groups, but did not reach statistically significant levels. The frequency of abnormalities (Table 6) was very high in patients group as around 90% of patients showed abnormal gains compared to around 25% of first degree relatives and 16% normal control group. It is observed also in Table 3 that first degree relatives did not differ significantly from normal controls in all tests except the right gain at 10 dls velocity.

Correlation study results are shown in Table 2 and Table 3. This study failed to find correlation between the illness variables (age of onset, duration of illness), sociodemographic variables (age, years of education), or the neuropsychological functioning results and the SPEM dysfunction in schizophrenia. The only significant correlation was between IPS-LNB and some items of SPEM. These are right and left gain at

Table 4
Frequency of Abnormal SPEM in Different Groups

	Accepted Norms	Patient Group (n= 15)		Relaties Group (n= 15)		Normal Control Group (n= 15)	
		No.	%	No.	%	No.	%
Phase	0-+2	7	46	4	33	2	16
Right gain in 10 dls	0.7	14	93	3	25	2	16
Right gain in 20 dls	0.6	13	86	3	25	1	8
Right gain in 30 dls	0.6	14	93	3	25	2	16
Left gain in 10 dls	0.7	14	93	3	25	2	16
Left gain in 20 dls	0.6	14	93	3	25	2	16
Left gain in 30 dls	0.6	14	93	3	25	2	16
No. of catch up saccades	40-50	7	46	7	58	3	25
Size of catch up saccades	2-3	5	33	2	16	4	33
Anticipatory saccades	1-2	13	86	7	58	3	25
Square wave	3-4	8	53	0	0	1	8

10 dls velocity (p= 0.05), left gain at 20 dls and 30 dls and size of catch up saccades (p= 0.02), anticipatory saccades (p= 0.03) and square wave (p= 0.01).

Discussion

This study was done on a small group of schizophrenics who were relatively young and with chronic duration of illness. The means of ten parameters (out of eleven) of SPEM were significantly different in schizophrenics and normal controls. The frequency of abnormalities in schizophrenics was between 33% to 93%. This is comparable to most of the studies in SPEM (Shagass et al., 1974; Holzman et al., 1974; Lipton et al., 1983). In a review study, Holzman (1987) estimated that between 50% to 85% of schizophrenics do poorly on SPEM. The wide range of frequency of SPEM dysfunction in schizophrenia could be attributed to the different methods both in the technique (starting with the pendulum in the early studies (Shagass et al., 1974; Holzman et al., 1974) to the advanced computerized techniques) and the method of assessment of SPEM track. In our study we followed a recent one adopted by Sweeney et al., (1992). Also, the definition of norms

differs from one study to another, our norms is that adopted in Kasr El-Aini audiology unit.

Similarly, the results of normal people control was in agreement with most previous studies (Siever et al., 1986; Holzman, 1987). Results of first degree relatives were doing perfectly in most of the parameters of SPEM and their means did not show significant difference from that of normal controls. In ten parameters (only one parameter was different-right gain in 10 dls and this is in significant in the matrix of whole results). This finding does not confirm the suggestion the SPEM dysfunction is a genetic marker in schizophrenia. However, the denial of this suggestion cannot be based on this limited number of first degree relatives in our study and a recommendation for inclusion of big number of first degree relatives is suggested.

This work tried to find a correlation between some sociodemographic and illness variables and the SPEM dysfunction. It was found the SPEM is not correlated to the age of the patient, nor age of onset of schizophrenia, nor duration of illness. This means that the impairment in SPEM starts from the start or may be before the illness and it is not a matter of aging or chronicity of the illness. Our results are in agreement with most of the previous studies in this area (Holzman et al, 1974; Siever et al, 1986; Holzman, 1987; Sweeney et al, 1992).

As regards the effect of neuropsychological impairment of SPEM, Sweeney et al (1992) found that the patients who did poorly on neuropsychological tests, that assess the prefrontal and left temporal areas, had more impairment on SPEM testing. Our results did not show such a correlation between any neuropsychological variables (WAIS or IPS of LNB) and the degree of performance in SPEM. This could be due to our use of tests that measure the whole intellectual functions and not choosing tests that assess specific brain regions.

