Shift of Attention in Schizophrenics, Siblings of Schizophrenics, and Depressed Patients

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This study investigated whether deviant types of attention, measured by reaction time, would characterize schizophrenics, depressives, and healthy siblings of the schizophrenic probands. Nineteen schizophrenics, 16 depressives, 15 healthy siblings of the schizophrenics, and 19 normals were tested on the cross-modal reaction time task. Two ways of conveying information about the impending imperative stimulus were compared to attempt to normalize schizophrenic cross-modal retardation. Deviant shift of attention was measured by cross-modal retardation, i.e., a greater than normal lengthening of reaction time to cross-modal vs. ipsilateral stimuli. Both schizophrenics and depressives showed deviant shift of attention to sound stimuli, but only schizophrenics were deviant to light stimuli. Anomalous cross-modal retardation was not normalized by the experimental manipulation. Both patient groups showed significantly slowed reaction time, suggesting deviances of general alertness. Depressed patients displayed anomalous sustained attention, indexed by significant lengthening of reaction time over trials. Siblings of schizophrenics did not differ significantly from controls on any aspect of reaction time performance. High magnitude of cross-modal retardation to sound and slow overall reaction time may be markers of psychopathological disorder rather than vulnerability. High cross-modal retardation to light may be a marker of schizophrenic disorder. Slowing of reaction time over trials is a potential marker of depressive disorder.

Reviews of the literature on schizophrenic information processing (2, 5, 6, 20, 27-30, 37, 40, 44) have consistently concluded that schizophrenics display disordered attention. However, it might be observed that contemporary research has done little more than validate the earliest clinical descriptions of deviant attention in schizophrenia (13, 16). Four important gaps persist in our knowledge of schizophrenic attentional disturbance: a) to what extent are attentional anomalies unique to schizophrenics vs. characteristic of a variety of psychopathologies? b) is attention dysfunction associated only with overt psychopathological disorder, or is it also a marker of underlying vulnerability to psychopathology? c) can different types of attention deviance be delineated and differentiated? d) can schizophrenic attentional disturbance be normalized by experimental manipulations?

Diagnostic Specificity

The problem of whether attentional deviance is specific to schizophrenia or general to many kinds of disorder is critical to evaluating the hypothesis that attentional disturbance plays a specific etiological role in the development of schizophrenia. Many (11, 18, 20, 22) have proposed that attentional disturbances lead to primary changes in the quality of sensory experience, to which specific schizophrenic symptoms are secondary sequelae. An alternative hypothesis is that attentional deviance itself has multiple etiologies and may serve as a final common pathway in the development of many disorders. An analogy might be drawn to elevated white blood cell count, which can arise from many agents or processes and indicate the onset of various medical disorders.

Research to determine whether attention disturbance is unique to schizophrenia requires explicit diagnostic criteria and a control group similar to schizophrenics in severity of illness.

Markers of Disorder or Vulnerability

In order to determine whether a disorder in shift of attention is a marker of vulnerability to schizophrenia,
healthy siblings of schizophrenics were tested in the present study. If the cross-modal task detected deviations in both the healthy relatives and the schizophrenic probands, this would suggest that difficulties in shifting attention may reflect a vulnerability to schizophrenia.

This research was guided by a model distinguishing between vulnerability to schizophrenia, a relatively permanent trait, and episodes of schizophrenic disorder that are waxing and waning states (45). Spring and Zubin (33) proposed a strategy for identifying test procedures that are markers of vulnerability or episodes of psychopathology. The complete strategy requires dovetailing findings from cross-sectional studies of psychiatric patients and their relatives with findings from prospective studies of patients followed from the acute episode until symptom remission. Since the current report pertains only to the cross-sectional stage of this research, an abbreviated strategy was used.

A performance characteristic was considered a potential marker of vulnerability to schizophrenia if it appeared predominantly in schizophrenics and their healthy relatives. If a performance deviation was prevalent in nonpsychotic psychiatric patients as well as in schizophrenics and their relatives, the characteristic was considered a general marker of vulnerability to psychopathology. A performance anomaly not characteristic of relatives of schizophrenics but prominent among both schizophrenic and depressed patients was considered a general marker of psychopathological disorder. Characteristics not found in relatives of schizophrenics but prevalent among schizophrenics or depressives were considered markers of schizophrenic disorder or markers of depressive disorder, respectively.

