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MULTIVARIATE STRUCTURE OF EYE MOVEMENT DYSFUNCTION DISTINGUISHING SCHIZOPHRENIA

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ABSTRACT

The study examined processes associated with eye-movement dysfunction (EMD) maximally discriminating paranoid and nonparanoid schizophrenic subjects from psychiatric and normal controls. Measures included the linear slope of saccade number over velocities of a ramp visual target (a measure shown effectively to distinguish schizophrenia-related EMD), along with several other measures of perceptual/cognitive processes potentially contributing to oculomotor tracking. The first significant dimension, of a multiple discriminant function analysis, was one of "EMD as related to spatial memory". Measures of the latter reflected efficiency in encoding properties of a visual stimulus array into a task-facilitative format. The paranoid schizophrenic subgroup had the highest scores on this dimension, normals, the lowest, and depressed psychiatric controls and nonparanoid schizophrenic patients, intermediate. A second significant dimension was one of "manual motor speed in following a ramp visual target". Depressives were slowest, and nonparanoid schizophrenic patients fastest. Speed of the nonparanoid patients was ascribed to extrapyramidal effects, especially tremor and akathisia. The results were discussed with respect to information processing involved in oculomotor tracking, and the effects of medication, or dyskinesia, on manual motor tracking.

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Studies of eye movement dysfunction (EMD), and those of dysfunctional memory, cognition and perception, by and large have represented parallel avenues of investigation (see e.g., Holzman, 1985; Spaulding & Cole, 1983). Recently, students of oculomotor control have contended that the processes involved in visually tracking a moving target, for example, overlap with those of perceptual and memory operations, including the transaction of traditional visual search and related tasks (Khurana & Kowler, 1987). Steinman (1986) emphasizes the need to consider the highest "cognitive" level whenever we evaluate control of eye movements. The assessment of EMD in terms of possible cognitive-perceptual correlates may inform us as to the functions involved in oculomotor control, and suggest sources of abnormalities.

The present study reflects an interest in the configuration of cognitive-behavioral processes defining EMD related to schizophrenia. The procedure is not unlike psychometrically defining the structure of a measure in terms of its association with other measures, often as is done using factor analytic methods. In the present case, however, multivariate methods addressed specifically the structure of measurement variance involved in the separation of target diagnostic groups.

It was important therefore to select as a representative from available measures of oculomotor control one which discriminates well EMD as related to schizophrenia. The measure of choice comprised the slope of the linear function relating the number of saccadic eye movements to the velocity of a ramp visual target. The measure effectively discriminates between schizophrenic subjects and others. An estimated 75% of schizophrenic subjects produce a negative slope, while 90% of nonschizophrenic psychiatric controls and 94% of nonpatient controls produce a positive slope. Furthermore, differences in linear trend account for 99% of the between-groups variance in saccade-number change over target velocities (Mather, Neufeld, Merskey, & Russell, 1992).

An additional reason for investigating the covariation among oculomotor-control and cognitive/perceptual measures with respect to inter-group discrimination, is as follows. Recently, EMD has been used as a behavioral phenotypic measure in constructing a mathematical model of heritability of schizophrenia (Holzman, Kringlen, Matthyse et al., 1988; Matthysse, Holzman, & Lang, 1986). The structure of this heuristic model (Matthysse & Holzman, 1989; McGue & Gottesman, 1989) expresses a Mendelian dominant-gene mechanism of transmission of a latent trait that tends to be manifest behaviorally as schizophrenia, EMD or both. It seems important to describe further the expression of the tentative latent trait by exploring related cognitive/perceptual processes.

Several processes were considered potentially to reflect components of tracking performance. Among others, these included "visual-spatial management" (Flach, Kaplan, Banglesdorf, Orlowski & Carmody, 1990). Subjects were given a spatial-memory task (based on Smith & Milner, 1981) which required the locations of an array of stimuli; they were required also to bisect lines of various lengths presented in various locations within the viewing field (Bowers & Heilman, 1980). Two other tasks thought to impinge on oculomotor tracking included stimulus tracking using a non-ocular response modality (manual-motor tracking),

and oculomotor reaction time to a suddenly changing target (Iacono, Tuason, & Johnson, 1981; Mather & Putchat, 1983).

In this study, then, we measured oculomotor control, as well as performance on putatively related tasks, among paranoid and nonparanoid schizophrenic patients, and psychiatric and nonpatient controls. Measures were submitted to a version of multiple discriminant function analysis originally developed by Cooley and Lohnes (1971) and Veldman (1967). The analytical method identifies the number and nature of orthogonal dimensions separating the groups. The dimensions ("discriminant dimensions") successively maximize the variance accounted for among the groups, with respect to the measurement battery. The method is not a stepwise procedure, thus avoiding the tenuous validity of a final solution developing from successive predictor selection (Derksen & Kesselman, 1992; Lees & Neufeld, 1994). It comprises, instead, simply the eigen-decomposition of the matrix product $(\boldsymbol{P}^{pxp})^{-1}\boldsymbol{A}^{pxp}$, where $\boldsymbol{P}^{pxp}$ is the within-groups matrix of deviation-score cross products, and $\boldsymbol{A}^{pxp}$ is the between-groups counterpart, p being the number of measures entering the analysis.

The procedure is constrained to throwing into relief the most salient aspects of inter-group variation. Of principal interest are the structures of the discriminant dimensions, as defined by their associated measures, and the degree to which the respective structures characterize the individual groups.