Etiology of SPEM dysfunction in schizophrenia is still a matter of debate. There are several possibilities that could explain it. The first possibility is that schizophrenics usually do poorly on any test that requires their cooperation and motivation. To rule out this possibility every researcher in schizophrenia, motivates his patients, to do their best and possibly do not include patients who fail to cooperate and this is what was done in our study. However this possibility remains in the mind of any interpreter of schizophrenic results on a test that requires cooperation and motivation.

The second possibility is the effect of medicine (neuroleptics). In our study, all were on their conventional treatments. This is because, nearly all previous studies had confirmed that neuroleptics on conventional therapeutic doses do not impair SPEM (Holzman et al, 1975; Levy et al, 1983; Mialet and Pichot, 1981; Siever et al, 1986).

The third possibility is that SPEM dysfunction results from a gene or genes that are associated with schizophrenic illness. This explains, why first degree relatives show higher SPEM dysfunction than general population. This also explains the higher concordance rate of SPEM dysfunction in MZ twin schizophrenics than dizygotic twin schizophrenics (Holzman, 1987). However, our study did not give support for this hypothesis.

The last possibility that makes sense is that SPEM dysfunction results from a type of impaired involuntary attention in schizophrenics. This is a type of attention that has inhibitory function to the insignificant extraneous stimuli allowing the person to follow his task uninterrupted. This ability of selective attention is possibly impaired in Schizophrenic patients and makes them

follow the target in a poor way in spite of being motivated and cooperative. So, our conclusion is that schizophrenics in a high frequency, show poor SPEM, and this is mostly related to their poor selective attention abilities. But, because of the small number of the sample and the selection criteria, we should be cautious of the generalization of these results.

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مقياس وكسلر بلفيو لذكاء الراشدين تأليف الدكتور/ ديفيد وكسلر، أقتباس
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Dysfonction du Movement Poursuit Regulier des Yeux Ches les Schizophreniques Chroniques et Ches Leurs Parents du Premier Degré

Cette étude comprend 15 schizophreniques chroniques prennants des medicaments, 12 de leurs parents et 12 personnes mormaux. L'étude de (SPEM) est le but de ce travail. Les résultats ont démontré que definitivement les schizophreniques performent mal sur ce devoir, avec une incidence de 90%. Les parents ont démontré la même performance que les personnes normaux. Il n'éxistait pas une correlation entre la dysfonction de (SPEM) et l'âge des patient, la durée de la maladie, ou le degré de la détérioration neuropsychologicale.

خلل الحركة السلسة للعين فى مرضى الفصام المزمن وأقاربهم من الدرجة الأولى

أجريت هذه الدراسة على عينة مختارة تتكون من خمسة عشر مريضا فصاميا مزمنا وتحت العلاج. وكذلك أثنى عشر من أقاربهم من الدرجة الأولى، وأثنى عشر شخصا طبيعيا، كان الهدف الرئيسى هو دراسة حركة العين المتابعة السلسة.

أظهرت النتائج أن مرضى الفصام يعانون من ضعف الأداء لإختبار حركة العين المتابعة السلسة. وكانت نسبة تواتر هذا الضعف بين المرضى حوالى تسعين فى المائة. كان أداء الأقارب نو الدرجة الأولى متقاربا من الأفراد الطبيعين، لم تكن ثمة علاقة أو إرتباط بين الاضطراب على إختبارات حركة العين المتابعة السلسة وعمر المرضى أو عمرهم عند بداية المرض أو مدة المرض أو درجة الاضطراب على إختبارات الوظائف النفسية.

تمت مناقشة الإحتمالات لتفسير اضطراب حركة العين المتابعة السلسة فى مرض الفصام وتم توضيح إحتمال أن ضعف الإنتباه المختار هو فى الغالب وراء هذا الاضطراب.