Different Types of Attention

A unitary concept of attention deviance has persisted in experimental psychopathology research despite the fact that measures of attention display minimal intercorrelation (1, 15, 23). Such lack of relationship may reflect the existence of different types of attention, separable conceptually and operationally, such that deficits of one type need not imply impairment of another (25). Tests of attention should be described in terms of the type of attention they measure, for example, alertness, vigilance, sustained, shift, or maintenance of selective attention. Correlations across attentional tasks or across different measures within a single task should not necessarily be expected unless both tasks or measures operationalize the same type of attention.

A finding that one attention task is a vulnerability marker does not imply that another attention task will also be a marker of vulnerability, particularly if the two operationalize different types of attention. The continuous performance test, measuring vigilance (26), and smooth pursuit eyetracking, measuring nonvoluntary attention (12), have both proven sensitive to vulnerability to schizophrenia. In contrast, findings for the set reaction time task (29), measuring sustained (34) or selective (28) attention, have been inconsistent (39).2,3 Despite superficial similarities to set reaction time (34), cross-modal reaction time measures a different kind of attention. No findings have previously been reported on whether attentional shifting is disordered in persons vulnerable to schizophrenia.

Cross-Modal Reaction Time

The cross-modal reaction time task was designed to test Mettler's (21) hypothesis that schizophrenic patients have difficulty shifting attention from one modality to another. Reaction times are compared to ipsimodal stimuli (i.e., stimuli preceded by a stimulus in the same modality) and cross-modal stimuli (i.e., stimuli preceded by a stimulus in a different modality). An anomaly in shift of attention is inferred from greater than normal discrepancy in response speeds to sequences that require attention to shift (cross-modal) vs. those that do not require attentional shift (ipsimodal). In previous studies (36, 38, 41), schizophrenics showed greater than normal lengthening of reaction time for cross-modal relative to ipsimodal sequences involving sound as the second stimulus. Whereas two studies (36, 38) failed to find significant schizophrenic cross-modal retardation to light, the one study (41) that equated sound and light stimuli for sensation level did find such an effect. This has been the first study of cross-modal reaction time to use explicit diagnostic criteria, to test a nonpsychotic psychiatric control group, and to test relatives of patients.

In addition to shift of attention, the cross-modal reaction time task yields measures of two other types of attention. Reaction time level, or the average speed of response, provides a measure of general alertness. Previous findings suggest that rapid vs. slow reaction time level is associated with increased responsiveness to environmental stimuli (43), regulation by stimuli arising from external vs. internal sources (14), and low vs. high stimulus redundancy acceptance levels (32). Finally, the degree to which reaction time lengthens over many trials provides a measure of difficulty in sustaining attention.


Strategies to Normalize Attentional Deviance

Previous research (41) has demonstrated that telling subjects what stimulus to expect reduced but did not eliminate schizophrenic cross-modal retardation. Verbally conveying expectancy information on each trial inherently augments the degree of social interaction between experimenter and subject during the task. In view of the theory (35) that schizophrenics experience anxiety over social interaction, it was reasoned that patients' anxiety might disrupt their tendency to profit from correct expectancy information. Therefore, the usual verbal means of conveying information was compared with another condition in which a sound or light presented by the reaction time machine conveyed expectancy information. It was expected that schizophrenic cross-modal retardation would be reduced in the machine vs. verbal information condition because the impersonal nature of the task would reduce the disruptive effects of social anxiety on schizophrenic attention.

Method

Subjects

Sixty-nine subjects were tested: 19 schizophrenic patients, 16 patients with major depressive disorder, 15 healthy siblings of the schizophrenic probands, and 19 normal control subjects. All subjects were males between the ages of 18 and 36 years. Schizophrenics, depressives, and controls were matched on age, ethnicity, and educational level.

Table 1 presents the average biographical details of the groups and the results of tests of differences on each biographical variable. The groups did not differ significantly in age or ethnicity. Siblings were better educated than both patient groups (Newman-Keuls' Test), and the proportion of married individuals was greater among nonpatients.

Patients were tested within 2 weeks of admission to Kingsboro Psychiatric Center, Brooklyn, New York. Charts were reviewed for all male patients in the appropriate age range admitted to cooperating wards. When a patient appeared suitable for the study, a screening interview was jointly administered by two psychologists. Patients were selected or excluded from the study and assigned Research Diagnostic Criteria (RDC) diagnoses based on the agreement of two psychologists evaluating abstracts of the chart review and screening interview.5

The patient groups did not differ significantly in number of previous hospitalizations. All patients had been diagnosed schizophrenic by hospital psychiatrists,6 and all were receiving antipsychotic medication.7 Schizophrenic and depressed patients did not differ significantly in medication dosages in terms of mean daily equivalents of chlorpromazine (4) expressed in milligrams per kilogram body weight. Similarity of medication served to reduce adventitious differences between schizophrenic and depressed groups due to differences in medication status.