If oculomotor control involves selected cognitive/perceptual processes (Steinman, 1986), then EMD may be expected to be related to deficits in cognitive/perceptual performance as tapped by the present measures. A discriminant dimension reflecting cognitive/perceptual processes conjoint with EMD, in turn, especially may be indicative of paranoid schizophrenic diagnostic status; these subjects in particular have displayed deficits in encoding spatial stimulus properties (Magaro, 1983; Neufeld, 1991), deficits possibly detracting from normal visual tracking of a moving target (e.g., Khurana & Kowler, 1987). The spatial memory task described above, for example, requires specifically the encoding and rehearsal of a visual array. Retardation in stimulus encoding, associated with paranoid schizophrenia (Neufeld, Vollick, & Highgate-Maynard, 1993) should reduce opportunities for rehearsal during the limited time of array presentation (Neufeld & Williamson, in press). To the degree that such encoding processes overlap with those involved in "eye tracking" abnormality, discriminant dimension structure(s) should evince an association between spatial memory and eye tracking performance, characterizing especially paranoid schizophrenic patients.

METHOD

Subjects

Nonpatient control subjects (n=16) were recruited from a pool of "mature summer school students" in Psychology at the University of Western Ontario. Schizophrenic patients (n=24) and depressed (n=10) psychiatric control subjects were selected from the inpatient population of the London Psychiatric Hospital, London, Ontario. All subjects were right-handed by Olfield's (1971) test, and between 18 and 50 years of age. They had a minimum of Grade 8 education, an estimated Weschler Adult Intelligence Scale Vocabulary IQ (Paitich & Crawford, 1970) of 80 or greater, and no indications of prior drug dependency or alcoholism. Cumulative hospitalization did not exceed 3.5 years, and history of brain

pathology and electroconvulsive therapy within 6 months prior to testing both were negative.

Patient groups were selected by clinical diagnosis and Research Diagnostic Criteria (RDC). The latter were established by systematic interview using the Symptom-Sign Inventory (Foulds, 1965; George & Neufeld, 1987). Diagnoses were paranoid schizophrenia (n=15), nonparanoid schizophrenia (n=9) and unipolar depressive affective disorder (n=10). Demographic characteristics of these groups are presented in Table 1. Significant differences in WAIS-Clarke IQ occurred between the nonpatient controls and the respective psychiatric groups ($p < .05$), the psychiatric groups not differing significantly from one another. The paranoid and nonparanoid schizophrenic patients each exceeded the psychiatric controls in chlorpromazine (CPZ) equivalence drug dosages ($p < .05$), but did not differ significantly from each other. A similar pattern of inter-group differences held for medication for neuroleptic side effects (in benztropine-equivalence dosages).

With respect to side effects themselves, Tardive Dyskinesia (TD; see Spohn, Coyne, Lacoursiere, Mazur & Hayes, 1985), as measured on the Abnormal Involuntary Movement Scale (AIMS; NIMH, 1974), was higher for the nonparanoid schizophrenic patients ($x = 0.50$), than for the paranoid schizophrenic patients or psychiatric controls ($x = 0.14$ and 0.17, $p < .05$). The scale was administered by the fourth author, blind to patient diagnosis. Full-scale AIMS scores were employed, rather than subdivisions of TD, in the face and extremities. Secondary analysis of data reported by Spohn et al. (1985) indicated that the subdivisions are parallel or statistically equivalent measures of a common dimension: differences in correlations with a variety of other measures generally were nonsignificant (and invariably so when Bonferroni adjustment for Type-I error was employed; Larzelere & Mulaik, 1977), while the subdivision scores were moderately and significantly correlated with each other (see Neufeld & Gardner, 1990).

An additional measure of extrapyramidal symptomatology, the Simpson-Angus Scale (Simpson & Angus, 1970), was administered. One item, "head dropping" was replaced with two items from a scale developed by Chouinard, Ross-Chouinard, Annable et al. (1980), considered to be more relevant in the present context. These items were bradykinesia (sluggishness of physical and mental processes), and akathisia (motor restlessness; inability to lie quietly or sleep). Nonparanoid schizophrenic patients were found to exceed the paranoid schizophrenic patients and the psychiatric controls with respect to 2 items: "tremor" and "akathisia", $p < 0.05$.

General Procedure

Laboratory tasks performed by subjects included oculomotor and manual motor tracking, spatial memory, and line bisection. These tasks were undertaken during 2 half-hour experimental sessions. Specific procedures and measures derived from these tasks are outlined below.

TABLE 1
DEMOGRAPHIC CHARACTERISTICS OF GROUPS

Characteristic		Paranoid Schizophrenic	Nonparanoid Schizophrenic	Unipolar Affective Disorder	Normal
No. of subjects:					
Males		7	5	3	9
Females		8	4	7	7
Age (years)	*Mean*	31.73	31.22	32.70	25.31
	sd	7.88	6.23	9.14	4.55
WAIS Vocab. IQ	*Mean*	102.33	102.78	107.00	111.56
	sd	10.33	11.49	8.23	6.51
Estim. *Verb* Premorbid IQ+	*Mean(sd)*	99.8 (8.16)	99.8 (5.53)	99.6 (7.85)	--
Perf	*Mean(sd)*	102.2 (5.90)	101.8 (3.94)	101.6 (6.07)	--
Education (years)	*Mean*	12.07	12.11	11.90	14.88
	sd	2.99	1.69	1.08	1.54
Social Position++	*Mean*	3.53	4.00	3.60	3.19
	sd	1.06	1.00	1.07	1.11
Cumulative hospitalization (mos)	*Mean*	7.90	13.78	12.75	--
	sd	10.34	13.13	18.73	--
Chlorpromaz. medication equivalencies*(mg)	*Mean*	517.91	336.69	8.35	--
	sd	441.58	390.33	18.02	--
Benztropine medication equivalencies**	*Mean*	2.08	1.17	0.00	--
	sd	1.90	1.00	0.00	--
Edinburgh Handedness Index#	*Mean*	63.33	66.67	77.00	88.12
	sd	69.45	64.81	31.99	17.60
Premorbid Adjustment##	*Mean*	0.88	0.85	0.5	--
	sd	0.33	0.23	0.2	--

\+ Barona, Reynolds & Chastain (1984)
++ Hollingshead (1957)
* Davis (1976); Wyatt & Torgow (1976)
** Krogh (1982)
\# Oldfield (1971)
Phillips & Zigler (1961)

Tasks and Measures

Oculomotor Control

Tracking. Subjects watched a red laser light that was projected on a curved screen in front of them 57 cm away, so that the light was always equi-distant from a point between their eyes. The light moved horizontally between 15° left and 15° right of centre, due to the pivoting motion of a mirror galvanometer. Light movement was computer controlled. The head was stabilized by a chin-forehead rest, so that only the eyes moved during tracking.

