The Modality Shift Effect in Schizophrenia:
Fact or Artifact?

Salvatore Mannuzza, Mitchell L. Kietzman,
Ira J. Berenhaus, Philip H. Ramsey,
Joseph Zubin, and Samuel Sutton
The Modality Shift Effect in Schizophrenia: 
Fact or Artifact?

Salvatore Mannuzza, Mitchell L. Kietzman,
Ira J. Berenhaus, Philip H. Ramsey,
Joseph Zubin, and Samuel Sutton

Received February 7, 1984; revised March 26, 1984

We investigated whether the disproportionate slowing of schizophrenic patients on cross-modal relative to ipsimodal sequences in a reaction time task ("modality shift effect") could be attributed to a psychometric artifact, as implied by Chapman and Chapman. Fifteen schizophrenic patients (Research Diagnostic Criteria) and 50 normal controls with no current or past history of psychiatric disorder were tested. Subjects made an identical finger-lift response to brief duration light and sound stimuli of different intensities presented in quasi-random order. The major finding was that psychometric artifact could not account for the disproportionate slowing of the reaction time of schizophrenic patients since the reliabilities and variances of the cross-modal and ipsimodal conditions did not differ in normal controls. Furthermore, the modality shift effect was highly significant for reaction times to both sound and light and for all intensities. Findings were the same when the schizophrenic patients were compared to a matched subgroup of slow-responding normal controls.

1This article is based on a doctoral dissertation submitted by the senior author to the Department of Psychology, City University of New York.
2Department of Psychophysiology, New York State Psychiatric Institute, New York, New York.
3Department of Psychology, Queens College of the City University of New York, Flushing, New York.
5Department of Psychiatry, University of Pittsburgh School of Medicine, and Veterans Administration Medical Center, Highland Drive, Pittsburgh, Pennsylvania.
Chapman and Chapman (1973a; 1973b; 1978) have provided a methodological critique which has been directed at many areas of psychiatric research. The Chapmans note that schizophrenic patients perform more poorly than normal controls on almost any experimental task. As a result of this poorer-than-normal or less adequate performance (the so-called schizophrenic “generalized performance deficit”), no special significance can be inferred from poorer performance on any single experimental measure. To deal with this problem, several investigators have resorted to a differential deficit methodology in which the performances of schizophrenic patients and normal controls on two tasks – an experimental and a control task – are compared. It is expected that, due to their generalized performance deficit, schizophrenic patients will perform more poorly than normal controls on both measures. The hypothesis tested in such experiments is that schizophrenic patients will be differentially affected by the experimental variable, i.e., they will show a greater performance deficit (relative to normal control subjects) on the experimental task than on the control task.

The Chapmans’ major objection to a differential deficit methodology is that unless experimental and control tasks are matched with regard to their ability to discriminate more competent from less competent normal subjects (referred to as the “discriminating power” of a task), poorer performance on the experimental task may simply reflect the psychometric characteristics of the particular tasks chosen, coupled with the tendency for schizophrenic patients as a group to perform less competently. The task yielding the greater dispersion of scores will show greater performance differences between subjects differing in general competence.

According to the Chapmans the problem can be circumvented if the two tasks are matched on reliability and variance of observed scores, the product of which yields true-score variance. Theoretically, tests have identical discriminating power if their distributions of error-free true scores are identical (Chapman and Chapman, 1978).

One approach to matching involves manipulating a variable that influences the difficulty of the tasks. If level of difficulty influences accuracy (as it typically does), both reliability and variance will be heavily influenced by its manipulation. The standardization sample used for matching the tasks should consist of a relatively large group of normal control subjects who display a wide range of competence. A substantial proportion of the standardization sample should perform as poorly as schizophrenic patients. The schizophrenic patients should not be included in the standardization sample because any true differential deficit in ability which characterizes the patients would affect the relative variance obtained for the two tasks. The equivalence of the two tasks should then be verified by recalculating reliability and variance for a subgroup of the normal standardization sample that scores at the level of the patient group.
Despite increasing awareness of the problems that the Chapmans discuss, only a few investigators (e.g., Oltmanns and Neale, 1975) have coped with these difficulties by employing appropriate research designs.