Normal controls were recruited from the New York State Employment office and were screened to exclude cases with a personal or familial history of psychiatric hospitalization. Information about hospitalization was confirmed by the Statistical Services Division of the New York State Department of Mental Hygiene. This agency provided data on history of inpatient and outpatient hospitalization for each subject and his first-degree relatives.

Apparatus

Stimulus sources for sound and light were 1-inch circles of brown and white, respectively, located at the subject's eye level. Response keys for sound and light were positioned in front of the appropriate stimulus

5 Of 566 male patients admitted to cooperating wards during the course of the study, 35 (6 per cent) served as subjects and 8 (1 per cent) were not approached for screening. The remaining patients were excluded from the sample primarily for the following reasons: a) inappropriate age; b) escaped or scheduled for discharge; c) asymptomatic or denied symptoms; d) schizophrenic patient lacking appropriate sibling (schizophrenic patients were only tested if they had a brother aged 18 to 36 never hospitalized for psychiatric disorder and residing in the New York metropolitan area; the criterion that siblings never have been hospitalized was adopted to ensure that the sibling sample, although vulnerable to schizophrenia, was not contaminated by cases of previous psychiatric disorder); e) diagnosis uncertain or inappropriate (this category includes schizophrenics who were not subchronic, having had at least one previous hospitalization, in order to exclude cases of transient, situationally induced emotional disturbances); f) recent drug or alcohol abuse; g) evidence of organic brain syndrome, epilepsy, venereal disease, or recent ECT; or h) transferred to Kingsboro Psychiatric Center from long term institutionalization.

6 Male patients in the study age range were rarely given hospital diagnoses of affective disorder. Of the hundreds of age-appropriate patients screened, three had been diagnosed manic or depressed. However, the current sample of depressed patients displayed symptoms that warranted a diagnosis of major depressive disorder according to the RDC.

7 The majority of individuals in both patient groups were receiving phenothiazine medication. Of the schizophrenics, 13 were receiving chlorpromazine only, two were receiving chlorpromazine and fluphenazine, one was receiving fluphenazine only, and one was receiving thioridazine only. Of the depressed patients, seven were receiving chlorpromazine only, six were receiving fluphenazine only, one was receiving fluphenazine and thioridazine, and one was receiving thioridazine only. Two schizophrenics and one depressed patient were receiving butyrophenones (haloperidol in all three cases).
TABLE 1
Biographical Details of Groups

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<tr>
<th>Variable</th>
<th>Schizophrenic</th>
<th>Depressed</th>
<th>Sibling</th>
<th>Control</th>
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<tr>
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<td>10.8</td>
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<td>14</td>
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<tr>
<td>SD</td>
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</table>

\*p < .05.

sources. A center key was designated as home key. Pressing the appropriate response key triggered a randomly variable preparatory interval of between 1.5 and 3.5 seconds and terminated by the sound or light stimulus.

Visual and auditory stimuli were equated and maintained at 2.0 log units of power above threshold for one normal subject. The auditory stimulus was a 10-millisecond white noise burst produced by a random noise generator (General Radio Company, Model 1381). The visual stimulus was a 10-millisecond flash produced by a glow modulator gas-discharge tube (Sylvania R1131C), collimated and focused on a diffusing plate. The light appeared at a viewing distance of 15 inches from the subject and subtended a visual angle of 3.93°. Flash intensity was 7.86 foot-lamberts. Background illumination was .00039 foot-lamberts. Background noise from an air conditioning unit masked extraneous noise.

Procedure

After 5 minutes of adaptation to the dark, six practice trials were given. Subjects began each trial by depressing the home key with the index finger of their writing hand. In the verbal information condition the tester then told the subject what stimulus to expect. In the machine information condition a sound or light forewarned the subject. At their own pace, subjects depressed the appropriate key, triggering a variable foreperiod terminated by the stimulus. The identical finger lift response was required to sound or light.

To maintain motivation, subjects were told that they would earn a 1-cent bonus each time their reaction time was very fast. A bonus was awarded every time a reaction time was faster than the previous one, and totals were reported to the subject after each block of trials.

Stimuli were presented in 10 blocks of 49 stimuli each. Within each block, stimuli were presented in quasi-random order, with the sole constraint that no more than 15 stimuli in the same modality appeared successively. Verbal and machine information conditions were alternated across blocks, with starting order counterbalanced across subjects. After the fifth block, stimulus sources and response keys were physically switched, to appear equally often to the subject's left and right. Starting position was counterbalanced across subjects.

Subjects were tested in two sessions of five blocks with 2-minute rests between blocks.