Eye movement was recorded by DC electro-oculography (EOG); to enhance recording accuracy, solver-silver chloride electrodes were worn by subjects for 1/2 hour before the experiment began. The EOG signal was calibrated during the preliminary period. In addition, eye settings were calibrated by having the subject look to the centre and to the end points of target motion. This precaution was used because a study of normals showed that different subjects' settings of "straight ahead" and target end points were highly idiosyncratic (Mather & Fisk, 1985).

A copy of the signals of eye and target positions were recorded on adjacent channels of a Grass Model 7 polygraph. Baseline and accuracy of eye movements were recalibrated after every 4 trials, and the signals were filtered by a 1/2 amp frequency filter set at 72 Hz and a 60 Hz notch filter. To minimize fatigue, subjects rested for 30 sec after each trial and for 5 mins halfway through the approximately 20-min task.

For the test of tracking, the laser light target moved horizontally at a constant velocity for 30°. A trial comprised 6 different velocities: 11, 22, 33, 44, 55, and 66°/s, presented once in each direction, and sampled randomly (12 moves). Between moves, the signal was stationary at end point for 500 ms. Thus, the target departure time and direction were predictable, but not the velocity. Two trials were presented with the room lights on and 2 with them off. Note that movement of a structured background across the retina slows smooth pursuit velocity and increases the number of saccades in normal subjects (Colleiwjh & Taminga, 1982).

Measures provided by this task included mean number of saccadic movements at each target velocity during each trial. The number of saccadic movements was measured by hand from the polygraph records. Movements were only accepted as a saccade if they fit the following criteria: a single smooth movement, over 100° velocity (over 50° if they were in reverse direction to the pursuit component), 1.5° or more in amplitude, and not followed by a return of 50% or more (characteristic of blinks or electrical noise). Saccades in the opposite direction from target movement were counted for number but their amplitude was not added to the total, since that would be negative. All subjects attempted to some extent to predict target path, and occasionally but not often made "backward" saccades to return to accurate position at low target velocity. Few square wave jerks (Levin et al., 1982) or large anticipatory saccades (Whicker et al., 1985) were present, so it was not necessary to categorize them separately; most saccade-like moves met the velocity requirement. The number of these saccades was not simply related to the breakdown of pursuit tracking. Schizophrenic subjects did not have significantly slower pursuit than normals, and saccade number and pursuit velocity were not significantly correlated (Mather et al., 1992).

Looking. A trial for the test of looking comprised 8 instantaneous movements of end points described above (i.e., 30 moves). The light was stationary at end point for either 500 ms or, randomly, 750 ms. Thus, the direction and amplitude of the target movements was known, but not the departure times. Two trials were given with the light continuously illuminated, followed by 2 trials of a "flash" condition, where the target was illuminated until 200 ms following a move, and again for 200 ms before the next move. Performance under the flash condition provided little additional information. Measures obtained, therefore, under the continuous-light condition included mean total amplitude (degrees) of saccades per target move, and mean saccadic reaction time to target movement (ms).

Spatial Memory

Subjects were asked to remember the locations of 16 small familiar items, according to procedures similar to those used by Smith and Milner (1981) in testing for memory deficits resulting from the ablation of portions of the hippocampus. The items were set on a plexiglass sheet covering a matching-sized 64 x 48 cm piece of white paper. Items were placed on 16 locations randomly selected from a grid of 32, which allowed them to be spaced apart from one another and yet not be seen to be arranged in any pattern. Each time the items were presented to a new subject, their locations were shifted one place, for a total of 16 possible arrangements. Eight items were always to the left of midline and eight to the right. The head was placed in a chin-rest during viewing, for stability. The item array was viewed for 3 mins, with the items being named in turn during the first minute. Subjects then turned away for a 2-min "forgetting period", during which the items and plexiglass sheet were removed, leaving the blank paper. They turned back and recalled as many item names as possible, with no time constraint but usually within one minute. At this point subjects were requested to replace the items exactly as initially shown. The disparity between the original and replacement positions were recorded. Measures included, first, the mean distance in cm between the original and replacement item positions, as measured from the approximate item centre points, and, second, the number of item names recalled.

Line Bisection

The task was to indicate the centre of balsa wood sticks placed in front of the subject (Bowers & Heilman, 1980). Variation in task conditions included: stick length (12, 18, 24 and 30 cm), hand used in bisection (left, right), position of the stick with respect to body midline (left, centre, right), and modality of incoming information (visual, and, for comparison, tactile). In the visual condition, subjects bisected the stick with a pencil stroke; in the tactile condition, they indicated with the index finger the centre of an unseen stick, after running the finger along the stick at liberty. Each bisection in the tactile condition commenced with the experimenter placing the subject's hand once on the left and once on the right end of the stick, considered important in balancing for direction-specific errors. These combinations made for 96 bisections in all. Measures for the tactile and visual conditions included the following: deviation of line bisection from midline, measured in mm and recorded with sign (rightward = positive), as Constant Error, and without sign as Variable Error.