The present experiment was designed to assess whether a particular reaction time performance anomaly could be due to psychometric artifact. In the cross-modal reaction time task, visual and auditory stimuli are presented in quasi-random order, and the subject is instructed to lift his index finger from a key as quickly as possible when either stimulus is presented. In the analysis of data from a cross-modal reaction time experiment, each trial is classified in terms of the sensory modality of the stimulus of the present trial and the preceding trial. For example, a "cross-modal/light" response is a reaction time to a visual stimulus, preceded by a trial in which the stimulus was auditory; an "ipsimodal/sound" response is a reaction time to an auditory stimulus, preceded by a trial in which the stimulus was auditory.

The general finding of cross-modal reaction time experiments has been that schizophrenic patients show a greater than normal increase in reaction time for cross-modal, relative to ipsimodal, stimulus sequences, i.e., schizophrenic patients are disproportionately slowed (significantly more than normal controls) when the modality of the stimulus of the current trial is different from the modality of the stimulus of the previous trial (Spring, 1980; Sutton et al., 1961; Sutton and Zubin, 1965; Waldbaum et al., 1975). The disproportionate slowing of the reaction time of schizophrenic patients on cross-modal trials is designated here as the "modality shift effect." The interpretation of this finding has been that schizophrenic patients are impaired in their ability to shift attention from one sensory modality to another (Zubin, 1975).

Regarding the Chapmans' methodological critique, an objection to this interpretation is that the disproportionate slowing of schizophrenic patients on cross-modal trials may be explained by their generalized deficit, coupled with the greater discriminating power of the cross-modal condition. To control for this possibility, the reliability and variance of the cross-modal and ipsimodal conditions must be assessed for a relatively large group of normal control subjects. If these two conditions differ with respect to reliability or variance, or both, then a matching procedure must be instituted to equate cross-modal and ipsimodal conditions on discriminating power. In the present research, it was intended to match cross-modal and ipsimodal conditions on discriminating power by manipulating stimulus intensity, since prior experiments have shown

---

6 The interested reader should consult Nuechterlein (1977) for a review of the reaction time literature in schizophrenia, Mannuzzo (1980) for a review of the cross-modal reaction time literature, and King (1975) for a more general discussion of psychomotor assessment in impaired groups.
a systematic relationship between reaction time and intensity over a wide range of values, for both auditory and visual stimuli (King, 1962; Teichner and Krebs, 1972; Woodworth and Schlosberg, 1954).

The current research is the only cross-modal reaction time experiment in which multiple intensities of stimuli have been employed, thus allowing an assessment of the relationship between cross-modal reaction time retardation and stimulus intensity. Furthermore, the present study is the only cross-modal reaction time experiment in which a large group of carefully screened normals has been tested. Finally, this research is the first evaluation of whether the modality shift effect can be attributed to psychometric artifact.

METHOD

Subjects

The 65 subjects who participated in the present experiment were 15 male schizophrenic patients who were hospitalized at the New York State Psychiatric Institute, and 50 normal male control subjects. All patients fulfilled criteria for a definite diagnosis of schizophrenia, and all control subjects were considered to be “never mentally ill” on the basis of the Research Diagnostic Criteria (RDC: Spitzer et al., 1978).

Demographic characteristics of patient and control groups, as well as patient information regarding drug dosage and duration of hospitalization, are shown in Table I. There were no significant differences between groups in age, education, or ethnicity. All patients were receiving some form of neuroleptic (antipsychotic) medication.