Results

Reaction times were transformed to base e logarithmic equivalents to normalize frequency distributions and reduce heterogeneity of variance resulting from a correlation between cell mean and standard deviation values.

Data were analyzed by a four-factor mixed model analysis of variance with group (schizophrenic, sibling, depressed, control) as a between factor, and modality shift (ipsi-modal, cross-modal), information (verbal,
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Analyses were conducted separately for reaction times to sound and light. For reaction times to sound, significant main effects were found for groups \( F[3,65] = 18.22, p < .001 \) and modality shift \( F[1,65] = 12.60, p < .01 \), and significant interactions were found for group \( \times \) modality shift \( F[3,65] = 2.76, p < .05 \) and group \( \times \) block \( F[12,260] = 3.18, p < .001 \). For reaction times to light, significant main effects were found for group \( F[3,65] = 16.46, p < .001 \) and modality shift \( F[1,65] = 13.97, p < .001 \), and a significant interaction was found for group \( \times \) block \( F[12,260] = 3.08, p < .001 \).

**Modality Shift**

Simple effects were analyzed to determine which groups showed significant lengthening of reaction time as a function of modality shift. Because of the interest in determining whether schizophrenics display cross-modal retardation to light as well as sound, simple effects were analyzed for both light and sound. Significant differences between ipsimodal and cross-modal reaction times were found only for schizophrenics' response to both sound \( F[1,65] = 11.83, p < .001 \) and light \( F[1,65] = 15.37, p < .001 \) and for depressives' responses to sound \( F[1,65] = 8.55, p < .01 \).

Figures 1 and 2 indicate the degree of cross-modal retardation found for each group by displaying the cumulative percentages of individuals in each group at different magnitudes of cross-modal retardation. Retardation was computed as the difference between antilog mean cross-modal reaction time (in milliseconds) and antilog mean ipsimodal reaction time (in milliseconds). Difference scores were based on antilogs because subtraction of logs is a division operation.

Supplementary contrasts were performed to compare pairs of groups on the effect of modality shift. No significant differences were found between normal controls and siblings of schizophrenics in modality shift effects for either sound or light reaction time. Since neither nonpatient group showed a significant simple effect of modality shift, the two groups were combined for further analyses.

For the effect of modality shift on sound reaction times, both schizophrenics and depressives differed significantly from the nonpatient group \( F[1,65] = 5.43, p < .05 \) for schizophrenics; \( F[1,65] = 4.04, p < .05 \) for depressives). Schizophrenics and depressives did not differ significantly. Results suggest that increased cross-modal retardation to sound stimuli is a general marker of psychopathological disorder.

Only the schizophrenic group differed significantly from nonpatients on the modality shift effect for light reaction times \( F[1,65] = 4.27, p < .05 \). This finding suggests that increased cross-modal retardation to light stimuli is a marker of schizophrenic disorder. However, its specificity is uncertain since schizophrenics and depressives did not differ significantly.

**Reaction Time Level**

For reactions to sound and light, both schizophren-
ics and depressives were significantly slower than either nonpatient group (Newman-Keuls' Multiple Range Test). There were no significant differences between schizophrenics and depressives, or between siblings and controls. Results suggest that slowed reaction time is a general marker of psychopathology.

**Slowing of Reaction Time over Trials**

The significant interaction between group and block for both sound and light reflects the fact that only depressed patients showed a consistent lengthening of reaction time across blocks. Figure 3 shows this effect for sound reaction time. Simple effects analyses revealed that depressives displayed a significant change in reaction time level across all 10 blocks ($F[9,585] = 4.61, p < .001$ for sound; $F[9,585] = 3.45, p < .001$ for light). The simple effect for block was insignificant for the remaining groups for both light and sound. Slowing of response speed over trials appears to be a marker of depressive disorder.

**Correlational Analyses**

Pearson product-moment correlations were computed to determine whether the patient groups' greater than normal cross-modal retardation could be explained by their slower overall reaction time level. The correlation between antilog overall reaction time and antilog cross-modal retardation was not significant for schizophrenics ($r = .01$ for sound; $r = .26$ for light) or depressives ($r = -.17$ for sound; $r = .13$ for light).

Correlations were also computed to determine whether overall reaction time level or cross-modal retardation were associated with medication dosage. There were no significant correlations between dosage and overall reaction time for schizophrenics ($r = .14$ for sound; $r = .23$ for light) or depressives ($r = .02$ for sound; $r = -.04$ for light). In addition, there were no significant relationships between dosage and cross-modal retardation for schizophrenics ($r = -.27$ for sound; $r = .41$ for light) or depressives ($r = .09$ for sound; $r = -.02$ for light).