Manual Motor Tracking

Manual tracking procedures were identical to those of eye tracking, apart from the following. Manual tracking was accomplished by the movement by the

dominant hand of a pointer which pivoted about a point below the chin. Hand movements were recorded by a constant voltage signal passing through a linear potentiometer below the base of the pivoting arm holder; the resulting change recorded the angle of the pivoting pointer. As with oculomotor tracking, pointer settings for "straight ahead" and end points were calibrated for each subject. Measures included time lag (ms) of the hand crossing the centre behind the target — leading the target was recorded as negative lag — and amplitude gain in percent.

ANALYTICAL METHODS

In order to describe the structure of differences among the diagnostic groups with respect to oculomotor tracking, measures of the latter, along with those of looking, spatial memory, line bisection, and manual motor tracking, were submitted to a version of multiple discriminant function analysis (MDFA) originally developed by Cooley and Lohnes (1971), and Veldman (1967). The MDFA was used to identify the number of significant discriminant dimensions along which subjects from the respective diagnostic groups were separated. Discriminant dimensions comprise linear combinations of the included measures, minimizing overlap among the groups. Group separation is maximum on the first dimension, and becomes smaller with each succeeding dimension. Where the MDFA is carried out on 4 groups, with 3 or more measures, 3 significant discriminant functions are possible.

Each dimension contains unique information about the nature of intergroup separation, in that each is orthogonal to the remainder. The structure coefficients — correlations between the original measures and the linear combination of measures associated with a discriminant dimension — describe the nature of group separation with respect to the dimension. Unlike certain other multivariate procedures, MDFA is constrained to throw into relief variance and covariance among the respective measures specifically associated with inter-group differences. In this way, the method of analysis is ideally suited to identifying the multivariate structure of oculomotor-tracking as it distinguishes the diagnostic groups (see e.g., Neufeld, 1977). Previous uses of this procedure for dealing with similar problems in psychopathology have been presented by Broga and Neufeld (1977; 1981).

Since MDFA is not formally a "confirmatory multivariate procedure" (e.g., "confirmatory factor analysis"), — whereby the hypothesized pattern of results is specified numerically *a priori* — the stability of obtained findings needs to be considered. Note that MDFA comprises "eigen-decomposition" of a matrix product. Details of the matrix multiplication, and the elements of a matrix product that is "decomposed", are available in a number of sources (e.g., Harris, 1985; Neufeld, 1977a). Stability of solutions from eigen-decomposition of data matrices is determined by (a) magnitudes of the obtained "eigenvalues" which reflect group discrimination on their associated dimensions, and (b) absolute size of the subject sample (Guadagnoli & Velicer, 1988). Such considerations preempt earlier rule-of-thumb criteria involving measure-to-sample size ratios (see, e.g., Gudagnoli & Velicer, 1988). In the case of MDFA, stability can be evaluated directly in terms of the levels of discriminant-dimension statistical significance

(see also Davies, 1970), a function of eigenvalue magnitude, total sample size, as well as numbers of groups and measures. Statistical significance in this context refers to the probability of the obtained or greater group separation under the null hypothesis of a "multivariate swarm" from a common population (e.g., Cooley & Lohnes, 1971).

RESULTS

The measures submitted to the MDFA are listed in Table 2. In each case, a preliminary ANOVA was performed to identify significant interactions between Diagnostic Groups and the conditions of task administration (e.g., for oculomotor tracking, light-dark room illumination). Averaging of scores across the respective levels of a condition occurred only if there was no significant interaction between groups and the condition, except for the following two measures. Scores for the oculomotor saccade numbers incorporated significant nonadditive effects of groups and target velocity. Here, the slope and intercept of the linear function relating an individual's saccade number to target velocity was employed. Saccade amplitude similarly was scored, although effects of groups and target-velocity in this case were additive according to conventional alpha levels (actual $p \approx .08$). For Constant Error of tactile line bisection, two significant higher-order interactions were observed with respect to groups and the following conditions: stick length and stick position, and the preceding 3 factors, in turn, with left-right end of hand location, and left-right bisecting hand. Together, these effects accounted for less than 0.025 of the dependent-variable variance. Therefore, scores on tactile line bisection used for the MDFA were averaged across levels of these conditions.

In a few instances, data for the Continuous-Target Looking Task was unscorable because of recording difficulties. This problem occurred with one paranoid and two nonparanoid schizophrenic patients, one psychiatric control patient, and one nonpatient control. Missing observations for the two measures obtained from this task (Continuous target total amplitude, and Continuous target reaction time) were replaced by estimates with degrees of freedom of significance tests reduced accordingly (Frane, 1985; Kirk, 1982; Tabachnick & Fidell, 1983). The results reported in Table 2 should be regarded as univariate descriptors, augmenting the multivariate findings (below). Their status is much the same as that of bivariate correlation matrices, typically presented alongisde omnibus multivariate analyses, such as factor analysis, cannonical correlation, or linear structural relations analysis.