Hospital charts were reviewed for all male patients in the appropriate age range who had been admitted to participating wards. Patients who appeared suitable were administered the Schedule for Affective Disorders and Schizophrenia (SADS: Endicott and Spitzer, 1978), and only patients who met RDC for definite schizophrenia were recruited for testing.7

Normal control subjects were recruited by a variety of means: contacting subjects who had participated in other experiments, making announcements at hospital staff meetings, notifying hospital personnel, etc. All prospective control subjects were administered the Schedule for Affective Disorders and Schizo-

7Of 54 patients screened, 15 (28%) were retained. The remainder were excluded for the following reasons: diagnoses of psychotic disorders other than definite schizophrenia (n = 20); substance abuse (n = 14); visual defect (n = 2); organic impairment (n = 1); presently not exhibiting psychotic symptoms (n = 1); and untestable (n = 1).
Table I. Biographical Characteristics of the Schizophrenic and Normal Control Subject Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Differencea</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patient (n = 15)</td>
<td>Control (n = 50)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>18–48</td>
<td>18–49</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>29.6</td>
<td>29.3</td>
</tr>
<tr>
<td>SD</td>
<td>8.2</td>
<td>7.8</td>
</tr>
<tr>
<td>Education (years)</td>
<td>8–16</td>
<td>9–19</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>11.9</td>
<td>12.7</td>
</tr>
<tr>
<td>SD</td>
<td>2.5</td>
<td>2.6</td>
</tr>
<tr>
<td>Ethnicity (n)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>7</td>
<td>27</td>
</tr>
<tr>
<td>Hispanic</td>
<td>5</td>
<td>13</td>
</tr>
<tr>
<td>Black</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Duration of hospitalization (days)</td>
<td>2 days–10 years</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>329.3</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>929.0</td>
<td></td>
</tr>
<tr>
<td>Daily drug dosage (mg/kg)b</td>
<td>7.3–49.9</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>19.4</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>12.8</td>
<td></td>
</tr>
</tbody>
</table>

a None of the comparisons were statistically significant.

b Daily dosages of different neuroleptic medications were translated into equivalent dosages of chlorpromazine (Thorazine) in accordance with a table presented in Davis (1976).
phrenia—Lifetime Version (SADS-L; Spitzer and Endicott, 1978). Individuals who provided evidence of either a past or a present episode of mental disorder were not included.⁸

**Apparatus and Stimuli**

The subject was seated at a table and a stimulus box was positioned directly in front of him at a distance of 16.5 inches. The front panel of the box contained a circular opening (1-inch diam.) covered by satin-finish ground glass (Edmund Scientific Company No. 2144), and surrounded by four dim red fixation lights. A response key was located directly in front of the subject, between the edge of the table and the front panel of the stimulus box.

The entire sequence of events during a block of trials (interstimulus interval, stimulus duration, reaction time registration, trial replacement, etc.) was automatically controlled by an AIM 6502 microcomputer (Rockwell International Corporation). The AIM was connected in common with two programmable attenuators. The auditory signal, a 2000-Hz sinusoid, was generated by an oscillator (Wavetek, Model 136) whose output was amplified and then passed through programmable Attenuator A (auditory). The output of the attenuator was delivered to the subject through a loudspeaker located within the stimulus box. Programmable Attenuator V (Visual) received input from a reference voltage. Following attenuation, a current source activated eight yellow (585 nm) light-emitting diodes (Monsanto No. MV 5353) located on the stimulus box and diffused by the ground glass.

Ten intensities of sound stimuli (S) and ten intensities of light stimuli (L) were used. The lowest intensity stimulus of each modality (S₁ and L₁) was set at 1.8 log units above the threshold values determined for one normal subject (discussed below). Subsequent values (S₂, S₃, ..., S₁₀ and L₂, L₃, ..., L₁₀) ranged from 1.9 to 2.7 log units above threshold, consecutive intensities differing by 0.1 log unit. All stimuli were 10 msec in duration.

The normal subject whose detection thresholds were obtained was a 21-year-old white male who had had two hearing and vision tests within a year of the testing date. Both tests showed 20/20 vision and hearing within normal limits. Neither the subject nor any of his family members had a hearing or vision problem.