**Discussion**

The results of this study replicate the finding of Waldbaum et al. (41) that schizophrenics show greater than normal cross-modal retardation even when subjects are certain of what stimulus to expect. This study has also shown that greater than normal cross-modal retardation is not unique to schizophrenics, at least insofar as reactions to sound are concerned. Although the magnitude of sound cross-modal retardation tends to be somewhat greater for schizophrenics than for depressives, the difference is not significant. However, for reaction times to light, only schizophrenics and not depressives showed significantly greater than normal cross-modal retardation. The finding of significant schizophrenic cross-modal retardation to light in the present study and in that of Waldbaum et al. contrasts with two (36, 38) previous failures to find such an effect. It is noteworthy that the two studies yielding positive results equated sound and light stimuli for sensation level.

It is necessary to ask whether the similar sound cross-modal retardation of schizophrenics and depressives might be explained by the fact that patients were receiving comparable medications whereas normal subjects were drug free. Might antipsychotic medications provoke deviances in attentional shifting? There are four reasons why this possibility seems unlikely. First, there were no significant correlations between dosage of antischizophrenic medication and degree of attentional deviance for either patient group. Second, despite the similarity in medication status and the resemblance in schizophrenic and depressed patients' responses to sound, the two groups differed in responses to light such that only schizophrenics displayed evidence of anomalous attentional shifting. Third, all previous studies of cross-modal reaction time have tested drug-free patients. The differences in

![Fig. 3. Mean log decisecond sound reaction times and standard errors for each group on each block. The means of the distributions are connected by solid lines. Crossbars indicate 1 standard error above and 1 standard error below the mean.](image-url)
cross-modal retardation between drug-free schizophrenics and controls in these earlier studies were somewhat greater than those in the current study. Fourth, most previous studies (9, 10, 24, 42) have revealed no effect of psychotropic drugs on reaction time performance. In the few cases (3, 32) in which drug influences have been found, the effect of drugs has been to improve patient reaction time performance and to reduce rather than provoke differences between patients and normal subjects.

Deviant reaction time was not normalized by conveying expectancy information by an energy as opposed to a verbal cue. Thus, there is no evidence that anxiety over social interaction impairs patients' ability to shift attention based on correct expectancy information.

The lack of difference between verbal and machine information conditions also suggests that cross-modal retardation is influenced primarily by the sequence of responded-to stimuli. In terms of the stimuli received, all trials in the machine information condition are ipsmodal because the expectancy signal is in the same modality as the imperative signal. The correlation between the magnitude of cross-modal retardation in the verbal and machine conditions was $r(67) = .34$, $p < .01$. This finding suggests that the effect of an ipsomodal or cross-modal sequence of imperative stimuli is similar, regardless of the type of informational stimuli inserted between the imperative stimuli.

The progressive lengthening of reaction times for depressed patients has previously been reported across shorter series of trials (7, 17, 19) and suggests an impairment in sustained attention. This phenomenon warrants further investigation as a potential marker of depressive disorder.

This investigation provided no evidence that siblings of schizophrenics resemble ill probands in showing either slowed overall reaction time level or increased magnitudes of cross-modal retardation to either sound or light stimuli. Thus, there is no evidence to suggest that these performance characteristics are potential markers of vulnerability to schizophrenia.

Genetic theory predicts that only some of the siblings can be expected to be genetically vulnerable to schizophrenia, and an even smaller proportion can be expected to be at risk for the phenotypic expression of this disorder. It is therefore important to consider whether the sample of siblings was too small to reveal differences between this group and controls. If the sibling group were comprised of some individuals who are vulnerable to schizophrenia and some who are not, and if the reaction time task adequately reflected vulnerability, one would expect greater within-group variability in the performance of siblings than in that of controls. Indeed, the within-group variability of the siblings was greater than that for controls for all of the reaction time measures reported here. However, these differences were not significant.

It is apparent that the reaction time performance of these siblings of schizophrenics ranged from normal to supernormal, displaying reduced reaction time, reduced cross-modal retardation, and reduced intra-individual variability. This observation highlights a question about the most appropriate characterization of the sibling sample. Although defined as a group of individuals vulnerable to schizophrenia by virtue of genetic and environmental liabilities shared with ill probands, the most vulnerable siblings (those who fell ill) were excluded from the sample. It is possible that the resulting experimental group included a substantial proportion of "invulnerables," that is, those who have developed superior skills that maintain healthy functioning despite the liabilities that caused their siblings to fall ill. In the long run, it may be of great importance for prevention and treatment to investigate the sources of adequate performance in individuals at risk for schizophrenia.

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