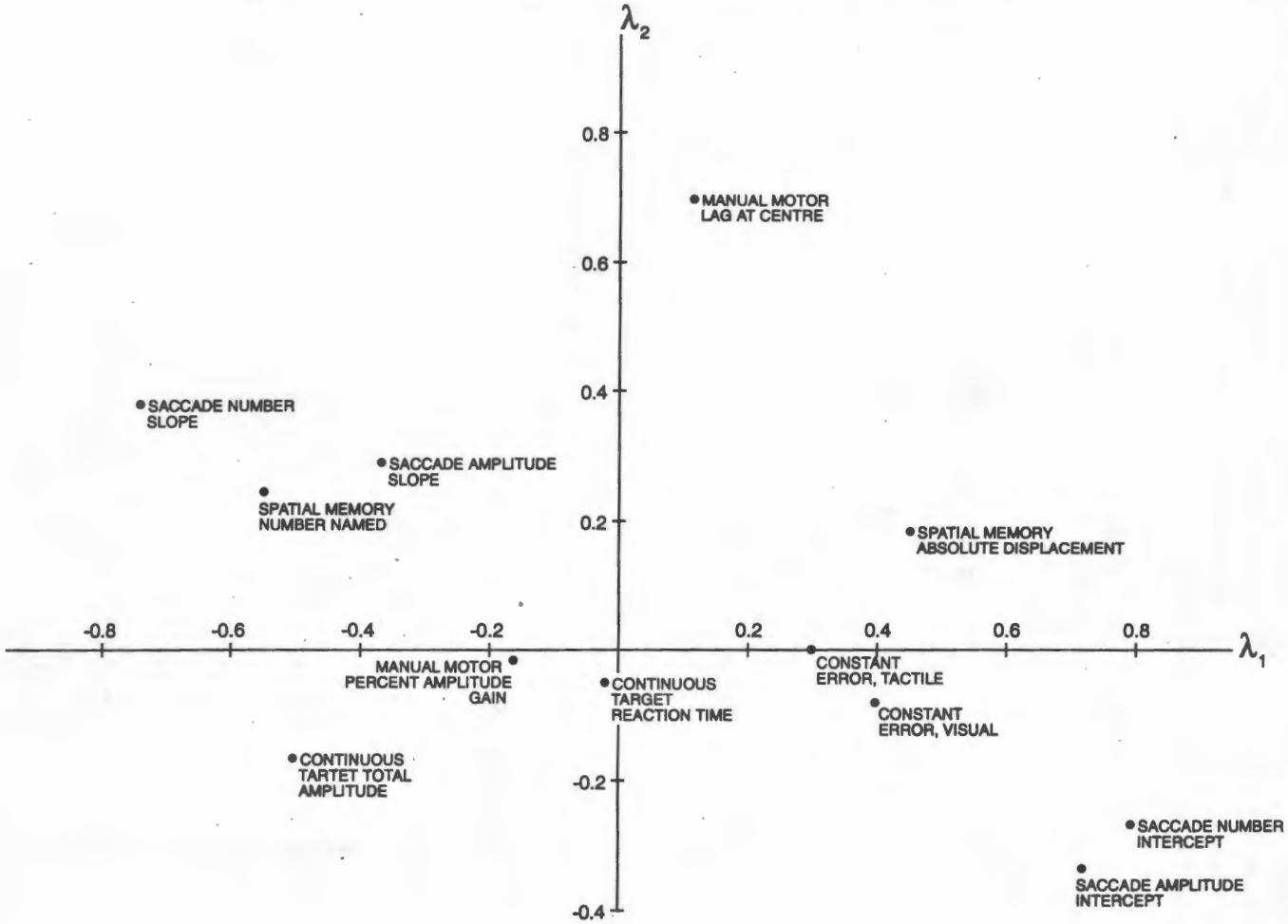
A one-way four-groups multivariate analysis of variance was applied to the measures listed in Table 2. The F-ratio approximation to Wilk's Λ was 3.170 (36/94), $p < 0.00009$, indicating one or more significant discriminant dimensions separated the groups. Group separation on the first dimension (Λ_1) was highly significant according to Rao's approximate chi^2 test, chi^2 (14) = 61.42, $p < 0.00009$; separation on the second dimension was significant also, chi^2 (12) = 24.65, $p \approx .0175$, unlike that on the third dimension, chi^2 (10) = 5.76. The first dimension accounted for 78% of the multivariate between-group separation, and the second dimension, for 19%.

Figure 1 presents structure coefficients for the two significant discriminant dimensions. The configurations of group separation on the 2-dimensional plane is

TABLE 2. Means, standard deviations (in parantheses) and univariate F ratios for measures submitted to MDFA.