First, a method of limits procedure was employed to obtain approximate estimates of the auditory and visual absolute thresholds. These provided the

---

⁸Only 4 of 54 prospective normal control subjects were rejected on the basis of the interview findings: one presented a questionable past history of affective disturbance, two fulfilled RDC for (probable) Drug Use Disorder, and one fulfilled RDC for Alcoholism.
values used in a subsequent signal-detection rating scale procedure. The signal-detection methodology provided estimates of sensitivity that are free of response bias.

**Procedure**

Testing began after 5 min of dark adaptation. The subject was instructed to lift his finger as quickly as possible whenever a light or a sound was presented. The identical finger-lift response was required on all trials, and the subject was not given any foreknowledge of the modality or intensity value of the forthcoming stimulus. There were ten blocks of 41 trials per block. Each stimulus (e.g., S6, L4, L2) was preceded by every other stimulus (including itself) once. The results of a runs test (Siegel, 1956) indicated that the sequence of light and sound stimuli within each block of trials did not differ from chance ($z = -0.16$).

The timing of events during a block of trials was independent of the subject’s response: i.e., the subject’s speed of response had no effect on interstimulus interval, and each trial was cycled automatically, with no warning signal and no subject initiation required. The interval between stimuli varied randomly between 2.0 sec and 3.5 sec.

Reaction times less than 100 msec or greater than 1 sec were not included in the analyses. These trials were replaced to assure that an equal number of cross-modal and ipsimodal light and sound sequences was maintained for all subjects.

**Data Analyses**

All reaction times were transformed to common (base 10) logarithmic equivalents in an effort to reduce skewness and to correct for the heterogeneity of variance often found in patient–normal comparisons of reaction time performance (King, 1954). Analyses were conducted on the geometric mean reaction times (i.e., the antilog of the mean of the logs) of each intensity and condition. Separate analyses were conducted for reaction times to light and sound stimuli. For the analyses of variance, the epsilon correction (Jennings and Woods, 1976) was used since repeated-measures designs may entail inflated degrees of freedom.

The following phases of analyses were planned. First, confirm that a significant interaction of subject group (patient, control) x modality shift (cross-modal, ipsimodal) is found. This should show a disproportionate slowing for schizophrenic patients on cross-modal relative to ipsimodal trials. Next, assess the discriminating power of the cross-modal and ipsimodal conditions for the 50 control subjects by comparing the reliabilities and variances. If estimates of discriminating power differed between conditions, implement a trial-deletion procedure in which reaction times to certain stimulus intensities are systemati-
cally deleted until a close match on reliability and variance is achieved. After determining whether estimates of discriminating power are comparable for cross-modal and ipsimodal conditions, assess whether patient/normal differences are retained for the total samples (or use a matching procedure if they are not). Next, conduct a subsequent analysis in which the patients are compared with a subgroup of normals who perform in the patient range. Finally, evaluate whether a disproportionate slowing for schizophrenic patients is found compared to the slow normals.

RESULTS

Initial Assessment of Cross-Modal Reaction Time Retardation Differences

Group geometric mean reaction times for each intensity value and modality shift condition are shown in Fig. 1 (light) and Fig. 2 (sound). By inspection it may be noted that (i) for both light and sound stimuli, both cross-modal and ipsimodal conditions, and both subject groups, reaction time tended to decrease as a function of increasing intensity (1 through 10); (ii) For both light and sound stimuli, for all intensity values, and both cross-modal and ipsimodal conditions, the patient group responded more slowly than the control group; (iii) For both light and sound stimuli, at all intensity values, the patient group

![Fig. 1](image-url)  
**Fig. 1.** Reaction times of patient and control groups to light stimuli, as a function of stimulus sequence (I = ipsimodal, C = cross-modal) and intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 50 normal controls.
Fig. 2. Reaction times of patient and control groups to sound stimuli, as a function of stimulus sequence (I = ipsimodal, C = cross-modal) and intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 50 normal controls.

responded more slowly on cross-modal trials than on ipsimodal trials. A similar tendency can be seen for the normal controls, but the magnitude of the difference between cross-modal and ipsimodal conditions is much smaller.

**Confirmation of the Modality Shift Effect**
**for Schizophrenic Patients**

Data were analyzed by a three-factor mixed-model analysis of variance with subject group (patient, control) as a between factor, and modality shift (cross-modal, ipsimodal) and intensity (1 through 10) as repeated-measures factors. For both light and sound stimuli analyzed separately, significant main effects were found for subject group (light: \( F(1, 63) = 38.49, p < 0.001 \); sound: \( F(1, 63) = 33.00, p < 0.001 \)), modality shift (light: \( F(1, 63) = 37.72, p < 0.001 \); sound: \( F(1, 63) = 47.37, p < 0.001 \)), and intensity (light: \( F(9, 567) = 50.38, p < 0.001 \); sound: \( F(9, 567) = 19.03, p < 0.001 \)). Also, a significant interaction was found for subject group \( \times \) modality shift (light: \( F(1, 63) = 28.50, p < 0.001 \); sound: \( F(1, 63) = 31.16, p < 0.001 \)).

The subject group \( \times \) modality shift interaction is displayed in Fig. 3. For both light and sound stimuli, it is clear that the patient group was disproportionately slowed on cross-modal trials. The magnitude of cross-modal reaction time retardation for light was 85 msec for the patient group as compared to 6 msec for the control group; cross-modal reaction time retardation
for sound was 87 msec for the patient group as compared to 9 msec for the control group. Within-group analyses were also performed. For reactions to both light and sound stimuli, the patient group was significantly slower on cross-modal trials than on ipsimodal trials (light: \( t(14) = 4.91, p < 0.001 \); sound: \( t(14) = 6.32, p < 0.001 \)), whereas the control group was not significantly slower as a function of these conditions (light: \( t(49) = 0.96, \text{ns} \); sound: \( t(49) = 1.37, \text{ns} \)).

**Modality Shift Comparisons Within and Across Intensities**

To assess whether the modality shift effect was consistent across intensities, the data for each intensity value were treated as if they constituted a separate experiment. A two-factor mixed-model analysis of variance, with subject group as a between factor and modality shift as a repeated-measure factor, was conducted for each intensity value. For both light and sound stimuli, all intensity values showed significant main effects (\( p < 0.01 \)) for group and modality shift, and a significant interaction (\( p < 0.01 \)) for group \( \times \) modality shift. Thus, the modality shift effect for schizophrenic patients was found regardless of modality or intensity of the responded-to stimulus.

A one-factor repeated-measures analysis of variance, with intensity as the repeated-measure factor, was conducted on the intrasubject cross-modal/ipsimodal difference scores separately for each group and modality. None of these analyses reached statistical significance: patients/light, \( F(9, 126) = 1.84 \); patients/sound, \( F(9, 126) = 0.81 \); controls/light, \( F(9, 441) = 1.36 \); controls/sound, \( F(9, 441) = 1.73 \). In summary, the magnitude of cross-modal reaction time retardation did not differ as a function of intensity of light or sound stimuli for either the schizophrenic patients or the normal controls.
The availability of ten intensities within each sensory modality permitted an analysis of reaction time as a function of intensity. Trend analyses were done on the ipsimodal reaction times separately by group for light and sound reaction times. A significant linear trend as a function of intensity was found for patients and normals for both light and sound stimuli (patients: light, \( F(1, 14) = 28.68, p < 0.001 \); sound \( F(1, 14) = 10.30, p < 0.01 \); controls: light, \( F(1, 49) = 121.22, p < 0.001 \); sound, \( F(1, 49) = 45.34, p < 0.001 \).