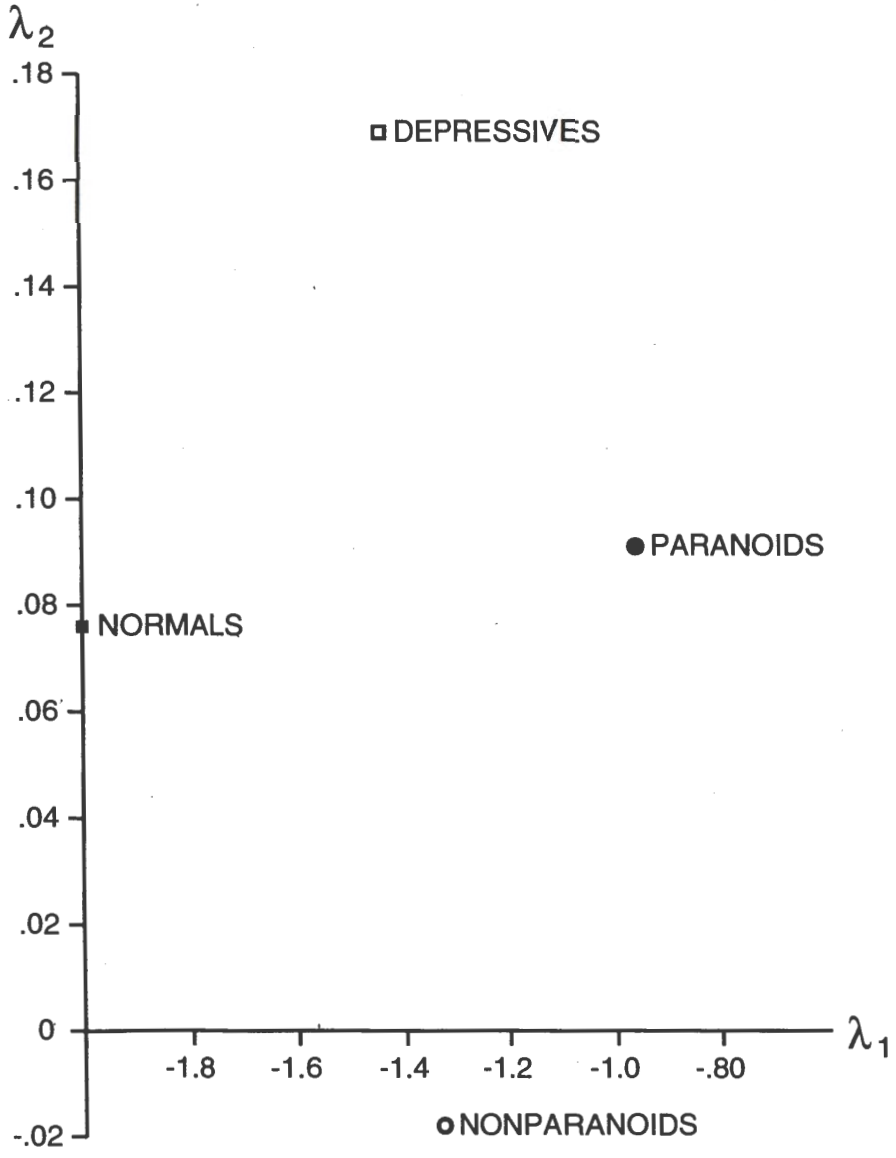
Measure	Paranoid Schizophrenics	Nonparanoid Schizophrenics	Psychiatric Controls	Nonpatient Controls	*F*	*df*	*p*
Oculomotor Control:							
Tracking:							
Slope for a saccade number as a function of target velocity	-0.0112 (0.0381)	-0.0052 (0.0178)	0.0189 (0.0187)	0.0285 (0.0227)	13.81	3/46	< 0.0001
Intercept for saccade number as a function of target velocity	3.19 (0.9765)	2.82 (0.7370)	1.87 (0.8350)	1.13 (0.8857)	16.17	3/46	< 0.0001
Slope for saccade amplitude as a function of target velocity	0.3034 (0.0413)	0.2941 (0.0645)	0.3437 (0.0941)	0.3554 (0.0802)	2.35	3/46	NS
Intercept for saccade amplitude as a function of target velocity (deg)	0.143 (1.7719)	0.289 (1.9481)	-2.274 (1.3416)	-3.306 (1.9792)	11.49	3/46	< 0.0002
Looking:							
Continuous-target total amplitude (degrees)	26.99 (3.1321)	28.41 (1.5317)	27.87 (2.4680)	29.71 (1.9225)	4.03	3/41	< 0.05
Continuous-target reaction time (msec)	190.00 (17.627)	193.55 (19.955)	192.22 (13.357)	191.33 (14.659)	< 1	3/41	NS
Spatial Memory:							
Absolute Displacement(cm)	9.50 (6.8298)	6.73 (3.8574)	8.15 (3.9274)	4.54 (2.2735)	3.16	3/46	< 0.04
Number Named	10.67 (2.1269)	10.44 (2.4552)	12.10 (1.7920)	13.44 (2.5025)	5.21	3/46	< 0.04
Line Bisection:							
Constant Error,Visual(mm)	0.0280 (0.1257)	0.667 (0.210)	0.0180 (0.1931)	-0.1294 (0.2323)	2.74	3/46	NS
Constant Error,Tactile(mm)	0.1720 (0.4373)	-0.0667 (0.3073)	-0.1010 (0.3881)	-0.0825 (0.3100)	1.74	3/46	NS
Manual Motor Tracking:							
Lag at Center (msec)	118.63 (67.936)	37.92 (62.892)	135.46 (64.378)	92.8 (48.2697)	4.85	3/46	< 0.006
Percent Amplitude Gain	94.55 (16.295)	101.52 (15.261)	102.82 (15.983)	100.45 (13.556)	< 1	3/46	NS

Figure 1. Structure coefficients of measures on Λ_1 and Λ_2.



presented in Figure 2. Locations on this plane correspond to the group centroids, which comprise the group-average discriminant scores; discriminant scores, in turn, are weighted sums of the constituent measures, the weights being determined by the corresponding discriminant function. For measures with negative structure coefficients in Figure 1 (e.g., saccade-number slope on Λ_1, and Continuous-target total amplitude on Λ_1), lower measure scores lead to higher discriminant scores (Figure 2). The group means, and univariate *F* ratios for the various measures are presented in Table 2.

Figure 2. Group centroids on Λ_1 and Λ_2.



Consideration of the structure coefficients (Figure 1) indicates that Λ_1 is described by measures of oculomotor control, and Λ_2 by a measure of manual motor control (lag at centre). Measures with the higher structure coefficients on Λ_1 include the slope of the linear function relating saccade number to tracking target

velocity. Paranoid schizophrenic patients had the highest negative slopes and normal controls, the highest positive slopes (Table 3). Neuman-Keuls *a posteriori* paired comparisons, pursuant to the significant F ratio presented in Table 2, indicated each subgroup of schizophrenic patients to be significantly different from each group of controls, $p < 0.0001$, other differences being nonsignificant. Similarly, the paranoid schizophrenic patients had the highest mean intercept of the saccade-number linear function, the nonpatients having the lowest. Paired comparisons revealed a pattern of inter-group differences like that for saccade-number slope, except now also the psychiatric and nonpatient controls differed significantly, $p < 0.01$. For the intercept of the linear function relating saccade-amplitude to tracking target velocity, the configuration of significant differences among the groups was identical to that for saccade-number slope.

TABLE 3
MEAN NUMBER OF SACCADES OVER TARGET VELOCITIES FOR EACH GROUP.