**Cross-Modal Reaction Time Retardation and Patient Variables**

Pearson product-moment correlations between the magnitude of cross-modal reaction time retardation in relation to drug dosage, number of previous hospitalizations, and duration of hospitalization were calculated. None were significant.

**Assessment of Discriminating Power**

Coefficient alpha, an estimate of reliability (Anastasi, 1982), was calculated for the cross-modal and ipsimodal conditions of the normal controls. The results of these analyses showed that all alpha coefficients were identical: alpha = 0.99 for both cross-modal and ipsimodal conditions in both sensory modalities.

A \( t \) test for the difference between correlated variances of normal controls (Guilford, 1965) was then conducted separately for each sensory modality. Results indicated that there were no significant differences between the variances of the cross-modal and ipsimodal conditions for reactions to light (\( t(48) = 1.02 \)) or to sound (\( t(48) = 1.62 \)).

In conclusion, when the data from all ten intensities for the control subjects are analyzed, cross-modal and ipsimodal conditions were found to be already matched on reliability and variance (for both modalities). However, as Chapman and Chapman have noted, matching on these variables must also be demonstrated for a subgroup of normal control subjects who score in the patient range. This procedure assures that the “tests” possess equivalent discriminating power at different levels of “competency.”

The 15 normal control subjects who exhibited the slowest average geometric mean ipsimodal reaction times in each sensory modality were chosen to constitute the two slow normal subgroups, one for each sensory modality. Results of \( t \) tests confirmed that the patient group and these slow normal subgroups did not differ significantly in the ipsimodal condition (light: \( t(28) = \)}.
0.47; sound; \( t(28) = 0.79 \). There were no significant differences between the patient group and either slow normal subgroup on age, education, or ethnicity.

Coefficient alphas were calculated from the reaction time results for the cross-modal and ipsimodal conditions of each slow normal subgroup. For reactions to both light and sound, alphas were high and differences between alphas were small: light/cross-modal, alpha = 0.98; light/ipsimodal, alpha = 0.99; sound/cross-modal, alpha = 0.98; sound/ipsimodal, alpha = 0.99.

A \( t \) test for the difference between correlated variances was then conducted separately for each modality. Results indicated that there were no significant differences between the variances of cross-modal and ipsimodal conditions for either light \( (t(13) = 0.65) \) or sound \( (t(13) = 1.07) \) analyses. Thus the equivalence of the two tasks under examination was demonstrated.

**Comparison of Patients and Slow Normals on Cross-Modal Reaction Time Retardation**

Data were analyzed by a three-factor mixed-model analysis of variance with subject group (patient, slow normal) as a between factor, and modality shift (cross-modal, ipsimodal) and intensity (1 through 10) as repeated-measures factors. For both the light and sound analyses, a significant interaction was found for subject group \( \times \) modality shift (light: \( F(1, 28) = 11.76, p < 0.002; \) sound: \( F(1, 28) = 13.39, p < 0.001 \). As shown in Fig. 4, patients, as compared to slow normals, were disproportionately retarded on cross-modal trials. Within-group comparisons showed that there was no significant difference between cross-modal and ipsimodal reaction times for both slow normal subgroups, light: \( t(14) = 0.35; \) sound; \( t(14) = 0.19 \).

**DISCUSSION**

The major finding of the current research was that the disproportionate reaction time slowing of schizophrenic patients due to shift of sensory modality
could not be attributed to psychometric artifact. After the modality shift effect was confirmed by a significant subject group × modality shift interaction, the reliabilities and variances of the cross-modal and ipsimodal conditions (for the normal control subjects and for the two slow normal subgroups) were shown to be comparable. Thus, the disproportionate slowing of patients on cross-modal trials could not be explained by differences in discriminating power between cross-modal and ipsimodal conditions coupled with the schizophrenic patients' generalized performance deficit.