	Target Velocity (°/s)					
Group	11	22	33	44	55	66
Paranoid Schizophrenic	3.21	2.75	2.88	2.81	2.69	2.47
Nonparanoid Schizophrenic	2.73	2.54	2.82	2.79	2.55	2.33
Psychiatric Control	1.86	2.14	2.90	2.95	2.95	2.82
Normal Control	1.42	1.65	2.08	2.08	2.77	2.72

Turning to performance on the looking task, the total saccade amplitude associated with the instantaneously shifting target was significantly lower for the paranoid schizophrenic patients than for the nonpatient controls, $p < 0.05$.

Measures of other behaviors associated with Λ_1 were those of spatial memory; number of items named, and displacement from original positions. Both paranoid and nonparnaoid schizophrenic patients named fewer items than nonpatient controls, $p < 0.05$, and the paranoid schizophrenic subgroup exceeded the nonpatient controls in displacing the items from their original positions, $p < 0.05$.

Based on the configuration of measures describing Λ_1, this dimension was considered to be one of "spatial-memory related oculomotor abnormality". The configuration, furthermore, was suggestive of oculomotor abnormality at lower target velocities comprising relatively higher-frequency, higher-amplitude saccades. Specifically, saccade-number and amplitude intercepts had appreciable positive structure coefficients for this dimension (.79 and .70, respectively), and the slope for saccade number had a substantial negative structure coefficient (-.73). Paranoid schizophrenic subjects obtained the highest discriminant scores

for this dimension, normals the lowest, with the nonparanoid schizophrenic patients and psychiatric controls having more intermediate values (Figure 2).

We consider, now, the second discriminant dimension. The salient descriptor was manual motor lag at center. The discriminant scores of the nonparanoid schizophrenic subjects and depressed psychiatric patients located these groups at opposite extremes on this dimension, the paranoid schizophrenic patients and nonpatient controls this time scoring in between.

The depressed patients displayed the most pronounced lag and the nonparanoid schizophrenic patients, the least. The mean lag of the nonparanoid schizophrenic group was significantly less than that of each other group, $p < 0.01$. The reduced lag for the nonparanoids was attributed not to superior tracking performance relative to others (including normal controls), but to elevated extrapyramidal symptoms as assessed by the AIMS, and the "tremor" and "akathisia" items of the modified Simpson-Angus scale, described above (Chouinard et al., 1980; Simpson & Angus, 1970). The constellation of findings indicated that extrapyramidal symptomatology was aligned with manual motor rather than oculomotor abnormalities, as registered on the current indices. This finding initially seems paradoxical. Most extrapyramidal disorders cause slowing of movement rather than acceleration, or else involuntary jerking. The movement disorder here is one of activation or increase (tremor, restlessness). Indeed two of the subjects actually showed a negative lag, moving ahead of the stimulus (although their scores on the extrapyramidal measures were not the largest in the group) and the rest of the nonparanoid group also demonstrated decreased lag. Manneristic movements and spasmodic, even choreiform phenomena, have long been recognized as part of the motor findings in schizophrenia (Kraepelin, 1919). The tendency for abrupt, advanced or excessive response by the non-paranoid patients, in whom the above phenomena are more prominent is thus likely to be part of the inherent pattern of the illness. It is not readily attributable to medication since the amounts taken of neuroleptics and anti-Parkinsonian drugs were intermediate in this group, between the paranoid schizophrenic group and the others.

Certain other observations augment this interpretation. First, with respect to Λ_1, the ordering of group centroids (Figure 2) did not accord with extrapyramidal symptoms means. Second, intragroup correlations of dependent variables with measures of extrapyramidal symptomatology, and with CPZ and benztropine dosage equivalencies, were nonsignificant throughout, $p > .10$ (procedures of Larzelere & Mulaik, 1977, for multiple tests of correlations applied group-wise). In the case of saccade-number slope, for example, sample intragroup correlations with dosage equivalencies either were 0 (e.g., paranoids, benztropine) or positive (e.g., 0.53, nonparanoids, benztropine), a direction opposite to that expected if psychotropic medication created the negative slope of schizophrenic subjects. The results from a multivariate analysis auxiliary to the present research questions (available from the first author) indicated that extrapyramidal symptomatology and manual motor lag define a unique dimension. These findings incidentally help, likewise, to discount the possibility that EMD overall is due to medication.

Anticholinergics may disrupt certain aspects of memory (Frith, 1984; Spohn & Strauss, 1989). Correlations between benztropine dosage equivalencies and measures from the spatial memory task ranged from -0.2017 (paranoids, number named) to 0.4529 (nonparanoids, absolute displacement); but only the paranoid

subgroup was markedly different from controls on absolute displacement, with a negligible correlation between benztropine and displacement (r=0.0268). All values were nonsignificant (p > at least 0.10), even by unadjusted, liberal tests.

The preceding represent correlational estimates of the effects of medication on obtained results. Such an approach constitutes the best, albeit fallible, precaution against incorrect attributions of the findings obtained, in the absence of other options (Neufeld & Broga, 1981). Strengths and weaknesses of this approach have been discussed at some length elsewhere (Neale & Oltmanns, 1980; Neufeld & Broga, 1981).

DISCUSSION

Multivariate analysis of the selection of oculomotor-control and related measures indicated a bidimensional separation among the diagnostic groups. The first dimension reflected EMD, the structure of which was defined most saliently by the slope and intercept of the linear function relating saccade number to target velocity. The second dimension was specific to lag in manual motor tracking of a target, a motor measure from the separate subsystem of hand tracking (Mather & Putchat, 1984). The two subgroups of schizophrenic subjects had the higher values on the first dimension, with the paranoid subjects exceeding the nonparanoid subjects. On the second dimension, the unipolar affective disorder patients had the highest values (the greatest manual-motor tracking lag) and the nonparanoid schizophrenic patients the lowest values. The reduced lag on the part of the latter group was attributed to the effects of an abrupt extrapyramidal movement disorder rather than to the side effects of medication on manual motor movement.