Several additional factors support this conclusion. First, the disproportionate slowing of schizophrenic patients was consistent across modalities: for reactions to both light and sound stimuli, the subject group × modality shift interaction was significant at the 0.001 level. In addition, within-intensity patient—control comparisons showed a disproportionate slowing at all intensity values for reactions to light and sound. Furthermore, the disproportionate slowing was still found when patients were compared to slow normals.

One advantage of using the ten intensity levels, a strategy used here for the first time in cross-modality research, was that it permitted some assessment of the patients' engagement in the task. For example, if the reaction times of patients were characterized by high variability, then a systematic relationship between intensity and reaction time would not be expected. The finding that the reaction times of patients (as well as those of control subjects) decreased as a function of increasing intensity suggests that patients were engaged in the task.

Consistent with other cross-modal reaction time experiments in which medicated patients were tested (Rist and Thurin, 1982; Spring, 1980), results of the current research showed that the correlation between drug dosage and cross-modal reaction time retardation was not significant for either sensory modality. These findings, together with the fact that the effect was demonstrated in drug-free patients in five earlier studies (Sutton et al., 1961; Sutton and Zubin, 1965, Experiments A, B, and C; Waldbaum et al., 1975), provide compelling evidence that psychotropic medication cannot account for the disproportionate reaction time retardation of schizophrenic patients.

The average magnitudes of cross-modal reaction time retardation for the schizophrenic patients in the current study (light, 85 msec; sound, 87 msec) were much larger than those found in prior studies (10 msec to 30 msec). Schizophrenic patients tend to show larger cross-modal reaction time retardation when they are not forewarned of the modality of the forthcoming stimulus (Waldbaum et al., 1975), and in the present study this may explain their greater magnitude of cross-modal slowing. Here, the subjects were uncertain about both the modality and intensity of the forthcoming stimulus. The present research included the additional factor of not only not knowing the modality of the forthcoming stimulus, but also not knowing the intensity of the stimulus that would be presented on each trial, i.e., subjects did not know which of ten intensities, in addition to not knowing which of two sensory modalities (a light or a sound), would be presented. Perhaps magnitude of cross-modal reaction
time retardation is related to the degree of uncertainty about the stimulus that is to be presented.

Two points about this hypothesis are worth noting: First, for normal control subjects, there appears to be no apparent relationship between degree of uncertainty and cross-modal slowing. In the current research, as in prior cross-modal reaction time experiments, mean cross-modal/ipsimodal difference scores for the control group have ranged from 0 msec to 9 msec. Thus the influence of uncertainty on cross-modal reaction time retardation seems to be limited to the patient group. Second, uncertainty alone cannot account for the total modality shift difference between patients and controls, since Waldbaum et al. (1975) and Spring (1980) also found greater cross-modal slowing for patients than controls when subjects were informed prior to each trial which stimulus would be presented.

One might speculate why a higher degree of uncertainty might result in a greater motor retardation in patients. Perhaps increased uncertainty for the patients results in increased arousal, which in turn exacerbates their attentional shift deficit. Note that this proposal implies that degree of arousal (or some other mechanism), rather than degree of uncertainty, is a key factor in increasing cross-modal slowing in patients. Thus, for example, if schizophrenic patients were administered a central nervous system stimulant, cross-modal reaction time retardation should increase, regardless of whether the forthcoming stimuli were known. Additional experiments could provide insight into these issues and, in turn, should advance our knowledge of the attentional deficit in schizophrenia.

ACKNOWLEDGMENTS

The authors would like to thank Drs. Rue L. Cromwell and Gad Hakerem for their helpful comments, Mr. Robert Laupheimer and Mr. Ben Djoletto for the construction and maintenance of the equipment, and Mr. Marvin Nalick and Mr. Michael Gelsomino for their artwork and photography of the figures. This research would not have been possible without the cooperation of Drs. Stanley Bone and Gregory Asnis, the psychiatrists in charge of the two participating wards of the New York State Psychiatric Institute.

REFERENCES

Modality Shift Effect in Schizophrenia