Measures of non-oculomotor behaviors associated with the first dimension included the number of items of the spatial memory task correctly named during immediate recall, and the accuracy of item relocation (based on Smith & Milner, 1981). The nature of this dimension can be considered further in the light of the properties of measures describing its structure, and with known cognitive-behavioral abnormalities among paranoid schizophrenic subgroups.

The spatial memory task taps, among other functions, the encoding of presenting stimulation into a format which facilitates rehearsal, and subsequent recall. Difficulties in encoding the properties of spatial and verbal stimuli to facilitate task performance have been prominent among schizophrenic patients in a number of past experiments involving choice reaction time and related tasks (e.g., George & Neufeld, 1984; Highgate-Maynard & Neufeld, 1986; Neufeld, 1977b). This deficit has been especially pronounced among paranoid schizophrenic subjects (Broga & Neufeld, 1981b; Neufeld, 1978; 1991), the subgroup scoring highest on the first discriminant dimension in the present study. Completion of a unit of encoding appears to entail a larger number of steps, or "subprocesses" of encoding (Townsend, 1984) among these subjects (Nefueld, Vollick, & Highgate-Maynard, 1993; Neufeld, 1992).

Negative mean slopes of linear functions of saccade-number (Table 2) reflect the relatively greater frequency of saccades at lower target velocities and slightly lower frequencies at higher velocities among the schizophrenic subjects (Table 3). The pronounced higher frequencies at lower velocities in particular are compatible with a deficit in stimulus encoding, for the following reasons. Oculomotor

tracking has been shown to entail stimulus encoding with respect to spatial information surrounding the target velocity (Katsanis & Iacono, 1991; Kohler, van der Steen, Taminga et al., 1984; Khurana & Kowler, 1987). In turn, in the cognitive psychology literature, discontinuous eye movements (fixations) have been linked directly to the encoding of spatial and other stimulus features (Just & Carpenter, 1976; 1987). Increased saccadic frequency would accord with reduced efficiency, as outlined above, in encoding tracking-facilitative spatial information. Such an effect should be more apparent at lower target velocities, before saccadic movements tend to dominate smooth oculomotor pursuits for all subjects (Collewijn & Tamminga, 1982).

Other functions involved in oculomotor tracking appeared to be intact among schizophrenic subjects. The mean velocity of smooth pursuit movements was similar among the present samples, regardless of target velocity (Mather, 1989). Increased saccade production, therefore, in the present instance, is not readily attributable to deficiencies in this subsystem of tracking. Subsidiary microanalysis of movement/target records indicated also that distance away form the target path upon saccade completion was similar among the groups of subjects; all differences were small and nonsignificant (Mather, 1989). In this way, normal accuracy of the saccade subsystem was observed among the schizophrenic subjects (Colleiwjn & Tamminga, 1982). Finally, oculomotor reaction time in the looking task, one considerably less laden with ongoing information-processing demands, was intact, similar to findings of Iacono, Tuason and Johnson (1981) and Mather and Putchat (1983).

Recently, a Mendelian dominant-gene mechanism has been advanced as a possible source of the joint heritability of schizophrenia and EMD (Holzman et al., 1988; Matthyse et al., 1986; cf., Nicholson & Neufeld, 1993). Meehl (1962; 1989) was the first to suggest such a mechanism for the genetic transmission of schizophrenia. The current version of this account postulates that an autosomal dominant gene has a high, though imperfect, likelihood of endowing a bearer with a given latent trait; the latent trait, in turn, tends to evince schizophrenic symptomatology and EMD. Furthermore, in the absence of the allele coding for the latent trait, the probability of the latter's occurrence is small, though not 0. Previous tests of the mathematical model expressing the Mendelian mechanism of transmission have supported the model's reliability, and to some extent, validity across demographically divergent samples (Holzman et al., 1988; Matthyse et al., 1986; however, cf. discussion by Clementz & Sweeney, 1990, p. 86). Note that manifestation of a putative latent trait technically is not restricted to EMD per se; other variables are eligible, although EMD has been of obvious value in the development and testing of models. In the present instance, EMD has pointed to other variables by implicating correlated cognitive/perceptual processes. This liaison permits such processes, then, potentially to bear on the genetic transmission model, and vice versa. The latent trait can express itself in the form of cognitive/perceptual deficits, especially as the latter mediate EMD. Resulting refinements of the gene-transmission model include the addition of parameters comprising conditional probabilities of (a) selected cognitive deficits (e.g., stimulus-encoding retardation), given the latent trait (and, secondly, given its absence), as well as (b) EMD, given the cognitive deficit (and its absence). Testing of an expanded model incorporating the above parameters requires measures of target

deficits and EMD to be obtained among families of schizophrenic probands. An expanded set of measures obtained from such a sample currently is being analyzed by Cromwell and co-workers (personal communication, 1989). In a previous study, Mather (1985) observed increased saccade production among children of schizophrenic probands. Similarly, Clementz, Sweeny, Hirt and Haas (1990) recently have reported elevated frequency of intrusive saccades among relatives of schizophrenic patients, especially those relatives with "schizophrenia spectrum disorder". However, the present cognitive-perceptual measures did not accompany eye-tracking measures in those studies.

A final word is in order concerning the stability of the multivariate pattern of results. As stated earlier, magnitude of obtained "eigenvalues" must be considered in conjunction with sample size (Guadagnoli & Velicer, 1988). In the case of multiple discriminant function analysis, this combination affects directly the level of statistical significance of the discriminant dimensions. The probability of obtained or greater group separation on the first discriminant dimension, given the null hypothesis, was less than 0.00009; that for the second dimension was approximately 0.0175. Collectively, the probability value associated with the obtained pattern of group separation was less than 0.00009. Clearly, the requirements for stability, according to these criteria, were met (see also Davies, 1970).

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